Introduction

Before the advent of diagnostic imaging and neurosurgical procedures, brain abscesses were a life threatening condition (1, 2). In human medicine, the most common microbial sources of brain abscess are contiguous foci related to ear, sinus, or dental infections; these account for more than 50% of cases in most series (1, 3). Metastatic spread of a distant haematogenous infection (25% of cases), trauma, or surgery are other possible sources of brain abscess (1, 3). According to several studies and reports, surgery is the treatment of choice for brain abscesses in humans (4–7). However, there is little information available regarding the treatment of cerebral abscesses in animals. Recently, the first surgical case of a canine intracranial abscess, presumably due to bite wound, was reported (8). The present case report details the successful surgical management of an intracranial abscess in a cat following a presumed bite wound.

Case report

A nine-year-old male castrated European Shorthair cat weighing 3.5 kg was presented with a six-day history of signs of progressive depression, pyrexia (39.6°C rectal temperature), and ataxic gait. Two weeks prior the owners noticed a small wound at the base of the right ear. At presentation no skin lacerations were visible. According to the owners, the cat regularly fought with its neighbourhood cats.

Because of the pyrexia, and possible pain, the cat had been treated with ceftiofur (8 mg / kg S.C.) and tolfenamic acid (4 mg/kg P.O.) initially by its referring veterinarian. The day after therapy had started, the body temperature decreased to within normal limits, but the signs of depression and ataxic gait did not improve.

On presentation the cat was responsive but appeared depressed. There was not any evidence of a wound on the ear, as had been reported by the owner. Vital parameters included a rectal temperature of 38.0°C, heart rate of 160 / min, and respiration rate of 40 / min. In addition, the cat had sticky mucous membranes. On neurological examination, the cat was responsive but appeared to be depressed. The cranial nerve examination revealed a decreased menace response of the left eye. Anisocoria was evident with presence of mydriasis of the right eye. Pupillary light reflex performed in a dark environment revealed an absent direct and consensual response from the right eye. No other abnormalities were detected. On gait and posture examination, the cat was ataxic on all limbs with intermittent circling to the right. Postural reactions were delayed in all limbs while spinal reflexes were adequate.

Based on the neurological deficits, the lesion was localised to the right side of the cerebral cortex (decreased menace in the left eye, and circling to the right), extending to the right oculomotor nerve (dilated right pupil unresponsive to light directed into the right temporal lobe lesion adjacent to a small defect in the temporal bone suggestive of a meningo-encephalitis with concurrent abscess formation. The site was surgically approached by a rostrotentorial craniectomy. A cerebral abscess was found and debrided. Histopathological examination of the removed tissue demonstrated a subacute to chronic purulent encephalitis with extensive necrosis of brain tissue. Neurological symptoms resolved completely within two weeks and full recovery was observed four weeks after surgery.

Keywords

Intracranial, abscess, bite wound, cat, Propionibacterium species

Summary

A nine-year-old male castrated European Shorthair cat was presented with a six-day history of progressive depression and ataxic gait. Neurological examination revealed depression, absent menace in the left eye, absent pupillary light reflex in the right eye, anisocoria, circling to the right, and delayed proprioception in all limbs. Magnetic resonance imaging showed a space-occupying lesion was localised to the right side of the cerebral cortex (decreased menace in the left eye, and circling to the right), extending to the right oculomotor nerve (dilated right pupil unresponsive to light directed into the right temporal lobe lesion adjacent to a small defect in the temporal bone suggestive of a meningo-encephalitis with concurrent abscess formation. The site was surgically approached by a rostrotentorial craniectomy. A cerebral abscess was found and debrided. Histopathological examination of the removed tissue demonstrated a subacute to chronic purulent encephalitis with extensive necrosis of brain tissue. Neurological symptoms resolved completely within two weeks and full recovery was observed four weeks after surgery.

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either eye). The differential diagnoses for rapidly progressive cerebral dysfunction in cats include inflammation – both infectious (Toxoplasma gondii, viral, bacterial, mycotic or parasitic infection) and non-infectious causes, as well as neoplasia.

The cat was stabilised in the intensive care unit with the administration of maintenance fluid therapy, amoxicillin and clavulanic acid\(^d\) (20 mg/kg I.V. TID), metronidazole\(^e\) (20 mg/kg I.V. TID), dexamethasone\(^f\) (0.4 mg/kg I.V. SID) and assisted feeding. A complete blood count and biochemical profile were obtained and revealed a leucytosis of 25.2 x 10^9/L (reference 6.3–19.6 x 10^9/L), differential count: neutrophils 81.1%, lymphocytes 9.1%, monocytes 2.8%, eosinophils 0.4%, basophils 0%, normoblasts 0%). The cat was tested feline immunodeficiency virus and feline leukaemia virus negative by Western Blot and immunofluorescence assay tests respectively.

