AFIP Wednesday Slide Conference 1979-80

Index

WSC 1979-80

Conf. #	Case #	Date of Conference	Contrib. #	AFIP #	Contributor	Species	Organ	Diagnosis
1	I	19 Sep 79	2711-79	1711326	University of Nebraska	Feline	Pancreas & Liver	Diabetes mellitus with hepatic lipidosis and islet degeneration (amyloidosis)
	II	"	520-78	1668481	Oregon State Univ.	Caprine	Thymus	Thymoma
	III	"	3-104	1667962	Montana State Univ.	Porcine	Small intestine	Enteritis (Coccidiosis)
	IV	"	MK79-420	1712741	National Institutes of Health	Marmoset	Liver	Hepatitis (Hepatitis B virus)
2	I	26 Sep 79	78-0627	1667114	University of Arizona	Iguana	Skin	Dermatitis (Dermatophilosis)
	II	"	78-529	1666532	Aberdeen Proving Grounds	Equine	Skin	Sarcoid
	III	"	78-127	1661469	National Animal Disease Center	Ovine	Nasal cavity	Enzootic nasal adenocarcinoma
	IV	"	79-1494	1712546	Syntex Research Inc.	Feline	Skin	Dermatitis (Sporotrichosis)

3	I	3 Oct 79	9981-79B	1712507	University of Missouri	Feline	Kidney	Membranous glomerulo-nephritis
	II	"	L77-2128	1667340	Pig Research Institute of Taiwan	Porcine	Kidney	Congenital renal cortical dysplasia (fibrosis)
	III	"	77-12	1619654	British Columbia Provincial Vet. Laboratory	Chicken	Kidney	Tubular nephrosis (Infectious bronchitis virus)
	IV	"	220	1666307	University of Missouri	Chicken	Kidney & muscle	Lymphosarcoma (Marek's disease herpesvirus)
4	I	10 Oct 79	79-1274B	1712746	University of Georgia	Porcine	Lung	Pneumonitis (Nocardiosis)
	II	"	10030	1667473	Oklahoma State Univ.	Bovine	Lung	Bronchopneumonia (Brucellosis)
	III	"	18863	1711330	Livestock Disease Diag. Ctr. Lexington, KY	Equine	Kidney	Nephritis (Actinobacillosis)
	IV	"	Y2174-2	1623576	Ohio State Univ.	Equine	Lung	Pneumonia (Corynebacterium equi)
5	I	17 Oct 79	78-844	1668451	South Dakota State University	Ovine	Brain	Encephalitis (Listeriosis)
	II	"	Y5911	1667111	Ohio State Univ.	Canine	Brain	Telangiectasis
	III	"	69249	1668144	Texas A&M	Canine	Spinal cord	Afghan hereditary myelomalacia
	IV	"	119	1712509	Upjohn Company	Rat	Brain	Granular cell Schwannoma

6	I	24 Oct 79	78D532	1693726	Maryland Dept. Agriculture	Bovine	Liver	Hepatitis (Candidiasis)
	II	"	(None)	1682621	Dept. of Vet. Path. AFIP	Goldfish	Body, Cross Section	Granulomas (Amoebiasis)
	III	"	79P446	1714169	Colorado St. Univ.	Canine	Brain	Meningitis (Cryptococcosis)
	IV	"	78-527	1667963	Center for Disease Control	Guinea pig	Colon	Colitis (Amoebiasis)
7	I	31 Oct 79	79M1831C	1710525	U.S. Environmental Protection Agency	Mouse	Colon	Transmissible colonic hyperplasia (Citrobacter freundii)
	II	"	M6187/335	1666182	Lilly Research Laboratories	Macaque	Brain	Fibrosis & cavitation
	III	"	28449N	1714165	Auburn University	Porcine	Ileum	Hyperplastic ileitis (Campylobacter sputorum subsp. mucosalis)
	IV	"	79-145	1713079	Naval Medical Res. Inst.	Mouse	Small intestine	Enteropathy (Hexamitiasis)
8	I	7 Nov 79	CP79-594	1716396	Southwestern Med. School	Canine	Brain	Encephalitis (Encephalitozoonosis)
	II	"	A31205	1714167	Pfizer, Inc.	Feline	Liver & Spleen	Myeloproliferative disease compatible with erythremic myelosis
	III	"	79-230-11	1714920	National Zoo	Wallaby	Lung & lymph node	Pneumonitis & lymphadenitis (Toxoplasmosis)
	IV	"	19176	1716402	Montana St. Univ.	Caprine	Liver & kidney	Cholangitis & Glomerulonephritis (Sarcocystosis)

9		14 Nov 79	78-602B	1667074	Vet. Diag. Lab. Tifton, GA	Equine	Brain	Meningoenceph itic arteritis (Equine rhino-pneumonitis virus)
	II	"	J3816	1664173	Univ. of Nebraska	Bovine	Brain & eye	Arteritis, Uveitis and Keratitis (Malignant catarrhal fever)
	III	"	MDA-Anim. Hlth. Lab.	1713318	Maryland Dept. of Agriculture	Parrot	Liver & kidney	Hepatitis and tubular nephrosis (Chlamydiosis)
	IV	"	7263	1712508	Upjohn Company	Bovine	Lung	Bronchopneumonia (Bovine Respiratory disease complex-Shipping fever)
10	I	28 Nov 79	A77-130	1682696	Angell Memorial Anim. Hospital	Feline	Lung	Tracheitis (Capillaria-sis)
	II	"	X390	1713323	University of Alabama	Canine	Lung	Tracheobronchitis (Filaroides osleri)
	III	"	28845-1	1713319	Letterman Army Inst. of Research	Canine	Scrotum	Hemangioma-like lesion of canine scrotum
	IV	"	6451K	1711329	Univ. of Penn.	Canine	Inguinal mass	Lymphangioma
11	I	12 Dec 79	BZ133-78	1667116	Anim. Medical Center	Touraco (bird)	Bone & Muscle	Osteomyelitis (Tuberculosis)
	II	"	77-3161	1712659	South Dakota State University	Porcine	Liver	Hepatitis (Tuberculosis)
	III	"	78D501	1693934	Maryland Dept. of Agriculture	Bovine	Intestine	Enterocolitis (Johne's disease)
	IV	"	78S156	1664302	Milton S. Hershey Medical Center	Armadillo	Tongue	Sarcocystosis

12	I	19 Dec 79	79-0616	1712578	Univ. of Arizona	Feline	Spleen	Mast cell tumor
	II	"	CP79-894	1716395	Southwestern Med. School	Canine	Spleen	Plasmacytosis (Ehrlichiosis)
	III	"	78-257	1711328	Brooks AFB	Macaque	Spleen	Splenitis (Microfilariasis)
	IV	"	SWRI Case I	1718122	Sterling-Winthrop Research Inst.	Rat	Heart	Necrosis (Tyzzer's disease)
13	I	9 Jan 80	66983	1666470	Bureau of Biologics, FDA	Chimp.	Colon	Enteritis (Strongyloidiasis)
	II	"	79-4692	1718554	Univ. of Illinois	Rabbit	Brain	Meningoencephalitis (Baylisascaris procyonis larvae)
	III	"	A77-342	1623521	Brooks AFB	Feline	Pancreas	Pancreatitis (Eurytrema procyonis)
	IV	"	79-364	1718322	Tuskegee Institute	Porcine	Ureter	Periureteritis (Stephanurus dentatus)
14	I	16 Jan 80	(None)	1719055	Eastern Fish Dis. Laboratory	Trout	Body, Cross Section	Epidermal hyperplasia (Ichthyophthirius multifiliis)
	II	"	(None)	1719054	Western Fish Dis. Laboratory	Salmon	Kidney	Granulomas (metacercaria of Nanophyetus salmincola)
	III	"	77-34	1666534	Pfizer, Inc.	Menhaden (fish)	Testis	Degeneration and fibrosis (Eimeria brevoortiana)
	IV	"	390-79	1689415	Oregon St. Univ.	Camel	Liver	Osteolipomatous metaplasia (Possible hypervitaminosis A)

15	I	23 Jan 80	78-257	1662196	Univ. of Penn.	Bovine	Mammary gland	Granulomas (T arculosis)
	II	"	77-2545-17	1633432	Kansas St. Univ.	Bovine	Mammary gland	Mastitis
	III	"	76816	1668874	Los Angeles County Dept. of Hlth. Services	Canine	Mammary gland	Carcinosarcoma
	IV	"	A19977	1714921	Animal Med. Center	Feline	Thymus	Adenocarcinoma (Possibly thymic carcinoid)
16	I	30 Jan 80	79-226	1714911	Univ. of Penn.	Porcine	Uterus	Endometritis (Staphylococcus aureus)
	II	"	PCSD-28097	1669223	Letterman Army Inst. of Research	Canine	Uterus & Ovary	Granulosa cell tumor with cystic endometrial hyperplasia
	III	"	69-457	1700620	Bio-Dynamics, Inc.	Macaque	Vaginal polyp	Endometriosis
	IV	"	76-326	1713950	Natl. Center for Toxicologic Research	Mouse	Ovary	Teratoma
17	I	6 Feb 80	C78-167	1718315	Univ. of Florida	Equine	Skin	Dermatitis with pseudo-epitheliomatous hyper-plasia
	II	"	79/700	1718319	Dept. of Agriculture, Wollongbar, New South Wales, Australia	Porcine	Skin	Dermatitis, vasculitis and parasitic folliculitis (Swine poxvirus, Salmonella typhimurium and Demodex sp.)
	III	"	78-897	1713991	Aberdeen Proving Grounds	Feline	Lip	Feline eosinophilic granuloma complex ("Rodent ulcer")
	IV	"	S79-1121	1713992	Angell Mem. Anim. Hospital	Feline	Skin	Plasmacytoma

18	-	13 Feb 80	18650	1667961	Montana State Univ.	Bear	Mandible	Odontogenic myxofibroma
	II	"	78-291	1666499	National Zoo	Bear	Small intestine	Choledochal adenocarcinoma
	III	"	79-94-3	1714919	National Zoo	Duiker (antelope)	Muscle	Rhabdomyolysis (Capture myopathy)
	IV	"	RA7-6043	1668788	Univ. of Georgia	Coyote	Liver	Hepatitis (Capillaria hepatica)
19	I	20 Feb 80	(None)	1642713	Univ. of Illinois	Canine	Eye	Retinitis (Canine distemper)
	II	"	28992	1693566	Letterman Army Inst. of Research	Rat	Ear	Carcinoma of Zymbol's gland
	III	"	(None)	1599982	Penn-Daw Anim. Hosp.	Canine	Eye	Adenocarcinoma of ciliary body
	IV	"	(None)	1581246	Univ. of Kentucky	Equine	Eye	Malignant medulloepithelioma
20	I	5 Mar 80	N78-384	1667920	School of Aerospace Med., Brooks AFB	Mouse	Heart	Epicardial mineralization
	II	"	C79-02	1712506	Eastman Kodak Co.	Rabbit	Heart	Epicarditis and myocarditis (Staphylococcus aureus)
	III	"	75-1956	1665272	Dow Chemical Co.	Rabbit	Heart	Necrosis (Adriamycin intoxication)
	IV	"	SW76-1095	1620680	Natl. Institutes of Health	Porcine	Heart	Necrosis (Vitamin E & Selenium deficiency)

21	I	12 Mar 80	28063	1669175	Letterman Army Inst. of Research	Chicken	Brain	Encephalomala (Vitamin E & Selenium deficiency)
	II	"	D70-5647	1718843	Univ. of Illinois	Caprine	Spinal cord	Meningomyelitis (Leuko-encephalomyelitis virus of goats)
	III	"	78-7585	1714908	Quebec Dept. of Agr.	Bovine	Spinal cord	Myelinodystrophy (Progressive ataxia of Charolais)
	IV	"	77-17001	1656673	Rollins Animal Dis. Diagnostic Laboratory	Canine	Spinal cord	Meningeal hemorrhage (Intervertebral disc prolapse)
22	I	26 Mar 80	6277	1667566	Murray St. Univ.	Canine	Brain	Meningoencephalitis (Histoplasmosis)
	II	"	79-153	1711327	Univ. of Penn.	Canine	Brain	Canine meningoencephalo-myelitis syndrome
	III	"	J1765	1664174	Univ. of Nebraska	Canine	Brain	Meningitis (Cryptococ-cosis)
	IV	"	78353-13	1716390	Natl. Anim. Dis. Ctr.	Ovine	Brain	Chorioencephalitis (Progressive pneumonia virus of sheep)
23	I	2 Apr 80	79-530	1714170	Univ. of Tennessee	Equine	Colon	Colitis (Corynebacte-rium equi)
	II	"	11587-17	1619768	Johns Hopkins Univ.	Guinea pig	Lymph node	Lymphadenitis (Freund's adjuvant injection)
	III	"	79-1495	1712547	Syntex Research Inc.	Rat	Subcutis	Panniculitis (Injection of aluminum stearate in sesame oil)
	IV	"	"AFIP 2"	1716397	Washington St. Univ.	Equine	Liver	Hepatitis & Protozoiasis (Graft versus host reaction; unidentified protozoa

24	I	9 Apr 80	A16838	1714914	Anim. Med. Center	Canine	Liver	[illegible] s of (Copper toxic Bedlington terriers)
	II	"	77/670	1718318	Dept. of Agriculture, Wagga Wagga, NSW, Australia	Ovine	Liver	Necrosis (Copper intoxication and pyrrolizidine plant poisoning)
	III	"	71953	1712662	Texas A&M	Feline	Liver	Hemosiderosis and hepatic atrophy (Portacaval shunt syndrome)
	IV	"	2-213	1716401	Montana State Univ.	Equine	Liver	Cholangiohepatitis and biliary hyperplasia (Possible salmonellosis with trifoliosis intoxication)
25	I	16 Apr 80	H79-378	1712747	Pig Research Inst. of Taiwan	Porcine	Placenta	Placentitis (Pseudorabies)
	II	"	79-119	1714917	Univ. of Penn.	Equine	Intestine	Enteritis & vasculitis (Rhinopneumonitis herpesvirus)
	III	"	H1145	1618990	Univ. of Nebraska	Porcine	Nasal turbinates	Rhinitis (Cytomegalovirus)
	IV	"	A79-170	1713946	Angell Mem. Anim. Hosp.	Myna bird	Liver	Hepatitis (Lankesterellosis)
26	I	23 Apr 80	79-1495	1712938	Syntex Research Inc.	Coyote	Intestine	Enteritis (Canine Parvovirus)
	II	"	79-1	1710129	Armed Forces Radiobiological Res. Inst.	Chicken	Heart	Myocarditis (Avian parvovirus)
	III	"	80A-956-4	1713372	Univ. of Georgia	Canine	Heart	Myocarditis (Canine parvovirus)
	IV	"	78-3679	1713320	Ohio State Univ.	Feline	Liver	Hepatitis (Feline parvovirus)

27	-	30 Apr 80	7187	1639438	Univ. of Alabama	Rat	Mammary gland	Anaplastic carcinoma
	II	"	(None)	1504164	Registry of Vet. Pathology, AFIP	Canine	Eye	Malignant melanoma
	III	"	(None)	1717347	Biomedical Ref. Laboratories, Inc.	Canine	Eye	Teratoid medulloepithelioma
	IV	"	(None)	1582044	Fitzsimmons Army Med. Ctr.	Canine	Eye	Multiple hereditary ocular anomalies
28	I	14 May 80	(None)	1737202	Division of Zoonotic Dis. Pathology, AFIP	Ovine	Liver	Hepatitis (Wesselsbron virus)
	II	"	(None)	1737276	Division of Zoonotic Dis. Pathology, AFIP	Bovine	Lung	Bronchopneumonia & Emphysema (Ephemeral fever rhabdovirus)
	III	"	X11841	1727726	Greenfield Laboratories	Siberian polecat	Skin	Granulomas (Hepatozoon mustelis)
	IV	"	77-2653 77-2785	1713327	Walter Reed Army Inst. of Research	Mouse	Brain	Choriomeninoencephalitis (Trypanosoma rhodesiense)
29	I	21 May 80	79-7035	1701507	Kansas State Univ.	Canine	Skull	Chondroma rodens
	II	"	S78-133	1664430	Naval Med. Res. Inst.	Rat	Stifle	Arthritis & Osteopetrosis (Possibly mycoplasmosis with hereditary osteopetrosis)
	III	"	7800-60	1711663	St. Louis Zoo & Monsanto Company	Deer	Antler	Fracture of antler in velvet
	IV	"	D79-442	1713328	Univ. of Georgia	Chicken	Tibia	Epiphyseal dyschondroplasia

30	I	28 May 80	C79-01	1712749	Eastman Kodak Company	Guinea pig	Lung	Pneumonia (*Pneumocystis carinii*)
	II	"	77D284	1664303	Milton S. Hershey Medical Center	Macaque	Lung	Bronchiolitis & Bronchiectasis (*Pneumonyssus semicola*)
	III	"	79N943	1727247	Univ. of California, Davis	Porcine	Skin	Dermatitis (*Sarcoptes scabiei*)
	IV	"	VRL A99718	1727250	Veterinary Reference Laboratory	Canine	Peritoneum	Peritonitis (Mesocestoides)

Schedule of Moderators
AFIP Wednesday Conferences
1979-1980

Date	Conference Number	Moderator
19 Sept 79	1	Maj. Toft
26 Sept	2	LTC Stedham
3 Oct	3	Col. Casey
10 Oct	4	Maj. Langloss
17 Oct	5	Dr. Garner
24 Oct	6	Dr. Migaki
31 Oct	7	Capt. Parker
7 Nov	8	Col. Jones
14 Nov	9	LTC De Paoli
21 Nov	(Thanksgiving Break)	
28 Nov	10	Lt. Col. Imes
5 Dec	(ACVP Meeting)	
12 Dec	11	Dr. Montali
19 Dec	12	COL Hildebrandt
26 Dec	(Christmas Break)	
2 Jan 80	(New Years Break)	
9 Jan	13	Maj. Toft
16 Jan	14	Dr. Migaki
23 Jan	15	Col. Casey
30 Jan	16	Lt. Col. Imes
6 Feb	17	LTC Stedham
13 Feb	18	Dr. Montali
20 Feb	19	LTC Trucksa
27 Feb	(IAP Meeting)	
5 Mar	20	Col. Jones
12 Mar	21	Capt. Parker
19 Mar	(C. L. Davis Conf., AFIP)	
26 Mar	22	Dr. Garner
2 Apr	23	MAJ Hall (T)
9 Apr	24	COL Hildebrandt
16 Apr	25	LTC De Paoli
23 Apr	26	Maj. Langloss
30 Apr	27	Col. Fetter (T)
7 May	(Comparative Pathology Course, AFIP)	
14 May	28	LTC Trucksa
21 May	29	Dr. Montgomery (T)
28 May	30	LTC Mellick (T)

(T) = Tentatively Designated Moderator.

Results
AFIP Wednesday Slide Conference - No. 1
19 September 1979

Case I - 2711-79 (AFIP 1711326).

History: A 6-year-old, neutered male, domestic short-nair cat presenting initially with anorexia and mild icterus. Two weeks later the cat was presented with vomiting and continuing anorexia. The animal was euthanized following serum chemistries:

Total protein	7.5	Glucose	250
Albumin	4.3	LDH	340
BUN	45	SGOT	61
Direct bilirubin	2.5	Alk Phos	310

Contributor's Diagnosis & Comments: Diabetes mellitus. Histologically, pancreatic amyloidosis and hepatic lipidosis were observed.

Attendees' Diagnoses & Comments: Morphologic Diagnoses: 1) Degeneration with amyloidosis, diffuse, moderate to severe, islet cells, pancreas; 2) Vacuolar change compatible with fatty metamorphosis, diffuse, severe, liver; 3) Cholangitis, acute, diffuse, mild to moderate, bile ducts of liver.

Etiologic Diagnosis: 1) Diabetes mellitus, 2) Amyloidosis.

Comments: Attendees were in agreement that the hyaloid material in the pancreas was morphologically compatible with amyloid. There was little evidence of inflammation in the pancreas leading the staff to consider degeneration of islet cells as the most likely cause of the diabetes mellitus. Disturbances in nutrient metabolism, initially with insulin-mediated carbohydrate utilization, leads to compensatory alterations in lipid metabolism, exemplified by the vacuolar changes in the liver. Protein catabolism, dehydration, diabetic glomerulitis and altered hepatocyte function were discussed as possible causes for the abnormal serum chemistries. An etiology for the cholangitis was discussed; relationship to the diabetes mellitus or normal aging processes were not considered likely for the bile duct lesion. In addition, lenticular, retinal, glomerular and vascular changes seen in man and dogs were compared to the findings in cats.

Contributor: Veterinary Sciences Department, University of Nebraska (East Campus), Lincoln, NE 68508.

References:

1. Glenner, G.C.: Amyloid, amyloidosis and amyloidogenesis. Int Rev Exp Path 15: 1-92, 1976.
2. Feldman, E.C.: Diabetes mellitus. In: Current Veterinary Therapy, 6th ed., Kirk, R.W., (ed), W. B. Saunders Co., Philadelphia, 1977, pp 1001-1009.
3. Schaer, M.: Diabetes mellitus in the cat. J. Am. Anim. Hosp. Assoc. 9: 548, 1973.

4. Johnson, K. H., Osborne, C. A., et al.: Intracellular substance with some amyloid staining affinities in pancreatic acinar cells of a cat with amyloidosis. Path. Vet. 7: 153, 1970.
5. Finn, J.P., Martin, C.L., et al.: Feline pancreatic islet cell hyalinosis associated with diabetes mellitus and lowered serum-insulin concentrations. J. Sm. Anim. Pract. 11: 607, 1970.
6. Gepts, W. and Toussaint, D.: Spontaneous diabetes in dogs and cats. A pathological study. Diabetologia 3: 249, 1967.

Case II - 0520-78 (AFIP 1668481).

History: Incidental finding in a 10-year-old female Saanen goat.

Contributor's Diagnosis & Comments: Thymoma.

Attendees' Diagnosis & Comments:

Diagnosis: Thymoma, involuted thymus, goat.

Comments: Epithelioid cells were considered to be the neoplastic cell in this tumor. There was a strong compliment of small lymphocytes which the staff considered normal. The frequent occurrence of this neoplasm in goats and sheep (30% of all neoplasms in goats examined at one abbatoir were reported to be thymomas in one study) prompted comments from the staff about etiology of this neoplasm. The lack of reports of metastases and malignancies of this tumor in the ovine and caprine species prompted staff members to speculate about the appropriateness of diagnoses such as nodular hyperplasia or adenomatous hyperplasia of epithelial-derived thymic parenchyma. The cystic spaces in this caprine thymus were compared to the cysts found in involuted rodent and canine thymuses.

Contributor: Department of Veterinary Medicine, Oregon State University, Corvallis, OR 97331.

References:

1. Hadlow, W. J.: High prevalence of thymoma in the dairy goat. Vet. Path. 15: 153-169, 1978.
2. Parker, G. A., Casey, H. W.: Thymomas in domestic animals. Vet. Path. 13: 353-364, 1976.

Case III - 3-104 (AFIP 1667962).

History: Tissues are from a male 15-day-old Hampshire cross pig with a history of diarrhea which developed at approximately 8 days of age. This pig is one of 20 similarly affected animals.

Laboratory Results: Cultures of gut content and virus isolation attempts were negative for known pathogens.

Contributor's Diagnosis & Comments: Morphologic Diagnosis: Enteritis, necrotizing, subacute, disseminated, moderate, with marked villous atrophy. Etiologic Diagnosis: Swine coccidiosis. The intracytoplasmic bodies in the superficial mucosal epithelium were considered to be coccidia merozoites.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: 1) Enteritis with villus atropny, subacute to chronic, diffuse, severe, small intestine; 2) Lymphoid necrosis, multifocal, mild to moderate, Peyer's patches, small intestine.

Etiologic Diagnosis: Coccidiosis.

Comments: Conference attendees agreed on the morphologic diagnoses, but were split in their opinions as to likely etiology. Many attributed the intestinal changes to a viral patnogen (coronavirus and rotavirus were most often mentioned) and considered the coccidia to be incidental. A few participants considered the enteritis to be compatible with changes induced by acute massive coccidial infestation alone. In this case of acute fatal swine coccidiosis, the remarkable absence of intestinal hemorrnage, so frequently reported with fatal coccidial infections of cattle (with Eimeria zurnii) and chickens (with E. tenella and E. necatrix), led many participants to consider a concurrent viral infection as contributory to the mucosal and lymphoid tissue changes.

Contributor: Montana State Veterinary Diagnostic Laboratory, P. O. Box 997, Bozeman, MT 59715.

References:

1. Bergland, M. E.: Necrotic enteritis in nursing piglets. Proc. 20th Annual Meeting Am. Assoc. Vet. Lab. Diagnosticians (1977): 151-158.
2. Sangster, L. T., Stuart, B. P., et al.: Coccidiosis associated witn scours in baby pigs. Vet. Med. Sm. Anim. Clin. 73: 1317-1319, 1978.
3. Veterling, J. M.: Coccidia (Protozoa: Eimeriidae) of swine. J. Parasitol. 51: 897-912, 1965.

Case IV - Mk79-420 (AFIP 1712741)

History: Sanguinus mystax was inoculated with 0.5 ml of a suspension of marmoset liver that had been infected with human hepatitis type A and passaged twice in marmosets. The animal became moribund and was exanguinated 68 days post-inoculation.

Laboratory Results: SGPT was elevated 40 days post-inoculation.

Contributor's Diagnosis & Comments: Cholangiohepatitis, subacute, diffuse, with necrosis of individual hepatocytes and multinucleated giant cell formation. There is disseminated mononuclear reaction that is for the most part confined to the periportal areas. There is diffuse swelling and granular degeneration of the hepatocytes with necrosis and loss of individual hepatocytes. There are numerous binucleate and trinucleate hepatocytes with an occasional cell containing multiple nuclei. Most bile canaliculi contain accumulated bile. This animal in addition to the hepatitis had a purulent peritonitis, the origin of which was undetermined but appeared to be due to a perforation in the colon. The histology is compatible with the changes described for human hepatitis A.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Hepatitis, subacute, diffuse, mild to moderate, liver. 2) Peritonitis, pyogranulomatous, moderate, liver. 3) Microfilariasis, sinusoidal and vascular, mild to moderate, liver.

Etiologic Diagnoses: 1) Human hepatitis A virus for hepatitis, 2) Miscellaneous enteric bacteria for peritonitis, 3) Dipetalonema sp and/or Tetrapetalonema sp for microfilariasis.

Comments: Three distinct morphologic and etiologic diagnoses were made by most participants. The peritonitis was most often attributed to an intraperitoneal injection or rupture of the gut as frequently occurs with acanthocephalid (Prosthenorchis sp) infestations. Several participants reported that microfilariasis is common in wild-caught marmosets; hepatic changes, if any, that could be attributed to circulating microfilaria were briefly discussed by attendees. Staff members reported that periportal infiltrates of inflammatory cells are often seen in marmosets, and their etiology is unknown. Hepatic changes, such as foci of degeneration, and portal tract inflammation were considered consistent with the changes reported for viral hepatitis A infection of marmosets.

Contributor: Comparative Pathology Section, Veterinary Research Branch, NIH, Bethesda, MD 20205.

References:

1. Shao-Nan Huang, L. D., et al.: Electron and immunoelectron microscopic study on liver tissues of marmosets infected with hepatitis A virus. Lab. Invest. 41: 63-71, 1979.
2. Deinhardt, F., Holmes, A. W. et al.: Studies on the transmission of human viral hepatitis to marmoset monkeys. I. Transmission of disease, serial passages and description of liver lesions. J. Exp. Med. 125: 673-688, 1967.
3. Chalifoux, L. V., Hunt, R.D., et al.: Filariasis in New World Monkeys: Histochemical differentiation of circulating microfilariae. Lab. Anim. Sci. 23: 211-220, 1973.
4. Moore, J. G.: Epizootic of acanthocephaliasis among primates. JAVMA 157: 699-705, 1970.
5. Porter, J.A.: Parasites of marmosets. Lab. Anim. Sci. 22: 503-506, 1972.

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Results
AFIP Wednesday Slide Conference - No. 2
26 September 1979

Case I - 78-0627 (AFIP No. 1667114)

History: A zoo in Arizona presented a live but moribund adult female iguana (Ctenosaura sp.) for diagnosis of a chronic skin disease. No history was provided. The reptile's skin was covered with irregularly outlined, dull tan elevations. All regions of the body were affected. There was a 1 cm diameter ulcer on the sternum.

Laboratory Results: Gram stained imprints and lactophenol blue stained wet mounts of skin lumps revealed no organisms, but the sternal ulcer contained many Gram positive cocci. Organisms interpreted as streptococci were cultured from the ulcer but not from skin lumps.

Contributor's Diagnosis & Comments:

Morphologic Diagnosis: Dermatitis, chronic, focal, with focal hyperkeratosis. Etiologic Diagnosis: Dermatophilus congolensis.

Comments: Failure to stain and culture the organism from skin lumps was probably due to inoculating from the subcutaneous surface of lesions rather than from the hyperkeratotic epidermal surface. Whether the streptococci cultured from the ulcer were actually D. congolensis or not remains a question, because plates were rapidly overgrown by Proteus sp. The observations of branching, Gram-positive, transversely and longitudinally dividing chains of coccoid organisms established the diagnosis.

Attendees's Diagnosis & Comments: Most diagnoses were in accord with that of the contributor, all concurred with the etiologic agent. The submitted electron photomicrograph demonstrated both transverse and longitudinal division, a unique characteristic of this organism.

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

References:

1. Montali, R. J., Smith, E. E. et al.: Dermatophilosis in Australian beaded lizards. JAVMA 167: 553-555, 1975.
2. Roberts, D. S.: The histopathology of epidermal infection with the Actinomycete Dermatophilus congolensis. J. Path. Bact. 90: 213-216, 1965.
3. Simmons, G. C., Sullivan, N. D. et al.: Dermatophilosis in a lizard (Amphibolorus barbatus). Austr. Vet. J. 48: 465-466, 1972.

Case II - 78-529 (AFIP No. 1666532).

History: A 3 cm diameter pedunculated mass removed from the head of a 13-year-old pinto mare. The mass was first noted 9 months earlier by the owner, who requested excision because of repeated traumatization.

Contributor's Diagnosis & Comments: Equine sarcoid.

Attendees' Diagnosis & Comments: Equine sarcoid. The staff preferred a diagnosis of equine sarcoid, but agreed that a diagnosis of fibroma is reasonable. One of the key features in differentiating equine sarcoid from fibroma is the extension of rete ridges or pegs deep into the spindled component of the sarcoid. The absence of much of the epithelium immediately abutting the spindled growth in this case made this feature less useful, although there was an area of limited rete ridge extension. Also, although much of the neoplasm was of more mature fibrous tissue several less mature (more cellular) areas of spindled cells were seen which many attendees considered more compatible with sarcoid. Another feature mentioned by one author (Baker & Leyland) as characteristic of sarcoid is the parallel arrangement of fibroblasts at the edge of the epidermis with the long axis of the fibroblasts perpendicular to the basement membrane. Loss of epithelium again hindered this evaluation.

Contributor: Comparative Pathology & Surgical Branch, Biomedical Chemical Systems Laboratory, Aberdeen Proving Ground, MD 21010.

References:

1. Baker, V. R., Leyland, A.: Histological survey of tumours of the horse, with particular reference to those of the skin. Vet. Record 96: 419-422, 1975.
2. Stannard, A. A., Pulley, L. T.: Tumors of the skin and soft tissues. In: Tumors in Domestic Animals, 2nd Ed., J. E. Moulton, ed., University of California Press, Berkeley, 1978, pp 18-22.

