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Conference Moderator: Dr. Don Nichols, DVM, Diplomate ACVP
Department of Pathology
US Army Medical Research Institute of Infectious Diseases
Fort Detrick, MD

CASE I – 06-1650 (AFIP 3027077).

Signalment: One-year-6-month old gelding donkey.

History: Generalized nodular dermatitis that was present for 2 months. The donkey was clinically normal. Skin biopsies were taken at time of castration.

Gross Pathology: None provided other than the history of generalized nodular dermatitis.

Histopathologic Description: Specimen is skin with numerous protozoan parasitic cysts in the dermis. These cysts contain many bradyzoites within a parasitophorous vacuolar membrane. The parasitophorous vacuoles appear to be surrounded by a layer of hypertrophied fibrocytes with an outer layer of collagen. Many of the cysts are degenerate/necrotic and associated with marked eosinophilic and granulomatous (epithelioid macrophages and multinucleated giant cells) inflammation that occasionally forms eosinophilic granulomas with central eosinophilic "abscesses". There is a widespread multifocal mild lymphoplasmacytic and eosinophilic superficial perivascular dermatitis with mild widespread acanthosis of the overlying epidermis.

Contributor's Morphologic Diagnosis: Multifocal marked eosinophilic granulomatous nodular dermatitis with intralesional protozoal cysts consistent with *Besnoitia bennetti.*

Contributor's Comment: Species of the genus *Besnoitia* are parasites of a wide range of domestic and wild species, including cattle, goats, reindeer, horses, donkeys, opossums, rabbits, rodents, and lizards.[1] *Besnoitia* species are unlike

other members of the coccidian family Sacrocystidae (i.e. *Sarcocystis, Toxoplasma,* and *Neospora*) in that the cysts containing bradyzoites are found mainly within fibroblasts in the skin, subcutaneous tissues, and fascia.[2] *Besnoitia besnoiti*, which infects cattle as the intermediate host, causes significant economic losses in countries outside the U.S. due to condemnation of hides at slaughter. The less common *B. bennetti* affects equids.[1] Over the past ten years, cases of *B. bennetti* have been infrequently reported in donkeys in the United States.[1,3]

Clinical signs of *B. bennetti* in donkeys are common with other forms of besnoitiosis, and include varying intensities of alopecia, lichenification, hyperpigmentation, exudative crusts, anorexia, and lethargy.[3] In donkeys, areas of lichenification and raised dermal nodules were most commonly seen on the head, base of the ears, withers, inner aspects of the hind limbs, and the perineal and perivulvar regions.[1,3] Cysts in the ocular sclera, submucosa of the inner lip, eyelids, and external nares were observed in some affected donkeys.[1] Pruritis was associated with the skin lesions in two published cases.[1,3] Clinical differentials for dermatitis in donkeys include chronic bacterial dermatitis, dermatophytosis, autoimmune dermatoses, sarcoidosis, parasitic dermatitis (i.e. pediculosis), unusual manifestation of multi-systemic disease, and nutritional deficiencies.[3]

Tissue cysts containing *B. bennetti* bradyzoites can reach up to 650 um in diameter and are visible to the naked eye as white to glistening white nodules embedded in the host tissue.[1,3] The thick walls of the cysts are comprised of three layers recognizable by light microscopy. The outermost layer consists of hyaline connective tissue, the middle layer encloses the nucleus and cytoplasmic organelles of the host fibroblast, and the thin, innermost layer represents the parasitophorous vacuolar membrane surrounding myriads of bradyzoites. The bradyzoites vary in size and shape and can be seen by TEM to contain a conoid, micronemes, rhoptries, a nucleus, amylopectine, a mitochondrium, and dense granules. The characteristics of these structures can be used to identify the organism to the genus level, while immunohistochemistry is useful in identifying the species.

