Surgical management of myelopathy caused by a solitary spinal osteochondroma in a young cat

Nikola Heblinski and Hugo Schmökel

Abstract

Case summary A 10-month-old, male castrated, domestic shorthair cat was presented with fast-progressing ataxia of the pelvic limbs. MRI and CT were performed, revealing a bony proliferation at T11, with mass effect and laterodorsal compression of the spinal cord. After hemilaminectomy and the removal of the bony mass, the cat recovered uneventfully. At the 1 year follow-up, the cat did not show any neurological deficits or signs of recurrence.
Relevance and novel information Solitary osteochondroma as a cause of neurological deficits in the pelvic limb has been described in dogs and humans, but, to the best of our knowledge, there are no reported feline cases described in the literature, in which CT and MRI were performed in combination with the successful removal of the lesion and an excellent outcome for the patient.

Introduction

Solitary osteochondromatosis of the spine is a rare disease described in people, horses and dogs, as well as one case involving a cat.[1,2] Osteochondroma (OC) is a benign tumour of the bones. It can appear in either single form or the multiple, hereditary form (osteochondromatosis or hereditary multiple exostosis).[1,3] Even in humans, the involvement of the thoracic spine is rare, with only 27 cases reported.[4] Although their aetiology is unclear, OCs originate from the separation of epiphyseal growth plate cells, followed by herniation through the periosteum adjacent to the growth plate; alternatively, the cells may originate from the separate secondary ossification centres in the spinal processes.[3,4] Throughout the process of growth, the exostoses are continuously replaced by spongiomatose bone tissue.[5–7]

Case description

A 10-month-old, castrated male indoor domestic shorthair cat was presented with a history of fast-progressing ataxia of the pelvic limbs. The referring veterinarian had prescribed pain management with opioids and nonsteroidal anti-inflammatory drugs (NSAIDs), without improvement after 2 days. A radiographic examination

of the thoracolumbar spine was unable to reveal the reason for the neurological deficits.

At presentation the cat was alert, and the general examination was unremarkable. The cranial nerves and front legs were normal. Further neurological examination revealed a non-ambulatory paraparesis, without the ability to bear weight on the pelvic limbs (modified Frankel score 3). In the pelvic limbs, proprioceptive responses were absent, and the sciatic and femoralis reflexes were hyperactive. The cat was able to urinate consciously. The examination indicated a T3–L3 myelopathy. The results of the haematology and serum chemistry were within the normal limits.

To further diagnose the lesion, an MRI scan with a low-field Esaote Vet-MR Grande was performed (T3–S3),

Evidensia Specialist Animal Hospital Strömsholm, Strömsholm, Sweden

Corresponding author:
Nikola Heblinski DVM, Evidensia Specialist Animal Hospital Strömsholm, Djursjukhusvägen 11, 73494 Strömsholm, Sweden
Email: nikola.heblinski@gmx.de

followed by a CT scan with a Phillips Brilliance CT 40 Channel, using the cervical spine helical program with 1mm slices and 0.5 mm increments (C5–L3). The MRI revealed a mass effect within the spinal canal of T11. There was a heterogenic hypointensity in the turbo three-dimensional and T2 sequence. The revealed mass showed contrast uptake that was visible in the T1 sequence. There was a T2-hyperintense signal change within the spinal cord at the level of the compression (Figure 1). The CT showed a bony proliferation originating from the left pedicle of T11, with laterodorsal compression of the spinal cord. No periosteal reactions or lysis of the adjacent bone indicating infection or malignant behaviour were noted (Figure 2).

With the presumptive diagnosis of a solitary OC, surgery was recommended to the owner. After premedication with methadone (0.3 mg/kg) and acepromazine (0.05 mg/kg), anaesthesia in the cat was induced with propofol (4 mg/kg) and maintained with isoflurane and oxygen. An electrocardiogram, capnography and oxygen saturation, blood pressure and body core temperature measurements with an oesophageal sensor were used to monitor the anaesthesia.

An extended left-sided hemilaminectomy T11 was performed, including the left facet joints T10–11 and T11–12 and the spinous process of T11. The proliferative bony mass was removed with small rongeurs, and bone samples were collected for histological examination. After flushing with sterile saline, the defect was covered with an autologous fat graft and the incision was closed routinely. Postoperative analgesia consisted of 0.3 mg/kg methadone every 2–4 h. A transdermal fentanyl patch was applied for 3 days together with an NSAID (meloxicam 0.05 mg/kg) every 24 h for 14 days.

The 8 week postoperative control showed a great improvement in the motoric function of the pelvic limbs, with the ability to walk and positive proprioceptive responses (ambulatory paraparesis, modified Frankel score 2). No pain in the operated area could be detected. One year after surgery, the cat was free of obvious neurological deficits, as seen in several video sequences provided by the owner.

