Surgical treatment of a traumatic intracranial epidural haematoma in a dog

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Summary
A 10-month-old Czech wolf dog was unconscious after being kicked in the head by a horse. The following day, the dog was atactic and collapsed after several steps. The level of consciousness was decreased. Cranial nerve examination was normal and right postural reactions were decreased. Spinal reflexes were intact in all limbs. The diagnostic work-up included a computed tomography (CT) scan of the head with IV contrast. A lenticular shaped, hyperdense, non-enhancing lesion was observed in the left fronto-parietal region. A diagnosis of intracranial epidural haematoma was made. Two craniotomies were performed on a different day and most of the haematoma was removed. Corticosteroids and antimicrobial therapy were administered. Fifteen days after the surgery, the clinical examinations were unremarkable. Fifteen months later, the owners considered the dog normal. Intracranial subdural or intra-par enchymal haematomas have been described in the veterinary literature. To the authors’ knowledge, this is the first report of the successful management of an intracranial epidural haematoma in the dog. In humans, these lesions are well described. Common locations are temporal, parietal and frontal regions or a combination of these regions. Patients can be asymptomatic, present with varying clinical signs, or be unconscious. Based on the human literature, following trauma to the head, a CT scan should be performed even if the patient is asymptomatic. Some authors believe that there are not any absolute indications for conservative management versus surgical management.

Keywords
Epidural, haematoma, intracranial, neurosurgery, dog

Introduction
Head trauma may result from many different sources, such as motor vehicle accidents, bites or kicks, and malicious wounding, including gunshot wounds (1, 2). Both physical and biochemical factors are involved in the genesis of parenchymal injury. At the time of an impact, spontaneous rupture of parenchymal tissue may result from shear forces. Brain tissue may also be torn by bony or fibrous edges within the cranial vault or by penetrating injuries. Considering the lack of proven effective therapies, management of head trauma in dogs or cats can be frustrating. The role of surgical intervention for head trauma in dogs and cats is still uncertain but may be beneficial in some cases (1, 3).

Brain injuries occurring after trauma can be divided into two categories, primary or secondary, depending upon when the damage was done. Primary injuries occur at the moment of impact, whereas secondary injuries are delayed, and are usually vascular in nature. They also can be classified as extra-axial or intra-axial, according to their relative location. Interior parenchymal injuries are referred to as intra-axial trauma, and those affecting exterior structures, such as the meninges or cerebral spinal fluid (CSF), are known as extra-axial. Epidural haematomas are classified as extra-axial lesions (4).

Case report
A 10-month-old female Czech wolf dog weighing 27 kg was admitted for evaluation after being kicked in the head by a horse on the previous day. It had remained unconscious for a while. The patient was treated a few hours after the incident with corticosteroids and intravenous fluids. It improved slowly during the first day but the referring veterinarian was concerned about its ‘degree of consciousness’.

At presentation to our clinic, physical examination was unremarkable, except for the presence of left-sided epistaxis. Deformations of the nasal bone and skull were not palpable. The level of consciousness was stable but still decreased from normal. Cranial nerve examination was normal. Proprioceptive ataxia was observed, characterized by repetitive falling and collapses to the floor. On neurological examination, postural reactions, including proprioception positioning and hopping, were decreased on the right front and right hind limbs. Spinal reflexes were present and normal in all of the limbs. Cutaneous truncal reflex was present and back/neck pain was not detected. Nociception and skin sensation were normal on all of the limbs. Based on the decreased mental status along with right-sided proprioceptive positioning deficits, it was considered that the lesion was localized in the left thalamocortex. Differential diagnoses for this lesion were: fracture of the skull, brain haematoma, focal or diffuse oedema, with or without a falx herniation, or intracranial haemorrhage.

A complete blood cell count, coagulation profile and a serum biochemical analysis were within normal limits. The diagnostic work-up included a computed tomographic examination (CT) of the brain with IV contrast. The patient was premedicated with diazepam (0.2 mg/kg) and general anaes-
Anaesthesia was induced with propofol \( \text{b} \) (6 mg/kg). Anaesthesia was maintained with isoflurane \( \text{c} \) in oxygen. A CT of the brain was performed (Figs. 1 and 2) in a transverse plane using a brain window with a thickness of 3 mm and an index of 5 mm before and after administration of intravenous contrast medium (54 ml Telebrix \( \text{d} \)). A helical acquisition was performed in order to obtain reconstructive images. A well delimited bifrontal lesion, hyperdense and mostly homogeneous was observed (Fig. 1A, B).

