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NPMS 2023, Case # 1

Authors: Lydia M. Hall, DVM; Jaime Landolfi, DVM, PhD, DACVP

History/Signalment: 42-year-old, male silverback gorilla (*Gorilla gorilla*) in managed care at a U.S. zoological institution. This individual had been clinically managed for multiple mild chronic-degenerative/age-related diseases including osteoarthritis, sinusitis related to nasal mites, and subclinical bilateral atrioventricular valvular insufficiency and atrial enlargement. He was housed with multiple conspecifics in an indoor exhibit who were all offered a similar diet with no recent novel enrichment items. In the summer of 2022, he presented to the veterinary team with a 1-day history of lethargy, cough, and hyporexia. Over the next 48-hours, he developed profuse mucoid nasal discharge, became minimally responsive, and began to show tremors of the hands and head. No other co-housed conspecifics were affected. During darting for emergent care, he became unresponsive with cardiopulmonary arrest within minutes. Resuscitative efforts were ineffective, and he was submitted for necropsy examination.

Gross: Relevant gross findings were minimal and included mild laryngeal air sacculitis, bilateral rhinitis, and thoracopulmonary changes including hemorrhage and congestion. At the time of gross exam, lung changes were attributed to terminal trauma associated with cardiopulmonary resuscitation attempts. Additionally, this individual also had mild to moderate musculoskeletal and cardiovascular changes consistent with clinically managed disease and geriatric status. No gross lesions were detected in the central nervous system.

Histologic Description: Regionally in the medulla (represented in the submitted slide) and pons and extending into caudal portions of the thalamus, the brain has multiple, small, often coalescing, nodular aggregates of many neutrophils and fewer macrophages (microabscesses). Affected areas have mild to marked loss, fragmentation and/or vacuolization of neuropil (rarefaction), small amounts of admixed cellular debris, moderate numbers of macrophages with slightly foamy cytoplasm (gitter cells), and moderately increased numbers of glial cells (gliosis). Few axon sheaths are dilated and contain a swollen axon (spheroid). Several neurons are hypereosinophilic and shrunken with angular margins and karyolysis (necrosis). Many intralesional vessels are lined by plump and rounded endothelial cells (reactive hypertrophy) and cuffed by moderate numbers of neutrophils, macrophages, and lymphocytes. Changes are most severe and extensive in caudal portions of the brain with bilateral distribution. In more cranial areas, lesions are minimal to mild and unilateral. In some distant sections of cerebral cortex and midbrain, few random vessels are cuffed by low to moderate numbers of lymphocytes, plasma cells, macrophages and rare neutrophils. Diffusely, meninges as well as the Purkinje layer of the cerebellum are expanded by moderate amounts of colorless space (presumed edema versus artifact). Throughout the brain, many neurons have small to moderate amounts of golden to dark brown, granular intracytoplasmic material (lipofuscin).

Many sections of lung are affected by a severe inflammatory process centered on medium-caliber airways and associated with intra-airway foreign material (aspiration bronchopneumonia). Many bronchioles are filled with abundant cellular debris, numerous necrotic and fewer viable neutrophils, macrophages, variable amounts of eosinophilic, homogenous to clumped fibrillar material (proteinic fluid and fibrin), clusters of mixed bacteria and/or fragments of irregular linear to angular, light brown, refractile and birefringent foreign (plant) material. Segmentally, bronchiolar epithelium ranges from attenuated to absent (ulcerated) or is obscured by inflammation. Inflammation extends into and partially to completely obscures surrounding alveoli and interstitium. Alveolar septa range from inapparent to hypereosinophilic and smudged with karyorrhexis (necrosis). Alveoli are filled with numerous neutrophils, fewer foamy macrophages, scattered extravasated erythrocytes (hemorrhage) and small amounts of proteinaceous fluid. In bordering areas, alveoli are dilated with abundant proteinaceous fluid and hemorrhage amongst fewer neutrophils and foamy macrophages. Few larger bronchioles/bronchi contain similar luminal inflammatory debris.

Morphologic Diagnoses:

1. Brain (medulla, pons, caudal thalamus): Encephalitis, necrosuppurative, multifocal, subacute, severe with microabscesses (rhombencephalitis)
2. Lungs: Bronchopneumonia, necrosuppurative, multifocal, acute, with intralesional foreign (plant) material and mixed bacteria (aspiration pneumonia)

Comment: Pathologic processes in the brain and lungs were instrumental in this gorilla's clinical course and ultimate demise. Changes in the lung were consistent with aspiration. The character of inflammation and associated changes indicated bronchopneumonia was acute; thus, aspiration was presumed a consequence of preexisting encephalitis. Encephalitis was characterized by a caudal, mostly brainstem, distribution and necrosuppurative inflammation with neuropil microabscesses. The character and distribution of the encephalitis was highly suspicious for an infectious etiology, and listeriosis was an important differential.[1,3,6] Subsequent ancillary testing was pursued and included Listeria monocytogenes PCR on fresh frozen cerebral frontal cortex, generic bacterial (16s) PCR on formalin-fixed, paraffin-embedded sections of affected brain, immunohistochemical labeling for several antigens (Listeria sp., Sarcocystis sp. Toxoplasma gondii., West Nile virus), histochemical staining to screen for bacteria (Gram, Ziehl-Neelsen), as well as examination of over 50 additional sections of brain. Additional investigation was unable to confirm the cause of the encephalitis. Several differentials remain. Paucimicrobial listeriosis was considered, with the inability to detect Listeria sp. antigen or DNA attributable to insufficient sensitivity of assay and/or inaccessibility of assay target due to formalin-fixation. Given the absence of metazoa, protozoa, or bacteria in examined sections, these differentials were considered less likely but could not be ruled out. Viral encephalitis was a possibility; however, a lack of illness in other conspecifics and indoor setting for this individual decreased the suspicion for a viral etiology.[2,5] Lastly, in the absence of any confirmed infectious cause for the described brainstem encephalitis, an idiopathic, immune-mediated pathogenesis for the lesion was also considered. Notably, a high percentage of brainstem encephalitis cases in humans is attributed to immune-mediated disease.[4,8,10]