Case III - 78-127 (AFIP No. 1661469).

History: Mass from the nasal cavity of a ewe from a flock with a low incidence of this condition.

Contributor's Diagnoses & Comments: Nasal adenocarcinoma of sheep. These tumors have been reported as epizootics in several countries. Although the tumor is apparently transmissible, the etiologic agent has not been isolated. Virions ("C-type") have been reported in tumor cells by Japanese workers.

Attendees' Diagnoses & Comments: Enzootic nasal adenocarcinoma of sheep. Attendees considered this tumor compatible with the described mucoepithelial tumor of sheep. Various sections presented different appearances to the tumor; many felt the mass had a papillary adenomatous form, with occasional regions of cystadenomatous morphology. Attendees were approximately split in designating this tumor benign or malignant, but by precedence agreed the tumor

should be designated an adenocarinoma. The morphology of nasal tumors of cattle and goats was compared to this ovine tumor. Reports in the literature linking the ovine enzootic nasal adenocarcinoma to viruses such as herpesvirus and the Visna-Maedi complex were mentioned as possible etiologies.

Contributor: National Animal Disease Center, USDA/ARS, Ames, Iowa 50010.

References:

1. Damodaran, S., Ramakrishnan, R., et al.: Neoplasms of the ethmoidal mucosa in bovines. Cheiron Tamil Nadu J Vet Sci Anim Husbandry 3: 1-7, 1974.
2. Duncan, J. R., Tyler, D. E., et al.: Enzootic nasal adenocarcinoma in sheep. JAVMA 151: 732-734, 1967.
3. Moulton, J. E.: Tumors of the alimentary tract. In: Tumors of Domestic Animals, 2nd Ed, J. E. Moulton, ed, University of California Press, Berkeley, 1978, pp 212-215.
4. Yonemichi, H., Ohgi, T., Fujimoto, J. et al.: Intranasal tumor of the ethmoid olfactory mucosa in sheep. Am. J. Vet. Res. 39: 1599-1606, 1978.
5. Young, S., Lovelace, S. A , et al.: Neoplasms of the olfactory mucous membrane of sheep. Cornell Vet. 51: 97-112, 1961.

Case IV - 79-1494 (AFIP No. 1712546).

History: A 3-year-old female DSH cat with skin lesions of 3 weeks duration located near the medial canthus of the left eye. The lesion was treated for 4 weeks with topical antibacterial, antifungal and steroid containing formulations. The cat was seen again 5 weeks after initial examination and had 5 more lesions on or about the tail, hind limbs and head.

Laboratory Results: Preliminary culture findings are compatible with cutaneous infection of Sporothrix schenckii (Sporotrichum schenckii).

Contributor's Diagnosis & Comments: Dermatitis, granulomatous, mycotic. The lesions were removed surgically and the cat remains asymptomatic.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Dermatitis, granulomatous, ulcerative, focally extensive, severe, skin, DSH, feline.

Comments: The etiologic agent most frequently mentioned by participants was mycobacterium associated with feline leprosy. Also mentioned were various deep fungal diseases such as histoplasmosis, sporotrichosis and phycomycosis. Staff members observed a few mononuclear cells with clear areas in the cytoplasm which were consistent with sporotrichosis. Special stains such as

PAS and GMS clearly demonstrated 3-8 by 1-3 microns, ovoid organisms in the inflammatory lesion. An acid fast stain failed to demonstrate organisms. The staff discussed differential features of sporotrichosis and feline leprosy. In several cases on file at the AFIP, the inflammatory cell infiltrate is virtually indistinguishable from the present case. In one case, the infiltrate in feline leprosy was more purely histiocytic.

Contributor: Syntex Research Inc., Department of Pathology, Palo Alto, California 94304.

References:

1. Kaplan, W., Ivens, M.S.: Fluorescent antibody staining of Sporotrichum schenckii in culture and clinical materials. J. Invest. Dermatol. 35: 151-159, 1960.
2. Lane, J. W., Garrison, R. G., et al.: Ultrastructural studies on the yeast-like and mycelial phases of Sporotrichum schenckii. J. Bact. 100: 1010-1019, 1969.
3. Lurie, H. I.: Histopathology of sporotrichosis. Arch. Pathol. 35: 421-437, 1963.
4. Scott, R. E., Bentinck-Smith, J., et al.: Sporotrichosis in three dogs. Cornell Vet. 64: 416-426, 1974.
5. Werner, R. E., Levine, B. G., et al.: Sporotrichosis in a cat. JAVMA 159: 407-412, 1971.

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Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 3
3 October 1979

Case I - 9981-79B (AFIP No. 1712507)

History: A 3-year-old, male, DSH cat was presented to a veterinarian because of swelling of the ventrum and legs (subcutaneous edema).

Laboratory Results: There was a 4+ proteinuria, predominantly albumin by electrophoresis. Total serum protein was 5.1 gm%. The cat was FeLV positive.

Contributor's Diagnosis & Comments: Membranous glomerulitis. At necropsy there was neither lymphoproliferative nor myeloproliferative disease in this cat. The enclosed electron photomicrograph shows this cat to have glomerular pathology indicative of immune complex glomerulitis (sub-epithelial electron dense deposits and associated changes). There is a well recognized association between lymphoproliferative neoplasms and glomerulitis in several species. However, it has been suggested that a primary manifestation of feline leukemia is immune complex glomerulitis. The evidence thus far is circumstantial.

Attendees' Diagnosis & Comments: Morphologic Diagnosis: Glomerulonephritis, membranous, diffuse, mild to moderate, kidney, DSH, feline. Etiology: Compatible with immune-complex glomerulonephritis induced by Feline Leukemia Virus infection. Comment: Attendees concur with the histopathologic and electron photomicrographic diagnoses of the contributor. Many attendees were impressed by the 4+ proteinuria and upon close examination of the electron photomicrograph concluded that effacement of the foot processes of the epithelial podocytes was also present.

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

References:

1. Cotter, S. M., Hardy, W. D. et al.: Association of feline leukemia virus with lymphosarcoma and other disorders in the cat. JAVMA 166: 449-454, 1976.
2. Krakowka, S.: Glomerulonephritis in dogs and cats. Vet. Clin. N. Amer. 8: 629-639, 1978.
3. Mackey, L.: Feline leukemia virus and its clinical effects in cats. Vet. Rec. 96: 5-11, 1975.
4. Slauson, D. O., Lewis, R. M.: Comparative pathology of glomerulonephritis in animals. Vet. Path. 16: 135-164, 1979.

Case II - L77-2128 (AFIP No. 1667340)

History: Tissue from a 5-month-old gilt from the Republic of China. This animal died suddenly, without signs.

Contributor's Diagnosis & Comments: Renal cortical hypoplasia with fibrosis. Grossly, both kidneys were symmetrically enlarged, firm and white. The cortex was narrower than normal, and lacked evidence of fetal lobulation. Microscopically, the glomeruli and tubules are reduced in number and size. There is tremendous fibrous deposition in the cortex and cortico-medullary junction. Numerous edematous foci with loose connective tissue are present in the stroma. The tubules are dilated, and some contain proteinaceous material with inflammatory components.

Attendees' Diagnosis & Comments: Morphologic Diagnosis: Fibrosis, cortical, diffuse, severe, kidney, porcine. Etiologic Diagnosis: Congenital renal cortical dysplasia.
Comments: Most attendees attributed the lesions to a congenital process. The staff expressed reservation in designating this kidney hypoplastic in as much as they were described as enlarged at necropsy. Renal dysplasia was a suggested alternate diagnosis to fibrous hypoplasia.

Contributor: Department of Veterinary Pathology, Pig Research Institute of Taiwan, Miaoli, Repuolic of China 350.

References:

1. Finco, D. R., Kurtz, H. J. et al.: Familial renal disease in Norwegian elknound dogs. JAVMA 156: 747-760, 1970.
2. Krook, L.: The pathology of renal cortical hypoplasia in the dog. Nord. Path. Vet. 2: 37-48, 1965.

Case III - 77-12 (AFIP No. 1619654)

History: A flock of 3-week-old Shaver layers were inoculated with an agent. All of the birds became lethargic and died quickly.

Laboratory Results: Five birds were submitted for necropsy; one was dead and 4 were alive. All birds were depressed and stunted. Kidneys were enlarged with marked whitish deposits in kidneys and ureters. There were no other significant lesions.

Contributor's Diagnosis & Comments: Toxic tubular nephrosis due to urate deposits. These birds were injected intratracheally with a nephrosis-producing strain of infectious bronchitis virus. The virus was recovered in tissue culture from all birds examined.

Attendees' Diagnosis & Comments: Morphologic Diagnosis: Nephrosis, tubular, acute, diffuse, moderate, with urate topni, kidney. Etiology: Compatible with avian coronavirus (Infectious bronchitis virus).
Comments: Most attendees were in accord with the contributor's diagnosis; the majority of attendees suggested avian coronavirus as the likely etiologic agent. Discussion revolved around the pathogenesis of the nephrosis, and whether the urate crystals precipitated following viral-induced destruction of renal tubules or urate crystals induced the observed toxic-like nephrosis. Staff members concurred that a diagnosis of nephritis was compatible with the observed inflammatory component in the tubules, but could not determine whether the inflammation was in reaction to the urates, the coronavirus, or both.

Contributor: British Columbia Provincial Veterinary Laboratory, Abbotsford, British Columbia.

References:
1. Heath, B. C.: Chemical pathology of nephrosis induced by an infectious bronchitis virus. Avian Dis. 14: 95-106, 1970.
2. Jones, R. C.: Nephrosis in laying chickens caused by Massachusetts-type infectious bronchitis virus. Vet. Red. 95: 319, 1974.
3. Cumming, R. B.: Infectious avian nephrosis (uremia) in Australia. Aust. Vet. J. 39: 145-147, 1963.

Case IV - 220 (AFIP No. 1666307)

History: Sections of kidney and breast muscle mass from a clinically normal 12-week-old chicken that was inoculated intraperitoneally with the Georgia isolate of Marek's disease virus 10 weeks previously.

Contributor's Diagnoses & Comments: 1) Marek's disease lymphosarcoma involving skeletal muscle. 2) Necrotizing vasculitis in renal vessels due to neoplastic cell infiltration.
Comment: The Georgia isolate of Marek's disease virus induces primarily visceral lymphosarcomas. An ovarian lymphosarcoma was noted at necropsy, and no gross or microscopic lesions were present in peripheral nerves.

Attendees' Diagnoses & Comments: Morphologic Diagnoses: 1) Lymphosarcoma, skeletal muscle, chicken. 2) Lymphosarcomatous infiltration, vascular, kidney, chicken. Etiologic Diagnosis: Marek's Disease. Etiology: Probably avian herpesvirus (Marek's disease virus).
Comments: The microslides examined during the Conference did not contain lesions with features of renal vascular necrosis, but were more typical of infiltrating immature lymphocytes. Among differential diagnoses, most participants mentioned lymphoid leucosis virus, but ruled it out primarily because it rarely affects skeletal muscle and the age of the birds.

Contributor: Department of Veterinary Patnology, College of Veterinary Medicine, University of Missouri, Columbia, MO 65201.

References:
1. Payne, L. N., Frazier, J. A. et al.: Pathogenesis of Marek's Disease. IN: International Rev. Exp. Pathol., Richter, G. W., Epstein, M. A., Eds., 1976, pp 59-154.
2. Sharma, J. M.: Immunosuppressive effects of lymphoproliferative neoplasms of chickens. Avian Dis. 23: 315-327, 1979.

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Results
AFIP Wednesday Slide Conference - No. 4
10 October 1979

Case I -79-1274B (AFIP 1712746).

History: Several late-term abortions or stillbirths were experienced in a one hundred brood-sow operation within a short period of time. Two aborted fetuses from one litter were submitted for diagnostic examination.

Laboratory Results: Nocardia asteroides was cultured from lung and stomach of both fetuses.

Contributor's Diagnosis & Comments: Nocardia abortion. Several nocardial abortion cases are seen each year in Georgia swine herds. Lesions in aborted fetuses are usually limited to an exudative bronchopneumonia. Giant cells are not always present. Fetal membranes are rarely available or are unsuitable for examination. Nocardia was cultured from the reproductive tract of the dam on one occasion.

Attendees' Diagnoses & Comments:
Morphologic Diagnosis: Pneumonitis, pyogranulomatous, diffuse, severe, fetal lung, porcine.
Etiologic Diagnosis: Nocardiosis.
Etiology: Compatible with Nocardia asteroides.
Comments: The inflammatory cell response was mixed, with a strong component of polymorphonuclear cells typical of inflammatory exudation. The Gram stain demonstrated Gram-positive bacilli in long branching chains, characteristic of Actinomycetes.

Contributor: Veterinary Diagnostic Laboratory, University of Georgia, P. O. Box 1389, Tifton, GA 31794.

Reference:
Cole, J. R., Holzinger, E. A.: Nocardia asteroides associated with swine abortion. Vet. Med. SAC 67: 496-498, 1972.

Case II - 10030 (AFIP 1667473).

History: Bovine fetus aborted at approximately 7 months gestation.

Laboratory Results: Culture of fetal organs Brucella abortus.

Contributor's Diagnosis & Comments: Bronchopneumonia.

Attendees' Diagnoses & Comments:
Morphologic Diagnosis: Bronchopneumonia, diffuse, severe, fetal lung, bovine.
Etiologic Diagnosis: Brucellosis.
Etiology: Compatible with Brucella abortus.

Comments: The attendees unanimously supported the contributor's diagnosis of brucella-induced bronchopneumonia. Keratinous squamous cells ("squames") observed in several bronchioles are a common finding in fetal lungs.

Contributor: Department of Patnology, College of Veterinary Medicine, Oklanoma State University, Stillwater, OK 74074.

References:

1. Smart, R. A.: Brucellosis species-induced abortion. Theriogenology 5: 102-106, 1976.
2. Jubb, K. V. F., Kennedy, P. C.: Patnology of Domestic Animals, 2nd Edition, Vol. I, Academic Press, New York, 1970, pp 528-530.

Case III - 18863 (AFIP 1711330).

History: Tissue from a 2-day-old foal.

Laboratory Results: Actinobacillus equuli was isolated from the lung, liver, kidney and spleen of this foal.

Contributor's Diagnosis & Comments: Embolic nephritis due to Actinobacillus equuli infection.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Nephritis, suppurative, acute, multifocal, severe, kidney, equine.

Etiologic Diagnosis: Actinobacillosis.

Etiology: Compatible with Actinobacillus equuli.

Comments: The concensus among participants was an etiologic diagnosis of Actinobacillosis but many considered the lesions to be compatible with streptococcal infection; Gram stain, demonstrated coccobacilli in glomeruli and other capillaries. Attendees and staff agreed the lesions were typical of "embolic suppurative nephritis" (3).

Contributor: Livestock Disease Diagnostic Center, Route 6, Newton Pike, Lexington, KY 40505.

References:

1. Shigeler, R. K., Kelly, A.: Foal septicemia (A case report). Vet Med. SAC 71: 1465-1468, 1976.
2. Webb, R. F., Cockram, F. A. et al.: The isolation of Actinobacillus equuli from equine abortion. Austr. Vet. J. 52: 100-1101, 1976.
3. Jubb, K. V. F., Kennedy, P. C.: Patnology of Domestic Animals, 2nd Edition, Vol. II, Academic Press, New York, 1970, pp 308-310.

Case IV - Y2174-2 (AFIP 1623576).

History: Tissue from one of several foals which had symptoms of pneumonia. This foal collapsed with severe dyspnea and died following a tracheal washing.

Laboratory Results: Corynebacterium equi and alpha hemolytic Streptococcus spp were isolated from the lung.

Contributor's Diagnosis & Comments:

Morphologic Diagnosis: Pneumonia, purulogranulomatous, multifocal.

Etiologic Diagnosis: Corynebacterium equi pneumonia. Radiography of the chest revealed multiple, well-circumscribed, radiopaque lesions throughout the lungs.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Pneumonia, purulogranulomatous, focally extensive, severe, lung.

Etiology: Compatible with Corynebacterium equi.

Comments: The necrosis and purulogranulomatous inflammation extensively effaced pulmonary architecture, typical of an abscess. This lesion is most often associated with Corynebacterium equi infection in the horse, but bacterial cultures would be necessary to eliminate other possible organisms such as Actinobacillus, Salmonella and Streptococcus.

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

References:

1. Bain, A. M.: Corynebacterium equi infections in the equine. Austr. Vet. J. 39: 116-121, 1963.

2. Cimprich, R. E., Rooney, J. R.: Corynebacterium equi enteritis in foals. Vet. Path. 14: 95-102, 1977.

3. Rooney, J. R.: Corynebacterium infections in foals. Mod. Vet. Pract. 47: 43-45, 1966.

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Results
AFIP Wednesday Slide Conference - No. 5
17 October 1979

Case I - 78-844 (AFIP # 1668451).

History: Seven ewes in a group of 500 silage-fed drylot animals became ill in a 1-week period. Affected animals wandered aimlessly, circled, had elevated temperatures (105°F), became recumbant and died. There were no gross necropsy lesions.

Laboratory Results: Listeria monocytogenes was isolated from brain tissue.

Contributor's Diagnosis & Comments:
Morphologic Diagnosis: 1) Encephalitis, suppurative, focal, severe, brain stem; 2) Meningitis, nonsuppurative, mild.
Etiologic Diagnosis: Listeriosis.

Attendees' Diagnoses & Comments:
Morphologic Diagnosis: Encephalitis, suppurative, multifocally disseminated, mild to severe, brain stem.
Etiologic Diagnosis: Listeriosis.
Etiology: Compatible with Listeria monocytogenes.
Comments: There was considerable variation in distribution and severity of the inflammatory reaction from slide to slide. Some microslides contained florid suppurative lesions with pronounced neuronal changes while others contained only small foci of mixed inflammatory cells. Most microslides contained regions of vascular changes and malacia with neuronophagia. All participants considered the signalment, species and histologic findings consistent with listeriosis.

Contributor: Department of Veterinary Science, South Dakota State University, Brookings, SD 57007.

References:
1. Borman, G., Olson, C. et al.: The trigeminal and facial nerves as pathways for infection of sheep with Listeria monocytogenes. Am. J. Vet. Res. 21: 993-1000, 1960.
2. Charlton, K. M., Garcia, M. M.: Spontaneous listeric encephalitis and neuritis in sheep: Light microscopic studies. Vet. Path. 14: 297-313, 1977.
3. Cordy, D. R., Osebold, J. W.: The neuropathogenesis of Listeria encephalomyelitis in sheep and mice. J. Inf. Dis. 104: 164-173, 1959.

Case II - Y5911 (AFIP # 1667111).

History: A 13-year-old, mixed breed, spayed female dog with a 4-month history of progressive epilepsy with numerous major seizures and multiple psychomotor seizures. Progressive personality changes were noted. Seizures were not responsive to therapy.

Laboratory Results: EEG displayed abnormality indicative of neoplasm. The CSF was normal.

Contributor's Diagnosis & Comments: Cerebral angiomatosis. Grossly, the cerebral cortex contained multiple red foci (1-10 mm diameter). Lesions did not protrude above the surface of the brain and were essentially spherical. They were located in the frontal and occipital lobes of the cerebrum.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Telangiectasis, capillary, focal, cerebral cortex.

Comment: The staff considered the endothelial cells to be within normal limits for capillaries. The lack of supporting mesenchymal elements around the vessels and the normal appearance of the neuropil adjacent to the prominent capillaries were helpful features in arriving at the diagnosis. Thorough examination of the entire central nervous system would be necessary to rule out the possibility of other concurrent problems in this animal.

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

References:

1. Dorn, A. S.: Radiographic interpretation of the abnormal canine cerebral angiogram. J. Am. Anim. Hosp. Assoc. 11: 484-490, 1975.

2. Robbins, S. L.: Pathologic Basis of Disease, W. B. Saunders Co., Philadelphia, 1974, p. 1514.

3. Rubenstein, L. J.: Tumors of the Central Nervous System. Atlas of Tumor Pathology, Fascicle 6, AFIP, 1972, pp 241-244, 246-256.

Case III - 69249 (AFIP # 1668144).

History: A 7-month-old female Afghan developed rear limb ataxia which progressed in a period of 2 weeks to complete posterior paresis with urinary and bowel incontinence. The dog died following a short period of respiratory difficulty.

Contributor's Diagnosis & Comments: Familial myelomalacia in Afghan hounds. Non-inflammatory myelomalacia is prominent in the ventral spinal columns. These lesions are most extensive in the thoracic segment of the cord. Sections are labeled "C", "T" and "L" for cervical, thoracic and lumbar sections, respectively.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Myelomalacia, diffuse, lateral and ventral funiculi, thoracic spinal cord.

Etiologic Diagnosis: Herido-familial myelopathy of Afghan hounds.

Comments: The staff was impressed by the selective vulnerability of the thoracic cord. The similarities to vascular-induced insults and vitamin B_{12} deficiency in other animals was discussed. The staff considered diagnoses of degenerative myelopathy and myelinolysis to be compatible with the lesions observed on H&E stained microslides.

Contributor: Department of Veterinary Pathology, Texas A&M University, College Station, TX 77843.

References:
1. Averill, D. R., Bronson, R. T.: Inherited necrotizing myelopathy of Afghan hounds. J. Neuropath. Exp. Neurol. 36: 734-747, 1977.
2. Cockrell, B. Y., Herigstad, R. R. et al.: Myelomalacia in Afghan hounds. JAVMA 162: 362-365, 1973.
3. Cummings, J. F., de LaHunta, A.: Hereditary myelopathy of Afghan hounds, a myelinolytic disease. Acta Neuropath. (Berl) 42: 173-181, 1978.
Done, J. T.: Developmental disorders of the nervous system in animals. Adv. Vet. Sci. Compar. Med. 21: 69-114, 1977.

Case IV - 119 (AFIP # 1712509).

History: Tissue from a lesion that was an incidental finding in a Sprague-Dawley rat that was a control animal in a carcinogenicity study. No clinical abnormalities were apparent.

Laboratory Results: Hematology and clinical biochemistry: All values were within normal ranges.

Contributor's Diagnosis & Comments: Schwannoma, granular cell variant. Neuropil artifacts are due to prolonged formalin storage.

Attendees' Diagnoses & Comments:
Morphologic Diagnosis: Granular cell tumor, cerebral cortex.
Comments: There was variation from slide to slide in the prominence of granules within cells; cells with granules tended to be most prominent at the margins of the tumor. The apparent increase in granular cell tumors in recent years in rats and dogs was noted by senior staff members. A Schwann cell origin is possible for granular cell tumors originating near innervated vessels of the leptomeninges.

Contributor: Pathology & Toxicology Research, Upjohn Company, Kalamazoo, MI.

References:
1. Burek, J. D.: Pathology of Aging Rats, CRC Press, West Palm Beach, FL, 1978, pp 145-148.
2. Derman, J. J., Rice, J. M. et al.: Granular cell variants in a rat schwannoma, evidence of neurogenic origin of granular cell tumor (myoblastoma). Vet. Path. 15: 725-731, 1978.
3. Hollander, C. F., Burek, J. D. et al.: Granular cell tumors of the central nervous system of rats. Arch. Pathol. 100: 445-447, 1976.
4. Mennel, H. D., Zulch, E.: Tumors of the central and peripheral nervous systems. In: Patholoy of Tumors in Laboratory Animals, Vol. I. - Tumors of the Rat, Part 2. Turosov, V. S., ed. WHO: IARC Sci. Pub. No. 6: 295-313, 1976.

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Results
AFIP Wednesday Slide Conference No. 6
24 October 1979

Case I - 78D532 (AFIP 1693726)

History: Tissues from one of two 4-month-old calves which had been purchased at a local auction about 4 weeks earlier. Clinical signs included anorexia and pyrexia.

Laboratory Results: Candida albicans was isolated from the gut, liver and urinary bladder. The isolates were sensitive to Nystatin. No other pathogens were isolated.

Contributor's Diagnosis & Comments: Systemic mycosis - Candida albicans. Postmortem lesions were generalized mycotic infection involving the liver and other organs of the digestive system, lymph nodes, urinary bladder and kidneys. The liver showed numerous discolored, circular, raised nodules. Special stains demonstrated the typical mass of tangled hyphae.

Attendees' Diagnoses & Comments: Hepatitis, necrotizing, acute to subacute, multifocal, moderate, with thromboses, liver.
Etiologic Diagnosis: Hepatic candidiasis.
Etiology: Compatible with Candida albicans.
Comments: Most participants were able to detect yeast forms and mycelia. Attendees remarked on the mild inflammatory reaction to the yeast and the coagulative necrosis, and considered this typical of systemic Candida infections. Participants mentioned the association of systemic candidiasis with ulcerative rumenitis and the over-zealous use of antibiotics, especially tetracyclines.

Contributor: Animal Health Laboratory, Maryland Department of Agriculture, Centreville, MD 21617.

References:

1. Cross, R. F., Moorhead, P. D. et al.: Candida albicans infection of the forestomachs of a calf. JAVMA 157: 1325-1330, 1970.
2. Mills, J. H. L., Hirth, R. S.: Systemic candidiasis in calves on prolonged antibiotic therapy. JAVMA 150: 862-870, 1967.
3. Seelig, M. S.: Mechanisms by which antibiotics increase the incidence and severity of candidiasis and alter the immunological defenses. Bact. Rev. 30: 442-459, 1965.

Case II - AFIP 1682621.

History: A 10-year-old goldfish from a privately owned aquarium had been sick for 2 weeks showing signs of abdominal distention and partial anorexia. The fish was constipated 3 days prior to death. (H&E and PAS stained sections are presented).

Contributor's Diagnosis & Comments: Granulomas, multiple, severe, generalized in abdominal organs and mesentery, goldfish, piscine; Etiology: organisms morphologically compatible with amoebae.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: 1) Granulomas, multifocally disseminated, severe, generalized in multiple abdominal organs, goldfish, piscine. 2) Enteritis, ulcerative and necrotizing, acute, multifocal, mild to moderate, intestine.

Etiologic Diagnosis: Generalized granulomatous amoebiasis.

Etiology: Compatible with Hartmanellidae.

Comment: Participants were unanimous in their morphologic and etiologic diagnoses. Minute amoeba were most easily seen at the margins of the granulomas and among the epithelioid macrophages; organisms were usually 2-4 microns in diameter and stained PAS positive.

Contributor: Department of Veterinary Pathology, AFIP, Wash, DC 20306.

References:

1. Noble, E. R., Noble, G. A.: Amebic parasites of fishes. J. Protozool. 13: 478-480, 1966.

2. Voelker, F. A., Anver, M. R. et al.: Amoebiasis in goldfish. Vet. Path. 14: 247-255, 1977.

Case III - 79P446 (AFIP 1714169).

History: A 2-year-old spayed female Labrador retriever had been showing signs of dementia, tetraplegia and severe weight loss over a 3-month period. Antemortem clinical pathological findings included:

Hemogram	
WBC	9,700/mm^3
Differential	
Neutrophils	82%
Lymphocytes	12%
Monocytes	3%
Eosinophils	3%

Cerebro-spinal Fluid	
Pandy	2+
Pressure	220mmHG
Cytology	Numerous neutrophils, lymphocytes, monocytes

Contributor's Diagnosis & Comments: Cryptococcal meningitis. The thick mucinous capsule around the organisms, that is responsible for the "soap bubble" appearance of the lesion was demonstrated with mucicarmine stain. The meningitis was present over the entire brain and proximal cervical spinal cord. It was most severe over the right olfactory lobe where there was also hemorrhage, malacia and inflammation in the subjacent cortex. Exudative lesions containing the organims were found on the nasal surface of the cribriform plate suggesting this was the route of entry. Disseminated foci of cryptococcal pneumonia were also present.

Attendees' Diagnoses & Comments:
Morphologic Diagnosis: Meningitis, granulomatous, diffuse, severe, meninges of cerebral cortex.
Etiologic Diagnosis: Cryptococcal meningitis.
Etiology: Cryptococcus neoformans.
Comments: Attendees unanimously agreed with the contributor's diagnosis. The clinical pathological data showed changes of significance in the CSF; the fluid protein (Pandy) was mildly elevated, and the distribution and numbers of inflammatory cells in the fluid were indicative of an infectious or necrotizing process. The units for the CSF pressure should most likely have been expressed as mm of water rather than mm of mercury; regardless, the pressure was elevated.

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

References:
1. Campbell, C. K., Naylor, D. C. et al.: Cryptococcosis in a cat. Vet. Rec. 87: 406-409, 1970.
2. Griner, L. A., Walch, H. A.: Cryptococcosis in Columbiformes at the San Diego Zoo. J. Wildlife Dis. 14: 389-394, 1978.
3. Wagner, J. L., James, R. P. et al.: Cryptococcus neoformans infection in a dog. JAVMA 153: 945-949, 1968.

CaseIV - 78-527 (AFIP 1667963).
History: A 350 gm guinea pig was experimentally inoculated with a live agent.

Contributor's Diagnosis & Comments: Entamoeba histolytica - Amebiasis. The guinea pig was inoculated intracecally with 30,000 trophozoites of E. histolytica and eutnanized 2 weeks later.

Attendees' Diagnoses & Comments:
Morphologic Diagnosis: Colitis, necrotizing, ulcerative, acute to subacute, diffuse, severe, colon.
Etiologic Diagnosis: Amebic colitis.
Etiology: Compatible with E. histolytica.
Comment: Most participants observed large amoeba up to 25 microns in diameter, associated with the margins of the ulcers. Several microslides contained characteristic flask-shaped ulcers. The enteric lesions were compared to the changes in the intestine of the goldfish (Case II) and the sizes of the two amoebae were noteworthy.

Contributor: Research Animal & Veterinary Pathology Section, Center for Disease Control, PHS, DHEW, Atlanta, GA 30333.

References:

1. Lynch, E. J.: Histopathological observation on the influence of a special diet used in experimental amebiasis in guinea pig. Am. J. Trop. Med. 6: 813-819, 1957.

2. Maegraith, B., Harinasuta, C.: Experimental amebiasis in the guinea pig. Trans. Roy. Soc. Trop. Med. Hyg. 47: 582, 1953.

3. Phillips, B. P., Wolfe, P. A. et al.: Studies on the ameba-bacteria relationship in amebiasis. Comparative results of the intracecal inoculation of germfree, monocontaminated, and conventional guinea pigs with Entamoeba histolytica. Am. J. Trop. Med. Hyg. 4: 675-692, 1955.

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Results
AFIP Wednesday Slide Conference - No. 7
31 October 1979

Case I - 79M1831C (AFIP 1710525).

History: A post-weanling female Webster mouse was presented with diarrhea and rectal prolapse. At necropsy the only significant finding was a thickened and rigid distal colon. (H&E and PAS microslides are presented.)

Laboratory Results: Citrobacter freundii and Escherichia coli were isolated from the colon.

Contributor's Diagnosis & Comments: "Transmissible murine colonic hyperplasia." This mouse was one of many that had this problem. The C. freundii isolates compare consistently well with the isolates from Argonne National Laboratory in their biochemical reactions. Some of the microslides showed Giardia sp. The significance of these protozoa in the pathogenesis of this murine entity is not known.

Attendees' Diagnoses & Comments:
Morphologic Diagnosis: Hyperplasia, mucosal, diffuse, severe, colon.
Syndrome: Transmissible murine colonic hyperplasia.
Etiology: Consistent with Citrobacter freundii infection.
Comment: Participants were impressed by the hypercellularity of the colonic mucosa but with marked reduction of PAS-positive goblet cells. Inflammatory cells were present in the mucosa but the concensus was that it was within normal limits for the murine colon. Some attendees observed protozoan organisms resembling Giardia sp. on the surface epithelium.