Inflammatory responses associated with the cysts can be seen histologically as perivascular to interstitial accumulations of lymphocytes and eosinophils. Ruptured cysts incite a granulomatous response. A study of a *B. bennetti* outbreak in a heard of donkeys reported that degenerating cysts were most common in animals undergoing trimethoprim-sulfamethoxazole treatment.[1] In a study by Davis et al, post-treatment skin biopsies showed the presence of cysts without histological evidence of inflammation, suggesting that chronic infection with *Besnoita* sp. may result in a subclincally affected state in some animals.[3]

Like other coccidian parasites, the *Besnoitia* life cycle involves both a definitive and intermediate host. Domestic cats have been recognized as the definitive host for

the three *Besnoita* species whose life cycles are known.[1] Transmission to the intermediate host involves ingestion of sporulated oocysts in feed or water contaminated by feces of the definitive host. However, only the tissue cyst and bradyzoite states of the *B. bennetti* life cycle have been described. Also, results of studies in donkeys suggest that domestic cats are unlikely to play a role in transmitting the disease. The presence of extracellular bradyzoites in the dermal crusts of infected donkeys lends support to the proposal of an arthropod vector.

AFIP Diagnosis: Haired skin and subcutis: Dermatitis, granulomatous and eosinophilic, multifocal, moderate, with protozoal cysts etiology consistent with *Besnoitia* sp., donkey (*Equus asinus*), equine.

Conference Comment: The contributor provides an excellent summary of *Besnoitia bennetti* in equids. Jubb, Kennedy, an Palmer includes 4 layers that comprise the mature cyst wall: 1) the outermost layer of compressed dermal collagen, 2) a very thin homogenous intermediate zone, 3) the host cell with peripheralized nuclei, and 4) the innermost parasitophorous vacuole filled with crescentic bradyzoites.[4]

Other species of *Besnoitia* and their intermediate hosts are listed below:[4,5,6]
B. besnoiti – cattle[1,4]
B. caprae – goats[1]
B. tarandi – caribou, reindeer, mule deer, musk ox[1,6]
B. wallacei, *B. akodoni* – rodents[1,4,5]
B. jellisoni – deer mice, kangaroo rat, opossum[6]
B. oryctofelisi – rabbits[1]
B. darlingi – opossum, lizard[1,5]

This case was reviewed in consultation with Dr. Chris Gardiner, AFIP consultant in veterinary parasitology.

Contributor: Department of Veterinary Biosciences, College of Veterinary Medicine, The Ohio State University, 1925 Coffey Road, Columbus, Ohio 43210
http://vet.osu.edu/biosciences.htm

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CASE II – GUVS 2 (AFIP 3032057).

Signalment: 6-year-old, female, Mediterranean spur-thighed tortoise, chelonian, *Testudo graeca*.

History: An adult captive bred Mediterranean spur-thighed tortoise (*Testudo graeca*) had a history of urolithiasis five years previously, at which time a urolith of undetermined composition had been removed from the urinary bladder by coeliotomy. After an uneventful recovery, the tortoise remained healthy until April 2006, when it became depressed, lethargic and anorexic over a one month period. On clinical examination in May 2006, it also appeared to be lame in the right forelimb. Haematological examination revealed anaemia and leucopaenia. There were markedly increased plasma concentrations of uric acid and urea, along with hyponatraemia, hypochloraemia and hyperkalaemia. Consistent hyperuricaemia was demonstrated on repeat biochemical examination during the next few days. Radiographic findings were unremarkable. The tortoise was given daily supportive fluid therapy (Critical Care, Leith Petwerks, 5 g powder in 20 ml water), along with enrofloxacin (Baytril 2.5% Oral Solution, Bayer, 10 mg/kg). Despite treatment, it became progressively more lethargic and was euthanased one week later by intracoelomic injection of barbiturate solution.

Gross Pathology: At postmortem examination, the tortoise weighed 521 g and the carapace was 17 cm long and 11 cm wide. There was little body fat. Both kidneys were firm and pale. Extensive pale yellow granular deposits were visible on the capsules and cut surfaces of the kidneys. There were pale yellow granular deposits in multiple appendicular joints, including the right and left coxofemoral, stifle, elbow, carpal and tarsal joints.

Laboratory Results:
Haematology (Reference ranges):
Packed cell volume: 19% (26-43%)
Total leucocyte count: 4.1 x 10^9/L (6.0-48.0 x 10^9/L)
Differential leucocyte count (Reference ranges not available):
Heterophils: 1.6 x 10^9/L
Lymphocytes: 1.2 x 10^9/L
Monocytes: 1.0 x 10^9/L
Eosinophils: 0.0 x 10^9/L
Basophils: 0.4 x 10^9/L