This lesion had a lenticular shape with the wide portion located on the bone and was measured (length: 30 mm, width: 13 mm and height 22 mm), and was hyperdense, mean 63 Hounsfield Units (SD ± 4), non contrast enhancing (mean value 64 Hounsfield Units, SD ± 4). It was located in the left fronto-parietal region dorsal to the meninges. This lesion caused a mass effect (ventro-medial displacement of the left ventricle) and a right midline shift of the brain. Diffuse oedema was located in the left hemisphere around the described lesion. The frontal sinus also had a similar hyperdense lesion.

Based on these considerations, a diagnosis of traumatic left fronto-parietal epidural haematoma and a left frontal sinusal haematoma, with many fissures of the frontal and parietal bones was made.

Based on the size of the extra-axial lesion diagnosed on the CT scan compared to the size of the brain, along with the presence of mass effect and secondary displacement of the left ventricle and the left hemisphere, it was decided to approach the epidural haematoma surgically. Although the dog had improved after having been unconscious, the removal of the haematoma could potentially speed recovery, and stop the secondary lesions caused by the compression of the left hemisphere. The lesion was located in a position accessible by surgery, which was performed the day after the CT. The dog was anaesthetized using the same protocol as used the previous day. Analgesia was administered with morphine \( \text{e} \) (0.1 mg/kg) before and after surgery. Corticosteroids (prednisone \( \text{f} \), 1 mg/kg/d) and antimicrobial therapy (cephazolin \( \text{g} \), 22 mg/kg/d) were also administered perioperatively. The dog was positioned in sternal recumbency with the head elevated and securely fastened on a sand bag. Care was taken not to compress the jugular veins in order to prevent intracranial hypertension. The head was turned slightly onto the right side to enable better exposure of the left craniotomy site. The surgical approach was located caudally in the left frontal bone. A curved skin incision was made followed by elevation of the temporal muscle. A craniotomy was performed using an air-powered drill to elevate a bone flap sufficiently large to permit extraction of the haematoma. Small holes were drilled into the left frontal bone with a 2 mm drill bit, outlining an ovoid-shaped flap which was removed with bone forceps (Fig. 2). The haematoma was difficult to extract because it was not yet organized; the majority of it was aspirated, followed by removal of small pieces with a curette. Although the haematoma was not adherent to the dura, it was not possible to remove it in its entirety. Bleeding from its location was not observed during the procedure, and the original bleeding could not be determined. Bone reconstruction could not be attempted. A standard closure with suture of the temporal muscle was performed. A second post-operative CT examination showed the craniotomy site covered with muscles. Most of the haematoma had been removed but some persisted rostral to the surgical site. A right midline shift and a compression of the left hemisphere was still present. The day after the first surgery, it was decided to surgically remove the persistent portion of the haematoma. Clinically, the dog continued to present an abnormal mental status, abnormal gait, and severe

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\( \text{f} \) Fluorochrome, Lyon, France.
\( \text{g} \) SoluMedrol \( \text{g} \), Pfizer, Paris, France.
\( \text{h} \) Sulfate de Morphine \( \text{h} \), Laboratoire Aguettant, Lyon, France.

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proprioceptive positioning deficits on the right front and right hind limbs. The original incision was opened and the osteotomy site was enlarged with rongeur forceps in order to allow better exposure. The frontal sinus was not penetrated to prevent post-operative complications. A post-operative CT examination was performed (Fig. 3A, B). A new haematoma, based on the level of intensity on CT (34 Hounsfield units), which was most likely secondary to the surgery, was observed. Further compression was not present and the midline of the brain hemisphere was in the normal position.

The day after the second surgery, the dog was able to walk. The physical and neurological examinations detected a significant improvement in the dog’s mental status, gait and postural reactions. Antimicrobial therapy was continued for 13 days, and corticosteroids therapy for six days. The dog was discharged three days after the second surgery.