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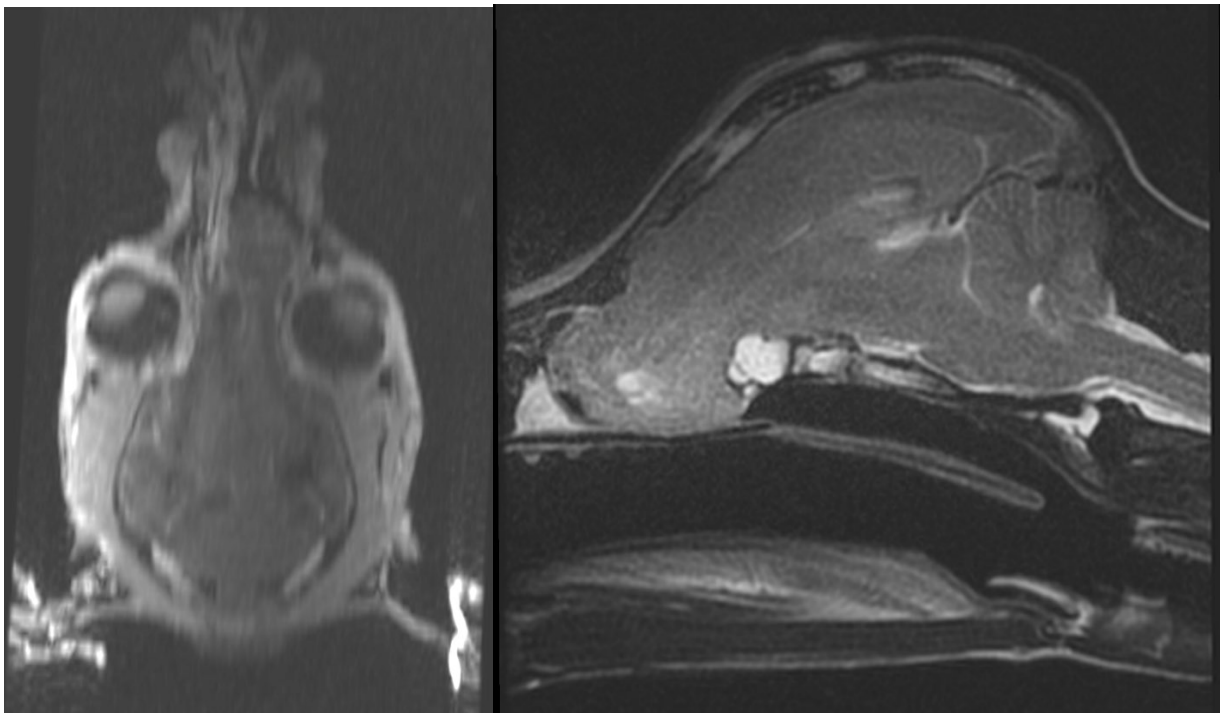
2023 NPMS Case 2

Authors: Gayle C. Johnson, DVM, DACVP, PhD (University of Missouri); Chris Levine, DVM, DACVIM (Neurology); Levine Veterinary Neurology, Bradenton, FL

History/Signalment: Stanley, a 7-month-old, neutered male Jack-Russel Terrier cross, was presented for evaluation of acutely increasing cluster seizures and a chronic history of left sided circling. About 3 months ago, Stanley was adopted from a shelter, where similar signs had been observed, but there was improvement in his new home. On the day of presentation, Stanley experienced numerous cluster seizures that consisted of full body convulsions and vocalizing (a total of 20 seizures, each lasting about 2-3 minutes). Stanley had a hypermetric gait, compulsive left-sided circling, and an absent menace response bilaterally. There was anisocoria present, with the left pupil larger than the right. Rhinorrhea was noted. A cisternal CSF sample revealed low grade mixed inflammation. Images of the brain are shown.
A postsurgical biopsy was submitted.



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Surgical Procedure: A large mass blended to the anterior brain and extended into the nasal cavity, especially on the right side, resting on the top of the hard palate. The connection between the brain and this mass was wide and poorly defined. Ethmoid and nasal turbinates were poorly developed. Surgical excision of the protruding tissue involved making a triangular flap over the dorsal nasal cavity, then placing the patient in dorsal recumbency. A balloon was placed beneath the mass. As it was slowly inflated, much of mass was returned to the calvaria. The tissue that remained attached to the nasal cavity was divided from the anterior brain. The dura was closed to cover non-herniated tissue and the residual brain and meninges were dissected, fixed in formalin and submitted as a biopsy. The dog was discharged after 2 days hospitalization.