A magnetic resonance imaging (MRI) study of the cranium was performed using an open magnet, 0.2 Tesla magnetic resonance unit\(^1\) on the second day of hospitalisation. Transverse spin echo T1-weighted, proton density weighted, and T2-weighted images were acquired before contrast injection. Transverse spin echo and gradient echo T1-weighted images were acquired after intravenous contrast injection (gadolinium, 0.3 ml/kg)\(^g\). An intracranial space occupying lesion was present in the temporal lobe of the right cerebral hemisphere causing a midline shift of the falx cerebri to the left (Fig. 1). Compression and displacement of the right lateral ventricle and adjacent brain tissue were also apparent. The lesion was not well-defined and heterogeneous areas of hypointense and slightly hyperintense signal (compared to the surrounding brain) were visible in the T1-weighted images. The lesion had a hyperintense signal (compared to the surrounding brain), and was surrounded by a streaky hyperintense signal at the junction of gray and white matter on the proton density and T2-weighted images. A defect approximately 2–3 mm in diameter was visible in the right temporal bone. The bone was thickened at the rim of the defect. A hypointense area was visible in all sequences in the adjacent right temporal muscles extending into the subcutis and cutis. A diffuse, poorly-defined, contrast enhancement of the lesion was visible, extending from the cutis at the right ear base via the temporal musculature to the periosteum of the temporal bone. Intracranial contrast enhancement was visible via the temporal bone defect to the meninges, and extending as a 14 x 17 x 11 mm amorphous lobular mass in the temporal lobe of the right cerebral hemisphere. At least four rounded hypointense areas were present in the mass. The largest was 8 mm in diameter and located in the caudo-dorsal aspect of the mass.

These findings were suggestive for a perforating trauma of the right temporal skin, musculature and bone with concurrent infection and inflammation of the right temporal lobe of the cerebrum and meninges, and compression on the oculomotor nucleus, which probably caused the anisocoria. Abscess formation was likely considering the hypointense areas in the mass. A diagnosis of meningo-encephalitis, brain abscess and brain oedema with concurrent increased intracranial pressure was concluded.

Supportive care at the intensive care unit was continued as had been started before the MRI study (Fig. 1). Treatment by surgical decompression and debridement of necrotic tissue was recommended.

The following day, the cat was premedicated with buprenorphine\(^h\) (20 µg/kg I.M.). Anaesthesia was induced with propofol\(^i\) (5 mg/kg IV) and maintained with fentanyl\(^j\) (continuous rate infusion 10–20 µg/kg/hr) and isoflurane\(^k\) in 100% oxygen delivered through auffed endotracheal tube. Controlled ventilation was provided, and electrocardiography, blood pressure, capnography, pulse oximetry and urine production were monitored. The intravenous administration of maintenance fluid therapy, and amoxicillin and clavulanic acid (20 mg/kg I.V. TID), and metronidazole (20 mg/kg I.V. TID) were maintained throughout the procedure and continued postoperatively during recovery.

The site of the right temporal bone was clipped and aseptically prepared. After clipping, multiple scars 1–2 mm in diameter were evident in the skin around the base of the right ear.

The cat was positioned for surgery in sternal recumbancy with the head elevated to reduce intracranial pressure and to optimise the blood flow to and from the brain. The site was surgically approached by a rostrotemporal craniectomy. A small defect in the temporal bone was encountered. The craniectomy was performed by enlarging the temporal bone defect using Kerrison rongeurs. During this approach, the dura mater started protruding through the osteotomy site which was indicative of increased intracranial pressure. After incising the inflamed and thickened dura mater, a viscous yellow discharge flowed out of the defect (Fig. 2).

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\(^{a}\) Amoxiclav: Sandoz, Almere, The Netherlands
\(^{b}\) Metronidazole: Baxter, Utrecht, The Netherlands
\(^{c}\) Rapidexon: Eurovet, Bladel, The Netherlands
\(^{d}\) Magnetom; Siemens N.V., The Hague, The Netherlands
\(^{e}\) Dotarem: Guerbet, Roissy Cedex, France
\(^{f}\) Fentanyl: Bipharma, Hameln, Germany
\(^{g}\) IsoFlo: Abbott Animal Health, Maidenhead, United Kingdom

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The inflamed dura mater was excised and necrotic brain tissue was removed. During and after the surgical procedure, the site was thoroughly lavaged with sterile lactated Ringer’s solution. The defects in the dura mater and temporal bone were left open, but covered with the temporal muscle. The temporal musculature was reattatched at its origin by simple interrupted sutures, and the subcutis and cutis were routinely closed.

*Propionibacteria* spp. cultured from the purulent fluid were sensitive to amoxicillin and clavulanic acid, ampicillin, clindamycin, erythromycin, trimethoprim/sulfamethoxazole, and resistant to metronidazole.

The histopathological diagnosis made by a board certified pathologist was subacute to chronic purulent encephalitis with extensive malacia of brain tissue debrided from the site.

The cat was transferred to the intensive care unit for postoperative supportive fluid therapy and assisted feeding.

The day after surgery, the cat was still ataxic, the palpebral and papillary reflexes were positive, and menace response was negative bilaterally. The cat was not eating spontaneously, but assisted feeding was effective. Two days after surgery the cat was alert, and the menace response, pupillary light reflex and palpebral reflex were positive. The cat was showing decreasing ataxia and increasing strength. As the cat was still not eating spontaneously, the placement of an oesophageal feeding tube was recommended to the owner, but was declined because assisted feeding was increasingly successful.

