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CASE REPORT
Diagnosis and surgical management of a fractured atlas in a cat

Monty S Bali Dr Vet Med Diplomate ECVS (SA)

Fachtierarzt für Kleintiere, Tierärztliche Klinik Stommeln, Nettegasse 122, 50259 Pulheim, Germany

A 6-month-old male-castrated domestic shorthair cat was presented shortly after being bitten on the neck by a large breed dog. On presentation the cat was non-ambulatory tetraparetic with preserved deep pain perception. Plain radiographs of the neck did not show any abnormalities. Computed tomography was performed and showed a right-sided depressed fracture of the dorsal lamina of the atlas. A partial dorsal laminectomy was performed to alleviate the spinal cord compression. The cat made a full recovery within 12 weeks of surgery.

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A 6-month-old male-castrated domestic shorthair cat was presented to our emergency service, after being bitten 2 h previously on the neck by a large breed dog. After the accident, the owners had not noticed any voluntary movement of the limbs nor spontaneous micturition. The animal was presented in lateral recumbency, the general condition was good with a rectal temperature of 38.6°C. The examination of the cardiovascular system was unremarkable. Several small puncture wounds were found on the dorsal neck. The animal exhibited severe tetraparesis, with complete loss of voluntary motor function on the right body half and only minimal voluntary motor function on the left. The neck seemed painful, but due to the suspicion of cervical instability we refrained from further manipulation. The spinal reflexes were normal to increased in all four extremities and deep pain sensation was retained in all limbs. At the time of examination, the bladder was large and was easily expressed manually. The lesion was localised to the upper cervical segment (C1–C5). Radiographs of the thorax, abdomen as well as cervical vertebral column were unremarkable in both planes. The cat was anaesthetised with ketamine (8 mg/kg IV, Ketavet; Pfizer) and xylazine (1.5 mg/kg IV, Xylazin 2%; CEVA Tiergesundheit). After intubation helical computed tomography (CT) (Somatom Emotion 16-slice, Siemens) was performed and revealed a right-sided depressed fracture of the dorsal lamina of the atlas with severe compression of the spinal cord (Fig 1). There were no findings suggestive of cervical instability. For the duration of the CT, as well as the surgical preparation, the cat received supplementary oxygen through the tracheal tube. Heart rate and haemoglobin saturation (SpO_2) were monitored with pulse oxymetry (NPB-40, Nellcor Puritan Bennett). Following the CT, the cat was routinely prepared for surgery. Buprenorphine (0.01 mg/kg IV, Buprenovet; Bayer) and potentiated amoxicillin (20 mg/kg IV, Augmentin; GlaxoSmithKline) were injected intravenously. General anaesthesia was maintained with isoflurane and the cat received a continuous rate infusion of lactated Ringer's solution (10 ml/kg/h IV, Ringer-Laktat; DeltaSelect). During surgery electrocardiography, respiratory rate, SpO_2, end-tidal carbon dioxide, rectal temperature and oscillometric non-invasive blood-pressure (NIBP) measurements were continuously monitored (multi-parameter monitor, PM-9000Vet, Mindray). The animal was positioned in sternal recumbency, with the neck fixed in a slightly flexed position. Care was taken not to occlude jugular venous return. A dorsal midline skin incision was performed from the occipital protuberance to the third cervical vertebrae. The dorsal lamina of the atlas was approached using sharp and blunt dissections. Care was taken not to harm the more laterally located vertebral artery. Minor haemorrhage was controlled using cold lactated Ringer's solution and locally applied epinephrine (0.1 ml, Adrenalin 1:1000; Jenapharm). The depressed fracture was complicated by several small fissures running adjacent to the fracture. To minimise the risk of iatrogenic spinal cord injury,

E-mail: montybali@hotmail.com

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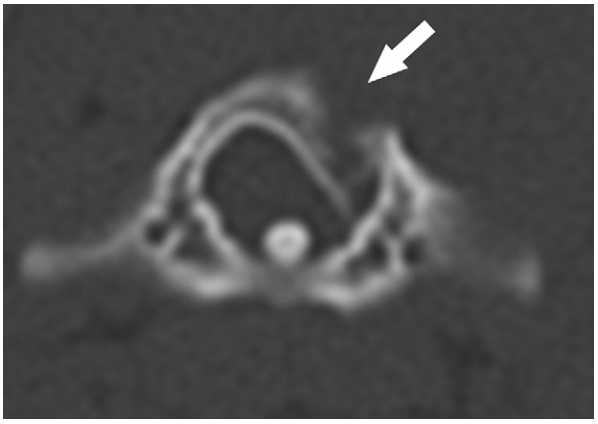

Fig 1. Preoperative transverse computed tomographic image of the atlas, depicting the impression fracture (white arrow).