Histopathology: Multiple areas from the mass were prepared for examination. Several specimens were bordered by a segment of ciliated respiratory epithelium (Fig. 1, HE). In illustrated section, the epithelium (arrow) covers a layer of dense, cell-poor collagen reminiscent of dura. Between the dura and the brain is less dense fibrous tissue containing blood vessels (arrowhead). Masson trichrome better defines this layer. Mildly increased collagen is also present around penetrating blood vessels derived from that layer (Fig. 2). Neurons were readily visible in sections of brain but did not have an arrangement suggestive of the olfactory lobe (Fig. 4).

Fig. 1

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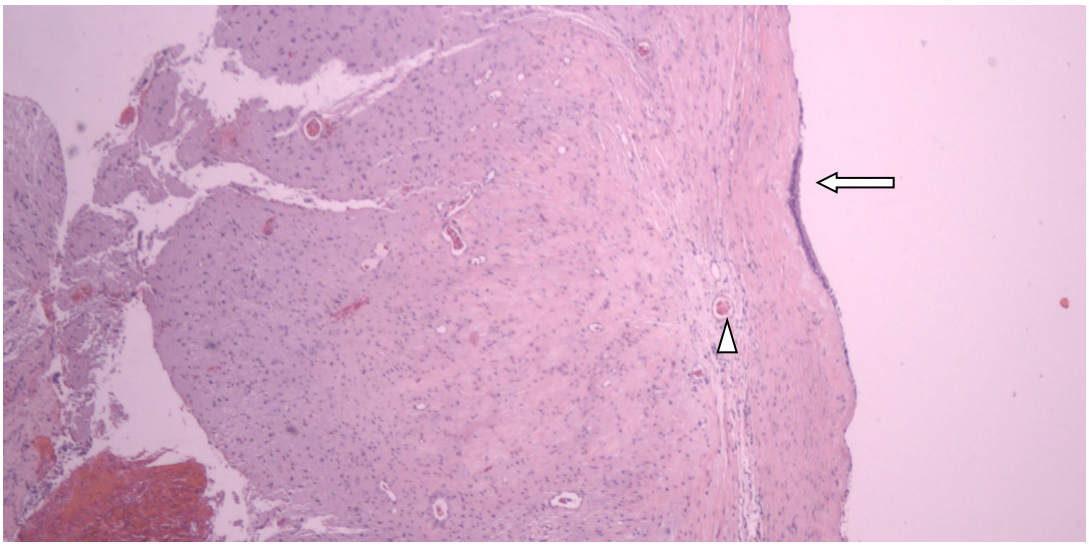
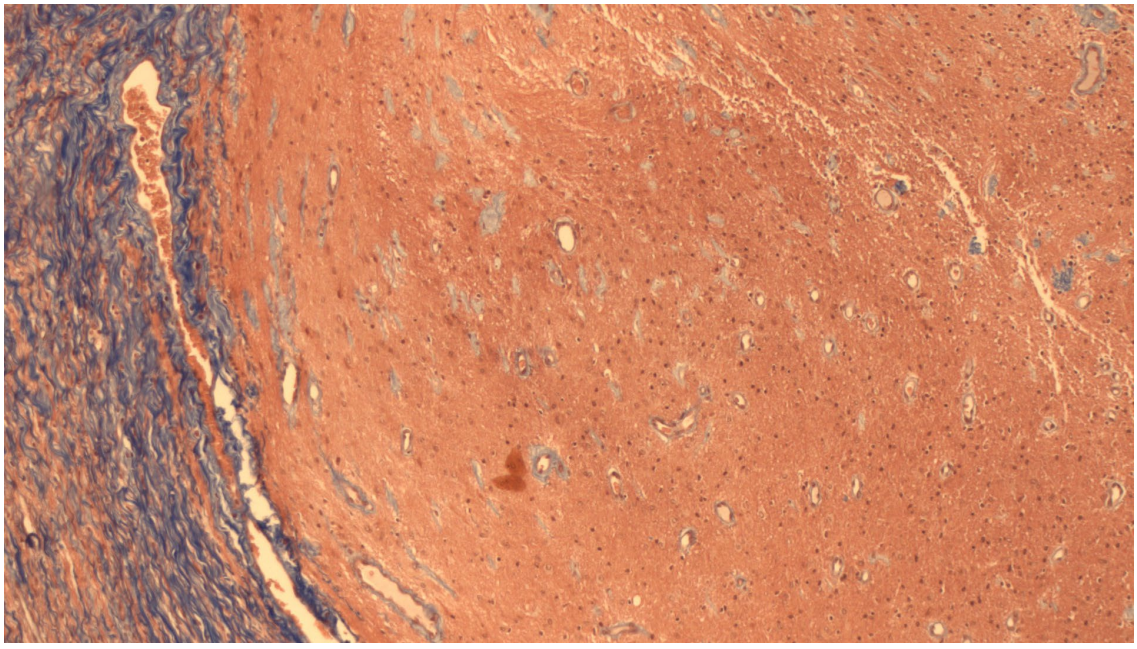


Fig. 2.



Neurons were visible in the brain tissue and stained positively with NeuN immunohistochemically (Fig 3, 4). GFAP staining was surprisingly mild along the pial surface and was present in the brain.

