AFIP Wednesday Slide Conference 1981-1982

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Conf. #	Case #	Date of Conference	Contrib. #	AFIP #	Contributor	Species	Organ	Diagnosis
1	I	16 Sept 81	71773	1802132	Bureau of Biologics, FDA	Marmoset	Small intestine, tongue	Intestinal pyogranulomas due to *Prosthenorchis sp.* Lingual candidiasis and sarcosporidiosis.
	II	"	D80-609	1804671	Univ of Illinois	Dog	Uterus	Normal placental site involution.
	III	"	80-1498-1	1804131	Univ of Tennessee	Dog	Perineum	Cystadenocarcinoma with osseous metaplasia, origin undetermined.
	IV	"	81-179-4 (X70)	1802811	Kansas State Univ.	Cat	Spinal cord	Syringomyelia.
2	I	23 Sept 81	81-1131	1804668	WRAIR	Rabbit	Kidney	Acute infarcts due to thrombi & bacterial emboli. Glomerular amyloidosis.
	II	"	CP77-1189	1802806	Southwestern Medical School	Guinea pig	Lung	Lipid pneumonia.
	III	"	DN-18213	1803087	Experimental Path. Labs.	Equine	Liver	Fetal hepatic necrosis due to equine herpesvirus (rhinopneumonitis abortion).
	IV	"	3-18	1795804	Bio/dynamics	Rat	Ovary	Gonadostromal tumor, Sertoli cell pattern.

3	I	30 Sept 81	81-768	1804140	Univ of Tennessee	Horse	Liver	Serum hepatitis.
	II	"	81-0133	1803021	Univ of Arizona	Dog	Lung	Barium sulfate foreign body pneumonia.
	III	"	804	1802812	C.E. Kord Animal Disease Lab	Dog	Small intestine	Coronavirus enteritis.
	IV	"	R28186	1804128	Bushy Run Research Center	Rat	Kidney	Carcinoma, solid, renal clear cell type, with osseous metaplasia.
4	I	7 Oct 81	80-479-3	1803359	Regional Vet Lab Australia	Pony	Liver, spleen, kidney	Hemoglobinuric nephrosis & and parasitemia due to Babesia equi.
	II	"	79-D-32	1802130	Penn State Univ.	Guinea pig	Liver	Pyogranuloma due to Staphylococcus aureus.
	III	"	S2298-80	1803012	Animal Med. Ctr.	Dog	Tongue	Oral eosinophilic granuloma of Siberian huskies.
	IV	"	08-010	1758399	Hazleton Labs	Dog	Abdominal cavity	Mesothelioma, abdominal cavity.
5	I	14 Oct 81	81P539	1803328	Colorado State Univ.	Foal	Thymus, spleen, lymph nodes	Combined immunodeficiency of horses.
	II	"	79-0025	1713952	US Army Biomedical Lab	Dog	Cerebellum & midbrain	Meningoencephalitis due to canine distemper.
	III	"	91284	1802815	L.A. County Dept of Health	Dog	Kidney	Glomerular thrombosis, nephrosis, & hemorrhage due to rattlesnake envenomation.
	IV	"	1	1804149	Ethicon Research Foundation	Rat	Site unspecified	Fascial granulomas due to surgical sponge implant.

6	I	21 Oct 81	1-ARB-004	1806689	Bureau of Vet Med, FDA	Dog	Liver	Glucocorticoid induced hepatopathy.
	II	"	80B600 & 801338	1803538	Univ. of Calif.	Bird	Kidney	Renal adenovirus infection of lovebirds.
	III	"	227648-6	1804249	Mich. State Univ.	Pheasant	Spleen	Marble spleen disease.
	IV	"	21-210	1803540	Okla. State Univ.	Ox	Colon	Coccidiosis due to Eimeria zurnii.
7	I	28 Oct 81	8767 (H&E)	1795815	Univ. of Alabama	Guinea pig	Lung, lymph node	Interstitial pneumonia due to guinea pig cytomegalovirus.
	II	"	81-360	1803321	Univ. of Texas	Cat	Kidney, perirenal fat	Lymphosarcoma due to FeLV liposarcoma possibly due to FeSV.
	III	"	81-942 (HPS stain)	1804672	Univ. of Montreal	Dog	Meninges, spinal cord	Necrotizing vasculitis and pyogranulomatous meningitis.
	IV	"	-	1801670	USUHS	Rat	Kidney	Gold nephropathy.
8	I	4 Nov 81	79-2	1710128	AFRRI	Dog	Coronary arteries	Atherosclerosis, coronary arteries.
	II	"	81-1	1562991	G. A. Parker	Puppy	Spinal cord	Globoid cell leukodystrophy.
	III	"	80-12522	1803333	Univ. of Kentucky	Bovine fetus	Brain	Encephalitis due to Sarcocystis bovicanis.
	IV	"	A80-230	1801474	Angell Mem. Anim. Hosp.	Dog	Kidney	Renal telangiectasia in Welsh Pembroke Corgi.

9	I	18 Nov 81	468	1803076	New Mexico Vet. Diag. Services	Cow	Mammary gland	Mastitis due to Nocardia asteroides.
	II	"	H81-49	1806827	Animal Industry Res. Inst., Taiwan	Pig	Lung, lymph node	Toxoplasmosis with hog cholera and Salmonellosis.
	III	"	N81-3252	1811026	Univ. of Saskatchewan	Horse	Small intestine	Enteritis due to M. avium.
	IV	"	C9-976	1734039	Anim. Hlth. Lab.	Feline	Lung	Pulmonary paragonomiasis.
0	I	2 Dec 81	N81-484	1802991	Univ. of Florida	Dog	Heart	Myocardial aspergillosis.
	II	"	30696-27	1811146	LAIR	Monkey	Lung	Pulmonary infarcts with thrombosis.
	III	"	19-2633	1802915	Lovelace Res Inst	Rat	Liver, spleen	Mononuclear cell leukemia of Fischer 344 rats.
	IV	"	80-3574	1757299	Life Sciences Res.	Mouse	Liver	Hepatitis due to Pseudomonas aeruginosa.
1	I	9 Dec 81	2553-1	1803314	Univ. of Nebraska	Puppy	Lung Liver	Canine distemper. Tyzzer's disease.
	II	"	9130-81	1803023	Univ. of Missouri	Dog	Liver	Subacute hepatitis, due to ICH.
	III	"	AB1651 or AB1758 or AB1534	1806696	ToxiGenics, Inc.	Guinea pig	Heart	Rhabdomyomatosis.
	IV	"	M20428	1810447	NCI, FCRC	Mouse	Spleen	Fibrosarcoma, due to Type C sarcoma virus.

12	I	16 Dec 81	2732	1803531	Syntex Research	Cow	Intermandibular soft tissue	Actinobacillosis.
	II	"	81-3083	1804673	Univ. of Montreal	Cow	Lung	Septic emboli due to Fusobacterium necrophorum.
	III	"	1791887	1791887	Sea World, San Diego	Duck	Liver	Schistosomiasis and amyloidosis.
	IV	"	81-14458	1810707	South Dakota State University	Pig	Liver & lung	Necrotizing and granulomamatous hepatitis due to Salmonella cholerasuis.
13	I	6 Jan 82	NB1-54-9	1802809	Univ. of Georgia	Dog	Colon	Granulomatous colitis due to Histoplasma capsulatum.
	II	" "	1971-E,H or 1981-I	1809912	USAF Aerospace Med. Res. Lab.	Baboon	Liver	Hepatocystis simiae, merocyst.
	III	"	81-166 (194-A)	1801534	Murdoch Univ.	Dog	Stomach	Hypertrophic gastritis.
	IV	"	S-10210	1811156	Merck Institute	Cat	Nasal cavity	Transitional carcinoma.
14	I	13 Jan 82	8721, 8605	1802979	Univ. of Alabama	Mice	Lung, lymph node	Peribronchiolar pneumonia Mycobacteria avium.
	II	"	076 412 076 464	1810369	Natl. Center for Toxicol. Res.	Mice	Kidney	Proliferative glomerulitis. Mouse mammary tumor virus.
	III	"	BG-1	1810710	Bowman Gray School of Medicine	Macaque	Adrenal gland	Adrenal microabscesses. Listeria monocytogenes.
	IV	"	A-36534	1810442	Natl. Cancer Inst.	Mouse	Harderian gland	Metastatic Harderian gland carcinoma to lung.

15	I	20 Jan 82	SPA-086	1802990	University of Pittsburgh	Pig	Skin	Pityriasis rosea.
	II	"	U2070 or W1475	1801671	Ohio State Univ.	Horse	Ethmoid region	Hemorrhagic nasal polyp.
	III	"	718-13	1812876	University of Maryland	Cat	Mammary gland	Total fibroadenomatous change.
	IV	"	79-824-1 or 79-824-3	1801674	Kansas State Univ.	Calf	Cecum	Typhlitis due to Trichuris discolor.
16	I	27 Jan 82	80N2051	1760315	Louisiana State U.	Alligator	Colonic serosa	Egg yolk peritonitis.
	II	"	D81-10131	1809929	Univ. of Minnesota	Mink	Kidney, spleen	Aleutian disease.
	III	"	C-80-01	1757301	Eastman Kodak Co.	Pigeon	Lung	Parasitemia due to Hemoproteus sp.
	IV	"	634	1803357	Hoeschst-Roussel Pharmaceuticals	Cat	Anterior mediastinum	Thymoma.
17	I	3 Feb 82	1803985	1803985	Ben White Animal Hospital	Dog	Eyelid	Chalazion.
	II	"	80-566	1803320	Nat'l Animal Disease Center	Dog	Eye	Hereditary microphthalmia and retinal dysplasia.
	III	"	1725431	1725431	Univ. of Illinois	Cat	Eye	Panophthalmitis due to FIP.
	IV	"	5255-80	1803062	Oregon State Univ.	Cat	Eye	Retinal degeneration due to taurine deficiency.

18	I	10 Feb 82	X-140MRI	1803093	Midwest Res. Inst.	Rabbit	Kidney	Tubulointerstitial nephritis due to Encephalitozoon cuniculi.
	II	"	527-1	1803312	Univ. of Nebraska	Cow	Liver, lung	Bronchopneumonia and hepatitis due to Brucella abortus.
	III	"	236618	1804247	Michigan State University	Equine	Brain	Eastern equine encephalomyelitis.
	IV	"	89748	1802813	L.A. County Dept of Health	Dog	Brain	Glioblastoma multiforme.
19	I	17 Feb 82	23447	1810441	Bureau of Foods, FDA	Dog	Spleen, colon	Lymphocytic depletion and colitis due to parvovirus infection.
	II	"	AFRRI #1	1782726	AFRRI	Monkey	Lumbar area	Parasitic nodule due to Edesonfilaria malayensis.
	III	"	Sp81-364 (H364-81A)	1816641	Univ. of Liverpool	Dog	Salivary gland	Necrotizing sialoadenitis.
	IV	"	A163717	1803526	Vet Reference Lab, Salt Lake City	Cat	Skin	Dermatitis due to Cryptococcus neoformans.
20	I	24 Feb 82	1-1190-81	1808316	Montana Veterinary Diagnostic Lab.	Steer	Heart	Myocardial degeneration due to monensin toxicity.
	II	"	FIA-2	1809915	Univ of Minnesota	Cat	Pancreas	Islet cell vacuolar change and insular amyloidosis.
	III	"	79-5396	1803529	Vet Diagnostic & Investigational Lab	Pig	Spinal cord	Poliomyelomalacia due to selenium toxicity.
	IV	"	Mk 81-853	1822342	Comp Path, NIH	Monkey	Aorta	Arteriosclerosis and aortitis.

21	I	17 Mar 82	1458-T9	1796386	Mobil Oil Corp.	Guinea pig	Heart	Basophilic (mucoid) myocardial degeneration.
	II	"	X13765	1803456	Eli Lilly Co.	Crane	Spleen, liver, lung, small intestine	Disseminated visceral coccidiosis due to Eimeria reichenowi.
	III	"	2702	1815112	Vet. Services Lab., Guelph	Gilt	Lung	Contagious porcine pleuropneumonia due to Hemophilus parahemolyticus.
	IV	"	3775	1805528	Univ. of Wisconsin	Dog	Ear	Ceruminous gland carcinoma.
22	I	24 Mar 82	80-5389	1805537	British Columbia Ministry Agri/Food	Goat	Brain	Caprine viral leukoencephalomyelitis.
	II	"	E-129-11	1827810	Upjohn Company	Dog	Liver, lymph node	Familial erythrocytic pyruvate kinase deficiency.
	III	"	82-02	1824528	AFIP & Onderstepoort Res. Lab.	Ewe	Lung	Pulmonary adenomatosis (Jaagsiekte).
	IV	"	16776	1803019	Johns Hopkins Un.	Cat	Skin	Mast cell tumor.
23	I	31 Mar 82	79695 or 79954	1776776	National Animal Disease Center	Chicken	Tibia	Hypovitaminosis D (Rickets).
	II	"	1800901	1800901	Crofton Animal Hospital	Guinea pig	Hind leg	Hypovitaminosis C (Scurvy). Scorbutic arthropathy.
	III	"	80-2268	1801675	Ohio State Univ.	Equine	Nasal septum	Fibrocartilagenous dysplasia.
	IV	"	N81-1130	1810706	University of Saskatchewan	Pig	Forelimb	Congenital hyperostosis (Diaphyseal dysplasia).

24	I	7 April 82	P-3916-79	1822362	Purdue University	Sow	Brain and meninges	Harding's cerebrospinal angiopathy.
	II	"	72848	1802135	Texas A&M Univ.	Dog	Lymph node	Lymphadenitis due to Phialophora sp.
	III	"	80-164	1806757	Pfizer Research	Rat	Liver	Hepatitis due to Kilham rat virus.
	IV	"	879	1802946	C.E.Kord Animal Dis. Diag. Lab.	Dog	Brain	Granular cell tumor.
25	I	14 April 82	M20324	1810449	FCRC, NCI	Monkey	Uterus, colon	Choriocarcinoma (colon). Stromal cell hyperplasia & epithelioid cytomorphosis (uterus).
	II	"	Mk81-879-AA	1822617	Comp. Path., NIH	Monkey	Kidney	Hemoglobinuric nephrosis & parasitemia due to Plasmodium knowlesi.
	III	"	81-38	1803081	NMRI, NNMC	Baboon	Kidney	Oxalate nephrosis.
	IV	"	SWRI2A72B	1802985	Sterling Winthrop Res. Inst.	Monkey	Uterus, ileum, colon	Endometriosis & fibro-leiomyoma (uterus only).
26	I	21 April 82	47540	1815104	Vet. Services Lab, Guelph	Equine	Skin	Molluscum contagiosum-like dermatitis.
	II	"	81-1238B	1737277	AFIP & Onderste-Vet. Res. Lab.	Bovine	Ear, esopha-gus, larynx	Sweating sickness.
	III	"	80-508	1805677	Univ. of Penn. New Bolton Center	Cow	Skin	Dermatitis due to Demodex bovis.
	IV	"	1481K	1619250	University of Pennsylvania	Dog	Skin	Malignant hair follicle tumor.

27	I	28 April 82	AFRRI #2	1791669	Comp. Path., AFRRI	Parrot	Liver	Avian chlamydiosis.
	II	"	80443	1803337	Nat'l. Animal Disease Center	Equine	Vagina & cervix	Contagious equine metritis.
	III	"	81-1349	1805545	Vet. Diag. Lab., Auburn, Alabama	Canine	Heart	Vasculitis & myocarditis due to encephalitozoonosis.
	IV	"	29935	1827311	USAMRIID	Snake	Subcutis	Malignant chromatophoroma.
28	I	12 May 82	N80-736	1802918	USAF/SAM, Brooks AFB, TX	Aardwolf	Heart	Chronic-active myocarditis due to Trypanosoma cruzi.
	II	"	BZ65-81	1803009	Animal Med. Ctr., New York	Nyala (antelope)	Heart	Myxomatous change of fat & interstitial fibrosis.
	III	"	6621 I&II	1830594	Zoological Park of East Berlin	Antelope	Liver, lymph node	Toxoplasmosis.
	IV	"	X1756	1803705	National Zoological Park	Sea lion	Skin	Dermatitis due to Fusarium sp.
29	I	19 May 82	81-775	1822536	Center for Disease Control	Armadillo	Kidney	Pyogranulomatous nephritis due to Sporothrix schenckii.
	II	"	80-9625	1801295	Washington State University	Cat	Stomach	Ollulanus tricuspis.
	III	"	81-001	1822361	Dow Chemical Co.	Hamster	Heart	Dissecting aortic aneurysm.
	IV	"	80F1886/ T24-A & B	1747664	North Carolina State University	Feline	Trachea, kidney	Tracheal epithelial carcinoma with renal metastasis.

30	I	26 May 82	AM-7530-82	1822364	Purdue University	Mynah	Liver, kidney	Iron storage hepatopathy (hemosiderosis).
	II	"	HPS 3754, 3755, 3758	1824529	10th Med. Lab.	Mouse	Lung, lymph nodes, abdominal organs	Pyogranulomas due to Coccidioides immitis.
	III	"	80-1942-5	1805542	Dow Chemical Co.	Canine	Muscle	Necrotizing and granulomatous myositis due to Toxoplasma gondii.
	IV	"	P80-031	1803517	Proctor & Gamble	Mouse	Lung, kidney	Clara cell necrosis and nephrosis due to the toxic intermediate of ethylcarbamoylfuran.

Results
AFIP Wednesday Slide Conference - No. 1
16 September 1981

Conference Moderator: John D. Toft II, DVM, MS
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Case I - 71773 (AFIP 1802132):

History: An adult Mystax marmoset (Saguinus mystax), one of shipment newly arrived from South America. This animal was not eating well and was found dead in the cage.

Contributor's Diagnosis & Comments: Multiple parasitism is still common in imported marmosets. Mycotic organisms compatible with Candida are present on the tongue surface. Sarcosporidia in the muscle of the tongue is a frequent finding with questionable clinical significance. Prosthenorchis elegans is frequently found in the ileum and occasionally will penetrate the intestinal wall with resulting peritonitis that is usually fatal.

AFIP Diagnosis: 1) Pyogranulomas, multifocal, severe, serosa and muscular tunics, small intestine, marmoset, nonhuman primate, (Saguinus mystax) - etiology compatible with acanthocephaliasis due to Prosthenorchus sp. 2) Glossitis, exudative and ulcerative, chronic with multifocal intraepithelial microabscess formation and numerous hyphal and yeast forms, moderate to severe, epithelium, tongue, - etiology compatible with Candida sp. 3) Sarcosporidiosis, multifocal, skeletal muscle, tongue.

Conference Note: Acanthocephalid eggs are present in some sections. Several participants observed microfilaria within small intestinal blood vessels compatible with the genus Dipetalonema.

Contributor: Bureau of Biologics, FDA, Bethesda, MD 20014.

References:

1. Bullock, B. C. et al.: New World Monkeys. In: Primates in Medicine, Vol. 2, 1969, pp 62-74.
2. Benirschke, K.: Diagnostic Exercise. Lab. Anim. Sci. 29 (1): 33-34, 1979.
3. Deinhardt, J. B. et al.: Marmosets as laboratory animals. Lab. Anim. Care 17 (1): 11-29, 1967.
4. Karr, S. L. et al.: A survey of Sarcocystis in nonhuman primates. Lab. Anim. Sci. 25 (5): 641-645, 1975.
5. Moore, J. G.: Epizootic of acanthocephaliasis among primates. JAVMA 157 (5): 699-705, 1970.
6. Takos, M. J. et al.: The pathology and pathogenesis of fatal infections due to an acanthocephalid parasite of marmoset monkeys. Am. J. Trop. Med. & Hyg. 7: 90-94, 1958.

Case II - D80-609 (AFIP 1804671):

History: A mature mix breed bitch used for teaching purposes at the Veterinary College had a mucopurulent vaginal discharge. Systemic signs were absent. The clinical diagnosis was pyometra. Surgery revealed numerous regularly spaced nodular thickenings in both uterine horns.

Contributor's Diagnosis & Comments: 1) Subinvolution of placental sites. 2) Cystic endometrial hyperplasia.
Comment: Subinvolution of placental sites may not be present in some specimens.

AFIP Diagnosis: 1) Endometritis, suppurative, diffuse, mild, uterus, mixed breed, canine. 2) Involution of placental attachment site characterized by multifocal fibrosis, hemorrhage, persistence of trophoblasts and dilated endometrial glands, endometrium, uterus.

Conference Note: Further history provided by the contributor at a later date indicated that the bitch was between two and three weeks postpartum when the surgery was performed. In view of this additional information most participants were of the opinion that the involution process was within normal limits.

Contributor: Department of Veterinary Pathobiology and Veterinary Diagnostic Medicine, University of Illinois, Urbana, IL: 61801.

References:

1. Al-Bassam, M. A. et al.: Involution abnormalities in the postpartum uterus of the bitch. Vet. Pathol. 18: 208-218, 1981.
2. Beck, A. M., McEntee, K.: Subinvolution of placental sites in the postpartum bitch. A case report. Cornell Vet. 56: 269-277, 1966.
3. Glenn, B. L.: Subinvolution of placental sites in the bitch. 18th Gaines Veterinary Symposium. Oct 1968, 7-10.
4. Schall, W. D. et al.: Spontaneous recovery after subinvolution of placental sites in a bitch. JAVMA 159: 1780-1782, 1971.

Case III - 80-1498-1 (AFIP 1804131):

History: A 10-year-old male miniature poodle had a right sided perineal hernia containing a 2.5 cm diameter subcutaneous bony cyst. Cyst content was clear reddish tinged watery fluid. The specimen was decalcified prior to being embedded in paraffin.

Contributor's Diagnosis & Comments: Mixed apocrine gland carcinoma.
Comments: Most of the cyst wall is comprised of stromal elements including reactive bone, necrotic bone and basophilic myxomatous connective tissue. Epithelium varying from squamous to low columnar lines portions of the cyst and forms irregular luminal structures within its wall (stromal invasion). In some areas cuboidal cells are piled up with loss of polarity. Mitotic figures are scant. The tumor appears to have been adequately excised. There was no recurrence 5 months later.

AFIP Diagnosis: Cystadenocarcinoma with osseous metaplasia, subcutis, perineum, poodle, canine.

Conference Note: Further history provided by the contributor at a later date indicated that a perineal hernia was also observed at the time of surgery. Only the rectum, however, was involved and it remained intact. The tumor shelled out easily.

Although some participants concurred with the contributor, most were of the opinion that the spindle cells present were not of myoepithelial origin, and that the bone was metaplastic. The inner-most accumulations of hyalinized collagenous tissue that are essentially acellular contained viable epithelial cells and stained intensely blue with the Masson Trichrome method. The AFIP Department of Orthopedic Pathology considered this material to be a specific type of osteoid often described as having a "cotton candy" appearance. It is commonly encountered in human cystic bone lesions such as bone cysts[1], cystic giant cell tumors, cystic osteoblastomas, and chondroblastomas. Some authors advocate its origin from fibrin coagulum becoming calcified and serving as a scaffold for ossification[1]. However, other possibilities include osteoid produced in a relatively ischemic environment or production by the epithelial cells themselves as has been documented in transitional cell carcinomas, melanomas, and rare skin tumors. We have also seen bone formation associated with keratin debris in intracutenaous cornifying epitheliomas and necrotizing and calcifying epitheliomas (pilomatrixoma, Malherbe's tumor).

Contributor: Department of Pathobiology, College of Veterinary Medicine, University of Tennessee, Knoxville, TN 37901.

References:

1. Sanerkin, N. G.: Old fibrin coagula and their ossification in simple bone cysts. The Jrnl of Bone & Joint Surgery 61-B (2): 194-199, 1979.

2. Stannard, A. A. et al.: Tumors of the skin and soft tissues. In: Tumors in Domestic Animals, 2nd Ed., University of California Press, Berkeley, 1978, pp 55-58.

3. Weiss, E. & Frese, K.: VII. Tumors of the Skin. In: Bull. Wld. Hlth. Org. 50: 79-100, 1974.

Case IV -81-179-4(X70) (AFIP 1802811):

History: A 14-month-old tailless cat was presented with severe chronic ulcerations of the distal dorsal and volar surfaces of the left hind leg and an abnormal gait.

Contributor's Diagnosis & Comments: Syringomyelia, lumbar and sacral spinal cord.

Comments: The cat's early history and breeding was unavailable, but it was likely congenitally tailless. That the cat was genetically part Manx is reasonably certain, suggesting that at least some degree of syringomyelia was congenital and one manifestation of the Manx syndrome. Brain and anterior spinal cord were normal. Taillessness and associated defects appear to be autosoma dominant with incomplete penetrance.

AFIP Diagnosis: Syringomyelia, segmental, moderate, tailless cat, feline.

Contributor: Department of Pathology, Kansas State University, Manhattan, Kansas 66506.

References:

1. Kitchen, H. et al.: Spina bifida, sacral dysgenesis and myelocele (Animal Model: Manx Cats). Am. J. Pathol. 66: 203-206, 1972.

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4. Leipold, H. W. et al.: Congenital defects of the caudal vertebral column and spinal cord in Manx cats. JAVMA 164: 520-523, 1974.

5. Martin, A. H.: A congenital defect in the spinal cord of the Manx cat. Vet. Pathol. 8: 232-238, 1971.

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Results
AFIP Wednesday Slide Conference - No. 2
23 September 1981

Conference Moderator: James B. Moe, DVM, PhD
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Case I - 81-1131 (AFIP 1804668):

History: Tissue from an adult, male, flemish giant, rabbit found dead. He was being used as a feeder for tsetse flies. On gross examination the left kidney was swollen, dark red in color, with multiple yellow-white foci 1-2 mm in diameter on the capsular surface and a white creamy material in the pelvis. (Gross photograph included).

Laboratory Results: Staphylococcus aureus and Escherichia coli were cultured from both kidneys, the mitral valve and external ear canals of both ears. Pasteurella multocida was cultured from the middle ear canal.

Contributor's Diagnosis & Comments: 1) Infarct, subacute, multifocal, marked, kidney, rabbit. 2) Thrombosis, multifocal, moderate, kidney. 3) Septic emboli, multifocal, mild, kidney. 4) Amyloidosis, multifocal, moderate, kidney.

Comment: Other gross findings in this animal included: A light brown crusty material in the ear canals which contained mites identified as Psoroptes cuniculi with a yellow-white creamy material beneath the crust; and a raised nodular mass 0.5 cm high attached to one leaf of the mitral valve. Microscopically, emboli and thrombi were present in the lung as well as the kidney. Staphylococcus aureus and Escherichia coli were cultured from the kidneys, heart valve lesion and the external ear canals. Unfortunately samples from the lung were not submitted for microbiological examination. The ability of P. cuniculi to precipitate systemic disease is not well established in the literature. Controversy even exists as to whether or not otitis resulting from secondary bacterial invaders can extend from the external ear canal into the middle and inner ear. This case supplies evidence indicating that systemic disease can result from otitis externa secondary to P. cuniculi as the same organisms were cultured from the external ear canals, heart valve lesion and the kidneys. Additionally, Pasteurella multocida was cultured from one of the middle ear canals. How the organism reached this location is not known. Amyloid deposits in the kidney were suspected on H&E slides and confirmed by use of Congo red stain and polarized light. Amyloid was also found in the pancreas, spleen, stomach, small intestine, sacculus rotundus and colon. The rabbit was uremic, a condition which has been associated with impaired immuno-responsiveness in other species.

AFIP Diagnosis: 1) Infarcts, acute, multiple, severe, kidney, rabbit, Lagomorph, etiology, multifocal thrombi and bacterial emboli. 2) Amyloidosis, glomerular tufts, moderate, and interstitium, multifocal, mild, kidney.

Conference Note: Some participants commented that subcutaneous abscesses or lymphadenitis due to Staphylococcus aureus are known to cause bacteremias in rabbits and could have been the primary site of infection in this case.

Contributor: Division of Pathology, WRAIR, WRAMC, Washington, DC 20012.

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2. Hinton, M.: Veterinary problems in a colony of rabbits used to feed tsetse flies. Br. Vet. J. 136: 33, 1980.

3. Raska, et al.: Humoral inhibitors of the immune response in uremia. Lab. Invest. 42: 636-642, 1980.

Case II - CP77-1189 (AFIP 1802806):

History: A 400 gm female Hartley guinea pig was injected with complete Freunds adjuvant and egg albumin in each foot pad 2-1/2 weeks prior to death. At time of foot pad injection the animal was also administered 20 ml of sterile mineral oil I.P.

Contributor's Diagnosis & Comments: Grossly, the peritoneal cavity was filled with mineral oil, and on both the parietal and visceral peritoneal surfaces there were multiple shaggy white plaques. Examination of the lungs revealed several patchy areas of consolidation. Microscopically, there was thickening of alveolar septa with numerous lipid laden macrophages, neutrophils and lymphocytes and distended capillaries, some of which contained lipid. It is felt the pneumonic process is associated with the intraperitoneal injection of mineral oil; whereby the mineral oil was absorbed by lymphatics, entered the blood stream and was carried to a number of tissues where it was phagocytized by macrophages. Bacteria were not recovered from cultures of either the lungs or peritoneal cavity. Therefore, the neutrophilic response was probably in response to the lipid.

AFIP Diagnosis: 1) Pneumonia, interstitial, granulomatous, characterized by numerous vacuolated macrophages, chronic, diffuse, severe, lung, guinea pig, rodent, etiology compatible with lipid pneumonia. 2) Vasculitis and perivasculitis, subacute, multifocal, moderate, lung.

Conference Note: In addition to the changes described by the contributor participants also noted the presence of giant cells and vasculitis in most sections. Many speculated that a type IV hypersensitivity had occurred compounding the lipid pneumonia. The possibility of the complete Freund's adjuvant gaining access to the lung by way of the circulation was considered as well as repeated exposure to an inhaled antigen. Acid fast bacteria, however, were not present. The brown pigment seen within macrophages was PAS positive and acid fast negative.

Contributor: Division of Comparative Medicine, Southwestern Medical School, 5323 Harry Hines Blvd., Dallas, TX 75235.

Reference:

Benirschke, K., et al.: Pathology of Laboratory Animals, Springer-Verlag, New York, 1978, p. 98.

Case III - DN 18213 (AFIP 1803087):

History: Tissue from a male equine fetus that was aborted at approximately 9-1/2 months gestation.

Laboratory Results: Bacterial culture attempts on selected tissues were negative.

Contributor's Diagnosis & Comments: Liver - focal necrosis with occasional cells that have eosinophilic intranuclear inclusions that are consistent with those described for equine rhinopneumonitis virus.

Comments: The original submission from the practitioner included portions of fetal lung, spleen, and liver for histopathologic examination. The lung section had several eosinophilic intranuclear inclusions in the bronchiolar epithelium. The spleen section had small foci of necrosis of lymphocytes in the lymphocytic follicles. The liver section had random areas of focal necrosis in which several affected cells had eosinophilic intranuclear inclusions. The vaccination history of the mare that aborted was not available at time of abortion.

AFIP Diagnosis: Necrosis, hepatocellular, with eosinophilic intranuclear inclusions, acute, multifocal, severe; and periportal hepatitis, subacute, multifocal, liver, foal, etiology compatible with equine herpes virus.

Conference Note: There was discussion concerning the nature of the periportal cellular infiltrates, particularly considering the relative lack of an inflammatory response to the foci of necrosis. Opinions included: extramedullary hematopoiesis; loss of hepatocytes with condensation of the remaining Kupffer cells and stroma; a lymphoid infiltrate; and an infiltrate of immature granulocytic cells. The morphologic changes in the fetal liver with EHV abortion as described by Jubb and Kennedy include leukocytic infiltrates within portal areas.

Contributor: Experimental Pathology Laboratories, P. O. Box 474, Herndon, VA 22070.

References:

1. Gillespie, J. H. et al.: Hagan and Bruner's Infectious Diseases of Domestic Animals. Cornell Univ. Press, Ithaca, NY, 1981, pp 563-568.

2. Horner, G. W.: Serological relationship between abortifacient and respiratory strains of equine herpesvirus 1 in New Zealand. New Zealand Vet. J. 29: 7-8, 1981.

3. Roberts, S. J.: Veterinary Obstetrics and Genital Diseases, Edwards Brothers, Inc., Ann Arbor, MI, 1971, pp 135-147.

4. Studdert, M. J.: Comparative aspects of equine herpesviruses. Cornell Vet. 64: 94-122, 1973.

Case IV - 3-18 (AFIP 1795804):

History: An incidental finding in a 2-year-old rat.

Contributor's Diagnosis & Comments: Granulosa cell tumor.

Comments: The ovarian mass consisted of tubular structures of varying sizes and shapes embedded in a mesenchymal stroma. The lining epithelium, arranged in columns, were Sertoli-like in appearance. The cytoplasm was clear with occasional coarse vacuoles, and the nuclei were round to oval. In some tubules, the cells formed clusters around small foci of proteinaceous material creating Call-Exner-like bodies.

AFIP Diagnosis: Gonadostromal tumor, Sertoli cell pattern, ovary, rat.

Conference Note: Although Sertoli cell tumor of the ovary was the most frequent diagnosis submitted, others included granulosa cell tumor with Sertoli cell pattern, arrhenoblastoma, gonadoblastoma, and Sertoli cell hyperplasia associated with senile follicular atrophy. True Call-Exner bodies were not thought to be present because well defined rosettes did not surround the eosinophilic material in the tubular structures.

Contributor: Bio/Dynamics, Inc., Mettlers Road, East Millstone, New Jersey 08873.

References:

1. Carter, R. L. et al.: Tumors of the ovary. In: W.h.O. Pathology of Tumors in Laboratory Animals, Vol. 2, IARC, Lyon, 1973, pp 189-200.

