COMPANION OR PET ANIMALS

Clinical, diagnostic imaging, surgical findings and long-term outcome in a Cavalier King Charles Spaniel with thoracolumbar vertebral articular process hyperplasia

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SUMMARY
A 2-year-old female spayed Cavalier King Charles Spaniel presented with 10-day history of progressive painful T3–L3 myelopathy. MRI of the spine revealed an extradural dorsal spinal cord compression at the level of T12–T13 vertebrae caused by hyperplasia of the caudal vertebral articular process (CVAP) of T12 vertebra. Similar changes were noticed at the level of the T13–L1 vertebrae, causing spinal cord compression. In order to plan surgical decompression, CT of the spine was performed. This revealed thickened T12 dorsal lamina, hyperplastic T12 CVAP, and narrowed and elongated T13 VAP. The patient underwent T12–T13 dorsal laminectomy and spinal cord decompression by removal of the hyperplastic CVAP of T12. Postoperative 6-year follow-up revealed a normal neurological examination. A combination of MRI and CT allowed optimal treatment selection and surgical planning for CVAP hyperplasia. Surgical treatment by dorsal laminectomy provided successful long-term outcome.

BACKGROUND
Vertebral articular process (VAP) dysplasia refers to congenital anomalies affecting the cranial or caudal VAP (CVAP) including absence (aplasia), incomplete formation (hypoplasia) and enlarged (hyperplasia) VAP formation, unilaterally or bilaterally.1–3 Caudal VAP hypoplasia or aplasia of the thoracic vertebrae has been reported as a common incidental finding in neurologically normal French Bulldogs, English Bulldogs and Pugs.1 Thoracolumbar VAP hyperplasia secondary to an underlying malformation has been described in a limited number of dogs.4,5 There is scarce information on outcome following surgical treatment in dogs with CVAP hyperplasia. This report highlights the importance of combination of advanced imaging modalities to plan surgical treatment for a successful long-term outcome.

CASE PRESENTATION
A 2-year-old female spayed Cavalier King Charles Spaniel (CKCS) presented 10 days after development of thoracolumbar kyphosis and pelvic limb ataxia since jumping off a car. Pre-referral 7-day oral meloxicam 0.1 mg/kg/24h (Metacam; Boehringer Ingelheim) treatment did not result in improvement. At presentation, general physical examination was unremarkable. Neurological examination revealed thoracolumbar kyphosis, ambulatory paraparesis and severe pelvic limb proprioceptive ataxia. Paw positioning and hopping were absent on both pelvic limbs. Spinal pain was detected on palpation of the thoracolumbar spine. The remainder of the neurological examination was normal. The neuro-anatomical localisation was to the T3–L3 spinal cord segments. Differential diagnoses included intervertebral disc (IVD) extrusion (compressive or non-compressive), deterioration of developmental abnormalities following trauma, inflammatory/ infectious disease of the central nervous system and, less likely, neoplasia.

INVESTIGATIONS
Haematology and serum biochemistry revealed no significant abnormalities. High-field (1.5 T GE Signa Echospeed; GE Medical Systems, Milwaukee, Wisconsin, USA) MRI of the thoracolumbar spine showed dorsal spinal extradural compression at the level of T12 and T13 vertebrae. An enlarged and irregularly shaped T12 CVAP was protruding ventrally into the vertebral canal resulting in attenuation of the dorsal subarachnoid and epidural space, and spinal cord compression (figure 1A,B,C). The spinal cord was hyperintense on T2W sequences relatively to normal spinal cord parenchyma consistent with intramedullary spinal cord oedema/ moderate obstructive syringomyelia cranial to the site of compression (figure 1A,B). Malacia or gliosis were considered less likely differential diagnoses for this finding based on the medical history. VAP dysplasia was observed also at the level of T13–L1, although no spinal cord compression was detected. Multiple IVD degeneration was seen (figure 1A). Administration of gadobutrol 0.1 mmol/kg (Gadovist; Bayer) showed no contrast enhancement. CT imaging of the thoracolumbar spine was performed using a 16 slice helical CT (GE Brivo; GE Medical Systems) in order to characterise more precisely the osseous anomaly, evaluate if there was concurrent ligamentum flavum hypertrophy and accurately plan surgical treatment. CT images were reformatted using a high-spatial-frequency algorithm in transverse, sagittal and dorsal planes. The CT showed hyperplastic dysplasia of the T12 CVAP ventrally protruding into the vertebral canal causing spinal cord compression. The adjacent T13 cranial VAP were narrowed and elongated as observed on...
Figure 1  (A) Midline sagittal T2W MRI of the thoracolumbar spine, (B) transverse T2W and (C) T1W MRI at the level of the T12–T13 intervertebral disc space. Note the enlarged caudal vertebral articular process and marked dorsoventral flattening of the spinal cord at the level of T12–T13, with associated attenuation of the hyperintense subarachnoid and epidural space. Note the intramedullary increased signal intensity at this level.