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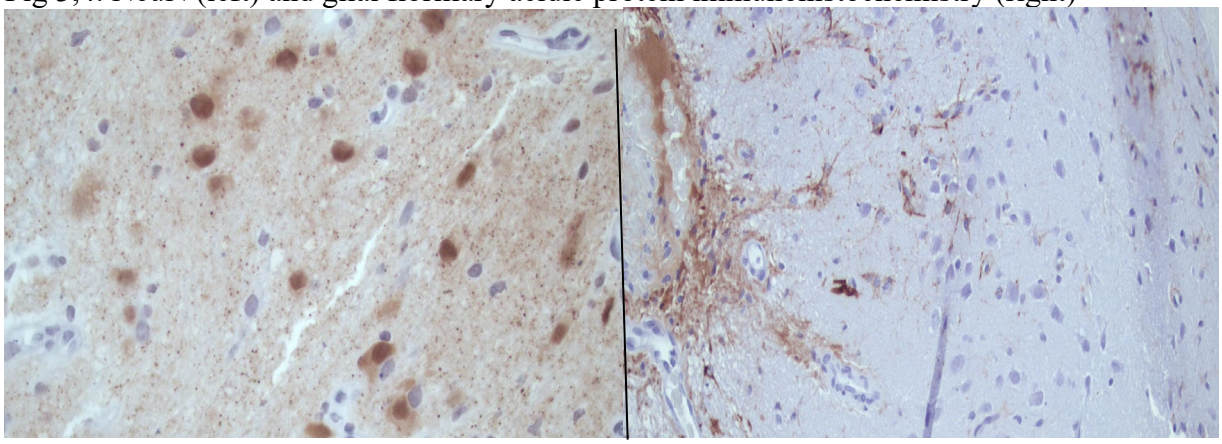
Fig 3,4. NeuN (left) and glial fibrillary acidic protein immunohistochemistry (right)



Diagnosis: Nasoethmoidal dysplasia with brain herniation

Diagnosis: Meningoencephalocele

Discussion: Meningoencephalocele is thought to be a result of partial anterior neuropore failure after neural tube closure, compared to the more severe cranial agenesis seen in anencephaly. The condition is uncommon in dogs but may be clinically silent throughout life[1]. The nasoethmoidal location is the most common type in dogs and humans.[1,2] The largest canine case series consisted of 22 dogs, 1 month-8 years of age. Of these dogs, 77% had generalized seizures (either with unilateral or bilateral lesions) and 31% had abnormal behavior, most commonly aggression.[1] With only medical treatment by anti-epileptic drugs, 11 dogs responded with reduced seizures, 9 were euthanized and 2 were lost to follow-up.

In two case descriptions, surgically treated nasoethmoidal meningoencephaloceles in adult dogs resulted in a reduction in seizures in one case.[3,4]

Patient seizures are thought to result from traction or compression of misplaced cortex, hemorrhage, white matter degeneration, and/or inflammation, all can contribute to cerebrocortical excitability.[1] The clinician must weigh the risk of ascending meningitis if rhinorrhea or intractable seizures are present vs. the surgical risk.

The classification of meningoencephalocele[5] has recently been revised, based on the location of the lesion:

1. Ethmoidal-nasal: may be above or below the nasal bones
2. Cranial Located: Herniation through the calvaria, subdivided into occipital, parietal, frontal, temporal and acrania
3. Orbital: lateral herniation displaces the globe anteriorly or posteriorly
4. Cleft: through any existing craniofacial cleft, and
5. Basal: Through the floor of the anterior cranium

Causes include toxic chemicals and heritable conditions, although most cases are of unknown etiology.

Toxic causes include: Griseofulvin,[6] methyl mercury[7] and triamcinolone acetate affecting rhesus monkeys fetuses dosed at 23-31 days gestational age.[8]
Hereditary forms include: Crested ducks[9] and presumably geese. In this condition heterozygotes are viable but have encephalocele, while homozygous recessive birds are lethally affected and do not hatch.
A kindred of Burmese cats have an autosomal recessive meningoencaphalocele,[10] which may no longer exist in the breed. Kittens in this kindred were selected for attenuated facial structures and some of them has facial bone abnormalities.[11]

Follow up: At 5 months post-operatively, the dog contributing this biopsy is presently seizure-free and neurologically normal.