Biochemistry (Reference ranges):
Sodium: 89 mmol/L (111-154 mmol/L)
Potassium: 12.6 mmol/L (2.2-6.1 mmol/L)
Chloride: 79 mmol/L (83-120 mmol/L)
Calcium: 5.0 mmol/L (2.0-3.5 mmol/L)
Phosphate: 0.9 mmol/L (0.5-3.5 mmol/L)
Urea: 129.2 mmol/L (0.2-16.0 mmol/L)
Uric acid: 3252 μmol/L (35-707 μmol/L)
Creatinine: 31 μmol/L (9-218 μmol/L)
Cholesterol: 2.4 mmol/L (0.7-6.1 mmol/L)
Total protein: 33 g/L (13-61 g/L)
Albumin: 11 g/L (5-28 g/L)
Globulin: 22 g/L (Reference range not available)
Aspartate aminotransferase: 327 U/L (18-534 U/L)

Histopathologic Description: The kidney has severe, generalised, urate nephrosis, with deposition of lightly basophilic, radiating, fibrillary, crystalline urates (gouty tophi) in renal tubules and the renal interstitium. There is necrosis of renal tubular epithelial cells and destruction of tubules. Interstitial oedema and fibroplasia are evident. Infiltrates of heterophils are present, especially in areas of necrosis. There are also mild, multifocal interstitial infiltrates of lymphocytes. Barbiturate change is evident at the periphery of the kidney.

Contributor's Morphologic Diagnosis: Kidney: Nephrosis, generalised, severe, with urate deposits (gouty tophi), Mediterranean spur-thighed tortoise, *Testudo graeca*

Contributor's Comment: This tortoise had bilateral urate nephrosis (renal gout) and generalised urate arthritis (articular gout). The renal gout was characterised by deposition of urate crystals (gouty tophi) in the kidney, accompanied by nephrosis and interstitial nephritis. Causes of renal disease in tortoises include dehydration, abnormal diets, viral, bacterial or parasitic infection and nephrotoxins such as aminoglycosides.[1] Many cases of renal gout are idiopathic. Obstruction of the

lower urinary tract in tortoises has been associated with urolithiasis, particularly deposition of urate crystals, and coelomic masses such as neoplasia.[1,2] The cause of the renal gout in this tortoise was unknown, but the history of urolithiasis may be significant, possibly indicating a predisposition to deposition of urate crystals in the urinary tract.

Clinical manifestations of renal disease in this case were depression, lethargy and anorexia, accompanied by anaemia, leucopaenia and elevated plasma concentrations of uric acid and urea. Concentrations of urea and creatinine are not reliable indices of renal disease in tortoises, whereas uric acid concentrations greater than 1000 μmol/l are usually due to renal insufficiency.[1] Elevated uric acid concentrations may also occur in tortoises with dehydration and hepatic disease.[1] Uric acid concentrations greater than 1500 μmol/l are associated with deposition of urate crystals in tissues.

The articular gout in this tortoise was associated with granulomatous inflammation and joint erosions in multiple appendicular joints. Arthritis in tortoises may be caused by deposition of urate crystals (articular gout) or other mineral salts (pseudogout) in or around joints, bacterial infection (septic arthritis) or degenerative joint disease related to trauma or aging. Visceral, articular and periarticular gout are common manifestations of renal disease, but may also be caused by prerenal factors, such as dehydration.[1,3]

AFIP Diagnosis: Kidney: Nephritis, tubulo-interstitial, chronic, diffuse, mild, with marked interstitial fibrosis, tubular loss, and numerous urate tophi (gout), Mediterranean spur-thighed tortoise (*Testudo graeca*), chelonian.

Conference Comment: Gout is the deposition of sodium urate crystals or urates in tissue and occurs in humans, birds, and reptiles (species that lack the enzyme uricase). There are no convincing reports of gout in either dogs or cats. Even in Dalmatian dogs, with their high serum uric acid concentrations, do not appear to develop gout. As previously stated by the contributor, causes of gout include impaired excretion by the kidneys (severe renal disease, postrenal obstruction, dehydration, nephrotoxic drugs) or overproduction of uric acid (high-protein diets) leading to elevated plasma uric acid concentration (hyperuricemia) resulting in the precipitation of urates on many visceral and/or articular surfaces. Additionally, vitamin A deficiency and excess dietary calcium may result in gout.[4,5,6,7,9]

There are two forms of gout in birds and reptiles: visceral and articular. Visceral gout is more common and presents grossly as a thin layer of gray granules or white/gray chalky patches on the visceral serosae, especially the parietal

pericardium and the kidneys. The articular form is rare and is characterized by swollen joints with white chalky deposits in and around joints. The joints of the extremities are most commonly affected.[4,6,8]