Two weeks after the second surgery, the physical and neurological examinations were normal. Six months and 15 months after the surgery, a telephone interview with the owners indicated that they were satisfied with the treatment and that they considered the dog to have a normal level of consciousness and gait; it was able to climb stairs and run without any clinical signs.

**Discussion**

Intracranial haematomas are classified based on imaging examinations (CT scan, magnetic resonance imaging [MRI]) as extradural, subdural or intra-parenchymal. The epidural space is located between the skull and outer layer of dura. A few cases of subdural and intra-parenchymal intracranial haematomas have been reported in the veterinary literature (5–9). Spinal epidural haematomas have also been reported, usually associated with intervertebral disk disease (IVDD) and von Willebrand’s disease, and diagnosed based on MRI examinations (10, 11). In those cases (10, 11), both epidural haematomas and disc extrusion (if an IVDD was present) compressed the spinal cord and surgery was necessary to remove the extruded disc and haematoma. To the authors’ knowledge, this is the first case report of intracranial epidural haematoma successfully treated by removal of the haematoma in the dog. Regardless of location, epidural haematomas rapidly form after haemorrhage from rupture of the venous sinuses. The haemorrhage causes a diffuse pain secondary to the inflammatory response (10–12). The stage of a haematoma can be evaluated on imaging examinations as hyperacute, acute, subacute or chronic. Acute haemorrhage appears hyperdense (+50 to +70 Hounsfield Units) (13), whereas subacute haemorrhage is more isodense (similar in signal intensity to the surrounding brain tissue).

An incidence of 1.5 extradural haematomas per 100,000 persons per year has been reported (14). The increased use of CT scanners has allowed detection in a higher number of epidural haematomas in asymptomatic human patients. Common locations (90 to 95%) are temporal, parietal and frontal regions, or a combination of these regions. Posterior fossa haematomas as well as occipital haematomas are also described but they are uncommon (14–17). Epidural haematomas in humans are most commonly secondary to trauma, but can also be secondary to vascular disease or tumour. Delayed epidural haematomas can occur after a surgery for decompression of another epidural (and are then evaluated in the same way as a traumatic haematoma), subdural or intracerebral haematoma, as was the case in our patient (a small epidural haematoma was found after the second surgery). In children, it is most commonly secondary to a fall from a height, whereas it is more commonly due to high velocity automobile accidents in adults (14, 17, 18). When it is traumatic, it is often associated with a skull fracture involving the squamous portion of the temporal bone with a tear of the middle meningeal artery or vein. Posterior fossa epidural haematomas are frequently due to bleeding from dural veins and sinuses. The venous epidural haematoma rarely enlarges, whereas arterial epidural haematoma tends to strip the dura further away from the bony calvaria resulting in enlargement. Most haematomas are from arterial bleeding (2, 12, 17).

Clinical signs associated with intracranial epidural haematomas can vary depending on the severity of the trauma, the location and the time elapsed from injury to presentation. Some patients can be asymptomatic for several days. A clinical investigation using imaging examinations as CT or MRI, if used, can help diagnose asymptomatic epidural haematomas. On the other hand, some patients who are asymptomatic only present for an evaluation when clinical signs appear (headache, persistent vomiting, pyramidal signs). In human medicine, some authors recommend performing a CT scan after a

![Fig. 3 A and B Cranial computed tomographic examination, transversal and longitudinal planes. The craniotomy had been performed caudal to this image. A recent haematoma (black arrow) due to the second surgery is present, but most of the traumatic epidural haematoma has been removed. No more compression is present and the normal midline position of the brain hemisphere is observed.](image-url)
head trauma even if the patient is asymptomatic in order to avoid a fast deterioration if a haematoma is present (16, 18). Our current practice is to perform a CT scan on animals who are admitted with clinical signs secondary to head trauma, but it is currently not our practice in an asymptomatic patient. Loss of consciousness can be initial and persistent, secondary to deterioration after a lucid interval, or followed by a regained consciousness. Our patient lost consciousness and then regained partial improvement in mentation (15, 16, 18). Patients can arrive in a conscious state but restless, or with neurological deficits (16). The passage from an asymptomatic phase to a comatose condition is probably due to an increase in the volume of the haematoma and, in particular, to the development of a marked shift in the midline structures of the brain (16).