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NPMS 2023, Case #3

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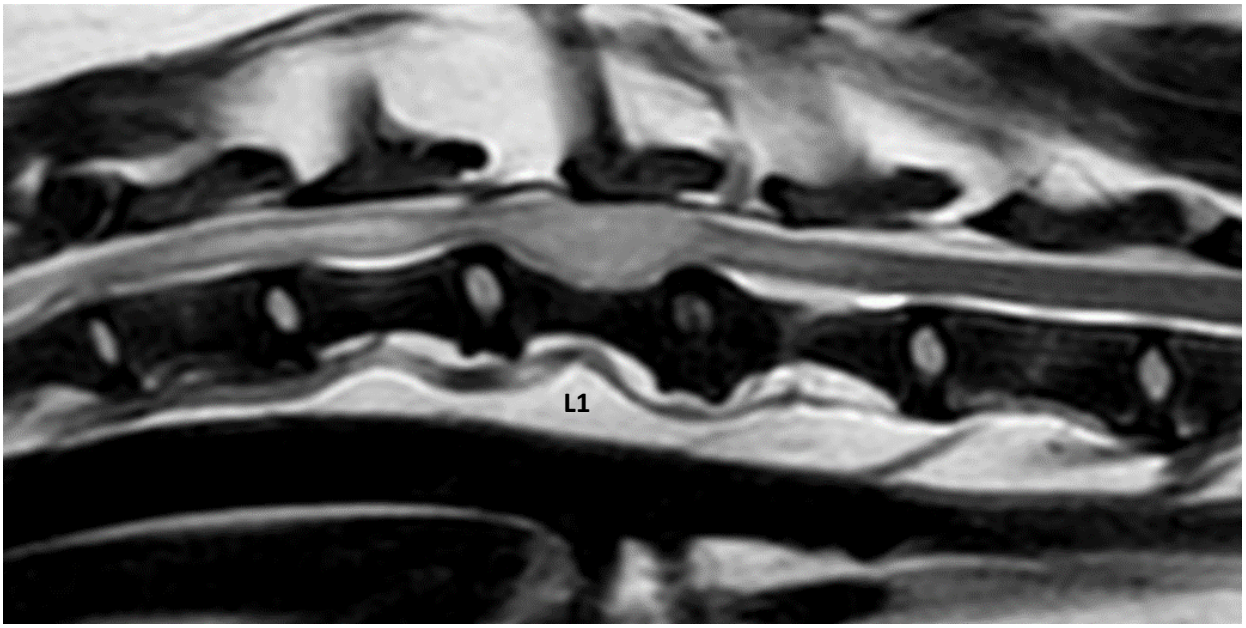
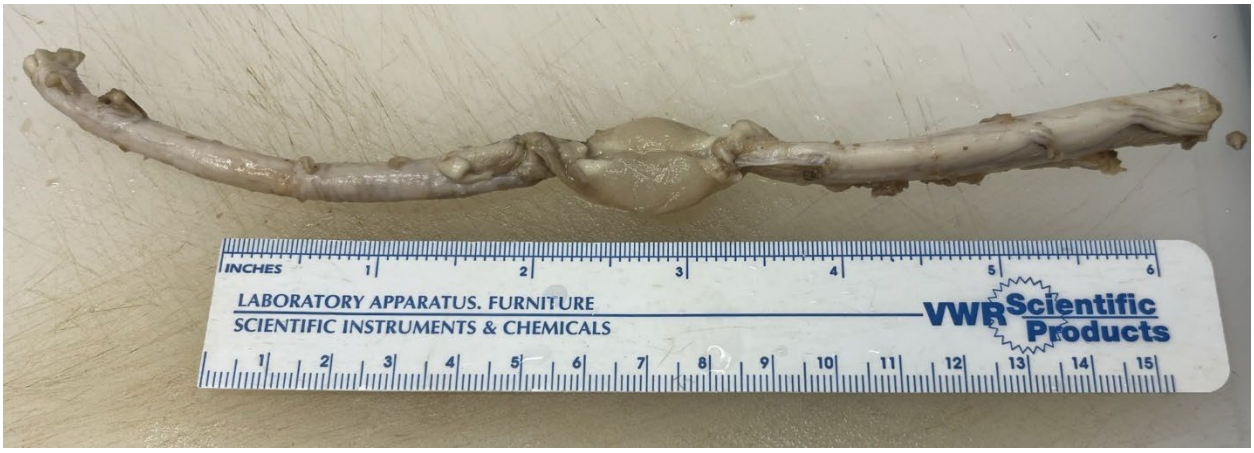
History/Signalment: 3.25-year-old, female spayed Brittany dog with acute hindlimb proprioceptive deficits. Irregular L1-L2 vertebrae were noted on radiographs at the rDVM. MRI revealed a hyperenhancing intramedullary spinal cord lesion at the level of the L1 vertebra, and suspected protrusion of the T13-L1 intervertebral disc (image below).



Gross Pathology: Within the spinal cord at the level of L1, a soft, light tan, ovoid, 1.5 cm diameter mass expands and effaces the neuroparenchyma.



Description:
Spinal cord, T12-L1: At the level of T13-L1, severely expanding and effacing the meninges, perineurium of spinal nerves, and invading along the arachnoid sheathes of vessels to efface up to 90% of the spinal cord parenchyma is a densely cellular, infiltrative mass composed of interlacing streams and whorls of spindle cells within a fine fibrovascular to myxomatous stroma. The cells are elongated with indistinct borders and small volumes of pale eosinophilic, fibrillar cytoplasm. Their nuclei are oval with vesicular chromatin and occasionally a single prominent, punctate nucleolus. Anisokaryosis is mild and there are 5 mitotic figures in ten 400x fields (2.37 mm^2). The walls of blood vessels throughout the mass are severely expanded and replaced with homogenous eosinophilic, acellular material (hyalinization). The scant remaining spinal cord white matter is moderately rarefied and there are frequent large, mildly fragmented spheroids within myelin sheathes swollen with clear to wispy pale eosinophilic fluid. Throughout the stroma are mildly increased numbers of astrocytes. Just cranial to the nodular mass, at the level of T12-T13, the meninges are minimally expanded by similar cells that dissect along the superficial arachnoid sheaths of white matter blood vessels. These vessels are similarly but more mildly hyalinized as previously described.