2. Engle, E. T.: Tubular adenomas and testis-like tubules of the ovaries of aged rats. Cancer Res. 6: 578-582, 1946.

3. Norris, H. J. et al.: Comparative pathology of ovarian neoplasms. Path. Vet. 6: 45-58, 1969.

4. Tavassoli, F. A. et al.: Sertoli cell tumors of the ovary. Cancer 46: 2281-2297, 1980.

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Results
AFIP Wednesday Slide Conference - No. 3
30 September 1981

Conference Moderator: James B. Nold, DVM
Major, USAF, BSC(VC)
Diplomate ACVP
Head, Comparative Pathology Division
Armed Forces Radiobiology Research Institute
Bethesda, MD 20014

Case I - 81-768 (AFIP 1804140):

History: A 13-year-old Arabian stallion was referred to our clinic with a history of anorexia and depression of 6 days duration. Clinical exam revealed marked icterus, ataxia, and dark brownish-red urine. There was no history of exposure to a toxin or recent vaccinations.

Laboratory Results: AST (SGOT) was 1000 IU/L; total bilirubin was 28 mg/dl of which 11 mg/dl was direct and BUN was 24 mg/dl.

Contributor's Diagnosis & Comments: Liver, hepatitis, nonsuppurative, necrotizing, severe, compatible with serum hepatitis.

Comments: Severe diffuse coagulation necrosis and loss of hepatocytes has resulted in collapse of hepatic parenchyma bringing portal triads into close apposition. Generally the only remaining hepatocytes correspond to lamina terminalis and are often regenerative. Portal triads are infiltrated by mononuclear cells. Abundant bilirubin is located in distended biliary canaliculi, hepatocellular cytoplasm and bile duct lumens.

AFIP Diagnosis: Hepatitis, necrotizing, acute to subacute, diffuse, severe, liver, Arabian, equine.

Conference Note: The majority of participants considered the morphologic changes most compatible with serum hepatitis. Differential diagnoses discussed included hepatitis due to pyrrolizidine alkaloids or other plant toxins, aflatoxin, phenothiazine, and EIA. One possible pathogenesis for serum hepatitis presented by the moderator was an organ specific Shwartzmann reaction. Special stains demonstrated the brown granular pigment prominent within the multinucleated hepatocytes to be hemoglobin. Intra-canalicular bile stasis also was confirmed.

Contributor: University of Tennessee, College of Veterinary Medicine, P. O. Box 1071, Dept of Pathobiology, Knoxville, TN 37901.

References:

1. Hjerpe, C.A.: Serum hepatitis in the horse. JAVMA 144: 734-740, 1964.
2. Howarth, L. & Shires, M.: Serum Hepatitis in a Horse, Iowa State University Veterinarian, 1976, No. 1, pp 28-32.
3. Jubb, K.V.F. & Kennedy, P.C.: Pathology of Domestic Animals, 2nd Ed., Vol. 2, 1970, p. 227.
4. Panciera, R.J.: Serum hepatitis in the horse. JAVMA 155: 408-412, 1969.

Case II - 81-0133 (AFIP 1803021):

History: A 6-month-old male poodle developed diarrhea and began vomiting. In 24 hours it became listless and developed an elevated temperature. It was taken to a veterinary emergency service where the dog died while being prepared for diagnostic radiography. The cadaver was presented for necropsy the next day. Chalky fluid was found in stomach and trachea and ran from cut surface of lung. The lung appeared collapsed. Most pieces of lung did not float in formalin.

Contributor's Diagnosis & Comments: Aspiration of barium sulfate suspension; pulmonary foreign body.

Comments: The case was selected as an example of exceptionally deep aspiration and as an example of an identified foreign substance. The emergency service veterinarian did not attempt to cover up the fact that the dog drowned.

AFIP Diagnosis: Anisotropic crystalline material, intra-alveolar and intra-bronchiolar, diffuse, severe, lung, poodle, canine.

Conference Note: Several participants commented that the presence of contrast material within so many alveolar spaces was unusual for simple aspiration and was indicative of introduction by positive pressure. The identity of the material was thought to be barium sulfate or kaolin. In situations where barium sulfate is a suspected foreign body its presence can be shown by placing a portion of the material on a platinum loop and inserting it into the flame of a Bunsen burner. The blue-green flame characteristic of the barium cation will be seen.

Contributor: Department of Veterinary Science, University of Arizona, Tucson, AZ 85721.

References:

1. Huston, J. et al.: Pulmonary reaction to barium sulfate in rats. Arch. Path. 54: 430-438, 1952.

2. Johnson, F. B.: Crystals in pathologic specimens. Path. Annu. 7: 321-344, 1972.

3. Kay, S.: Tissue reaction to barium sulfate contrast medium. Arch. Path. 57: 279-284, 1954.

Case III - 804 (AFIP 1802812):

History: Ten-week-old dog with history of vomiting, diarrhea and dehydration.

Contributor's Diagnosis & Comments: Corona - viral enteritis.

Comments: Direct fluorescent antibody staining of intestinal mucosa was positive for corona virus and negative for distemper and parvovirus. Negative staining of stool specimen revealed virus consistent with corona.

AFIP Diagnosis: Enteritis, with villous blunting and fusion, subacute, diffuse, moderate, small intestine, canine.

Conference Note: One of the primary differential diagnoses considered was resolving parvovirus enteritis. Several participants, however, who had previously examined sections of small intestine from canines recovering from CPV stated that the morphologic appearance was much different from corona virus enteritis: no villi were present, the remaining lamina propria was extensively fused, and the crypts continued to regenerate producing bizarre epithelial cells.

Contributor: C. E. Kord Animal Disease Laboratory, P.O.Box 40627, Melrose Station, Nashville, TN 37204.

References:

1. Appel, M.J.G. et al.: Canine viral enteritis. I. Status report on corona- and parvo-like enteritides. Cornell Vet. 69: 123-133, 1979.

2. Keenan, K. P. et al.: Intestinal infection of neonatal dogs with canine coronavirus 1-71: Studies by virologic, histologic, histochemical, and immunofluorescent technics. Am. J. Vet. Res. 37: 247-256, 1976.

3. Mebus, C. A. et al.: Pathology of neonatal calf diarrhea induced by a coronavirus-like agent. Vet. Path. 10: 45-64, 1973.

Case IV - R28186 (AFIP 1804128):

History: The animal was a 768-day-old Fischer 344 rat used as a control in a 2-year feeding study. At necropsy, the left kidney had a 1 cm in diameter cream colored mass involving the anterior pole. The mass had a gritty sensation on cutting.

Contributor's Diagnosis & Comments: Renal adenocarcinoma with osseous metaplasia.

Comments: This tumor is of interest because of the prominent ossification that is present. Some foci of tumor cells have a transitional cell appearance, but the tumor location and the presence of ossification would be more consistent with the chosen diagnosis.

AFIP Diagnosis: 1) Carcinoma, solid, renal clear cell type, with osseous metaplasia, kidney, F344 rat, rodent. 2) Glomerulopathy, chronic, diffuse, and interstitial nephritis, chronic, multifocal, minimal, kidney.

Conference Note: Most participants concurred with the contributor. The two other differential diagnoses considered were transitional cell carcinoma and adrenal cortical carcinoma. One participant commented that transitional cell carcinomas in his experience are typically more invasive, located in the vicinity of the renal pelvis, and are rare.

Contributor: Bushy Run Research Center, R.D. 4, Mellon Road, Export, PA 15632.

References:

1. Bennington, J. L. et al.: Tumors of the kidney, renal pelvis, and ureter. Fascicle 12, 2nd series, Atlas of Tumor Pathology, AFIP, Wash., DC, 1975.

2. Hard, G. C.: Tumors of the kidney, renal pelvis, and ureters. In: WHO Pathology of Tumors in Laboratory Animals, IARC, Lyon, 1973, pp 83-90.

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Results
AFIP Wednesday Slide Conference - No. 4
7 October 1981

Moderator: Alexander De Paoli, DVM, PhD
Colonel, VC, USA
Diplomate ACVP
Chief, Pathology Division
U.S. Army Medical Research Institute of Infectious Disease
Fort Detrick, MD 21701

Case I - 80/479/3 (AFIP 1803359):

History: A three-year-old pony gelding was splenectomised and transfused with 400 ml of equine blood.

Laboratory Results:

Day	Temp. (°C)	P.C.V. (%)	W.B.C. X10^6/L	Parasitemia (thin smear)	Clinical Signs
-3					Horse splenectomised
0	39.1				Inoculated i/v 400 ml blood
1	39.2	36		7/5 minutes	Normal
2	38.8			75/5 minutes	Normal
3	38.6	37		0.8%	"
4	39.3	36		4.5%	"
5	40.0	36		25.0%	Lethargic
6	39.0	23	19,690	54%	Depressed
7	38.6	9	22,220*	70%	Very depressed, frank icterus, hemoglobinuria, died 5 p.m.
			Band 8%		
			Neut 54%		
			Lymph 31%		
			Mono 7%		

*Marked erythrophagocytosis by neutrophils.

Contributor's Diagnosis & Comments: Acute fatal Babesia equi infection.
Comments: This was an experimental infection for production of antigen slides for I.F.A.T. Parasitemia was detected the day after infection. Leucocytosis, erythrophagocytosis and parasitemia are prominent in liver and kidney sections.

AFIP Diagnosis: 1) Parasitemia, erythrocytic, severe, red blood cells, liver and spleen, pony, equine, etiology, Babesia equi. 2) Nephrosis, hemoglobinuric, acute, diffuse, mild to moderate, kidney. 3) Hepatitis, periportal, and cholestasis, subacute, diffuse, mild, liver. 4) Erythrophagocytosis, diffuse, moderate to severe, liver and kidney.

Conference Note: The hepatitis was not thought to be related to the Babesiosis.

Contributor: Regional Veterinary Laboratory, Wollongbar, N.S.W. 2480, Australia.

References:

1. Allen, P. C. et al.: Experimental acute Babesia caballi infections. I. Red blood cell dynamics. Exp. Parasitol. 37: 67-77, 1975.

2. Allen, P. C. et al.: Experimental acute Babesia caballi infections. II. Response of platelets and fibrinogen. Exp. Parasitol. 37: 373-379, 1975.

3. Blood, Henderson, Rodostits: Veterinary Medicine, Fifth Edition, Lea & Febiger, Philadelphia, 1979, pp 728-734.

4. Jubb & Kennedy: Pathology of Domestic Animals, Vol. I, Academic Press, New York, 1970, pp 321-322.

5. Mahoney, D. F.: Babesia of domestic animals. In: Parasitic Protozoa, Vol. IV, J. P. Kreier, Ed., Academic Press, New York, 1977, pp 1-43.

6. Roby, T. O. et al.: The hereditary transmission of Babesia caballi in the tropical horse tick, Dermacentor nitens Neumann. A.J.V.R. 25: 105, 1964.

7. Ward, P. A. et al.: The entry process of Babesia merozoites into red cells. Am. J. Pathol. 102 (1): 109-114, 1981.

Case II - 79-D-32 (AFIP 1802130):

History: A female 28-month-old smooth black guinea pig died unexpectedly. She was one of a group of guinea pigs with diabetes mellitus, which were being studied.

Laboratory Results: Hemolytic coagulase positive Staphlococcus aureus was cultured from two specimens of liver and from heart blood.

Contributor's Diagnosis & Comments: Hepatic botryomycosis.

Comments: There is focal necrosis with numerous heterophils and macrophages surrounded by fibrous connective tissue. There are plasma cells, and hyperplasia of bile ducts adjacent to the abscessed areas. A Gram stain demonstrated gram-positive large cocci organisms arranged in clusters. GMS and PAS stains were not demonstrable. There is also mild bile duct hyperplasia and plasma cells are present in occasional portal areas. Hepatocytic vacuolar change is present.

AFIP Diagnosis: 1) Pyogranuloma, focal, moderate, liver, smooth black guinea pig, rodent. 2) Hepatitis, periportal, subacute, diffuse, mild, liver.

Conference Note: Although participants observed most of the changes described by the contributor, bacterial granules were not seen in any sections. Accordingly, the diagnosis of botryomycosis could not be made.

Contributor: Dept of Comparative Medicine, The Milton S. Hershey Medical Center, Pennsylvania State University, Hershey, PA.

References:
1. Benirschke, K. et al.: Pathology of Laboratory Animals, Springer-Verlag, New York, 1978, pp 1451-1453.
2. Binford, C. H. et al.: Diseases caused by fungi and actinomycetes. In: Pathology of Tropical and Extraordinary Diseases, AFIP, Washington, DC, 1976, p. 561.
3. Blackmore, D. K. et al.: The apparent transmission of staphylococci of human origin to laboratory animals. J. Comp. Path. 80: 645-651, 1970.
4. Ganaway, J. R.: Bacterial, mycoplasma, and rickettsial diseases. In: The Biology of the Guinea Pig, J.E. Wagner & P.J. Manning, Eds., Academic Press, New York, 1976, p. 128.
5. Gupta, B. N. et al.: Osteoarthritis in guinea pigs. Lab. Anim. Sci. 22 (3): 362-368, 1972.
6. Shults, F. S. et al.: Staphylococcal botryomycosis in a specific pathogen-free mouse colony. Lab. Anim. Sci. 23 (1): 36-42, 1973.

Case III - S2298-80 (AFIP 1803012):

History: A 5-year-old female Siberian husky. Chronic proliferative oral lesions not responsive to antibiotics.

Contributor's Diagnosis & Comments: Eosinophilic collagenosis / Oral eosinophilic granuloma in Siberian husky dogs.

Comments: The cause of this chronic lesion is not known. It appears to be specific for the Siberian husky dog and is remarkably responsive to corticosteroid administration.

AFIP Diagnosis: Granulomas, eosinophilic, linear, multiple, focally extensive, severe, ventrolateral tongue, Siberian husky, canine.

Conference Note: One participant thought that in the eosinophilic centers of the granulomas the collagen undergoing degradation was surrounded by Splendore-Hoeppli material. The morphologic similarity to the linear eosinophilic granuloma of the cat was discussed.

Contributor: The Animal Medical Center, 510 East 62nd St., New York, N.Y. 10021.

References:
1. Bucci, T. J.: Intradermal granuloma associated with collagen degeneration in three cats. JAVMA 148: 794-800, 1966.
2. Madewell, B. R. et al.: Oral eosinophilic granuloma in Siberian husky dogs. JAVMA 177: 701-703, 1980.
3. Potter, K. A. et al.: Oral eosinophilic granuloma of Siberian huskies. JAAHA 16: 595-600, 1980.

Case IV - 08-010 (AFIP 1758399):

History: Collie dog, 7-year-old male, unilaterally castrated 3 years earlier (veterinarian performed a laparotomy and could not locate a retained testicle). Dog was presented with complaint of lethargy, anorexia and weight loss and a progressively enlarging abdomen. At this time, a clinical workup was done and the dog was tentatively diagnosed as having hepatitis, unknown origin. The dog was treated with steroids, various antibiotics and megestrol acetate to stimulate appetite. The abdomen continued to enlarge and 1 week later was full of clear to yellow cysts of varying size. An irregular, firm, 10x8x8 cm mass was also found within the abdomen.

Laboratory Results:

SGOT - 44	PCV - 39	WBC - 29,300		URINALYSIS - Trace protein	
LDH - 287	RBC - 5.2	Segs - 93%	Lymphs - 4%		
		Bands 0	Mono - 3%		

Contributor's Diagnosis & Comments: Mesothelioma and in the retained testicle Sertoli cell tumor and seminoma.

Comments: Mesotheliomas are rare in dogs[1] and other domestic animals, but occur with some frequency in laboratory rodents.[2] They have been reported to arise from the tunical vaginalis of the testes as well as from other serosal sites. The classic "tombstone" appearance of the mesothelial cover cell layer is evident in several locations in the neoplasm and arborescent growths over a stalked stroma can be found. The cuboidal lining cells demonstrates the pleuripotent nature of mesothelial cells which also line all of the Muellerian duct and the germinal epithelium of ovaries.

AFIP Diagnosis: Mesothelioma, abdominal cavity, collie, canine.

Conference Note: Sections vary considerably with respect to cellularity. Participants unanimously concurred with the diagnosis of mesothelioma. Part of the discussion was concerned with the identity of the cystic structures. The possibilities considered, in addition to being part of the neoplasm, included lymphatics, preexistent omental stroma, and dilated tubular remnants of the retained testis. One attendee commented that mesotheliomas in cattle can be cystic. The moderator stated that electron microscopy is often helpful in diagnosing difficult cases; mesothelial cells have tight junctions, lack basement membrane, and often have cilia.

Contributor: Hazleton Lab., Inc., 9301 Leesburg Turnpike, Vienna, VA.

References:

1. Benirschke, K. et al.: Pathology of Laboratory Animals, 1978.
2. Jubb, K.V.F. et al.: Pathology of Domestic Animals, Vol. 2, 1970.
3. Moulton, J. E.: Tumors in Domestic Animals, 1978.

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Results
AFIP Wednesday Slide Conference - No. 5
14 October 1981

Conference Moderator: John M. Langloss, DVM, PhD
Diplomate ACVP
Chief, Division of Immunopathology
Armed Forces Institute of Pathology
Washington, DC 20306

Case I - 81P539 (AFIP 1803328).

History: This tissue is from a 3-week-old male Arabian foal that had mild diarrhea for two weeks.

Contributor's Diagnosis & Comments: 1) Thymic hypoplasia. 2) Lymphoid hypoplasia, lymph nodes and spleen.

Comments: WBC 5500; Lymphocytes <400; IgM <5 mg%; IgG 400 mg%; Peripheral blood lymphocytes were nonresponsive to phylolectins.

The lesions seen in this foal are compatible with a diagnosis of combined immunodeficiency. The diagnosis was confirmed by the lack of responsiveness of peripheral blood lymphocytes. Changes (collections of inflammatory cells and debris) in Hassall's corpuscles are unexplained but have been previously described (McGuire et al. 1976).

AFIP Diagnosis: Hypoplasia, lymphoid, diffuse, severe, thymus, spleen, lymph nodes, Arabian foal, equine, etiology compatible with combined immunodeficiency of horses.

Conference Note: Participants unanimously diagnosed lymphoid hypoplasia and considered combined immunodeficiency the primary etiologic diagnosis. Part of the discussion was concerned with whether hypoplasia and lymphoid depletion can be differentiated strictly morphologically. Participants commented that they had seen lymphoid tissues with a similar histologic appearance in irradiated animals and in chronic disease states with lymphoid depletion.

Contributor: Department of Pathology, College of Veterinary Medicine, Colorado State University, Ft. Collins, CO 80521.

References:

1. McGuire, T.C. et al.: Alterations of the thymus and other lymphoid tissue in young horses with combined immunodeficiency. Am. J. Pathol. 84: 39, 1976.

2. Splitter, G. et al.: Combined immunodeficiency of horses: A review. Developmental and Comparative Immunology 4: 21-32, 1980.

Case II - 79-0025 (AFIP 1713952).

History: Brain from a 9-month-old male sheep dog that was presented with progressive CNS disease including ataxia, incoordination, and hyperreflexia. The dog was euthanatized.

Contributor's Diagnosis: Encephalomeningitis, nonsuppurative, diffuse, moderate to severe, cerebellum with intranuclear inclusions consistent with a diagnosis of canine distemper.

AFIP Diagnosis: Meningoencephalitis, nonsuppurative and demyelinating with multiple eosinophilic intranuclear inclusions, chronic, multifocal, moderate to severe, cerebellum and midbrain, sheep dog, canine, etiology compatible with canine distemper.

Conference Note: Participants unanimously concurred with the contributor and agreed that the morphologic changes were classic for canine distemper.

Contributor: U.S. Army Biomedical Laboratory, (SGRD-UV-VC), Aberdeen Proving Ground, MD 21010.

References:

1. Appel, M.: Pathogenesis of canine distemper. A.J.V.R. 30 (7): 1167-1182, 1969.

2. Jubb & Kennedy: Pathology of Domestic Animals, Vol. 1, Academic Press, New York, 1970, pp 215-221.

3. Vandevelde, M. et al.: Chronic canine distemper virus encephalitis in mature dogs. Vet. Pathol. 17: 17-29, 1980.

4. Watson, A. et al.: The ultrastructure of inclusions in blood cells of dogs with distemper. J. Comp. Pathol. 84: 417-426, 1974.

5. Wisniewski, H. et al.: Observations on viral demyelinating encephalomyelitis, canine distemper. Lab. Invest. 26 (5): 589-599, 1972.

Case III - 91284 (AFIP 1802815).

History: An 18-month-old female German shepherd dog was presented to the hospital with a temp. of 102.4, swelling of the muzzle, and hemorrhage in the right eye after being bitten on the nose by a Mojave Desert rattlesnake. Treatment was given, and the dog appeared to be doing well for three days. On the 4th hospital day, the dog vomited several times and refused to eat. The dog had a WBC of 17.5, RBC of 3.5, BUN of 225, creatinine of 9 on the 4th day. Vomition continued and the owner elected euthanasia on day 5. Necropsy examination was unrewarding, with only tiny dark foci seen in the renal cortices.

Contributor's Diagnosis & Comments: Disseminated intravascular coagulation (DIC) following rattlesnake envenomation.

Comments: The dog had been bitten on the nose by a Mojave Desert rattlesnake. Systemic pathologic effects following rattlesnake bite have been poorly studied in

the dog. This type of lesion has not been described in man, nor has it apparently been reported in dogs. The desert rattlesnake venom does contain material which has thrombin-like activity, as well as numerous other factors.

AFIP Diagnosis: 1) Thrombosis, glomerular tufts, acute, segmental to diffuse, severe, kidney, German shepherd, canine, etiology, Crotalus cerastes cerastes (Mojave Desert Rattlesnake) venom. 2) Nephrosis, with interstitial hemorrhage, acute, multifocal, severe, kidney.

Conference Note: Most participants submitted a diagnosis of glomerular thrombosis due to DIC.

The pathogenesis of the glomerular lesions was of particular interest. Although DIC was discussed as a possible etiology, the morphologic appearance was not considered typical because the primary changes were restricted to the glomeruli. The AFIP Department of Nephropathology described the lesion as a mesangiolytic glomerulopathy in which there was initial destruction of mesangial cells and subsequent replacement of the glomeruli by hemorrhagic cysts and thrombi. The nephrosis was considered secondary to ischemia and analagous to the "lower nephron nephrosis" seen with acute renal failure. Another explanation included increased capillary permeability, an effect described with crotalid venom. This change could have resulted in loss of protein and erythrocytes into the mesangium and subsequent obliteration of segments of the glomerular tuft due to increased osmotic pressure. Electron microscopic evaluation of the glomeruli was considered necessary to completely characterize the lesion.

Contributor: L.A. County Dept of Health, Section Comparative Pathology, 12824 Erickson Ave., Downey, CA 90242.

References:

1. Amorim, M. F. et al.: Intermediate nephron nephrosis from snake poisoning in man: Histopathologic study. Am. J. Pathol. 30: 479-499, 1954.

2. Chugh, K. S. et al.: Acute renal failure following snakebite. Am. J. Trop. Med. 24 (4): 692-697, 1975.

3. Chugh, K. S. et al.: Acute renal failure due to intravascular hemolysis in the North Indian patients. Am. J. Med. Sci. 274 (2): 139-146, 1977.

4. Russell, F. E.: Sanke Venom Poisoning, J. B. Lippincott Co., Philadelphia, 1980, pp 168-213.

Case IV - #1 (AFIP 1804149).

History: Tissue excised from a Sprague-Dawley rat.

Contributor's Diagnosis & Comments: Foreign body granuloma.

Comments: The foreign body in this case was a sterile cotton gauze sponge implanted for 14 days. The severity and apparent chronicity of the response is remarkable in the contributor's opinion. Since gauze sponges occasionally remain in patients, knowledge of the tissue response to and appearance of implanted cotton fibers may be valuable to the diagnostic pathologist.

AFIP Diagnosis: Granulomas, with anisotropic foreign material, chronic, multifocal, focally extensive, fascia, site unspecified, Sprague-Dawley rat, rodent.

Conference Note: The most common morphologic diagnoses submitted were fasciitis and foreign body granuloma. The changes seen were thought to be due to mechanical irritation versus a response to an antigen. The cotton fibers stained positively with Sirius red.

Contributor: Ethicon Research Foundation, Somerville, NJ 08876.

References:

1. Varma, S. et al.: Tissue reaction to suture materials in infected surgical wounds: A histopathologic evaluation. A.J.V.R. 42 (4): 563-570, 1981

2. Varma, S. et al.: Further studies with polyglycolic acid (Dexon) and other sutures in infected experimental wounds. A.J.V.R. 42 (4): 571-574, 1981.

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Results
AFIP Wednesday Slide Conference - No. 6
21 October 1981

Conference Moderator: Robert D. Furrow, D.V.M.
Diplomate ACVP
Veterinary Pathologist
Animal Resources Branch
Div. of Veterinary Medical Research
Food & Drug Administration
Beltsville, MD 20205

Case I - 1-ARB-004 (AFIP 1806689).

History: An obese eight-year-old male castrate poodle with lethargy and polyuria for one month. The urine S.G. was 1.010, alk. phosphatase 343, cholesterol 477, SGPT 463. At laparotomy the liver was enlarged and was covered with dark brown areas.

Contributor's Diagnosis & Comments: Vacuolar degeneration, diffuse, severe, liver, secondary to hyperadrenocorticism.

Comments: ACTH stimulation: pre-plasma cortisol = 14, post stimulation = 70. The dog was treated with lysodrin and the clinical signs improved. Post lysodrin ACTH stimulation: preplasma cortisol = 12, post stimulation = 11. The animal is now clinically much improved but continues to have polyuria secondary to chronic renal insufficiency. Alkaline phosphatase, cholesterol, and SGPT have returned to normal levels.

AFIP Diagnosis: Vacuolar change, periportal and midzonal, diffuse, severe, liver, poodle, canine, etiology, glycogen deposition due to hyperadrenocorticism.

Conference Note: Virtually all participants concurred with the contributor's morphologic diagnosis and considered hyperadrenocorticism as the most likely etiology. Diabetes mellitus was the most frequent differential diagnosis submitted although clinical signs were not thought to be typical of the disease. The PAS technique demonstrated many PAS positive, diastase sensitive, glycogen granules within the vacuolated cells. Many cells, however, retained a vacuolar appearance. Although the literature ascribes the latter to intracellular edema, several participants stated that glycogen could have been lost during processing. The biopsy was fixed in buffered formalin.

Contributor: Division of Veterinary Research, Bureau of Veterinary Medicine, FDA, Bldg 328A, BARC-E, Beltsville, MD.

References:

1. Badylak, S. F. et al.: Sequential morphologic and clinicopathologic alterations in dogs with experimentally induced glucocorticoid hepatopathy. AJVR 42: 1310-1318, 1981.

2. Rogers, W. A. et al.: A retrospective study of probable glucocorticoid-induced hepatopathy in dogs. JAVMA 170: 603-666, 1977.

Case II - 80B600 & 80B1338 (AFIP 1803538).

History: Tissue from one of a group of 1000 lovebirds of two species, Agapornis roseicollis and A. personata. The birds had been obtained from various sources and were housed in groups of 12 in newly constructed flight cages. Losses began after a few months and continued at a rate of 20-40 birds per week. Clinical signs were exercise intolerance with fine tremors developing shortly before death, or sudden death with no premonitory signs. Chlamydia were isolated from several of the birds. Other birds had severe candidiasis. (Two electron micrographs included).

Laboratory Results: In addition to hepatic, splenic and enteric lesions of psittacosis, several birds had giant nuclei with granular basophilic to amphophilic inclusions in renal tubular epithelium. These inclusions were sometimes accompanied by tubular degeneration and mononuclear or mixed interstitial infiltrate. In some instances no evidence of renal disease other than the presence of the inclusions was found. Virions, which were round to hexagonal with an average diameter of 70-80 nm, were seen within the enlarged nuclei. Paracrystalline arrays were not found. In degenerating cells virions in the cytoplasm were not enveloped. The characteristics are those of an adenovirus. Attempts to isolate the virus through chicken egg inoculations failed.

Contributor's Diagnosis & Comments: Renal adenovirus infection of lovebirds.

Comments: Although adenovirus infections of birds are fairly common, they are usually hepatic or respiratory. The renal inclusions have not been seen in other species of birds from the same establishment, but similar inclusions, with adenovirus particles demonstrable on electron microscopy, have been seen in the intestinal epithelium of Amazon parrots suffering from psittacosis and poxvirus infection. It is assumed that the lovebird and the Amazon inclusions represent activation of a latent adenovirus as has been described in other species.

AFIP Diagnosis: Nephritis, centrolobular, with basophilic intranuclear inclusions and karyomegaly, acute, multifocal, mild, kidney, lovebird, avian, etiology, adenovirus.

Conference Note: Although most participants considered the etiologic agent an adenovirus there were several morphologic diagnoses submitted. The interstitial cellular infiltrates were interpreted by the majority of attendees as a response to the viral destruction of epithelial cells. Others, however, considered them to be foci of extramedullary hematopoiesis. The moderator stated that in most birds, interstitial infiltrates are normally present but are distributed haphazardly throughout renal lobules, whereas in this case they were located in the vicinity of the viral inclusions. Several participants described tubular necrosis but the latter was considered a postmortem change.

Contributor: Department of Veterinary Pathology, University of California, Davis, CA.

References:

1. Mcferran, J. B. et al.: Avian adenoviruses - A review. Avian Path. 6: 189-217, 1977.

2. Ward, J. M. et al.: Latent adenovirus infection of rats: Intranuclear inclusions induced by treatment with a cancer chemotherapeutic agent. JAVMA 169: 952-953, 1976.

Case III - 227648-6 (AFIP 1804249).

History: Tissue from 30-week-old Ring-neck pheasants found dead with no symptoms prior to death (January 1981). The pheasants all came from separate pens. Average number per pen was 2,000. Over a period of 1 week approximately 900 birds were lost. Some oat and hay packs were found in the crops but the latter were not impacted. The same feed was fed to Chukar pheasants and Bob-white quail. A new batch of birds was brought in from Pennyslvania in December 1980.

Gross Lesions: Live birds were quite unsteady on their feet and had difficulty maintaining balance prior to being euthanatized. Four out of the 10 birds had markedly enlarged spleens with hundreds of white roundish nodular lesions measuring 1-2 mm in diameter incorporated inside them. Spleens were enlarged anywhere from 5 to 10 times normal and had a rather granular appearance over these grayish-white lesions. Other birds had spleens that were not quite as enlarged but also with the white lesions present. Two of the 4 birds had multifocal variably sized grayish-white nodular lesions present inside the liver. These measured from 1-2 mm in diameter and varied anywhere from a few to quite numerous lesions. Several of the birds had lungs that were slightly more edematous than normal.

Laboratory Findings: Microbiologic examination: The liver and spleen yielded a heavy growth of Pasteurella sp. which was sensitive to gentamycin, neomycin, nitrofurantoin, polymyxin B, and tetracycline but resistant to ampicillin, cephalothin, chloramphenical, linocomycin, penicillin, streptomycin and Vetisulid/ Sonilyn.

Histopathologic examination: A section of liver had multifocal areas of fibrinoid necrosis as well as multifocal areas of mixed inflammatory infiltrate and some necrosis present. Sections of spleen had multifocal areas of fibrinoid necrosis that were seen literally everywhere one looked in the spleen. Occasional large cells in the middle of these necrotic areas, as well as cells around the periphery, contained basophilic intranuclear inclusion bodies. This lesion was extremely widespread, severe, and striking. A section of lung had occasional focal areas of necrosis as well as colonies of bluish-staining bacteria present.

Contributor's Diagnosis & Comments: Marble spleen disease; fowl cholera.

Comments: This was a natural outbreak in an unvaccinated flock on a privately owned game farm. The combined infections produced severe losses totalling about 2000 birds by the time the problem had run its course. Although both of these diseases are relatively common in pheasants, this is the first time we had seen the combined infections in this laboratory, and they have not been reported in the literature as dual infections.

AFIP Diagnosis: 1) Depletion, lymphoid, with deposition of eosinophilic fibrillar and homogenous material and rare basophilic intranuclear inclusions, acute diffuse, severe, Schweigger-Seidel sheaths, spleen, pheasant, avian, etiology marble spleen disease (adenovirus). 2) Hyperplasia, reticuloendothelial cells (fixed mononuclear macrophages), diffuse, moderate, red pulp, spleen.

Conference Note: Participants unanimously diagnosed marble spleen disease. Morphologically reticuloendothelial cell hyperplasia and lymphoid depletion were most commonly described. The eosinophilic material was thought by many attendees to be amyloid. Others considered it to have a variable appearance: either lightly eosinophilic and fibrillar, or homogenous, more intensely eosinophilic, and

compatible with a proteinaceous material other than amyloid. Nuclei undergoing karyomegaly and containing large basophilic intranuclear inclusions were not present in any of the sections examined, although several participants observed occasional inclusions in nuclei of normal size. Bacteria were not described by any attendees. The moderator indicated there appeared to be an increased number of Schweigger-Seidel sheaths present.

Contributor: Animal Health Diagnostic Laboratory, Michigan State University, Lansing, MI.

References:

1. Bygrave,d A. C. et al.: Marble spleen disease in pheasants (Phasianus colchicus). Vet. Rec. 92: 534-535, 1973.

2. Carlson, H. C. et al.: Marble spleen disease of pheasants in Ontario. Can. J. Comp. Med. 37: 281-286, 1973.

3. Domermuth, C. H. et al.: A naturally occurring infection of chickens with a hemorrhagic enteritis/marble spleen disease. Avian Dis. 23: 479-484, 1979.