Discussion

In contrast to dogs and humans, where the growth of the exostoses ceases with skeletal maturity, OCs in cats proliferate beyond physeal closure.[8–12] Most commonly, solitary OCs affect older cats and are also often related to feline leukaemia virus diseases.[10,12–14] No breed or sex predisposition has been identified.[12] Nevertheless, in dogs, the possibility of an inherited predisposition has been discussed.[6,8,15–18]
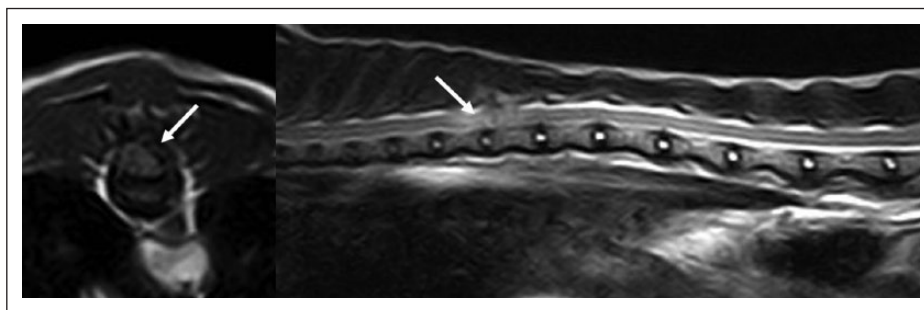
Figure 1 Transverse and sagittal T2 MRI sequence of the thoracolumbar spine. The heterogenous, hypointense mass compressing the spinal cord from the left laterodorsal side is marked by the arrow
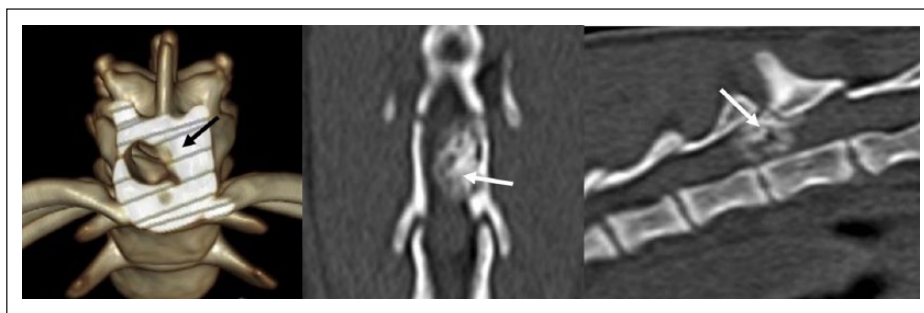
Figure 2 Transverse three-dimensional reconstruction and dorsal and sagittal CT scans revealed a boney proliferation compressing the spinal cord from the left, laterodorsal at the level of T10–11 (arrows)

Even although these benign lesions are usually clinically insignificant, malignant transformation into osteosarcoma, chondrosarcoma or parosteal sarcoma is possible.[13,17,19–21]

Most OCs are asymptomatic, but mechanical signs can occur, depending on the location and size of the OC. Clinical signs usually occur if an OC creates pressure on neighbouring structures, such as tendons, vessels, the spinal cord or the trachea.[2,10,11,14,22–27] OC lesions can be amorphous (or sometimes linear or oval-shaped), heterogeneous mineralised formations, which may or may not be attached to the skeleton.[2,9–11,24,25,27–29] Consequently, cartilaginous exostoses in the spine may be revealed to be connected to a physeal plate radiographically.[13]

To the best of our knowledge, solitary OCs triggering neurological signs have been described in dogs and humans, but there is only one such case report for a cat reported in the literature.[2,24,30] The present case report describes a 10-month-old, castrated male, domestic shorthair cat with progressive ataxia of both hindlimbs. Radiographs of the skeletal system did not reveal any pathological findings to explain the clinical signs. The unique composition of hyaline cartilage, with its low number of cells and high water content, allows for a good MRI display of OCs. MRI is therefore considered the best diagnostic modality for OCs.[7] In addition, MRI can be used to determine the exact localisation and extent of any potential compression of the surrounding soft tissue. Another advantage is the possibility of monitoring the thickening of the cartilage caps, and this may allow detection of the early transformation to malignancy.[7,17] In the literature on humans a thickness of the cartilage cap of more than 2 cm may indicate a malignant transformation,[31] but this has not been evaluated in affected animals thus far.[13] Nevertheless, the MRI of the cat described here suggests an OC of the T11 vertebra (Figure 1).

In human medicine, CT is used to evaluate the physical characteristics of an OC.[32] For optimal preoperative planning and to further rule out possible differential diagnoses, such as periosteal chondromas, calcinosis circumscripta or osteochondrosarcoma, a CT was performed in addition to the MRI.[25,31] The findings of the CT further supported the existence of an OC. No other pathological findings were seen (Figure 2).

Despite the fact the spinal cord from C1 to C4 has not been evaluated via either MRI or CT, the preoperative neurological examination suggests a T3–L3 myelopathy. Additionally, postoperative recovery suggests the conclusion of a lesion affecting a single vertebra and therefore the presence of a solitary OC.

Given the continuing growth of OCs in cats beyond skeletal maturity and the marked neurological changes these create, the surgical removal of the mass and the decompression of the neuronal tissue are the treatments of choice.[13] According to the literature, a histopathological examination is mandatory to confirm the diagnosis.[13] The histopathology of the tissue removed from the cat described here revealed bone tissue composed of well-differentiated osteoid tissue. There were no findings suggestive of necrosis in the osteoid tissue and no signs of malignancy. Assuming complete removal of the tumour and no malignancy detected at histopathology, the prognosis is good and there is minimal likelihood of local recurrence. Postoperatively, the cat already showed an improvement in motor function and normal proprioception of the pelvic limbs after 8 weeks. One year later, the cat had an unremarkable gait, and no signs of recurrence were seen on the video sequences provided by the owner.

Conclusions

Spinal solitary osteochondroma is rarely observed in cats. This case report describes the diagnostic imaging findings, including CT and MRI, of a solitary OC in the spinal cord of a cat. The cat was successfully treated with surgery and was free of clinical signs 1 year after surgery. Advanced imaging was essential for surgical planning and delineating the surgical margins. Histopathology should be used to confirm the diagnosis and indicate the long-term prognosis.

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