Contributor: U. S. Environmental Protection Agency, HERL, Research Triangle Park, NC.

References:
1. Barthold, S. W., Coleman, G. L., et al.: Transmissible murine colonic hyperplasia. Vet. Path. 15: 223-236, 1978.
2. Barthold, S. W., Osbaldiston, G., et al.: Dietary, bacterial and host genetic interactions in the pathogenesis of transmissible murine colonic hyperplasia. Lab. Anim. Sci. 27: 938-945, 1977.
3. Silverman, J., Chavannes, J. M., et al.: A natural outbreak of transmissible murine colonic hyperplasia in A/J mice. Lab. Anim. Sci. 29: 209-213, 1979.

Case II - M6187/335 (AFIP 1666182).

History: A mature rhesus monkey on a drug safety evaluation study for approximately one year died suddenly with no observed clinical signs. The animal had been given an immunosuppressive compound daily via nasal gavage. The following hemograms are presented:

Date	Hb g/dl	PCV %	RBC $10^6/\mu l$	RBC $10^3/\mu l$	Platelets $10^5/\mu l$
8/77	12.5	39	5.1	17.5	480
6/78	10.3	33	4.1	1.4	29

Contributor's Diagnosis & Comments: Multifocal cerebral cortical necrosis with cavitation. The pathogenesis of the lesion was unclear, but was perhaps caused by cerebral vascular obstruction and resultant ischemic infarction, or hemorrhage into the cerebral cortex with necrosis and cavitation. The WBC and platelet counts decreased gradually over the last month before death; the decrease in platelets may have impaired the coagulability of the blood leading to a cerebral vascular accident. PAS and silver stains failed to reveal fungal organisms.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Fibrosis, with cavitation, focal, severe, cerebrum.

Etiologic Diagnosis: Undetermined.

Comment: Participants were impressed by the amount of fibrous tissue, paucity of inflammatory cells and absence of hemosiderin. The staff noted a lack of active degeneration or necrotizing processes. These findings are consistent with a healed lesion of many months duration. Several participants noted ependymal cells in areas of cavitation, leading them to suspect the original lesion may have been adjacent to a lateral ventricle. The hemograms indicated a significant reduction in red and white blood cell counts. The units for platelets were confusing, and probably represent a typographical error. Units of 10^3 platelets/μl would be consistent with the overall reduction of other blood elements.

Contributor: Pathology Department, Lilly Research Laboratories, Greenfield, Indiana 46140.

Case III - 28449N (AFIP 1714165).

History: Ten of a group of 100 bred gilts experienced episodes of bloody diarrhea during a 3-week period. Following 2 to 6 days of clinical illness, 4 animals died. Medication with several antibacterial drugs did not appear to be effective. Two diseased pigs, one dead and one moribund, were submitted for examination.

Laboratory Results: Gross and microscopic lesions were similar in both gilts. There was staining of the perineal areas by dark red feces, and the carcasses were dehydrated. Gross visceral lesions were limited to the posterior one-third of the small intestine. The serosal surface of the ileum had a convoluted appearance and there was a marked palpable thickness. The mucosa was rugose and covered with free blood that washed off readily, leaving a slightly granular but otherwise normal epithelial surface. The lumen contained long strands of clotted blood.

Contributor's Diagnosis & Comments: [No diagnosis submitted.] Numerous bacteria were observed in the apical cytoplasm of cryptal epithelial cells in tissue stained by the Wartnin-Starry technique.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: Ileitis and hyperplasia, hemorrhagic, acute to subacute, diffuse, moderate to severe, ileum.

Syndrome: Proliferative hemorrhagic enteropathy of pigs.

Etiology compatible with Campylobacter sputorum subsp mucosalis.

Comment: The participants observed marked necrosis and blunting of the intestinal villi with prominent mucosal hyperplasia and abscessation of the crypts. Attendees speculated on the relationship between this syndrome and intestinal adenomatosis of swine and transmissible ileal hyperplasia of hamsters.

Contributor: Department of Patnology and Parasitology, School of Veterinary Medicine, Auburn University, Auburn, AL 36830.

References:

1. Jacoby, R. O.: Transmissible ileal hyperplasia of hamsters. I. Histogenesis and immunocytochemistry. Am. J. Path. 91: 433-450, 1978.

2. Love, D. N., Love, R. J.: Pathology of proliferative hemorrhagic enteropathy in pigs. Vet. Path. 16: 41-48, 1979.

3. Love, R. J., Love, D. N., et al.: Proliferative hemorrhagic enteropathy in pigs. Vet. Red. 100: 65-68, 1977.

4. Rowland, A. C., Lawson, G. H. K.: Intestinal adenomatosis in the pig: A possible relationship with a hemorrhagic enteropathy. Res. Vet. Sci. 18: 263-268, 1977.

Case IV - 79-145 (AFIP 1713079).

History: An epizootic disease occurred in post-weanling mice from a production colony of NIH/Nmri CV. The disease was characterized by failure to gain weight, lethargy, enteropathy and death.

Contributor's Diagnosis & Comments: Enteritis, parasitic; etiology: Spironucleus (Hexamita) muris. Large numbers of trophozoites are seen within the crypts and coating the villous surface of the small intestine. Electron microscopy (see reference) confirmed the presence of the protozoan organisms.

Atttendees' Diagnoses & Comments:

Morphologic Diagnosis: Enteropathy, protozoal, diffuse, small intestine.

Etiologic Diagnosis: Intestinal hexamitiasis.

Etiology: Consistent with Spironucleus (Hexamita) muris.

Contributor: Experimental Pathology Department, Naval Medical Research Institute, Bethesda, MD 20014.

Reference:

Eisenbrandt, D. L., Russell, R. J.: Scanning electron microscopy of Spironucleus (Hexamita) muris infection in mice. Scanning Electron Microscopy 3: 23-27, 1979.

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Results
AFIP Wednesday Slide Conference No. 8
7 November 1979

Case I - CP79-594 (AFIP 1716396).

History: An 8-week-old puppy had shown signs of disorientation and weight loss for about 10 days. The animal had a staggering gait and fell easily; it sometimes became trapped in corners of the room. (H&E and B&B Gram stains of brain tissue are presented.)

Contributor's Diagnosis & Comments: Canine encephalitozoonosis. The disease has been reported several times in dogs in South Africa but only once in dogs from the U. S. Numerous Gram-positive parasites occur in cytoplasmic vacuoles in cerebral endothelial cells and astrocytes. There is a moderate nonsuppurative encephalitis with focal gliosis. (Presented tissue is from an experimentally infected dog that showed the same signs as those seen in the natural disease.)

Attendees' Diagnoses & Comments:

Morphological Diagnoses: 1) Encephalitis, granulomatous, disseminated, mild to moderate, brain. 2) Vasculitis, granulomatous, segmental, mild to moderate, brain.

Etiologic Diagnosis: Cerebral encephalitozoonosis.

Etiology compatible with Encephalitozoon cuniculi.

Comments: Participants were unanimous that the Gram-positive ovoid organisms associated with glial nodules and/or granulomatous vascular lesions were typical of Encephalitozoon. Electron microscopy by AFIP staff has recently revealed that viable organisms are consistently associated with endothelial cells and phagocytosed organisms tend to be degenerating forms.[1] Some microslides also demonstrated foci of malacia.

Contributor: Division of Comparative Medicine, Southwestern Medical School, University of Texas, Dallas, TX 75235.

References:

1. Van Dellen, A. F., Botha, W. S., et al.: Light and electron microscopical studies on canine Encephalitozoonosis: Cerebral vasculitis. Onderstepoort J. Vet. Res. 45: 165-186, 1978.

2. Basson, P. A., McCully, R. M., et al.: Nosematosis: Report of a canine case in the Republic of South Africa. J. S. Afr. Vet. Med. Assoc. 37: 3-9, 1966.

3. Nordstoga, K., Westbye, K.: Polyarteritis nodosa associated with nosematosis in blue foxes. Acta Pathol. Microbiol. Scand. (A) 84: 291-296, 1976.

4. Plowright, W.: An encephalitis-nephritis syndrome in the dog probably due to congenital encephalitozoon infection. J. Comp. Path. 62: 83-92, 1952.

5. Shadduck, J. A., Bendelle, R., et al.: Isolation of the causative organism of canine encephalitozoonosis. Vet. Path. 15: 449-469, 1978.

Case II - A31205 (AFIP 1714167).

History: A 3-year-old female domestic snorthair cat was presented with signs of lethargy and anorexia for about 1 month. In addition, the cat was thin, partially dehydrated, icteric and had very pale mucous membranes. A nemogram yielded the following results: PCV of 12%; Hb of 4.5 gm/dl; many nucleated RBCs were observed in the peripheral blood smear.

Contributor's Diagnosis & Comments: Myeloproliferative disease compatible with erythremic myelosis. The pleomorphic population of cells in the hepatic portal regions with differentiation toward erythroid elements would warrant the diagnosis of erythremic myelosis. There is general agreement, however, that this entity is part of a dynamic process encompassing several disease entities which include reticuloendotheliosis, erythremic myelosis and erythroleukemia.

Attendees' Diagnosis & Comments: Myeloproliferative disease compatible with erythremic myelosis. Most microslides demonstrated a mild diffuse fibrinous capsulitis of the liver, suggestive of a concurrent infection with Feline Infectious Peritonitis virus.

Contributor: Pfizer, Inc., Eastern Point Road, Groton, CT 06340.

References:

Harvey, J. W., Shields, R. P., et al.: Feline myeloproliferative disease - Changing manifestations in the peripheral blood. Vet. Patn. 15: 437-448, 1978.

Case III - 79-230-11 (AFIP 1714920).

History: Tissue from a 2-year-old White-fronted wallaby (Macropus parma) that was received from a zoo in Arizona and died 6 weeks after arrival in the Washington, DC area.

Contributor's Diagnosis & Comments: Toxoplasmosis. The lung and mesenteric lymph node showed necrosis and mononuclear cell infiltrates associated with tachyzoites that are both free and within cells. Structures resembling cyst-forms were also evident. The brain contained organisms associated with glial nodules. Diagnosis was based on histopathologic findings as serum for testing was not available. Australian marsupials appear to be rather susceptible to toxoplasmosis, with overt disease being more common in them than in other zoological species. The source of the infection was not determined, but contamination of food with cat feces is suspected.

Attendees' Diagnoses:

Morphological Diagnoses: 1) Pneumonitis, necrohemorrhagic, acute, diffuse, severe, lung. 2) Lymphadenitis, necrohemorrhagic, acute, diffuse, severe, lymph node.

Etiologic Diagnosis: Disseminated toxoplasmosis.

Etiology compatible with Toxoplasma gondii.

Contributor: National Zoological Park, Wasnington, DC 20008.

References:

1. Dobos-Kovacs, M., Meszaros, J., et al.: Studies on source of Toxoplasma infection in captive kangaroos. Acta Vet Acad Sci Hungaricae 24: 293-301, 1974.
2. Dubey, J. P.: A review of Sarcocystis of domestic animals and other coccidia of cats and dogs. JAVMA 169: 1061-1078, 1976.

Case IV - 19176 (AFIP 1716402).

History: Tissue from a domestic goat which died 24 days after being fed material from a wild carnivore. Four days prior to death the goat became pyrectic (106°F); 2 days before death the goat was icteric.

Contributor's Diagnoses & Comments:

Morphological Diagnoses: 1) Cholangitis, subacute, disseminated, mild, with moderate cholestasis, liver. 2) Leukocytosis, sinusoidal, diffuse, mild to moderate, liver. 3) Glomerulonephritis, protozoan, multifocal, minimal, kidney.

Etiologic Diagnosis: Generalized coccidiosis. (Caprine Dalmeny Disease). Etiology compatible with Sarcocystis spp.

Comments: Most participants reported observing tachyzoite-like organisms in renal glomeruli and capillaries of the medulla, but not in the liver. Staff observed that the normal caprine glomeruli are relatively hypercellular compared to other domestic animals, and that histologic evaluation of the glomeruli in this case would best be performed with control animals, biopsies, or both. In addition to the listed hepatic changes, staff members appreciated subtle degeneration of individual hepatocytes.

Contributor: Marsh Diagnostic Laboratory, Department of Veterinary Science, Montana State University, Bozeman, MT 59717.

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, 1976.
3. Fayer, R., Johnson, A. J., et al.: Oral infection of mammals with Sarcocystis fusiformis bradyzoites from cattle and sporocysts from dogs and coyotes. J. Parasitol. 62: 10-14, 1976.
4. Leek, R. G., Fayer, R.: Sheep experimentally infected with Sarcocystis from dogs. Cornell Vet. 68: 108-123, 1978.

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Results
AFIP Wednesday Slide Conference - No. 9
14 November 1979

Case I - 78-602B (1667074).

History: Twelve horses were stricken with a disease marked by intermittent fever, slight nasal discharge, anorexia, para- and tetra-paresis, and paralysis. Two horses became recumbant and another severely ataxic. The presented tissues are from a horse that was euthanized after several days of lateral recumbancy and urinary incontinence.

Laboratory Results: Three of 12 horses had a significant rise in serum antibody titer (4-fold or greater) against Equine Herpesvirus 1. EHV1 was isolated from lung and spinal cord of one horse which died but not the horse from which lesions are demonstrated.

Contributor's Diagnosis & Comments: Disseminated necrotizing encephalitis due to Equine Rhinopneumonitis Virus. Lesions were limited to the brain and spinal cord and did not involve mesenteric arteries as seen in equine viral arteritis. Both recumbant horses had similar lesions, but they varied in severity. The ataxic horse recovered. There were no abortions, as no horses were pregnant.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: Arteritis with malacia and gliosis, diffuse, mild to moderate, choriomeninges and cerebrum, brain.

Syndrome: Meningoencephalitic equine rhinopneumonitis infection.

Etiology: Equine Herpesvirus 1.

Comment: The primary lesion and principle event in this case is fibrinoid necrosis with thrombosis of medium-sized arterioles. Involvement of medium-sized arterioles is commonly associated with several immune-complex vasculitides, and maybe the result of subacute to chronic viral infections. Gliosis and malacia with swollen axons were observed by most participants, but there was some variability between microslides. Lesions in the neuropil resembled changes associated with vascular embarrassment, and are therefore probably secondary to the vasculitis.

Contributor: Veterinary Diagnostic Laboratory, Pathology Department, Box 138, Tifton, GA 31794.

References:

1. Cochrane, D. G., Dixon, F. J.: Antigen-antibody complex induced disease. IN: Textbook of Immunopathology, P. A. Miescher & H. J. Muller-Eberhard (Eds), Grune & Stratton, New York, 1976, pp 137-156.

2. Jackson, T. A., Kendrick, J. W.: Paralysis of horses associated with Equine Herpesvirus 1 infection. JAVMA 158: 1351-1357, 1971.

3. Little, P. B., Thorsen, J.: Disseminated necrotizing myeloencephalitis: A herpes-associated neurological disease of horses. Vet. Path. 13: 161-171, 1976.

4. Saxegaard, F.: Isolation and identification of equine rhinopneumonitis virus from cases of abortion and paralysis. Nord. Vet. Med. 18: 504-512, 1966.

Case II - J3816 (AFIP 1664173).

History: Multiple tissues (2 microslides) from a 3-year-old Hereford steer which was on a feeding trial. The animal was in an individual pen directly across an alleyway from a group of sheep. Retrospectively, caretakers reported that the dead steer had shown vague intermittent gastrointestinal signs and "irritation around the eyes" for several weeks.

Laboratory Results: Routine virological procedures failed to grow a herpesvirus and rabbit inoculation was innocuous.

Contributor's Diagnosis & Comments: Malignant Catarrhal Fever. Necropsy revealed bilateral corneal opacities, peripheral deep corneal vascularization, hypopyon, erosions on the hard palate and in the esophagus, catarrhal rhinitis, abomasal hyperemia, enlarged retropharyngeal and mesenteric lymph nodes and, increased quantities of turbid synovial fluid with flakes of fibrin in several joints. Histologic lesions included widespread vasculitis, including brain, spleen, lymph nodes, liver and retina. There was periportal infiltration of lymphoreticular cells and a few small lymphoid accumulations in the kidneys and adrenals. Original brain cuts showed scattered perivascular cuffing in addition to the vasculitis. The ocular vasculitis may be absent in some microslides.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Arteritis, fibrinoid, segmental, moderate, eye and brain. 2) Keratitis, interstitial and erosive, with edema, diffuse, severe, cornea. 3) Conjunctivitis, chronic, diffuse, moderate, palpebral and bulbar conjunctiva. 4) Uveitis, anterior, chronic, diffuse, severe, eye.

Syndrome: Malignant Catarrhal Fever.

Etiology: Herpesvirus type B (cell associated).

Comment: Vascular lesions in this bovine were similar to the lesions in the previous equine case, except that involvement of slightly smaller vessels was evident. The ocular lesions in this case are associated with multiple viruses, but the characteristic vascular lesions are very suggestive of the contributor's diagnosis.

Case III - MDA-ANI HLTH LAB (AFIP 1713318).

History: A Loly crown parrot died 2 weeks after being released from an import quarantine station in Philadelphia. Three days prior to death the bird had diarrhea and partial anorexia.

Laboratory Results: At necropsy the ingluvies was empty; there was a slight amount of yellow fluid in the pericardium; the liver was enlarged with white foci; and the spleen was enlarged. Bacterial cultures were negative for pathogens including Salmonella. An embryo-lethal agent was detected from lung and liver tissue which was identified by electron microscopy as Chlamydia psittaci at the NADL in Ames, IA. Special stain (Pinkerton's method) revealed small round red bodies forming clusters in the necrotic areas of the liver and in the cytoplasm of hepatocytes.

Contributor's Diagnosis & Comments: Necrotizing hepatitis and renal degeneration caused by Chlamydia psittaci infection. The gross and microscopic lesions were suggestive of psittacosis which was confirmed by the presence of LCL bodies as well as isolation of chlamydia from the psittacine tissues.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Hepatitis, necrotizing, acute, multifocal, moderate, liver. 2) Nephrosis, tubular, acute, multifocal, mild, kidney.

Etiologic Diagnosis: Hepatic chlamydiosis.

Etiology compatible with Chlamydia psittaci.

Comment: While the renal lesion was considered a non-specific degenerative process, the hepatic necrosis was considered consistent with chlamydiosis by the majority of participants. Many attendees also considered Salmonella, viral agents and mixed infections as compatible with the hepatic lesions.

Contributor: Animal Health Laboratory, Maryland Department of Agriculture, College Park, MD 20740.

References:

1. Burkhart, R. L., Page, L. A.: Chlamydiosis (Ornithosis-Psittacosis). IN: Infectious and Parasitic Diseases of Wild Birds. J. W. Davis, R. C. Anderson, et al. (Eds), Iowa State University Press, Ames, IA, 1971, pp 118-140.

2. Panigrahy, B., Grimes, J. E., et al.: Zoonotic diseases in psittacine birds: Apparent increased occurrence of chlamydiosis (psittacosis), salmonellosis, and Giardiasis. JAVMA 175: 359-361, 1979.

Cas IV - 7263 (AFIP 1712508).

History: This 6-week-old calf was one of several animals with diarrhea which had failed to respond to antibiotic treatment.

Laboratory Results: At necropsy, mucopurulent material was present throughout the upper respiratory tract. Forty percent of lung parenchyma, in an anteroventral region, was firm and deeply reddened. Bronchial lymph nodes were swollen, dark, and contained excessive fluid. Bacterial cultures isolated Pasteurella multocida and viral cultures isolated Bovine Herpesvirus I from lung tissues.

Contributor's Diagnoses & Comments: Acute necrotizing bronchopneumonia compatible with Bovine Herpesvirus I and Pasteurella multocida. In sections, intranuclear inclusions were more easily identified than bacteria. Involvement of other tissues was not appreciated.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Bronchopneumonia, necrotizing, with fibrinous pleuritis, diffuse, severe, lung.

Syndrome: Bovine Respiratory Disease Complex (Shipping Fever).

Comment: The majority of participants considered the pneumonic lesion typical of Pasteurella multocida infection, but were impressed by the scant fibrinous exudation. A few staff members reported observing inclusion bodies compatible with a viral infection.

Contributor: Pathology and Toxicology Research, The Upjohn Company, Kalamazoo, MI.

References:

1. Irwin, M. R., McConnell, S., et al.: Bovine respiratory disease complex: A comparison of potential predisposing and etiologic factors in Australia and the United States. JAVMA 175: 1095-1099, 1979.

2. Jensen, R., Pierson, R. E. et al.: Shipping fever pneumonia in yearling feedlot cattle. JAVMA 169: 500-506, 1976.

3. Schieffer, B., Ward, G. W., et al.: Correlation of microbiological and histological findings in bovine fibrinous pneumonia. Vet. Path. 15: 313-321, 1978.

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ADDENDUM TO RESULTS
Wednesday Slide Conference - No. 9
14 November 1979

Case II - J3816 (AFIP 1664173).

Comment on Etiology: The initial results for this conference listed herpesvirus type B (cell-associated) as the etiology of bovine Malignant Catarrhal Fever. A cell-associated herpesvirus designated bovid herpesvirus 3 is accepted as the etiology of the African form of the disease but numerous viruses have been isolated from natural occurring cases of bovine Malignant Catarrhal Fever in North America, including herpesviruses but cell-free transmission of the disease has not been accomplished.[1]

Reference:

1. Storz, J., Okuna, N., et al.: Virologic studies on cattle with naturally occurring and experimentally induced Malignant Catarrhal Fever. Am. J. Vet. Res. 37: 875-878, 1976.

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Results
Wednesday Slide Conference - No. 10
28 November 1979

Case I - A77-130 (AFIP 1682696).

History: An 8-year-old, castrated male domestic shorthair cat was presented for enteritis and weight loss for several months duration. There was partial improvement following medication for ancylostomiasis and ascariasis.

Laboratory Results: Fecal analysis revealed ova of ascarids, hookworms and capillarids by flotation method.

Contributor's Diagnosis & Comments: Tracheal capillariasis. The cat failed to improve satisfactorily and the owner requested euthanasia. No medication for capillaria infection was administered. Hundreds of capillaria nematodes were found in the trachea and primary bronchi. At autopsy eleven unsuspected adult taenia tapeworms were found in the small intestine along with several ascarids. It is of interest to note that at least 3 stool specimens had been examined without finding taenia eggs; although taenia eggs were found in mature segments but not in the feces at autopsy.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Tracheitis, chronic-active, diffuse, mild to moderate, with squamous metaplasia, trachea and bronchi.

Etiologic Diagnosis: Tracheal capillariasis.

Etiology: Capillaria aerophila.

Comments: Most microslides demonstrated adult nematodes and luminal as well as submucosal ova compatible with the Capillaria aerophila. The bi-polar plugs and thick shell of the ova are characteristic of capillarids. In addition, numerous basophilic bodies, morphologically compatible with a developmental stage of a protozoan were observed in the cytoplasm of some mucosal epithelial cells. These organisms could not be identified from the presented tissues, and their contribution to the clinical symptomatology of the cat remains unclear.

Contributor: Angell Memorial Animal Hospital, 350 South Huntington Ave., Boston, MA 02130.

Case II - X390 (AFIP 1713323).

History: An adult female fox hound was purchased from a vendor for use in a research project and was found to have Dirofilariasis. Euthanasia was performed and incidental findings included these lesions in the trachea and bronchi.

Contributor's Diagnosis: Filaroides osleri of the trachea and bronchi.

Attendees' Diagnoses & Comments:
Morphologic Diagnosis: Bronchitis/Tracheobronchitis, nodular, chronic-active with focal granulomas, moderate to severe, trachea and bronchi.
Etiologic Diagnosis: Tracheobronchial filaroidiasis.
Etiology: Filaroides osleri.
Comments: Attendees were unanimous in attributing the lesions to F. osleri, based primarily on morphology and location of the adult nematodes in the trachea.

Contributor: Department of Comparative Medicine, University of Alabama Medical Center, Birmingham, AL 35294.

Case III - 28845-1 (AFIP 1713319).
History: On routine physical examination of a 3-1/2 year old, male German shepherd, a thickened scrotum with focal reddish, hyperpigmented, raised macules was noted. Over a 2-3 month period macules enlarged and became pedunculated. The largest mass was excised and cauterized. A serous, slightly milky fluid was expressable from the cut surface of the soft, fluctuant mass.

Contributor's Diagnosis & Comments: Hemangioma-like lesion (varicose tumor) of the scrotum. The presence of numerous muscular arteries as well as cross sections of nerves (surrounded by thick, finely fibrillar eosinophilic collars) compounded by the multiple nature and history of recent development presented a confusing picture. The mass did not fit neatly into a diagnosis of capillary hemangioma. The owners elected not to have the remaining masses removed, and refused a recommendation for castration of the animal. One year later telephone follow-up with the owners revealed that all masses except two small pinpoint foci had regressed spontaneously.

Attendees' Diagnoses & Comments:
Diagnosis: Tumor-like[1] (Hemangioma-like)[2] lesion of the canine scrotum.
Comment: Staff and attendees considered the maturity of the stromal connective tissue, lack of mitotic figures and dermal hyperpigmentation to be consistent with the contributor's diagnosis. The clinical course of the untreated remaining masses further supports this diagnosis. This lesion was originally described as "varicose tumor of the scrotum".[3]

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

References:
1. Weiss, E.: International histological classification of tumors of domestic animals. VIII. Tumors of the soft (mesenchymal) tissues. Bull. WHO 50: 101-110, 1974.
2. Moulton, J.: Tumors in Domestic Animals, 2nd Ed., University of California Press, Berkely, 1978, pp 33-35.
3. Weipers, W. L., Jarrett, W. F. H.: Hemangioma of the scrotum of dogs. Vet. Rec. 66: 106-107, 1954.

Case IV - 6451K (AFIP 1711329).

History: A 20 cm X 15 cm X 5 cm pendulous mass was excised from the inguinal area of a 3-year-old Siberian husky. The mass had been excised 18 months previously. The specimen was soft, edematous and oozed a clear fluid from the cut surface.

Contributor's Diagnosis & Comments: Lymphangioma. The history, clinical signs and histology are typical of a lymphangioma.

Attendees' Diagnoses & Comments:

Diagnosis: Lymphangioma.

Comments: Attendees unanimously concurred with the contributor's diagnosis. This tumor is rare in the dog and other domestic animals and little is known of its biological behavior, although local recurrence may be a feature of some lymphangiomas, as in this case.

Contributor: Laboratory of Pathology, School of Veterinary Medicine, University of Pennsylvania, 3800 Spruce St., Philadelphia, PA 19104.

References:

1. Stambaugh, J., Harvey, C. E., et al.: Lymphangioma in the dog: Four case reports. JAVMA 173: 759-761, 1978.

2. Turk, J. R., Gallina, A. M., et al.: Cystic lymphangioma in a colt. JAVMA 174: 1228-1230, 1979.

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Contributor's Diagnosis & Comments: Hepatitis, granulomatous, multifocal; M. avium tuberculosis abortion. Acid-fast bacilli were demonstrated in granulomas by Ziehl-Neelsen stain.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Hepatitis, granulomatous, multifocal, moderate, liver.

Etiology: Possibly tuberculosis.

Comment: There was some variation from slide to slide but attendees were unanimous in their morphologic diagnosis. Necrosis and giant cell formation was evident in the granulomatous foci, but because of autolysis it was difficult to determine which cell types were involved. Acid fast stains revealed positive bacilli within the granulomatous areas.

Contributor: Department of Veterinary Science, South Dakota State University, Brookings, SD 57007.

Reference:

Ellsworth, S. R., Kirkbride, C. A., et al: Mycobacterium avium abortion in a sow. Vet. Path. 16: 310-317, 1979.

Case III - 78D501 (AFIP 1693934).

History: This 1½ year old male domestic bovine was the third animal to die on a Maryland farm within one year. At presentation, the animal was emaciated and dehydrated.

Laboratory Results: Impression smears made from the lymph nodes and intestinal mucosa showed acid fast organisms. Mycobacterium paratuberculosis was isolated from the fecal samples; no other pathogens were isolated from blood and intestinal samples. Virus isolation attempts were non-productive.

Contributor's Diagnosis & Comments: Johne's Disease (Mycobacterium paratuberculosis). On postmortem examination the lesions were restricted to the digestive system and mesenteric lymph nodes. The mucosa of the posterior part of the intestinal tract was thickened and thrown into folds. The mesenteric lymph nodes were greatly enlarged, pale and edematous.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Enterocolitis, granulomatous, diffuse, severe, small intestine and colon.

Syndrome: Johne's Disease.

Etiology consistent with Mycobacterium paratuberculosis.

Comment: Again, the attendees unanimously agreed with the contributor's diagnosis. An acid fast stain demonstrated macrophages filled with acid fast bacilli.

Contributor: Animal Health Laboratory, Maryland Department of Agriculture, Centreville, MD 21617.

References:
1. Hale, N. H.: Johne's Disease. Adv. Vet. Sci. 4: 341-387, 1958.
2. Thoen, C. O. and Muscoplat, C. C.: Recent development in diagnosis of paratuberculosis (Johne's Disease). JAVMA 174: 838-847, 1979.
3. Buergelt, C. D., Hall, C. E., et al: Lymphocyte transformation: An aid in the diagnosis of paratuberculosis. AJVR 38: 1709-1715, 1977.
4. Johnson, D. W., Muscoplat, C. C., et al: The use of lymphocyte transformation for the diagnosis of paratuberculosis (Johne's Disease) in infected cattle. Proc. 81st Annu. Mtg. US Anim. Health Assoc., 1978, pp 467-469.

Case IV - 78S156 (AFIP 1664302).

History: Tissue from the head of an adult armadillo which died of hyperthermia (heat stroke).

Contributor's Diagnosis & Comments: Tongue muscle showing sarcocysts containing spores of sarcosporidia.

Attendees' Diagnosis & Comments:
Diagnosis: Sarcocystosis, intramuscular, multifocal, moderate, tongue.
Comments: Sarcocystosis is a common finding in many wild and zoo birds, reptiles and mammals. Life cycles for most Sarcocystis sp. have yet to be elucidated.

Contributor: Department of Comparative Medicine, Milton S. Hershey Medical Center, Hershey, PA 17033.

Reference:
Anderson, J. M. and Benirschke, K.: The armadillo, Dasypus novemcinctus, in experimental biology. Lab. Anim. Care 16: 202-216, 1966.

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References:

1. Nielsen, S. W.: Spontaneous hematopoietic neoplasms of the domestic cat. In: Comparative Morphology of Hematopoietic Neoplasms. Natl. Cancer Inst. Monogr. 32: 73-90, 1969.

2. Stannard, A. A. and Pulley, L. T.: Tumors of the skin and soft tissues. In: Tumors in Domestic Animals, 2nd Ed., 1978, J. E. Moulton (Editor), Univ. of California Press, Berkeley, pp 16-74.

3. Jarrett, W. F. H. and Mackey, L. J.: International histological classification of tumors of domestic animals. II. Neoplastic diseases of the hematopoietic and lymphoid tissues. Bull. World Health Org. 50: 21-34, 1974.

4. Confer, A. W. and Langloss, J. M.: Long-term survival of two cats with mastocytosis. JAVMA 172: 160-161, 1978.

5. Jones, S. R., MacKenzie, W. F., et al: Comparative aspects of mastocytosis in man and animals with report of a case in a baboon. Lab. Anim. Sci. 24: 558-562, 1974.