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5. Iltis, J. P. et al.: Demonstration of an avian adenovirus as the causative agent of marble spleen disease. Am. J. Vet. Res. 38: 95-100, 1977.

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7. McFerran, J. B. et al.: Avian adenoviruses - A review. Avian Path. 6: 189-217, 1977.

8. Wyand, D. S. et al.: Marble spleen disease in ring-necked pheasants: Histology and ultrastructure. Avian Dis. 16: 319-329, 1972.

Case IV - 81--210 (AFIP 1803540).

History: Colon and abomasum from an 8-year-old ox that was ill four days with mild diarrhea and mucoid, blood-flecked stools. Gross necropsy findings included mucosal hemorrhage in both the small and large intestine, flecks of blood in the intestinal lumen, diffuse but irregular adherent fibrinonecrotic debris in the proximal colon, and whitish, opaque, slightly elevated foci 0.2-1.5 cm diameter in the abomasal mucosa.

Laboratory Results: Colonic mucosal scrapings revealed oocysts of Eimeria zurnii.

Contributor's Diagnosis & Comments: Coccidiosis (confirmed). Bovine virus diarrhea (presumptive).

Comments: It is somewhat unusual for a mature cow to be fatally afflicted with coccidiosis and/or BVD. The relative roles of those infections in disease suffered by this animal are not clear. The slides contain nice examples of lesions attributable to each disease. At gross necropsy our thoughts were preoccupied by the coccidial infection. Because there were no upper alimentary lesions, nor Peyer's Patch lesions, BVD was not anticipated and virologic studies to confirm the presence of BVD antigen were not conducted. Some sections of ileum and colon had marked downgrowth of crypt epithelium into submucosal lymphoid follicles.

AFIP Diagnosis: 1. Colitis, mucinous and necrotizing, with intra and extra-epithelial protozoa, subacute, segmental, mild to moderate, colon, ox, etiology, coccidiosis (Eimeria zurnii). 2. Necrosis, multifocal, mild, gastric glands and mucus neck glands, abomasum, ox.

Conference Note: The number of oocysts and presence of abomasal necrosis varies in different sections. The elevated foci in the abomasal mucosa described by the contributor were not thought to be present in any of the sections examined by the participants. Lymphoid necrosis also was not observed by any attendees and caused some to question whether the diagnosis of BVD could be made in this case based strictly on histologic changes.

Contributor: Department of Pathology, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK 74074.

References:

1. Davis, L. R. et al.: The endogenous development of Eimeria zurnii, a pathogenic coccidium of cattle. AJVR 18: 569-574, 1957.

2. Jubb & Kennedy: Pathology of Domestic Animals, Vol 2, Academic Press, New York, 1970, pp 14-23.

3. Kent, T. H. et al.: Comparative pathogenesis of some enteric diseases. Vet. Path. 10: 414, 1973.

4. Ramsey, F. K.: The Pathology of a Mucosal Disease of Cattle. Proc. 91st Meeting Am. Vet. Med. Assoc., 1954, pp 162-166.

5. Stockdale, P.H.G. et al.: Production of bovine coccidiosis with Eimeria zuernii. Can. Vet. J. 17: 35-37, 1976.

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Results
AFIP Wednesday Slide Conference - No. 7
28 October 1981

Conference Moderator: Miriam R. Anver, DVM, PhD
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Case I - 8767(H&E) (AFIP 1795815).

History: 5-month-old Hartley strain guinea pig, weight 520 gms, control animal in an experiment. Found in an extremely weakened condition and was euthanatized.

Laboratory Results: At necropsy the lungs were beige colored, rubbery in texture and failed to collapse when the chest was opened. Multiple pale foci up to 2 mm in diameter were present in the spleen. Petechial hemorrhages were present in subcutis and mesenteric fat. Lymph nodes were dark red in color. Escherichia coli and Klebsiella pneumoniae were cultured from spleen and liver.

Contributor's Diagnosis & Comments: Pneumonia, interstitial, diffuse, probably due to cytomegalovirus.

Comments: This case is compatible with severe generalized cytomegalovirus infection in a presumably immunocompromised host (no proof of this available), with terminal gram-negative septicemia. Inclusions compatible with those of cytomegalovirus were found in salivary gland, lung, mediastinal nodes, thymus, stomach, small intestine, large intestine, liver, kidney, pancreatic islets, mesenteric nodes, ovary, bone marrow and meninges. Severe morphologic alterations were present in many of these organs. The bone marrow and spleen had extensive acute necrosis. Many nodes were severely effaced and hemorrhagic. There was obvious bacterial overgrowth in the intestinal wall, and large colonies of bacteria were present in necrotic areas of the spleen.

AFIP Diagnosis: 1) Pneumonia, interstitial, with numerous intranuclear eosinophilic inclusions, acute, diffuse, moderate, lung, guinea pig, rodent, etiology compatible with guinea pig cytomegalovirus. 2) Lymphadenitis, necrotizing, with multiple syncytial giant cells and intranuclear eosinophilic inclusions, acute, diffuse, moderate, lymph node.

Conference Note: Participants unanimously concurred with the contributor. Inclusions were observed in alveolar epithelium, macrophages, and endothelial cells. Topics discussed included cytomegalovirus and other herpesvirus infections in various species. One attendee indicated that the histologic appearance of the lungs in this case was very similar to those of human neonates congenitally infected with human cytomegalovirus causing severe multisystem disease. These infants often died within the first week of life.

Contributor: Department of Comparative Medicine, School of Medicine and Dentistry, University of Alabama, University Station, Birmingham, AL 35294.

References:
1. Hsiung, G. D. et al.: Viruses of guinea pigs: Considerations for biomedical research. Microbiol. Rev. 44: 468-490, 1980.
2. Van Hoosier, G. L., Jr. et al.: Chapt 10, Viral and Chlamydial Diseases. In: Biology of the Guinea Pig, Wagner and Manning (eds), Academic Press, NY, 1976.
3. Robbins: Pathologic Basis of Disease, Saunders, Philadelphia, 1979, pp 569-572.
4. Weller, T. H.: The cytomegaloviruses: Ubiquitous agents with protean clinical manifestations. New Eng. J. Med. 285: 203-212, 1971.
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Case II - 81-360 (AFIP 1803321).

History: A 4-month-old domestic long-hair cat had a movable, radiographically uniform, soft tissue mass in the left ventral neck that had been growing as the kitten grew. An encapsulated soft mass was removed and two months later, it recurred. Two courses of Adriamycin (6.6 mg/m^2) were given. A moderate but therapeutically refractive anemia ensued. Splenectomy did not help. The spleen showed lymphoid hyperplasia and marked extramedullary hematopoiesis. The CBC is given below. The cat continued to deteriorate and was euthanatized at 13 months of age. Necropsy results showed tumor masses in the liver and kidney. The thymus was inapparent and lymph nodes were small. Both the kidneys and liver were enlarged and pale.

Laboratory Results: At first admission SGPT was 331 IU and LDH was 570 IU. After splenectomy, hematology showed:

Hb	4.0-6.5 g/dl
WBC	22,800-33,900/mm^3
PMN's	70-80%
Lymph	11-20%
Monos	3-5%

Contributor's Diagnosis & Comments: 1) Lymphosarcoma, multicentric, liver, kidney, bone marrow, feline leukemia virus. 2) Liposarcoma, neck, recurrent with metastasis to liver and kidney, feline sarcoma virus? or feline leukemia virus?

Comments: The first tumor measured 6.5 x 7 cm and floated. A diagnosis of liposarcoma was made on histopathology; C-type virus particles were found on EM. Two subsequent serum samples were positive for feline leukemia virus by ELISA. At necropsy, liposarcomas were found at the original site and in the liver and kidney. Lymphosarcoma was found in the liver kidney and bone marrow. Electron microscopy of both neoplasms showed C-type particles. The diseases in this cat can be explained by feline leukemia virus. Liposarcomas have been previously associated with feline leukemia virus[1] but under experimental transmission studies. They thought that a sarcoma virus could be involved because the virus caused transformation of cultured cells -- a trait of sarcoma virus. The feline sarcoma viruses are incomplete and require a helper virus to reproduce. This naturally occurring case could be demonstrating that relationship between feline leukemia and sarcoma viruses.

AFIP Diagnosis: 1) Lymphosarcoma, poorly differentiated, kidney, cat, feline, etiology, feline leukemia virus. 2) Liposarcoma, perirenal fat.

Conference Note: Virtually all participants concurred with the diagnosis of lymphosarcoma in the kidney but there was disagreement concerning the perirenal mass. Attendees either concurred with the contributor or felt that the tumor was another manifestation of the lymphosarcoma. The small hyperchromatic cells observed scattered throughout the neoplastic lymphocytes were thought to be mature lymphocytes and metarubricytes. The moderator pointed out the morphologic similarity between the liposarcoma in this case and the ones described by Rickard et al.

Contributor: University of Texas Medical School, 6431 Fannin, Houston, Texas 77025.

References:

1. Rickard, C. G. et al.: A transmissable virus induced leukemia of the cat. J. NCI 42: 987-1014, 1969.
2. Dr. John Post. Personal communication (Contributor reference).
3. Essex, M.: Feline leukemia and sarcoma viruses in viral oncology. In: Viral Oncology, George Klein (ed), Raven Press, 1980, pp 205-229.
4. Hardy, W. D.: Feline leukemia virus. Hardy, Essex, and McClelland (eds), Elsevier, North Holland Inc., 1980, pp3-114.
5. Essex, M.: Feline oncornaviruses. Adv. Canc. Res. 21: 235-237, 1975.

Case III - 81-942(HPS stain) (AFIP 1804672).

History: Spinal cord from a 7-month-old female boxer. For about two weeks prior to admission, this dog had shown evidence of severe pain in the cervical area. Necropsy revealed massive leptomeningeal hemorrhage affecting mainly the ventro-lateral zones of the cervical region of the spinal cord.

Contributor's Diagnosis & Comments: Histologically, lesions were confined mainly to spinal meninges. Segmental or diffuse fibrinoid necrosis was present in several meningeal arterioles and small arteries and variable numbers of neutrophils were observed invading the wall of the affected vessels. Extensive leptomeningeal hemorrhage was accompanied by focal accumulations of neutrophils and macrophages which were more pronounced in the area of affected vessels. Similar cases of meningeal polyarteritis in dogs have been reported recently; we were unable to determine the cause of this lesion in our animal.

AFIP Diagnosis: 1) Vasculitis, characterized by fibrinoid necrosis, subacute, multifocal, severe, muscular arteries, meninges, spinal cord, boxer, canine. 2) Meningitis and radiculitis, pyogranulomatous, subacute, focal, severe, spinal cord. 3) Hemorrhage, subdural, acute, severe, spinal cord.

Conference Note: Although morphologic diagnoses submitted by participants varied, there was general agreement with the contributor. Due to the type of inflammation present, the size of vessels affected, and the extensive necrosis and hemorrhage seen, the lesions were thought to be similar to the necrotizing vasculitis associated with immune complex disease. A specific etiology could not be determined in this case. Causes of vasculitis in various species were discussed.

Contributor: Faculty of Veterinary Medicine, University of Montreal, St-Hyacinthe (Quebec) Canada J2S 7C6.

References:

1. Hoff, E. J. et al.: Case report: Necrotizing vasculitis in the central nervous systems of two dogs. Vet. Pathol. 18: 219-223, 1981.

2. Easley, J. R.: Necrotizing vasculitis: An overview. JAAHA 15: 207-211, 1979.

3. Kelly, D. F. et al.: Polyarteritis in the dog: A case report. Vet. Rec. 92: 363-366, 1973.

4. Fankhauser, R. et al.: Cerebrovascular disease in various animal species. Ann. NY Acad. 127: 817-860, 1965.

Case IV - (AFIP 1801670).

History: S-D rat kidney removed two days after a single intraperitoneal injection of 75 ug/Kg b.w. of gold sodium thiomalate. A section from the kidney of a control rat is included for comparison.

Laboratory Results: Plasma creatinine and urea nitrogen levels were markedly elevated over controls.

Contributor's Diagnosis & Comments: Acute proximal tubular necrosis, kidney, rat, following intraperitoneal injection of gold sodium thiomalate.

Comments: Administration of gold salts to rats results in acute necrosis of the terminal segment of the proximal convoluted tubule. Due to delays in macrophotography, the specimens (control and experimental kidneys) were kept at room temperature in physiological saline for 3 hours prior to immersion fixation with 10% buffered formalin.

AFIP Diagnosis: Necrosis, acute, tubular, diffuse, moderate to severe, cortex, kidney, rat, rodent, etiology, gold sodium thiomalate.

Contributor: Department of Pathology, School of Medicine, USUHS, Bethesda, Maryland 20014.

References:
1. Payne, B. J. (ed): The toxicity of 3 gold-containing compounds in laboratory animals. Vet. Pathol. 15: Suppl 5, 1978.
2. Nagi, A. H. et al.: Gold nephropathy in rats - Light and electron microscopic studies. Exp. & Molec. Path. 15: 354-362, 1971.
3. Yarom, R. et al.: Nephrotoxic effect of parenteral and intraarticular gold. Arch. Pathol. 99: 36-43, 1975.

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Results
AFIP Wednesday Slide Conference - No. 8
4 November 1981

Conference Moderator: George A. Parker, DVM
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Case I - 79-2 (AFIP 1710128).

History: Tissue from a 12-year-old male poodle that was presented moribund.

Contributor's Diagnosis & Comments: Atherosclerosis and arterial mineralization.

Comments: Arteries throughout the dog's body were affected, with the epicardial arteries being most severely involved.

AFIP Diagnosis: 1) Atherosclerosis, chronic, diffuse, severe, coronary arteries, poodle, canine. 2) Fatty infiltration, multifocal, mild, myocardium, heart.

Conference Note: Attendees unanimously concurred with the contributor. The extensive replacement in the arteries of both the media and intima by the atheromatous plaques is considered typical in the dog. Canine arterial smooth muscle cells in cell culture exposed to LDL's are known to accumulate approximately four times more cholesterol as compared with other species.[4] This difference in metabolism may account for the significant involvement of the media. Although the reason for the disease in this case was not known, atherosclerosis in dogs is usually associated with hypothyroidism. The Department of Cardiovascular Pathology stated that the changes seen in human cases are primarily intimal, usually focal and do not extend into the intra-myocardial branches. Foam cells are not commonly observed in the adventitia, and with an equivalent degree of circumferential thickening, the vessels are usually more severely reduced in diameter.

Contributor: Veterinary Medicine Division, Armed Forces Radiobiological Research Institute, Bethesda, MD 20014.

References:

1. Clarkson, T. B.: Animal models of atherosclerosis. Adv. Vet. Sci. Comp. Med. 16: 151-173, 1972.

2. Kinlough-Rathbone, R. L. et al.: Atherosclerosis. Am. J. Surg. 141: 638-643, 1981.

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4. Mahley, R. W. et al.: Canine hyperlipoproteinemia and atherosclerosis. Am. J. Pathol. 87: 205-219, 1977.

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Case II - 81-1 (AFIP 1562991).

History: Tissue from a beagle puppy that exhibited a progressive neurologic deficit. Some of the littermates were similarly affected. (2 electron micrographs included).

Contributor's Diagnosis & Comments: Canine globoid cell leukodystrophy (Krabbe's disease).

Comments: Globoid cell leukodystrophy, sometimes familial, has been reported in a number of breeds of dogs. Both the canine and human diseases appear to result from an inherited deficiency of galactocerebroside B-galactosidase. The present case was from a breeding establishment that was selectively propagating a "lemon" coat color. The clinical history suggested that several littermates and progeny from previous matings were affected, though tissue from only one dog was available for study. The diagnosis was based on characteristic light and electron microscopic findings.

AFIP Diagnosis: Radiculoleukodystrophy, histiocytic (globoid cell), chronic, multifocal, mild, beagle, canine, etiology, deficiency of galactocerebroside B-galactosidase.

Contributor: George A. Parker, D.V.M., 11101 Streamview Court, Great Falls, VA 22066.

References:

1. Fletcher, T. F. et al.: Globoid cell leukodystrophy (Krabbe type) in the dog. JAVMA 149: 165-172, 1966.

2. Yunis, E. J. et al.: The morphologic similarities of human and canine globoid leukodystrophy. Am. J. Pathol. 85: 99-114, 1976.

3. Fletcher, T. F. et al.: Animal model: Globoid cell leukodystrophy in the dog. Am. J. Pathol. 66: 375-378, 1972.

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5. Kurtz, H. J. et al.: The peripheral neuropathy of canine globoid-cell leukodystrophy (Krabbe-type). Acta Neuropath 16: 226-232, 1970.

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7. Fletcher, T. F. et al.: Ultrastructural features of globoid-cell leukodystrophy in the dog. Am. J. Vet. Res. 32: 177-181, 1971.

8. Zaki, F. A. et al.: Globoid cell leukodystrophy in a miniature poodle. JAVMA 163: 248-250, 1973.

9. Johnson, G. R. et al.: Globoid cell leukodystrophy in a beagle. JAVMA 167: 380-384, 1975.

10. Jolly, R. D. et al.: Inherited lysosomal storage diseases: An essay in comparative medicine. Vet. Rec. 92: 391-400, 1973.

11. Oehmichen, M. et al.: Cytochemical markers for mononuclear phagocytes as demonstrated in reactive microglia and globoid cells. Acta Histochem. 66: 243-252, 1980.

Case III - 80-12522 (AFIP 1803333).

History: Tissue from an aborted 7 to 8 month bovine fetus.

Laboratory Results: Indirect FA test for Sarcocystis bovicanis was positive on frozen fetal tissue.

Contributor's Diagnosis & Comments: Granulomatous encephalitis due to sarcocystosis.

Comments: Protozoan parasites compatible with Sarcocystis spp were present in vascular endothelial cells throughout the brain. Both mature and immature schizonts were observed.[2,4] In some instances, the zoites were arranged in rosette or palisade fashion, with zoites oriented about the periphery of the schizont. These various forms or stages of the schizont plus the obvious tropism for endothelial cells are consistent with Sarcocystis spp. Mouse inoculation of the fetal tissues and serology on the dam were negative for Toxoplasma gondii. Abortion has been reported when pregnant animals were infected with Sarcocystis spp both spontaneously and experimentally.[1-5] Congenital infection, however, with demonstrable organisms in the fetal tissues is not commonly reported.[4] The placenta was not available for examination in the present case.

AFIP Diagnosis: Encephalitis, perivascular, with intraendothelial protozoal cysts, subacute, multifocal, mild, brain, fetus, bovine, etiology, Sarcocystis cruzi (bovicanis).

Conference Note: Attendees essentially concurred with the contributor although many submitted Toxoplasma gondii as the most likely etiology. The moderator commented that differentiation between Sarcocystis and Toxoplasma tissue cysts is very difficult in histologic sections.

Contributor: Livestock Disease Diagnostic Center, University of Kentucky, 1429 Newtown Pike, Lexington, KY 40511.

References:

1. Corner, A. H. et al.: Dalmeny disease. An infection of cattle presumed to be caused by an unidentified protozoan. Can. Vet. J. 4: 252-264, 1963.

2. Dubey, J. P.: A review of Sarcocystis of domestic animals and of other coccidia of cats and dogs. JAVMA 169: 1061-1078.

3. Fayer, R. et al.: Abortion and other signs of disease in cows experimentally infected with Sarcocystis fusiformis from dogs. J. Infect. Dis. 134: 624-628, 1976.

4. Munday, B. L.: Suspected Sarcocystis infection of bovine placenta and foetus. Z. Parasiteuk. 51: 129-132, 1976.

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Case IV - A80-230 (AFIP 1801474).

History: A 13-year-old spayed female Welsh Pembroke Corgi dog presented with severe, constant hematuria. The dog had recurrent hematuria for at least four years and had first been examined at this hospital four years ago. Intravenous pyelogram at that time revealed unilateral nephrocalcinosis. The dog had been treated with a variety of antibiotics and the hematuria had been intermittent for the four years prior to this presentation. During one week of hospitalization the dog's hematocrit fell from 23% to 15%. The owner elected euthanasia rather than further diagnostic or therapeutic workup.

Contributor's Diagnosis and Comments: Telangiectasis of the kidney of the Welsh Pembroke Corgi dog; chronic mononuclear interstitial necphritis.

Comments: This condition has been diagnosed by histological evaluation in 8 Welsh Pembroke Corgis at Angell Memorial Animal Hospital. Six of the dogs had complete autopsies. The condition involved both kidneys and various other sites, including subcutaneous tissue, spleen, anterior mediastinum, retroperitoneal space, duodenal wall, and midbrain.

Multiple, firm to fluctuant, red-black masses several centimeters in diameter, were seen on the capsular surface of the kidneys. On cross sections multiple, red-black, sometimes cystic foci were seen within the medulla, clustering at the corticomedullary junction. The overlying cortex is decreased in width. Intrapelvic blood clots were frequent.

The following clinical signs are characteristic of the disease - lifelong recurrent hematuria with the passage of long linear clots in the urine. Intravenous pyelograms showed pyelorenal backflow and occasional nephrocalcinosis. These signs in a Welsh Pembroke Corgi are diagnostic of this syndrome. Several additional cases have been diagnosed clinically.

AFIP Diagnosis: Telangiectasia with infarction, chronic, focally extensive, severe, Welsh Pembroke Corgi, canine.

Conference Note: Diagnoses submitted included cavernous hemangioma, thrombosis with hemorrhage, and telangiectasia. The size, number, and proximity of the vascular spaces varies between sections, but extremely compressed parenchyma is evident between some of them as would be expected with telangiectasia.

Contributor: Angell Memorial Animal Hospital, Department of Pathology, 350 S. Huntington Ave., Boston, MA 02130.

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Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 9
18 November 1981

Conference Moderator: George D. Imes, Jr., DVM, MS
Lt Colonel, USAF, BSC(VC)
Diplomate ACVP, DIPLOMATE ACVPM
Chief, Division of Veterinary Pathology
Armed Forces Institute of Pathology

Case I - 468 (AFIP 1803076).

History: The tissue submitted is from a cow from a large dairy herd which had a high incidence of mastitis over a 2-year period. Prior to the increase in the incidence of mastitis a "homemade" mammary infusion was used. Milk cultures taken several times over the 2-year period failed to reveal an etiologic agent. (H&E and B&B provided).

Contributor's Diagnosis & Comments: Mastitis, chronic, suppurative, necrotizing. Nocardia asteroides etiology.

Comments: Nocardia asteroides was isolated from milk samples and mammary tissue from multiple affected animals in the herd. A Gram stain of mammary gland sections revealed numerous gram-positive branching filamentous organisms. Possible explanations for the negative culture results obtained by other laboratories over the 2-year period include 1) failure to incubate plates long enough to grow the organism, and 2) destruction of the organism by freezing. Milk samples were routinely frozen for shipment.

Control of the mastitis was achieved primarily by strict udder palpation and culling. Estimated losses from mastitis in this 3300 cow herd were 950 cows (450 died and 500 culled) over a 2-year period.

AFIP Diagnosis: Mastitis, necrotizing and pyogranulomatous, with gram-positive filamentous bacteria, lobular, severe, mammary gland, cow, bovine, etiology, Nocardia asteroides.

Conference Note: The bacteria were acid-fast and did not form "grains" in any of the sections examined. These characteristics are compatible with Nocardia asteroides. The morphologic appearance of sections is somewhat variable. Acini filled with recognizable neutrophils and necrotic debris surrounded by palisading macrophages and giant cells are present in some slides.

Contributor: New Mexico Veterinary Diagnostic Services, 700 Camino de Salud, NE, Albuquerque, NM 87106.

References:

1. Pier, A.C. et al.: Nocardia asteroides as a mammary pathogen of cattle. I. The disease in cattle and the comparative virulence of 5 states. Am. J. Vet. Res. 22: 502-517, 1961.

2. Pier, A.C. et al.: Nocardia asteroides as a mammary pathogen of cattle. II. The sources of nocardial infection and experimental reproduction of the disease. Am. J. Vet. Res. 22: 698-703, 1961.

3. Bushnell, R.B. et al.: Clinical and diagnostic aspects of herd problems with nocardial and mycobacterial mastitis. Amer. Assoc. Vet. Lab. Diag. 22nd Annual Proceedings, 1-2, 1979.

4. Hibbs, C.M. et al.: Nocardia mastitis in a large dairy herd. Amer. Assoc. Vet. Lab. Diag. 23rd Annual Proc., 73-78, 1980.

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Case II - H81-49 (AFIP 1806827).

History: A 5-month-old mixed breed pig from a group of 1200 pigs that became ill in a 5-day period. The affected animals showed a purplish discoloration of ears and nasal and abdominal skin. Nervous signs including incoordination, tremors and convulsions were often observed. The pigs had an elevated temperature of 40.5 to 41.5°C. (Two H&E sections provided).

Laboratory Results: A mild to pronounced leucopenia was evident in most affected pigs (including this case). Circular button ulcers of the colonic mucosa, hemorrhagic infarcts of the spleen and nonsuppurative meningoencephalitis were seen in most cases. Fluorescent antibody (FA) tests on frozen sections of tonsillar tissue were positive for hog cholera virus.

Contributor's Diagnosis & Comments: Toxoplasmosis in association with hog cholera and salmonellosis.

Comments: Impression smears of the hepatic lymph nodes stained with Giemsa stain were positive for Toxoplasma gondii. This case is representive of acute lesions where trophozoites and cysts with toxoplasma are numerous in the affected areas. However, the positive FA test on the frozen sections of the tonsillar tissue, and the gross and microscopical features of the visceral organs reveal that hog cholera is involved in the infection and may play a primary role in this case.

AFIP Diagnosis: 1) Pneumonia, interstitial, acute to subacute, diffuse, moderate, with multifocal alveolar necrosis and protozoa, lung, pig, porcine, etiology, Toxoplasma gondii. 2) Lymphadenitis, necrotizing with protozoa, acute, diffuse, severe, lymph node, etiology same as one.

Conference Note: Many participants believed that the history, the extensive numbers of Toxoplasma organisms, and the severe necrosis present indicated a predisposing viral infection. Most, however, did not feel that a definitive diagnosis could be made of hog cholera or African swine fever based upon the morphologic changes observed in these sections. Hemorrhage within the subcapsular space of the lymph node was not seen, and although necrotizing vasculitis was evident, it may have been secondary as most affected vessels were located within large areas and foci of parenchymal necrosis usually containing Toxoplasma organisms.

Contributor: Dept of Veterinary Medicine, Animal Industry Res. Institute, TSC No. 1, Tapu, Chunan, Miaoli, Taiwan, Republic of China.

References:
1. Dunne, H.: Hog cholera. IN Diseases of Swine, The Iowa State University Press, 1975, pp 189-255.
2. Siegmund, O.H. et al.: Toxoplasmosis. IN The Merck Veterinary Manual, Merck & Co., Inc., 1979, pp 466-469.
3. Gillespie, J.H. et al.: The Togaviridae. IN Hagan & Bruner's Infectious Diseases of Domestic Animals, Cornell University Press, Ithaca, NY, 1981, pp 665-675.
4. Jubb & Kennedy: Pathology of Domestic Animals, Vol. II, 2nd Ed., Academic Press, NY, 1970, pp 668-671.

Case III - N81-3252 (AFIP 1811026).

History: A 17-year-old mare was submitted for necropsy with a history of illness and marked weight loss over a 6-week period. Gross postmortem findings included enlargement of all thoracic and abdominal lymph nodes, ulceration of the mucosal surface of the cecum and terminal ileum, and multiple nodules scattered throughout the liver and lungs.

Contributor's Diagnosis & Comments: Granulomatous enteritis (mycobacterial).

Comments: Suspensions of lymph node given to chickens (i.v.) caused typical lesions of avian tuberculosis and death in 6 weeks. Similar material given intramuscularly and subcutaneously to 2 guinea pigs produced microscopic granulomas in multiple organs after 6 weeks but no acid-fast organisms were visible except at the inoculation sites. Guinea pigs and chickens were positive on intradermal skin tests using avian type tuberculin 3 weeks post-inoculation. Pure cultures of Mycobacteria were isolated on mycobacterial agar from the horse and inoculated chickens. The above studies confirm that the organism belongs to the Mycobacterium avium/Intracellulare group. Chickens are susceptible to M. avium, whereas guinea pigs are susceptible to M. tuberculosis, M. bovis, but are only slightly susceptible to M. avium.

AFIP Diagnosis: Enteritis, granulomatous with submucosal pyogranulomas, chronic, transmural, severe, small intestine, horse, equine, etiology Mycobacterium avium.

Conference Note: An acid-fast stained section is included with these results. Participants unanimously concurred with the contributor's morphologic diagnosis and considered one of the Mycobacterium species as the most likely etiologic agent. The moderator commented that the presence of nodular foci and necrosis in the submucosa are not typically seen with paratuberculosis. An auramine -o- rhodamine fluorescent technique for acid-fast organisms demonstrated intensely positive bacteria within macrophages and giant cells. In many of the latter, the density of organisms was so great that individual bacilli could not be discerned.

Contributor: Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, Saskatchewan, Canada S7N OWO.

References:
1. Griffith, A.S.: Types of tubercule bacilli in equine tuberculosis. J. Comp. Path. & Ther. 50: 159-172, 1937.
2. Lesslie, I.W. et al.: Tuberculosis in a horse caused by the avian type tubercle bacillus. Vet. Rec. 70: 82-84, 1958.
3. Baker, J. R.: A case of generalized avian tuberculosis in a horse. Vet. Rec. 93: 105-106, 1973.
4. Larsen, A.B. et al.: Susceptibility of horses to Mycobacterium paratuberculosis. AJVR 33: 2185-2189, 1972.

Case IV - C9-976 (AFIP 1734039).

History: Tissue submitted from a young feline with a history of severe dyspnea. The patient died post-operatively after exploratory surgery for intestinal obstruction.

Laboratory Results: Grossly the pulmonary vessels and bronchioles were thrombosed. The lungs revealed edema and pneumonia with congestion.

Contributor's Diagnosis: Pulmonary trematodiasis due to Paragonimus kellicotti.

AFIP Diagnosis: Bronchiolitis and bronchiolectasia, chronic, ulcerative, with squamous metaplasia and peribronchiolar gland hyperplasia, multifocal, severe, lung, domestic short hair, feline, etiology, Paragonimus kellicotti.

Conference Note: Several participants commented that multiple points were seen at the end of some tegmental spines consistent with P. kellicotti versus P. westermanii. The peribronchiolar interstitial pneumonia evident was considered to be an extension of the bronchiolitis. Medial hypertrophy of pulmonary arteries also is present in some sections and has been reported by a few authors to be related to the presence of these parasites as well as Aelurostrongylus abstrusus.

Contributor: Animal Health Laboratory, Centreville, MD 21617.

References:
1. Bisgard, G.E. et al.: Paragonimiasis in a dog and cat. JAVMA 144: 501-507, 1964.
2. Dubey, J.P. et al.: Induced paragonimiasis in cats: Clinical signs and diagnosis. JAVMA 173: 734-742, 1978.
3. Herman, L.H. et al.: Paragonimiasis in a cat. JAVMA 149: 753-757, 1966.
4. Wallace, F.G.: Lung flukes of the genus Paragonimus in American mink. JAVMA 78: 229-234, 1931.

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Results
AFIP Wednesday Slide Conference - No. 10
2 December 1981

Moderator: Ralph M. Bunte, D.V.M.
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Diplomate ACVP
Staff Pathologist
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AFIP, Wash, DC 20306

Case I - N81-484 (AFIP 1802991).

History. A 3-year-old female German shepherd dog presented with ataxia of a few weeks duration. The condition worsened to hindlimb paralysis and paresis of forelimbs. A bilateral panuveitis was noted. The rectal temperature was 103.6° F and persisted. Previous therapy had included steroids. (H&E and GMS provided).

Laboratory Results. Laboratory results showed a neutrophilia and increased levels of SGOT, SGPT, alkaline phosphatase, CPK, creatinine, and BUN. A vitreous aspirate showed septated hyphae. CSF fluid revealed an increased number of inflammatory cells, primarily monocytes. *Aspergillus fumigatus* was cultured from the spleen, kidney, heart, and eye.

Contributor's Diagnosis & Comments. Myocarditis, granulomatous, multifocal, severe, right ventricle. Etiology, fungal (*Aspergillus spp*).

Comment. Fungal hyphae and granulomatous lesions were found in the heart, kidneys, spleen, liver, skeletal muscles, eyes, meninges, and bone. The primary site of infection could not be determined. The respiratory, integumentary, and gastrointestinal systems were free from mycotic disease. Treatment with steroids might have contributed to dissemination of the fungus.

AFIP Diagnosis. Myocarditis, necrotizing, pyogranulomatous, subacute, multifocal, severe, heart, German shepherd, canine, etiology, *Aspergillus sp*.

Conference Note. Differential etiologic diagnoses submitted by participants included aspergillosis, candidiasis, and phycomycosis. The presence of dilated bulbous structures was considered somewhat unusual for *Aspergillus sp*. These are thought to be vesicular swellings of the hyphae and to occur when growth conditions are less than optimal. The characteristic "sunburst" pattern or actinomycetoid form of the fungus is present in most GMS stained sections.

Contributor. Division of Comparative Pathology, Box J-103, University of Florida, Gainesville, Florida 32610.

References.

1. Chandler, E. A.: *Aspergillus* infection of the nasal cavity of the dog: Diagnosis and treatment. Vet. Rec. 96: 156, 1975.