Gout is a disease of purine (adenine, guanine) metabolism. Uric acid and urates are the end products of purine metabolism and are eliminated as semisolid urates in birds and reptiles. The degradation of purines to uric acid is outlined in the figure below.[4,8]

Adenine > hypoxanthine > xanthine > uric acid
Guanine > xanthine > uric acid

Both of these pathways require xanthine oxidase to form xanthine from hypoxanthine and uric acid from xanthine.[8]

Microscopically, aggregates of acicular birefringent urate crystals (tophi) or the spaces left after the crystals dissolve during preparation of paraffin embedded histologic sections are pathognomonic of gout and are usually surrounded by numerous neutrophils, macrophages, and giant cells.[4] Since formalin fixation leaches out most of the water-soluble urate deposits, collection of tissues in absolute ethyl alcohol is preferable.

True gout must be distinguished from pseudogout in which crystals other than sodium urate, such as calcium pyrophosphate dehydrate or hydroxyapatite, are deposited in joints. Grossly, pseudogout appears as cream-colored gritty material surrounding the joint capsule. This is in contrast to urates which are found inside the joint capsule and within the synovial fluid. Tophi are not present in pseudogout. Additionally, urates are radiolucent, whereas calcium deposits are radiopaque. True gout affects the kidneys, pericardium, liver, and other internal organs, whereas pseudogout only affects the joints and does not appear to occur in other locations. Pseudogout has been reported in humans, Rhesus macaques, dogs, and turtles.[3,6,8]

Contributor: Division of Pathological Sciences, Institute of Comparative Medicine, University of Glasgow Veterinary School, Glasgow G61 1QH, Scotland, United Kingdom, http://www.gla.ac.uk/faculties/vet

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CASE III – 06-0150-2 (AFIP 3050733).

Signalment: Adult, male, Northern water snake (*Nerodia sipedom*).

History: Refused food one week before death.

Gross Pathology: The small and large intestines are mildly distended to 10 mm in diameter, and the lumen is filled with necrotic debris. The serosal blood vessels are congested. The liver has multiple 2 mm in diameter white plaques. There are many up to 5 x 2 x 2 mm dark brown to black nodules within the hepatic and renal parenchyma and overlying the capsules.

Gross morphologic diagnoses:
1. Necrotizing enteritis, colitis, gastritis and hepatitis.
2. Pentastome parasites within liver and kidney.

Laboratory Results: Heart blood culture: *Providencia ruttgeri*

Cytology (stomach, fecal material), fecal screening: Amoebic trophozoites; minimal histiocytic inflammation the gastric sample and none in feces

Histopathologic Description: Intestine: Diffusely and transmurally there is necrosis of all sections on the slide. The lumen is filled with necrotic debris and the mucosa is replaced by eosinophilic and karyorrhectic debris mixed with cross sections of cestodes with calcareous corpuscles, and many bacteria. The submucosa, tunica muscularis and serosa contain numerous up to 30 um in diameter amoebic trophozoites, eosinophilic and karyorrhectic debris and some viable macrophages and fewer heterophils.

Contributor's Morphologic Diagnosis: Small intestine and large intestine: Enteritis and colitis, necrotizing, transmural, subacute, marked with many amoebic trophozoites and some cestodes.

Contributor's Comment: Amoebiasis in reptiles, caused by *Entamoeba invadens*, is an important disease in captive snakes, lizards, and chelonians. The disease is characterized by ulcerative colitis and hepatitis, and is one of the most common gastrointestinal diseases in snakes.[2] *E. invadens* is morphologically, or biologically similar to *E. histolytica* in humans, but the two species are distinguished by the host species and temperature tolerance.[5] *E. invadens* prefers an optimum temperature of 80-84.2 degrees F and cannot be transmitted to warm-blooded animals.[4] The biological host for *E. invadens* is thought to be the herbivorous turtles, in which a symbiotic relationship without any pathogenicity may be observed. In the intestine of turtles, amoebic protozoa take nourishment from ingested plants to form cysts, and complete their life cycle without being pathogenic to the host. However, in the intestine of the other carnivorous reptiles, the amoeba is unable to take the specific nourishments it requires, and has to invade the intestinal mucosa to survive, resulting in harmful infection of the host.[5] The immune status of the host also plays a role in protozoal pathogenicity.[2]