Computed tomography has different advantages compared with MRI for the examination of patients with head trauma (17, 19). It is widely available, fast, and accurate for detecting acute haemorrhage. High resolution CT is excellent for evaluating facial and skull fractures. Epidural haematomas are readily detected by CT, and can be differentiated from subdural haematomas. However, CT has a number of disadvantages when used to evaluate head injuries and particularly epidural haematomas. Isodense or low-density acute haemorrhages can be seen in patients who are severely anaemic or who suffer from disseminated intravascular coagulopathy. A small epidural (or subdural) haematoma may not be detected if the appropriate setting for window width and level is not used (4, 12, 17, 19). The advantage of MRI, in head trauma, is that it reveals not only any abnormal fluid collection but its entire extent over the cerebral convexity. Therefore, medial displacement of the superficial cerebral veins is considered to be a conclusive indication of an extracerebral fluid collection. Direct images in many different planes, without patient manipulation, with MRI help to differentiate peritentorial and subtentorial extracerebral haematomas from intra-axial lesions. Also, the volumetric perception of extracerebral fluid collection is much more accurate with multiplanar nuclear magnetic resonance images (12, 17, 19, 20). Due to the greater availability and the shorter imaging time, we believe that CT scan is better suited in cases of head trauma in dogs and cats. In order to diagnose an epidural haematoma, a lenticular shaped, biconvex, high-density extra-axial mass that does not cross suture margins is usually readily observed on a CT examination. In humans, an associated skull fracture is seen in 90% of the cases (17, 20). In our case, the haematoma was associated with skull fractures and an important shift of the midline of the brain. A lower incidence of fractures is observed in children because of their elastic bones (18, 21). A CT finding that can also be used to predict rapid expansion of an epidural haematoma is the presence of low-density areas within high-density lentiform collection. The low-density areas are thought to represent swirling blood in active bleeding (4, 12, 17).

There are not any concrete guidelines for the conservative management of epidural haematomas. In general, patients who are asymptomatic or who display minor symptoms, with small haematomas (15 to 20 mm), and with a lack of, or modest, midline shift, might be candidates for conservative treatment (21–23). Due to the variability in brain size in dogs, it is impossible to consider the absolute size of the haematoma as an indication for surgery. However, it is appropriate to evaluate the proportion of the lesion compared to the rest of the brain in order to determine if a mass effect is present, and most importantly, to assess the clinical and neurological status of the patient, as well as its evolution since the trauma. In our patient, the neurological status did not improve after having initially recovered consciousness, and the size of the lesion appeared to be causing a clinically significant mass effect on the left ventricle. It is thought that there is an absence of absolute indication for surgery, and that some dogs who were treated with a surgical removal of the haematoma may improve with conservative management. However, until further studies have been completed, we share the opinion that the removal of a lesion causing a mass effect may speed up recovery and potentially reduce the secondary effects of the mass on the brain.

One factor which remains unclear is the role of skull fracture in resorption of haematomas. In humans, one study (21) showed that resorption rate was higher in patients with a skull fracture. It is thought that the clot might be pushed into the skull diploic bone through the fracture and be resorbed, or integrated by bone healing. Survival from traumatic epidural haematomas in humans is between 70 to 95% (14–16, 18). The outcome in children is distinctly better, when compared to their adult counterparts with a similar neurological examination, with a mortality varying from 3.4% to 10% (18).

**Conclusion**

To the authors’ knowledge, this is the first report of the successful treatment of an intracranial epidural haematoma following trauma in the dog. Since the haematoma was large and had resulted in a midline shift of the brain, and the dog ceased to improve after regaining consciousness, surgery was performed to remove most of the haematoma, thus allowing the dog to make a complete recovery.

**Acknowledgement**

The authors thank Dr. Jennifer Lansdowne for editorial assistance with this manuscript.

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**Vet Comp Orthop Traumatol 5/2008**

460 Cabassu et al.

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