Case II - CP79-894 (AFIP 1712578).

History: On 1 August, an adult male Australian shepherd was initally presented for lethargy, weakness, pyrexia (104.3°F) and pale mucous membranes. The dog was treated for uncinariasis and discharged. Two weeks later the dog was again presented for the same continuing problems: lethargy, weakness, pyrexia (104.1°F) and pale mucous membranes. In additon, the animal had lost weight and had developed a hemorrhagic stool. The dog died before tests on 15 August were completed.

Laboratory Results:

Hemogram	1 Aug	15 Aug
PCV	30	21
RBC	4.4	3.0
Hb	10.5	6.7
WBC	5.6	3.35
Neutro's	86%	58%
Lympho's	10%	37%
Mono's	4%	1%
Eosino's	0%	4%
Nucl RBCs		9/100 WBC
Reticulocytes		1%
Platelets	250,000	10,000

Chemistries	1 Aug	15 Aug
Tot Protein	5.4	6.8
Albumin		1.9
BUN	10	10
Bleeding time		8 min
Coagulation time		<4 min
Prothrombin time		8 sec
Parasites		
Microfilaria	Neg	Neg
Fecal	Heavy hookworm	Heavy hookworm

Contributor's Diagnosis & Comments: Canine Ehrlichiosis. Serum submitted for canine ehrlichial titration had a titer of 1:1280 which was the highest dilution tested by the laboratory. E. canis inclusion bodies were not identified with certainty within leucocytes on blood films.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Plasmacytosis, diffuse, severe, spleen, canine.

Etiologic Diagnosis: Consistent with Ehrlichiosis.

Comments: The presented clinical pathological data and splenic lesion led participants to strongly consider Ehrlichiosis. A prolonged bleeding time with essentially normal coagulation time and prothrombin time is compatible with the thrombocytopenia; in addition, panleukopenia and anemia are typical findings in chronic infections of Ehrlichiosis. Generalized plasmacytosis is very suggestive of E. canis infection, especially when the meninges, retina, and kidneys are involved.[2] The rickettsial organisms are very difficult to demonstrate in tissue sections, but may be visualized on impression smears of the lung stained with Giemsa stain. The German shepherd breed is more prone to show overt severe symptomatology with E. canis infections than other small breeds.

Contributor: Southwestern Medical School, 5323 Harry Hines Blvd., Dallas TX 75235.

References:

1. Buhles, W. C., Huxsoll, D. L., et al: Tropical canine pancytopenia. Clinical, hematologic and serologic response of dogs to Ehrlichia canis infection, tetracycline therapy and challenge inoculation. J. Inf. Dis. 130: 357-367, 1974.

2. Hildebrandt, P. K., Huxsoll, D. L., et al: Pathology of canine ehrlichiosis (tropical canine pancytopenia). Am. J. Vet. Res. 34: 1309-1320, 1973.

3. Huxsoll, D. L., Hildebrandt, P. K., et al: Tropical canine pancytopenia. JAVMA 157: 1627-1632, 1970.

Case III - 78-257 (AFIP 1711328).

History: Splenic tissue from a cynomolgus monkey (Macaca fascicularis). Numerous pale nodules 2-5mm in diameter were present throughout the spleen; some elevated the capsule.

Contributor's Diagnoses & Comments:

Morphologic Diagnoses: 1) Splenitis, granulomatous, multifocal, characterized by giant cells and eosinophils: etiology is microfilaria. 2) Splenitis, granulomatous, diffuse, red pulp, characterized by eosinophils: associated with microfilaria.

Comments: The pathologist did not have a complete set of necropsy specimens which might have permitted identification of adult nematodes. Degenerating microfilaria were observed primarily in multinucleated giant cells within the splenic granuloma. Filarial nematodes are common parasites of primates and at least 40 species have been described from apes and monkeys. Significant pathological changes are not associated with the adult worms in the subcutis of non-human primates. Granulomatous nodules originating in the splenic red pulp have been reported in drills infected with Loa.[1] The nodules were attributed to the destruction of microfilaria. Treatment of the infection and clearing of microfilaria from the peripheral blood resolves the lesions.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Splenitis, granulomatous and eosinophilic, nodular and diffuse, moderate to severe, spleen, macaque.

Etiologic Diagnosis: Parasitic splenic granulomatous inflammation associated with microfilaria of an unidentified filarid nematode.

Comments: Participants most often mentioned Loa loa, Brugia sp. and Dipetalonema sp. as the likely filarid nematodes responsible for the microfilaremia. Due to the presence of multinucleated giant cells, acid fast stains were performed, but failed to demonstrate bacteria. A combination of eosinophils and giant cells is suggestive of helminthic parasitism. Many of the microslides did not contain microfilaria.

Contributor: School of Aerospace Medicine, Brooks Air Force Base, TX 78235.

References:

1. Duke, B. O. L.: Studies on Loiasis in monkey. III. The pathology of the spleen in drills infected with Loa. Ann. Trop. Med. Parasit. 54: 141-146, 1960.

2. Orihel, T. C. and Seibold, H. R.: Nematodes of the bowel and tissue. In: Pathology of Simian Primates, Part II, 1972, R. N. T-W-Fiennes, Editor, S. Karger, Basel, pp 76-103.

Case IV - SWRI Case I (AFIP 1718122).

History: This 8 week old, female, albino Charles River rat died while on a 2 week, 3 dose level toxicology study.

Contributor's Diagnosis & Comments: Necrosis and inflammation, subacute to chronic, multifocal, moderate, heart. Etiology: Tyzzer's disease. Liver lesion consisted of foci of coagulation-type necrosis with inflammatory cell infiltration, and presence of intracytoplasmic bacilli suspected to be Bacillus piliformis. A Giemsa stain demonstrated intracytoplasmic bacilli in cells at the periphery of the lesions in both the heart and liver compatible with Bacillus piliformis. It is interesting to note that only high and mid-dose animals, under stress, developed the disease. Also, the heart was equally involved which is not a very common site for this disease.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Myocarditis, necrotizing, multifocal, moderate, heart, rat.

Etiologic Diagnosis: Tyzzer's disease, Bacillus piliformis.

Comments: Most participants observed faint clusters of elongated bacilli within the cytoplasm of degenerating myocardial cells at the periphery of the necrotic foci. The small number of inflammatory cells in the necrotic foci, a typical feature of Tyzzer's disease, led some staff members to consider myocardial necrosis to be an acceptable alternative diagnosis. Involvement of the myocardium, while not as common as the enterohepatic form of the disease, is, nevertheless, well documented and is a useful diagnostic feature of generalized Tyzzer's disease.[1,2,4]

Contributor: Sterling-Winthrop Research Institute, Rensselaer, NY 12144.

References:

1. Ganaway, J. R., Allen, A. M., et al: Tyzzer's disease. Am. J. Path. 64: 717-730, 1971.

2. Stedham, M. A., Bucci, T. J., et al: Tyzzer's disease in the rat. Lab. Invest. 20: 604, 1969.

3. Jonas, A. M., Percy, D. H., et al: Tyzzer's disease in the rat. Arch. Path. 90: 516-528, 1970.

4. Schaich-Fries, A. and Svendsen, O.: Studies on Tyzzer's disease in rats. Lab. Anim. 12: 1-4, 1978.

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Results
AFIP Wednesday Slide Conference - No. 13
9 January 1980

Case I - 66983 (AFIP 1666470).

History: An approximately 12-month-old recently imported chimpanzee was found dead in its cage after several days of anorexia.

Contributor's Diagnoses & Comments: Strongyloidiasis. Strongyloides stercoralis or S. intestinalis are usually incriminated. In addition to strongyloidiasis, this animal had a severe moniliasis of the tongue and esophagus. Fatal strongyloidiasis has been reported in chimpanzees.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Enteritis, chronic-active, diffuse, severe, small intestine.

Etiologic Diagnosis: Enteric strongyloidiasis.

Etiology: Consistent with Strongyloides fulleborni.

Comments: Numerous rhabditiform larvae and eggs typical of Strongyloides sp. were present in dilated crypts and interepithelial tunnels. In some sections larvae were seen in the submucosa and Peyer's Patches where they had illicited a granulomatous response. While the adult parthenogenic female strongyloidids are normally restricted to the small intestine, few diagnostic sections of adults were observed in multiple sections; hence the exact identification of the parasite was difficult. Strongyloides fulleborni is the most common species reported to occur in chimpanzees and other great apes.[1]

Contributor: Bureau of Biologics, Food & Drug Administration, 8800 Rockville Pike, Bethesda, MD 20014.

References:

1. De Paoli, A., Johnsen, D. O.: Fatal strongyloidiasis in gibbons (Hylobates lar). Vet. Path. 15: 31-39, 1978.

2. McClure, H. M., Strozier, L. M., et al.: Strongyloidosis in two infant orangutans. JAVMA 163: 629-632, 1973.

Case II - 79-4692 (AFIP 1718544).

History: Several juvenile rabbits in one litter began showing central nervous system symptoms characterized by torticollis and ataxia. The affected animals were destroyed by the owner and one was submitted for necropsy. No significant lesions were observed on gross examination.

Contributor's Diagnoses & Comments: Meningoencephalitis, eosinophilic, malacic, verminous. Significant microscopic lesions were limited to the brain and spinal cord. The metazoan parasites in sections of the brain are nematodes. The presence of lateral alae suggests that they are probably ascarid larva which are undergoing aberrant migration in an unusual host.

Attendees' Diagnoses & Comments:
Morphologic Diagnosis: Meningoencephalitis, subacute, multifocal, severe, cerebellum and medulla.
Etiologic Diagnosis: Encephalitic ascariasis.
Etiology: Larval nematodes suggestive of Baylisascaris procyonis.
Comments: Nearly all microslides demonstrated the presence of ascarid larvae. The larvae were frequently located away from the areas of intense inflammatory changes, and often had no inflammatory cells around them. The inflammatory cells consisted of heterophils, immunocytes and gitter cells. The most severe changes consisted of multifocal areas of malacia and associated cellular infiltration. The presence of these ascarid larvae in the rabbit are highly suggestive of contamination of the environment or food with raccoon feces. The raccoon ascarid was previously reported as Ascaris columnaris, but the correct designation for this parasite is Baylisascaris procyonis.[1]

Contributor: Laboratory of Diagnostic Medicine, College of Veterinary Medicine, University of Illinois, Urbana, IL 61801.

References:
1. Jacobson, H. A., Scanlon, P. F., et al.: Epizootiology of an outbreak of cerebrospinal nematodiasis in cottontail rabbits and woodchucks. J. Wildl. Dis. 12: 357-360, 1976.
2. Church, E. M., Wyand, D. S., et al.: Experimentally induced cerebrospinal nematodiasis in rabbits. Am. J. Vet. Res. 36: 331-335, 1975.

Case III - A77-342 (AFIP 1623521).
History: Incidental finding in a 1-1/2 year old female research colony cat which died with acute hemorrhagic cystitis and urethral calculi obstruction.

Laboratory Results: The flukes were removed from fixed tissues and tentatively identified as Eurytrema procyonis.

Contributor's Diagnoses & Comments: Pancreatic ductal trematodiasis due to Eurytrema procyonis.

Attendees' Diagnoses & Comments: Pancreatitis, interstitial and periductal, chronic-active, diffuse, moderate, with atrophy of acini, pancreas.
Etiologic Diagnosis: Pancreatic ductal trematodiasis.
Comments: Attendees were impressed by the diffuseness and severity of the pancreatic change associated with the presence of the trematodes. Tissue reaction to pancreatic ductal parasites is generally mild, but pronounced fibrosis has been reported.[2] The trematode could not be positively identified from tissue sections, but two flukes were considered the most likely species in cats in the continental United States: Eurytrema procyonis and Platynosomum concinnum.

Contributor: School of Aerospace Medicine, Comparative Pathology Branch, Veterinary Services Division, Brooks AFB, TX 78235.

References:
1. Burrows, R. B., Lillis, W. G.: Eurytrema procyonis Denton, 1942 (Trematoda: Dicrocoeliidae), from the domestic cat. J. Parasitol. 46: 810-812, 1960.
2. Sheldon, W. G.: Pancreatic flukes (Eurytrema procyonis) in domestic cats. JAVMA 148: 251-253, 1966.

Case IV - 79-364 (AFIP 1718322).

History: Peri-ureteral tissues from a 6-month-old female porcine. The animal died in a holding pen at a local auction and was submitted for necropsy.

Laboratory Results: There were no antemortem clinical laboratory tests. The parasite was keyed out as Stephanurus dentatus.

Contributor's Diagnoses & Comments: Stephanuriasis inducing severe periureteritis. This is a very common incidental (pig sale and slaughter) finding in swine from the Tuskegee area. The parasite is responsible for significant economic loss every year.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Periureteritis, granulomatous, multifocal, moderate to severe, (periureteral) adipose tissue.

Etiologic Diagnosis: Periureteral stephanuriasis.

Etiology: Compatible with Stephanurus dentatus.

Comments: The attendees concurred with the contributor's diagnosis. The morphology of the parasite, the eggs and its location were considered diagnostic.

Contributor: Department of Pathology, School of Veterinary Medicine, Tuskegee Institute, Tuskegee, AL 36088.

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Results
AFIP Wednesday Slide Conference - No. 14
16 January 1980

Case I - AFIP 1719055.

History: Body section from a hatchery-raised brown trout. Many fish in the hatchery were affected. Some were observed rubbing themselves against the sides or bottom of the pond.

Contributor's Diagnosis: "Ich".

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Hyperplasia, epidermis, parasitic, diffuse, moderate, skin.

Etiologic Diagnosis: Ichthyophthiriasis.

Etiology: Ichthyophthirius multifiliis.

Comments: All attendees reported the presence of ciliated organisms within the hyperplastic/hypertrophied cuticle (some being present between the epidermis and corium). The size of the trophozoite, the presence of cilia, the crescent to horseshoe-shaped macronucleus (best seen on wet scrapings) and the hyperplastic epidermal response were considered diagnostic of Ichthyophthirius sp. infection.[1,2,3,4] The life cycle of this ciliate has been well elucidated. A few microslides which contained visceral organs of the fish demonstrated multiple bacterial colonies in the renal tubule lumina; the significance of these bacteria was not known.

Contributor: Glenn L. Hoffman, Eastern Fish Disease Laboratory, Leetown, WV 25430.

References:

1. Mawdesley-Thomas, L. E., Jolly, D. W.: Diseases of Fish. II. The goldfish (Carassius auratus). J. Sm. Anim. Pract 8: 533-541, 1967.

2. Hoffman, G. L.: Ciliates of Freshwater Fishes. IN Parasitic Protozoa II. Intestinal Flagellates, Histomonads, Trichomonads, Amoeba, Opalinids, and Ciliates. J. P. Kreier (editor), Academic Press, New York, 1978, pp 585-632.

3. Allison, R., Kelly, H. D.: An epizootic of Ichthyophthirius multifiliis in a river fish population. Progr. Fish-Cult 25: (3), 1963.

4. Davis, H. S.: Culture and Diseases of Game Fishes, University of California Press, Berkeley, 1967, pp 209-212.

Case II - AFIP 1719054.

History: Tissue from a wild-caught, free-living moribund salmon found on a stream bank in the Pacific Northwest.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Granulomas, metacercarial, multifocal, moderate to severe, kidney, fish.

Etiologic Diagnosis: Renal metacercariasis.

Etiology: Metacercaria of Nanophyetus salmincola.

Comments: Sections of the kidney demonstrated numerous piriform, encysted trematodes. The cyst wall was thin and usually surrounded by a rim of mononuclear inflammatory cells with a thin fibrous capsule around the leucocytes. The cysts were considered small (micro-cercous) compared to most metacercaria. The metacercaria had two suckers (distomate) of roughly equal size. These morphologic features, in addition to the lack of a forked tail and histology of the excretory bladder[3] were useful features in keying out the metacercarial parasites of this trematode. Geographic region and host species are also very useful, where provided. The metacercaria of this trematode may occur in almost all tissues of the fish, but are usually most numerous in the kidneys, muscles and fins.[4] Heavy infections can result in death of fresh-water fish. These parasites have economic importance to salmonid fry and serve as the vector for the rickettsial organism Neorickettsia helmintheca, which causes a serious disease in the final host, wild and domestic canids.

Contributor: W. T. Yasutake, Western Fish Disease Laboratory, USDI, Seattle, WA 98115.

References:

1. Millemann, R. E., Knapp, S. E.: Pathogenicity of the "salmon poisoning" trematode, Nanophyetus salmincola, to fish. IN A Symposium on Diseases of Fishes and Shellfishes. S. F. Snieszko (editor), American Fisheries Society Special Pub. No. 5, Washington, DC, 1970, pp 209-217.

2. Wood, E. M., Yasutake, W. T.: Histopathology of Fish. IV. A granuloma of brook trout. Prog. Fish-Cult. 18: 108-112, 1956.

3. Hoffman, G. L.: Parasites of North American Freshwater Fishes, University of California Press, Berkeley, 1967, pp 161-201.

4. Needham, T., Wootten, R.: The parasitology of teleosts. IN Fish Pathology, R. J. Roberts (editor), Bailliere Tindall, London, 1978, pp 1144-1182.

Case III - 77-34 (AFIP 1666534).

History: Tissue from an adult menhaden (Brevoortia tyrannus) netted in a Rhode Island estuary.

Contributor's Diagnosis & Comments: Testicular degeneration and fibrosis due to Eimeria brevoortiana infection. The parasite is somewhat unusual in having the sporogonic stages of its development within the testes of the host and the gametogonic stages in the pyloric ceca.

Attendees' Diagnoses & Comments: Morphologic Diagnosis: Orchitis, chronic, protozoal, with interstitial fibrosis and atrophy, diffuse, severe, testis.

Etiologic Diagnosis: Testicular coccidiosis.

Etiology: Eimeria brevoortiana.

Comments: The testicular tissue is markedly atrophied and the tubules containing large number of oocysts. A few microslides demonstrated an occasional seminiferous tubule with germinal cells in stages of spermatogenesis. The oocysts were typical of Eimera, having four sporocysts per oocyst, and two closely compacted sporozoites within each sporocyst. Species' name is derived from the host. The life cycle has been briefly described[1,2] and it is worth noting the absence of schizonts and gametogony in the testicle.

Contributor: Central Research, Pfizer Inc., Groton, CT 06340.

References:

1. Hardcastle, A. B.: Eimeria brevoortiana, a new sporozoan parasite from menhaden (Brevoortiana tyrannus), with observations on its life history. J. Parasitol. 30: 60-68, 1944.

2. Pellerdy, L.: Coccidia of Marine Fishes. In Coccidia and Coccidiosis, Akademiai Kiado, Budapest, 1974, pp 87-94.

Case IV - 390-79 (AFIP 1689415).

History: Liver tissue from an aged Bactrian camel (Camelus bactrianus) which died of acute bloat.

Contributor's Diagnosis & Comments: Multifocal hepatic osteolipomatous metaplasia. Gross lesions were confined to the liver. Approximately one-fourth of the total volume of the liver was involved.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Metaplasia, osteolipomatous, multifocally disseminated, severe, liver.

Comments: The participants unanimously agreed that the lesion was a metaplastic change rather than a neoplasm. The lesions in this liver were compared to the myelolipoma of wild and domestic felids but there were significant differences: There was no myeloid component in this liver as is a criteria of myelolipoma; similarly, there were no nodules or compressive growth typical of myelolipoma; bone formation is not reported as a component of the myelolipoma. Most participants considered the fat cells to be histologically compatible with normal lipocytes rather than hepatocytes undergoing severe fatty metamorphosis. It was speculated that the origin of the lipocytes and bone forming cells was a primitive or undifferentiated mesenchymal cell within the liver. Fat-storing cells (lipocytes, Ito cells)[3,4] have been described histologically and ultrastructurally in animals (pig,[4] rabbits,[5] rat[6]) as well as man.[4] They may number one per every 20 hepatocytes in routine sections. Little is known about these cells, however, there are reports in the literature of human patients with massive accumulation of lipid in fat-storing Ito cells as the result of chronic hypervitaminosis A. An assay for vitamin A in this camel might be of interest. The etiology for the osseous metaplasia remains unclear.

The contributor, in subsequent studies on livers of camels, has indicated that this condition is relatively common histologic findings in aged animals. Dr. R. J. Montali (Pathologist National Zoological Park) reported that one of two camels recently necropsied at the National Zoological Park had multiple areas of adipose tissue without bony formation.

Contributor: Veterinary Diagnostic Laboratory, School of Veterinary Medicine, Oregon State University, Corvallis, OR 97331.

References:

1. Lombard, L. S., Fortna, F. M., et al.: Myelolipomas of the liver in captive wild felidae. Vet. Path. 5: 127-134, 1968.

2. Gourley, I. M., Popp, J. A., et al.: Myelolipomas of the liver in a domestic cat. JAVMA 158: 2053-2057, 1971.

3. Bronfenmajer, S., Schaffner, F., et al.: Fat-storing cells (lipocytes) in human liver. Arch. Path. 82: 447-453, 1966.

4. Ito, T., Nemoto, M.: Ueber die Kupfferschen Sternzellen und die "Fettspeicherungszellen" ("fat-storing cells") in der Blutkapillarenwand der menschlichen Leber. Okajima Folia Anat Jap 24: 243-258, 1952.

5. Yamagishi, M.: Electron microscopic studies on the fine structure of sinusoidal wall and fat-storing cells of rabbit livers. Arch. Histol. Jap. 18: 223-261, 1959.

6. Tamikawa, K., Yoshimura, K., et al.: Fine structure of the reticulo-endothelial cells in the normal rat liver: Morphological classifications. Kurume Med. J. 12: 139-147, 1965.

7. Hruban, Z., Russell, R. M. et al.: Ultrastructural changes in livers of two patients with hypervitaminosis A. Am. J. Pathol. 76: 451-461, 1974.

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Results
AFIP Wednesday Slide Conference - No. 15
23 January 1980

Case I - 78-257 (AFIP 1662196).

History: A mature Guernsey originated in Indiana and was added to a milking herd in Maryland. She lost weight for several months after the shipment, had mastitis and a chronic cough. She was diagnosed as having lymphosarcoma and was destroyed.

Laboratory Results: Mycbacterium bovis was isolated from several tissues.

Contributor's Diagnosis & Comments: Tuberculous mastitis. This case involved lungs, pleura, liver, peritoneum, mammary glands and lymph nodes, including the mammary lymph nodes. Nieberle & Cohrs (1967) note that in cases of tuberculous mastitis the regional nodes are generally not involved. Gross changes in the udder were not particularly prominent. The gland was diffusely firm and slightly granular and contained watery, light brown, flocculent secretions. Acid fast organisms were present in ducts. Cases of tuberculous mastitis should always be regarded as patent and the zoonotic potential for those consuming raw milk is significant.

Attendees' Diagnosis & Comments:
Morphologic Diagnosis: Granulomas, coalescing, multiple, severe, mammary gland.
Etiologic Diagnosis: Consistent with tuberculous mastitis.
Etiology: Consistent with Mycobacterium bovis.
Comments: Participants unanimously agreed with the contributor's diagnosis. Epithelioid macrophages (with a tendency toward spindling shape and elongate nuclei) and both Langhan's and foreign body giant cells surrounding foci of mineralizing caseous material are highly suggestive of tuberculosis in cattle. Acid fast stain revealed numerous red bacilli, an unusual finding in bovine tuberculosis, where acid fast bacilli tend to be scattered and few in number.

Contributor: New Bolton Center, School of Veterinary Medicine, University of Pennsylvania, Kennett Square, R.D. 1, PA 19348.

Reference:
Nieberle, K., Cohrs, P.: Textbook of the Special Pathological Anatomy of Domestic Animals, Pergamon Press Ltd, Oxford, 1967, pp 783-788.

Case II - 77-2545-17 (AFIP 1633432).

History: Tissue taken from a 5-month-old Hereford heifer maintained on native grass pasture in Kansas. Four other heifers, ranging in age from 5 to 18 months, were similarly affected. The 5 heifers received diethylstilbestrol at birth and a growth-promoting implant at 6 days of age.

Laboratory Results: Clinical pathological findings consisted of a moderate neutrophilia and fibrinogen greater than 900 mg%. Corynebacterium pyogenes was the principle bacterial isolate from necropsy tissues. An alpha Streptococcus sp. and a Peptostreptococcus sp. were also isolated.

Contributor's Diagnosis & Comments: Gangrenous mastitis. In addition to the bacterial isolates, an unidentified mycoplasma-like-agent was recovered from affected tissue. Herpetiform mammillitis was considered. Attempts to transmit this condition by teat scarification was successful in one of two recipient animals; vesicles ensued which progressed to scab formation. Attempts to isolate a herpesvirus have not been successful.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Mastitis, necrotizing, focally extensive, mammary gland.

Comments: The histologic changes are compatible with a gross diagnosis of gangrene. Some sections had only necrosis of the dermis; a morphologic diagnosis of necrotizing dermatitis was felt to be most appropriate for those microslides. An etiology for this lesion could not be determined from the sections. In the absence of an inflammatory reaction, the numerous colonies of cocci in the necrotic material have an uncertain significance: the bacteria may represent postmortem growth or antemortem contaminants in necrotic tissue. Participants considered a pathogenesis by estrogen-induced thrombosis, as is reported in man, however, this has apparently not been reported in the bovine species. Acute ergotism was also considered a possible etiology, especially in those sections that demonstrated only dermal necrosis.

Contributor: Department of Pathology, School of Veterinary Medicine, Kansas State University, Manhattan, KS 66502

References:

1. Natterman, H., Horsch, F.: Zur Pathogenese der Corynebacterium pyogenes Mastitis des Rindes. Monatshfte Vet Med 32: 342-345, 1977.
2. Schalm, O. W.: Pathological changes in the milk and udder of cows with mastitis. JAVMA 170: 1137-1140, 1977.
3. Bovine mastitis produced by Corynebacteria. JAVMA 170: 1164-1165, 1977.
4. Jasper, D. E.: Mycoplasma and mycoplasma mastitis. JAVMA 170: 1167-1172, 1977.
5. Boughton, E.: Mycoplasma bovis mastitis. Vet Rec 103: 70-71, 1978.
6. Counter, D. E.: A severe outbreak of bovine mastitis associated with Mycoplasma bovigenitalium and Acholeplasma laidlawii. Vet Rec 103: 130-131, 1978.
7. Gourlay, R. N., Wyld, S. G., et al.: Isolation of Mycoplasma canadense from an outbreak of bovine mastitis in England. Vet Rec 103: 74-75, 1978.

Case III - 76816 (AFIP 1668874).

History: A rapidly growing mass was excised from a posterior abdominal mammary gland of an 11-year-old, female, Terrier-cross breed.

Contributor's Diagnosis & Comments: Carcinosarcoma. The sections contain a neoplasm composed of well-differentiated squamous cell carcinoma, and pleomorphic and anaplastic sarcomatous tissue. Both components of the tumor show malignant features. The connective tissue component appears to be fibroblastic in some

areas; although generally quite pleomorphic, there are areas in which definite striated muscle cells may be found. PTAH stain demonstrated cross-striations in some of the tumor cells, which have the appearance of strap cells.

Attendees' Diagnoses & Comments:
Diagnosis: Carcinosarcoma.
Comments: Attendees agreed there were two distinct populations of cells composing this tumor. One was a well-differentiated squamous cell carcinoma, and the other, a sarcomatous spindle cell element with occasional cross-striations. Although this particular combination of mesenchymal and epithelial malignant cells is not specifically described in standard veterinary oncology references, squamous cells and muscle cells have clear origins, hence, a diagnosis of carcinosarcoma would be appropriate.

Contributor: Comparative Medical & Veterinary Services, Los Angeles County Department of Health Services, 12824 Erickson Ave., Downey, CA 90242.

References:
1. Hampe, J. F., Misdorp, W.: International Histological Classification of Tumors of Domestic Animals. IX. Tumors and dysplasias of the mammary gland. Bull. WHO 50: 111-133, 1974.
2. Moulton, J. E.: Tumors of the mammary gland. In: Tumors in Domestic Animals, 2nd Ed., J. E. Moulton (Ed.), University of California Press, Berkeley, 1978, pp 346-271.

Case IV - A19977 (AFIP 1714921).
History: A 9-year-old female Siamese cat was presented with a dry, non-productive cough, dyspnea and poor appetite. Physical examination revealed cyanosis, harsh lungs sounds and gallop rhythm. A serosanguinous fluid filled the chest cavity.

Contributor's Diagnosis & Comments: Thymic carcinoid. A 2x2x2 cm pale, firm, encapsulated mass was present in the anterior mediastinum. There was marked ventricular hypertophy and passive congestion of the liver and lungs. Histologic features and positive argyrophilic staining established the diagnosis. Carcinoids of thymic origin have been described in man and this is possibly the first report in the cat at this site. A paper reporting six cases (including this case) has been submitted by the contributing institution.

Attendees' Diagnoses & Comments:
Diagnosis: Adenocarcinoma.
Comments: None of the participants reported seeing a tumor comparable to this one. The majority of participants considered the lesion to be a well-differentiated adenocarcinoma of undetermined origin. Several attendees, however, believed the tumor had benign histologic features, such as nominal mitotic rate, retention of basement membranes around tubulo-alveoli and good cellular differentiation. Some participants remarked on the resemblance of the glandular structures to the polycystic tumor seen infrequently in the thymic region of aged rats.[3,4] It was also suggested that the glandular tissues of this tumor may be (metaplastic) remnants of thymic epithelial cells of branchial cleft origin.

Special stains revealed PAS-positive-diastase-resistent material within the tubulo-alveolar lumens and as minute granules within the luminal columnar cells. AMP stain failed to demonstrate acid mucopolysaccharides/hyaluronic acids within cells or tubular lumens. Argyrophilic staining (Sevier-Munger method) was inconclusive. In consultation with AFIP's Pulmonary & Mediastinal Pathology Branch, the tumor did not resemble human thymic tumors and it was suspected to be an adenocarcinoma. Consultation with the contributing institution confirms that a paper describing thymic carcinoids in the dog and cat has been submitted for publication.

Contributor: Pathology Department, The Animal Medical Center, 510 East 62 Street, New York, NY 10021.

References:

1. Rosai, J., Higa, E.: Mediastinal endocrine neoplasms of probable thymic origin, related to carcinoid tumors. Cancer 29: 1061-1074, 1972.

2. Levine, G. D., Rosai, J.: A spindle cell variant of thymic carcinoid. Arch Pathol Lab Med 100: 293-300, 1976.

3. Burek, J. D.: Pathology of Aging Rats, CRC Press, West Palm Beach, FL, 1978, pp 110-116.

4. Burek, J. D., Neihuizen, S. P.: Age-related characteristics of thymuses from BN/Bi rats. In: Proc 5th Eur Symp Basic Res Gerontol, Schmidt, Bruschke, et al. (Eds), Verlag, Erlangen, 1977, p. 167.

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Registry of Veterinary Pathology
Department of Veterinary Pathology

Results
AFIP Wednesday Slide Conference - No. 16
30 January 1980

Case I - 79-226 (AFIP 1714911).

History: Four gilts out of a herd of 120 were showing thick, inspissated vaginal discharge, anorexia and pyrexia (106° F.).

Laboratory Results: Bacterial cultures produced a pure growth of Staphylococcus aureus.

Contributor's Diagnosis & Comments: Necrotizing pyogranulomatous endometritis due to staphylococcal organisms. Gross necropsy findings revealed a very thickened mucosa with reduction of luminal diameter. The surface contained grey, friable, necrotic tissue up to 0.5 cm thick, with numerous pinpoint yellow foci. Uterine lymphadenopathy was prominent. Histologically, there are numerous pyogranulomatous foci containing Gram-positive cocci.