Two days after the surgery the cat was sent home and the owner continued the assisted feeding at home for another two days. Meloxicam (0.1 mg/kg P.O. SID) was administered for seven days, and amoxicillin/clavulanic acid (12.5 mg/kg P.O. BID) for 30 days. Because the cultured *Propionibacteria* spp. were resistant to metronidazole, this was discontinued.

A week after the surgery the cat was active and the ataxic gait had almost resolved completely. The cat was able to jump on the couch and ate without assistance. Neurological symptoms had resolved completely within two weeks after surgery. Four weeks after the surgery the cat was fully recovered.

**Discussion**

To the authors’ knowledge, successful surgical treatment of an intracranial cerebral brain abscess due to a presumed bite wound in a cat has not previously been reported in literature. Although the owners had not witnessed a fight, an episode of bite wound trauma was a plausible explanation for the development of the brain abscess, due to the appearance of the small wound at the right ear base seen by the owners, the location of the typical scars, the pin-point hole in the *os temporale*, the abscess formation and culture of *Propionibacteria* spp. Surgical treatment might have been the key to the successful outcome of this clinical case. According to the literature, humans with a brain abscess that are treated medically have a poorer outcome than patients treated surgically (6, 7). In addition, surgical excision is associated with a lower recurrence rate and shorter hospitalisation than treatment by aspiration in cases of human brain abscessation (9, 10). Surgical treatment followed by antimicrobial therapy has been advised in treating human brain abscesses (11, 12). Other important factors that might have contributed to the favourable outcome in our case were the short time between diagnosis and surgery, and use of appropriate antibiotics. In a clinical analysis of 53 human cases, improved outcome was positively correlated with the early diagnosis using computed tomography or MRI, short time between diagnosis and surgery, and the appropriate use of antibiotic therapy (7).

The surgical approach by a rostrotemporal craniectomy was ideal because it permitted access to the abscess and immediate removal of its contents, thereby allowing definitive microbiological confirmation and histopathological diagnosis (2, 4–7, 13, 14).

In our case, there were not any neurological deficits remaining after surgery, although a considerable amount of necrotic brain tissue had been removed. In human medicine, partial removal of brain tissue does not necessarily result in major neurological deficits. Temporal lobe resection in 14 adult human patients with temporal lobe tumours resulted in mild permanent neurological deficits in only two patients (15). In addition, it was reported that neurological damage was evident in only eight percent of 176 humans who underwent temporal lobe resection for the treatment of refractory temporal lobe epilepsy (16).

In general, *Propionibacteria* spp. are aerotolerant, anaerobic Gram-positive, non-pathogenic bacteria that can be isolated from the infidibulum of the sebaceous glands. However, certain species of *Propionibacteria* can cause infections if they contaminate blood and other body fluids. They can cause a number of infections like the common skin disease acne vulgaris, and in certain cases *Propionibacteria* spp. have been associated with osteomyelitis and arthritis in a dog, multifocal abscession and placentitis in a cow, and granulomatous lesions involving the head, thorax, abdomen, pelvic area, and skin in cattle (17–21). In human neurosurgery, *Propionibacteria* spp., especially *P. acnes*, are regularly reported in causing postoperative intracranial infections and brain abscessation (22–28).

In the present case *Propionibacteria* spp. cultured from the purulent fluid showed in...
vitro sensitivity to amoxicillin and clavulanic acid. Antimicrobial drug distribution into the central nervous system is influenced by physical barriers and physiochemical (lipophilicity protein binding, molecular weight, ionisation, active transport) barriers. Tight junctions within the blood-brain barrier and blood- cerebrospinal fluid barrier limit diffusion of antibiotics with molecular weights higher than 500 Dalton (Da) (such as vancomycin and amphotericin B) through the intact barrier. In addition, antibiotic drugs that are highly protein bound have limited access to the central nervous system (13). Amoxycillin (419.46 Da) and clavulanic acid (237.25 Da) are capable of passing the blood-brain barrier, achieving the minimum inhibitory concentration in cerebrospinal fluid (4, 5, 14).

Although dexamethasone was administered preoperatively in this case, the use of corticosteroids in neurosurgical patients is contentious. There is very little evidence to support the use of glucocorticoids in neurosurgery. According to one review article, the administration of corticosteroids should not be routine in cases of brain injury because it has been associated with increased mortality of patients (29). However, another study showed that dexamethasone has protective effects by regulating the levels of NF-kappaB and relieved a secondary injury caused by the inflammatory cytokines in rat brain tissue after experimental traumatic brain injury (30). Although our case was not particularly acute but more subacute to chronic, and also involved infection, it is questionable if the use of corticosteroids affected or contributed to the positive outcome.

In conclusion, this clinical case presentation describes the successful surgical treatment of an intracranial cerebral brain abscess due to a presumed bite wound in a cat.

References


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