Morphologic Diagnoses:
Spinal cord (T12-L1): Malignant nerve sheath tumor, presumptive, with vascular hyalinization and axonal degeneration

Final Diagnosis: T12-L1 spinal cord – Malignant nerve sheath tumor (presumptive)

Comments:
Histologic evaluation reveals highly invasive mesenchymal neoplasm that appears to arise in the extramedullary space, most suggestive of a malignant nerve sheath neoplasm or meningeal sarcoma. Meningeal sarcoma and malignant nerve sheath tumor are each rare in dogs, particularly in young animals, and both are associated with aggressive behavior and a grave prognosis. A differential diagnosis of meningioangiomatosis was also initially considered in this case. However, formation of a discrete mass and the highly invasive nature of the proliferative spindle cell population are not typical for this entity.

Prominent vascular hyalinization throughout the mass and affecting vessels cuffed by neoplastic cells in the adjacent segments of spinal cord is considered likely reactive in nature. This is reminiscent of a change reported in cutaneous schwannomas in horses and in human schwannomas. However, as noted during the session discussion for the case, this finding is not specific to nerve sheath tumors and may also be seen in other neoplasms.

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NPMS 2023, Case #4

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2. Tri-Institutional Training Program in Laboratory Animal Medicine and Science, Memorial Sloan Kettering Cancer Center, Weill Cornell Medicine, and The Rockefeller University, New York, NY.

History/Signalment: Five 1.5-month-old intact female NOD.Cg-*Prkdc*^{scid} *Il2rg*^{tm1Wjl}/SzJ (NSG) mice were subcutaneously engrafted in the right flank with a human pancreatic tumor patient-derived xenograft (PDX). Approximately 2 months later, 2/5 mice, including the current case, presented with sudden bilateral hind limb paralysis and were euthanized by CO_2 overdose. Terminal blood was collected via cardiac puncture for complete blood count and serum chemistry analyses, and complete necropsies with histopathology examination were performed.

Gross findings: The nutritional condition was underconditioned (body condition score: 2/5; body weight: 18.491 g). The right flank had a 1.3 x 0.5 x 0.3 cm, white, soft, and homogeneous subcutaneous mass (xenograft tumor).

Histologic description:

Spinal cord and spinal nerve roots (thoracolumbar and lumbosacral), and peripheral nerves (hind limbs): Predominantly the sciatic nerves and the ventral spinal nerve roots, and to a lesser extent the ventral spinal cord's white and grey matter exhibit multifocal to coalescing areas of Wallerian-type degeneration, composed of mild to marked myelin sheet dilation with intralesional flocculent axonal/myelin eosinophilic debris, swollen and hypereosinophilic axonal spheroids, and digestion chambers with vacuolated macrophages. Affected regions have mild to moderate gliosis, mainly in the spinal cord grey matter.

Skeletal muscle (hind limbs): The myofibers are multifocally mildly to moderately reduced in size (atrophy), occasionally have pale eosinophilic and vacuolated sarcoplasm with loss of cross striations (degeneration), or exhibit mild to moderate nuclear crowding, centralization, and/or rowing with rare cytoplasmic basophilia (myopathic changes and/or rare regeneration).

Morphologic diagnoses:

Spinal cord and spinal nerve roots (thoracolumbar and lumbosacral), and peripheral nerves (hind limbs): Degenerative neuropathy and myelopathy with myelin sheath dilation, axonal degeneration, and gliosis, moderate to marked, multifocal to coalescing

Skeletal muscle (hind limbs): Myofiber atrophy, degeneration, and rare regeneration, mild to moderate, multifocal

Laboratory Results: PCR was positive for Lactate Dehydrogenase-Elevating Virus (LDV) in the spinal cord and xenograft tumor samples.

Etiologic diagnosis: Arteriviral degenerative neuropathy and myelopathy, with secondary neurogenic myofiber atrophy

Discussion: Hind limb paresis/paralysis in mice generally originates from dysfunction of the nervous and/or musculoskeletal system from various causes such as infectious diseases, traumatic injuries, neoplasia, intervertebral disk/degenerative joint disease, toxic compounds, genetic diseases, autoimmune diseases, and/or ischemia. In this case, the history/signalment, and gross and microscopic findings helped us rule out most of these causes. Also, most infectious agents that cause hind limb paresis/paralysis in laboratory mice are part of our institution's murine sentinel program and were not detected in the room where the affected mice were housed in at least the past three years.