2. Wood, J. L. and Heish, D. C.: Disseminated aspergillosis in a dog. JAVMA 172: 704, 1978.

3. Lane, J. G. and Warnock, D. W.: The diagnosis of *Aspergillus fumigatus* infection of the nasal chambers of the dog with particular reference to the value of the double diffusion test. J. Sm. Anim. Pract. 18: 169, 1977.

4. Chandler, F. W.: Histopathology of Mycotic Diseases. Yearbook Medical Publishers Inc., Chicago, 1980, pp 34-38.

Case II - LAIR 30696-27 (AFIP 1811146).

History. The tissue is from an adult male African Green monkey (Cercopithecus aethiops) that had been maintained in a research colony for 6 years. In Nov 1975 and May 1976 the animal was used for brief pharmacokinetic studies of DEHP, a material used in the manufacture of plastics. No other experimental procedures were done to this animal. In Feb 1979 a grade 4 heart murmur was detected on routine physical examination. Except for the murmur, the animal remained clinically normal until 8 May 1981 when anorexia, depression, and dyspnea were noted. These signs became progressively more severe over a period of 2 weeks. No treatment was given and the animal was found dead in its cage on 27 May 1981.

Contributor's Diagnosis & Comment.

Morphologic diagnoses: 1) Thrombosis, subacute, diffuse, severe, pulmonary arteries, with focally severe pulmonary infarction, lung, African green monkey, (Cercopithecus aethiops). 2) Subintimal fibroelastosis and medial hypertrophy, chronic, multifocal, severe, muscular arteries, lung.

Syndrome diagnosis: Chronic congestive heart failure with pulmonary artery thrombosis and pulmonary infarction.

Comment. Gross necropsy revealed a large globoid heart that contained large thrombi in the right atrium and both ventricles. Both mitral and tricuspid valves were grossly thickened. The main branches of the pulmonary artery were almost completely filled by large mural thrombi. The lobes of the left lung did not collapse and the ventral aspect of these lobes were firm, dark red and sharply demarcated from more normal appearing dorsal areas. The liver was swollen and had an accentuated lobular pattern on both capsular and cut surfaces.

Histologically, there were severe diffuse degenerative changes in all areas of the myocardium and 1 large (2 cm in diameter) well-demarcated area of fibrosis extending throughout the thickness of the left ventricular wall that appeared to be a healed infarct. The liver had histologic lesions compatible with those of chronic passive congestion.

Thrombosis of the main branches of the pulmonary artery occurs in cases of 1) reduced blood flow; 2) clotting abnormalities; 3) damage to vessel walls. Because of the dual blood supply to the lungs, infarction does not usually result from thrombosis of large pulmonary arteries unless there is one or more of the following: 1) obstruction of venous return; 2) very low cardiac output, or 3) sepsis. The presence of numerous "heart failure cells" in the lung, typical hepatic lesions of chronic passive congestion, and the diffuse severe chronic degenerative changes in the heart of this monkey indicates that decreased cardiac output was the probable cause. The lesions in small and intermediate sized muscular arteries in the lung are of special interest. Paradoxically, lesions of this type may be produced by either reduced cardiac output or by chronic pulmonary hypertension. In both instances, sustained vasoconstriction is thought to be responsible for the medial hypertrophy and intimal fibroelastosis of these vessels. In this case, the lesions in the small arteries were probably a further consequence of long-standing reduction in cardiac output since there was no evidence of venous thrombosis, mitral stenosis or other lesions that would compromise venous return. The pathogenesis of these interesting vascular lesions is discussed in detail in the referenced book.

AFIP Diagnosis. 1) Infarcts, chronic, multifocal, severe, lung, African green monkey (Cercopithecus aethiops), nonhuman primate, etiology, multifocal thrombi. 2) Proliferation, intimal, and hypertrophy, medial, chronic, multifocal, mild to moderate, arteries, lung. 3) Hemosiderosis, chronic, diffuse, moderate, lung.

Contributor. Division of Research Support, Letterman Army Institute of Research, Presidio of San Francisco, CA 94129.

Reference.

Spencer, H.: Pathology of the Lung (3rd Ed.), Vol. II, Chapt 15, Pulmonary thrombosis, fibrin thrombosis, pulmonary embolism and infarction, and Chapt 16, Chronic pulmonary hypertension. Pergamon Press, New, 1977, pp 543-649.

Case III - 19-2633 (AFIP 1802915).

History: A moribund, 744-day-old male Fischer 344 rat was submitted for necropsy. The animal was icteric. At necropsy, the spleen was found to be markedly enlarged.

Laboratory Results:

RBC - 0.94 x $10^6/mm^3$
WBC - 50.7 x $10^3/mm^3$
Hb - 3.8g%
PCV - 11.5%
Total bilirubin - 33 mg%

Contributor's Diagnosis & Comments. Mononuclear leukemia.

Comment. Mononuclear leukemia is a common disease of aged Fischer 344 rats. The neoplasm appears to originate in the spleen; bone marrow is rarely affected. Involvement of the liver and lymph nodes is common. The cell line from which the neoplasm originates has not been identified. A profound hemolytic anemia always accompanies the disease.

AFIP Diagnosis. 1) Leukemia, mononuclear cell, liver and spleen, F344 rat, rodent. 2) Hyperplasia, bile ducts, multifocal, mild, liver.

Contributor. Lovelace Inhalation Toxicology Research Institute, P.O. Box 5890, Albuquerque, New Mexico 87185.

References.

1. Coleman, G. L., Barthold, S. W., Osbaldiston, G. W.: Pathological changes during aging in barrier-reared Fischer 344 male rats. J. Gerontol. 32: 258-278, 1977.

2. Davey, F. R., Moloney, W. C.: Postmortem observations on Fisher rats with leukemia and other disorders. Lab. Invest. 23: 327-334, 1970.

3. Moloney, W. C., Batata, M., King, V.: Leukemogenesis in the rat: Further observations. J. Natl. Cancer Inst. 46: 1139-1144, 1971.

4. Moloney, W. C., Boschetti, A. E., King, V. P.: Spontaneous leukemia in Fischer rats. Can. Res. 30: 41-43, 1970.

5. Moloney, W. C., King, V. P.: Reduction of leukemia incidence following splenectomy in the rat. Can. Res. 33: 573-574, 1973.

6. Squire, R. A. et al.: Tumors. Chapt 12, Pathology of Laboratory Animals, Vol. II, Benirschke, K. et al. (ed), Springer-Verlag, New York, 1978, pp 1117-1118.

Case IV - 80-3574 (AFIP 1757299).

History: Nude mouse, male. One of several nude mice which appeared listless and weak 3 weeks after arrival. On necropsy, livers of some animals had a rough surface whereas others had abscesses.

Laboratory Results. Pseudomonas aeruginosa was isolated in almost pure culture. A limited number of Staphylococcus aureus colonies also were isolated and were thought to be present due to collection technique.

Contributor's Diagnosis & Comments. Severe subacute to chronic multifocal necrotizing hepatitis.

Comment. Pseudomonas aeruginosa infections in mice usually localize in the ear causing head tilt and circling. In these nude mice the primary target organ was the liver.

AFIP Diagnosis. Hepatitis, pyogranulomatous and necrotizing, subacute, multifocal, moderate, liver, nude mouse, rodent.

Conference Note. Several participants thought the mice may have been in the recovery stage of mouse hepatitis virus. However, intracytoplasmic inclusion bodies were not found within hepatocytes, and the portal and perilobular distribution of the lesions is more suggestive of a bacterial infection.

Contributor. Life Sciences Research, 10 W. 35th St., Chicago, Illinois 60616.

References.

1. Benirschke, K., Garner, F. M. and Jones, T. C.: Pathology of Laboratory Animals, Springer-Verlag, New York, 1978, p. 251; pp 1439-1442.

2. Ediger, R. D. et al.: Circling in mice caused by Pseudomonas aeruginosa. Lab. Anim. Sci. 21: 845-848, 1971.

3. Pollack, M.: Pseudomonas aeruginosa exotoxin A. N. Eng. J. Med. 302: 1360-1361, 1980.

4. Bartell, P. F. et al.: The lethal events in experimental Pseudomonas aeruginosa infection of mice. J. Inf. Dis. 118: 165-172, 1968.

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Results
AFIP Wednesday Slide Conference - No. 11
9 December 1981

Conference Moderator: William C. Hall, VMD, PhD
Director, Veterinary Pathology
Microbiological Associates
5221 River Road
Bethesda, MD 20016

Case I - 2553-1 (AFIP 1803314).

History. Unvaccinated coonhound puppy, 6 weeks of age, submitted to referring veterinarian with complaint of purulent rhinitis, conjunctivitis, anorexia, and depression. Clinical examination revealed moist rales with dyspnea, a temperature of 104°F, and a white blood count of 2,200.

Laboratory Results. At necropsy whitish foci less than 1 mm in diameter were noted throughout the liver. Diffuse atelectasis of the apical and cardiac lung lobes was seen.

Contributor's Diagnosis & Comment. 1) Pneumonitis, interstitial, mild, diffuse. 2) Hepatitis, multifocal, severe.

Comment. Eosinophilic inclusion bodies are seen in the cytoplasm of the alveolar lining cells and occasionally the bronchiolar epithelium. Rarely are the inclusions intranuclear. Clusters of filamentous organisms morphologically similar to Bacillus piliformis are seen in hepatocytes at the periphery of necrotic foci. Etiologic diagnosis: 1) Canine distemper, lung. 2) Tyzzer's disease, liver.

AFIP Diagnosis. 1) Pneumonia, interstitial, subacute, diffuse, mild to moderate, with intracytoplasmic and rare intranuclear eosinophilic inclusions, lung, coonhound, canine, etiology, canine distemper virus (morbillivirus). 2) Hepatitis, necrotizing, acute, multifocal, severe, with intracytoplasmic bacilli, liver, etiology, Bacillus piliformis (Tyzzer's disease).

Contributor. Department of Veterinary Science, University of Nebraska, Lincoln, Nebraska 68583.

References.

1. Poonacha, K. B. and Smith, H. L.: Naturally occurring Tyzzer's disease as a complication of distemper and mycotic pneumonia in a dog. J. Am. Vet. Med. Assoc. 169 (4): 419-420, 1976.

2. Qureshi, S. R., Carlton, W. W. and Olander, H. J.: Tyzzer's disease in a dog. J. Am. Vet. Med. Assoc. 168 (7): 602-604, 1976.

Case II - 9130-81 (AFIP 1803023).

History: A 9-week-old Bassett hound had 3 episodes of illness over a one-month period of time. Between the episodes the puppy appeared to be in good health. The first time the pup was listless. The second time it had diarrhea. The third time the pup was vomiting. The WBC was 28,000 and the serum was very icteric. The dog went into a coma and died. The puppy was vaccinated against ICH 5 days before the first episode of illness.

Contributor's Diagnosis & Comment. Hepatic fibrosis with central to central and central to portal bridging; widespread vacuolar change; single cell necrosis; parenchymal inflammation with scattered neutrophils and lymphocytes; bile duct proliferation.

Comment. The histopathology is suggestive of postnecrotic cirrhosis.[2] In this case it is probably due to ICH in a partially immune dog. The disease in this dog has been called chronic active hepatitis in the literature.[5]

AFIP Diagnosis. Hepatitis, subacute, with vacuolar change and individual hepatocellular necrosis, diffuse, moderate, liver, Bassett hound, canine.

Conference Note. Most participants submitted a diagnosis of subacute hepatitis with diffuse vacuolar change. Etiologies included infectious canine hepatitis virus, toxins, and glycogen storage disease. Attendees were unsure if any fibrosis was present based only upon H&E stained sections, and had varying opinions concerning the identity of the mononuclear cells located between hepatocytes and along the distorted sinusoids. These were thought to be a combination of extremely attenuated and squeezed hepatocytes, circulating lymphocytes and macrophages, Kupffer cells in increased numbers due to condensation of stroma following loss of parenchyma, and possibly fibroblasts. Plasma cells were not seen in any sections. The AFIP Masson's trichrome stained section did not reveal fibrosis as described by the contributor. The severe vacuolar change was believed to represent glycogen infiltration either induced by endogenous or exogenous corticosteroids, or as a degenerative process associated with the hepatitis.

Contributor. Department of Veterinary Pathology, College of Veterinary Medicine, University of Missouri, Columbia, Missouri 65211.

References.

1. Bishop, L., Strandberg, J. D., Adams, R. J. et al.: Chronic active hepatitis in dogs associated with leptospires. Am. J. Vet. Res. 40: 839-844, 1979.

2. Gall, E. A.: Posthepatic, postnecrotic and nutritional cirrhosis. Am. J. Pathol. 36: 241-258, 1960.

3. Gocke, D. J., Morris, T. Q., and Bradley, S. E.: Chronic hepatitis in the dog: The role of immune factors. J. Am. Vet. Med. Assoc. 156: 1700-1705, 1970.

4. Meyer, D. J., Iverson, W. O., Terrell, T. G.: Obstructive jaundice associated with chronic active hepatitis in a dog. J. Am. Vet. Med. Assoc. 176: 41-44, 1980.

5. Strombeck, D. R. and Gribble, D.: Chronic active hepatitis in the dog. J. Am. Vet. Med. Assoc. 173: 380-386, 1978.

Case III - AB1651 or AB1758 or AB1534 (AFIP 1806696).

History. Heart from a 3-month-old guinea pig. The animal was clinically normal.

Laboratory Results. Hematology and a battery of 12 clinical chemistries including SGOT, SGPT, and Alkaline Phosphatase were normal.

Contributor's Diagnosis & Comment. Rhabdomyomatosis.

Comment. Similar myocardial lesions were present in three additional guinea pigs from a group of 40 animals. The exact nature of this lesion is apparently unknown. The vacuolation of the myocytes is primarily due to glycogen accumulation. The lesion has been variously designated as degenerative, a storage disease, and as congenital. Similar lesions in humans have been termed rhabdomyomas. (Some slides show only a small portion of the lesion.)

AFIP Diagnosis. Rhabdomyomatosis, multifocal, moderate, myocardium, heart, guinea pig, rodent.

Contributor. Department of Pathology, ToxiGenics, Inc., 1800 East Pershing Road, Decatur, Illinois 62526.

References.

1. Hueper, W. C.: Rhabdomyomatosis of the heart in a guinea pig. Am. J. Pathol. 17: 121-124, 1941.

2. Rooney, J. R.: Rhabdomyomatosis in the heart of the guinea pig. Cornell Vet. 51: 388-394, 1961.

3. Vink. H. H.: Rhabdomyomatosis (nodular glycogenic infiltration) of the heart of guinea pigs. J. Pathol. 97: 331-334, 1969.

Case IV - M20428 (AFIP 1810447).

History: 4-week-old NFS/N mouse injected at birth with a C-type virus into the peritoneal cavity. A greatly enlarged spleen was noted grossly. No other lesions were reported.

Contributor's Diagnosis & Comment. Viral-induced peritoneal sarcoma with metastasis to the spleen. With special stains the tumor cells produced collagen and thus, the diagnosis is fibrosarcoma. Note the presence of giant cells and inflammation.

Comment. The necropsy was performed by technicians and virologists who noted the major and only lesion was an enlarged spleen. After a histogenesis study by a pathologist, the sarcomas were seen to arise on mesothelial surfaces of the omentum, mesentery, and diaphragm and subsequently metastasized to the spleen. When the spleen was involved the splenic tumors were always much larger than the primary tumors. The primary tumors were usually small and may have und rgone regression.

AFIP Diagnosis. Fibrosarcoma, spleen, NFS/N mouse, rodent, etiology, C-type sarcoma virus.

Conference Note. Rhabdomyosarcoma was the most frequent diagnosis submitted based upon the presence of multinucleated tumor cells and apparent strap cells. Other diagnoses included anaplastic sarcoma, reticulum cell sarcoma type A (composite lymphoma), granulomatous splenitis, fibrosarcoma, and leiomyosarcoma. The pleomorphic and atypical appearance of many of the spindle cells was not considered compatible with a granulomatous response. A Masson's trichrome stain revealed a substantial amount of collagen and other special stains failed to demonstrate cross-striations. The C-type virus utilized is a sarcoma virus which has not been reported previously in the literature. Studies are being performed currently to characterize it further.

Contributor. Laboratory of Comparative Carcinogenesis, National Cancer Institute-FCRC, Building 538, Frederick, Maryland 21701.

References.

1. Benirschke, K., Garner, F., Jones, T. C.: Pathology of Laboratory Animals, Vol. II, Springer-Verlag, New York, 1978, pp 1068-1069, 1112-1113, 1307-1308.

2. Harvey, J. J., Rager-Zisman, B., Wheelock, E. F. et al.: Factors affecting responses to murine oncogenic viral infections. Br. J. Canc. 41: 577-586, 1980.

3. Siegler, R.: Pathogenesis of virus-induced murine sarcoma. I. Light microscopy. J. Nat'l. Canc. Inst. 45: 135-147, 1970.

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Results
AFIP Wednesday Slide Conference - No. 12
16 December 1981

Conference Moderator: George Migaki, D.V.M.
Diplomate, ACVP
Registry of Comparative Pathology
Armed Forces Institute of Pathology
Washington, DC 20306

Case I - 2732 (AFIP 1803531).

History: A 4-5 year old range cow had a firm, well circumscribed, pedunculated tissue mass protruding from below the intermandibular space. It was surgically removed without complications or subsequent recurrence.

Contributor's Diagnosis & Comment. Pyogranulomatous inflammation consistent with actinobacillosis.

Comment. Bacteria consistent with Actinobacillus sp. were demonstrated with special stains and with electron microscopy. The Splendore-Hoeppli reaction around organisms is well developed.

AFIP Diagnosis. Granulation tissue with focal and coalescing granulomas, chronic, focally extensive, severe, intermandibular soft tissue, cow, bovine, etiology, probably Actinobacillus lignieresi.

Contributor. Syntex Research, 3401 Hillview Ave., Palo Alto, CA 94304.

References.

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Case II - 81-3083 (AFIP 1804673).

History: Lung from a 4-year-old cow which died suddenly. She was from a herd of 40 dairy cows kept on pasture. At necropsy, the lungs were congested and did not collapse. There were foci of alveolar emphysema and bronchi were filled with a white foamy material. A large abscess was present in the liver.

Contributor's Diagnosis & Comment. Generalized pulmonary embolism from a ruptured liver abscess.

Comment. Histological examination of the lungs revealed generalized congestion and alveolar edema and hemorrhages. Several small pulmonary vessels were obstructed with gram-negative filamentous bacteria (Fusobacterium necrophorum) and necrotic debris. Inflammatory cells were not present in the lung parenchyma. Generalized massive pulmonary embolism resulting from the rupture of a liver abscess should always be considered in the differential diagnosis of sudden deaths in cattle. Death seems to occur rapidly. In most of the cases we have seen, inflammation was absent despite the presence of the septic emboli (bacteria and pus) in the microcirculation. These animals die because of acute respiratory failure.

AFIP Diagnosis. Emboli, septic, pulmonary vessels, with hemorrhage and edema, peracute, multifocal, moderate, lung, cow, bovine, etiology, Fusobacterium necrophorum.

Conference Note. Bacteria were observed within alveolar capillaries as well as larger pulmonary vessels. The pulmonary edema and hemorrhage were considered secondary to thromboemboli and endothelial damage from a concommitant septicemia. One attendee commented that postmortem overgrowth of organisms resulting from an agonal bacteremia should be considered differentially in cases with multiple bacterial thromboemboli.

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Case III - (AFIP 1791887).

History: Liver from a male (300 gm) harlequin duck from Sea World, San Diego. Animal had no apparent previous illness and was found dead in the water. At necropsy, the duck appeared to have had weight loss, and the liver was brown-gray with a white film over the capsule.

Contributor's Diagnosis. 1) Amyloidosis. 2) Schistosomiasis. Note the schistosome eggs with associated granulomas.

AFIP Diagnosis. 1) Amyloidosis, vascular, sinusoidal, and capsular, diffuse, moderate, liver, Harlequin duck, avian. 2) Granulomas, multifocal, mild to moderate, liver, etiology, schistosomatid eggs. 3) Vasculitis, intramural and perivascular, subacute, diffuse, mild to moderate, liver. 4) Vacuolar change, diffuse, moderate to severe, liver. 5) Capsulitis, subacute, multifocal, mild, liver.

Conference Note. Participants unanimously concurred with the contributor. Parasite eggs containing a miracidium were evident, but without adults the genus and species could not be determined. The presence of spines was considered doubtful and the projections observed were due most likely to a folded shell. Spines are difficult to identify in tissue section and when seen appear as a thickened projection of the egg shell. Although the presence of amyloid may have been related to the schistosome infection, its frequent occurrence in waterfowl lacking the parasite and its absence in some birds that are parasitized make such a correlation difficult. The distribution of amyloid in this case demonstrates that deposition occurs first within vessel walls and subsequently within the sinusoids. Primary amyloidosis has not been reported in birds. Although some participants thought that migration of parasite eggs through the vessel walls caused the multifocal intramural vasculitis, similar inflammatory cells were seen perivascularly throughout the parenchyma and within the capsule. Therefore, these changes could not be considered consclusively a result of the schistosome infection.

Contributor. Sea World, San Diego, California.

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Case IV - 81-14458 (AFIP 1810707).

History: A group of 250 2-3 month old feeder pigs became sick over a 2-week period. Nearly all pigs appeared ill and 10 had died. Mild diarrhea without blood was evident in a few pigs. Purple discoloration of the skin on ears and bellies was sometimes evident.

Laboratory Results. Microscopic lesions in addition to those of the liver were focal nonsuppurative meningitis, focal interstitial pneumonia and mild atrophic enteritis. Salmonella cholerasuis was isolated from internal organs of 3 pigs submitted. Hemophilus parasuis also was isolated from the lung. FAT and negative contrast EM examination were positive for rotavirus.

Contributor's Diagnosis. Hepatitis, necrotic, multifocal, disseminated, severe, etiology--Salmonella cholerasuis.

AFIP Diagnosis. Hepatitis, necrotizing and granulomatous, subacute, multifocal, moderate, with diffuse triiditis, pig, porcine, etiology, Salmonella cholerasuis.

Conference Note. The amount of necrosis in sections is variable. Based upon the clinical signs, age of animals, and morphologic changes in the liver, participants unanimously considered hepatic salmonellosis the most likely etiologic diagnosis. Special stains failed to demonstrate bacteria. Porcine salmonellosis unlike the disease in calves, rarely results in recognizable bacterial colonies in liver sections.

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Results
AFIP Wednesday Slide Conference - No. 13
6 January 1982

Conference Moderator. Robert M. Sauer, D.V.M.
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Case I - NBl-54-9 (AFIP 1802809).

History. Tissue from an 8-year-old female Dachshund with a 6-month history of chronic diarrhea. The dog originated in Memphis, Tennessee.

Laboratory Results. Histoplasma sp. organisms were seen on a rectal biopsy and scraping.

Contributor's Diagnosis & Comment. Histoplasmosis.
The liver, adrenal glands and regional lymph nodes were also involved..

AFIP Diagnosis. Colitis, granulomatous, transmural, chronic, diffuse, severe, colon, dachshund, canine, etiology, Histoplasma capsulatum.

Conference Note. There was unanimous agreement with the contributor. The histologic changes are essentially similar to those described in Jubb & Kennedy.[2]

Contributor. Department of Veterinary Pathology, University of Georgia, Athens, Georgia 30602.

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Case II - 1971-E, H, or I-1981 (AFIP 1809912).

History. An incidental finding at necropsy in an experimental baboon (Papio).

Contributor's Diagnosis & Comments. Morphological - Granulomas, eosinophilic-leukocyte, liver, baboon (Papio). Etiological - Protozoan hepatitis (hepatocystosis). Etiologic agent - Hepatocystis kochi.

Comments. Some of the granulomas contained central multiloculated cysts (merocystic megaloschizonts) with a peripheral zone filled with myriads of coccoid to pyriform basophilic organisms consistent with merozoites of the asexual reproductive cycle of Hepatocystis kochi. The cyst centers contained vacuoles and amorphous eosinophilic material. The locules of the cysts were surrounded by a dense perimeter of mixed inflammatory cells including eosinophils, macrophages, lymphocytes, and plasma cells. Multinucleated giant cells were present in some of the sections. Other granulomas contained only remnants of ruptured cysts, scattered merozoites, refractile eosin-staining lancelet and rectangular crystals, erythrocytes, fibrin, and a pleocellular inflammatory infiltrate. In many sections periportal regions of the liver contained inflammatory cells of the same types already described.

Hepatocystis kochi is a malarial parasite that is endemic to several parts of Africa. The protozoan is transmitted by midges (Culicoides sp) found in the endemic regions. Gametocytes develop in erythrocytes after merozoites penetrate the erythrocyte and undergo metamorphosis. The gametocytes are ingested by the insect host during a blood meal and sporogony ensues in the gut of the insect to produce the infective sporozoites. The parasite is not transmitted in the United States because we apparently lack the specific insect vector. Consequently all the parasitized primates in the colonies in this country are considered to have been infected prior to their arrival. Therefore, to explain the presence of varying developmental stages of this parasite in American baboons, some investigators believe that the asexual state apparently perpetuates itself by continued cycles of schizogony in the hosts' liver. Gametocytes can also be seen in blood smears within the hosts' erythrocytes.

AFIP Diagnosis. 1) Granuloma, eosinophilic, solitary, with a central protozoal merocyst, liver, baboon (Papio sp), nonhuman primate, etiology, Hepatocystis sp. 2) Hepatitis, periportal, subacute, multifocal, mild, liver.

Conference Note. The morphological characteristics of the merocysts were considered most compatible with Hepatocystis simiae in accordance with the criteria reported by Garnham.[2] These include a multilocular mature merocyst which attains a size of 300 μ and contains polyhedral crystals similar to those present in this case. In contrast, the mature cyst of H. kochi attains a diameter of 100 μ, is simple, has a rim of merozoites surrounding a single large vacuole, and does not contain crystals. The crystals in this case were anisotropic. Garnham states that they have been determined cytochemically to consist of protein including the amino acids tyrosine, tryptophan, and arginine. The presence of extensive numbers of eosinophils suggested that they could have been Charcot-Leyden crystals, but the latter are not anisotropic. The merocysts in some sections were thought to have ruptured and made the diagnosis difficult. The stages in the invertebrate host are considered similar for both H. kochi and H. simiae. Since Hepatocystis sp. does not undergo schizogony within erythrocytes it is considered distinct from the closely related Plasmodium genus.

Contributor. Pathology Branch, Toxic Hazards Division, Air Force Aerospace Medical Research Laboratory, Wright-Patterson AFB, OH 45433.

References.

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Case III - 81-166 (194-A) (AFIP 1801534). Gross photograph included.

History. A 5-year-old crossbred dog had an 8-week history of lethargy, weight loss, and occasional vomiting which on one occasion contained a small amount of blood. Mucous membranes were pale. Radiography revealed a large mass lesion in the stomach. The lesion illustrated was removed by partial gastrectomy. Six months after surgery the dog had returned to good health.

Laboratory Results. Severe iron deficiency anemia, low serum iron and elevated total iron binding capacity, hypovitaminosis B_{12} and low serum folate.

Contributor's Diagnosis & Comment. Idiopathic hypertrophic gastritis.

Comment. There are marked gastric mucosal hyperplastic and metaplastic changes with an associated infiltrate of chronic inflammatory cells in the lamina propria. The case is presented as an example of a canine lesion similar to Menetrier's disease in man, with associated severe depletion of body iron, Vitamin B_{12} and folate. Similar lesions have been described in Basengi, boxer and bull terrier dogs.

AFIP Diagnosis. Gastritis and mucosal hyperplasia, chronic, focally extensive, moderate to severe, stomach, mixed breed, canine.

Conference Note. All previous reports of this syndrome in dogs describe normal proportions of chief cells, parietal cells and mucus cells. The parietal cells in this case were thought to outnumber the chief cells extensively, a characteristic of the Zollinger-Ellison Syndrome in man. Although the chronological relationship between the hyperplasia and gastritis could not be substantiated histologically, the latter was thought probably to have occurred first.

Contributor. School of Veterinary Studies, Murdoch University, Murdoch, Western Australia, 6150.

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Case IV - S-10210 (AFIP 1811156).

History. An 11-13 year old male castrate domestic shorthair cat had dyspnea and a three month history of chronic unilateral nasal discharge which was sometimes bloody. Treatment with amoxycillin and flucytosine for three weeks was unrewarding. The animal was euthanatized.

Laboratory Results. Titers for Penicillium sp. and Aspergillus sp. were negative. CBC's performed were normal. A Feleuk Test was negative. Culture of the nasal exudate was negative and only inflammatory cells were seen with cytology. Radiographs revealed a soft tissue mass in the right nasal passage. No bone lysis was evident.

Contributor's Diagnosis & Comments. Carcinoma of the nasal cavity.

Comment. Carcinomas of the nasal cavity are uncommon in domestic animals. They are categorized as squamous cell, undifferentiated, adenocarcinoma, mucous cell or transitional carcinoma. The anaplasia, cell size, polyhedral shape, lack of intercellular bridging and arrangement of cells in tightly packed sheets and cords are characteristic of transitional carcinomas. Mucicarmine and PAS-Alcian blue stains were negative for mucus.

AFIP Diagnosis. Carcinoma, transitional, right nasal cavity, domestic short-hair, feline.

Conference Note. The two most frequent diagnoses submitted were undifferentiated carcinoma and histiocytic lymphosarcoma. The transitional cell carcinoma is a classification used by the WHO to designate a type intermediate between the squamous cell and undifferentiated carcinoma. In addition to the characteristics described by the contributor these neoplasms have distinctive oval vesicular nuclei with peripheral concentrations of chromatin and frequent mitotic figures. They are uncommon in domestic animals.

Contributor. Merck Institute for Therapeutic Research, West Point, PA 19486.

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Results
AFIP Wednesday Slide Conference - No. 14
13 January 1982

Conference Moderator: George D. Imes, Jr.
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Case I - 8721, 8605 (AFIP 1802979).

History. Tissue from C57BL/6 female retired breeder mice (6-8 months old). The mice were submitted from a large breeding facility and were clinically normal.

Laboratory Results. At necropsy the lungs had multiple, tan, 2-3 mm foci with darkened centers.

Contributor's Diagnosis & Comments. Pneumonia, multifocal, Mycobacterium avium. Comments. These mice were submitted from a large, commercial breeding facility of barrier design for routine health surveillance. On repeated sampling of the affected subpopulation, virtually all retired breeder C57BL/6 females examined had similar lung lesions and suppurative otitis media. Special stains revealed an acid fast organism in lung alveolar macrophages and in the middle ears. Mycobacterium avium was isolated and the identity of the organism was confirmed by the Center for Disease Control in Atlanta. Cultures were negative for other bacterial pathogens and Mycoplasmas, and virus serologies were negative. The source of infection of this colony with M. avium is presently a mystery.

AFIP Diagnosis. 1) Pneumonia, peribronchiolar, granulomatous, multifocal, mild, lung, C57BL/6 mouse, rodent, etiology, Mycobacterium sp. 2) Hyperplasia, lymphoid, and sinus histiocystosis with occasional intracellular acid fast bacilli, diffuse, mild, tracheobronchial lymph node.

Conference Note. The majority of participants considered a bacterial agent the most likely etiology based upon the presence of faintly staining bacillary forms within some macrophages. Foreign body pneumonia and mycoplasmosis were other etiologies submitted. Langhans' type giant cells were observed in some sections. The peribronchiolar distribution was suggestive of airway exposure to the agent. Acid fast stains demonstrated a moderate number of organisms within the macrophages associated with the peribronchiolar infiltrates and a few within occasional macrophages in the lymph node. Single foamy alveolar macrophages located away from the pulmonary infiltrates did not contain organisms and were considered within normal limits for a mouse lung. Although no published reports of M. avium in mice were found, several geriatric mice from a single colony were submitted previously to our Department with lung lesions similar to those in this case (although more severe). There were numerous acid fast organisms present which were morphologically compatible with a Mycobacterium species other than M. bovis or M. tuberculosis. The origin for these organisms was unknown and culture had not been performed prior to submission.

Contributor. Department of Comparative Medicine, University of Alabama, School of Medicine & Dentistry, University Station, Birmingham, Alabama 35294.

References.
1. Bergman, R., and Holmberg, O.: Occurrence and characterization of "Avium-like" Mycobacteria isolated from animals in Sweden. Acta Path. Micro. Scand. Section B 87: 363-369, 1979.
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Case II - 076 412 and 076 464 (AFIP 1810369). Two H&E slides provided.

History. Both cases are from untreated virgin yellow female A^{VY}/A mice produced by mating the mammary tumor virus positive C3H/HeNlCrWf females with (A^{VY}/A) males of the VY/Wf strain.

076 412 is 196 days of age and had four mammary adenocarcinomas (Dunn's type B). The tumors were first observed 42 days prior to necropsy.

076 464 is 194 days of age and had no mammary adenocarcinomas.

Contributor's Diagnosis & Comments. 076 412 glomerulonephritis, proliferative, severe. 076 464 glomerulonephritis, proliferative, very minimal.

Comments. Both mice were from the same experiment which consisted of 465 untreated mice, of which 401 were available for analysis. Two hundred and thirty three mice developed one or more mammary adenocarcinomas of which the vast majority were of the Dunn's type B variety. The renal lesion, a proliferative glomerulonephritis, varied in severity in direct relationship to the number of mammary tumors, their size, and the length of clinical time they were observed. The severe renal lesion, as illustrated by case 076 412, was not observed in any mice which did not develop mammary tumors. Unfortunately there is not E.M. or serological data to provide additional support information to this suspected immunologically mediated lesion.