Attendees' Diagnoses & Comments:
Morphologic Diagnosis: Metritis, suppurative, diffuse, severe, uterus.
Etiologic Diagnosis: Septic metritis associated with coccal bacteria.
Etiology: Staphylococcus aureus.
Comments: The principle inflammatory cell was the neutrophil accompanied by areas of necrosis and endometrial ulceration. Extension of inflammatory process into the myometrium was present in most sections warranting a diagnosis of metritis. Participants reported numerous bacterial colonies compatible with staphylococci within the lesion.

Contributor: New Bolton Center, School of Veterinary Medicine, University of Pennsylvania, Kennett Square, R.D. 1, PA 19348.

References:
1. Fennestad, K. L., Stoulback, P., et al.: Staphylococcus aureus as a cause of reproductive failure and so-called Actinomycosis in swine. Nord Vet Med 7: 929-947, 1955.
2. Thorne, H., Nilsson, P. O.: Staphylococcus aureus as the cause of abortion in swine. Acta Vet Scand 2: 311, 1961.

Case II - PCSD-28097 (AFIP 1669223).

History: A 13-year-old, intact female poodle was presented to a veterinarian because of a mass in a mammary gland. Thoracic radiographs were unremarkable, but abdominal radiographs suggested the presence of a mass in the pelvic cavity. Surgical specimens are presented.

Contributor's Diagnosis & Comments: 1) Granulosa cell tumor, ovary, canine. 2) Cystic endometrial hyperplasia, diffuse, marked, uterus, canine. The changes in the uterine endometrium are probably a result of steroidal hormone production by the ovarian neoplasm.

Attendees' Diagnoses & Comments:

Diagnoses: 1) Granulosa cell tumor, cystic, ovary, canine. 2) Hyperplasia, endometrial, cystic, diffuse, moderate, uterus, canine.

Comments: The attendees unanimously agreed with the contributor's diagnoses and comments. Some staff members commented that the uterine glands at this stage of the lesion were primarily in the cystic stage rather than hyperplastic. The polycystic nature of the tumor is noteworthy, but the cells lining the cystic spaces were distinctly endocrinoid and compatible with granulosa cells. Call-Exner bodies, a feature of this tumor in humans, were lacking in multiple sections of this canine tumor.

Contributor: Letterman Army Institute of Research, Presidio of San Francisco, Calif. 94129.

References:

1. Dow, C.: The cystic hyperplasia-pyometra complex in the bitch. J. Comp. Pathol 69: 237-250, 1959.

2. Dow, C.: Experimental reproduction of the cystic hyperplasia-pyometra complex in the bitch. J. Path Bact 78: 267-278, 1959.

3. Norris, H. J., Garner, F. M., et al.: Comparative pathology of ovarian neoplasms. IV. Gonadal stromal tumors of canine species. J. Comp. Pathol 80: 399-405, 1970.

4. Nielsen, S. W., Misdorp, W., et al.: International Histological Classificataion of Tumors of Domestic Animals. XV. Tumors of the ovary. Bull. WHO 53: 203-215, 1976.

5. McEntee, K., Nielsen, S. W.: International Histological Classification of Tumors of Domestic Animals. XVI. Tumors of the female genital tract. Bull. WHO 53: 217-226, 1976.

Case III - 69-457 (AFIP 1700620).

History: Section of a surgically excised, pear-shaped vaginal polyp which was slightly protruding between the vulvar lips of an adult rhesus monkey.

Contributor's Diagnosis & Comments: Endometriosis. This rhesus monkey also had a palpable abdominal mass. Microscopically, the growth consisted of endometrial tissues, glandular and stromal, in the lamina propria of the vagina. Individual endometrial cells were tall columnar. Cystically dilated endometrial glands contained pink-staining precipitate. The stroma was highly vascularized and hemorrhagic. A large part of the overlying mucosa was eroded.

Attendees' Diagnosis & Comments:

Diagnosis: Endometriosis, vagina.

Comments: Attendees unanimously agreed with the contributor's diagnosis. There was considerable variation in morphology from slide to slide. Most participants reported the presence of both cellular elements necessary to make the diagnosis of endometriosis: glandular and stromal cells. Considerable amounts of blood in the tissue sections often obscured histologic detail. The blood may represent trauma to the vaginal polyp or, that the macaque was menstruating, or both.

Contributor: Bio-Dynamics, Inc., Mettlers Road, East Millstone, NJ 08873.

References:
1. MacKenzie, W. F., Casey, H. W.: Animal model of human disease: Endometriosis. Am. J. Path. 80: 341-344, 1975.
2. Splitter, G. A., Kirk, J. H., et al.: Endometriosis in four irradiated rhesus monkeys. Vet. Path. 9: 249-262, 1972.
3. Folse, D. S., Stout, L. C.: Endometriosis in a baboon (Papio doguera). Lab. Anim. Sci. 28: 217-219, 1978.
4. MacKenzie, W. F., Splitter, G. A., et al.: Endometriosis in primates. Med. Primatol. 1: 288-297, 1972.
5. McClure, H. M., Ridley, J. H., et al.: Disseminated endometriosis in a rhesus monkey (Macaca mulatta): Histogenesis and possible relationship to irradiation exposure. J. Med. Assoc. Georgia 60: 11-13, 1971.

Case IV - 76-326 (AFIP 1713950).

History: Incidental finding in a yellow, 196-day-old female mouse weighing 57.6 grams. A single, hard, greyish mass measuring approximately 2 mm was noted in association with the ovary.

Contributor's Diagnosis & Comments: Teratoma of the ovary. The mass consisted of a large quantity of keratin and lesser amounts of ciliated columnar epithelium and neural tissue. Several poorly developed hair follicles and hair shafts were found in a few sections. The yellow mouse is an obese mouse, thus the high weight.

Attendees' Diagnosis & Comments:

Diagnosis: Teratoma, didermal, ovary, mouse.

Comments: Participants agreed with the contributor's diagnosis and histologic observations. By definition, however, the teratoma should contain tissue elements representative of all three major germ layers of the embryo. Consequently, some staff members preferred the additional descriptor "didermic" in view of the apparent absence of tissues distinctly of mesodermal origin (i.e.: bone, muscle, cartilage, fat, etc.).

Contributor: Pathology Services Project, National Center for Toxicological Research, University of Arkansas for Medical Sciences, Jefferson, AR 72079.

Reference:

Squire, R. A., Goodman, D. G., et al.: Tumors. In: Pathology of Laboratory Animals, Vol. II, K. Benirschke, F. M. Garner, et al. (editors), Springer-Verlag, New York, 1978, pp 1179-1180, 1217.

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Results
AFIP Wednesday Slide Conference - No. 17
6 February 1980

Case I - C78-167 (AFIP 1718315).

History: A firm fibrous growth had been present on the right front pastern of a 10-year-old Tennessee Walker stallion for 3 years before surgical removal. The mass increased and decreased in size several times during the 3 years, but with no apparent seasonal predilection.

Contributor's Diagnosis & Comments: 1)Epidermal hyperplasia and acanthosis, diffuse, severe, with ballooning degeneration, skin. 2) Dermatitis, acute, moderate, skin. The epidermal lesions are compatible with infectious verrucae (warts) though the site of the lesion is very unusual. There was no clear evidence for viral inclusion bodies to support the morphologic impression.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Dermatitis, chronic-active, with pseudoepitheliomatous hyperplasia, severe, skin.

Comment: The staff considered the deep rete pegs with anastomosing processes to be a reactive epithelial hyperplasia secondary to chronic inflammation. Squamous papilloma and infectious verrucae were also considered in the differential. Some attendees speculated on the possibility of illicit iatrogenic trauma to the pastern of this Tennessee Walker.

Contributor: Division of Comparative Pathology, University of Florida, Box J-145, JHMHC, Gainesville, FL 32610.

Case II - 79/700 (AFIP 1718319).

Ten of 30 adult pigs died during a 5-day period showing signs of septicemia. Many of the pigs had extensive proliferative skin lesions.

Laboratory Results: Salmonella typhimurium was recovered in heavy pure growth from heart blood, liver and kidney of this sow. Poxvirus was demonstrated in these lesions by electron microscopy.

Contributor's Diagnoses & Comments: Swine pox, severe, with Demodex sp. Numerous intracytoplasmic inclusions are present within the hyperplastic epithelium. By electron microscopy, poxviruses were demonstrated.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Dermatitis, proliferative and necrotizing, focally extensive, skin. 2) Vasculitis, necrotizing, with thrombosis, acute, multifocal, moderate to severe, subcutis. 3) Folliculitis, parasitic, multifocal, moderate, skin.

Etiologic Diagnoses: 1) Swine pox dermatitis.
2) Bacterial vasculitis.
3) Demodecosis.

Etiologies: 1) Swine poxvirus.
2) Bacillary bacteria.
3) Demodex phylloides.

Comments: Attendees agreed with the contributor's diagnoses. The epidermal proliferation was distinct and well delineated. While there was variation in microslides, most sections clearly demonstrated the intracytoplasmic inclusion and ballooning degeneration of cells. The vascular thrombosis was associated with uniform populations of bacilli as opposed to the mixed population of the epidermis. Participants considered Erysipelothrix insidiosa, Pasteurella multocida, Salmonella spp, and Hemophilus parahemolyticus as possible etiologic agents of the septicemia. Most microslides demonstrated markedly dilated follicles filled with Demodex mites.

Contributor: Regional Veterinary Laboratory, Department of Agriculture, Wollongbar, New South Wales, Australia, 2480.

References:

1. Miller, R. B., Olson, L. D.: Epizootic of concurrent cutaneous streptococcal abscesses and swinepox in a herd of swine. JAVMA 172: 676-680, 1978.
2. Meyer, R. C., Conroy, J. D.: Experimental swinepox in gnotobiotic piglets. Res. Vet. Sci. 13: 334-338, 1972.
3. Wilcock, B. P., Armstrong, C. H., et al.: The significance of serotype in the clinical and pathological features of naturally occurring porcine salmonellosis. Can. J. Comp. Med. 40: 80-88, 1976.
4. Nordstoga, K.: Porcine salmonellosis. I. Gross and microscopic changes in experimentally infected animals. Acta Vet. Scand. 11: 361-369, 1970.
5. Lawson, G. H. K., Dow, C.: Porcine salmonellosis. J. Comp. Path. 76: 363-371, 1966.

Case III - 78-897 (AFIP 1713991).

History: This mass was removed from the lower lid of a 6-year-old female domestic short-haired cat. The cat had a history of acne on the chin. The mass had been present for 2 weeks prior to removal.

Contributor's Diagnosis & Comments: Dermatitis, necrotizing, chronic-active, diffuse, moderate, lip; consistent with a diagnosis of feline granuloma complex. Collagen necrosis, giant cells, macrophages and eosinophils are present in the mass.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Chelitis, necrotizing, eosinophilic-granulomatous, diffuse, severe, lip.

Disease Syndrome: Feline eosinophilic granuloma complex. ("rodent ulcer").

Comments: Participants unanimously agreed with the contributor's diagnosis and comments. Of the three main categories within the feline eosinophilic granuloma complex, this lesion meets the criteria of the eosinophilic ulcer (Scott, 1975).

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

Reference:

Scott, D. W.: Observations on the eosinophilic granuloma complex in cats. J. Am. Anim. Hosp. Assoc. 11: 261-270, 1975.

Case IV - S79-1121 (AFIP 1713992).

An 11-year-old castrated male Persian cat was presented on 25 March for examination of 3 slightly firm, raised, non-ulcerated, well-circumscribed skin masses over the left foreleg and left thorax. The masses were haired and non-pruritic. The cat returned 4 weeks later with many new 1-10 mm diameter masses all over the body. Two masses were ulcerated, reddened and glistening. A biopsy stained with the PAS reaction is presented.

Laboratory Results: (Antemortem clinical laboratory findings)

Hemogram	21 Apr	15 May
PCV	28%*	20%*
WBC	2.5*	3.8*
Neutro's	55%	69%
Lympho's	33%	24%
Mono's	9%**	2%
Eosino's	3%	5%

Urinalysis	May
Color	Dark yellow**
Sp. Grav.	1.036
pH	5
Protein	100 g/dl**
Glucose	Neg
Ketone	Neg
RBC/HPF	Packed**
WBC/HPF	1-5
Bacteria	None
Bence Jones	Neg

Chemistry	May
Tot. Prot.	10.8**
Albumin	2.6
BUN	31
Creatinine	2**
Amylase	355
Dir. Bilirub.	.03
SGOT i.u./L	31
SGPT i.u./L	36
LDH i.u./L	79
Alk Phos U/L	25**

Contributor's Diagnosis & Comments: Multiple plasmacytomas of skin and subcutis. At autopsy 2 months later, multiple myeloma was found with involvement of bone marrow, viscera and skin. Immunoelectrophoresis revealed the neoplastic plasma cells were producing IgG.

Attendees' Diagnosis & Comments:

Diagnosis: Plasmacytoma, skin.

Comments: Those clinical laboratory findings with a single asterisk (*) indicate those values that were abnormally low, and double asterisks (**) indicate those results that were abnormally elevated. These clinical values are highly suggestive of myeloma. Histologically, Mott cells, binucleate cells and bizarre mitotic figures were observed. Many neoplastic cells had round hyperchromatic nuclei which were eccentrically placed in basophilic cytoplasm, compatible with plasma cells. Methyl Green-Pyronin clearly demonstrated magenta-red material in the cytoplasm of many tumor cells, indicative of high content of RNA.

Contributor: Angell Memorial Animal Hospital, 350 South Huntington Ave., Boston, MA 02130.

References:

1. Farrow, B. R. H., Penny, R.: Multiple myeloma in a cat. JAVMA 158: 606-611, 1971.

2. Jarrett, W. F. H., Mackey, L. J.: International Histological Classification of Tumors of Domestic Animals. II. Neoplastic disease of the hematopoietic and lymphoid tissues. Bull. WHO 50: 21-34, 1974.

3. Shull, R. M., Osborne, C. A., et al.: Serum hyperviscosity associated with IgA multiple myeloma in two dogs. JAAHA 14: 58-70, 1978.

4. Osborne, C. A., Perman, V. et al.: Multiple myeloma in the dog. JAVMA 153: 1300-1317, 1968.
5. Cornelius, C. E., Goodbury, R. F., et al.: Plasma cell myelomatosis in a horse. Cornell Vet. 49: 478-493, 1959.
6. Pascal, R. R.: Plasma cell myeloma in the brain of a rabbit. Cornell Vet. 51: 528-535, 1961.

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Results
AFIP Wednesday Slide Conference - No. 18
13 February 1980

Case I - 18650 (AFIP 1667961):

History: Tissue is from a large cauliflower-like lesion involving the anterior third of the left mandibular ramus of a yearling male grizzly bear. The bear was thin and approximately half the size of his twin brother.

Contributor's Diagnosis & Comments: Odontogenic myxofibroma. This well-defined mass involved the anterior third of the left mandible and the cross sectional area of the mass exceeded a corresponding area of the right mandible by 2 to 3 times. Radiographs revealed an expansile lesion with central radiolucent areas and peripheral radiodense spicules. All premolar teeth were displaced with erosion of the anterior root of the first molar. The histology of the mass varied considerably. In the most predominant part of the lesion were bundles and sheets of myxomatous tissue. In some sections myxomatous tissue was found associated closely with or originating from mesenchymal cells of the tooth bud pulp. Other neoplasms considered in the differential diagnosis included adamantinoma (ameloblastoma), hamartoma and ameloblastic fibroma.

Attendees' Diagnoses & Comments:

Diagnosis: Odontogenic tumor with fibromatous and myxomatous components.

Comments: On the basis of the single section of this large tumor, the lesion is interpreted as an odontogenic process possessing two mesenchymal components (fibromatous and myxomatous) and an odontogenic epithelial component. Within the mesenchymal components were cords and islands representative of odontogenic epithelium, characterized by cords and rows of epithelial cells on distinct basement membranes. Spindle cells were located centrally in some cords. Participants varied in their interpretation of cords of odontogenic epithelium. Some considered the epithelium to be a proliferation of dental lamina normally found during odontogenesis; others considered the epithelium to be a hamartomatous overgrowth of odontogenic epithelium; and finally, some participants considered the cords to be the neoplastic epithelial component of an odontogenic tumor. Based on the supplemental clinical and radiographic information, an odontogenic neoplasm was favored by most participants. Of the odontogenic tumors described in domestic animals,[1] the odontogenic myxofibroma would be most compatible with this lesion. Odontogenic fibromas and myxofibroma are apparently very rare tumors in both man and animals.[4]

Contributor: Marsh Laboratory, Montana State University, Bozeman, MT 59717.

References:

1. Head, K. W.: International histological classification of tumors of domestic animals. XI. Tumors of the upper alimentary tract. Bull WHO 53: 145-166, 1976.

2. Effron, M., Griner, L. et al.: Nature and rate of neoplasia found in captive wild mammals, birds and reptiles at necropsy. J Natl Cancer Inst 59: 185-198, 1977.

3. Bernier, J. L.: Tumors of the odontogenic apparatus and jaws. Atlas of Tumor Pathology, 1960, Fascicle 102, Armed Forces Institute of Pathology, Washington, DC, pp 24-60.
4. Gorlin, R. J. and Meskin, L. H. et al.: Odontogenic tumors in man and animals: Pathologic classification and clinical behavior. A review. Ann NY Acad Sci 108: 722-771, 1963.

Case II - 78-291 (AFIP 1666499):

History: A 10-year-old male sloth bear (Melursus ursinus) that was born and exhibited at the National Zoo developed ascites and died one week later with jaundice.

Contributor's Diagnosis & Comments: Adenocarcinoma, metastatic to small intestine from the extrahepatic bile ducts. The tumor obstructed and appeared to arise from the cystic duct of the gallbladder. It was very scirrhous in some areas and produced mucin as confirmed by special stains. It had spread extensively into the liver parenchyma around the gallbladder and metastasized to most all peritoneal surfaces, the mesenteric lymph nodes and the lung. Tumors of the gallbladder and extrahepatic biliary tract have been reported in other bears and a cluster (three cases) occurred in sloth bears at the San Diego Zoo in the early 1960's.

Attendees' Diagnosis & Comments:

Diagnosis: Adenocarcinoma, serosal surface, small intestine, possibly metastatic, sloth bear.

Comments: Most participants noted the two cellular components of the tumor, the tubuloalveolar epithelium and the desmoplastic or scirrhous component. The tubuloalveolar pattern of the neoplastic epithelial cells, was suggestive of duct origin, but intestinal mucosal origin also had to be considered.

Contributor: National Zoological Park, Washington, DC 20008.

References:

1. Dorn, C. R.: Biliary and hepatic carcinomas in bears at the San Diego Zoological Gardens. Nature 202: 513-514, 1964.
2. Effron, M., Griner, L. et al.: Nature and rate of neoplasia found in captive wild mammals, birds and reptiles at necropsy. J Natl Cancer Inst 59: 185-198, 1977.

Case III - 79-94-3 (AFIP 1714919):

History: Tissue from a 6-year-old, male, yellow-backed duiker (Cephalophus sylvicultor) that died 48 hours after being transported by air in a small crate. Serum chemistries performed in the agonal antemortem period were as follows:

Serum Chemistries	a.m. 14 March	p.m. 14 March	a.m. 15 March
BUN	49 mg/dl	53 mg/dl	68 mg/dl
Creatinine	1.7 mg/dl	1.7 mg/dl	3.6 mg/dl
LDH	-	24,000 i.u.	36,550 i.u.
SGOT	-	35,700 i.u.	49,950 i.u.
CPK	-	851,000 i.u.	1,020,000 i.u.

Contributor's Diagnosis & Comments: Exertional rhabdomyolysis (capture myopathy). Muscles of the pectoral limbs and pelvic girdle showed changes that ranged from loss of cross striations to hyalinization and frank necrosis of myofibrils. There was also similar but milder involvement of skeletal muscles elsewhere, plus diaphragm and tongue muscles. There were degenerative changes in the myocardium. Acute tubular necrosis with numerous hyaline and cellular cast formations, as well as a mild acute diffuse pneumonia with hyaline membrane formation was also seen. The significance of the latter change is not known and had not been described previously in this condition.

Attendees' Diagnoses & Comments:
Morphologic Diagnosis: Rhabdomyolysis, acute, diffuse, severe, skeletal muscle.
Syndrome: Exertional rhabdomyolysis (capture myopathy).
Comment: Attendees unanimously agreed with the contributor's diagnosis. In the clinical pathological tests, all serum enzymes were considered massively elevated and highly suggestive of massive muscular and/or neuromuscular insult. Attendees compared the disease syndrome to crushing-trauma syndrome of man and so-called "Monday morning sickness" of draft horses.

Contributor: National Zoological Park, Washington, DC 20008.

Reference:
Bartsch, R. C., McConnell, E. E., et al.: A review of exertional rhabdomyolysis in wild and domestic animals and man. Vet Path 14: 314-324, 1977.

Case IV - RA7-6043 (AFIP 1668788):
History: Tissue from an adult coyote trapped during a wildlife study in Louisiana.

Contributor's Diagnosis & Comments: *Capillaria hepatica* infection. Twenty-one of 71 coyotes were infected, but none of 31 red foxes or 20 gray foxes trapped from the same areas were infected with *Capillaria hepatica*. The infection was more frequent in older coyotes. The inflammatory response to the ova was more severe in this liver than was seen in most infected animals.

Attendees' Diagnoses & Comments:
Morphologic Diagnoses: 1) Granulomas, parasitic, multifocal, moderate, liver. 2) Microfilaremia, sinusoidal, diffuse, mild, liver.
Etiologic Diagnoses: 1) Hepatic capillariasis. 2) Microfilaremia.
Etiologies: 1) *Capillaria hepatica*. 2) Compatible with *Dirofilaria immitis*.
Comments: The presence of thick, brown, double operculated eggs in the hepatic parenchyma is considered highly suggestive, if not pathognomonic, of *Capillaria hepatica*. Most attendees ascribed the microfilaria in the hepatic sinusoids as being consistent with canine heartworm infestation (*D. immitis*) but also considered *Dipetalonema* spp.

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

References:

1. Crowell, W. A., Klei, T. R., et al.: *Capillaria hepatica* infection in coyotes of Louisiana. JAVMA 173: 1171-1172, 1978.

2. Wobeser, G., Rock, T. W.: *Capillaria hepatica* (Nemstoda: Trichuridae) in a coyote (*Canis latrans*). J Wildlife Dis 9: 225-226, 1973.

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Results
AFIP Wednesday Slide Conference - No. 19
20 February 1980

Case I - (AFIP 1642713).

History: A 5-month old male Labrador-German shepherd cross was presented for an upper respiratory tract infection manifested by a bilateral mucopurulent discharge from the nose and eyes. A diarrhea of 3 days duration with moderate dehydration was also noted.

Contributor's Diagnosis & Comments: Canine distemper. Although somewhat variable from section to section, the majority of the lesions are in the retina, which consists of folds, mild retinitis, central chromatolysis of the retinal ganglion cells and pigment migration from the pigmented retinal epithelium into the photoreceptor retina. In addition, a mild iridocyclitis is present. Although not present in the submitted slides, eosinophilic intracytoplasmic inclusions were found in the glandular epithelial cells of an accessory lacrimal gland and transitional epithelium of the urinary bladder.

Retinitis and optic neuritis occurs frequently in canine distemper. As an early response to the infection, the retinal ganglion cells degenerate and the pigmented epithelium proliferates and degranulates into the retina. Acute retinitis is characterized by congestion and edema of the optic fiber layer and inner plexiform layer. Perivascular lymphocytic cuffing occurs in some, but not all, cases. The extent and severity of the retinitis varies from focal to almost complete destruction. The focal lesions, as in this case, may affect only the layer of rods and cones, frequently causing the retina to detach focally, resulting in folds suggestive of a rosette. In other cases, not represented in these slides, the atrophy may be severe enough to lead to loss of layer organization, with only remnants of the inner layer remaining.

Intranuclear inclusions may occur in the retinal glia, but more often are observed in the epithelium or, as in this case, a lacrimal gland.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: Retinitis, with marked degenerative changes, subacute, multifocal, retina.

Etiology: Compatible with lesions produced by the canine distemper virus (Paramyxoviridae: Morbillivirus).

Comments: Participants unanimously agreed with the contributor's diagnosis and comments. While the lesions are highly suggestive of canine distemper infection, changes such as retinal folds, central chromatolysis, and lymphocytic perivascular cuffs are non-specific and could occur with other processes.

Contributor: Registry of Veterinary Pathology, AFIP. (Case contributed by Department of Ophthalmology, College of Veterinary Medicine, University of Illinois, Urbana, IL 61805).

Reference:

Saunders, L. Z., Rubin, L. F.: Ophthalmic Pathology of Animals, 1975. S. Karger, Basel, pp 144-145.

Case II - 28992 (AFIP 1693566).

History: Tissue from a 2½-year-old male Sprague-Dawley rat presented for necropsy because of a 3cm X 3cm ulcerated mass in the right mandibular region. This rat was never used experimentally. The mass had a balloon-like consistency and sanguinous fluid mixed with yellowish flakes was aspirated.

Contributor's Diagnosis: Carcinoma, probably of auditory sebaceous gland (Zymbol's gland), right mandibular area, rat.

Attendees' Diagnoses & Comments:

Diagnosis: Carcinoma, with predominantly squamous differentiation, Zymbol's gland.

Comments: Of the tumors of the auditory sebaceous glands (Zymbol's gland) described in rats (Pliss, 1973), this tumor most closely resembles a squamous cell carcinoma with keratinization. Most attendees also reported seeing isolated cells with clear cytoplasm or multiple clear vacuoles resembling sebaceous glands. The majority of spontaneous tumors of Zymbol's gland tend to demonstrate squamous cell differentiation.

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

Reference:

Pliss, G. B.: Tumours of the auditory sebaceous glands. In: Pathology of Tumours in Laboratory Animals. Vol. I. Tumours of the rat, 1973. VS Turusov, ed. WHO/IARC Scientific Pub. No. 5, Lyon, pp 23-30.

Case III - (AFIP 1599982).

History: A collie, age and sex unspecified, was presented with a massively enlarged and painful eye. The contralateral eye appeared normal.

Contributor's Diagnoses & Comments: Adenocarcinoma, ciliary body, nonpigmented epithelium, eye. There is extensive invasion of the ciliary body, cornea, iris and sclera. The lens capsule has multiple fractures and there is severe diffuse liquifactive and degenerative change of the cortex. Massive acute subretinal hemorrhage attended by total retinal detachment with retinal adhesions to the posterior lens capsule are also present.

This tumor arises from the iridociliary epithelium and is characterized by pleomorphic cells that tend to form solid sheets, gland-like structures and papillary forms. In the dog, pigmented tumors of the ciliary body epithelium are much less common than nonpigmented tumors. Even in the tumors arising from the nonpigmented epithelium, it is common to find variable degrees of pigmentation due to preexisting melanocytes which degranulate into the surrounding neoplastic growth. This tumor is locally aggressive but has little tendency to metastasize.

Differential diagnosis should include metastatic adenocarcinomas.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Adenocarcinoma, ciliary body, nonpigmented epithelium, eye. 2) Detachment and degeneration, retinal, acute, diffuse, severe, eye. 3) Hemorrhage, vitreal, acute, diffuse, severe, eye. 4) Episcleritis with anterior synechia, subacute, focally extensive, eye. 5) Cataractous change, liquifactive, severe, lens, eye.

Comments: Participants considered the changes in the retina, vitreous, lens, iris, sclera and cornea to be secondary to the neoplasm. In addition, several participants reported degeneration of the ganglion and inner nuclear cell layers of the retina to be compatible with early lesions of glaucoma, which was probably induced by the tumor occluding the filtration angle.

Contributor: Registry of Veterinary Pathology, AFIP. (Case contributed to AFIP by Dr. Seth Koch, Penn-Daw Animal Hospital, 6223 Richmond Highway, Alexandria VA 22309).

Case IV - AFIP 1581246.

History: An 18-month-old Standardbred filly, blind in the left eye, was referred for ophthalmic examination. The left eye was inflamed and congested. The left pupil was fixed and mid-dilated. The left eye lacked both direct and consensual reactions. Although the vitreous of the left eye was slightly hazy, the retina was folded inwardly and a solid pink to grey-white lobular mass occupied about one-third of the posterior segment. The right eye was clinically normal. (2x3 oversize microslide)

Contributor's Diagnosis & Comments: Malignant medulloepithelioma, optic nerve, left eye. The tumor is composed of pleomorphic, columnar, neural-like cells forming cords, tubules, clefts and occasional rosettes. The stratified sheets of cells have some degree of polarity. The basal segments of the cords rest on prominent thin bands of hyalinized collagenous stroma resembling very thick basement membranes. The apical cells lining the central clefts and rosettes show a definite external limiting membrane. The collagenous stroma is moderately PAS and alcian blue positive (and hyaluronidase sensitive), however, acid mucopolysaccharide material is not present within the lumina of the rosettes. The cells are nonpigmented with moderate amounts of eosinophilic cytoplasm. The nuclei are vesicular and pleomorphic, with prominent limiting membranes, peripheral margination of chromatin and occasional large nucleoli. Mitotic figures are moderate to numerous. Invasion into the optic nerve, the mitotic rate and the cellular pleomorphism warrant a malignant designation. These are rare tumors occasionally reported in dogs, horses and a single case in a goldfish.

Attendees' Diagnosis & Comments:

Diagnosis: 1) Malignant medulloepithelioma, optic nerve, left eye. 2) Detachment and degeneration, retinal, focally extensive, left eye.

Comments: Attendees agreed with the contributor's diagnosis and comments.

Contributor: Registry of Ophthalmic Pathology, AFIP. (Case contributed by Department of Veterinary Science, University of Kentucky, Lexington, KY).

References:

1. Eagle, R. C., Font, R. L., et al: Malignant medulloepithelioma of the optic nerve in a horse. Vet. Pathol. 15: 588-594, 1978.

2. Bistner, S. I.: Medulloepithelioma of the iris and ciliary body in a horse. Cornell Vet. 64: 588-595, 1974.

3. Lahar, M., Albert, D. M.: Medulloepithelioma of the ciliary body in the goldfish. Vet. Pathol. 15: 208-212, 1978.

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Results
AFIP Wednesday Slide Conference - No. 20
5 March 1980

Case I - N78-384 (AFIP 1667920).

History: An incidental finding in a 6-week-old, female BALB/c mouse which was euthanized for quality control procedures. There were diffuse irregular, white thickenings of the epicardium primarily over the right ventricle.

Contributor's Diagnosis & Comments: Dystrophic cardiac calcinosis. This condition has been observed in a number of young BALB/c mice from the same supplier, and, as reported by Eaton, Custer, et al., is confined to the epicardium in this strain. Concomnitant calcinosis in other organs has not been observed.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Mineralization, epicardial, segmental, moderate, right ventricle, heart, BALB/c, mouse.

Comments: Attendees concurred with the contributor's diagnosis. This lesion was probably inconsequential, but in some cases the mineralization reaches such extensive proportions that it may lead to congestive heart failure. Multiple strains of mice have variable sites of involvement: in BALB/c mineralization is usually confined to the epicardium; in C3H it is confined to the myocardium, and DBA/2 there may be concurrent distribution [Eaton, Custer, et al., 1978].

Contributor: School of Aerospace Medicine [USAF/SAM VSP], Brooks AFB, TX 78235.