Lactate Dehydrogenase-Elevating Virus (LDV; family: Arteriviridae) is an important cause of infectious hind limb paresis/paralysis in laboratory mice, but it is not part of our institution's murine sentinel program, as it is inefficiently transmitted amongst mice and serologic methods are generally inefficient to detect anti-LDV antibodies (1). A series of recently published LDV cases in NSG mice reported similar clinical signs and microscopic changes to those in the current case (2). Serum LDH levels were high in the current case (3368.5 U/L; normal general rage: 360-550 U/L (3)), and this used to be the gold standard for LDV diagnosis in mice as LDV specifically infects and destroys a subset of F4/80-positive macrophages responsible for serum LDH clearance. However, high serum LDH levels are not specific to LDV infections as this enzyme can be falsely elevated by hemolysis or secondarily due to cell destruction in various tissues (1, 2). An important means of natural transmission of LDV is through bite wounds in fighting mice, but in a laboratory setting, it is often transmitted by contamination and introduction of biologic materials of mouse origin such as culture reagents, xenograft/allograft tumor implants, cell cultures, etc. (1, 2) Therefore, we tested and confirmed positivity for LDV using RT-PCR in the spinal cord and xenograft tumors of the affected mice, confirming that this is a case of Arteriviral degenerative neuropathy and myelopathy with secondary neurogenic myofiber atrophy, presumably caused by the introduction of an infected xenograft tumor in this mouse. Different manipulations can help eradicate LDV from transplantable tumors, such as transplantation of infected tumors into non-murine models (e.g., nude rats), disassociation and subculture of infected tumors in vitro, and fluorescence-activated cell sorting (2).

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UCDAVIS
VETERINARY MEDICINE

NPMS 2023, Case #5

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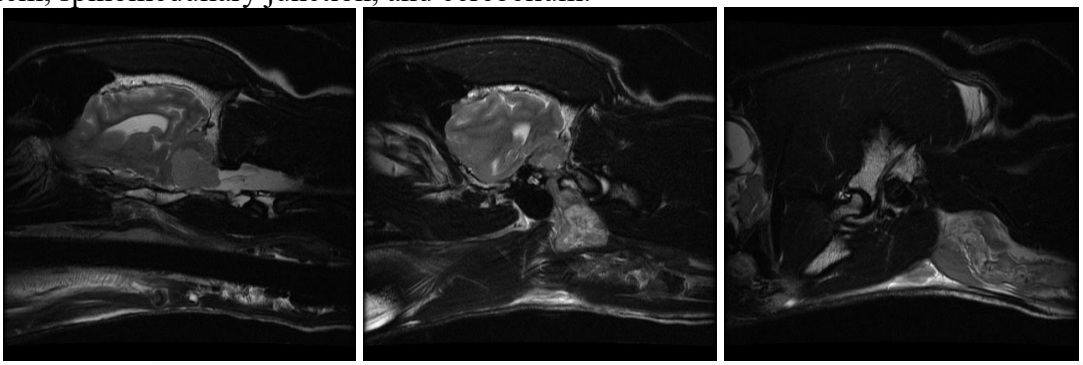
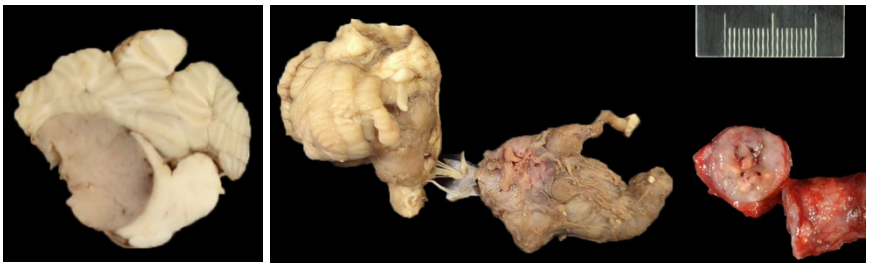
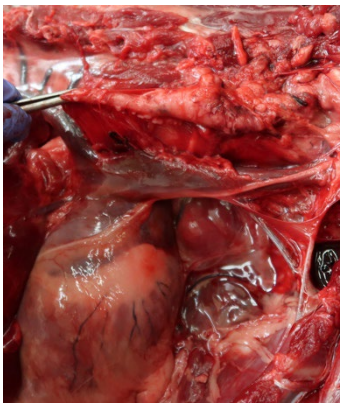
History/Signalment: 1.5-year-old male-intact standard poodle mix. 5 days prior to presentation developed a head tilt and circled to the right. 3 days prior: progressed to falling to the right with decrease sensation in pelvic limbs. 1 day prior: laterally recumbent, could not lift head, obtunded, with apneustic respiration. Neuroexamination revealed non-ambulatory tetraparetic, head tilt to the right, horizontal nystagmus with fast phase to the right consistent with central paradoxical vestibular dysfunction. MRI revealed an extensive T2-weighted hyperintense mass that spanned from the right side of mid-brain, through the petro-occipital fissure, along the right carotid artery, and down into the thoracic inlet. The mass was causing severe right sided compression of the brainstem, spinomedullary junction, and cerebellum.



Gross findings: Extending from the first branch of the aorta cranially into the calvarium is a linear, multinodular, pale tan to grey, semi-firm mass that passes through the cranial mesenteric ganglion, right thoracic inlet, jugular foramen and hypoglossal canal into the skull. It severely compresses the brainstem and cerebellum and communicates with the 4th ventricle. It expands and effaces the vagosympathetic trunk but does not invade the right carotid. On section, the mass has multifocal hemorrhage and pale tan firm granules (mineralization). It is 2cm wide at the largest diameter.