MRI (figure 2). Vacuum phenomenon was seen within the T12–T13 intervertebral disc. Remodelling of the T13–L1 VAP and multiple mineralised IVDs, without any further sites of spinal cord compression, were seen. The T10 dorsal spinous process was hypoplastic (figures 2 and 3).

DIFFERENTIAL DIAGNOSIS
Based on the MRI and CT findings, the main differential diagnosis was a spinal cord compression secondary to hyperplastic dysplasia of T12 CVAP.

TREATMENT
A T12–T13 dorsal laminectomy (figure 4) was performed and the spinal cord was decompressed (figures 5 and 6). Intraoperatively, the T12 dorsal lamina and T12 CVAP were hyperplastic; no ligamentum flavum hypertrophy was noticed. Moreover, intraoperative assessment revealed no signs of spinal instability. Histopathology of T12 dorsal arch and T12 CVAP were consistent with VAP hyperplasia due to lack of pathological abnormalities and presence of thickened T12 lamina and enlarged CVAP.

Figure 2  (A) Sagittal and (B) dorsal CT images of the thoracolumbar junction. Note the enlarged T12 caudal vertebral articular process (CVAP) (arrow) and the vacuum phenomenon within the T12–T13 intervertebral disc. Dysplasia of the VAP at T13–L1 is also noticed (arrowhead). The spinous process of T10 is hypoplastic (asterisk).

Figure 3  3D CT reconstruction of the thoracolumbar junction. The abnormally shaped caudal vertebral articular process (CVAP) of T12 (arrow) and T13–L1 (arrowhead) are seen, compared with the normal CVAP of the adjacent L1–L2 joint (asterisk).

Figure 4  Dorsal approach to the T12–T13 spine showing the hyperplastic T12 caudal vertebral articular process during dorsal laminectomy.

OUTCOME AND FOLLOW-UP
The day after surgery, the patient showed pelvic limb proprioceptive ataxia and ambulatory paraparesis. On discharge 3 days after surgery, the degree of ataxia and paresis were mildly improved compared with preoperative examination. After 4 weeks of strict restriction in exercise and postsurgical analgesia with meloxicam 0.1 mg/kg/24 h (Metacam; Boehringer Ingelheim) and gabapentin 10 mg/kg/8 h (Gabapentin Teva; Teva), the patient had a normal posture and a mild degree of proprioceptive ataxia. Paw positioning and hopping were mildly delayed on the pelvic limbs. At 2-month postoperative follow-up, the patient’s postural reactions were normal and the gait was slightly ataxic in the pelvic limbs. Follow-up at 1 year, 3 years and 6 years after surgery revealed normal posture, normal gait and normal
proprioception on both pelvic limbs. The patient remained comfortable, exercising without restrictions.

DISCUSSION

This is the first report of a successful outcome after surgical treatment for focal T3–L3 myelopathy caused by thoracic CVAP hyperplasia in a young CKCS with a 6-year long-term follow-up. A previous case series reporting dysplastic CVAP in four dogs included a 10-month-old CKCS with signs of T3–L3 focal myelopathy. In the CKCS reported by Penderis et al., MRI of the spine revealed an hourglass spinal cord compression at the level of the last two thoracic vertebrae. The compression was caused by ventral bulging of the dorsal annulus fibrosus and dorsally by ligamentum flavum hypertrophy with or without VAP new bone formation. Preoperative differentiation between new bone formation and ligamentous structure was not possible without CT of the spine. Such findings lead to the hypothesis that the VAP hyperplasia and the other spinal abnormalities might have been secondary to chronic microinstability.