This protozoa moves and feeds by forming pseudopodia, changing shape while in the trophozoite state. The cyst is a resting stage in which a wall is produced by the trophozoite to encapsulate and protect the parasite while it is in the abiotic environment.[2]

The characteristic microscopic lesions induced by *E. invadens* are severe intestinal erosion, ulceration, and inflammation often with a fibrinonecrotic pseudomembrane. The ileum and colon are the most severely affected intestinal segments. Hepatic necrosis can also be present.[1]

This water snake had classic lesions of amoebiasis, including enteritis and gastritis. The presence of organisms and necrotic lesions within other organs (liver, kidney, pancreas, spleen, epididymis, stomach) was a result of blood invasion and spread.

There are several genera of cestode parasites which affect reptiles. *Acanthotaenia, Crepitdobothrium, Ophiotaenia, Spirometra, Bothriocephalus, and Bothridium* are described affecting snakes. *Ophiotaenia* is the most common in North American snakes and is acquired by ingestion of infected frogs.[3]

This snake was also infected with pentastomes, a relatively common snake lung parasite which can be found in a nymph stage within other tissues. This parasite has an indirect life cycle that involves fish, amphibians or mammals. Heavy infestations in snakes may cause respiratory problems. No adult pentastomes were seen in the lungs of this individual, but hyperplasia of the pulmonary smooth muscle could be related to the previous presence of pentastomes within the lumen. Parasites were present in the liver and kidneys.

A pure culture of *Providencia ruttgeri*, an opportunistic organism in reptiles, grew from heart blood culture. Bacterial septicemia is a common finding in snakes infected with *Entamoeba invadens* as erosion of the intestinal mucosal barrier allows passage of bacteria from the gut lumen into the bloodstream.

No bacteria were isolated from the liver, and no *Salmonella* or *Shigella* grew from intestinal culture.

AFIP Diagnoses: 1. Intestine: Enteritis, necrotizing, transmural, acute to subacute, diffuse, severe, with fibrin, edema, and numerous amoebic trophozoites, Northern water snake (*Nerodia sipedom*), reptile.
2. Intestine: Intramural cestodes, few.
3. Intestine: Intramural pentastomes, few.

Conference Comment: The contributor provides a thorough summary of *Entamoeba invadens* as well as providing additional information about cestodes that parasitize reptiles and pentastomes in snakes.

Entamoeba histolytica causes amebic dysentery in humans and nonhuman primates (especially Old World), and rarely infects other species (dogs, cats, pigs, cattle). Many infections are asymptomatic. Microscopically, flask-shaped ulcers in the colon, with a narrow neck through the mucosa and a broad base in the submucosa are characteristic. Trophozoites may disseminate to other organs, especially the liver and brain, where they form amebic abscesses. A distinctive feature of the

necrotizing lesions is the almost complete lack of a cellular inflammatory response.[6,7,8,9]

Amebic trophozoites and cysts are PAS and GMS positive.[5,8]

Contributor: Department of Pathology, National Zoological Park, 3001 Connecticut Ave NW, Washington D.C. 20008

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CASE IV – 040166 43 (AFIP 3027300).

Signalment: Three-year-old, female, Nubian goat (*Capra hircus*).

History: This animal was part of a herd of goats used for the production of antibodies to various antigens. The goats are housed outdoors on pastures and have free access to open sheds for shelter.

This goat arrived at USAMRIID on 30 December 2003 and was placed in a separate pasture away from the resident herd for a 30-day quarantine period. It appeared to be healthy during a physical exam 6 days after its arrival. However, 25 days later, the goat was noted to be vocalizing and having difficulty walking. When it did walk, it consistently circled to the left. Physical examination at this time revealed that its body temperature, pulse, and respiration rate were unremarkable. The goat was placed in an indoor stall and treatment with an anti-inflammatory drug (flunixin meglumine) and antibiotics (enrofloxacin and ampicillin) was begun.

Although the animal ate and drank normally, there was no improvement in its locomotion. Two days after initial clinical presentation, the goat also had left horizontal nystagmus. The following day, it was noted to be repeatedly rubbing an area of alopecia and scaliness on the right side of the neck and shoulder.