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Histologic Description: Examined is a section of the intercranial mass with the brain stem and a separate section of the mass where it travels through the jugular foramen. The intracranial mass is unencapsulated, compressive, and moderately cellular. The mass is composed of sheets, streams, and bundles of two distinct population of cells and interspersed pale eosinophilic wispy to granular matrix that frequently aggregates into circles resembling neuropil. The majority are small round cells with a round nucleus, dense chromatin, and mild amounts of eosinophilic cytoplasm (consistent with neuroblasts, germ cells/progenitor cells). Admixed are larger rhabdoid cells with an eccentrically placed round nucleus with stippled chromatin and a magenta nucleolus. The cell has granular to finely vacuolated amphophilic cytoplasm with a paler perinuclear clearing (resembling ganglion but not well-differentiated neurons). Within each cell population, anisokaryosis and anisocytosis are moderate. Individual cell necrosis is frequent. There are 38 mitotic figures seen in 2.37mm^2 (ten 400x fields).

Immunohistochemical stains:

- Positive: Microtubule associated protein 2 (MAP2), Synaptophysin (Syp), Neuron specific enolase (NSE), Neuronal nuclei (NeuN)
- Negative: Oligodendrocyte transcription factor 2 (OLIG2), Neurofilament (NF200)

Morphologic Diagnosis: RIGHT VAGOSYMPATHETIC TRUNK, CERVICAL GANGLION, BRAINSTEM: PERIPHERAL GANGLIONEUROBLASTOMA

Final Diagnosis: Peripheral ganglioneuroblastoma

Comment:

An embryonal peripheral neuroblastic tumor, ganglioneuroblastoma, is confirmed. These are very rare tumors (<1%) and arise in young animals. Grossly, these tumors are grey, firm and well-demarcated. Histologically, ganglioneuroblastomas are comprised of small blue cells (neuroblasts) with ganglion-cell differentiation.

Given the rarity of these tumors and embryonal nature, suspected immunohistochemical staining is based on human literature and rare veterinary case reports. The majority of the neuronal immunohistochemical markers were positive in this tumor. Although neurofilament is expected to have been positive, it is possible a different neurofilament isotype not captured by the veterinary immunohistochemical stain is present.

Ganglioneuroblastomas can arise from the CNS or the PNS. Peripheral ganglioneuroblastomas tend to grow rapidly and have a high mitotic rate, which this tumor had. Additionally the peripheral ganglioneuroblastomas are associated with peripheral, spinal, or sympathetic ganglia. This tumor was closely associated with the cervical ganglia.We hypothesize that the tumor started in the thoracic cavity then spread up the cervical vagosympathetic trunk before entering the brain and compressing the brainstem.

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NPMS 2023, Case #6

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History/Signalment: This 3-year-old River Vine Snake was submitted to the Michigan State University Veterinary Diagnostic Laboratory with a history of subcutaneous parasites. This snake was captive bred at an Association of Zoos and Aquariums (AZA) accredited aquarium. River Vine Snakes are terrestrial snakes indigenous to mangroves and tidal rivers along the coast of Myanmar. Their diet is composed entirely of fish.

Gross findings: No gross lesions were recorded at the time of postmortem examination.

Acknowledgement: Dr. Ryan Colburn DVM; John Ball Zoo, Grand Rapids, MI.

Histologic Description, Morphologic Diagnosis, Final Diagnosis, and a 1-2 paragraph comment about the entity with select references (or if not known, what the likely differentials are).

Histologic Description:
Approximately 30% of the calvarium is expanded by two parasite profiles that dorsally displace the neural parenchyma. The parasites have a thin undulating and pseudosegmented eosinophilic cuticle that is segmentally lined by rows of fine spines and multiple sclerotized openings. These openings stain black with a Movat's Pentachrome stain. Underlying the cuticle, there is a layer of striated skeletal muscle. There is a centrally located digestive tract that is closely associated with bright eosinophilic acidophilic glands. The parasite profiles are surrounded by a fibrotic capsule that is variably mineralized. There are minimal to absent changes to the neural parenchyma.

Morphologic and Final Diagnosis: Brain - Pentastomiasis

Discussion:
Pentastomids are a group of obligate parasitic arthropods that are molecularly related to crustaceans[1]. Grossly, they range from 1-14cm in length and have distinctive annulations[2]. The larvae have a single ventrally oriented mouth that is flanked by two pairs of hooks, giving the appearance of "five mouths"[3]. Histologically, pentastomids have a thin, eosinophilic, pseudosegmented, annulated cuticle that is underlaid by striated skeletal muscle and a central digestive tract. Distinctive histologic features include: 1. sclerotized pores that stain black with a Movat's Pentachrome stain, and 2. bright acidophilic glands that surround the gastrointestinal tract[4].

Pentastomes have an indirect lifecycle. The adults are found in the respiratory tracts of their respective definitive hosts, which are commonly reptiles[5]. In the respiratory tract, females lay eggs that are excreted into the environment via respiratory secretions or feces. The eggs hatch into larvae and are ingested by intermediate hosts, which are commonly rodents and fish. In the intermediate host, the larvae migrate through the viscera and encyst in various tissues. The cycle completes once a definitive host ingests the intermediate host, and the larvae migrate to the respiratory tract of the definitive host to molt into adults[5]. Pentastomiasis is a zoonotic disease that affects both humans and primates[2-3]. Three commonly referenced species in human and veterinary medicine are *Armillifer armillitus*, *Porocephalus crotali,* and *Linguatula serrata*.

Snakes are common definitive host for pentastomids and are uncommonly reported as intermediate hosts. In this case, the aberrant visceral migration of the larvae suggests that this snake was an intermediate host.

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