Two other lesions occurred in these mice irrespective of whether or not they had mammary tumors or renal lesions. They were hyperplasia of the pancreatic islets (363/401) and a chronic steatitis of the peritoneal unilocular fat 381/401. The fat lesion is illustrated in 076 412.

AFIP Diagnosis. (076 412): 1) Glomerulitis, proliferative, subacute, diffuse, mild to moderate, kidney, female, A^{VY}/A mouse, rodent. 2) Necrosis with granulomatous inflammation, multifocal, minimal, perirenal fat.
(076 464): Glomerulitis, proliferative, subacute, diffuse, minimal, kidney.

Conference Note. Variable numbers of neutrophils were observed within multiple glomeruli of case 076 412. The lack of proteinaceous material within tubular lumina or of degenerative changes involving the tubular epithelium was considered unusual when taking into account the severity of the glomerular changes. The pathogenesis of glomerulonephritis in association with mouse mammary tumor virus is discussed by Pascal et al.[4] In his study circulating immune complexes involving both viral antigens and tumor cell membrane associated antigens are incriminated. Since intact virions were not demonstrated within glomeruli, formation of immune complexes do not occur *in situ* as has been described with murine leukemia virus.

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Case III - BG-1 (AFIP 1810710).

History. Tissue from a 3-day-old, stumptail macaque (Macaca arctoides). The body weight at necropsy was 500 gms. The animal was found dead in its pen.

Contributor's Diagnosis & Comments. Necrosis, liquefactive (abscessation), multifocal, moderate to severe, adrenal gland, etiology, Listeria monocytogenes.

Comments. Listeria monocytogenes has been reported to cause infection in man and other mammals, birds, fish and even arthropods and crustaceans. Encephalitis, meningitis, septicemia and/or abortion may be caused by this organism. Listeriosis has been reported three times in nonhuman primates as the cause of spontaneous disease. McClure and Strozier[3] reported perinatal listeric septicemia in a Celebese black ape. Zwart and Donker-Voet[6] reported a case involving a marmoset. Vetesi, et al.[5] reported an abortion in a Gray's monkey. This case is the first one recorded at our institution. L. monocytogenes was also cultured from the liver, spleen, lung and heart blood at the time of necropsy. The liver and spleen had similar lesions to those demonstrated in the adrenal glands. The lung had few lesions demonstrable with H&E, however, many gram-positive rods were present in the lung, as demonstrated by a Taylor's stain. Numerous organisms were evident also in the liver, spleen and adrenal glands stained by this technique. The dam showed no evidence of clinical illness antemortem or postpartum, consistent with reports in mothers of human infants with Listeria infections.[1] The placenta was not retrieved in this case, therefore we can only speculate that it might have also showed lesions similar to those reported by McClure & Strozier.[3] A vaginal swab was cultured at 3 weeks postpartum, but we failed to recover Listeria by this procedure.

AFIP Diagnosis. Microabscesses, multifocal, severe, adrenal gland, stumptail macaque (Macaca arctoides), nonhuman primate, etiology, gram-positive bacilli (cultured Listeria monocytogenes).

Conference Note. Although the lesions and morphology of the organisms were not specific for listeriosis, L. monocytogenes was considered the most likely gram-positive etiologic agent based upon the age of the animal. The morphologic changes and large numbers of organisms in this case are similar to neonatal listeriosis in man.[2]

Contributor. Comparative Medicine Department, Bowman-Gray School of Medicine, Winston-Salem, North Carolina 27103.

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Case IV - A-36534 (AFIP 1810442).

History. Tissue from a strain C57BL/N female mouse aged 21 months. A large 1.5 X 1.5 X 1.5 cm. diameter friable, multilobulated, pearly white mass caused protrusion of the orbit (left). The lungs contained a number of 1-3 mm diameter nodules visible on the surface. The animal was untreated and was the subject of a study to determine the incidence of tumors occurring during aging. Unfortunately, the bulk of the primary tumor was lost during processing but several sections of the skull showed the presence of a Harderian gland adenocarcinoma.

Contributor's Diagnosis & Comments. Adenocarcinoma, lung, metastatic, multiple, from Harderian gland.

Comments. Harderian gland neoplasms are not unusual in colonies of aging mice. An incidence of 4.5% has been reported by some authors. An incidence of 6% has been reported following thermal radiation. As with many murine neoplasms, metastasis of spontaneously occurring Harderian gland tumors is less common than with the same tumors induced by irradiation or other agents. Features differentiating this neoplasm from other primary and secondary tumors of the lung are the pleomorphic cells and the occurrence of cytoplasmic vacuoles (probably fat), and also the large cystic spaces.

AFIP Diagnosis. Carcinoma, solid, metastatic, multiple, C57BL/N mouse, rodent, origin, Harderian gland (as stated by the contributor).

Conference Note. Diagnoses submitted included carcinoma metastatic from either the adrenal gland, a skin sebaceous gland, or the Harderian gland, as well as a metastatic liposarcoma. A few sections also contained a solitary alveologenic carcinoma. The lack of poryphyrin pigment is typical of most Harderian gland neoplasms.

Contributor. Registry of Experimental Cancers, NCI-NIH, Landow Bldg. 1D-16A, Bethesda, Maryland, 20205.

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Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 15
20 January 1982

Conference Moderator: Michael A. Stedham, D.V.M.
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Veterinary Pathologist
Tracor Jitco, Inc.
1776 East Jefferson Street
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Case I - SPA-086 (AFIP 1802990). Two gross photographs included.

History. Several 10-week-old female Yorkshire pigs presented with multiple, variably sized (1-4 cm) lesions on the ventral abdomen and inguinal areas, characterized by a circular appearance, with raised erythematous circumferences, and depressed crusted centers.

Contributor's Diagnosis & Comments. Superficial dermatitis and epidermitis, eosinophilic, acute, severe, characterized by a) an extensive infiltrate of acute inflammatory cells, primarily eosinophils, into the superficial dermis and epidermis, b) sub-corneal pustule formation (some sections), c) a large central region of hyperkeratosis with impetiginization, and d) mild edema of the superficial dermis.

Comment. The gross appearance, and microscopic findings are consistent with a diagnosis of Pityriasis rosea.

AFIP Diagnosis. Dermatitis and epidermitis, eosinophilic, characterized by acanthosis, edema, and pustule formation, acute, focally extensive, moderate, Yorkshire, porcine.

Conference Note. After evaluating multiple sections, the honeycomb appearance of the epidermis was thought to be due to a function of cut rather than pseudo-epitheliomatous hyperplasia. The extensive number of eosinophils characteristic of this condition in pigs is not seen in man in which the infiltrate consists of a mixture of lymphoid cells, neutrophils, eosinophils, and histiocytes.

Contributor. University of Pittsburgh, Central Animal Facility, A-115 Scaife Hall, Pittsburgh, PA 15261.

References.

1. Altman, N. H., Andrews, E. J., and Ward, B. C.: Spontaneous Animal Models of Human Disease, Vol. II, Academic Press, New York, 1979, pp 20-21.
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Case II - U2070 or W1475 (AFIP 1801671).

History. Tissues from the nasal sinuses of two adult horses with histories of epistaxis for one year. Endoscopic examination revealed obstructive masses in the ethmoid region of both horses.

Contributor's Diagnosis & Comments. Morphologic diagnosis: Granulomatous rhinitis and hemosiderosis associated with ferruginous and calcareous encrusted connective tissues.

Comments. Both horses were euthanatized due to inoperable nasal obstructions. Significant postmortem findings were limited to the presence of green-yellow-brown mottled polypoid masses in the posterior nasal cavity. A cause of the lesions was not determined. There is considerable variability in the amount of pigment, macrophages and giant cells in the sections. These masses are compatible with Progressive Haematomas of the Ethmoid Region[1] and Haemorrhagic Nasal Polyps[2] of the horse.

AFIP Diagnosis. Rhinitis, granulomatous and hemorrhagic, with mineralization and ferruginous bodies, focally extensive, severe, ethmoid region, horse, equine.

Conference Note. Several participants thought the ferruginous bodies looked similar to pollen grains and that the changes present were a foreign body response. These bodies have been analyzed by Platt[2] and found to contain ferric iron, bilirubin, cholesterol, and sudanophilic lipid.

Contributor. Department of Veterinary Pathobiology, The Ohio State University, 1925 Coffey Road, Columbus, OH 43210.

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1. Cook, W. R., and Littlewort, M. C. G.: Progressive haematoma of the ethmoid region of the horse. Equine Vet. J. 6: 101-108, 1974.

2. Platt, H.: Haemorrhagic nasal polyps of the horse. J. Path. 115: 51-55, 1975.

Case III - 718-13 (AFIP 1812876).

History. The sections are from a 6-year-old neutered male cat with an approximately 3-week history of an abdominal subcutaneous mass. Megestrol acetate had been given intermittently since 1976 to treat a skin condition.

Contributor's Diagnosis & Comments. Mammary gland; feline mammary hypertrophy.

Comments. Sections of the submitted tissue reveal a fibroglandular proliferation of the mammary gland arranged in a lobular pattern. The histologic appearance is typical of feline mammary hypertrophy[1] and resembles pericanalicular fibroadenoma of women. The condition has recently been reported in neutered male cats given progestins.[2]

AFIP Diagnosis. Total fibroadenomatous change, mammary gland, male, cat, feline, etiology, megestrol acetate.

Conference Note. Total fibroadenomatous change is the nomenclature used by the W.H.O. for this condition. Other names in addition to feline mammary hypertrophy are fibroepithelial hyperplasia, fibroadenomatosis, and fibroadenoma. A PAS technique demonstrated discontinous ductal basement membranes, and a Masson trichrome stain revealed very sparse thin collagen fibrils coursing within the loose periductal spindle cells. These cells do not arise from myoepithelial cells as occurs in the dog with complex and mixed mammary tumors.[6] Nimmo and Plummer in an ultrastructural study[5] demonstrated them to be of fibroblastic origin. They observed hyperplastic myoepithelial cells arranged circumferentially around and close to the ductal epithelium. Basement membranes were scanty. Myoepithelial cells can be seen surrounding ducts in this case with their nuclei oriented at right angles to the epithelial cells.

Contributor. University of Maryland, School of Medicine, Department of Pathology/Comparative Medicine, Baltimore, Maryland 21201.

References.

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2. Hayden, D. W., Johnston, S. D., Kiang, D. T., et al.: Feline mammary hypertrophy of fibroadenoma complex: Clinical and hormonal aspects. Am. J. Vet. Res. 42: 1699-1703, 1981.
3. Hinton, M., and Gaskell, C. J.: Non-neoplastic mammary hypertrophy in the cat associated either with pregnancy or with oral progestagen therapy. Vet. Rec. 100: 277-280, 1977.
4. Moulton, J. E.: Tumors in Domestic Animals. University of California Press, Berkeley, 1978, p. 368.
5. Nimmo, J. S., and Plummer, J. M.: Ultrastructural studies of fibroadenomatous hyperplasia of mammary glands of two cats. J. Comp. Path. 91: 41-50, 1981.
6. Pulley, L. T.: Ultrastructural and histochemical demonstration of myoepithelium in mixed tumors of the canine mammary gland. Am. J. Vet. Res. 34: 1513-1522, 1973.

Case IV - 79-824-1 or 79-824-3 (AFIP 1801674).

History. A 4-month-old Holstein calf (steer) was submitted for chronic unthriftiness and scours.

Laboratory Results. Gross postmortem findings included a bronchopneumonia from which Pasteurella multocida was recovered, pediculosis, and a cecum and colon with thick walls and reddened mucosal surfaces.

Contributor's Diagnosis & Comments. Verminous cecitis (Trichuriasis).

Comments. The parasites in the cecum were identified as Trichuris discolor. Although this parasite is usually considered relatively nonpathogenic it was felt from the large numbers present and the clinical history that they were at least contributing to the clinical syndrome in this calf.

AFIP Diagnosis. Typhlitis, subacute, diffuse, minimal to mild, with multiple nematodes, Holstein, bovine, etiology, Trichuris sp.

Conference Note. A few sections had a single focus of mucosa containing coccidial schizonts and gametocytes. The presence of a pseudocoelomic body cavity, a considerably smaller cross-sectional anterior diameter, a single bacillary band, a stichosome and thick-shelled eggs with bipolar plugs is characteristic of Trichuris sp. Speciation cannot be determined histologically and requires the whole worm. Capillaria sp. can be differentiated by the presence of two or more bacillary bands and by its smaller uniform cross-sectional diameter - equal to the anterior end of Trichuris sp.

Contributor. Diagnostic Laboratory, Kansas State University, Manhattan, Kansas 66506.

References.

1. Binford, C. H., and Connor, D. H.: Pathology of Tropical and Extraordinary Diseases, Vol. II, AFIP, Washington, D.C., 1976, pp 415-420.

2. Georgi, J. R., and Whitlock, J. H.: Fatal Trichuris discolor infection in a Holstein heifer. Cornell Vet. 62: 58-60, 1972.

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Results
AFIP Wednesday Slide Conference - No. 16
27 January 1982

Conference Moderator: Samuel Machotka, D.V.M.
Diplomate ACVP
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Case I - 80N2051 (AFIP 1760315).

History. On 10 Jul 80, an adult wild 35.4 Kg, 2.23 meter female alligator was caught by hook and line and killed with a shotgun near her nest in the Rockefeller Wildlife Refuge of southwestern Louisiana. The nest contained about 40 eggs.

As part of a reproductive biology project, the reptile was necropsied by wildlife biologists who reported the following findings. The carcass was in good flesh, with large amounts of food (furballs, crabs, crayfish) in the stomach. The only abnormality noted was a diffuse, yellow-white finely granular 1-2 mm thickening of the mesentery and serosa of the stomach, intestine, spleen, kidney, ovaries and oviducts. The serosal surfaces of liver, heart and lungs were not affected. A regressed corpus luteum was present on one ovary.

Contributor's Diagnosis. Chronic, diffuse, marked, reactive peritonitis (serositis); probable etiology - egg yolk contamination of thoracoabdominal cavity.

AFIP Diagnosis. Peritonitis, granulomatous, chronic, diffuse, severe, serosa, colon, alligator, (Alligator mississippiensis), Reptilia, etiology, egg yolk.

Conference Note. The history of recent egg laying and the histologic presence of egg yolk material in the thickened peritoneum were considered compatible with egg yolk peritonitis. Other diagnoses submitted included steatitis associated with vitamin E/selenium deficiency and lipomatosis. One participant stated that the changes in this case were very similar to those he had observed in birds with chronic egg peritonitis. The appearance of the yolk material varies from intracytoplasmic foci of deeply amphophilic material to small lightly eosinophilic intracytoplasmic globules to extensive amounts of clear cytoplasm present within the reactive phagocytes, and probably represents stages in its digestion. No specific references of reptilian egg peritonitis were located and the contributor indicated that the condition had never been seen in a total of 200-300 alligator necropsies performed at all seasons for several years. The pathogenesis is presumably similar to the condition in birds.

Contributor. Department of Veterinary Pathology, School of Veterinary Medicine, Louisiana State University, Baton Rouge, Louisiana 70803.

Reference. Petrak, M. L.: Diseases of Cage and Aviary Birds. Lea & Febiger, Philadelphia, 1969, pp 325-328.

Case II - D81-10131 (AFIP 1809929).

History. This mink rancher has lost 50, mostly adult female mink after weaning. He has 550 breeding females and has greatest mortality in his demi-buff mink but some pastel mink also are affected.

Laboratory Results.

	(vol.%) pcv/hct	(g/ms%) hb/mcv	(/cmm) RBC	(/cmm) WBC	(gm/dl) Tot.Prot.	(gm/dl) albumin	(mU/ml) alk.phos	(mg/dl) BUN
D81-10131	29/32.4	11.0/54u^3	5.99x10^6	4500	13.3	2.3	1300	93
Normal	45/42.5	16.4/55u^3	7.65x10^6	7400	6.6	2.9	110	28

Contributor's Diagnosis & Comment. Aleutian mink disease.

Comment. Kidney - glomerulonephritis (glomerular tuft avascularity and hypercellularity, increased PMN's, increased amorphous to granular pink material) and multifocal chronic interstitital nephritis (mainly plasma cell infiltrates with possible early granuloma formation). Cortical tubules contain eosinophilic (proteinaceous) casts. Arteritis also is present.
Spleen - marked plasmacytosis. In addition to these typical lesions the counter-immunoelectrophoresis test for Aleutian mink disease also was positive.

AFIP Diagnoses. 1) Glomerulitis, proliferative, subacute, diffuse, minimal to moderate, kidney, mink, Mustelid, etiology, Aleutian disease (Parvovirus). 2) Nephritis, interstitial, plasmalymphocytic, subacute, multifocal, moderate, kidney, etiology, same as #1. 3) Arteritis, with fibrinoid necrosis, subacute, multifocal, moderate, muscular arteries, kidney, etiology, same as #1. 4) Plasmacytosis, diffuse, minimal to mild, red pulp, spleen, etiology, same as #1. 5) Extramedullary hematopoiesis, diffuse, mild to moderate, red pulp, spleen.

Conference Note. The changes within the glomeruli and involving the muscular arteries varied considerably between sections. Without thin sections the presence of a membranous glomerulonephritis could not be substantiated. The amount of extramedullary hematopoiesis present in the spleen was considered typical for most Mustelids in our experience.

Contributor. Veterinary Diagnostic Laboratories, College of Veterinary Medicine, University of Minnesota, St. Paul, Minnesota 55108.

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1. Benirschke, K., Garner, F. M., and Jones, T. C.: Pathology of Laboratory Animals, Springer-Verlag, New York, 1978, pp 126-127, 1022-1024, 1352.
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Case III - C-80-01 (1757301).

History. A pigeon was noticed to be weak, listless, and reluctant to fly, but otherwise normal. The pigeon was anesthetized, bled by cardiac puncture and autopsied.

Contributor's Diagnosis & Comment. Leucocytozoon infection.

Comment. Large numbers of leucocytozoon megaloschizonts were found within the capillary walls and small arteries of the lung where thrombosis of small arteries occurred. Megaloschizonts were also present in the liver, kidney and heart. Vectors of leucocytozoon parasites are black flies of the Simulium genus. Malarial pigment was not observed within the liver or spleen. The megaloschizonts ranged from 120 μ to 200 μ in diameter. Primary hepatic schizonts were also seen.

AFIP Diagnosis. 1) Parasitemia, hemoprotozoal, characterized by intra-erythrocytic pigmented gamonts, and vascular schizonts and cytomeres, blood vessels, lung, pigeon, avian, etiology, Hemoproteus sp. 2) Cuffing, perivascular, lymphocytic, multifocal, minimal to mild, muscular arteries, lung.

Conference Note. One of three possible etiologic agents was considered responsible for the protozoal structures observed in the lung including Leukocytozoon sp., Hemoproteus sp. and Plasmodium sp. The size of the schizonts described by the contributor in the liver, kidney, and heart was considered compatible with Leukocytozoon sp. However, several participants identified round to oval gamonts within erythrocytes that usually approximated the size and shape of the nucleus and always contained anisotropic black granular pigment. These were believed compatible with either Hemoproteus sp. or Plasmodium sp. The irregular, tortuous, shape of many of the protozoal structures which branched along capillaries and appeared bifurcate and trifurcate is characteristic of Hemoproteus sp. Two different asexual forms are evident in this case: 1) The schizont which is present within vessels (endothelial cells), is characterized by being 20 μ to 40 μ in diameter, and is subdivided into 10 to 20 round basophilic granular cytomeres; and 2) Maturing cytomeres which have broken out of the schizont, are present free in the blood or have lodged in capillaries, and are responsible for the irregular tortuous structures described above. Three stages in the maturation of these cytomeres are evident. Early forms have a foamy appearance, intermediate forms are characterized by condensing nuclear material and multiple small clefts, and mature forms are filled with typical merozoites. The megaloschizonts of Leukocytozoon sp. in contrast to Hemoproteus sp. are consistently round and contain cytomeres located peripheral to a conspicuous central body (either parasitic primordium or hypertrophied host nucleus).

While Hemoproteus columbae is a common entity in pigeons, Leukocytozoon sp. infection in these birds is extremely rare and has never been reported in North America to our knowledge. A species of Sarcocystis has been described within the endothelial cells of birds (Desser, personal communication) and could present diagnostic difficulties if early asexual stages are not evident.

We wish to thank Dr. Sherman Desser, Department of Microbiology and Parasitology, Univ. of Toronto, Ontario, Canada for his assistance with this case.

Contributor. Toxicology Section, Health, Safety & Human Factors Laboratory, Eastman Kodak Company, Rochester, New York 14650.

References.
1. Adam, K.M.G., Paul, J., and Zaman, V.: Medical and Veterinary Protozoology, Churchill Livingstone, London, 1971.
2. Cowan, A. B.: Reactions against the megaloschizonts of Leukocytozoon simondi mathis and leger in ducks. J. Infect. Dis. 100: 82-87, 1957.
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6. Levine, N. D.: Protozoan Parasites of Domestic Animals and of Man, Burgess Publ. Co., Minneapolis, 1973, pp 259-287.

Case IV - 634 (AFIP 1803357). Electron micrograph included.

History. A cat was presented with a history of sudden-onset respiratory distress.

Laboratory Results. Radiographically, a mass was noted in the anterior mediastinum. At necropsy the mass filled the anterior thoracic cavity. The mass was moderately firm.

Contributor's Diagnosis & Comment. Thymoma.
Comment. Epithelial cells are evident on the electron micrograph.

AFIP Diagnosis. Thymoma, predominantly epithelial, anterior mediastinum, breed unspecified, cat.

Contributor. Hoeschst-Roussel Pharmaceuticals, Inc., Route 202-206 North, Somerville, New Jersey 08876.

References.
1. Hadlow, W. J. High prevalence of thymoma in the dairy goat. Vet. Path. 15: 153-169, 1978.
2. Jarrett, W.F.H., and Mackey, L. J.: Neoplastic diseases of the hematopoietic and lymphoid tissues. Bull. Wld. Hlth. Org. 50: 33, 1974.
3. Mackey, T.: Clear-cell thymoma and thymic hyperplasia in a cat. J. Comp. Path. 85: 367-371, 1975.
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Results
AFIP Wednesday Slide Conference - No. 17
3 February 1982

Conference Moderator: Robert C. Trucksa, D.V.M.
Lt Colonel, VC, USA
Diplomate ACVO
Department of Veterinary Pathology
Armed Forces Institute of Pathology
Washington, DC 20306

Case I - (AFIP 1803985).

History. A tumor from the upper eyelid of a 10-year-old female poodle-mix dog.

AFIP Diagnosis. Blepharitis, granulomatous, focally extensive, moderate, eyelid, mixed-breed poodle, canine.

Conference Note. The histological features, consisting of clear vacuolated spaces surrounded by granulomatous inflammatory cells including foreign body giant cells and numerous PAS positive macrophages, are compatible with an obstructed Meibomian gland of the eyelid with secondary inflammation in response to extruded sebaceous material. Clinically this lesion is often called a chalazion.

A hordeolum is localized suppurative inflammation of the glandular portion of the eyelid. An external hordeolum or a sty involves the glands of Zeis or Moll. The condition occurs predominantly in young animals and recurrence is common until sexual maturity. An internal hordeolum or chalazion involves the Meibomian glands and occurs primarily in middle-aged dogs.

Contributor. Ben White Animal Hospital, Inc., 2403 West Ben White Blvd., Austin, Texas 79704.

References.

1. Gelatt, K. N.: Veterinary Ophthalmology. Lea & Febiger, Philadelphia, 1981, pp 292-294.

2. Hogan and Zimmerman: Ophthalmic Pathology - An Atlas and Textbook, W. B. Saunders, Philadelphia, 1962, pp 190-192.

Case II - 80-566 (AFIP 1803320).

History. Three female Australian shepherd puppies out of a litter of ten presented with microphthalmia and an abnormal haircoat color. The rest of the litter was normal.

Laboratory Results. RBC and WBC counts were performed every other day for a period of 20 days. Temperature and clinical appearance were recorded daily in the three affected dogs and a normal littermate. All animals remained normal during the whole period of the survey.

Contributor's Diagnosis & Comments. Hereditary microphthalmia and retinal dysplasia of the Australian shepherd.

Comments. Ocular abnormalities associated with both merle and white haircoat colors have been documented in Australian shepherd dogs. In this case, bilateral microphthalmia and iridal discoloration with fibrovascular bands were the prominent lesions. The condition has been described also in association with equatorial scleral ectasia; it is inherited as an autosomal recessive trait.[2] Similar lesions have been seen in cyclic neutropenia. Observation of the animals and blood monitoring for 3 weeks ruled out the latter condition.

AFIP Diagnoses. 1) Microphthalmia, severe, eye, Australian shepherd, canine. 2) Cataract, diffuse, severe, fetal nucleus and cortex, lens. 3) Dysplasia with multifocal scleral ectopia, diffuse, severe, retina, eye. 4) Scleral ectasia, severe, equatorial, eye.

Contributor. National Animal Disease Center, P. O. Box 70, Ames, Iowa 50010.

References.

1. Saunders, L. Z., and Rubin, L. F.: Ophthalmic Pathology of Animals. Karger Publishing Col., 1975, pp 190-191.

2. Gelatt, K. N., and Veith, L. A.: Hereditary multiple ocular anomalies in Australian shepherd dogs. Vet. Med./Sm. Anim. Clin. 65: 39-42. 1970.

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Case III - (AFIP 1725431).

History. A two-year-old male castrate domestic shorthair cat lost its vision bilaterally several months prior to death.

AFIP Diagnoses. 1) Panophthalmitis, pyogranulomatous, with necrotizing vasculitis, diffuse, severe, bilateral, eyes, domestic shorthair, feline, etiology compatible with feline infectious peritonitis (FIP) virus infection. 2) Detachment, subacute, segmentally diffuse, bilateral, retinae. 3) Keratitis, subacute, multifocal, mild, bilateral, corneas. 4) Degeneration, with gliosis and vacuolation, diffuse, mild to moderate, bilateral, optic nerves. 5) Cataract, subcapsular, multifocal, mild, lenses.

Conference Note. The pyogranulomatous panophthalmitis and vasculitis are consistent with a diagnosis of feline infectious peritonitis (FIP) virus infection. Careful examination and special stains failed to demonstrate an etiologic agent. Some of the systemic gross and histopathological findings noted in the anamnesis also supported the diagnosis. These included: nonsuppurative inflammatory lesions in the heart and thyroid gland, fibrous pleural adhesion, pleural edema, cerebralpontinegliosis, and lymphoid hyperplasia of several lymphoid organs.

Contributor. Ophthalmology Section, Department of Veterinary Clinical Medicine, University of Illinois, Urbana, Illinois 61801.

References.

1. Doherty, M. J.: Ocular manifestations of feline infectious peritonitis. J. Am. Vet. Med. Assoc. 159: 417-424, 1971.

2. Gelatt, K. N.: Iridocyclitis - panophthalmitis associated with feline infectious peritonitis. Vet. Med./Sm. Anim. Clin. 68: 56-57, 1973.

3. Gelatt, K. N.: Veterinary Ophthalmology. Lea & Febiger, Philadelphia, 1981, pp 551-553, 709-710.

4. Slauson, D. O., and Finn, J. P.: Meningoencephalitis and panophthalmitis in feline infectious peritonitis. J. Am. Vet. Med. Assoc. 160: 729-734, 1972.

Case IV - 5255-80 (AFIP 1803062).

History. A 4-year-old domestic shorthair female cat was donated to the referring veterinarian because it was blind. The owner had two other blind cats that had been euthanatized in the past 6 months. Retinal atrophy was diagnosed in one; the other had lymphosarcoma. There were two cats remaining in the household but their state of health was unknown. None of the cats were related. The cat's pupils were dilated and responded slowly to changes in light. The optic discs were sunken and the retinal vessels were much smaller than normal. The cat was in good flesh and was otherwise normal.

Laboratory Results. Histologic findings: In the retinas of both eyes there was partial to complete loss of the layer of rods and cones, outer nuclear layer, and outer plexiform layer. The inner nuclear layer was also somewhat narrower than normal.

Contributor's Diagnosis & Comments. Bilateral diffuse retinal degeneration and atrophy caused by taurine deficiency.

Comments. Retinal atrophy has been reported in cats on taurine-deficient diets. Taurine is almost absent in vegetables but abundant in meat and milk. The retinal changes can be stopped by correcting the diet, but lesions already present are not reversible. A more precise dietary history could not be obtained in this case. Recently, the National Research Council tentatively recommended that feline diets contain at least 500 ppm taurine; earlier NRC requirements did not list taurine as an essential ingredient.

AFIP Diagnoses. 1) Degeneration, diffuse, moderate to severe, outer segments, retina, eye, domestic shorthair, feline. 2) Cyst, focal, mild, ciliary process, eye.

Conference Note. The clinical history and histologic changes support the diagnosis of taurine deficiency. Vitamin A deficiency, inherited progressive retinal atrophy, and toxicity by zinc chelating agents (e.g. dandruff shampoos containing zinc pyridinethione) were considered differentially. The lack of corneal vascularization or squamous metaplasia of the corneal epithelium, the presence of multiple, unrelated, similarly affected cats, and the lack of tapetal cell necrosis ruled out these respectively. Although certain dog foods known to be severely taurine deficient were incriminated in the first reports of this entity, unconfirmed later reports suggest that some cat foods are also taurine deficient. Because the minimum required taurine level for the cat is not known with certainty, cat food manufacturers are conducting investigations into this area.

The lens contained severe disruptive changes which were not consistent with the recognized morphologic features indicative of a cataract. They were interpreted as postmortem or cutting artifacts.

Contributor. Veterinary Diagnostic Laboratory, Oregon State University, P. O. Box 429, Corvallis, Oregon 97339.

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1. Aguirre, G. D.: Retinal degeneration associated with the feeding of dog food to cats. J. Am. Vet. Med. Assoc. 172: 791-796, 1978.

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3. Gelatt, K. N.: Veterinary Ophthalmology. Lea & Febiger, Philadelphia, 1981, pp 657-660.

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5. Rubin, L. F., and Lipton, D. E.: Retinal degeneration in kittens. J. Am. Vet. Med. Assoc. 162: 467-469, 1973.

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Results
AFIP Wednesday Slide Conference - No. 18
10 February 1982

Conference Moderator: F. M. Garner, D.V.M.
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Case I - X-140 MRI (AFIP 1803093).

History. This lesion was an incidental finding in a rabbit that was used in a kidney function experiment. The kidneys were enlarged with tightly adhered capsules.

Contributor's Diagnosis. Encephalitozoon cuniculi.

AFIP Diagnosis. Nephritis, tubulointerstitial, subacute, multifocal, moderate, kidney, rabbit, Lagomorpha, etiology, Encephalitozoon cuniculi.

Conference Note. Tissue Gram stains revealed free Encephalitozoon cuniculi spores within a solitary granuloma in the medulla of one section. Organisms, however, were not observed in any of the H&E sections evaluated by the participants. The moderator commented that in his experience approximately 30% of the rabbits he routinely examines have lesions compatible with this condition. He rarely has observed schizonts or free spores in these cases. When present the organisms are best found in collecting duct epithelium. E. cuniculi spores and schizonts are much more prevalent in infected mice and rats.

Contributor. Midwest Research Institute, 425 Volker Boulevard, Kansas City, Missouri.

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Case II - 527-1 (AFIP 1803312).

History. Several abortions were noted in a range cow herd. Abortions occurred during the last trimester of pregnancy and affected both cows and heifers.

Laboratory Results.

Necropsy: The fetus submitted was approximately in the eighth month of gestation. The thoracic cavity contained 1500 ml of clear straw-colored fluid. The lungs were firm on palpation and were sectioned easily.

Contributor's Diagnosis & Comments. 1) Fetal pneumonia, fibrinopurulent, diffuse, severe. 2) Cholangiohepatitis, chronic, diffuse, moderate.

Brucella abortus, biotype 1, was isolated from cultures of lung, liver, and thoracic fluid.

AFIP Diagnoses. 1) Bronchopneumonia, histiocytic and fibrinous, subacute, diffuse, moderate to severe, lung, fetus, bovine, etiology, Brucella abortus biotype 1, (as specified by the contributor). 2) Hepatitis, subacute, multifocal, minimal, liver, etiology, same as #1. 3) Congestion, acute, mild, liver. 4) Hematopoiesis, extramedullary, periportal and portal, diffuse, moderate, liver.

Conference Note. Virtually all participants considered Brucella abortus the most likely etiology based upon the characteristic changes in the lungs. The foci of hepatitis were small and contained hepatocellular necrosis, mononuclear cells including lymphocytes, and occasionally fibrin. These foci usually were located adjacent to a central vein or midzonally. The presence of congestion may have been related to the hydrothorax described grossly. The multifocal, empty, dilated, sinusoids present in all sections formed symmetrical bands which were most obvious at low magnification. They were thus thought to be artifactual.

Contributor. Department of Veterinary Science, University of Nebraska, Lincoln, Nebraska 68583.

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Case III - 236618 (AFIP 1804247).

History. This 4-year-old Appaloosa mare had signs of illness on 31 July 1981 and by noon of the following day had clinical signs of encephalitis, was down, and had a temperature of 103° F. The following morning she was totally nonresponsive.

Contributor's Diagnosis. Eastern equine encephalomyelitis.

Laboratory Findings. Viral isolation of the EEE alphavirus (togavirus) was confirmed by the CDC Laboratory at Ft. Collins, Colorado. Fluorescent antibody examination for rabies was negative.

Histopathologic examination: There was extensive perivascular cuffing in nearly all the brain tissue examined which was less severe in the cerebellum. Most of the cells were lymphocytes, although there were a few neutrophils as well. Neuronal changes were not marked in these tissues, but in the cerebral cortex some of the neurons appeared to be shrunken and to be more eosinophilic than normal.