On the fourth day after clinical presentation, the goat was recumbent and unable to rise. Euthanasia was then performed and the carcass was submitted for a complete necropsy

Gross Pathology: The only lesion noted grossly was a large area of alopecia over the right shoulder; within this area there was cutaneous ulceration covered by a scab.

Laboratory Results: Hematology and serum chemistry results from blood samples collected on the first day of clinical signs and just before euthanasia were unremarkable.

Serology results on samples collected on the day of euthanasia were negative for antibodies against caprine Arthritis-Encephalitis virus and pseudorabies virus.

Histopathologic Description: Spinal cord (transverse and sagittal sections): Multifocally within the white matter there are degenerate axons (characterized by axonal swelling and increased eosinophilia) and spongiform change caused by axonal loss and myelin sheath ectasia, often accompanied by astrocytic hypertrophy and hyperplasia. Multifocal necrosis and microcavitation of the white matter is also present and is accompanied by infiltrates of low to moderate numbers of macrophages with abundant foamy cytoplasm (gitter cells); these lesions are more abundant and prominent in slides from histology block 42. Multifocal perivascular infiltrates of lymphocytes, plasma cells, and fewer macrophages are present in the meninges and white matter – with occasional extension into adjacent gray matter; numbers of such inflammatory cells vary from low to moderate to numerous.

Contributor's Morphologic Diagnosis: Spinal cord: Multifocal axonal degeneration and loss, moderate, with astrocytosis, white matter necrosis and histiocytic inflammation, and nonsuppurative perivascular meningomyelitis

Contributor's Comment: In addition to the lesions in the spinal cord, multiple foci of white matter necrosis with cavitation, gitter cell infiltration, and nonsuppurative perivascular inflammation are also present within the brain. Within the white matter of the cerebrum, a single nematode parasite was found. The morphology of this nematode is characteristic of a metastrongyle and is consistent with *Parelaphostrongylus tenuis* (*P. tenuis*).[1]

The normal hosts of *P. tenuis* are white-tailed deer (*Odocoileus virginianus*). Adult worms reside in the subarachnoid space of the brain or spinal cord of the deer; this location is the basis for a common nickname for *P. tenuis*: "the meningeal worm".[2] Eggs produced by female worms may be carried by the venous circulation to the lungs where they hatch or the eggs may hatch on the meninges and then the first-stage larvae enter the venous circulation to be carried to the lungs.[3] Either way, first-stage larvae in the lungs migrate up the bronchial tree and are coughed up, swallowed, and eventually passed in the feces. A wide variety of gastropod species can serve as intermediate hosts in which the larvae mature to third-stage larvae. Deer become infected when they accidentally ingest gastropods containing third-stage larvae. These larvae are released from the gastropod tissues in the abomasum and they then migrate into the peritoneal cavity, eventually following spinal nerves to enter the dorsal horn of the spinal cord.[2,4] Worm maturation to the adult stage occurs within the spinal cord in 20-30 days and the parasites then follow the dorsal nerve roots to enter the subarachnoid space.[2]

White-tailed deer are well-adapted hosts for *P. tenuis* and rarely display any clinical signs associated with infections. However, serious to fatal neurologic disease due to *P. tenuis* infection has been reported in numerous other ruminant and ruminant-like species.[2,4,5] Among domestic species, cattle are highly resistant to infection whereas llamas are particularly susceptible.[2] Goats and sheep appear to be intermediate in susceptibility.

The neurologic disease associated with *P. tenuis* infections in non-adapted hosts is caused primarily by prolonged and aberrant migration by the parasites within the spinal cord and/or brain; CNS damage secondary to the associated inflammation also plays an important role.[2,4] Trauma caused by the migrating worms produces multifocal necrosis and cavitation, sometimes with acute hemorrhage, followed by inflammation and axonal degeneration adjacent and distal to these foci. The inflammatory cell infiltrates usually consist primarily of lymphocytes, macrophages, and plasma cells, but may also include eosinophils.[2,3,5]

Clinical signs typically present initially as paresis or paralysis of one or more limbs – usually beginning with the hindlimbs – and reflect spinal cord injury. These may progress slowly or rapidly to recumbency; however, some animals may remain static or even recover.[2-4]

In goats, infection of the brain has been reported to cause circling – as in this case.[3] Larval migration through dorsal nerve roots has caused pruritus leading to self-trauma in goats.[3] Although nerve root involvement was not documented in this case, the goat was noted to have an area of apparent pruritus over the right shoulder with secondary alopecia and cutaneous ulceration from self-trauma.