Reference:

Eaton, G. J., Custer, R. P., et al.: Dystrophic cardiac calsinosis in mice. Genetic, hormonal and dietary influences. Am. J. Pathol. 90: 173-182, 1978.

Case II - C79-02 (AFIP 1712506).

History: 1-year-old male New Zealand White rabbit stopped eating and showed difficulty breathing. Within 24 hours the animal died.

Laboratory Results: On necropsy examination, the pericardium was thickened, the pericardial sac contained a thick fibrinopurulent fluid and the myocardium contained multiple yellow-white foci. Pododermatitis involved both hindlimbs. Culture of the pericardial fluid yielded pure colonies of hemolytic Staphylococcus aureus coagulase-positive organisms.

Contributor's Diagnosis & Comments: Staphylococcal pericarditis and myocarditis. The source of the organism may have been the pododermatitis. A Gram stain of the myocardial lesion showed Gram-positive cocci within the necrotic foci.

Attendees' Diagnosis & Comments:

Morphological Diagnosis: Epicarditis and myocarditis, fibrinopurulent, septic diffuse, severe, heart.

Etiologic Diagnosis: Bacterial epicarditis and myocarditis.

Etiology: Compatible with Staphlococcus aureus and/or Pasterella multocida.

Comment: Most participants considered the grape-like clusters of bacterial colonies to be consistent with staphylococcal organisms. Heterophils and fibrinous exudation were the predominant feature, with some microslides also demonstrating septic fibrinous thrombi. Some histologic sections also demonstrated mild fatty infiltration of atrio-ventricular valves.

Contributor: Toxicology Section, Health, Safety and Human Factors Laboratory, Building 320 Kodak Park, Eastman Kodak Co., Rochester, NY 14650.

References:

1. Carlton, W. W., Hunt, R. D.: Bacterial Diseases. In Pathology of Laboratory Animals, Benirschke, K., Garner, F. M., et al., Eds., Springer-Verlag, New York, 1979, pp 1451-1453.

2. Snyder, S. B., Fox J. G., et al.: Disseminated staphylococcal disease in laboratory rabbits (Oryctolagus cuniculus). Lab. Anim. Sci. 26: 86-89, 1976.

Case III - 75-1956 (AFIP 1665272).

History: In a teratogenesis study, pregnant rabbits were treated with a drug on post-insemination days 6 through 18. Many rabbits aborted on or before the 25th day of gestation. Death generally occurred 1 to 3 days later. Prior to death rabbits had frothy fluid from the nostrils and mouth.

Laboratory Results: Serum lactic acid dehydrogenase and creatinine phosphokinase levels were elevated in the treated animals. Necropsy revealed hydrothorax, hydropericardium, pulmonary congestion and edema, mottling of the heart, subepicardial grey-white areas and hepatic centrilobular congestion.

Contributor's Diagnosis & Comments: Adriamycin-induced myocardial degeneration. Adriamycin is a potent chemotherapeutic agent used to treat some types of neoplasms in man. Cardiotoxicity is an unfortunate side-effect that occurs in experimental animals, such as the rabbit, and has also been reported in man.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Necrosis, myocardial, acute, multifocal, moderate, heart.

Etiologic Diagnosis: Anthracycline-antibiotic-induced cardiomyopathy.

Etiology: Consistent with adriamycin intoxication.

Comments: Participants considered adriamycin to be the most likely causative agent, but could not rule out other related glycosidic anthracycline antibiotics such as daunomycin. In the presence of myocytolysis, participants felt the dense eosinophilic regions of some myofibers were true lesions typical of hyper-contraction bands.[1] Attendees discussed the two primary but distinctly separate cytotoxic effects of adriamycin on tumor cells and cardiac tissue. In tumor cells adriamycin is believed to exert part of its toxic effect through intercalation with cell DNA causing fragmentation of DNA and inhibition of DNA synthesis[2] (mainly, the early S-phase).[3,4] In non-dividing cardiac myocytes, however, the toxic effect of

adriamycin appears linked to free radical formation by altered lipid peroxidation[2] and by inhibition of sodium-potassium-activated adenosine triphosphatase (Na-K-activated ATPase).[5] Interestingly, adriamycin causes a nearly complete inhibition of potassium ion reaccumulation in cardiac myocytes while failing to effect sodium ion extrusion.[5] Subsequent imbalances in water content are probably evidenced histologically by the vacuolar myocytolysis.[6]

Contributor: Pathology Division, Dow Chemical Co., 9550 North Zionsville Road, P. O. Box 68511, Indianapolis, IN 46268.

References:

1. Van Vleet, J. F., Ferrans, V. J., et al.: Ultrastructural alteration in nutritional cardiomyopathy of selenium-vitamin E deficient swine. I. Fiber lesions. Lab. Invest. 37: 188-200, 1977.
2. Myers, C. E., McGuire, W. P., et al.: Adriamycin: The role of lipid peroxidation in cardiac toxicity and tumor response. Science 197: 165-167, 1977.
3. Lee, Y. C., Byfield, J. E.: Induction of DNA degradation in vivo by adriamycin. J. Natl. Cancer Inst. 57: 221-224, 1976.
4. Barrance, S. C., Gerner, E. W., et al.: Survival and cell kinetics effects of adriamycin on mammalian cells. Cancer Res. 33: 11-16, 1973.
5. Gosalvez, M., van Rossum, G. D. V., et al.: Inhibition of sodium-potassium-activated adenosine 5'-triphosphatase and ion transport by adriamycin. Cancer Res. 39: 257-261, 1979.
6. Olson, H. M., Young, D. M., et al.: Electrolyte and morphologic alteration of myocardium in adriamycin-treated rabbits. Am. J. Pathol. 77: 439-450, 1974.
7. Thompson, D. J., Molello, J. A., et al.: Teratogenicity of adriamycin and daunomycin in the rat and rabbit. Teratol. 17: 151-158, 1978.
8. Jaenke, R. S.: An anthracycline antibiotic-induced cardiomyopathy in rabbits. Lab. Invest. 30: 292-304, 1974.
9. Jaenke, R. S.: Delayed and progressive myocardial lesions after adriamycin administration in the rabbit. Cancer Res. 36: 2958-2966, 1976.

Case IV - SW76-1095 (AFIP 1620680).

History: A 1-month-old male miniature swine was used in an atherosclerosis research project. The animal was on an experimental diet consisting of 15% cottonseed oil and 1.5% cholesterol. There were no symptoms prior to death.

Laboratory Results: Gross Pathology: There was diffuse pulmonary congestion and edema with disseminated hemorrhage. The pericardial sac contained 30-40cc of clotted blood. The myocardium had 3 irregular round hemorrhagic lesions 1-3 cm in diameter. The cut section of the right ventricle revealed diffuse white streaking. There were numerous focal areas of hemorrhage in the fundus of the stomach and duodenum. History: Diffuse edema and hyalinization of myocardial muscle fibers were present with coalescing foci of necrosis and mineralization. Mixed inflammatory cell response in the epicardium was present with the fibrin deposition.

Contributor's Diagnosis & Comments: Acute nutritional cardiomyopathy. The lesions are consistent with those observed in selenium-vitamin E deficiency of swine. This animal was 1 of 4 observed with similar lesions from the test group. The diet consisted of an abnormally high level of unsaturated fatty acids, and there was no supplementation with vitamin E to provide an increased level of anti-oxidant activity.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Necrosis, myocardial, with mineralization, acute to subacute, focally extensive, heart.

Etiology: Consistent with vitamin E-selenium deficiency.

Comments: Attendees concurred with the contributor's diagnosis. The principle lesion was that of myocardial necrosis with secondary inflammatory cell reaction.

Contributor: Comparative Pathology Section, Veterinary Resources Branch, Building 28A, National Institutes of Health, Bethesda, MD 20014.

References:

1. Van Vleet, J. F., Ferrans, V. J., et al.: Ultrastructural alteration in nutritional cardiomyopathy of selenium-vitamin E deficient swine. I. Fiber lesions. Lab. Invest. 37: 188-200, 1977.

2. Van Vleet, J. F.: Current knowledge of selenium-vitamin E deficiency in domestic animals. JAVMA 176: 321-325, 1980.

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Results
AFIP Wednesday Slide Conference - No. 21
19 March 1980

Case I - 28063 (AFIP 1669175).

History: A group of 29 Leghorn chicks was placed on an experimental ration of torula yeast, casein, cornstarch and stripped lard. At six weeks of age, 3 of 29 chicks showed staggering and incoordination.

Contributor's Diagnosis & Comments: Encephalomalacia, subacute, multifocal, marked, cerebellum, chicken; caused by vitamin E deficiency. At necropsy there were petechial hemorrhages and 1 mm indented grey area on the cerebellum. Hemorrhages also were present on the pectoral and thigh muscles. Feeding selenium at 0.1 ppm is reported to protect chicks against exudative diathesis associated with vitamin E deficiency.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Encephalomalacia, acute, focally disseminated, moderate, cerebellum, brain.

Etiologic Diagnosis: Consistent with cerebellar hypovitaminosis E.

Etiology compatible with vitamin E deficiency.

Comment: Attendees noted the selective vulnerability of the cerebellum in this disease. There was eosinophilic degeneration of Purkinje cells with a few empty basket cells, and focally extensive spongiform degeneration of folia. Several participants also noted vascular thromboses near the necrotic folia.

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

References:

1. Cheville, N. F.: The pathology of vitamin E deficiency in the chick. Path. Vet. 3: 208-225, 1966.

2. Adamstone, F. B.: Brain degeneration in young chicks reared on an iron-treated vitamin E-deficient ration. Arch. Pathol. 31: 603-612, 1941.

3. Adamstone, F. B.: Histologic comparison of the brains of vitamin A-deficient and vitamin E-deficient chicks. Arch. Pathol. 43: 301-312, 1947.

4. Hoekstra, W. G.: Biochemical function of selenium and its relation to vitamin E. Fed. Proc. 34: 2083-2089, 1975.

5. Combs, G. F., Noguchi, T. et al.: Mechanisms of action of selenium and vitamin E in protection of biological membranes. Fed. Proc. 34: 2090-2095, 1975.

6. Hill, C. H.: Interrelationships of selenium with other trace elements. Fed. Proc. 34: 2096-2100, 1975.

7. Van Vleet, J. F., Ferrans, V. J.: Ultrastructural changes in skeletal muscle of selenium-vitamin E-deficient chicks. Am. J. Vet. Res. 37: 1081-1089, 1976.

8. Van Vleet, J. F.: Current knowledge of selenium-vitamin E-deficiency in domestic animals. JAVMA 176: 321-325, 1980.

Case II - D70-5647 (AFIP 1718843).

History: A 2-1/2 month old male Nubian kid was presented with respiratory disease and posterior ataxia with intermittent paralysis. Temperature was 103.5 F. The kid was alert and responsive to external stimuli but progressed to quadraplegia over several days.

Contributor's Diagnoses & Comments:

Diagnoses: 1) Myelitis, nonsuppurative, regional (upper thoracic), malacic, severe. 2) Meningitis, nonsuppurative, spinal cord, regional, severe.

Comments: The lesions observed are compatible with those described for viral leukoencephalomyelitis of goats (VLG), except that in previously reported cases the primary central nervous system lesions were observed in the cervical and lumbar spinal cord and in the brain. Mild perivascular cuffs were noted in several cervical cord segments but were absent in posterior thoracic or lumbar cord segments. No lesions were noted in multiple brain sections. A patchy bronchopneumonia was also diagnosed. VLG affects kid goats ranging from 1-4 months of age. Clinical signs are unilateral or bilateral posterior paresis and/or ataxia that may progress to quadraplegia. The kids usually are afebrile, are alert and responsive to stimuli, and have good appetites. Infection appears to occur in utero or shortly after birth. CNS lesions are usually accompanied by a variable interstitial pneumonia often with a secondary bronchopneumonia. The disease can be transmitted to newborn kids by inoculation with 220 nm millipore filtrate of nervous tissue from a naturally infected goat. Bacteriologic isolations are negative. The causative agent is thought to be a virus, but isolation in tissue culture has produced no cytopathologic effect. Transmission electron microscopy has demonstrated the lesion of VLG to be one of primary demyelination without damage to the associated neuron. A single virus particle suggestive of the nucelocapsid-like material of a myxovirus was also observed. In many aspects, VLG lesions resemble those of post-infectious encephalomyelitis of man which is suspected of having an immunologic origin secondary to a viral infection.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Meningomyelitis, nonsuppurative, focally disseminated, severe, spinal cord.

Syndrome: Viral leukoencephalomyelitis of goats.

Comment: Many participants considered the inflammatory reaction to be granulomatous and have some features of canine granulomatous meningo-encephalomyelitis syndrome. There were lesions of the neuropil characterized by swollen axons and chromatolytic neurons, as well as vascular changes characterized by infiltrations of mononuclear inflammatory cells, resembling post-viral and/or experimental allergic encephalitides.

Contributor: Veterinary Diagnostic Laboratory, University of Illinois, P. O. Box U, Urbana, IL 61801.

References:

1. Cork, L. C., Hadlow, W. J. et al.: Infectious leucoencephalomyelitis of young goats. J. Inf. Dis. 129: 134-141, 1974.

2. Cork, L. C., Davis, W. C.: Ultrastructural features of viral leukoencephalomyelitis of goats. Lab. Invest. 32: 359-365, 1975.

3. Cork, L. C.: Differential diagnosis of viral leukoencephalomyelitis of goats. JAVMA 169: 1303-1306, 1976.
4. Crawford, T. B., Adams, D. S., et al.: Chronic arthritis in goats caused by a retrovirus. Science 207: 997-999, 1980.
5. Cork, L. D., Hadlow, W. J., et al.: Pathology of viral leukoencephalomyelitis of goats. Acta Neuropath. 29: 281-292, 1974.

Case III - 78-7585 (AFIP 1714908).

History: A pure-bred Charolais heifer, 6-months-old, was submitted for necropsy. The animal had been symptomatic for 2-3 weeks; clinical signs were stiff gait, stumbling, progressive incoordination, dog-sitting position and paresis of the hind limbs.

Contributor's Diagnosis & Comments: Progressive ataxia of Charolais cattle. Microscopic lesions were confined to the white matter of the brain, cerebellum and spinal cord. The changes were characterized by eosinophilic plaques which, in haematoxylin-phloxin-&-saffran stains (HPS), appear acellular, granular and fibrillar. These lesions are similar to those described in previous reports. This heifer was relatively young, and was, typically, a female. The disease was first noted in England and has since been recognized in Canada, New Zealand and, more recently, in France and the United States.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Myelinodystrophy (leucodystrophy), focally extensive, spinal cord.

Syndrome: Progressive Ataxia of Charolais.

Comment: Participants unanimously agreed with the contributor's diagnosis, but there was discussion of a proper morphologic diagnosis in this case. Dysmyelinogenesis was considered inappropriate inasmuch as the term implies pathogenesis, which cannot be adequately interrupted from a single microslide at the light microscopic level. Similarly, diagnoses using the terms degeneration or malacia tacitly imply there may have been normal myelin which has undergone changes, when it is uncertain whether normal myelin existed around the axons. Hence, myelinodystrophy is the preferred diagnosis, suggesting only the presence of unusual myelin. In addition to the circular eosinophilic fibrillar plaques, most microslides demonstrated subtle changes in glial cells; some astrocytes resembled early gemistocytes.

Contributor: Animal Pathology Laboratory, Quebec Department of Agriculture, St. Hyacinthe, Quebec J2S 7X9, Canada.

References:

1. Blakemore, W. F., Palmer, A. C. et al.: Progressive ataxia of Charolais cattle associated with disordered myelin. Acta Neuropath. 29: 127-139, 1974.
2. Bonisset, S., Daviaud, L. et al.: Un cas d'ataxie progressive chez un bovin Charolais. Neuvieme congres international, maladies du betail Paris tome 3: 1159-1161, 1976.
3. Palmer, A. C., Blakemore, W. F. et al.: Progressive ataxia of Charolais cattle associated with a myelin disorder. Vet. Rec. 91: 592-594, 1972.
4. Patton, C. S.: Progressive ataxia in Charolais cattle. Vet. Path. 14: 535-537, 1977.

Case IV - 77-17001 (AFIP 1656673).

History: A female 3-year-old cocker spaniel was presented in acute pain in the lumbo-sacral region and without patellar responses. The dog was euthanized.

Contributor's Diagnosis & Comments: Intervertebral disc protrusion. Hemorrhage and other degenerative changes seen in the spinal cord are consequences of prolapsed disc. In some histo slides all these alterations are not evident.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Hemorrhage, meningeal, acute, segmental, moderate to severe, with dysplaced intervertebral disc material, spinal cord.

Comment: Participants agreed with the contributor's gross diagnosis. Histologically the hemorrhage was confined to the ventral meninges with most slides demonstrating epidural intervertebral disc material. Within the spinal cord, there were acute changes such as axon swelling ("torpedo" formations), perivascular edema and hemorrhage and minimal infiltration of polymorphonuclear leucocytes. These changes are consistent with spinal cord trauma as may occur with acute prolapse of intervertebral discs.

Contributor: Rollins Animal Disease Diagnostic Laboratory, Box 12223, Raleigh, NC 27605.

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Results
Wednesday Slide Conference - No. 22
26 March 1980

Case I - 6277 (AFIP 1667566).
History: Tissue from a 2-year-old female Schnauzer.

Contributor's Diagnosis: Multifocal granulomatous meningoencephalitis, etiology Histoplasma capsulatum.

Attendees' Diagnoses & Comments:
Morphologic Diagnosis: Meningoencephalitis, granulomatous, multifocal, moderate, cerebrum.
Etiologic Diagnosis: Mycotic meningoencephalitis.
Etiology: Consistent with Histoplasma capsulatum
Comment: All attendees reported seeing numerous small, approximately 3 micron diameter spherical organisms within macrophages in the regions of granulomatous inflammation.

Contributor: Veterinary Diagnostic and Research Center, Murray State University, Hopkinsville, KY 42240.

Case II - 79-153 (AFIP 1711327).
History: A 2-year-old, spayed female dog was showing signs of weakness which progressed to seizures over a 10-day period.

Laboratory Results: Cerebral Spinal Fluid (CSF) contained 40 mg% protein and 90 cells per cubic millimeter.

Contributor's Diagnosis: Granulomatous encephalitis.

Attendees' Diagnoses & Comments:
Morphologic Diagnosis: Meningoencephalitis, granulomatous, multifocal, moderate, cerebellum and medulla.
Syndrome: Canine granulomatous meningoencephalomyelitis.
Comment: Participants noted the multifocal nature of the lesions. There were perivascular lymphocytic cuffs and foci of epithelioid cells in the neuropil. Staff members noted that this entity has had numerous names in the recent past, including primary reticulosis of the central nervous system, inflammatory reticulosis and perithelioma. Participants compared this multifocal lesion to some cases previously labeled primary reticulosis in which the lesion was generally single, large, invasive and expansile; the possibility of two forms of canine granulomatous meningoencephalomyelitis or two different diseases was mentioned.

Contributor: School of Veterinary Medicine, University of Pennsylvania, 3800 Spruce Street, Philadelphia, PA 19104.

References:
1. Cordy, D. R.: Canine granulomatous meningoencephalomyelitis. Vet. Path. 16: 325-333, 1979.
2. Koestner, A., Zeman, W.: Primary reticuloses of the central nervous system in dogs. Am. J. Vet. Res. 23: 381-393, 1962.
3. Braund, K. D., Vandevelde, M. et al.: Granulomatous meningoencephalomyelitis in six dogs. JAVMA 172: 1195-1200, 1978.
4. Fankhauser, R., Fatzer, R., et al.: Reticulosis of the central nervous system in dogs. Adv. Vet. Sci. Comp. Med. 16: 35-71, 1971.

Case III - J1765 (AFIP 1664174).

History: A 2-year-old female pug was presented with "fainting spells" which progressed to grand mal seizures. The dog died within several days of initial presentation.

Laboratory Results: At necropsy, the only gross lesions were pulmonary congestion and a "prune" sized, round, firm mass in the thoracic inlet.

Contributor's Diagnosis & Comments: Granulomatous meningitis due to Blastomyces dermatitidis. The thoracic mass was a lymph node with granulomatous lymphadenitis. Budding, yeast-like cells similar to those in the brain were present in the lymph node. Lesions, other than the noted congestion and edema, were not found in the lungs.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Meningitis, pyogranulomatous, diffuse, mild to moderate, cerebrum.

Etiologic Diagnosis: Meningeal cryptococcosis.

Etiology: Consistent with Cryptococcus neoformans.

Comments: Participants were unanimous in their morphologic diagnosis, however, mycotic agents were generally sparse on histosiides. A few slides demonstrated yeast forms lacking internal structure but were budding, thus indicating viability. The yeast forms were often variable in size and sometimes collapsed, features associated with cryptococcal rather than blastomycotic organisms. Phase contrast microscopy clearly demonstrated the organisms and their clear, thick mucinous capsules. A decolorized restained slide, using the mucicarmine technique, was positive for mucin capsules. Meningeal blastomycosis is considered very unusual in domestic animals, while meningitis is a common presentation of cryptococcosis. This case demonstrates that inflammatory reaction to cryptococcal organisms can be very intense. The difficulties encountered in the histologic identification of organisms in this case emphasize the importance of special staining and culturing procedures in providing a definitive diagnosis.

Contributor: Veterinary Diagnostic Center, University of Nebraska, Lincoln, NB 68583.

References:
1. Jungerman, P. F., Schwartzman, R. M.: Blastomycosis. In Veterinary Medical Mycology. Lea & Febiger, Philadelphia, 1972, pp 124-138.
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Case IV - 78353-13 (AFIP 1716390).

History: An aged, emaciated ewe was culled from a large flock of normal sheep. No clinical signs were apparent.

Contributor's Diagnosis & Comments: Periventricular lymphocytic encephalitis with necrosis; etiology: ovine progressive pneumonia virus. OPP virus was isolated from the lung; brain isolation was not attempted. Heavy lymphocytic infiltrates, characteristic of Ovine Progressive Pneumonia, were present in this ewe.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Chorioencephalitis, periventricular, nonsuppurative, diffuse, moderate, cerebrum.

Syndrome: Ovine Progressive Pneumonia.

Etiology: Compatible with ovine lentivirus (Ovine Progressive Pneumonia Virus).

Comment: Most participants also observed mild multiple foci of malacia in the periventricular white tracts. Characteristically, the inflammatory cells were predominantly lymphocytes.

Contributor: National Animal Disease Center, USDA/ARS, P. O. Box 70, Ames, IA 50010.

References:

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3. Cutlip, R. C., Jackson, T. A. et al.: Prevalence of Ovine Progressive Pneumonia in a sampling of cull sheep from Western and Midwestern United States. Am. J. Vet. Res. 38: 2091-2093, 1977.

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Results
AFIP Wednesday Slide Conference - No. 23
2 April 1980

Case I - 79-530 (AFIP 1714170).

History: A female Arabian foal, born to a mare that previously had a combined immunodeficient foal, had pneumonia for 3 weeks prior to death. Necropsy findings consisted of large consolidated nodules scattered throughout all lung lobes and numerous 0.5 to 1.0 cm ulcers throughout the ileum and colon.

Laboratory Results: Corynebacterium equi was isolated from the lung and liver.

Contributor's Diagnosis & Comments: Colitis, ulcerative and pyogranulomatous, due to Corynebacterium equi. C. equi is most often associated with respiratory disease in the foal. This animal did have large nodules of granulomatous pneumonia. There was no history of enteric signs. Gram-positive bacilli could be identified within submucosal macrophages. These lesions have been compared to the lesions of Whipple's disease in man which is characterized by an extensive cellular infiltrate into the submucosa and lamina propria of macrophages filled with Gram-positive bacilli. In Whipple's disease, focal accumulations of macrophages have been found in other organs such as liver, lung and spleen.

Attendees' Diagnoses & Comments:
Morphologic Diagnosis: Enterocolitis, pyogranulomatous, multifocal, moderate to severe, ileum and colon.
Etiologic Diagnosis: Consistent with Corynebacterial enterocolitis.
Etiology: Compatible with Corynebacterium equi.
Comments: Some histoslides contained sections of small intestine while others had only colon. Most participants considered the history and necropsy findings to be very suggestive of Corynebacterial pneumonia. In the sections of the intestines, most participants observed macrophages filled with bacilli which were morphologically consistent with corynebacteria.

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

Reference:
Cimprich, R. E., Rooney, J. R.: Corynebacterium equi enteritis in foals. Vet. Path. 14: 95-102, 1977.

Case II - 11587-17 (AFIP 1619768).

History: A female Hartley strain guinea pig was injected in the foot pad with an experimental inoculum 2 weeks prior to death.

Contributor's Diagnosis & Comments: Granulomatous lumbar lymphadenitis from overdose of Freund's complete adjuvant. Acid fast material is present in the footpads, lymph nodes and in some of the abdominal viscera.

Attendees' Diagnoses & Comments:
Morphologic Diagnosis: 1) Lymphadenitis, granulomatous, diffuse, severe, with subcapsular lipid droplets, lymph node. 2) Degeneration, myofiber, acute, with vascular hyaline degeneration, multifocal, moderate, muscle.
Etiologic Diagnosis: Consistent with Freund's adjuvant-induced lymphadenitis, and injection site reaction.
Etiology: Compatible with Freund's adjuvant.
Comments: Attendees noted the nonspecific diffuse histiocytosis of the lymph nodes, consisting of polymorphonuclear cells, lymphocytes and epithelioid macrophages. Many participants noted particles of material within macrophages. The presence of subcapsular lipid droplets and mild capsular fibrosis of the lymph node is often observed in lymph nodes draining body sites injected with Freund's adjuvant. The particles of material within some macrophages possibly represent fragments of mycobacteria cell walls which would stain positively with an acid-fast stain.

Contributor: Department of Pathology, Johns Hopkins University, Baltimore, MD 21205.

Case III - 79-1485 (AFIP 1712547).
History: These soft tissue masses were found in the dorsal thorocolumbar subcutis of a rat following experimental procedures.

Contributor's Diagnosis: Granulomatous steatitis, subcutis, rat.
Cause: injection of aluminum stearate in sesame oil.

Attendees' Diagnoses & Comments:
Morphologic Diagnosis: Panniculitis (steatitis), granulomatous, multinodular, multifocally extensive, subcutis.
Etiologic Diagnosis: Compatible with foreign body injection site steatitis.
Etiology: Possibly lipid material.
Comments: The nodules consisted of foci of fat necrosis with larger droplets probably from the injected foreign lipids. The inflammatory reaction was granulomatous and probably directed at exogenous and degenerating endogenous lipids.

Contributor: Department of Pathology, Syntex Research Inc., 3401 Hillview Avenue, Palo Alto, CA 94304.

Case IV - AFIP 2 (AFIP 1716397).
History: This Arabian foal had been diagnosed as having a severe disease characterized by marked lymphopenia (less than 1000 lymphocytes per mm^3), absence of IgM in the serum, and inability of peripheral blood leucocytes to respond to phytomitogens. On postnatal day 28, it was given liver and thymic cells from an unrelated 94-day-old Shetland pony fetus. The Arabian foal died at the age of 83 days.

Laboratory Results: At the time of death sorbital dehydrogenase was 18.6 and SGOT was 605 units. At necropsy there were dermal ulcers and a rounded, olotony liver.

Contributor's Diagnosis & Comments: Chronic-active, nonsuppurative hepatitis, predominantly periportal, with bile duct proliferation. Graft versus host (GVH) reaction is the most common sequela to transplantation of immunologically competent cells in humans. For GVH reaction to occur: 1) the recipient must be immunoincompetent; 2) there must be a histocompatibility mismatch between donor and recipient; 3) immunocompetent cells must be transplanted into the recipient. The fetal liver is a putative source of stem cells for such transplantations because it is rich in stem cells capable of developing into immunocompetent cells. If fetal liver is transplanted at 80 days, in some instances enough competent cells are presented to partially restore immunologic function. If transplanted tissue is from a fetus greater than 90 days of age, GVH reaction is likely to result, presumably due to the number of mature reactive T-cells within the graft. The lesions present in foals with GVH reaction are similar to those described in man and the dog.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: 1) Hepatitis, periportal, granulomatous, severe, with hepatocellular necrosis, liver. 2) Parasitism, protozoal, intrahepatocytic, diffuse, moderate, liver.

Etiologic Diagnoses: 1) Hepatic graft versus host reaction. 2) Hepatic protozoan parasitism (probably a coccidian parasite).

Comments: The presented clinicopathological data is diagnostic of severe combined immunodeficiency (CID) in the Arabian foal. Interpretation of the inflammatory reaction in the liver was complicated by the protozoan parasitism. Most attendees observed changes compatible with those described for graft versus host (GVH) reaction, such as individual hepatocyte necrosis, periportal distribution of macropnages and lymphocytes, and biliary duct hyperplasia. However, in view of the parasitism, participants were reluctant to ascribe these changes solely to a GVH reaction. The presence of numerous blastic and immature lymphocytoid cells further complicated histologic interpretation. Most participants considered the intrahepatic parasites to be consistent with coccidian protozoa. A definitive etiologic diagnosis was not possible at the light microscopic level and several possibilities were discussed. Due to the diagnosis of severe CID, attendees could not rule out equine protozoa such as Kossiella equi, Sarcocystis fayeri[6], or protozoan species normally not capable of causing patent infections in immunocompetent foals. Participants noted that some species of subfamily Sarcocystinae[7] are known to infect hepatocytes of their intermediate hosts.

Contributor: Department of Veterinary Pathology and Microbiology, Washington State University, Pullman, WA 99163.

References:

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2. Perryman, L. E., Liu, I.K.M.: Graft versus host reactions in foals with combined immunodeficiency. AJVR 41: 187-192, 1980.

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Results
AFIP Wednesday Slide Conference - No. 24
9 April 1980

Case I - A16838 (AFIP 1714914).

History: A 10-year-old, male Bedlington terrier was presented for ascites, weight loss, anorexia and jaundice of 3 months duration.

Laboratory Results: Copper analysis of various organs: Brain - 27; kidney - 290: liver - 6,000 µg/gram dry weight.

Contributor's Diagnosis & Comments: Chronic active hepatitis with cirrhosis, Bedlington Terrier copper toxicity. Normal lobular structure of liver has been replaced by extensive interlobular fibrosis and regenerative nodules. Moderate infiltration of inflammatory cells (mainly lymphocytes, plasma cells and neutrophils), proliferation of bile ducts, and dilated lymphatics are evident in portal triads. Abundant pigment granules and vacuoles are present in the hepatocytes.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Fibrosis, hepatocellular necrosis, and chronic-active hepatitis, diffuse, moderate to severe, with nodular hepatocellular hyperplasia and bile duct reduplication, liver.

Syndrome: Copper toxicosis of Bedlington terriers.

Etiology: Compatible with copper toxicity.

Comment: Participants unanimously concurred with the contributor's diagnosis. Attendees noted the prominent disruption of the hepatic cords and individualization of hepatocytes. Necrosis of hepatic cells probably resulted in the numerous small endothelial-like lined spaces and small nodules of hyperplastic hepatocytes. Collections of biliary ductules could be due to loss of hepatocytes and condensation ductules and/or hyperplasia of ductules. This disease in Bedlington Terriers was compared to Wilson's disease (hepatolenticular degeneration) of man, but in the dog, Kayser-Fleischer rings in the eye and lenticular nucleus degeneration have apparently not been reported.

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

Reference:

Twedt, D. C., Sternlieb, I. et al.: Clinical, morphologic and chemical studies on copper toxicosis of Bedlington Terriers. JAVMA 175: 269-275, 1979.

Case II - 77/670 (1718318).