Comment. The lesions are consistent with those of equine encephalomyelitis. The lack of marked neuronal changes may have been due to the acute nature of the disease in this animal.

Michigan had 20 cases of EEE confirmed in 1980, and another 90-100 cases given a presumptive diagnosis of EEE clinically. Mosquito populations were estimated as being approximately 5-10 fold more than usual due to an extremely wet year. As of 15 August 1981, 6 cases of EEE had been confirmed in Michigan.

AFIP Diagnosis. Encephalitis, nonsuppurative, characterized by perivascular cuffs and glial nodules, subacute, multifocal, mild to moderate, brain, equine, etiology, Eastern Equine Encephalomyelitis (as specified by the contributor).

Contributor. Animal Health Diagnostic Laboratory, Michigan State University, Lansing, Michigan.

References.

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Case IV - 89748 (AFIP 1802813).

History. An aged male German shorthaired Pointer was picked up as a stray by the Department of Animal Control. It appeared somewhat depressed, and was found dead in the kennel the following morning. At necropsy examination, a large, asymmetric, irregular, hemorrhagic mass was found, primarily in the cerebral white matter, extending from the level of the septum pellucidum to rostral to the hippocampus.

Contributor's Diagnosis & Comments. Glioblastoma multiforme.

The lesion was found to have features which are characteristic of glioblastoma multiforme, namely the prominent vascularity and the immature tumor cells which have oval to spindle-shaped morphology, and tend to form a palisading pattern around areas of degeneration. The capillary endothelial proliferation is typical. Secondary tumor foci, separate from the larger primary mass are seen in some sections. An immunoperoxidase stain for glial fibrillary acidic protein (GFAP) was positive for this antigen in the processes of the spindle-shaped tumor cells. This antigen is specifically associated with this tumor. The reagents used were from DAKO, Santa Barbara, Calif., and were specific for human GFAP.

AFIP Diagnosis. Glioblastoma, cerebrum, brain, German shorthair pointer, canine.

Contributor. Los Angeles County Department of Health, Section of Comparative Pathology, 12824 Erickson Avenue, Downey, California 90242.

References.

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Results
AFIP Wednesday Slide Conference - No. 19
17 February 1982

Conference Moderator: Paul K. Hildebrandt, D.V.M.
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Case I - 23447 (AFIP 1810441).

History. Tissue from a 7-week-old male cocker spaniel from Ingalls, Arkansas. The dog had diarrhea and was seen by a veterinarian who treated it for coccidiosis. Recently, the tomato farm on which this dog lived had been sprayed with a fungicide/herbicide. It was shortly (2 days) after this spraying that the dog appeared to have acute gastric pain followed by death. At necropsy a large intra-abdominal mass was palpated prior to making any abdominal incisions. (Two H&E slides provided).

Contributor's Diagnosis & Comments. 1) Atrophy and blunting, villous, diffuse, moderate, small intestine, cocker spaniel, canine; compatible with parvovirus infection. 2) Depletion, lymphoid, diffuse, severe, spleen. 3) Congestion, periportal, diffuse, mild, liver. 4) Pigment, periportal, mild, liver; compatible with hemosiderin. 5) Hematopoiesis, extramedullary (EMH), diffuse, mild, spleen.

The intra-abdominal mass palpated at gross necropsy was an acute intussusception at the ileocecal junction.

Laboratory Results.
Organophosphate - Negative.
Chlorinated Hydrocarbons - Negative.
F/A - Direct-Positive for Parvovirus.

AFIP Diagnoses. 1) Depletion and necrosis, lymphocytic, subacute, diffuse, severe, spleen and Peyer's patches, cocker spaniel, canine, etiology, canine parvovirus (as specified by the contributor). 2) Colitis, mucoid, acute to subacute, diffuse, moderate, with multifocal crypt dilatation and crypt cell necrosis, large intestine. 3) Peritonitis, serofibrinous, acute, diffuse, moderate, colon.

Conference Note. The contributor indicated that multiple blocks of intestinal tissue were used including cecum and small intestine. We had no sections of small intestine to evaluate. Some slides contained sections of heart. The extensive lymphocytic depletion and necrosis evident in the spleen and Peyer's patches were considered highly suggestive of a parvoviral infection since radiation exposure of this animal was unlikely. Some of the colonic changes seen probably were related to the acute intussusception located more proximally.

Contributor. Food & Drug Administration, Division of Pathology, Bureau of Foods, 200 C Street, S.W., Washington, D.C. 20204.

References.
1. Arpel, M. J., Cooper, B. J., Greisen, H., et al.: Canine viral enteritis. I. Status report on corona and parvo-like enteritides. Cornell Vet. 69: 123-133, 1979.
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Case II - AFRRI #1 (AFIP 1782726).

History. An adult male cynomolgous monkey was euthanatized and necropsied at the termination of an experiment. A large retroperitoneal mass, approximately 7 X 4 centimeters in area, was found extending from the right pericentral lumbar region cranially to beneath the right kidney and adrenal gland.

Contributor's Diagnosis & Comments. Chronic inflammation and fibrosis, caused by Edesonfilaria malayensis.

Comment. A microfilaremia had been noted previously in this monkey, but it exhibited no untoward clinical symptoms, and this parasitic lesion was considered an incidental finding. The retroperitoneal mass had a smooth glistening surface and contained semitranslucent areas with numerous slender white worms. A characteristic feature of Edensonfilaria malayensis, identifiable both grossly and microscopically, is the greatly dilated glandular esophagus. This appears as a large, granular, basophilic body which occupies almost the entire pseudocoelom of the worm and is contiguous with the esophageal lumen. Intensely basophilic giant bodies, of unknown function and not present in all the slides, are often evident in the periphery of this part of the esophagus. In addition to its glandular esophagus, the parasite also contains a reticular intestine and has brown pigment associated with the lateral and ventral nerve cords.

Several other filarial nematodes have been reported to occur in cynomolgus monkeys in addition to E. malayensis. Dirofilaria magnilarvatum, D. repens, Dipetalonema digitatum, Brugia malayi, and Macacanema formosana are reported in the literature. None of these has the characteristic esophagus as noted with Edesonfilaria except Macacanema, and differentiation between the two cannot be made histologically. Macacanema is an uncommon parasite and the spicules of the male are much smaller than those in Edesonfilaria.

Adult Edesonfilaria usually are found free in the abdomomal cavity or in the retroperitoneal tissue. Except for microfilaremia clinical signs of infection are usually absent. Microfilariae are unsheathed, have a pointed tail, and average 140 microns in length by 3-5 microns in diameter. The life cycle and intermediate host of E. malayensis have not been identified, but transmission probably involves a blood-sucking arthropod. Filariasis is recognized frequently in Old World and New World nonhuman primates but it results in little, if any, clinically recognizable disturbances. There is little information on the treatment of filariasis in nonhuman primates.

AFIP Diagnoses. 1) Fibrosis and parasitic migration tracts with lymphoplasmacytic inflammation and nematode parasite, focally extensive, severe, retroperitoneal lumbar area, cynomolgus monkey (Macaca fascicularis), nonhuman primate, etiology, Edesonfilaria malayensis (as specified by the contributor). 2) Lymphoid hyperplasia, diffuse, moderate, lymph node.

Contributor. Division of Comparative Pathology, Armed Forces Radiobiology Research Institute, Bethesda, Maryland 20814.

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Case III - Sp81-364 (H364-81A) (AFIP 1816641).

History. A 4-year-old crossbred female dog presented with recurrent pyrexia 105-106° F.) and anorexia. Physical examination revealed painful swelling of the submandibular region. The section is from a surgically resected salivary gland.

Contributor' Diagnosis & Comments. Salivary gland necrosis with inflammation, fibrosis and ductal epithelial hyperplasia.

Comment. The changes present were representative of those found in both submandibular glands. Some sections include thrombosed arteries with haemorrhagic necrosis of the tunica media.

AFIP Diagnosis. Sialoadenitis, necrotizing, with ductular hyperplasia and squamous metaplasia, acute and chronic, multifocal, moderate to severe, salivary gland, mixed breed, canine.

Conference Note. Although most participants concurred with the contributor, one differential diagnosis discussed was an underlying neoplasm with secondary necrosis, inflammation, and fibrosis. The histologic changes and pathogenesis in this case, as discussed by Kelly et al., are similar to those described for necrotizing sialometaplasia in man. The latter is a benign self-limiting condition of the minor salivary glands. Extensive squamous metaplasia of the ducts and acini

is the predominant feature along with lobular necrosis and an accompanying inflammatory reaction. The major problem associated with necrotizing sialometaplasia is its clinical and histologic similarity to either squamous cell carcinoma or mucoepidermoid carcinoma. Unnecessary and destructive surgery, such as wide surgical excisions and maxillectomy, has been performed on patients with this benign disease process. If left alone it will resolve by itself within three to ten weeks without recurrence. As stated by Kelly et al. in reference to this case, the etiology of necrotizing sialometaplasia is not known but is thought to involve a compromised blood supply.

Contributor. Department of Veterinary Pathology, University of Liverpool, P.O. Box 147, Liverpool L69 3BX, England.

References.

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Case IV - A163717 (AFIP 1803526).

History. This mass was removed from a 13-year-old female domestic shorthair cat with multiple cutaneous masses.

Laboratory Results. Histopathology was perfromed - there were numerous large, foamy epithelioid macrophages with numerous scatterings of neutrophils and lymphocytes. Occasional structures suggestive of fungi were observed and subjected to special stains.

Contributor's Diagnosis & Comments. Multifocal chronic pyogranulomatous dermatitis (Cryptococcosis).

Comment. This case was a surgical submission. The cat was euthanatized after the diagnosis was rendered. Involvement of other systems is unknown. It is our impression and apparently the same impression of members of AFIP that we are seeing more of this type of reaction with some cases of Cryptococcosis. This is somewhat different than the classical case with massive numbers of organisms and almost pure epithelioid cell response.

Conference Note. The majority of participants considered Cryptococcus neoformans the most likely etiologic agent with Blastomyces dermatiditis as the primary differential etiology. The presence of yeast forms 4 u to 8 u in diameter that were sometimes folded and were surrounded by a 2 u to 3 u thick clear space was considered highly suggestive for C. neoformans. A carminophilic capsule was confirmed with a mucicarmine stain. B. dermatiditis also will stain positively with a mucicarmine stain and a Gomori methenamine silver stain sometimes must be used to make the diagnosis of C. neoformans if the latter has a thin capsule. B. dermatiditis does not fold easily, has a thicker wall, is usually larger, is multinucleated, and has broad-based budding. Primary cutaneous cryptococcosis is rare in man and probably in cats and other animals as well. Wilkinson comments that the size of the yeast precludes extensive aerogenous exposure of the lower

respiratory tract. The tissues of the head, oral cavity, and upper respiratory tract are more probable portals of entry with hematogenous dissemination to other organs including the skin. The inflammatory response in the skin is variable but, at least in man, is not indicative of the host's immune status; anergic individuals (identified by skin tests and usually afflicted with multiple diseases) can produce an extensive inflammatory response in the skin while having inflammatory free cerebral cryptococcosis.

Contributor. Veterinary Reference Laboratory, P. O. Box 30633, Salt Lake City, Utah 84130.

References.

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Results
AFIP Wednesday Slide Conference - No. 20
24 March 1982

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Case I - 1-1190-81 (AFIP 1808316).

History. Approximately 350 head of Angus heifers, weighing 450-500 pounds were being fed in one pen of a large feedlot. The day following a change in feed, the total feed consumption dropped to 25% of the preceding day's amount and the cattle developed dark, watery diarrhea. Two animals died on the second day after going off feed and by the end of the third week 66 animals had died.

Laboratory Results. Clinical chemistry results from sixteen animals at nine days following the outbreak:

	High	Low	Mean
SGOT	239	82	145
CPK	3139	192	1170
LDH	1612	736	1077

Contributor's Diagnoses and Comments.

Morphologic Diagnoses: 1) Cardiomyopathy, subacute, disseminated, necrotizing, moderately severe. 2) Pulmonary hemorrhage, acute, interlobular-disseminated, moderately severe.

Laboratory Diagnosis: Cardiomyopathy, consistent with monensin toxicosis.

Comment: The supplement being used was in liquid form and was supposed to have contained 350-400 gm of monensin sodium per ton. The rest of the ration was composed of alfalfa hay, corn silage and barley. The supplement tended to layer out upon storage probably producing unequal distribution of the monensin sodium.

AFIP Diagnoses. 1) Degeneration and vacuolization, subacute to chronic, multifocal, moderate, myocardium, right ventricle, heart, angus, bovine, etiology, monensin toxicity (as specified by the contributor). 2) Pneumonia, interstitital, fibrinous, subacute, diffuse, moderate, lung. 3) Congestion, diffuse, severe, lung. 4) Sarcocystosis, multifocal, myocardium, heart.

Conference Note. Monensin toxicity was considered the most likely etiologic agent based upon the presence of the animals within a feedlot, and the lack of histologic changes indicative of an infectious condition. Differential etiologies considered included plants causing degeneration of cardiac and skeletal muscle such as coffee senna, coyotillo, and hairy vetch, as well as vitamin E/selenium toxicosis or deficiency. A Masson trichrome stain demonstrated collagen formation within several foci of myocardial degeneration.

Several participants commented that the lesions in the lung bore some resemblance to bovine atypical interstitital pneumonia, and to subacute shock lung with early hyaline membrane formation as described by Robbins et al.

There was general consensus that the pulmonary changes were at least partially a result of a direct toxic effect by the monensin, versus being entirely secondary to the cardiac lesions. The moderator commented that teflon could induce similar changes in the lungs of experimental animals by damaging capillary endothelium.

Contributor. Montana Veterinary Diagnostic Laboratory, Diagnostic Laboratory Bureau, Department of Livestock, P. O. Box 997, Bozeman, Montana 59715.

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Case II - FIA-2 (AFIP 1809915).

History. A 14-year-old neutered male cat with a history of polyuria, polydypsia and progressive weight loss.

Laboratory Results.
4+ glucosuria (2 gm/100 ml) and a fasting blood glucose value of 254 mg%. There was no rise in serum insulin in response to a high dose i.v. glucose tolerance test.

Contributor's Diagnosis & Comments. Diabetes mellitus and insular (islet) amyloidosis.
Comment: Amyloid deposits largely replace some islets of Langerhans. This form of amyloid is localized to the islets and can be distinguished histochemically from secondary systemic amyloid (which is composed primarily of protein AA). Other islets show marked vacuolar change which apparently is a reflection of glycogen deposition in association with hyperglycemia. Changes in the exocrine tissues are likely related to aging.

AFIP Diagnoses. 1) Vacuolar change, multifocal, moderate to severe, islets of Langerhans, pancreas, breed unspecified, feline. 2) Amyloidosis, multifocal, moderate to severe, islets of Langerhans, pancreas. 3) Inflammation, subacute, multifocal, mild, pancreatic ducts, pancreas. 4) Hyperplasia, nodular, multifocal, mild, exocrine, pancreas.

Contributor. Department of Veterinary Pathobiology, College of Veterinary Medicine, University of Minnesota, St. Paul, Minnesota 55108.

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Case III - 79-5396 (AFIP 1803529).

History. This transverse section of spinal cord is from a two-month-old feeder pig which was paralyzed. Three more similarly effected pigs from this farm also were examined. Pigs on two more farms in the same general area developed a similar condition at approximately the same time.

Laboratory Results. Chemical analysis revealed 19 to 24 ppm Selenium in the feeds being used on all three farms.

Contributor's Diagnosis & Comments. Bilateral symmetrical poliomyelomalacia due to selenium toxicosis.

Comment: Lesions were found only in the cervical and lumbar intumescences. The lesions are remarkably symmetrical. In most sections, there is marked loss of neurons within the centers of the lesions with chromotolytic, degenerate, and normal appearing neurons at the margins. Little cellular response is evident, although an occasional perivascular cuff is present and there is mild vascularization and glial cell reaction.

Other than in an article by Herigstad et al., a thorough search of the literature revealed no known relationship of this type lesion with Se toxicosis. However, a few reports of paralysis associated with poliomyelomalacia in swine and sheep were found. In these reports lesions were described which are remarkably like those in the study case but no etiology was determined. Following the discovery of toxic levels of selenium in the feeds from each of the involved farms, we conducted an experiment using added Se to a control feed at levels bracketing the amounts detected in the incriminated batches of feed. The levels of Se added and the occurrence of poliomyelomalacia in the experimental subjects is reported in Table 1.

Table 1 — Sodium selenite added to feed

Group	Treatment	No. in group	Results
A	10 ppm Se	5	2/5 with poliomyelomalacia[2]
B	20 ppm Se	6	2/6 " "
C	30 ppm Se	6	5/6 " "
D	Control[1]	3	0/3 " "

1. Commercial grower ration; analysis showed 0.3 ppm Se.
2. Group A. One pig removed during experiment because it developed severe eperythrozoonosis.

The lesions found in the experimental subjects are indistinguishable from the "natural" cases.

AFIP Diagnosis. Poliomyelomalacia, subacute, bilateral, symmetrical, moderate, ventral horns, spinal cord, breed unspecified, porcine, etiology, selenium toxicity (as specified by the contributor).

Contributor. Veterinary Diagnostic & Investigational Laboratory, P. O. Box 1389, Tifton, Georgia 31793.

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Case IV - MK 81-853 AL (AFIP 1822342).

History. Tissues from a wild-caught, adult, female squirrel monkey (Saimiri sciureus) that died spontaneously during quarantine. At necropsy the major arteries were markedly thickened and noticeably rigid. Irregular elevations and roughened foci were found along the intimal surfaces.

Contributor's Diagnoses & Comments. 1) Atherosclerosis, diffuse, chronic, severe. 2) Aortitis, nonsuppurative, diffuse, chronic, mild to moderate. 3) Infiltrates, lymphocytic, perivascular, focal, periadventitial adipose tissue (in some sections).

Comment. Naturally occurring and experimentally induced arterial lesions have been studied and described extensively in the squirrel monkey. It is known that atherosclerosis and aortitis can occur spontaneously alone or concurrently in the squirrel monkey. When aortitis is present the degree of induced atherosclerosis is increased and regression of the induced lesion is decreased. Therefore, aortitis can complicate experimental results. To date the cause of spontaneous aortitis is unknown, but some workers suggest that migrating larval parasites may be the etiologic factor.

AFIP Diagnoses. 1) Arteriosclerosis, chronic, diffuse, severe, intima, aorta, squirrel monkey, (Saimiri sciureus), primate. 2) Aortitis, subacute, diffuse, moderate, media and adventitia, aorta.

Conference Note. An initial report of arterial lesions in squirrel monkeys indicated that aortic fatty streaks occurred with relatively high frequency in the free-living animals. The histologic appearance of the lesions described in this report was typical of fatty streaks: A thickened fibromuscular intima composed of smooth muscle cells and elastic fibers containing extracellular and intracellular lipid droplets, but no large accumulations of foam cells or lesions resembling human fibrous plaques. The changes in this case were not considered compatible with a fatty streak nor with typical atherosclerosis (due to the lack of atheroma formation). Although aortitis and arteriosclerosis are considered separate entities in squirrel monkeys and can occur concurrently, the diffuse circumferential involvement of the intima and the even, severe, nature of the lesion imply that the arteriosclerosis could have been secondary to the aortitis or was affected significantly by its presence as has been described by Hayes et al.

Contributor. Comparative Pathology Section, Building 28A, Room 111, National Institutes of Health, Bethesda, Maryland 20205.

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Results
AFIP Wednesday Slide Conference - No. 21
17 March 1982

Conference Moderator: James L. Stookey, DVM
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Case I - 1458-T9 (AFIP 1796386).

History. This tissue is from a 3.5-year-old male Dunkin Hartley strain guinea pig found dead on a routine check. The lesion of interest was not grossly visible and not directly related to the cause of death (pneumonia). Approximately 26 additional animals of this group exhibited similar lesions. There was no apparent sex predilection or experimental influence associated with the lesion. Ages of the affected animals ranged from two to six years. (H&E and PAS provided).

Contributor's Diagnosis & Comments. Basophilic degeneration of myocardium (auricle).

Comment. This condition is frequently encountered in human pathology[2,3] and has been reported in some animals.[1,3,4] The degenerative substance is believed to be either a glucan (polyglucosan)[1] or a muco-glycoprotein (acid mucopolysaccharide).[3] Areas of basophilic myocardial degeneration are PAS-positive before and after diastase treatment, and are positive with the alcian blue stain. Toluidine blue, von Kossa, Congo red, methyl green pyronine, and Feulgen stains are all negative. The morphological appearance and staining reactions are similar to those reported for human basophilic myocardial degeneration. Only auricles were affected in the guinea pigs we examined. Degenerative changes in the ventricles are age related. The deeply basophilic areas within ventricular myocardial fibers evident on the PAS stained section are intensely eosinophilic and lack striations in H&E sections.

AFIP Diagnoses. 1) Basophilic (mucoid) degeneration, multifocal, moderate to severe, myocardium, auricle, heart, Dunkin Hartley guinea pig, rodentia.
2) Degeneration and vacuolation, multifocal, mild, myocardium, ventricle, heart.
3) Epicarditis, subacute, diffuse, mild, heart.

Contributors. Mobil Oil Corporation, Mobil Environmental & Health Science Laboratory, Toxicology Division, P.O. Box 1026, Princeton, NJ 08540 and U.S. Environmental Protection Agency, Health Effects Research Laboratory, Research Triangle Park, NC 27711.

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Case II - X13765 (AFIP 1803456).

History. The tissue sections are from one of four sandhill (Grus canadensis) crane chicks placed in a pen where other chicks had died at 8 days of age. This chick was killed 24 days later.

Laboratory Results. At necropsy, there was mottling with circular (0.2 cm) pale tan foci on the surface and cut sections of the liver; dark red lungs; and a few erythematous areas in the small intestine. Fecalysis by flotation technique revealed nematode eggs and coccidial oocysts.

Contributor's Diagnosis & Comments. Disseminated visceral coccidiosis.

Comment. Eimeria reichenowi and E. gruis were reported in cranes as early as 1935[5] but information on the endogenous stages has been meager. Recently, severe intestinal coccidiosis with widespread systemic involvement was reported in captive whooping (G. americana) cranes at the Patuxent Wildlife Research Center in Laurel, Maryland.[1] Parasitic granulomas occurring in various organs and tissues of adult sandhill cranes at the center also were associated with a concurrent intestinal infection with E. reichenowi and E. gruis.[2] Exposure of six incubator-hatched and hand-reared sandhill crane chicks to pooled sporulated oocysts of both Eimeria species (artificially in two chicks and naturally in four chicks) resulted in typical infection of intestinal epithelium by both species, and invasion by E. reichenowi of subepithelial tissues extending to the muscular layer with widespread extra-intestinal development.[4] Asexual and sexual stages occurred primarily in macrophages in the liver, spleen, heart (not included in section), and lung. In the lung, oocysts were found in bronchial exudate and epithelial lining cells. Tissue responses to the parasite include focal granulomatous inflammation and necrosis. Life cycle studies are continuing.

AFIP Diagnoses. 1) Enteritis, subacute, multifocal, minimal, with protozoal schizonts and gamonts, small intestine, sandhill crane (Grus canadensis), avian, etiology, Eimeria reichenowi and Eimeria gruis. 2) Pneumonia, interstitial, granulomatous, subacute, multifocal, minimal, with protozoal schizonts, gamonts, and oocysts, lung, etiology, Eimeria reichenowi. 3) Splenitis, granulomatous and necrotizing, subacute, diffuse, moderate to severe, with protozoal schizonts and gamonts, spleen, etiology, same as 2. 3) Hepatitis, granulomatous, portal and periportal, with individual hepatocellular necrosis and protozoal schizonts and gamonts, subacute, diffuse, mild, liver, etiology, same as 2. 5) Hepatitis, granulomatous, subacute to chronic, multifocal, moderate, with protozoal schizonts and gamonts, liver, etiology, same as 2.

Conference Note. Histologic changes vary considerably between sections.

Contributor. Eli Lilly & Company, Greenfield, Indiana.

References.

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Case III - 2702 (AFIP 1815112).

History. A recently purchased gilt died after clinically apparent respiratory disease of one day duration.

Laboratory Results. A moderate number of Haemophilus pleuropneumoniae and a small number of non-haemolytic E. coli were isolated from the lung.

Contributor's Diagnosis. Severe, diffuse, fibrinous, necrotizing, pleuropneumoniae.

AFIP Diagnosis. Pleuropneumonia, fibrinohemorrhagic and histiocytic, acute and chronic, diffuse, severe, breed unspecified, gilt, porcine, etiology, Hemophilus parahemolyticus (pleuropneumoniae) (as specified by the contributor).

Conference Note. Contagious pleuropneumonia due to Hemophilus parahemolyticus was considered the most likely etiology based upon the presence of extensive hemorrhage, a striking mononuclear cell response, and numerous aggregates of bacteria. Pasteurella multocida was listed as the primary differential etiologic agent and cannot be distinguished with assurance from H. parahemolyticus histologically. However, more necrosis, less hemorrhage, and a predominantly neutrophilic cell response would be expected with porcine pasteurellosis. The mononuclear cells present were almost entirely macrophages and often were arranged in a characteristic streaming and swirling pattern that has been described with H. parahemolyticus infections (Sanford et al.). Lesions associated with contagious pleuropneumonia usually are limited to the thoracic cavity although the organism can be cultured from multiple tissues in affected animals including tonsil, spleen, liver, kidney, and joint fluid, and sporadic abortions have been reported in the third trimester of gestation. Both porcine and human strains of H. parahemolyticus exist. Based on the marked difference between their biochemical reactivity, several researchers consider the porcine strains to be a different species and use the nomenclature, H. pleuropneumoniae, as originally suggested by Shope.

Contributor. OMAF-VSB, Box 3612, Guelph, Ontario, Canada N1H 6R8.

References.

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Case IV - 3775 - (AFIP 1805528).

History. This tissue is from a rapidly growing mass in the ear canal of a three-year-old dog.

Contributor's Diagnosis & Comments. Ceruminoma.
Comment. The tumor was confined entirely to the ear canal and did not involve any of the underlying salivary gland tissue. The dog is presently healthy. There has been no recurrence of the tumor.

AFIP Diagnosis. Carcinoma, tubulosolid, external auditory canal, ear, breed unspecified, canine, probably of ceruminous gland origin.

Conference Note. A ceruminous gland tumor was considered the most likely diagnosis due to morphologic characteristics and location. The cellular atypia, areas of necrosis, large size and mitotic rate were compatible with a malignant designation. Most adenomas are well differentiated and contain characteristic copper colored secretory material. Several areas in the mass had a myxomatous appearance suggestive of cartilaginous matrix or osteoid deposition, but the lack of identifiable myoepithelial cells prevented the tumor from being described as mixed.

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Reference. Moulton, J. E.: Tumors in Domestic Animals, University of California Press, Berkeley, California, 1978, pp 58-59.

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Results
AFIP Wednesday Slide Conference - No. 22
24 March 1982

Conference Moderator: John D. Strandberg, DVM, PhD
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Case I - 80-5389 (AFIP 1805537).

History: A three-month-old female Alpine goat had signs of stiffness and ataxia at birth. There was subsequently sudden onset of head tilt to the left, left ear drooping, and aimless wandering. The temperature reached 103° F. There were no gross lesions at necropsy.

Laboratory Results. Extensive nonsuppurative meningoencephalitis is present with large areas of mineralization. Lesions are most severe in the white matter. Demyelination is evident in some areas.

Contributor's Diagnosis. Viral leukoencephalomyelitis of goats.

AFIP Diagnosis. Leukoencephalitis, necrotizing, mineralizing, and nonsuppurative, subacute, diffuse, severe, with multifocal, mild, meningitis, brain, Alpine goat, caprine, etiology, leukoencephalomyelitis - arthritis virus (Lentivirus, Retroviridae).

Conference Note. Caprine leukoencephalomyelitis - arthritis virus (VLG) was considered the most likely etiologic agent due to the age of the animal, and characteristic histologic changes including extensive involvement of white matter with relative sparing of gray matter, a nonsuppurative response, and the extensive granulomatous cuffs around vessels. Lesion distribution and the type of cellular response differentiates this disease from Toxoplasmosis and Listeriosis.. The moderator consulted with his colleague, Dr. L. C. Cork, concerning this case prior to the conference. This case is one of the most severe examples of the disease they have seen involving the brain. More mineralization is present than has been observed previously. There is evidence of axonal necrosis, confirmed by a Bodian stain, which is not a typical change. In some areas the lesions are suggestive of infarction, possibly a result of the thick perivascular cuffs. Although the brain lesions are severe, clinical signs probably were related to spinal cord involvement. The virus cannot be seen in tissue section by electron microscopy. Tissue cultures must be performed to demonstrate virions. Transmission of the disease appears to occur via the colostrum versus transplacentally. The virus is not strictly neurotropic and has been shown to infect several cell types including reticuloendothelial macrophages. Microglia may be part of the pathogenesis within the CNS. The Lentivirus causing maedi-visna in sheep results in similar lesions and is related antigenically to VLG. Although VLG virus can infect sheep, high doses must be injected intracerebrally; natural transmission between goats and sheep probably does not occur.

Contributor. British Columbia Ministry of Agriculture & Food, Box 100, Abbotsford, British Columbia, Canada V2S 4N8.

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2. Cork, L. C., and Narayan, O.: The pathogenesis of viral leukoencephalitis - arthritis of goats. I. Persistent viral infection with progressive pathologic changes. Lab. Invest. 42: 596-602, 1980.

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5. Summers, B. A., Appel, M.J.G., Greisen, H. A., et al.: Studies on viral leukoencephalomyelitis and swayback in goats. Cornell Vet. 70, 372-390, 1980.

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Case II - E-129-11 (AFIP 1827810). (Two H&E slides included).

History: The tissues are from a 17-month-old Basenji female that was part of a litter study. The animal had shown anemia all of her life but was otherwise clinically normal.

Representative CBC:	
Total plasma protein (g/dl)	5.9
Hemoglobin (g/dl)	5.9
Hematocrit (%)	20.0
WBC (x10°)	30.7
Segs (%)	70.0
Lymphs (%)	23.0
Monos (%)	4.0
Eosinophils (%)	2.0

6 nucleated RBC/100 nucleated cells. Reticulocytes: 40%, few poikilocytes, 4+ anisocytosis, 3+ leptocytes, slightly toxic neutrophils.

Contributor's Diagnosis & Comments. 1) Hepatosis, chronic, with portal fibrosis and hemosiderosis, secondary to chronic anemia. 2) Erythrophagocytosis and hyperplasia, lymph nodes, with hemosiderosis. 3) Bone marrow hyperplasia, long bone section, probably consequential to chronic anemia. 4) Myeloid metaplasia, spleen and liver, probably consequential to chronic anemia.

Comment. This case presents several of the cardinal signs of the pyruvate kinase deficiency reported in Basenjis. Another littermate had similar but milder changes. Bone marrow sclerosis was not predominant and myeloid metaplasia of the lymph nodes was not apparent.

AFIP Diagnoses. 1) Hepatitis, portal and periportal, chronic, with bile duct proliferation and siderophage aggregation, multifocal, mild to moderate, liver, Basenji, canine, etiology, familial erythrocytic pyruvate kinase deficiency (as specified by the contributor). 2) Extramedullary hematopoiesis, chronic, multifocal, minimal to severe, liver, spleen, lymph nodes, etiology, same as one. 3) Hemosiderosis, chronic, diffuse, minimal to moderate, liver, spleen, lymph nodes, etiology, same as one. 4) Hyperplasia, lymphoid, and erythrophagocytosis, diffuse, mild, lymph nodes.

Conference Note. Histologic changes in the liver, spleen, and lymph nodes, are similar to those described by Searcy et al. for canine familial erythrocytic pyruvate kinase deficiency. These changes plus the presence of a 40% reticulocyte count and the breed of the dog are highly suggestive of this condition. However, a definitive diagnosis can be made only with erythrocytic pyruvate kinase assay values. Cholestasis was evident in the liver. A bone section, including marrow elements, contained marked erythropoietic cell hyperplasia and a myeloid to erythroid ratio of approximately 1:4. Osteosclerosis and myelofibrosis are changes typically observed in terminal canine cases but are not a feature of the human disease. They were not seen in this animal probably due to the stage of the disease. Most dogs die by three years of age due to severe myelofibrosis and nonregenerative anemia.

Contributor. Toxicology and Pathology Research, The Upjohn Company, Kalamazoo, Michigan 49001.

References.

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2. Harvey, J. W.: Canine hemolytic anemias. J. Am. Vet. Med. Assoc. 176: 970-974, 1980.

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5. Tasker, J. B., Severin, G. A., Young, S., et al.: Familial anemia in the Basenji dog. J. Am. Vet. Med. Assoc. 154: 158-165, 1969.

Case III - 82-02 (AFIP 1824528).

History: The tissue is from a 4-year-old ewe which died shortly after being driven from one pasture to another.

Contributor's Diagnosis & Comments. "Jaagsiekte".

Comment. This case is particularly severe. Physical stress usually causes individuals with severe involvement to die -- thus the Afrikaans name for the disease which translates to "chasing or driving sickness". Once thought to be caused by a herpes virus, research has now established that a Retroviridae virus (in the Oncovirus Genus) is the causative agent (Verwoerd, et al.). This virus is distinct from the maedi/visna or ovine progressive pneumonia virus which is also a Retrovirus, but in the Lentivirus Genus.

AFIP Diagnosis. Pulmonary adenomatosis, (bronchiolo-alveolar adenocarcinoma), lung, breed unspecified, ewe, ovine, etiology, oncovirus (Retrovirus).