the resection of the dorsal lamina was started caudally with a Kerrison rongeur. After the decompression was completed, the surgical field was rinsed with lactated Ringer's solution. The individual muscle bellies as well as the subcutaneous tissues were reapposed in a routine fashion, using polydioxanone absorbable suture material (4-0 USP Surgicryl Monofilament, SMI). The skin incision was closed with polyamide sutures (4-0 USP Trulon, Sutures India). A postoperative CT was conducted to confirm the decompression (Fig 2). During the entire anaesthesia, ventilation remained spontaneous and monitoring never suggested hypoventilation or respiratory compromise. During recovery the cat was monitored extensively, including regular SpO_2 and NIBP measurements. Postoperatively the cat received buprenorphine (0.01 mg/kg IV q 8 h), potentiated amoxicillin (20 mg/kg IV q 12 h), meloxicam (0.01 mg/kg SC q 24 h, Metacam; Boehringer Ingelheim) and lactated Ringer's solution (4 ml/kg/h IV). To prevent decubital ulcers the patient was turned over every 3–4 h. Physical therapy was initiated the day after surgery (passive movements and massage five times daily). At this point the cat was already showing the return of spontaneous micturition and slight voluntary motor function in all four limbs. It was returned to the owners 2 days later with instructions to administer meloxicam and potentiated amoxicillin (12.5 mg/kg q 24 h, Synulox; Pfizer) for another 5 days. At this point the cat had recovered enough to turn itself and demonstrated controlled micturition. The owners were instructed and advised to continue with the physical therapy (passive movements, massage and proprioceptive exercises five times daily). The cat showed a rapid improvement of neurological function, 2 weeks after the surgery, it was already able to walk, although still exhibiting a pronounced tetraparesis. At the final control 12 weeks after surgery, the cat was completely normal with no residual neurological deficits.

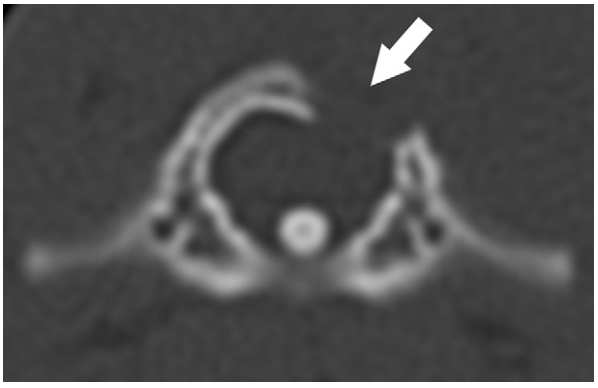

Fig 2. Postoperative transverse computed tomographic image of the atlas, after the partial laminectomy (white arrow).

Vertebral fractures are common in cats. In a study of 100 consecutive feline trauma patients, spinal injuries occurred in 26% of all patients.[1] Feline spinal injury most often results from motor vehicle trauma and high-rise syndrome, whereas bite wounds and gunshot wounds are uncommon causes.[2–4] Most injuries occur at the thoracolumbar and lumbosacral junction, whereas the cervical vertebral column is only very rarely affected.[2] In an effort to estimate the incidence of feline cervical vertebral fractures, the literature was reviewed by the author. Of a total of 515 cats with spinal injuries, only six had cervical vertebral fractures.[1–10] The axis (C2) was affected in three animals[4,10] and the fifth cervical vertebrae (C5) in another.[7] Two more feline cervical fractures were not further localised.[3] This stands in contrast to canine studies, which describe an involvement of the cervical vertebral column in 7–20% of cases.[2,7,11] Possible explanations for this disparity between species could be differences in aetiology and body size.[2] Anatomical differences might further contribute, but comparative experimental biomechanical studies are lacking.

It was not unexpected that the radiographs of the cervical vertebral column were inconspicuous. In a recent study, that evaluated radiographic sensitivity and negative predictive value for acute canine spinal trauma, radiography only had a moderate sensitivity for vertebral fractures (72%) and subluxations (77.5%). Low negative predictive values were found for the presence of vertebral canal narrowing (58%) and fracture fragments within the vertebral canal (51%). It was concluded that radiography cannot be used to reliably rule out acute vertebral lesions, and further imaging is indicated in patients with a high risk of such injuries.[12]

Hypoventilation is a serious potential complication in any animal with a severe cervical spinal cord injury. Three main mechanisms have been proposed. The resulting cervical myelopathy may be extensive enough to interrupt conduction along all motor fibres of the spinal cord, resulting in near-paralysis of the respiratory muscles. Haemorrhage and oedema may secondarily affect the respiratory centres of the medulla and C1 spinal cord, leading in turn to a decreased respiratory drive. Lesions involving the C5 spinal cord segment

or the corresponding nerve roots, may result in diaphragmatic paralysis due to phrenic lower motor neuron damage.[13] In our case, ventilation remained spontaneous during the entire anaesthesia and monitoring never suggested hypoventilation or respiratory compromise. The location of the lesion was too far cranially for a direct phrenic nerve injury to occur, but the first two mechanisms might have resulted in hypoventilation. Most probably the spinal cord injury was not severe enough, otherwise hypoventilation might have required mechanical ventilatory support.

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