A tentative diagnosis of cerebrospinal parelaphostrongylosis may be made based on clinical signs, possible exposure to infected gastropods, and/or typical CNS lesions. However, the definitive diagnosis currently requires finding larvae with the appropriate morphology within the CNS; this is often difficult due to the low numbers of parasites present in aberrant hosts. An antigen-capture ELISA for detecting *P. tenuis* antigens in the cerebrospinal fluid has been developed but it is not yet commercially available.[2]

According to the vendor from which this goat was purchased, this animal was housed indoors and fed a commercial pelleted diet for 9 months before its shipment to USAMRIID; these conditions mean it is highly unlikely that the goat was infected at the vendor's facility. In natural *P. tenuis* infections of goats, the latest reported onset of clinical signs is 9 weeks after the animals were removed from pasture; therefore the possibility that this goat was infected before its being housed indoors at the vendor's facility is remote.[3]

This animal initially presented with clinical signs of neurologic disease 31 days after its arrival and placement into the outdoor pasture. Experimental studies of white-tailed deer have shown that *P. tenuis* can reach the spinal cord in as little as 6 days after ingestion of third-stage larvae.[2] Experimental infection of goat kids produced neurologic signs 11 to 52 days after infective larvae were inoculated into the peritoneal cavity.[3] Therefore, the timing of the development of clinical disease in this case appears to be consistent with infection after its arrival.

Although white-tailed deer had not been seen in the pasture where this goat was housed at the time it developed initial clinical signs, a high deer population is present in the fields that adjoin this pasture and *P. tenuis* infection is highly prevalent in deer from this area of Maryland. The goat's pasture and the surrounding fields are low-lying and moist; these conditions favor gastropods and transmission of *P. tenuis* to the intermediate hosts.[3] This goat most likely became infected when it ingested a snail or slug that had been infected in the adjacent fields and then crossed over into the goat pasture.

Measures that can be taken to try to control problems with *P. tenuis* infections include reducing deer populations and deer access to pastures. Reducing habitats favorable to gastropods – especially along fence lines – can also be beneficial. Frequent de-worming of livestock during seasons when gastropods are active is also recommended in order to kill migrating third-stage larvae before they can reach the CNS.[2]

AFIP Diagnosis: Spinal cord, white matter: Axonal degeneration and loss, diffuse, marked, with digestion chambers, gitter cells, and mild lymphocytic meningomyelitis, Nubian goat (*Capra hircus*), caprine.

Conference Comment: The contributor provides an extensive and thorough review of *Parelaphostrongylus tenius*. Residents considered caprine arthritis encephalitis (CAE) virus and copper deficiency as differentials for this case.

Aberrant larval migration of nematode parasites through the CNS is referred to as cerebrospinal nematodiasis. Grossly, nematode larval migration often appears as necrohemorrhagic serpentine or linear tracts in the affected tissue. Included below is a chart of nematodes that cause central nervous system disease in animals.[6]

Parasite	Normal Host	Aberrant Host
NEMATODE MIGRATION IN ABERRANT HOST		
Angiostrongylus cantonensis	Rat	Dog
Baylisascaris procyonis	Raccoon	Dog
Elaphostrongylus rangiferi	Reindeer	Sheep, goat
Parelaphostrongylus tenius	Deer	Sheep, goat
Setaria digitata	Cattle	Sheep, goat, horse
ABERRANT NEMATODE MIGRATION IN NORMAL HOST		
Angiostrongylus vasorum	Dog (coyote)	
Dirofilaria immitis	Dog (cat)	
Stephanurus dentatus	Pig	
Strongylus spp.	Horse	

This case was reviewed in consultation with Dr. Chris Gardiner, AFIP consultant in veterinary parasitology.

Contributor: U.S. Army Medical Research Institute of Infectious Diseases (USAMRIID), Pathology Division, 1425 Porter Street, Ft. Detrick, MD 21702-5011
http://www.usamriid.army.mil/

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**Opinions, interpretations, conclusions, and recommendations are those of the author(s) and are not necessarily endorsed by the U.S. Army.

Michelle E. Thompson, DVM
Captain, Veterinary Corps, U.S. Army
Wednesday Slide Conference Coordinator
Department of Veterinary Pathology
Armed Forces Institute of Pathology
Registry of Veterinary Pathology*

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