Conference Note. Jaagsiekte is considered neoplastic versus metaplastic by virtue of its ability to produce extrathoracic and intrathoracic metastases. Pulmonary adenomatosis was chosen for the morphologic diagnosis because not only does this term relate the distinctive histologic appearance of the neoplasm and imply the responsible etiologic agent, but it elminates possible confusion with a spontaneous lung neoplasm.

Contributors. Division of Zoonotic Disease Pathology, Armed Forces Institute of Pathology, Washington, D.C. 20306, and Onderstepoort Veterinary Research Institute, P.O. Box 12502, Onderstepoort 0110, Republic of South Africa.

References.

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Case IV - 16776 (AFIP 1803019).

History: A mass was removed from the dorsolateral neck region of a 13-year-old male domestic short hair cat. No other history was provided with the biopsy.

Contributor's Diagnosis & Comment. Mastocytoma.

Comment. Numerous metachromatic granules were present in the small cells of this neoplasm following toluidine blue staining. Fewer granules were seen in the large anaplastic cells.

AFIP Diagnosis. Mast cell tumor, skin, dorsolateral neck, domestic shorthair, feline.

Contributor. Division of Comparative Medicine, Johns Hopkins University, 720 Rutland Avenue, Baltimore, Maryland 21205.

References.

1. Moulton, J. E.: Tumors in Domestic Animals. University of California Press, Berkeley, 1978, pp 31 & 195-196.

2. Weiss, E.: VIII. Tumors of the Soft (Mesenchymal) Tissues. Bull. Wld. Health Org. 50: 101-110, 1974.

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Results
AFIP Wednesday Slide Conference - No. 23
March 31, 1982

Conference Moderator: Arthur W. Fetter, DVM, PhD
Colonel, USAFR
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Route 22
Somerville, NJ 08876

Case I - 79695 or 79954 (AFIP 1776776).

History. The tissue is from one of a group of five-week-old chickens. The affected chicks were emaciated, lame, and had fine muscle tremors.

Laboratory Results. Hypocalcemia, hyperphosphatemia.

Contributor's Diagnosis & Comment. Vitamin D deficiency rickets. One-day-old White Leghorn chicks were placed on a vitamin D deficient diet. The control group was given vitamin D_3. At five weeks, deficient chicks were near death due to hypocalcemic tetany, loss of fat and muscle, and marked bone deformities. Parathyroid mass increased to 7.5 times normal. The length of the epiphyseal growth cartilage was within normal limits, but the metaphysis was lengthened due to deep irregular projections of unmineralized cartilage into the large metaphyseal spaces with loss of orderly progression of cartilage to primary spongiosa. Large unmineralized cartilaginous cores surrounded by wide bands of osteoid occurred deep in the metaphysis. Wide osteoid seams lined resorption cavities in the cortical bone as well as metaphyseal traceculae. Fibrous osteodystrophy characterized by immature fibrous tissue associated with large numbers of osteoclasts was present subperiosteally, within resorption cavities in the cortical bone, and between trabeculae in the metaphysis.

AFIP Diagnosis. Rickets, osteomalacia, and fibrous osteodystrophy, diffuse, moderate, White Leghorn chicken, avian, etiology, hypovitaminosis D with secondary hyperparathyroidism.

Conference Note. Morphologically rickets implies a widened, irregular growth plate due to extensions of unmineralized cartilage into the proximal metaphysis and the presence of unremodeled primary trabeculae containing unmineralized cartilaginous cores in the distal metaphysis. These changes are indicative of failure of endochondral ossification. The avian metaphysis normally contains tongues of mineralized hypertrophic chondrocytes adjacent to the ascending metaphyseal vessels. In this bird, however, the hypertrophied cartilage extends twice the normal depth into the metaphysis and is unmineralized. Three differential diagnoses were considered for this case including hypovitaminosis D, phosphorus deficiency, and calcium deficiency/calcium-phosphorus imbalance. They can be differentiated, however, histologically. The presence of rickets and osteomalacia implies an inability to mineralize cartilaginous matrix or osteoid. It occurs in phosphorus deficiency and hypovitaminosis D but not calcium deficiency/calcium-phosphorus imbalance. The presence of fibrous osteodystrophy implies hyperparathyroidism. It occurs in hypovitaminosis D and calcium deficiency/calcium-phosphorus imbalance, but not phosphorus deficiency (since calcium levels are normal). The presence together of rickets, osteomalacia, and fibrous osteodystrophy is indicative, therefore, of hypovitaminosis D.

Contributor. National Animal Disease Center, P. O. Box 70, Ames, Iowa 50010.

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5. Wise, D. R., and Jennings, A. R.: The development and morphology of the Growth plates of two long bones of the turkey. Res. Vet. Sci. 14: 161-166, 1973.

Case II - 1800901 (AFIP 1800901).

History. The tissue is from a guinea pig purchased from a department store in January 1981. It was fed only guinea pig pellets obtained from a supermarket. The animal presented acutely paralyzed in the hind limbs on 27 July 1981. The appetite was normal. On physical examination the hind leg muscles were firm, the legs were stiff, and reflexes could not be elicited. Pain perception was normal to increased. Soft stool was noted around the anus.

AFIP Diagnoses. 1) Osteoporosis with physeal atrophy and cortical subperiosteal hyperostoses, chronic, diffuse, moderate, bilateral femora and tibiae, guinea pig, rodent, etiology, chronic hypovitaminosis C (scurvy). 2) Degeneration, hemorrhage, and fibroplasia, severe, multiple skeletal muscles, etiology same as one. 3) Fibrous ankylosis, and periarticular fibroplasia (pseudoankylosis), bilateral stifle joints, etiology, same as one. 4) Hyperplasia, erythropoietic cells, diffuse, severe, diaphysis, epiphysis, metaphysis and ankylosed joint space, femora and tibiae.

Conference Note. The noted musculoskeletal lesions are all manifestations of chronic scurvy. This animal had scorbutic arthropathy as described by Pirani, et al. affecting both stifle joints. The fibrous ankylosis (not evident in all sections) developed from the organization of earlier intra-articular hemorrhage, and the extensive periarticular fibrosis (pseudoankylosis) resulted from joint instability. Together they produced clinical paralysis with apparent loss of reflexes. The histologic changes in the bones of scorbutic animals are variable. In early cases with severe deficiency, bone formation at the growth plate is suppressed while cartilage production persists resulting in a thickened, irregular, mineralized, cartilaginous lattice. In chronic cases the growth plate becomes thin and uneven with distortion and loss of chondrocyte seriation. The latter changes were considered present in this animal although they may have been superimposed upon age related attenuation of the growth plate. This variable appearance of the physis apparently is due to chondrocytes being less sensitive than osteoblasts to decreased levels of vitamin C (Bonucci). The long term lack of endochondral bone formation in chronic cases results in extensive loss of trabecular bone. The resultant weakened growth plate and metaphysis are not only susceptible to

distortion and microfractures (evident in some sections of this case) but also can result in attempts to form subperiosteal new bone along the diaphysis to provide compensatory support. In both early and chronic cases of scurvy, the metaphyseal marrow spaces usually contain hypercellular immature connective tissue which fails to produce collagen. The lack of its presence in this animal was considered unusual. Similar immature fibrous tissue also is seen in chronic cases around joints, within muscle bundles, and subperiosteally. The presence of hemorrhages within multiple tissues is a hallmark of scurvy and is thought to occur from depletion of subendothelial collagen and separation of endothelial cells resulting in leakage (Gore, et al.). The extensive erythropoietic cell hyperplasia seen probably was related to numerous hemorrhagic episodes.

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Case III - 80-2268 (AFIP 1801675). (H&E oversize slide).

History: A three-year-old quarter horse gelding had an inspiratory noise when worked. The nasal septum appeared thickened on radiologic examination. The section submitted is a frontal section of the surgically removed nasal septum.

Contributor's Diagnosis & Comments. Dysplasia of the cartilage of the nasal septum with focal endochondral ossification.

Comment. Although this condition is known among equine clinicians, it is not well documented in the literature. It is described as congenital longitudinal thickening of the nasal septum. Two other cases of this condition examined by us in the last two years were essentially similar grossly and microscopically. Post-surgically, these horses show marked improvement and are able to work normally.

AFIP Diagnosis. Dysplasia, fibrocartilaginous, with multifocal ossification, nasal septum, quarterhorse, equine.

Conference Note. The mass was not considered a hyperplastic process since the cells had a disorderly arrangement and since it consisted of fibrocartilage. The septum normally is composed of hyaline cartilage. Neoplasia, e.g. chondroma, was ruled out because the cells were well differentiated and not hypertrophied. Most chondromas also consist of hyaline cartilage. The variegated appearance is due to a combination of areas which are predominantly fibrous with ones which are predominantly cartilaginous. The intense basophilia of the ground substance in the latter areas is related to its acidity and does not represent mineralization.

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Reference. Boles, C.: Abnormalities of the upper respiratory tract. Vet. Clinics of North America 1: 91-92, 1979.

Case IV - N81-1130 (AFIP 1810706). (One gross photograph included).

History: Three stillborn piglets were submitted for necropsy. The forelimbs of all three piglets were thickened especially over the length of the radius and ulna.

Contributor's Diagnosis & Comment. Congenital hyperostosis (Diaphyseal dysplasia).

Comment. Piglets with congenital hyperostosis are often stillborn; pectoral and/or pelvic limbs may be affected. Enlargement of the limb is mainly the result of new periosteal bone formation which takes the form of radially orientated bony spicules. The lesions bear some resemblance to those seen in infantile cortical hyperostosis in man.

AFIP Diagnoses. 1) Hyperostosis, subperiosteal, diffuse, severe, radius and ulna, breed unspecified, pig, etiology, congenital (autosomal recessive trait). 2) Fibroplasia, myxomatous, supraperiosteal, diffuse, severe, extraosseous connective tissues, forelimb, etiology, same as one.

Conference Note. The histologic changes and the gross appearance of this case are similar to those described by Jubb and Kennedy. The presence, however, of muscle hypoplasia could not be substantiated histologically without a control section. An infiltrate of cells is evident at the level of the periosteal cambium layer extending for a short distance into the spaces between the bone spicules. The majority of the cells are round, have an eccentric nucleus, and contain a few eosinophilic intracytoplasmic granules. These were interpreted as osteoblasts due to their similarity to other cells lining the adjacent bony spicules. Rare neutrophils are present as well as unidentifiable small mononuclear cells. A periostitis, however, was not thought to be present as occurs with infantile cortical hyperostosis in man. The cortical bone was considered to be of normal density and appearance for a neonate.

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Results
AFIP Wednesday Slide Conference - No. 24
7 April 1982

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Case I - P-3916-79 (AFIP 1822362).

History. The tissue is from a three-year-old Yorkshire sow presented with a history of recent loss of condition, posterior weakness, and neuromuscular tics.

Laboratory Results. Viral and bacterial isolation procedures were negative.

Contributor's Diagnosis & Comment. Harding's Cerebrospinal Angiopathy.

Comment. Changes observed in various brain sections examined included arteritis, thrombosis and multifocal perivascular edematous cavitation. Vascular lesions of apparent varying duration often were found in the same brain. Acute lesions included endothelial swelling, subintimal fibrinoid necrosis and adventitial edema. More chronic lesions were present as cavernous spaces surrounding small arteries, wide perivascular cuffs consisting mainly of lymphocytes and plasma cells and recanalized thrombi of larger vessels.

AFIP Diagnosis. Vasculitis, necrotizing, with perivascular edema, lymphoplasmacytic cuffs, and eosinophilic droplets, subacute, multifocal, mild to moderate, brain and meninges, Yorkshire sow, porcine.

Conference Note. Histologic changes vary between sections. The perivascular eosinophilic droplets were PAS positive. The subcommissural organ is present on the dorsal surface of the cerebral aqueduct in most sections. Swine cerebrospinal angiopathy is thought to be a subacute to chronic manifestation of edema disease. Lesions similar to the ones in this case have been reproduced experimentally with E. coli extracts or supernatants (Kurtz, et al., 1976) and have been observed in pigs surviving acute E. coli diarrhea and enterotoxemia (Nakamura, et al.). Although vasculitis in the CNS associated with PAS positive perivascular eosinophilic droplets is considered characteristic of cerebrospinal angiopathy, focal demyelination and malacia also have been described. The latter changes, however, were not observed in the sections we examined. The droplets are thought to originate from astrocytes located around affected vessels (Nakamura, et al.).

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Case II - 72848 (AFIP 1802135). (H&E and Gridley fungal stain included).

History. This animal had become lame in the left forelimb about five weeks prior to its presentation at the Small Animal Clinic. The dog had lived in El Paso, Texas, for the first two years of its life and then had been moved to the owner's new residence in the Dallas area. The lameness had been treated with corticosteroids, but no improvement was noted. On initial examination at the Small Animal Clinic, the animal was depressed, lame in the left front leg, had a fever of 103.2° F., and weighed 19 Kg.

Laboratory Results.

Clinical pathology: Total WBC - 8,100/IU/L. Serum alkaline phosphatase - 170 IU/L. SGPT - 67 IU/L

Radiographs of bone: Lesions were identified in the distal left humerus, proximal ends of both humeri, right ilium, thoracolumbar vertebrae, left coxofemoral joint, right distal femur, and sternum. The lesions were characterized radiologically as periosteal new bone formation and/or intramedullary densities. A tentative diagnosis of mycotic osteomyelitis, possibly coccidioidomycosis, was made.

Culture: Bone biopsies were submitted for culture and histopathological examination. On primary isolation, colonies of slow-growing mycelia, initially white but later black, were found. Subcultures grew faster, had a black surface and reverse pigmentation. The organism grew readily at 37° C on subculture. The conidia were identified as Phialophora sp., most compatible with P. compacta.

Gross necropsy: Nodular exostoses were observed in the bones as described under radiographic results. Additionally, granulomatous foci were noted in lungs, liver, kidney, and lymph nodes.

Microscopic examination: Skeletal lesions were widespread in the bones identified by radiographic examination. The lesions consisted of periosteal hyperplasia, with new bone spicules and fibroplasia the predominating features. Granulomas occurred both within and outside of the bone cortices. These granulomatous foci consisted of small central cores of necrosis surrounded by epithelioid cells, macrophages, and lymphocytes. Giant cells were not observed,

and H&E stains did not reveal the agent. The Gridley fungal stain revealed tangled masses of hyphae with bulbous enlargements along their course. There was no pigment in any of the fungal colonies growing in tissue. Granulomas also were found in widely distributed lymph nodes, in the lung, liver, and kidney.

In general the lesions in the lymph nodes consist of discrete granulomas, many encapsulated, with a central core of coagulative necrosis in which variable amounts of amorphous protein material is easily discernible. Surrounding the central core are macrophages, lymphocytes, fibroblasts, and plasma cells. In the medullary sinuses there are multinucleated giant cells and plasma cells, some with Russell bodies, as well as lymphocytes. At times hemosiderin-laden macrophages are evident. The etiologic agent is difficult to discern in the H&E stained sections. The Gridley fungal stain reveals variable numbers of hyphae containing large spores along their course within the granulomas.

Contributor's Diagnosis. Granulomatous lymphadenitis, multifocal, chronic, canine, etiology, Phialophora sp., probably P. compacta.

AFIP Diagnosis. Lymphadenitis, granulomatous, with multifocal and coalescing fibrocaseous granulomas, chronic, diffuse, severe, German shepherd cross, canine, etiology, Phialophora sp. (as specified by the contributor).

Conference Note. Etiologic agents submitted by the participants included Paecilomyces sp., Ajellomyces (Blastomyces) dermatitidis, Candida albicans, and Aspergillus sp. Although the hyphae and chlamydospores are morphologically compatible with Phaeohyphomycosis (chromomycosis) other aspects of the case are not consistent with the diagnosis. The fungus is not dematiaceous in unstained deparaffinized sections which is highly unusual for chromomycosis. Lesions almost always are confined to the skin and subcutaneous tissues, and metastatic dissemination is very rare. Finally, the tissue reaction is usually a mixed purulent and granulomatous type similar to that observed with blastomycosis, sporotrichosis, and coccidioidomycosis. Culture was considered necessary to determine the diagnosis in this case.

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Case III - 80-164 (AFIP 1806757).

History: The tissue is from one of three moribund and icteric rats newly arrived from a reputable supplier.

Contributor's Diagnosis & Comment. Morphologic diagnosis: Hepatitis, acute, diffuse, moderate. Etiologic diagnosis: Parvovirus (Kilham Rat Virus).

Comment. The rats were approximately five to six weeks old (weanlings). Typical intranuclear inclusions can be seen in degenerating hepatocytes, Kupffer cells, and infrequently, endothelial cells. Other salient lesions were severe necrotizing hemorrhagic orchitis (which is virtually pathognomonic for Kilham Rat Virus infections), widespread subserosal, cerebral and pulmonary hemorrhages, and a moderate accumulation of serous abdominal fluid. Inclusions were found consistently in the liver and occasionally within the endothelium of the testes but not in other tissues. Serology on another shipment of rats from the same supplier (and from the same breeding room) revealed titers to KRV of 160 to 320 in 6/10 animals. Serology done by the supplier revealed titers to KRV of 40-80 in 7/10 rats submitted from that room.

The activation of latent infections, the possibility of vertical and transplantable tumor transmission, the extreme contagiousness and resistance of this parvovirus, and often subtle reproductive effects make this a serious disease of laboratory animals, and especially producers of rats. We find it surprising that natural outbreaks of this condition are not reported more frequently in the literature.

AFIP Diagnosis. Hepatitis, acute and chronic, with hemorrhage, individual hepatocellular necrosis, and basophilic intranuclear inclusions, diffuse, mild to moderate, liver, rat, rodent, etiology, Kilham Rat Virus (parvovirus).

Conference Note. A focal capsulitis is present in some sections. The eosinophilic fibrillar material coursing through most of the sinusoids and surrounding many individual hepatocytes consisted primarily of collagen as demonstrated by the Masson's trichrome stain. An increase in reticular fibers was evident in areas of parenchymal collapse but only accounted for a small portion of the fibrillar material.

Contributor. Pfizer Central Reseach, Groton, Connecticut.

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Case IV - 879 (AFIP 1802946). (Two electron micrographs included).

History: The tissue is from a ten-year-old female miniature schnauzer with a two-month history of seizures.

Contributor's Diagnosis & Comment. Cerebral granular cell tumor (myoblastoma). Comment. Necropsy revealed a white granular tumor mass invading from the meninges into the occipital area of cortex. The tumor cells contained a PAS positive diastase resistant granular cytoplasm. Electron microsopy revealed these granules to be nonhomogenous, variable-sized electron-dense granules consistent with those associated with granular cell tumor.

AFIP Diagnosis. Granular cell tumor, occipital cerebrum and meninges, brain, schnauzer, canine.

Conference Note. Participants unanimously concurred with the contributor. Spindle cells which were located at the periphery of nodules and scattered throughout the main mass of the tumor, (but not in the stroma) contained large, intracytoplasmic, intensely PAS positive granules compatible with angulate bodies. There is no consensus on the histogenesis of the granular cell. Histiocytes, smooth muscle cells, fibroblasts, and Schwann cells have been proposed as its precursor. Currently, "myoblastoma" and schwannoma cells are thought to stem from a common, multi-potential, mesenchymal cell (interstitial cell) located within the stroma (Sobel et al., 1973). Granular cells, probably histiocytic, also occur in nonneoplastic lesions, often sites of previous trauma. They can be differentiated from "myoblastoma" cells by ultrastructural and histochemical characteristics including intracytoplasmic granules which are strongly PAS positive, acid fast, and iron positive (Sobel et al., 1974). In the dog there have been five reported granular cell tumors in the tongue and one in the cerebrum (Parker, et al.).

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Results
AFIP Wednesday Slide Conference - No. 25
14 April 1982

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Case I - M20324 (1810449).

History: A three-year-old female Patas monkey was given an intravenous injection of ethylnitrosourea over 1.5 years ago while pregnant. Gross findings included a swollen endometrium and a large mass attached to the wall of the uterus and the serosa of the colon. The colonic epithelium and endometrial lining appear normal. Evidence of tumors in other organs was not observed.

Contributor's Diagnosis & Comment. Choriocarcinoma.
Comment. The uterus in many sections does not provide good evidence of the origin of the tumor; however, the stroma is not normal and in some slides foci of large trophoblasts are seen. The mass attached to the colon is a poorly differentiated carcinoma and contains areas of syncytial formation. In another section of uterus but not on these slides, areas of large trophoblast-like cells were seen; however, the lesions are small and not identical to the mass on the colonic serosa. One vessel in the endometrium appeared invaded by the large trophoblast-like cells. Seven other cases in this study showed a progression of typical uterine lesions to those of uncertain origin as in this case. The spectrum resembles the human disease.

AFIP Diagnoses. 1) Choriocarcinoma, metastatic to the serosa and tunica muscularis, colon, Patas monkey (Erythrocebus patas), nonhuman primate, etiology, ethylnitrosurea. 2) Hyperplasia, stromal cells, with endothelial hypertrophy and hyperplasia of the superficial spiral arterioles (epithelioid cytomorphosis), endometrium, uterus.

Conference Note. Choriocarcinoma and undifferentiated carcinoma were the two most frequent diagnoses submitted for the colonic neoplasm. Both endometrial carcinoma and stromal sarcoma were submitted for the uterus.

This case was part of a study done by Rice, et al. The neoplasms were not seen in males, in untreated control females, in nonpregnant females exposed to ethylnitrosurea (ENU), or in exposed or control offspring of either sex. Of 59 pregnant monkeys injected with ENU at various dosages, seven died or were euthanatized with a widely disseminated neoplasm within six months after receiving the last injection. Tumor nodules were found in the lungs of all affected animals and were morphologically compatible with a choriocarcinoma. Sections of uterus were unremarkable grossly and no exophytic tumor mass was ever seen. Microscopically, diffuse or well demarcated foci of tumor tissue resembling that of the pulmonary metastases were observed infiltrating the endometrial stroma and forming

endovascular tumor deposits in the veins. Attempts to detect chorionic gonadotropin in the urine or sera of normally pregnant or tumor bearing Patas monkeys by immunoassays that have been used with other primate species were unsuccessful. Rice et al. commented that use of this parameter to confirm histological interpretations will require development of an assay specific for the Patas hormone. They also commented that chorionic gonadotropin can be measured in other nonhuman primate species, such as the rhesus, only during the first few weeks of pregnancy. This pattern is different from the persistence of the hormone throughout pregnancy in the great apes and women. The Patas could be similar to the rhesus and accordingly tumors of trophoblastic origin also may not continue to produce chorionic gonadotropin.

The presence in the colon of large anaplastic cells with vesicular nuclei compatible with cytotrophoblasts and of multinucleated cells with hyperchromatic nuclei compatible with syncytiotrophoblasts, associated with hemorrhage and necrosis was consistent with the features of a choriocarcinoma as described by Robbins et al. Our attempts to confirm this diagnosis by demonstrating chorionic gonadotropin within the syncytial cells using an immunoperoxidase technique for HCG were unsuccessful. Changes in the endometrium were difficult to interpret. Multiple round to irregular tubular structures were observed along the superficial border and were lined by one to three layers of large, round to polygonal cells. Some of these structures apparently were surrounded by smooth muscle. Three possible origins for these cells were discussed: Endovascular invasion by normal or neoplastic cytotrophoblasts; glandular epithelium proliferating in response to the presence of chorionic tissue (atypical endometrial change or the Arias-Stella effect); and endothelial hypertrophy and hyperplasia of the superficial spiral arterioles (epithelioid cytomorphosis). In the experience of the moderator the last interpretation was the most likely explanation. This endothelial reaction normally is seen in the endometrial vessels immediately below the placental implantation site. It occurs during the first month of pregnancy but is not observed in late gestation. The reaction apparently is caused by the hormone relaxin being released from endometrial granulocytes (Dallenbach-Hellweg). The latter cells are not hematopoietic in origin but reportedly are derived from endometrial stromal cells. They were seen in this case and were characterized by hyperchromatic, slightly indented, eccentric nuclei and by numerous intracytoplasmic PAS positive eosinophilic granules. Endometrial granulocytes of the monkey virtually are identical to those of the human uterus and to the granular cells of the mesometrial decidua and metrial gland of the pregnant rat (Dallenbach-Hellweg). Histochemically, however, a mucopolysaccharide prosthetic group is attached to the protein in the nonhuman primate's granulocytes (Dallenbach-Hellweg); this feature may account for its PAS positive cytoplasm. The endometrium in this case was thickened markedly and the stroma was extensive. Changes of both the glands and stroma were suggestive of long term exposure to progestins and were similar somewhat to the decidual change seen in nonhuman primates that are pregnant (Ramsey et al.) or on long term birth control therapy. Additionally if the neoplasm was functional, exposure to chorionic gonadotropin with feedback to the ovary may have resulted in prolonged secretion of progesterone.

In a study of the placental implantation site in women, rhesus monkeys and baboons, Ramsey et al. demonstrated that the human trophoblast is much more invasive than its nonhuman counterparts. This characteristic resulted in comparatively deeper implantation and a highly irregular fetal-maternal junction. This lower invasive potential may account for the extreme rarity of choriocarcinoma in nonhuman primates (Ramsey et al.).

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Case II - Mk81-879-AA (AFIP 1822617).

History. The tissue is from an adult rhesus monkey experimentally inoculated with an infectious agent. The monkey died shortly after inoculation. At necropsy the spleen was markedly enlarged and severely congested; the liver was slightly enlarged and uniformly slate-grey; and the kidneys were diffusely dark red and slightly swollen.

Laboratory Results. Peripheral blood smears obtained at necropsy were stained with Giemsa. Examination revealed Plasmodium sp. in nearly 100% of the circulating erythrocytes.

Contibutor's Diagnosis & Comment. Nephrosis, hemoglobinuric, acute, experimentally-induced.

Comment. The renal lesions were typical of a severe acute malarial infection. Later it was learned that the investigator had inoculated the monkey with Plasmodium knowlesi.

AFIP Diagnoses. 1) Parasitemia, hemoprotozoal, characterized by intraerythrocytic pigmented trophozoites, diffuse, severe, blood vessels, kidney, rhesus monkey (Macaca mulatta), nonhuman primate, etiology, Plasmodium knowlesi (as specified by the contributor). 2) Nephrosis, hemoglobinuric, acute, diffuse, moderate to severe, kidney, etiology, same as one.

Conference Note. Histologic changes seen in this case virtually are identical to those described by Rosen et al. The hyaline eosinophilic droplets within the tubular epithelial cells were demonstrated to be hemoglobin by special stains. Numerous small to medium sized clear vacuoles also were seen at the base of many tubular epithelial cells. These were described by Rosen et al. and shown to be lipid. The authors commented that high levels of free fatty acids occur within the

blood in P. knowlesi infections and postulated that they may have diffused into the base of the cells from the peritubular capillaries. Alternative origins considered were focal cytoplasmic degradation or products of hemoglobin digestion. (Compare the vacuoles seen in this case with the ones evident in Case III of this Conference.) The malarial pigment (hemozoin) present in association with the trophozoites and within some interstitital macrophages cannot be differentiated from acid hematin histochemically. However, it is iron positive after microincineration. The trophozoites were seen to contain a central vacuole (ring form) in Giemsa stained sections. No schizonts were observed. The renal changes in rhesus monkeys infected with P. knowlesi are similar to those seen in man with blackwater fever due to P. falciparum (Connor, et al.).

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Case III - 81-38 (AFIP 1803081).

History: An adult male baboon was found dead in his cage the morning after injection with an experimental drug (cardiodilator).

Contributor's Diagnosis. Oxylate nephrosis. The drug was dissolved in ethylene glycol.

AFIP Diagnoses. 1) Nephrosis, characterized by intraluminal and intraepithelial oxalate crystals, acute, multifocal, severe, kidney, baboon (Papio sp.), etiology, ethylene glycol (as specified by the contributor).
2) Nephritis, interstitial, chronic, multifocal, mild to moderate, kidney.
3) Nephritis, tubular, acute, multifocal, mild to moderate, kidney.

Conference Note. The crystals were considered characteristic of oxalates based upon their morphology, their radial arrangement within tubular lumina, and their multicolored appearance upon polarization. The inflammatory changes in the renal parenchyma were not thought to be related to the crystal deposition, and the occasional presence of crystals within aggregates of luminal inflammatory cells was considered incidental. Although the acute tubular nephritis was probably a manifestation of a pyelonephritis, the latter diagnosis could not be made without examining the renal pelvis. Eosinophilic intranuclear inclusions were noted within epithelial cells. They were acid fast negative. They were not considered typical of viral inclusions or cytoplasmic invaginations.

Contributor. Naval Medical Research Institute, National Naval Medical Center, Bethesda, Maryland.

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Case IV - SWRI 2A72B (AFIP 1802985). (Two H&E slides included).

History: The tissues are from an aged female rhesus monkey with abdominal distention and constipation. The animal died while in preparation for radiography. At necropsy the large intestine was distended with gas and feces. A large red multilobulated mass was observed involving the uterus, bladder and intestine.

Contributor's Diagnosis & Comment. Uterus: leiomyoma and endometriosis. Colon and ileum: endometriosis.

Comment. The immediate cause of death was stricture of the ileum and colon related to endometriosis. This condition is very common in our colony; these monkeys often present with gastrointestinal signs. An association of leiomyomas with endometriosis has been made in humans.

AFIP Diagnoses. 1) Endometriosis, serosa, ileum, colon, and uterus, rhesus monkey (Macaca mulatta), nonhuman primate. 2) Fibroleiomyoma, myometrium, uterus.

Conference Note. An extensive amount of collagen was demonstrated within the myometrial mass, a typical characteristic of leiomyomas in primates. Fibroleiomyomas are similar to the "fibroid" seen in the uterus of human females. Current theories for the pathogenesis of endometriosis include regurgitation of endometrium through the infundibulum of the oviduct into the peritoneal cavity during the normal menstrual cycle, and metaplasia of coelomic epithelium into endometrial tissue. A definitive histologic diagnosis of endometriosis requires two of the three following features: Glands, stroma, or hemosiderin pigment (Robbins, et al.).

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Results
AFIP Wednesday Slide Conference - No. 26
21 April 1982

Conference Moderator: Michael H. Goldschmidt, MSc, BVMS, MRCVS
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Case I - 47540 (AFIP 1815104).

History. An 18-month-old thoroughbred filly had severe, generalized seborrhea with alopecia. In the inguinal and axillary areas, multiple, small, round, grey papules were present measuring approximately 2-4 mm in diameter. The papules often were umbilicated and some had a tiny central pore, which if squeezed extruded a caseous core. This section is taken from inguinal skin.

Laboratory Results. Electron microscopy: The inclusion bodies in the keratinocytes contain very large numbers of poxvirus virions. Negative staining of extracts from the lesions showed virus particles with morphology consistent with that of molluscum contagiosum virus.

Contributor's Diagnosis & Comments. Molluscum contagiosum-like dermatitis. Comment. The lesion is grossly, histologically and ultrastructurally typical of human molluscum contagiosum which is an uncommon skin condition caused by an unclassified poxvirus (4-6). Molluscum contagiosum-like lesions have been described in a kangaroo (1), horse (3) and in captive chimpanzees (2).

AFIP Diagnosis. Hyperplasia and superficial degeneration, with eosinophilic intracytoplasmic inclusions, multifocal, moderate, epidermis, skin, thoroughbred, equine, etiology, poxvirus (molluscum contagiosum).

Conference Note. The eosinophilic intracytoplasmic inclusions, also called molluscum bodies, are not discrete collections of virions. Ultrastructurally they consist of many vacuoles filled with mature viruses. These vacuoles compress the surrounding cytoplasm into thin trabeculae which contain immature spherical virions and ribosomes (Lutzner). In addition to the case reports mentioned by the contributor, a probable molluscum contagiosum-like pox infection also was described in two South American sea lions by Wilson et al. (8)

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Case II - 81-1238B (AFIP 1737277). (Two gross photographs included).

History: A single Africaner calf became severely ill; its haircoat was moist to touch, and erosive lesions appeared over bony prominences. The calf became progressively depressed within a 24-hour period and died.

Contributor's Diagnosis & Comments. Sweating sickness with secondary cutaneous dermatophilosus.

Comment. No ticks were found on this animal but dipping was reported to be performed on an irregular basis. Lesions represented are from the skin of the ear, the esophagus, and the pharynx/larynx area. Only stratified squamous epithelium and epidermis are involved. Photographs submitted show skin lesions on the live calf and postmortem esophageal and laryngeal lesions. The skin lesions in this case look very similar to toxic epidermal necrolysis as described in man, dogs, cats, primates, and horses. The latter syndrome is either idiopathic or occurs following exposure to certain drugs.

AFIP Diagnoses. 1) Dermatitis, necrotizing, acute, diffuse, severe, epidermis and papillary dermis, skin, pinna, ear, Africaner, bovine, etiology, sweating sickness toxin with secondary Dermatophilus congolensis and other bacteria. 2) Esophagitis, necrotizing, acute, diffuse, severe, mucosa, esophagus, etiology, sweating sickness toxin with secondary bacteria. 3) Laryngitis, necrotizing, acute, diffuse, severe, mucosa, larynx, etiology, same as two.

