Constrictive Myelopathy: a cause of hind limb ataxia unique to Pug dogs?
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BACKGROUND
Recently, a previously unreported condition termed “constrictive myelopathy” was described in 11 adult Pug dogs (J Am Vet Med Assoc 2013; 242:223-229). The paper reported a progressive incoordination and weakness of the hind limbs resulting from a constriction of the spinal cord at the thoracolumbar junction, and associated with malformations of the articulations of vertebrae in this area. The degenerative condition often progressed to paraplegia, with urinary and/or fecal incontinence. Despite surgical treatment, neurologic disease persisted or progressed. This myelopathy is seemingly unique to and reportedly rare in purebred Pug dogs, although anecdotal evidence suggests that the vertebral malformations (hypoplasia and/or aplasia of caudal articular processes) are relatively common in the breed, as supported by imaging studies. Authors of the published study hypothesize that the vertebral anomalies may represent a heritable condition in Pugs, and that instability at the thoracolumbar junction associated with the anomalies leads to the formation of a circumferential fibrous band which constricts the spinal cord. A case description of a Pug diagnosed with constrictive myelopathy at age 6.5 years, and euthanized at age 14 years is presented.

CASE DESCRIPTION
A spayed female purebred Pug dog, was initially observed at age 6.5 years to have reluctance climbing stairs and urinary and fecal incontinence. Neurologic examination revealed bilateral hind limb weakness and ataxia, with increased tone in the left hind. Hind limb proprioceptive deficits were present bilaterally, and the cutaneous trunci response was absent caudal to T13. Radiographs and computed tomography (CT) suggested hypoplasia of caudal articular processes of T10-T12, and MRI suggested spinal cord compression at T12-T13. A diagnosis of “pug myelopathy” was made, and a dorsal laminecctomy was performed in the area of compression. At surgery, a circumferential band of mature fibrous tissue, seen to compress the spinal cord, was removed. After surgery, the dog had improved hind limb function, and better control of urination and defecation. Approximately 6-7 months after surgery, however, the hind limb ataxia worsened, and a CT/myelogram suggested a demyelinating condition. The dog was treated with various doses of prednisone and underwent acupuncture therapy for 3 months at age 7.5 years. By age 8, the dog had complete urinary retention incontinence, and by age 9.0, would walk only if supported, and required front limbs to pull herself along. At age 12, a DNA sample was tested at University of Missouri for the degenerative myelopathy (DM) gene mutation, and results were negative. Approximately 1 week prior to euthanasia, the dog began having difficulty using one front limb, and euthanasia was elected.

Complete necropsy was done at the Michigan State University Diagnostic Center for Population and Animal Health (DCPAH). There was marked bilateral atrophy of the caudal thigh muscles, muscles over the pelvis, and epaxial muscles of the thoracic and lumbar spine. Slight scoliosis of the vertebral column to the right was noted at the level of T6-T7, and there was mild bridging spondylosis on the ventral aspect of the vertebral bodies at the T6-T7 intervertebral space. The entire vertebral column, containing the spinal cord, was placed in 10% neutral buffered formalin, and following fixation, the spinal cord was removed and vertebrae were disarticulated and examined. It was difficult to draw definitive conclusions regarding the caudal articular processes of the T10-T13 vertebrae, as there was extensive bony remodeling, presumably associated with age and surgery 9 years prior. However, the caudal articular processes of T10 and T11 seemed small (hypoplastic) bilaterally, and there appeared to be asymmetry with respect to size for the paired articular processes (right vs. left) of T10 and T13. Histologically, there was severe segmental chronic myelomalacia in the T12 and T13 spinal cord segments, with Wallerian degeneration cranial and caudal to this area. The leptomeninges were moderately to markedly thinned by dense fibrous tissue from T10-T13, with areas of arachnoid hyperplasia and dural fibrosis. Focal poliomyelomalacia in the C6 spinal cord segment was noted, and close inspection of the cervical vertebral column revealed dry, flaky intervertebral disc material at C5-C6 and C6-C7, suggesting disc degeneration. The final diagnosis was severe segmental chronic degenerative myelopathy at T12-T13, with meningeal fibrosis (T10-T13) and Wallerian degeneration. This appeared to be the major lesion, consistent with the 7-8-year history of progressive hind limb weakness, ataxia, and paralysis, and consistent with what was described by the surgeons who treated the dog. The more recent spinal cord lesion in the C6 segment involved primarily the gray matter and was consistent with an acute intervertebral disc extrusion that then became chronic.

REFERENCES
• http://www.pugs.org/.

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