Diagnosis

1a. Morphologic diagnosis: Spinal cord (subarachnoid space [T11-L7]), focally extensive varicose veins with thrombosis and intralesional nematodes, male, mixed breed, young adult cat.

1b. Morphologic diagnosis: Spinal cord (meninges [T11-L7]), multifocal, chronic, mild lymphocytic and eosinophilic leptomeningitis with intralesional nematodes, male, mixed breed, young adult cat.

1c. Morphologic diagnosis: Spinal cord ([T11-L7]), multifocal, moderate wallerian degeneration, male, mixed breed, young adult cat.

2. Etiologic diagnosis: Meningeal parasitary vasculopathy.

3. Etiology: Gurltia paralysans.


Typical gross findings: At necropsy all affected cats showed variable degrees of pelvic muscle atrophy that was easily observed after removal of the skin. In the cat of this report, the muscle atrophy was so severe that could be observed even without removing of the skin. Two out of three cats showed cutaneous ulcers partially covered by crusts in the metatarsal and digital areas. The duramater between T11 and L7 was markedly reddened (Fig.1). Initially this finding was interpreted as subarachnoid hemorrhage, but a closer view of the affected area revealed that it consisted of numerous small blood vessels in a typical varicose pattern similar to a vascular hamartoma. These varicosed vessels did not extend into the nervous tissue within the examined sections.
**Typical microscopic findings:** Marked distension of the subarachnoid spaces caused by multifocal, dilated and tortuous channels that were partially or completely occluded by blood or thrombi was observed between T11 and L7 (Fig. 2). These channels were lined by a single layer of flattened cells with indistinct cytoplasm and nuclei composed of aggregated chromatin (vascular endothelium [tunica intima]). These endothelial cells were arranged over a fine, almost imperceptible layer of smooth muscle cells (tunica media) that were in turn surrounded by a fine layer of fibrous connective tissue with small numbers of fibroblasts (tunica adventitia). These morphologic features were consistent with those of blood vessels. The absence of a lamina elastica interna and externa on HE-stained slides indicated that the described vessels were veins, and their small size suggested that they were venules. In some sections, these venules showed mild smooth muscle hypertrophy, moderate adventitial fibroplasia, and marked subintimal fibrosis (phlebosclerosis). These changes were severe enough to cause concentric thickening and complete luminal obstruction in some of the affected venules. Other venules contained multiple, endothelial-lined, intraluminal papilliform projections supported by collagen stalks, giving an arboriform aspect to the vascular lumen (Fig. 3). Thrombi in different stages of organization were observed obstructing normal and dilated venules in these areas. These thrombi contained either small, narrow channels between the vascular wall and the thrombus (initial neovascularization) or newly formed and stenotic blood vessels with a thick wall containing abundant fibrous connective tissue (Fig. 4). Randomly, there were multifocal areas of perivascular accumulation of lymphocytes (Fig. 5) and fewer mature eosinophils, with rare plasma cells and macrophages. Serial histological sections revealed intravascular cross sections of nematodes with approximately 200 μm in diameter, pseudocoelom, fine eosinophilic cuticle, coelomyarian musculature, digestive tract composed of multinucleated cells, lateral chords, and separated sexual organs, with females having two ovaries (Fig. 6). Based upon these morphologic features, nematodes were classified as metastrongyles, and considering the affected site (meninges) and animal species (cat), they were presumptively identified as Gurltia paralysans. The adjacent spinal cord had variable degrees of wallerian degeneration characterized by areas of axonal swelling (axonal spheroids) (Fig. 7), axonal fragmentation, digestion chambers, and microcavitation. Varicose veins were also observed throughout the white matter, but always close to the surface and associated to the meninges.