History: This aged Merino wether was 1 of 20 animals to die in a mob of 500 in Southwestern Australia. Most sheep were found dead in the paddock, while others showed depression, anorexia, hemoglobinuria and jaundice for 1-2 days before death. The mortality occurred during the dry summer and for the preceding 6 months while the sheep had been grazing a pasture containing Echium lycopsis lycopsis ("Paterson's curse" or "Lady Campbell weed").

Laboratory Results: Copper analysis of various organs: Liver - 1600ppm (normal 100-800ppm); kidney - 1692ppm (normal 20-30ppm).

Contributor's Diagnosis & Comments: Pyrrolizidine alkaloidosis with superimposed hepatogenous chronic copper poisoning. In the liver there is diffuse megalocytosis, periacinar necrosis and hemorrhage, fatty change, biliary hyperplasia, bile stasis and Kupffer cells distended with copper-containing pigment. The typical gross postmortem picture of jaundice, enlarged yellow liver, enlarged dark red friable spleen, urinary bladder distended with red urine and green-black renal cortices also was observed. Five pyrrolizidine alkaloids or their N-oxides, the most plentiful of which are echiumine and echimidine, have been demonstrated in Echium lycopsis.

Attendees' Diagnoses & Comments:

Morphlogic Diagnosis: Necrosis and vacuolar degeneration, hepatocellular, centrilobular, diffuse, moderate to severe, with biliary stasis and megalocytosis liver.

Etiologic Diagnosis: 1) Compatible with copper toxicosis. 2) Suggestive of pyrrolizidine alkaloid toxicosis.

Comments: The antemortem symptoms are very suggestive of copper toxicosis. Histologically, pigment-laden hepatocytes and Kupffer cells, as well as a few megalocytes were observed. Since both copper and pyrrolizidine alkaloids may induce centrilobular necrosis, the pigments were considered suggestive of copper, while the megalocytes were suggestive of pyrrolizidine alkaloidosis. Several participants noted large eosinophilic Councilman-like inclusion bodies in the hepatocytes; these bodies or globules have been previously reported in association with pyrrolizidine alkaloidosis.[7] Chronic copper poisoning in sheep is known to be associated with plants containing pyrrolizidine alkaloids, such as Echium spp.[8] The synergistic action of the two substances has no clear pathogenesis as yet, but some suggest disruption of the copper storage proteins by the

pyrrolizidine alkaloids leading to copper accumulation in the liver,[8] or to the direct formation of copper-pyrrolizidine complexes.[9] Special stains used to demonstrate copper in hepatocytes and Kupffer cells include rhodanine and rubeanic acid.[10]

Contributor: Regional Veterinary Laboratory, Department of Agriculture, Wagga Wagga, New South Wales 2480, Australia.

References:

1. Todd, J. R., Thompson, R. H.: Studies on chronic copper poisoning. II. Biochemical studies on the blood of sheep during the hemolytic crisis. Brit. Vet. J. 119: 161-169, 1963.

2. Huxtable, R. J.: New aspects of the toxiclogy and pharmacology of pyrrolizidine alkaloids. Gen. Pharmacol. 10: 159-167, 1979.

3. McLean, E. K.: The toxic actions of pyrrolizidine (senecio) alkaloids. Pharmacol. Rev. 22: 429-483, 1970.

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5. Tandon, H. E., Tandon, B. N., et al.: An epidemic of veno-occlusive disease in India: Pathologic study. Indian J. Med. Res. 65: 679-684, 1977.

6. Everist, S. L.: Poisonous Plants of Australia, 1st ed., 1974, Angus & Robertson Ltd, Sydney, p. 306.

7. Bull, L. B.: The histologic evidence of liver damage from pyrrolizidine alkaloids: Megalocytosis of the liver cells and inclusion globules. Austr. Vet. J. 31: 33-40, 1962.

8. St. George-Grambauer, T. D., Rac, R.: Hepatogenous chronic copper poisoning in sheep in South Australia due to consumption of Echium plantagineum (Salvation Jane). Austr. Vet. J. 38: 288-293, 1962.

9. Farrington, K. J., Gallagher, C. H.: Complexes of copper with some pyrrolizidine alkaloids and some of their esterifying acids. Austr. J. Biol. Sci. 13: 600-603, 1960.

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Case III - 71953 (AFIP 1712662).

History: A 2-year-old female cat suffered recurrent bouts of depression.

Laboratory Results: There was elevated blood ammonia and BSP retention was 12.5% after 45 minutes.

Contributor's Diagnosis & Comments: Hepatic atrophy associated with portacaval shunt syndrome. Particularly noteworthy in this liver is the apparent high density of portal triads due to overall parenchymal atrophy.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Hemosiderosis, diffuse, moderate, liver.

Comments: There was considerable discussion of this disease entity. All participants detected aggregates of hemosiderin-laden macrophages in the liver. Special stains clearly demonstrated iron in hepatocytes and Kupffer cells. Several participants reported performing necropsy examinations of dogs with this condition, with the consistent finding being a liver normal in shape but usually less than one-third normal size. These participants suggested that keys to the diagnosis of portacaval shunt syndrome rest in being aware of the elevated blood ammonia in the clinical history and the markedly reduced size of the liver at gross examination. After this discussion, participants were able to appreciate the increased density of portal triads noted by the contributor.

Contributor: Department of Veterinary Pathology, College of Veterinary Medicine, Texas A&M, College Station, TX 77843.

References:

1. Cornelius, L. M., Thrall, D. E., et al.: Anomolous portosystemic anastomoses associated with chronic hepatic insufficiency in six young dogs. JAVMA 167: 220-228, 1975.

2. Breznock, E. M.: Surgical manipulation of portosystemic shunts in dogs. JAVMA 174: 819-826, 1979.

3. Vulgamott, J. C.: Hepatic encephalopathy associated with acquired portacaval shunt in a dog. JAVMA 175: 724-726, 1979.

Case IV - 2-213 (AFIP 1716401).

History: Tissues from a 20-year-old mare that was dead on presentation to the diagnostic laboratory. Clinical signs prior to death included weight loss, dark urine and edema of all 4 limbs. The animal was thin and had icteric mucous membranes upon presentation. The horse was 1 of 6 with similar signs in a herd of 130 in Montana. (Two microslides are submitted.)

Contributor's Diagnosis & Comments: Cholangiohepatitis, necropurulent, subacute, diffuse, severe, with marked biliary hyperplasia. Lesions compatible with alsike clover toxicosis. No laboratory tests were available.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: 1) Cholangiohepatitis, periportal, chronic-active, diffuse, severe, liver. 2) Megalocytosis, hepatocellular, multifocal, moderate, liver. 3) Hyperplasia, biliary, diffuse, moderate, liver.

Etiologic Diagnoses: 1) Suggestive of hepatic salmonellosis. 2) Hepatic toxicosis suggestive of trifoliosis.

Comments: Participants considered two processes to be the most likely cause for the observed changes in the liver. Megalocytosis, periportal fibrosis and biliary hyperplasia are considered features of equine trifoliosis.[1] Participants observed numerous colonies of bacteria within hyperplastic ductules with an acute inflammatory reaction. Whereas polymorphonuclear cells are usually absent in trifoliosis, the inflammatory cell reaction in this case was ascribed to the bacteria. Attendees discussed the antemortem symptoms given in the history: the limb edema is compatible with alsike-induced photosensitization[2] or skin reaction reminiscent of "dew poisoning"[2] of the limbs and muzzle.[3] Special stains demonstrated the intrabiliary bacteria to be Gram negative bacilli; Salmonella spp. were considered a likely etiologic agent.

Contributor: Marsh Laboratory, Montana State University, Bozeman, MT 59717.

References:

1. Jubb, K.V.F., Kennedy, P. C.: The liver and biliary system. In Pathology of Domestic Animals, Vol II, 2nd Ed., 1970, Academic Press, New York, p. 216.

2. Kingsbury, J. M.: In Poisonous Plants of the United States and Canada, 1964, Prentice-Hall Inc., Englewood Cliffs, NJ, pp 359-360.

3. Pammel, L. H.: Alsike clover poisoning. Am. J. Vet. Med. 15: 437, 1920.

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Results
AFIP Wednesday Slide Conference - No. 25
16 April 1980

Case I - H79-378 (AFIP 1712747).

History: In a swine herd of more than 1900 sows in Taiwan (Republic of China), an abortion storm occurred in the late fall. Abortions occurred in 207 sows; 48 farrowed mummified fetuses and 51 stillbirths were observed. In addition, 258 neonatal piglets died.

Laboratory Results: Virological Isolation: Pseudorabies virus was isolated from liver, lung, spleen, tonsil and brain of these pigs. Fluorescent antibody tests demonstrated pseudorabies virus-specific fluorescence in sections of the lung, liver, and tonsil from affected piglets. At necropsy there were multiple areas of necrosis in the liver, spleen, lung, adrenal, tonsil and lymph nodes of aborted fetuses, neonatal pigs and suckling pigs. The surface of the placental membrane was dull and slightly rough in appearance.

Contributor's Diagnosis & Comments: Placentitis, necrotizing, acute, diffuse, severe, characterized by Cowdry herpesvirus intranuclear inclusion bodies, porcine; etiology: Pseudorabies virus. Necrotic placentitis with intranuclear inclusions has not been described in veterinary literature, however, these lesions commonly were observed in placentas aborted during this outbreak of pseudorabies caused by the Taiwan-strain of the virus. Although viral isolation from the placenta has not been attempted, pseudorabies virus was the only virus consistently isolated from the visceral organs of aborted fetuses. Pathologic manifestations of the Taiwan-strain of pseudorabies virus are very similar to those observed in swine from the Midwestern United States. It is suggested that the virus in Taiwan may have been introduced from the United States, and that these lesions should be observed in U.S. swine. The intranuclear inclusion bodies varied from eosinophilic droplets to basophilic masses in the placenta as well as other visceral organs. Basophilic inclusions have not been reported in previous cases of pseudorabies. The inclusions in this outbreak of swine pseudorabies infection are similar to those seen in the herpes virus of infectious bovine rhinotracheitis in cattle.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Placentitis, necrotizing, acute, diffuse, severe, placenta, porcine.

Etiologic Diagnosis: Herpesviral placentitis.

Etiology: Consistent with pseudorabies herpesvirus.

Comments: All attendees reported seeing Cowdry type A intranuclear inclusion bodies in the epithelial cells of the chorionic villi. Intranuclear inclusion bodies also were observed in subepithelial stromal cells. Subepithelial inflammatory cells were very sparse.

Contributor: Pig Research Institute of Taiwan, P. O. Box 23, Chunan, Miaoli, Taiwan, Zip Code 350, Republic of China.

References:
1. Csontos, L., Heijj, L. et al.: A contribution to the aetiology of Aujeszky's disease in the pig. Foetal damage and abortion due to the virus. Acta Vet Hung 12: 17-23, 1962.
2. Hsu, F. S., Lin, B. C., et al.: Pathology of prenatal and neonatal pseudorabies in swine. Chinese Soc of Vet Med 5: 1-7, 1979.
3. Kennedy, P. C., Richards, W.P.C.: The pathology of abortion caused by the virus of infectious bovine rhinotracheitis. Path. Vet. 1: 7-17, 1964.
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6. Molello, J. A., Chow, T. L. et al.: Placental pathology. V. Placental lesions of cattle experimentally infected with infectious bovine rhinotracheitis virus. Am. J. Vet. Res. 27: 907-915, 1966.
7. Olander, H. J., Saunders, J. R. et al.: Pathologic findings in swine affected with a virulent strain of Aujeszky's virus. Path. Vet. 3: 64-82, 1966.
8. Wohlgemuth, K., Leslie, P. F. et al.: Pseudorabies virus associated with abortion in swine. JAVMA 172: 478-479, 1978.

Case II - 79-119 (AFIP 1714917).

History: On a small breeding farm 7 live healthy foals were born and one abortion occurred. Tissues are from another foal that died 8 hours after birth. The colt was born of a 10-year-old mare that was barren the previous year. Delivery was characterized as normal and easy. The foal would not stand, nurse, nor take milk from a bottle. Colostrum was given by stomach tube prior to death.

Contributor's Diagnosis & Comments: Rhinopneumonitis. Histopathological changes are considered diagnostic of rhinopneumonitis. The lung, thymus, liver and adrenals had foci of necrosis with frequent eosinophilic intranuclear inclusion bodies in respective parenchymal cells. Lymphoid necrosis and inclusion bodies were seen in the splenic white pulp.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: 1) Enteritis, necrohemorrhagic, multifocal, moderate, with severe lymphoid necrosis of Peyer's patches, small intestine, equine. 2) Vasculitis, necrotizing, with thrombosis and hemorrhage, acute, multifocal, moderate, submucosa of small intestine, equine.

Etiologic Diagnosis: Herpesviral lymphadenitis and enteritis.

Etiology: Consistent with Equine Herpesvirus I (Equine rhinopneumonitis virus).

Comments: Attendees unanimously agreed with the contributor's diagnosis. The most prominent lesion was the severe acute necrosis of lymphoid cells in the gut-associated lymphoid tissue. In the intestinal mucosa, acute necrosis of the epithelium with formation of multinucleated syncytial cells and intranuclear inclusions were evident. Predominantly in the venules of the submucosa, acute necrotizing vasculitis was accompanied by thrombi formation. The lesions in the veins of this foal are distinctly different from the immune complex vasculitis reported in small arteries of older animals infected with herpesviruses.

Contributor: Department of Pathology, New Bolton Center, University of Pennsylvania, 382 W. Street Road, Kennett Square, PA 19348.

References:

1. Charlton, K. M., Mitchell, D. et al.: Meningoencephalomyelitis in horses associated with Equine Herpesvirus I infection. Vet. Path. 13: 59-68, 1976.

2. Grewal, A. S., Rouse, B. T. et al.: Mechanisms of resistance to herpesviruses: Comparison of the effectiveness of different cell types in mediating antibody-dependent cell-mediated cytotoxicity. Infect. Immunol. 15: 698-707, 1977.

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Case III - H1145 (AFIP 1618990).

History: Two 4-week-old Hampshire pigs were among a group of 80 that were having breathing difficulties. At necropsy, the nasal turbinates contained a greyish exudate.

Contributor's Diagnosis: Inclusion body rhinitis.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Rhinitis, with megalocytosis, subacute, diffuse, severe, nasal turbinates, porcine.

Etiologic Diagnosis: Herpesviral rhinitis.

Etiology: Consistent with Herpesvirus Type B (cytomegalovirus) of swine.

Comments: Participants unanimously agreed with the contributor's diagnosis. Attendees observed intracytoplasmic and intranuclear inclusions within mucosal epithelial and glandular cells. Epithelial and glandular megalocytes were accompanied by mononuclear inflammatory cells and small abscesses in the glandular crypts. The lesions in the nasal mucosa induced by pseudorabies (Aujeszky's disease) virus infection are typically acute and necrotizing with formation of Cowdry type A inclusions rather than large basophilic inclusions as seen in this animal.

Contributor: Department of Veterinary Science, University of Nebraska, Lincoln, NB 68503.

References:

1. Baskerville, A., McCracken, R. M., et al.: The histopathology of experimental rhinitis in pigs produced by a strain of Aujeszky's disease virus. Res. Vet. Sci. 12: 323-326, 1971.

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3. Duncan, J. R., Ramsey, F. K. et al.: Electron microscopy of cytomegalic inclusion disease of swine (inclusion body rhinitis). Am. J. vet. Res. 26: 939-947, 1965.

Case IV - A79-170 (AFIP 1713946).

History: A 15-month-old female Myna was purchased in June 1979. The bird seemed normal until 6 weeks later when lethargy and absence of vocalism were noticed by the owner. The next day the bird started to breath heavily and move her head up and down as though she were choking. The bird died enroute to the clinic. At necropsy the liver was enlarged, pale, yellow-brown and had petechia on the capsular surface.

Contributor's Diagnosis & Comments: Diffuse severe acute mononuclear hepatitis with multifocal hepatic necrosis associated with schizonts in lymphoid-macrophages compatible with Lankesterella. There was also moderate fatty change and autolysis. Electron microscopy demonstrated protozoal organisms typical of coccidia, but which could not be definitively classified. Further studies are pending.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Hepatitis, necrotizing, diffuse, severe, liver.

Etiologic Diagnosis: Compatible with protozoal hepatitis.

Etiology: Suggestive of coccidian parasite such as Lankesterella, Toxoplasma, or Isospora.

Comments: All participants observed 1-3 micron diameter organisms within mononuclear inflammatory cells, attended by extensive regions of coallescing coagulative necrosis of hepatocytes. In their differential diagnosis, participants considered various coccidian and malarial protozoans, including Lankesterella (Atoxoplasma), Toxoplasma gondii, Hemoproteus and mixed infections. The histologic similarities of these organisms in tissue sections emphasizes the importance of transmission and/or isolation studies as well as electron microscopy in rendering definitive diagnoses of many avian protozoan infections.

Contributor: Angell Memorial Animal Hospital, 350 South Huntington Ave., Boston, MA 02130.

References:

1. Box, E. D.: Lankesterella (Atoxoplasma). IN Infectious and Parasitic Diseases of Wild Birds, Davis, J. W. & Anderson, R. C., Eds., Iowa State University Press, Ames, Iowa , 1971, pp 309-312.

2. Box, E. D.: Influence of Isospora infections on patency of avian Lankesterella (Atoxoplasma, Garnham, 1950). J. Parasitol. 53: 1140-1147, 1967.

3. Lainson, R.: Atoxoplasma, Garnham, 1950, as a synonym for Lankesterella, Labbe, 1899. Its life cycle in the English sparrow (Passer domesticus, Linn). J. Protozool. 6: 360, 1959.

4. Petrak, M. L.: Diseases of Cage and Aviary Birds, Lea & Febiger, Philadelphia, 1969, pp 400-402.

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Results
AFIP Wednesday Slide Conference - No. 26
23 April 1980

Case I - 79-1495 (AFIP 1712938).

History: An orphaned female coyote was reared from a few days of age to time of death at 16 weeks of age. The pup was immunized at 10 and 13 weeks for distemper and hepatitis. The pup was exposed to pound dogs and 3 to 5 days later developed anorexia, depression, diarrhea and dehydration. The animal died in spite of antibiotic and fluid therapy. Antemortem hemogram revealed the following results:

WBC	$3600/mm^3$	Differential	
HCT	38%	Neutrophils	14%
Total Protein	6	Lymphocytes	60%
RBC	Slight anisocytosis	Mononuclears	18%
		Eosinophils	6%

Contributor's Diagnosis: Necrotizing enteritis compatible with canine parvovirus infection.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Enteritis, necrotizing, subacute, diffuse, severe, small intestine, coyote.

Etiologic Diagnosis: Consistent with viral enteritis.

Etiology: Compatible with canine parvovirus.

Comments: Attendees unanimously agreed with the contributor's diagnosis. Necrosis was most evident in the crypt regions and gut-associated lymphoid tissue. Necrosis of the actively mitotic cells in the crypts probably resulted in the pronounced villus atrophy observed by participants. The lesions have a remarkable similarity to the changes seen in the intestine of cats infected with feline parvovirus (feline panleukopenia virus). Similar to the feline counterpart, the hemogram of this coyote reflects a pronounced leukopenia attributable, primarily, to an absolute neutropenia. Mild hypoproteinemia also was suggested by the hemogram results. Conference participants from the National Zoological Park report canine parvovirus infects a variety of adult wild canids; they have observed parvoviral enteritis with some fatalities in Maned wolves (Chrysocyon brachyurus),[3] bush dogs (Speothos venaticus) and a crab-eating fox (Cerdocyon thous).

Contributor: Veterinary Disease Laboratory, Syntex Research Inc., 3401 Hillview Avenue, Palo Alto, CA 94304.

References:

1. Pletcher, J. M., Toft, J. D. et al.: Histopathologic evidence for parvovirus infection in dogs. JAVMA 175: 825-828, 1979.

2. Cooper, B. J., Carmichael, L. E. et al.: Canine viral enteritis. II. Morphologic lesions in naturally occurring parvovirus infection. Cornell Vet. 69: 134-144, 1979.

3. Fletcher, K. C., Eugster, A. K. et al.: Parvovirus infection in Maned wolves. JAVMA 175: 897-900, 1979.

4. New Veterinary Biological Products. JAVMA 176: 730, 1980.

Case II - 79-1 (AFIP 1710129).

History: Tissue from 1 of 6 adult White Leghorn chickens to die in a research colony. The birds were blood donors and were not used experimentally.

Contributor's Diagnosis & Comments: Viral myocarditis, compatible with parvovirus infection. Six adult White Leghorn chickens were involved in this outbreak.[1] Electron microscopy revealed 18 to 20 nm particles in the inclusions. Attempts to isolate the virus and reproduce the disease by inoculation of tissue homogenates were unsuccessful. The lesions resemble those seen in influenza of geese, a parvoviral disease of young geese in Europe.[2] The lesions are also similar to those seen in parvoviral myocarditis of puppies. The occurrence of parvovirus replication in the supposedly mitotically inactive myocardium poses an interesting question with regard to the widely accepted theory that nonadeno-associated parvoviruses require the DNA synthesis of host cells for their own replication. Alternatively, there may be more DNA synthesis in the myocardium than is commonly assumed.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Myocarditis, lymphocytic, diffuse, severe, heart, White Leghorn chicken.

Etiologic Diagnosis: Viral myocarditis.

Etiology: Consistent with parvovirus.

Comments: All participants reported seeing basophilic intranuclear inclusions in the myocardial nuclei, typical of parvoviral inclusions. There was diffuse infiltration of predominantly lymphocytes around the degenerating cardiac myocytes. The degenerating myocytes were pale, granular and fragmented.

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

References:

1. Parker, G. A., Stedham, M. A. et al.: Myocarditis of probable viral origin in chickens. Avian Disease 21: 123-132, 1977.

2. Nagy, Z., Derzsy, D.: A viral disease of goslings. II. Microscopic lesions. Acta Vet. Acad. Sci. Hung. 18: 3-18, 1968.

Case III - 80A-0956-4 (AFIP 1713372).

History: Two miniature Schnauzer puppies from a litter of three died acutely at 5 to 6 weeks of age. The submitted tissue is from the third puppy which died acutely at 10 weeks of age.

Contributor's Diagnosis & Comments: Multiple myocardial fibrosis probably secondary to canine parvovirus-induced myocarditis. Histologic examination of the myocardium from the first two puppies revealed multifocal nonsuppurative myocarditis with large basophilic intranuclear inclusions. No intestinal lesions were detected.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Myocarditis, chronic-fibrosing, multifocal, moderate, heart, Miniature Schnauzer, canine.

Etiologic Diagnosis: Suggestive of viral myocarditis.

Etiology: Consistent with canine parvovirus.

Comments: The principal histologic changes noted by the participants were myocardial degeneration with early fibrosis. A few widely scattered interstitial and perivascular lymphocytes were also present. While nuclear inclusions were not observed, attendees considered the changes to be compatible with those previously reported in canine parvovirus infection. Some participants also mentioned recent work with gnotobiotic dogs, in which a strain of canine distemper virus also caused myocarditis.[3]

Contributor: Department of Pathology, College of Veterinary Medicine, University of Georgia, Athens, GA 30602.

References:

1. Hayes, M. A., Russell, R. G. et al.: Sudden death in young dogs with myocarditis caused by parvovirus. JAVMA 174: 1197-1203, 1979.

2. Jezyk, P. F., Haskins, M. E. et al.: Myocarditis of probable viral origin in pups of weaning age. JAVMA 174: 1204-1207, 1979.

3. Higgins, R. J., Krakowka, S.: Canine viral myocarditis (Letter). JAVMA 175: 415-417, 1979.

Case IV - 78-3679 (AFIP 1713320).

History: Four kittens from a household with 60 cats developed diarrhea with rapid dehydration and died within 24-48 hours. The specimen is from a 2-month-old Himalayan kitten.

Contributor's Diagnosis & Comments: Hepatocellular degeneration, diffuse, moderate, characterized by individualization of hepatocytes and viral-like intranuclear inclusion bodies compatible with Feline Panleukopenia virus infection. The large and small intestine had diffuse severe mucosal epithelial erosion with hyperplasia of residual crypt epithelium, "diagnostic" for Feline Panleukopenia virus infection. Individualization of hepatocytes has been reported to be more suggestive of feline parvovirus infection than hepatic intranuclear viral-like inclusions.[1] Individualization of hepatocytes as in the present case must be interpreted with caution since post-mortem changes likely can contribute to this appearance.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Hepatitis, acute, diffuse, moderate to severe, liver, Himalayan, feline.

Etiologic Diagnosis: Compatible with viral hepatitis.

Etiology: Compatible with feline parvovirus (Feline panleukopenia virus).

Comments: All attendees reported observing basophilic intranuclear inclusion bodies in degenerating hepatocytes, thus warranting the morphologic diagnosis of hepatitis. Individualization of hepatocytes was considered a real change in this liver. Although many participants considered feline herpesvirus (feline rhinotracheitis virus) infection to be high among their differential diagnoses, it was noted that the preeminent histologic lesion associated with herpesviral hepatitis

is multiple discrete foci of necrosis rather than diffuse necrosis with individualization of hepatocytes, as was observed in this tissue. Whereas th[illegible] are excellent papers in the literature in which hepatic lesions were absent[2] or nonspecific[3] in cases of feline panleukopenia, it is suggested that lesions in this case may be due to an uncommon strain of feline parvovirus or perhaps to an immuno-compromised host.

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

References:

1. Jubb, K. V. H., Kennedy, P. C.: Pathology of Domestic Animals, Vol. 2, 1970. Academic Press, New York, pp 132.

2. Carlson, J. H., Scott, F. W. et al.: Feline Panleukopenia. I. Pathogenesis in germfree and specific pathogen-free cats. Vet Path 14: 79-88, 1977.

3. Langheinrich, K. A., Nielsen, S. W.: Histopathology of feline panleukopenia: A report of 65 cases. JAVMA 158: 863-872, 1971.

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Results
AFIP Wednesday Slide Conference - No. 27
30 April 1980

Case I - 7187 (AFIP 1639438).

History: This subcutaneous mass occurred on the abdomen of a 5-month-old female F344 rat. The mass was 5x5x4 cm and was an incidental finding.

Contributor's Diagnosis & Comments: Anaplastic mammary carcinoma. The majority of mammary tumors in rats are fibroadenomas. This neoplasm is extremely cellular in some areas, being composed of cells with a modest amount of cytoplasm and rounded, vesicular nuclei with many mitotic figures. In some fields there are attempts at acinar formation. Distant metastases were not found, but there was definite invasion of adjacent structures, particularly skeletal muscle. Metachromatic granules were not seen in tumor cells after staining with toluidine blue.

Attendees' Diagnosis and Comments:

Diagnosis: Carcinoma, anaplastic, solid, subcutis, probable mammary origin, rat.

Comments: Participants were impressed by the high mitotic rate, lack of cellular patterns and extensive areas of necrosis in this tumor. While most histoslides failed to demonstrate convincing glandular formations, the polygonal shape of cells, large nuclei with prominent nucleoli, and subepidermal regions of myxomatous differentiation were suggestive of carcinoma, rather than sarcoma. In the absence of distinct spindle-shaped cells, which are necessary for malignant mesenchymal mammary tumors in the international classification of tumors of the rat (Young, et al., 1973), participants considered the tumor most compatible with an anaplastic carcinoma of mammary gland origin. The lack of hair follicles and adnexal structures in the skin overlying the neoplasm was attributed to reactive proliferative growth of skin in attempt to cover the expansile nodular neoplasm.

Contributor: Department of Comparative Medicine, The Medical Center, University of Alabama, Birmingham, AL 35194.

Reference:

Young, S., Hallowes, R. C.: Tumours of the mammary gland. In: Pathology of Tumours in Laboratory Animals, Vol. I. Tumours of the Rat, Part 1; V.S. Turusov (ed.), International Agency for Research on Cancer, WHO; Lyon, France, 1973, pp 31-74.

Case II - (AFIP 1504164).

History: A 12-year-old female Dachshund was treated for nonresponsive iritis. After a few months the eye became enlarged and painful.

Contributor's Diagnosis & Comments: Malignant melanoma, anterior uvea, with secondary glaucoma, eye, canine.

The tumor cells are morphologically compatible with the epithelial type of melanoma as described in the Callender classification used by physicians. Even without a melanin bleach, the tumor cells clearly have large nuclei with a prominent nucleolus and coarse, often peripheralized, chromatin, a large nucleus to cytoplasmic ratio, pleomorphism, and a moderate mitotic rate. The entire iris and ciliary body are involved and the neoplastic cells are invading the sclera as well as infiltrating along the corneal endothelium. The filtration angles are obliterated. The massive destruction of the ciliary body and resulting decrease in production of aqueous humor, probably accounts for the minimal glaucomatous changes in the retina. In this case, the inner layers of the retina show minimal degeneration, whereas in an eye with such total occlusion of the filtration angle, severe loss and/or degeneration of the inner retinal layers would normally be observed.

There is mild corneal epithelial erosion and acute patchy keratitis. The lens is artefactually absent.

This dog died two years following enucleation; a complete necropsy failed to show any evidence of metastasis. It has been the experience of AFIP that the histo-morphologic prognostic criteria utilized in the diagnosis of intraocular melanomas in man do not appear to apply in the dog. Most intraocular melanomas in man begin in the posterior uvea (choroid). Only a few such cases have been reported in the dog. Most primary canine intraocular melanomas arise in the anterior uvea. In humans anterior uveal melanotic tumors are usually benign behaviorally, while most posterior uveal melanomas have a great malignancy potential and carry a grave prognosis. In our experience with canine intraocular melanomas, regardless of cellular morphology, site of origin, or degree of invasiveness (provided the neoplasm has not penetrated the globe), these melanotic tumors seldom metastasize.

Attendees' Diagnosis & Comments:

Diagnosis: Malignant melanoma, epitheloid, anterior uvea, eye, canine.

Comments: Participants unanimously agreed with the contributed diagnosis.

Contributor: Registry of Veterinary Pathology, AFIP, Washington, DC 20306.

References:

1. Gamel, J., McLean, W. el al.: Uveal melanomas. Correlation of cytologic features with prognosis. Cancer 41: 1897-1901, 1978.

2. Rones, B., Zimmerman, L. E.: The prognosis of primary tumors of the iris treated by iridectomy. Arch. Ophthalmol. 60: 193-205, 1958.

Case III - (AFIP 1717347).

History: This mass was found within the eye of a 2-year-old male Malamute.

Contributor's Diagnosis & Comments: Teratoid medulloepithelioma, eye, canine.

These are rare neoplasms of the canine and bovine eye which contain tissues resembling brain, cartilage, rhabdomyoblasts and premature mesenchyme, in addition to the poorly differentiated neuroepithelial and neuroblastic elements (rosettes) characteristic of medulloepitheliomas. The stroma of this tumor contained alcian blue positive material, which was sensitive to hyaluronidase digestion, a characteristic of medulloepitheliomas. In addition, silver stains clearly demonstrated axon-like fibers; cartilage also is readily seen. A PTAH stain for muscle fibers equivocally demonstrated bundles of rhabdomyoblasts.

The aggressive and pleomorphic features of this neoplasm warrant a designation of malignant. However, to our knowledge, there are no reported cases of metastasis of these tumors. Therefore, until more is known of these neoplasms, they should probably simply be designated teratoid medulloepitheliomas.

Attendees' Diagnosis & Comments:

Diagnosis: Teratoid medulloepithelioma, eye, canine.