Conference Note. Sections of larynx were not present on all slides. Differential diagnoses considered were sweating sickness and malignant catarrhal fever. Several participants noted focal vasculitis and thrombi. In personal communication between the moderator and J. A. Lawrence, several unpublished observations concerning the etiology of sweating sickness were discussed. Current research indicates that Hyaloma transiens (truncatum) ticks capable of transmitting the disease are infected with a rickettsial organism which resides in their

salivary glands. Upon feeding, a toxin produced by the Rickettsia enters the host. The amount of toxin injected determines the severity of the disease and is dependent upon the time period of engorgement of adult female ticks; ticks feeding for periods of 144 hours or longer are usually fatal. Male ticks carry the toxin but feed intermittently and inject much less saliva than females. A necrotic stomatitis-nephrosis syndrome without cutaneous lesions in Rhodesian cattle also has been described by Lawrence, who originally thought it was a form of sweating sickness (Lawrence, 1973). This syndrome is now believed to represent a different disease with a similar pathogenesis. Nephrosis is not a feature of sweating sickness. Both of these conditions are characterized by severe neutropenia with a left shift including metamyelocytes and toxic neutrophils.

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Case III - 80-508 (AFIP 1805677).

History: The tissue is from a one-year-old Holstein cow with numerous nodules 3 mm to 1 cm in diameter.

Contributor's Diagnosis & Comment. Dermatitis - subacute to chronic, granulomatous, focal, etiology, Demodex bovis.

Comment. Demodex bovis can cause significant damage to the hides of cattle and in rare instances death due to secondary bacterial infection. The mites parasitize the hair follicles and sebaceous glands which become cystic. If the lining of the cyst becomes disrupted, a focal granulomatous inflammatory reaction results.

AFIP Diagnosis. Dermatitis, subacute, with multinucleated giant cells, nodular, focal, moderate to severe, dermis, Holstein, bovine, etiology, Demodex bovis.

Conference Note. The lesion was not considered granulomatous due to an insufficient number of histiocytes. The diagnosis of Demodex bovis was based upon observing cross-sections of arthropods with an elongated abdomen, a minimal amount of musculature, and four pairs of coxae originating contiguously from the cephalothorax. In other genera of mites, the anterior two pairs and posterior two

pairs of coxae are separated by a short distance. The small basophilic nuclei .75 to 1 μ in diameter that are present in some cross-sections are portions of salivary gland. The abdomen of Demodex sp shrinks slightly during processing and is disproportionately small in section. The extensive number of lymphocytes seen in this case was considered unusual. The appearance of mite fragments within multinucleated giant cells and the lack of identifiable adnexal epithelium are consistent with the early healing stage described by Bwangamoi. Transmission studies of Demodex bovis indicate that prenatal transmission does not occur and that calves become infested within a few days of birth, most likely while nursing.

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Case IV - 1481K (AFIP 1619250).

History: An ulcerated mass measuring three fourths inch by one inch was noted on the foreleg of a ten-year-old female spayed poodle over the radius.

Microscopic Description. The lesion is elevated above the level of the surrounding skin, and is partially covered by a hyperplastic epidermis with a central area of ulceration which is covered by a fibrinopurulent pseudomembrane. Extending from the epidermis into the underlying dermis are cords of basophilic cells, some showing abrupt central keratinization. These extend into the deep dermis where they evoke a fibroblastic response by the host.

The invading tumor islands are of variable size. Many have a peripheral zone of basophilic basaloid cells with a central eosinophilic origin, in which ghost cells are present. Other areas form small squamous eddies resembling hair follicles. Rupture of a neoplastic focus and release of keratin has induced an acute inflammatory response in this area, with marked neutrophilic infiltration.

Contributor's Diagnosis & Comment. Tumor of hair cortex cell with differentiation towards trichoepithelioma and pilomatrixoma.

Comment. This lesion shows the formation of typical hair structures, and contains areas more suggestive of a pilomatrixoma (necrotizing and calcifying epithelioma of Malherbe). The cell of origin is a primitive basal cell of the epidermis. The lesion is invasive and induces a sclerotic host response. A guarded prognosis should be given as recent cases of malignant pilomatrixoma have been described in the dog.

AFIP Diagnosis. Hair follicle tumor, malignant, skin, poodle, canine.

Conference Note. Diagnoses submitted included trichoepithelioma, invasive trichoepithelioma, pilomatrixoma, and basosquamous carcinoma. Histologic features of both a trichoepithelioma and pilomatrixoma were present and resulted in using the more general term, hair follicle tumor. The lack of gradual keratinization, keratin pearls, and intercellular bridges ruled out basosquamous carcinoma. The moderator commented about his experience with malignant hair follicle tumors. Islands and epidermal downgrowths of basal type cells often containing central areas of keratin are seen extending deep into the dermis and are surrounded by an edematous fibroblastic response. The latter response is characteristic in these neoplasms. Features of both the trichoepithelioma and pilomatrixoma can be seen. The mitotic activity is usually low; if high, the associated lymph nodes and lungs should be evaluated. The prognosis is reasonably good with wide surgical excision.

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Results
AFIP Wednesday Slide Conference - No. 27
28 April 1982

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Case I - AFRRI #2 (AFIP 1791669). (2 electron micrographs included).

History. The tissue is from a Senegal parrot which died acutely.

Laboratory Results. Electron microscopy demonstrated multiple intracellular clusters of organisms in the spleen and in the liver (Kupffer cells). Initial, elementary, and intermediate bodies, typical of Chlamydia sp., were identified.

Contributor's Diagnosis & Comment. 1) Hepatitis, necrotizing, multifocal, severe, liver; caused by Chlamydia psittaci (psittacosis). 2) Microfilaremia, liver.

Comment. This parrot had been released from government quarantine two weeks previously. Within the cytoplasm of numerous sinusoidal lining cells (Kupffer cells) in the liver are clusters of small basophilic bodies. Although present on routine H&E staining, they are more clearly recognized with special stains: Pinkerton, Giemsa, and Brown and Hopps Gram stain. Other lesions in this parrot included a serositis and necrotizing splenitis. Organisms could also be identified within these other tissues. Electron microscopy demonstrated the basophilic bodies to have morphologic characteristics typical for Chlamydia sp. The microfilaria cannot be speciated, but microfilaria have been found previously in this host.

AFIP Diagnoses. 1) Hepatitis, necrotizing, acute to subacute, multifocal, moderate, liver, Senegal parrot, avian , etiology, Chlamydia psittaci.
2) Microfilaremia, liver.

Conference Note. Intracytoplasmic inclusions were seen occasionally within hepatocytes as well as within circulating monocytes and Kupffer cells. Although chlamydiosis in birds causes individual hepatocellular necrosis of variable severity, multiple foci of necrosis are only reported in parrots (and mice). The pathogenesis of the multifocal necrosis was not discussed in the literature reviewed. Possible mechanisms considered were lysis by the proliferating developmental forms and necrosis due to ischemia.

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Case II - 80443 (AFIP 1803337).

History. Reproductive tract samples were obtained from a 5-year-old quarterhorse mare. This animal received an intrauterine inoculation of a bacterium 48 hours prior to necropsy. Within 24 hours a discharge was seen at the ventral labial commissure. At necropsy the entire uterus, cervix and anterior vagina were covered with a grey, tenacious, opaque exudate.

Laboratory Results. A pure population of a gram negative coccobacillus was cultured from the reproductive tract.

Contributor's Diagnosis & Comment. Contagious equine metritis.
Comment. The intensity of the uterine lesions will vary with location of the sample. Cervical samples show the most remarkable lesions.

AFIP Diagnosis. Endometritis, vaginitis, and cervicitis, acute to subacute, diffuse, mild, endometrium, vagina and cervix, quarterhorse, equine, etiology, Hemophilus equigenitalis (as specified by the contributor).

Conference Note. Contagious equine metritis caused by Hemophilus equigenitalis was considered the most likely diagnosis based upon the history and gross lesions. Differential etiologic agents included Klebsiella pneumoniae var genitalium, Streptococcus zooepidemicus, and Pseudomonas aeruginosa. Although focal epithelial hyperplasia has been described in the endometrium it was not present in the sections we examined. The mild severity of the lesions was unexpected considering the clinical signs. A Brown and Hopps stain revealed aggregates of gram negative coccobacilli associated with eosinophilic material within the lumina of multiple endometrial glands.

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Case III - 81-1349 (AFIP 1805545).

History. The tissues are from 1 of 3 littermates that ranged in age from 4 to 6 weeks. All puppies had died or had been euthanatized in extremis. The most common signs were seizures and hypothermia. The healthy, 11-year-old female had lost one pup immediately after birth and a second pup within one week. The pups had been whelped and maintained in an abandoned, dirt floor garage that was frequented by neighborhood cats.

Laboratory Results. Necropsy results were hepatomegaly, focal pneumonia, and mesenteric lymph node enlargement. The renal cortices were pale. Gross lesions were not evident in the brain or spinal cord. Histopathological results included nonsuppurative encephalitis and interstitial nephritis, hepatitis, focal retinitis, gastritis, lymphadenitis, and myocarditis with necrotizing vasculitis. A gram positive microsporidium type of organism was demonstrated in the brain, myocardium, spleen, liver, stomach, and lymph nodes. Transmission electron microscopy revealed both intra- and extracellular organisms that were morphologically similar to the microsporidium, Encephalitozoon. (Dr. F. Chandler, CDC, Atlanta, GA).

Contributor's Diagnosis & Comment. 1) Necrotizing arteritis. 2) Myocarditis. 3) Encephalitozoonosis.

Comment. The fibrinoid necrosis of the myocardial small and medium size arteries is similar to that described in the blue fox. Some arteries had small granulomas within the wall, and rarely arteries were surrounded by exudate consisting of granulocytes, lymphocytes, plasma cells, and macrophages. Arterial necrosis was not evident in other organs, including the brain. Severe diffuse nonsuppurative reactions and abundant organisms were seen in the brain and renal cortices.

Encephalitozoon was distinguished by poor staining with H&E, gram positive reactions with Brown & Brenn and Goodpasture's stains, a PAS reacting granule that had a polar location, and transmission electron micrographs showing a polar filament. Toxoplasma is gram negative, has a PAS positive granule located in a subpolar position, and does not have a polar filament.

AFIP Diagnosis. Vasculitis, necrotizing, and myocarditis, acute to subacute, multifocal, moderate to severe, heart, breed unspecified, canine, etiology, Encephalitozoon cuniculi.

Conference Note. Spores were not present in all the sections we examined. The gram negative appearance of some spores may have been due to early degenerative changes. The pathogenesis of vasculitis in encephalitozoonosis has not been completely determined and is possibly due to a combination of factors. Nordstoga et al., 1976 suggested that the arterial lesions in blue foxes were morphologically similar to classical polyarteritis nodosa. Infected foxes have been shown to

exhibit a pronounced hypergammaglobulinemia. Van Dellen et al., however, commented that the distribution and severity of the vasculitis in dogs was variable even in an animal with subacute disease in which a state of hypersensitivity should have been established. They believed that the necrosis could have been caused by vascular ischemia resulting from two possible mechanisms. Parasitism of multiple endothelial cells at two different but close points in small vessels could result in vascular occlusion and ischemia, and focal parasitism of endothelial cells of the vasa vasorum could result in segmental ischemia of larger vessel walls. Nosema sp. has not been reported in dogs to date, although some early literature incorrectly used this genus instead of Encephalitozoon.

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Case IV - 29935 (AFIP 1827311). (Two electron micrographs included).

History. The tissue is from a mass which was surgically removed from the left side of a female gopher snake (Piterophis catenifer var. sayi). This excision was the fourth time a mass was removed from this site.

Contributor's Diagnosis & Comment. Malignant chromatophoroma, subcutis, gopher snake.

Comment. The masses were all rust color. The electron photomicrographs contain structures compatible with melanosomes and pterinosomes within the same tumor cells. This arrangement is indicative of mosaic chromatophores. This tumor has recurred locally, but metastasis has not been diagnosed. When compared to the previous masses, this one is morphologically the most suggestive of malignancy. The behavior of this neoplasm must await the death and necropsy of the snake.

AFIP Diagnosis. Chromatophoroma, malignant, subcutis, gopher snake (Piterophis catenifer var. sayi), Reptilia.

Conference Note. Special stains demonstrated melanin within scattered cells of the neoplasm. The concentration of melanin in positive cells was not heavy and granules were dispersed evenly throughout the cytoplasm.

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Results
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Case I - N80-736 USAFSAM (AFIP 1802918).

History. A young aardwolf (Proteles cristalus) was found moribund in its exhibit cage. Shock therapy was initiated but the animal died on the table.

Laboratory Results: Indirect FA - positive.

Contributor's Diagnosis & Comment. Myocarditis, necrotizing, granulomatous,. multifocal, severe, heart, aardwolf, etiology Trypanosoma cruzi.

Comment. Microscopically there was severe multifocal to diffuse necrotizing granulomatous myocarditis. Amastigotes of T. cruzi were seen often within the inflammatory cell foci. Nests of amastigotes, numbering up to 25 or more, were generally within myocardial but were also extracellular and within macrophages. The myofibers were often necrotic and had variably eosinophilic sarcoplasm, loss of cross striation, fragmentation, and nuclear degeneration. Necrotic myofibers were being replaced by granulation tissue. The inflammatory cell infiltrate was composed of macrophages, lymphocytes and plasma cells. TEM - The distinguishing morphologic features of T. cruzi amastigotes seen were the kinetoplast, basal body, periplast, flagellum and nucleus.

AFIP Diagnosis. Myocarditis, chronic-active, diffuse, moderate, with multifocal intracellular amastigotes, myocardium, heart, aardwolf (Proteles cristalus), etiology, Trypanosoma cruzi.

Conference Note. The inflammation was not considered granulomatous due to the lack of adequate numbers of macrophages within cellular infiltrates. A Masson trichrome stain demonstrated diffuse interstitial fibrosis. Several characteristics of T. cruzi allow differentiation from Leishmania sp. T. cruzi amastigotes are approximately one and one-half times larger, have a prominent, intensely basophilic kinetoplast which usually is aligned parallel to the nucleus and typically are found in parenchymal cells. Leishmania sp. amastigotes usually are located within phagocytes and have a kinetoplast which lies perpendicular to the nucleus. The kinetoplast of Leishmania sp. often is difficult to visualize.

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Case II - BZ 65-81 (AFIP 1803009).

History. A male adult Nyala (Tragelaphus angasi) was inactive for weeks, had atrial fibrillation at EKG examination, dependent pitting edema, venous engorgement, and chronic dermatitis. It was immobilized with M99 and Rompun. Treatment included antibiotics, steroids, diuretics, and vitamin E and selenium, but the animal died.

Laboratory Results. Serum T3 was less than 0.1 (normal - 2.5 ± 0.5 mg%) and serum T4 was 1.5 (normal - 7.6 ± 1.4 mg%). Normal values were established by testing several clinically normal nyalas. Vitamin E and selenium levels in the blood were normal.

Contributor's Diagnosis & Comment. Myocardial fibrosis and myxedema, hypothyroidism.

Comment. The nutritional condition of the animal at necropsy was considered adequate. Gross lesions included absence of both thyroid glands and chronic passive congestion of the liver. Myxedematous degeneration of adipose tissue was observed in the atrioventricular groove, in the subcutis, and in the mesentery. Interstitial myocardial fibrosis and myocytolysis were only evident subadjacent to areas of myxedema. The lesions in the heart probably caused cardiac failure and were responsible for the clinical signs. Generalized myxedema has been observed in human patients, dogs, and pigs with hypothyroidism.

AFIP Diagnoses. 1. Myxomatous change, diffuse, severe, epicardium, heart, nyala (Tragelaphus angasi). 2. Fibrosis, interstitial, diffuse, severe, myocardium, heart.

Conference Note. Although adequate evidence to support the diagnosis of subepicardial myxedema exists, the morphologic appearance of this tissue also is compatible with mucoid degeneration of fat as described by Smith, Jones, and Hunt. Special stains demonstrated the presence of acid mucopolysaccharides in the myxomatous area of the epicardium. Although the interstitial fibrosis was seen only in association with the myxomatous epicardial tissue, a correlation between the two changes could not be made nor could the pathogenesis of the fibrosis be determined.

Contributor. The Animal Medical Center, 510 East 62nd Street, New York, New York 10021.

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Case III - 6621 I & II (AFIP 1830594). (Two H&E sections included).

History. The tissues are from one of six adult Saiga antelopes (Saiga tatarica) which died peracutely. Severe bronchopneumonia and hemorrhagic, catarrhal enteritis were noted at necropsy.

Laboratory Results. Bacterial culture of affected tissues was negative.

Contributor's Diagnosis & Comment. Disseminated toxoplasmosis.

Comment. Three acute lethal cases of toxoplasmosis in Saiga antelope were described by Bulmer in 1971 which were histologically and clinically similar to the ones we encountered. These cases demonstrate that the Saiga antelope are not representative of other Artiodactylids in their susceptibility to T. gondii infections. Toxoplasmosis in most Artiodactylids is chronic and found incidentally at necropsy. The source of the organisms in our antelope could not be determined. Deep frozen (-30°F) liver and lymph node tissues from three different animals were injected into mice. Three to ten days post-inoculation the mice were euthanatized. Giemsa stained smears of peritoneal fluid revealed toxoplasma gondii trophozoites within the cells of one mouse, demonstrating the ability of the organism to survive freezing.

AFIP Diagnoses. 1. Hepatitis, necrotizing, acute to subacute, multifocal, moderate, liver, Saiga antelope (Saiga tatarica), etiology Toxplasma gondii. 2. Lymphadenitis, necrotizing, acute to subacute, diffuse, severe, lymph node, etiology, same as one.

Contributor. Research Department of Vertebrates, Zoological Park of East Berlin.

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Case IV - X1756 (AFIP 1803705). (One gross photograph included).

History. The tissue is from a California sea lion (Zalophus californianus) with cutaneous nodules.

Contributor's Diagnosis & Comment. Mycotic dermatitis due to Fusarium sp. most probably F. solani.

Comment. Dermatitis associated with Fusarium sp. infection developed in three California sea lions (Zalophus californianus) and three gray seals (Hauchoerus grypus) at the National Zoological Park in Washington, D.C. The lesions were papular or nodular and were distributed mainly on the face, trunk, and flippers. One sea lion died six weeks after extensive cutaneous involvement. The lesions regressed after one mild exacerbation in the other two sea lions. In the gray seals, the skin condition appeared to worsen during the summer and to regress during the winter despite oral and topical miconazole and thiabendazole.

Fusarium sp. was repeatedly isolated from biopsy specimens of lesions. Hyperplasia of epidermal and follicular epithelium was associated with acute and chronic inflammation and fungal hyphae. The species of the fungus in one of the gray seals was determined to be F. solani, a type occasionally associated with keratitis and opportunistic infections in human beings (Forster, et al.).

Initial excessive chlorination and high fluctuating pool temperature, attributed to a faulty water treatment system, were considered as factors in promoting fungal growth.

AFIP Diagnosis. Dermatitis, with acanthosis and intraepidermal abscesses, subacute, focally extensive, moderate, skin, California sea lion (Zalophus californianus), etiology, Fusarium sp. (as specified by the contributor).

Conference Note. Differential etiologic agents submitted by attendees included Fusarium sp., Dermatophilus congolensis, Candida sp., Aspirgillus sp., and one of the dermatophytes. Special stains demonstrated septate hyphae with parallel walls. Ranging from 2 u to 5 u in diameter within the outer layers of the hyperplastic epithelium of both the epidermis and distended hair follicles. Montali et al. commented that pinnipeds are relatively resistant to superficial mycoses particularly dermatophytes. Some of this resistance in the Northern fur seal (Callorhinus ursinus) has been attributed to fatty acid secretions, which apparently act as natural barriers to fungal penetration. Focal dermatitis with a seasonal prevalence has been reported in California sea lions, with similar histologic features to this case but the cause was unknown (Sweeney). Nematode larvae were observed within follicles in a few sections.

Contributor. Department of Pathology, National Zoological Park, Washington, D.C. 20008.

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Results
AFIP Wednesday Slide Conference - No. 29
19 May 1982

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Case I - 81-775 (AFIP 1822536).

History. The tissue is from a wild caught, nine-banded armadillo (Dasypus novemcinctus) which died while being conditioned for laboratory use. The tissue presented is stained with GMS and counterstained with H&E. The armadillo had been losing weight since capture. Necropsy findings included enlarged, diffusely mottled kidneys.

Contributor's Diagnosis & Comment. Sporotrichosis (Sporothrix schenkii). Comment. Sporotrichosis has been reported in many animal species. This is the first case in an armadillo. The disease is generally limited to the skin, subcutaneous tissues and lymphatics where linear nodules are formed. Disseminated sporotrichosis with visceral involvement is very rare, but cases in man involving lung, bone and other organs have been reported. In this section of kidney, numerous randomly scattered and variably sized granulomas are present. The granulomas are invested with fibrous tissue in some areas. Numerous Sporothrix schenkii organisms are seen in different sizes and shapes with some cigar forms and budding. The organisms are scattered amongst epithelioid cells, neutrophils, and are often seen within giant cells. On H&E sections the organisms are not visible, and only faint suggestive profiles are seen, often within giant cells. The use of the GMS and H&E stain enables visualization of the causative organism and characterization of the associated inflammatory response. A mixed, purulent and granulomatous response with a variable amount of fibrosis and the demonstration of characteristic yeast cells is suggestive of sporotrichosis. This case was confirmed by culture and the use of direct F.A. on paraffin imbedded tissue sections.

AFIP Diagnosis. Nephritis, pyogranolomatous, chronic, multifocal, severe, kidney, armadillo (Dasypus novemcinctus), Edentata, etiology, Sporothrix schenkii.

Contributor. Center for Disease Control, 1600 Clifton Road, N.E., Atlanta, Georgia 30333.

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Case II - 80-9625 (AFIP 1801295).

History. The tissue is from a 2-year-old female cat positive for feline leukemia virus. The cat was culled from the cattery.

Contributor's Diagnosis & Comment. Gastric nematodiasis due to Ollulanus tricuspis.

Comment. In our experience, Ollulanus tricuspis is associated with the presence of many globule leukocytes and an increase in size and number of lymphoid aggregates in the gastric mucosa. In long standing cases there is also an increase in fibrous connective tissue. By size, O. tricuspis adults and larvae can be differentiated from other nematodes that may occur in the feline stomach with the exception of Aelurostrongylus abstrusus larvae. Wet mounts need to be made to differentiate A. abstrusus larvae from O. tricuspis parasites.

AFIP Diagnoses. 1) Trichostrongyle nematodes, multiple, lumen, stomach, feline, etiology, Ollulanus tricuspis. 2) Fibrosis, diffuse, minimal, lamina propria, stomach.

Conference Note. The presence of a pseudocoelomic body cavity, platymarian musculature, and evenly spaced external longitudinal cuticular ridges is indicative of a trichostrongyle. Adults were observed in some of the sections we examined. O. tricuspis, unlike other trichostrongyles, is viviparous and does not deposit eggs.The minimal fibrosis evident could not be correlated morphologically with the presence of parasites; its distribution was diffuse and in the deep lamina propria, few inflammatory cells were present, and parasites were not observed within the mucosa in the sections we examined.

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Case III - 81-001 (AFIP 1822361).

History. The tissues are from Golden Syrian hamsters. In order to obtain a sufficient number of slides, sections had to be obtained from three different animals. (4) and (11) were females which died spontaneously at about 16 months of age. (51) was a male which died at 24 months of age. All hamsters were controls in an inhalation chronic toxicity study. Pertinent gross lesions included hemothorax and/or hemopericardium in (4) and (51) but not (11).

Contributor's Diagnosis & Comment. Medial necrosis, aortic, with dissecting aneurysm.

Comment. A few sections from (11) also have the heart present. In these sections there is multifocal myocardial degeneration, subacute, to chronic, slight; and a mural thrombus.

We have noted dissecting aortic aneurysms in a low percentage of hamsters. This is slightly more frequent in females. Affected animals usually die between 1-2 years of age. The aorta is usually affected between its origin and the arch. In the sections which include the heart, the necrotic area appears to encircle the aorta just above the coronary arteries. The coronary arteries, as well as other major systemic arteries, are not typically involved.

A pathogenesis of dissecting aortic aneurysm in man has been proposed.[2] The progression appears similar in hamsters and various aspects of the development may be noted depending on the section examined. There first appears to be focal medial necrosis. In man, the delicate vasa vasorum may be disrupted with subsequent intramural hemorrhage, but we have not noted this in hamsters. In any event, the necrotic area eventually communicates with the lumen, filling the area with blood and forming an aneurysm. The aneurysm may expand, dissecting along the plane of the muscularis or into the adventitia. With prolonged survival, these channels may even become endothelialized. The aneurysm may rupture leading to hemopericardium and/or hemothorax. Although we could not find references to dissecting aneurysms in untreated hamsters, a high incidence (51 of 77) was noted for hamsters given cortisone acetate.[5]

AFIP Diagnoses. 1) Aneurysm, dissecting, secondary to medial necrosis, focally extensive, severe, ascending aorta, Golden Syrian hamster, rodent. 2) Degeneration, hyaline, multifocal, minimal to mild, media, small intramyocardial coronary arteries, heart. 3) Degeneration, subacute to chronic, multifocal, minimal, myocardium, heart.

Contributor. Toxicology Research Laboratory, The Dow Chemical Company, Midland, Michigan 48640.

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Case IV - 80F1886T24-A* 80F1886T24-B* (AFIP 1747664). (Two H&E sections included).

History. The tissue is from an 11-1/2 year female DSH feline. The animal was found dead (no previous illness). Gross lesions at postmortem included massive abdominal hemorrhage and numerous raised firm, white, nodules within the lung. A nodular thickening was noted at the mid-cervical region which involved the trachea and esophagus.

Contributor's Diagnosis. Tracheal carcinoma with renal metastasis.

AFIP Diagnoses. 1) Carcinoma, epidermoid, trachea, with metastasis to the renal vessels and kidney, domestic short hair, feline. 2) Hyperplasia, follicular, nodular, solitary, thyroid.

Conference Note. Differential diagnoses submitted were adenocarcinoma or squamous cell carcinoma of the trachea with renal metastasis, and transitional cell or renal cell carcinoma of the kidney with tracheal metastasis. The circumferential involvement of the tracheal mucosa and the presence of more differentiated cells within the tracheal mass were considered evidence that the tumor was of tracheal origin. There was general consensus that the neoplasm most likely arose from the submucosal glandular epithelium. Metastasis to the esophagus, lungs, bronchial lymph nodes, and adrenal also was reported by the contributor. Metastasis and vascular thrombosis in the renal area resulted in the acute hemorrhage noted grossly and probably were the immediate cause of death. Primary tracheal neoplasms in domestic animals are rare.

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Reference. Moulton, J. E.: Tumors in Domestic Animals. University of California Press, Berkeley, 1978, p. 216.

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Results
AFIP Wednesday Slide Conference - No. 30
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Case I - AM-7530-82 (AFIP 1822364). (Iron stain included).

History. The tissues are from an adult male Rothschilds mynah found dead in its cage without premonitory symptoms of illness. Gross necropsy examination revealed mild ascites, hepatomegaly and an empty gastrointestinal tract.

Contributor's Diagnosis & Comments. Hepatopathy associated with excessive iron storage.

Comment. Hepatocytes, Kupffer cells and macrophages in liver sections contain an abundance of iron containing Prussian blue positive, brown granules. Periportal fibrosis and chronic pericholangitis are mild; Kupffer cell and macrophage numbers are diffusely increased. Numerous renal tubular epithelial cells contain the brown pigment, however, renal inflammation is not present.

AFIP Diagnoses. 1) Hemosiderosis and Kupffer cell hyperplasia, chronic, diffuse, severe, liver, Rothschild's mynah, avian. 2) Hemosiderosis, chronic, diffuse, moderate to severe, tubular epithelium, kidney.

Conference Note. Iron storage disorders are characterized by excessive accumulation of hemosiderin in various organs. In man when this accumulation is accompanied by tissue damage including fibrosis and cirrhosis in the liver, the condition is called hemochromatosis. When damage is not evident it is called hemosiderosis. In this case a Masson trichrome stain demonstrated minimal periportal fibrosis which did not disrupt the limiting hepatocellular plate around portal areas. With the absence of significant fibrosis, hemosiderosis was considered the most accurate morphologic diagnosis. However, the relatively small amount of hemosiderin observed within the hyperplastic Kupffer cells of this case is a feature similar to primary hemochromatosis in man, in which there may be an inability by RE cells to synthesize ferritin or to accept iron from transferrin for ferritin synthesis. The resultant low serum ferritin cannot exert negative feedback on the intestinal mucosal epithelium and thus excessive absorption occurs. Additionally, in early stages of primary hemochromatosis, fibrosis may be minimal (Robbins). The exact toxic effect of chronic iron accumulation is not understood completely; however, membrane peroxidation is thought to occur in acute iron toxicity. Iron may induce susceptibility of the liver to other agents and also enhance collagen formation secondary to injury (Lowenstine et al.). Hemochromatosis or parenchymal hemosiderosis rarely is reported in nonhuman mammals. Although avian iron metabolism is similar to that of mammals, mynahs and many other species of birds typically store histochemically detectable iron in parenchymal hepatocytes and other organs (Lowenstine et al.). Although the hepatopathy associated with mynah birds as described by Lowenstine et al. and by Randell et al. may represent hemochromatosis, the authors comment that hemosiderosis superimposed upon other conditions cannot be excluded completely.

Contributor. Animal Disease Diagnostic Laboratory, Purdue University, West Lafayette, Indiana 47907.

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3. Robbins, S. L., and Cotran, R. S.: The Pathologic Basis of Disease. W. B. Saunders, Philadelphia, 1979, pp 343-352.
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Case II - Blocks HPS 3754,3755,3758 (AFIP 1824529).

History. The tissues are from a mouse inoculated with cultured infectious material from a 35-year-old male U.S. Armed Forces member stationed in Germany.

Laboratory Results. A radiographically apparent lung nodule was surgically removed. On fungal culture of the nodule, an organism with arthrospores was isolated. Six weanling white mice were inoculated IP with the spores. All mice became depressed and anorectic and died or were killed 14 days PI. At necropsy, the peritoneal cavity and lungs contained disseminated, minute, grey-yellow foci.

Contributor's Diagnosis & Comment. Pyogranulomas, disseminated, severe, lungs, lymph nodes, abdominal organs, mice, etiology compatible with Coccidoides immitis.

Comment. The mycotic organism with characteristic arthrospores was tentatively identified as Coccidioides immitis. The diagnosis was confirmed by finding the typical tissue phase on histopathologic examination of mouse tissues. The clinical history of the patient includes a previous assignment in California. The patient has received systemic antifungal therapy and remains free of clinical signs.

AFIP Diagnosis. Pyogranulomas, subacute, disseminated, severe, lungs, lymph nodes, abdominal organs, mouse, rodent, etiology, Coccidioides immitis.

Contributor. 10th U. S. Army Medical Laboratory, Landstuhl, Germany.

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Case III - 80-1942-5 (AFIP 1805542).

History. The tissue is from a 3-month-old Afghan hound displaying progressive, ascending paresis. At necropsy, the major adductor muscles of both hind limbs were pale and shrunken. Multifocal, white, gritty areas were present throughout the affected muscle masses.

Contributor's Diagnosis & Comment. Severe, necrotizing, myositis due to Toxplasma gondii infection.

Comment. Other lesions in this dog included a severe meningoencephalomyelitis with numerous Toxoplasma cysts in the central nervous system. A serum antibody titer of 1:64 to T. gondii was demonstrated.

Toxoplasmosis is claimed to be the most commonly reported cause of myositis in dogs (3). A brief review of the recent literature did not substantiate this claim, although concurrent myositis and encephalomyelitis appears to be a common manifestation of canine toxoplasmosis (2,4).

Our appreciation to Drs. J. E. Cook and R. S. Rahaley, Department of Veterinary Pathology, Kansas State University, for helping with this case.

AFIP Diagnosis. Myositis, necrotizing and granulomatous, subacute, multifocal, severe, muscle, Afghan hound, canine, etiology, Toxoplasma gondii.

Contributor. Department of Toxicology, Dow Chemical Company, P. O. Box 68511, Indianapolis, Indiana 46268.

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4. Hartley, W. T., Lindsay, A. B., and Mackinnon, M. N.: Toxoplasma meningoencephalitis and myositis in a dog. New Zealand Vet. J. 6: 124, 1958.

Case IV - P80-031 (AFIP 1803517).

History. The tissues presented are from a mouse treated I.P. with 1-(N-ethylcarbamoylhydroxymethyl)-furan.

Contributor's Diagnosis & Comment. Clara cell necrosis and renal tubular necrosis secondary to the toxic intermediates of carbamoylmethyl-furan.

Comment. This case illustrates a key concept, specifically, that in which a chemical not toxic itself is metabolized to a toxic compound which injures cells with metabolizing capability. Chemicals which reproducibly induce an acute, specific pulmonary toxicity by routes of administration other than inhalation are relatively rare. It is now realized that the lung can metabolize foreign chemicals in addition to hepatocytes and renal proximal convoluted tubular epithelium. Specifically, pulmonary mixed-function oxidase activity can be localized in Clara cells. Many xenobiotics, such as furan derivatives (one cause of bovine atypical interstitial pneumonia, Boyd 1976), are now known to produce tissue injury after metabolic conversion (oxidative metabolism) to highly reactive intermediates, usually at the site of conversion, hence potentially inducing Clara cell necrosis, centrilobular hepatic necrosis, and/or renal necrosis.

AFIP Diagnoses. 1) Necrosis, bronchiolar epithelium, acute, multifocal, mild to moderate, terminal bronchioles, lung, mouse, rodent, etiology, toxic intermediate of 1-(N-ethylcarbamoylhydroxymethyl)-furan (as specified by the contributor). 2) Nephrosis, acute, diffuse, severe, epithelium, proximal convoluted tubules, kidney, etiology, same as one.

Contributor. The Procter & Gamble Company, P. O. Box 39175, Cincinnati, Ohio 45247.

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