While MRI images of our case were similar to the CKCS reported by Penderis et al., CT and intraoperative findings revealed that the spinal cord compression was solely osseous in origin and no ligamentous hypertrophy or DJD was associated to the CVAP hypertrophy. These findings would support the hypothesis that VAP hyperplasia in our case is likely due to developmental anomaly more than being secondary to the suspected microinstability. Moreover, in our case, intrasurgical spinal instability/microinstability was not recognised during laterolateral and dorsoventral movement evaluation. In the CKCS reported by Penderis et al., no treatment or long-term follow-up was reported. It is likely that the clinical signs in the reported case might have been cause by spinal cord concussion/ transient exacerbation of the compression during the reported jump off the owner’s vehicle. On CT, vacuum phenomenon was seen within the T12–T13 IVD space. Clinically, vacuum phenomenon has been associated with normal joint motion, degeneration of the IVDs and trauma. It is thought that vacuum phenomenon is caused by expansion of an enclosed tissue space as a rebound phenomenon after an external impact. Such impact causes the volume within an enclosed space to increase causing the pressure within the space to decrease. The solubility of the gas in the enclosed space will decrease as the pressure of the space decreases. Decreased solubility allows a gas to leave a solution causing vacuum phenomenon.

Mc Donnell et al. reported a family of Shiloh Shepherd Dogs (breed derived from the German Shepherd) with signs of progressive T3–L3 myelopathy caused by vertebral process degenerative joint disease. In this family, abnormalities of the synovial joints and bony proliferation of the involved articular processes and DJD were identified post mortem. The articular processes and joint capsules were thickened, and the cartilaginous surfaces were extremely irregular and appeared to be eroded. This suggested the possibility that such abnormalities were secondary to chronic malarticulation. The authors proposed the disorder being inherited in origin in this large dog breed.

In our patient, a CKCS of 10kg of body weight, no changes compatible with DJD were identified supporting the hypothesis that this patient was more likely affected by a primary form of CVAP hyperplasia. The authors consider that this is also supported by the fact that the similar changes seen at T13–L1 did not cause any clinical signs over the long-term follow-up.

Thoracolumbar CVAP aplasia/hypoplasia was reported in 11 Pugs, 5 of which with no neurological deficits and 6 with focal signs of T3–L3 myelopathy, associated with fibrous constrictive myelopathy. These finding led the authors to the hypothesis that in Pugs, fibrous constrictive myelopathy is a consequence of VAP hypoplasia/aplasia due to chronic instability resulting in formation of fibrous bands affecting the dura mater. More recently, Driver et al. reported concurrent CVAP hypoplasia in 17 Pugs and aplasia in 1 Pug affected with thoracolumbar myelopathy due

Learning points

- Thoracolumbar caudal vertebral articular process (CVAP) hyperplasia could cause significant spinal cord compression and secondary neurological deficits.
- Prompt recognition of thoracolumbar CVAP hyperplasia and assessment of its effect on the spinal cord can be obtained with a combination of MRI and CT.
- Surgical treatment of thoracolumbar CVAP hyperplasia in small-size dogs can lead to successful long-term outcome.
to four different putative diagnosis (IVD protrusion, subarachnoid diverticulum, pia-arachnoid fibrosis, vertebral instability). While this report is describing a single case and therefore no general information can be assumed, no imaging/intraoperative changes compatible with fibrous constrictive myelopathy were identified in association with the CVAP hyperplasia reported in this case. This case report provides the description of clinical signs and diagnosis, treatment and long-term follow-up of a CKCS with caudal VAP.

Contributors All authors have contributed and agreed to submitted case report.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement All data relevant to the study are included in the article or uploaded as supplementary information.

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