Comments: Attendees unanimously agreed with the contributed diagnosis. Most histoslides clearly contained tissues representative of retinal neurons, cartilage, lens epithelial cells and material, and neurons. Rhabdomyoblasts were not clearly identifiable on the H&E prepared histoslides.

Contributor: Contributed to the Registry of Veterinary Pathology by Biomedical Reference Laboratories Inc., 1447 York Court, Burlington, NC 27215.

References:

1. Lahav, M., Albert, D. M. et al.: Malignant teratoid medulloepithelioma in a dog. Vet. Path. 13: 11-16, 1976.

2. Langloss, J. M., Zimmerman, L. E. et al.: Malignant intraocular teratoid medulloepithelioma in three dogs. Vet. Path. 13: 343-352, 1976.

Case IV - (AFIP 1582044).

History: A 6-week-old male Australian shepherd puppy was presented because the eyes looked small and it appeared to be blind. (One histoslide and one 2x2 photomicrograph are presented).

Contributor's Diagnosis & Comments: Hereditary multiple ocular anomalies in the Australian shepherd dog.

This condition is suspected to be an autosomal recessive trait linked to the amount of white in the coat of this breed. The multiple anomalies can consist of microphthalmia, iridal heterochromia, cortical cataracts, equatorial staphylomas (in approximately 50% of cases), aberrant retinal tissues in the sclera, retinal folds and dysplasia, and colobomas. Most slides show cortical cataractous change, retinal detachment with folds, rosettes, dysplasia, equatorial scleral thinning and almost total absence of choroidal tissue. Many histoslides, but not all, had foci of intrascleral retinal tissue near the ora

ciliaris retina. However, since this important feature was absent in some histoslides, the 2x2 photomicrograph was submitted to demonstrate this anomaly.

Microphthalmia and cataracts often are observed in association with retinal dysplasia in other breeds as a congenital anomaly, but not always as an inherited trait. The lack of pupil in many of the sections is a function of cut.

Attendees' Diagnoses & Comments:

Morphologic Diagnoses: Microphthalmia (subgross diagnosis) attended with: a) Retinal dysplasia, detachment and ectopia, retina; b) Cataractous change, cortical, lens; c) Posterior synechia, lens; d) Hypoplasia, anterior uvea; e) Hypoplasia, choroid; f) Hypertrophy, retinal pigmented epithelium, etiology; secondary to retinal detachment.

Syndrome: Hereditary multiple ocular anomalies.

Comments: Participants concurred with the contributed diagnosis, and noted that inherited retinal dysplasias have also been reported in the Labrador retriever, Bedlington terrier, Sealyham terrier, and the Springer Spaniel.

Contributor: Contributed to the Registry of Veterinary Pathology by the Department of Pathology, Fitzsimmons Army Medical Center, Denver CO 80240.

References:

1. Gelatt, K. N., Veith, L. A.: Hereditary multiple ocular anomalies in Australian shepherd dogs. VM/SAC 65: 39-42, 1970.

2. Gelatt, K. N., McGill, L. D.: Clinical characteristics of microphthalmia with colobomas of the Australian shepherd dog. J.A.V.M.A. 162: 393-396, 1973.

3. Saunders, L. Z., Rubin, L. F.: Ophthalmic Pathology of Animals. S. Karger, New York, 1975, pp 190-194.

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Results
AFIP Wednesday Slide Conference - No. 28
14 May 1980

Case I - (AFIP 1737202).

History: Tissue from a 1-3 day-old Dorper lamb. The lamb first showed signs of pyrexia (41.5°C), followed by listlessness, increased respiratory rate, anorexia and general weakness. Bright yellow to orange feces were passed. Death occurred 36 hours after the first signs of listlessness. During the course of illness, neutrophilia and lymphopenia were recorded. Remarkable changes at necropsy consisted of moderate to severe icterus and a soft, friable liver. (The animal is from a country in Southern Africa).

Contributor's Diagnosis & Comments: Wesselsbron disease of lambs (experimental). This lamb was inoculated intradermally in 5 sites with a 0.5 ml suspension of $10^{6.6}$ MLD_{50}/ml of Wesselsbron virus.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Hepatitis, necrotizing, acute, diffuse, moderate to severe, liver, Dorper lamb.

Etiologic Diagnosis: Viral hepatitis.

Etiology: Consistent with Wesselsbron virus infection.

Comments: Most participants noted the prominent loss of hepatic cord architecture, the diffuse necrosis of individual hepatocytes and the formation of many cytoplasmic Councilman-like bodies. Although most participants considered the virus of Rift Valley fever in their differential, all agreed there was little histologic similarity between the two patterns of necrotizing hepatitis seen in these viral diseases: Rift Valley fever has distinct foci of coagulative necrosis in paracentral regions of the liver, while Wesselsbron causes diffuse necrosis of individual hepatocytes. The gross finding of prominent icterus also is commonly associated with Wesselsbron in sheep.

Contributor: Division of Zoonotic Disease Pathology, Armed Forces Institute of Pathology, Washington, DC 20306.

Reference:

Coetzer, J. A. W., Theodoridis, A. et al.: Wesselsbron Disease. Pathological, haematological and clinical studies in natural cases and experimentally infected newborn lambs. Onderstepoort J. Vet. Res. 45: 93-106, 1978.

Case II - (AFIP 1737276).

History: A number of dairy cows from a herd in the Republic of South Africa were noted to be reticent to move and walked with a stiff gait. Several of these cows developed severe respiratory signs and died in a matter of hours. Tissue is presented from one of the dead cows.

Contributor's Diagnosis & Comments: Probable ephemeral fever (Three-day stiff sickness). Severe pulmonary emphysema with rapid death has been reported in this disease. Although viral isolation was not attempted in this case, the history and histopathologic changes in the lung are very suggestive. The necropsy was conducted by a local practitioner and the joints were not examined.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Bronchopneumonia, fibrinopurulent, acute, disseminated, mild, with severe bullous emphysema, lung, bovine.

Etiology: Compatible with pulmonary form of Ephemeral fever rhabdovirus infection.

Comments: Most participants considered the mild bronchopneumonia and severe bullous emphysema which dissected between interlobular septae to be very suggestive of the dry bullous form of ephemeral fever. The regional history and clinical history were critical in making this diagnosis. Many participants also noted a mild vasculitis in the interlobular areas. This foreign animal disease would have to be differentiated from acute atypical interstitial pneumonia and contagious pleuropneumonia of cattle. The latter disease, however, is usually accompanied by severe pulmonary edema and fibrinous exudation.

Contributor: Division of Zoonotic Disease Pathology, Armed Forces Institute of Pathology, Washington, DC 20306.

References:

1. Theodoridis, A., Coetzer, J. A. W.: Subcutaneous and pulmonary emphysema as complications of bovine ephemeral fever. Onderstepoort J. Vet. Res. 46: 125-127, 1979.

2. Burgess, G. W., Spradbrow, P. B.: Studies on the pathogenesis of bovine ephemeral fever. Australian Vet. J. 53: 363-368, 1977.

Case III - X-11841 (AFIP 1727726).

History: Skin is submitted from 1 of 7 male Siberian polecats (Mustela eversmanni satunini) which died in a 5 day period. Clinical signs included anorexia, lethargy, dehydration and anemia.

Contributor's Diagnosis & Comments: Hepatozoon mustelis infection. Necropsy of the 7 polecats revealed generalized dermal petechia and edema with petechia in the brain. Hepatozoon organisms also were present in the liver, heart, lung, kidney, lymph nodes and skeletal muscle. Schizonts, merizoites, and gametocytes were all present in the skin. Inflammation was present only where the schizonts had ruptured. Encephalitozoon cuniculi organisms also were found in the skin, brain, kidney, heart, lung, lymph node, skeletal muscle, spleen and intestine. All 7 polecats had similar lesions.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Granulomas, protozoal, multifocal, subcutis, , skin, Siberian polecat.

Etiologic Diagnosis: Dermal protozoiasis.

Etiology: Consistent with Hepatozoon mustelis.

Comments: Most conference participants had attended the symposium in which this new disease was recently reported.[1] Only a few attendees reported observing minute granulomas which lacked hepatozoon organisms; these may have been foci of Encephalitozoon infection. The hepatozoon granulomas had elicited minimal inflammatory reaction, and in the absence of readily identifiable epithelioid macrophages, some attendees suggested protozoal cysts could be an alternate diagnosis. The small size of these protozoal cysts, the lack of an induced host cell wall, and the lack of karyomegalic host cell nuclei within a cyst wall all help to differentiate this organism from Besnoitia spp.[3]

Contributor: Eli Lilly and Company, Greenfield Laboratories, P. O. Box 708, Greenfield, IN 46140.

References:

1. Novilla, M. N., Carpenter, J. W. et al.: Dual infection of Siberian polecats with Encephalitozoon cuniculi and Hepatozoon mustelis n. sp. In: Comparative Pathology of Zoo Animals, 1978, R. J. Montali, G. Migaki (eds.), Smithsonian Institution Press, Washington, DC. (In press, 1980).

2. McCully, R. M., Basson, P. A. et al.: Observations on naturally occurring acquired hepatozoonosis of wild carnivores and dogs in the Republic of South Africa. Onderstepoort J. Vet. Res. 42:117-135, 1975.

3. Frenkel, J. K.: Besnoitia wallacei of cats and rodents: With a reclassification of other cyst-forming isosporoid coccidia. J. Parasitol. 63: 611-628, 1977.

Case IV - 77-2653 or 77-2785 (AFIP 1713327).

History: Tissue from mice surviving a long-term experiment with an infectious agent. Antemortem clinical laboratory tests revealed the mice were anemic, hypoglycemic, hypoalbuminemic and hyperglobulinemic.

Contributor's Diagnosis & Comments: Meningoencephalitis, lymphoplasmacytic, diffuse, severe, brain, mouse. Etiology: Trypanosoma rhodesiense. The purpose of the experiment was to study the sequential development of lesions in mice caused by trypanosomiasis in an attempt to establish a suitable animal model for the chronic form of the disease in man (African trypanosomiasis, African sleeping sickness). The mice were injected intraperitoneally with 1000 Trypanosoma rhodesiense (LVH 18) organisms which were originally isolated from a human patient in Kenya. The mice were sequentially sacrificed during a 140-day experiment. Mice presented in the slides were killed at 126 or 140 days post-inoculation. In addition to the brain lesions, a lymphoplasmacytic infiltrate was observed in the liver, heart, kidney, epididymis, and pancreas. The lymph nodes and spleen were depleted of lymphocytes and reticuloendothelial cells were prominent. The lesions observed in these mice were very similar to those described in chronic trypanosomiasis in man and other animals.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Choriomeningoencephalitis, lymphoplasmacytic, diffuse, moderate, brain, mouse.

Etiologic Diagnosis: Choriomeningoencephalitic protozoiasis.

Etiology: Compatible with Trypanosoma sp. infection.

Comments: Participants considered the lesions very similar to those observed in human cases of African trypanosomiasis (African sleeping sickness).[1] Most attendees reported seeing minute, ovoid (2-3 micron) organisms in the choroidal interstitium; these structures appeared aflagellate but with a small polar body. These features are the typical presentation of trypanosomes in tissue sections. Large plasma cells (Mott cells, morular cells) with prominent paranuclear cytoplasmic material (Russell bodies) were common in the choroid and subependymal regions. Mott cells (or morular cells), although not specific, are characteristic of African trypanosomiasis in man. Demonstration of trypanosomes in tissue sections is difficult, and not considered a practical approach to diagnosis.[1] Investigators always must use extreme care and controls to ensure that lesions in the CNS of mice, rats or rabbits on any experiment are not due to or complicated by concurrent infection with Encephalitozoon cuniculi or lymphocytic choriomeningitis virus.

Contributor: Department of Comparative Pathology, Walter Reed Army Institute of Research, Walter Reed Army Medical Center, Washington, DC 20012.

References:

1. Connor, D. H., Neafie, R. C. et al.: African Trypanosomiasis. In: Pathology of Tropical and Extraordinary Diseases, Vol. 1, 1976, C. H. Binford, D. H. Connor (eds.), AFIP Press, Washington, DC, pp 252-257.

2. Losos, G. J., Ikede, B. O.: Review of pathology of diseases in domestic and laboratory animals caused by Trypanosoma congolense, T. vivax, T. brucei, T. rhodesiense and T. gambiense. Vet. Pathol. 9 (Supplementum): 1-71, 1972.

3. Murray, M.: Pathology of African trypanosomiasis. Prog. in Immunol. II, 40: 181-192, 1974.

4. Ormerod, W. E.: Pathologenesis and pathology of Trypanosomiasis in Man. In: African Trypanosomiasis, 1970, Allen & Unwin, London, pp 587-601.

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Results
AFIP Wednesday Slide Conference - No. 29
21 May 1980

Case I - 79-7035 (AFIP 1701507).

History: A 4½-year-old, mixed-breed dog was presented with a growth near the external occipital protuberance. The mass was noticed 5 months earlier and had grown slowly. The surgical specimen is submitted.

Contributor's Diagnosis & Comments: Chondroma rodens. This tumor also has been called multilobular osteoma and chondroma of the canine skull.[1] These tumors are locally aggressive and may become malignant. This tumor did not show histologic evidence of malignancy.

Attendees' Diagnosis & Comments:

Diagnosis: Chondroma rodens, occiput, canine.

Comments: Participants unanimously concurred with the contributor's diagnosis. In the past this tumor in canids has been considered an analogue of the juvenile aponeurotic fibroma of man based on some histologic similarities. However, there are distinct differences between the two tumors which involve the anatomic location of the growths, their propensity to involve bone, the age of the individual and some morphologic and organizational features. In the dog, chondroma rodens occurs primarily at the occiput in aged bitches, while the human aponeurotic fibroma is generally found in the musculature or other soft tissues of the hands, wrists or feet of juvenile males. Histologically, aponeurotic fibromas tend to be less circumscribed and are not organized into the discrete islands and cords so characteristic of chondroma rodens. While the human counterpart often has a substantial fibroblastic component, the formation of bone, seen commonly in the canine tumors, is not considered a feature of the aponeurotic fibroma.[2]

Contributor: Department of Pathology, College of Veterinary Medicine, Kansas State University, Manhattan, KS 66502.

References:

1. Pool, R. R.: Tumors of bone and cartilage. In: Tumors in Domestic Animals, 2nd ed., 1978, J. E. Moulton (ed.), University of California Press, Berkeley, pp 99-103.

2. Pletcher, J. M., Koch, S. A. et al.: Orbital chondroma rodens in a dog. J.A.V.M.A. 175: 187-190, 1979.

3. Misdorp, W., Van Der Heul, R. O.: International histological classification of tumours of domestic animals. XXI. Tumours of bones and joints. Bull. W.H.O. 53: 265-282, 1976.

Case II - S78-133 (AFIP 1664430).

History: This stifle is from a 38-day-old, Osborne-Mendel tl (toothless) rat. Joints of all 4 limbs had been swollen for several days.

Laboratory Results: Cultures were negative for bacteria and mycoplasma.

Contributor's Diagnoses & Comments: 1) Arthritis, fibrinopurulent; 2) Osteopetrosis. Lesions were restricted to bones and joints. The joint capsules were distended due to the accumulation of fibrinopurulent exudate. Surrounding tissues were edematous and contained early fibroplasia. In rats, mycoplasma and Streptobacillus moniliformis are frequent causes of arthritis. In this case, bacteria were not observed in the tissues. Possible mishandling of joint fluid samples may have resulted in negative cultures.

Osteopetrosis is an hereditary disease in humans, dogs, cattle, mice, rabbits and rats. The severity of the osteopetrosis varies among these species from a transitory condition to a fatal disease. Osteopetrosis in the tl rat results in unerupted dentition and, thus, the "toothless" designation. These rats have a normal longevity. Osteopetrosis is characterized by defective bone remodeling. Chondro-osseous tissue persists in the diaphysis and results in a reduction of the marrow cavity.

Attendees' Diagnoses & Comments: 1) Arthritis, fibrinopurulent, acute, diffuse, severe, stifle; 2) Periosteitis, fibrinopurulent with early pannus of articular cartilage, acute, diffuse, severe, stifle; 3) Osteopetrosis, diffuse, moderate to severe, tibia and femur, rat.

Etiologic Diagnoses: 1) Consistent with arthritic mycoplasmosis; 2) Hereditary osteoclast malfunction.

Etiologies: 1) Consistent with Mycoplasma arthritidis infection. 2) Hereditary recessive trait.

Comments: Attendees agreed with the contributor's diagnoses. Most participants considered M. arthritidis to be the most likely cause of the arthritis, but also considered mixed infections with streptococcus and streptobacillus. Special stains, however, failed to demonstrate bacterial agents. Most attendees also commented on the intense granulocytosis in the metaphyseal bone marrow, but were reluctant to attribute the imbalanced erythroid:myeloid ratio to stimulation by the infection, inasmuch as rats with the tl trait have concurrent hematopoietic dyscrasias which are closely associated with the osteopetrotic condition. Preliminary studies of the distribution of acid phosphatase activity within tl osteoclasts indicate that this enzyme was not localized next to the bone surface as in normal osteoclasts. Osteoclasts are reduced in number and size in tl rats, and the number of cytoplasmic vacuoles adjacent to the ruffled borders are fewer than in normal rats. Thus, deficient osteoclastic activity results in the failure to remodel bone and mineralized cartilage at the epiphyseal plate. Inadequate bone and cartilage remodeling results in filling of the medullary cavity with cancellous bone and failure of remodeling at the cut-back zone (seen as thickened sub-epiphyseal diaphyses). The majority of multinucleated giant cells in the marrow cavities were megakaryocytes, rather than osteoclasts. The toothless feature is due to failure of osteoclastic remodeling of the maxilla and mandible, resulting in impaired dental eruption.

Contributor: Experimental Pathology Department, Naval Medical Research Institute, Bethesda, MD 20014.

References:

1. Cotton, W. R., Gaines, J. F.: Unerupted dentition secondary to congenital osteopetrosis in the Osborne-Mendel rat. Proc. Soc. Exp. Biol. Med. 146: 554-561, 1971.

2. Marks, S. C., Schneider, G. B.: Evidence for a relationship between lymphoid cells and osteoclasts: Bone resorption restored in ia (Osteopetrotic) rats by lymphocytes, monocytes and macrophages from a normal littermate. Am. J. Anat. 152: 331-342, 1978.

3. Cole, B. C., Ward, J. R. et al.: Chronic proliferative arthritis of mice induced by Mycoplasma arthritidis. I. Induction of disease and histopathological characteristics. Infect. Immun. 4: 344-355, 1971.

4. Hannan, P. C. T., Hughes, B. O.: Reproducible polyarthritis in rats caused by Mycoplasma arthritidis. Ann. Rheum. Dis. 30: 316-321, 1971.

Case III - 7800-60 (AFIP 1711663).

History: A fawn was hand-raised to the age of 6 months at which time it was released. The deer remained semi-tame and was seen frequently. At 8 months of age the deer had a pair of small, normal antlers, but 1 month later the right antler was replaced by a pedunculated, skin-covered mass approximately 8 x 4 inches. The mass was surgically excised and found to be firm and rubbery except for the attaching stalk which was soft and fleshy. (One histoslide and two 2x2 photoslides are presented).

Contributor's Diagnosis & Comments: Fracture of antler with sequestration, bone survival, and massive hyperplasia and collagenous metaplasia of periosteum. The mature antler bone does not normally contain marrow, hence, the dense fibrous inner structure is normal.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Subperiosteal new bone formation with periosteal fibroplasia, focally extensive, antler, deer.

Condition: Compatible with healing fracture of an antler in velvet.

Etiology: Probably trauma.

Comments: Most attendees also noted mild vasculitis in the sub-epithelial region. Most or all of the submitted histoslides failed to demonstrate bone sequestration, i.e. necrotic bone; this underscores the complexity of large bone lesions and emphasizes the need for caution in assuming a single, limited section accurately depicts an entire lesion. Most sections demonstrated a central region of preexisting bone with about an equal-sized region of periosteal new bone growth. The two regions of bone blend almost imperceptively, but the new bone generally had greater distances between the bony trabeculae. Between the periosteum and epidermis was an intense region of fibroplasia. Before the epidermis (antler velvet) was shed, the region of dense collagenous fibroplasia probably would have become bony antler.

Contributor: St. Louis Zoological Park and Monsanto Company, 800 N. Lindbergh Blvd., St. Louis, MO 63166.

References:

1. Taylor, D. O. N., Thomas, J. W. et al.: Abnormal antler growth associated with hypogonadism in white-tailed deer in Texas. Am. J. Vet. Res. 25: 179-185, 1964.

2. Wislocki, G. B., Waldo, C. M.: Further observations on the histological changes associated with the shedding of antlers of the white-tailed deer (Odocoileus virginianus). Anat. Rec. 117: 353-375, 1953.

Case IV - D79-442 (AFIP 1713328).

History: In a flock of 18,500 Hubbard broiler chickens, 5 percent of the birds were reluctant to move. When forced to move, the birds exhibited a painful, jerky gait. At necropsy affected 5½-week-old birds had subperiosteal hemorrhage on the anterior and medial aspects of the proximal tibia. Some birds had anterior bowing of the tibia. (One oversized histoslide).

Contributor's Diagnosis & Comments: Tibial dyschondroplasia. This lesion is believed to represent a failure of normal vascular invasion of the physis. Riddell has reproduced the lesion by aseptically inserting Saran-wrap^{R} in the metaphysis interrupting the metaphyseal blood supply to the physis. Poulos, et al, consider the underlying defect to be disturbed differentiation of the cells of the transitional (pre-hypertrophied) bone leading to cessation of endochondral ossification. The incidence of this lesion is also influenced by chloride level in the ration, energy level of the ration and, inherent rate of growth of the bird.

Attendees' Diagnosis & Comments:

Morphologic Diagnosis: Dyschondroplasia, epiphyseal, diffuse, moderate to severe, tibia, chicken.

Comments: Attendees agreed with the contributor's diagnosis. The principle histologic changes in this tibia included widening of the epiphyseal plate by disorganized, hypertrophied chondrocytes, lack of progression to the formation of orderly trabecular bone, lack of secondary bone formation and partial failure of cartilage to mineralize. The principle biochemical problem appears to be a decreased synthesis and degradation rate of proteoglycans in the zone of hypertrophied cartilage.[1] The lesion involves the epiphyses of most long bones, and hence, might more appropriately be considered epiphyseal osteochondrosis, and not simply a tibial dyschondroplasia.

Contributor: Department of Veterinary Pathology, College of Veterinary Medicine, University of Georgia, Athens, GA 30602.

References:
1. Lowther, D. A., Robinson, H. C. et al.: Cartilage matrix components in chickens with tibial dyschondroplasia. J. Nutr. 104: 922-929, 1974.
2. Riddell, C.: The development of tibial dyschondroplasia in broiler chickens. Avian Dis. 19: 443-461, 1975.
3. Riddell, C.: Studies on the pathogenesis of tibial dyschondroplasia in chickens. Part I, II & III. Avian Dis. 19: 473-505, 1975.
4. Poulos, P. W., Reiland, S. et al.: Skeletal lesions in the broiler with special reference to dyschondroplasia (osteochondrosis). Pathology, frequency and clinical significance in two strains of birds on high and low energy feed. Acta Radiologica 358: 229-277, 1978.

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Results
AFIP Wednesday Slide Conference - No. 30
28 May 1980

Case I - C79-01 (AFIP 1712749).

History: A 9-month-old, male Hairless Hartley strain guinea pig developed respiratory distress over a 4 day period. Treatment with fluids and antibiotics had little effect and the animal died. At necropsy, the lungs failed to collapse, were firm, and the airways contained frothy sanguinous fluid. (H&E and GMS histoslides are presented).

Contributor's Diagnosis & Comments: Pneumocystis carinii pneumonia. This strain of guinea pig is a mutant which is both hairless and immunodeficient. The immunodeficiency stems from aplasia or hypoplasia of the thymus and hypogammaglobulinemia. In addition to Pneumocystis, these guinea pigs are very susceptible to cytomegalovirus and Balantidium infections.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Pneumonitis, interstitial, acute to subacute, diffuse, severe, lung, guinea pig.

Etiologic Diagnosis: Consistent with pneumonic pneumocystosis.

Etiology: Pneumocystis carinii.

Comments: Attendees unanimously agreed with the contributor's diagnosis. Many participants considered a diagnosis of alveolitis to be acceptable, inasmuch as the trophozoites of Pneumocystis are restricted to the terminal airways.

Contributor: Toxicology Section, Health, Safety and Human Factors Laboratory, Eastman Kodak Company, B-320 Kodak Park, Rochester, NY 14650.

References:

1. Reed, C., O'Donoghue, J. L.: A new guinea pig mutant with abnormal hair production and immunodeficiency. Lab. Anim. Sci. 29: 744-748, 1979.

2. Frenkel, J. K.: Pneumocystosis. In: Pathology of Tropical and Extraordinary Diseases, Vol. 1, C. H. Binford and D. H. Connor (eds.), AFIP Press, Washington, DC, 1976, pp 303-307.

3. Frenkel, J. K., Good, J. T. et al.: Latent pneumocystis infection of rats, relapse, and chemotherapy. Lab. Investigation 15: 1559-1577, 1966.

4. Long, G. G., White, J. D. et al.: Pneumocystis carinii infection in splenectomized owl monkeys. J.A.V.M.A. 167: 651-654, 1975.

5. Seibold, H. R., Munnell, J. F.: Pneumocystis carinii in a pig. Vet. Pathol. 14: 89-91, 1977.

Case II - 77-D-284 (AFIP 1664303).

History: Tissue is submitted from a female Stump-tailed macaque being held in a breeding colony. Soon after regrouping the colony, she sustained numerous deep bite wounds which were considered the immediate cause of death.

Contributor's Diagnosis & Comments: Lung mite (Pneumonyssus semicola) infestation. The lesion is considered an incidental finding.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Bronchiolitis, parasitic, chronic-active, focally extensive, with bronchiectasia, lung, macaque.

Etiologic Diagnosis: Pneumonic pneumonyssiasis.

Etiology: Pneumonyssus semicola.

Comments: Most histoslides clearly demonstrated the lung mites in the ectatic and purulent bronchioles. The parasitized and ectatic bronchioles are often referred to as "mite-houses". In addition to the principal lesion, there was diffuse pulmonary congestion and edema with multiple foci of para-bronchiolar anthracosilicotic pigmentation. Both the anthracosilicotic pigments and digested blood pigments adjacent to the mites were birefringent.

Contributor: Department of Comparative Medicine, The Milton S. Hershey Medical Center, Hershey, PA 17033.

References:

1. Hall, W. B.: Respiratory mite parasites in nonhuman primates. Lab. Anim. Care 20: 402-406, 1970.

2. Chitwood, M., Lichtenfels, J. R.: Identification of parasitic metazoa in tissue sections. Exp. Parasitol. 32: 407-519, 1972.

Case III - 79N943 (AFIP 1727247).

History: This 2-week-old female piglet had patches of roughened and reddened skin over the head and body.

Contributor's Diagnosis & Comments: Cutaneous acariasis compatible with Sarcoptes scabiei. The skin lesions of this piglet were considered an incidental necropsy finding. Unfortunately, the mites were not especiated, but Sarcoptes scabiei was considered to be the most likely agent.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Dermatitis, parasitic, subacute, diffuse, mild, skin, porcine.

Etiologic Diagnosis: Cutaneous acariasis.

Etiology: Consistent with Sarcoptes scabiei.

Comments: Participants unanimously agreed with the contributor's diagnosis. The presence of spines on the dorsal exoskeleton were considered a key feature in identifying this genus of mites.

Contributor: Department of Pathology, School of Veterinary Medicine, University of California at Davis, Davis, CA 95616.

References:
1. Sabiiti, C. K., Wescott, R. B. et al.: Sarcoptic mange in swine in Northwestern United States. J.A.V.M.A. 175: 818-819, 1979.
2. Cargill, C. F., Dobson, K. J.: Experimental Sarcoptes scabiei infection in pigs: I. Pathogenesis. Vet. Rec. 104: 11-14, 1979.
3. Sheahan, B. J.: Experimental Sarcoptes scabiei infection in pigs: Clinical signs and significance of infection. Vet. Rec. 94: 202-209, 1974.

Case IV - VRL A99718 (AFIP 1727250).

History: This spayed female dog lost weight for several weeks. Clinical examination revealed prominent ascites. Clinicopathological data were essentially normal except for a WBC count of 27,000. Laparotomy was performed and tissue from the abdominal cavity is presented.

Contributor's Diagnosis: Chronic disseminated granulomatous peritonitis secondary to Mesocestoides infection.

Attendees' Diagnoses & Comments:

Morphologic Diagnosis: Peritonitis (Omentitis), parasitic, pyogranulomatous, diffuse, severe, abdominal cavity, canine.

Etiologic Diagnosis: Peritoneal cestodiasis.

Etiology: Consistent with Mesocestoides sp.

Comments: Attendees unanimously agreed with the contributor's diagnosis. Sections contained parasites with calcareous bodies which immediately characterize this organism as a cestode. The life cycle of this cestode is complex, as it involves at least two intermediate hosts. Carnivores may serve as an intermediate host, as in this case, or as the final host for the cestode.

Contributor: Veterinary Reference Laboratory, P.O. Box 30633, Salt Lake City, UT 84125.

References:
1. Greve, J. H., Hanson, R. L. et al.: Treatment of parasitic ascites in a dog. J.A.V.M.A. 174: 828-829, 1979.
2. Orthoefer, J. G., Baker, N. F. et al.: Peritonitis due to an intermediate stage of cestode in a dog with lymphosarcoma. J.A.V.M.A. 165: 537-538, 1974.
3. Reid, W. A. and Reardon, M. J.: Mesocestoides in the baboon and its development in laboratory animals. J. Med. Primatol. 5: 345-352, 1976.
4. Williams, J. F., Westheimer, J. et al.: Mesocestoides infection in the dog. J.A.V.M.A. 166: 996-998, 1975.

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DEG/mk
ADDRESS REPLY TO THE DIRECTOR
ATTN: AFIP- CPU-V

30 May 1980

Participants
AFIP Wednesday Slide Conference

This Conference, No. 30, brings an end to the 1979-1980 AFIP Wednesday Slide Conference Series. As Coordinator for this year's Conference, I want to thank you for your participation and cooperation in making this year's program successful.

Concerning this year's Conference I have compiled some data which I thought might be of interest to you:

1. There were 78 individuals and/or institutions listed as contributors. This will be expanded to over 90 contributors in the 1980-1981 conference year.
2. Seven new contributors were added to the list this year, and 9 old contributors were removed. Removal was by the participant's request, or effected because of failure to contribute cases during the previous year.
3. In the 30 weekly conferences, 120 cases were presented. Contributors submitted 141 new cases this year. Nine cases were taken from the teaching files of the Registry of Veterinary Pathology at the AFIP. Seven participants failed to contribute cases this year.
4. Forty-four submissions were duplicates of other contributions in this year or previous years. Unused cases are retained and may be used in next year's conferences.
5. This year, for the first time, many conferences were devoted to themes, such as diseases of an organ, diseases caused by similar etiologic agent, or similar inflammatory responses in an organ system. The purpose was to stimulate discussion of the similarities and differences in host responses to etiologic agents. If contributors have comments, pro or con, about this approach to the Wednesday Slide Conference, I would be happy to receive them.

Within the next few weeks, when compiled, a complete summary and tabulation of this year's conference cases will be forwarded to you.

Captain Timothy P. O'Neill has been selected as Conference Coordinator for the 1980-1981 year. We will be contacting you shortly concerning preparation of cases for next year's program and disposition of unused submitted cases.

Sincerely,

D. Earl Green

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