**Discussion:** Parasitism by *G. paralysans* (Order Strongylida, Superfamily Metastrongylidea, Family Angiostrongylidae) in the spinal veins of cats was initially described in Chile by Dr. Kurt Wolffhügel in 1933 (Wolffhügel 1933) and referred to as feline crural parasitic paraplegia (Wolffhügel 1934). This condition has not been often cited in textbooks of veterinary pathology (Jones et al. 2000) or parasitology (Levine 1968, Bowman et al. 2002), and therefore remains unknown by many veterinary clinicians, pathologists, and parasitologists. The disease remained forgotten since its first description, but additional cases have been described in Chile in the last few years (Gómez et al. 2010, Moroni et al. 2012). Apparently, cases of the disease used to be restricted to Chile, but a few cases were
reported in Uruguay (Rivero et al. 2011) and Argentina (Guerrero et al. 2011) in 2011. Evidences of a possible occurrence of *G. Paralysans* infestation outside South America have been reported in Ithaca, NY by Dr. Georgi at Cornell University in 1933, but this case has never been published and has been only mentioned in textbooks of veterinary medicine (Bowman et al. 2002). Although very little is know about the pathogenesis of *G. Paralysans* infestation, it has been proposed that domestic cats are accidental hosts and that a wild felid (*Leopardus guigna*) locally known in Chile and Argentina as *guiña* and elsewhere as *kodkod* is the definitive host that bears the adult nematode in the lungs (Wolffhügel 1933). This is the first report of feline crural parasitary paraplegia in Brazil, but it seems that the disease has been affecting cats in the South part of the country for the last 20 years.

The severe vascular dilation and tortuosity associated to the presence of thrombi, compensatory changes such as medial hypertrophy and adventitial hyperplasia, and reparative changes such as phlebosclerosis were features utilized to morphologically classify the described vascular changes as varixes, also referred to as varicose venules, microvarixes, telangiectasia, reticular veins, or vascular spiders. By definition, a varix is a venous vascular dilation that occurs primarily or secondarily to an insulting event. Primary varixes are a multifactorial condition commonly affecting humans. Secondary varixes occur in decurrence to vascular obstruction mainly caused by venous thrombosis and consequent blood stasis (Nicolaides 2000). It is probable that the thrombi and the nematodes that partially or completely obstructed the venular lumina led to blood stasis and increased vascular pression in the cat of this report, predisposing to the occurrence of varicose changes, similar to what is described in humans affected by myelopathy and radiculopathy secondary to spinal intradural varicose veins (Moonis et al. 2003, Paldor et al. 2010).

Although varicose veins are one of the most common vascular problems in humans, they are rarely described in veterinary medicine, and hence this changes are rarely if ever mentioned in veterinary pathology textbooks (Maxie 2007, McGavin & Zachary 2007). The most common examples of varicose veins in humans include those occurring in the legs and in the hemorrhoidal plexus (hemorrhoids), but several other locations have been described, including meningeal varicose veins (Moonis et al. 2003, Paldor et al. 2010) similar to those described in these cats. The rare descriptions of varicose veins in the veterinary literature include those occurring in the eye (Adkins et al. 2005), urinary bladder (Littman et al. 1990), and esophagus (Bertolini et al. 2007) of dogs, although in this species esophageal varicose veins do not occur secondary to portal hypertension (Germain 1975), unless in experimental cases (Tamiya & Thal 1960, Ross et al. 1961, Jensen et al. 1983). Other reports of varicose veins in animals include those affecting the reproductive tract, such as testicular veins (varicocele) of stallions, bulls, rams, and dogs (Ezzi et al. 1987, Foster et al. 1989), uterine veins of mares (Foster et al. 1997) and rabbits (Bray et al. 1992), and vaginal veins of mares (White et al. 1984). To the best of our knowledge there are no descriptions of varicose veins in cats.

This diagnostic exercise had as its primary objective to describe a case of parasitary infestation rarely reported in veterinary medicine, and also to call attention to the sporadic occurrence of varicose veins in animals, a
fact that explains why the veterinary pathologist, in contrast to the human pathologist, may be unaware of the morphologic features of this change.

References and Recommended literature:


Please send your comments/questions to the whole LCPG list by hitting “reply to all”.

A final document containing this material with answers and a brief discussion will be posted on the C. L. Davis website by the end of the current month (http://www.cldavis.org/lcpg_english.html).