TITLE: Retrospective evaluation of the clinical presentation, magnetic resonance imaging findings, and outcome of dogs diagnosed with intracranial empyema (2008–2015): 9 cases

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Abstract

Objective – To describe the clinical presentation, advanced imaging findings, and short and long term outcomes in dogs with intracranial empyema.

Design – Retrospective case series

Animals – Client owned dogs diagnosed with intracranial empyema

Methods – Medical records from 2 referral hospitals were searched for dogs diagnosed with intracranial empyema. To be included in this study, dogs had to fulfill 1 or more of the following 3 inclusion criteria: a magnetic resonance imaging (MRI) scan with space occupying accumulation of extra-axial material suggestive of empyema, a cerebrospinal fluid analysis suggestive of empyema, or direct visualization of purulent material during intracranial surgery.

Results – Nine dogs with intracranial empyema were included, with a median age of 3.5 years (range 4 months-12.5 years). All presented as emergencies with 7 of the 9 dogs showing neurological abnormalities and 2 of the 9 with retro-bulbar swelling and exophthalmos. Six had surgical intervention, one was medically managed and the remaining two dogs were euthanized. Typical MRI findings included extra-axial, T1-weighted hypo to isointense, T2-weighted hyperintense material compared to grey matter with varying degrees of contrast enhancement, with 6/8 showing evidence of contiguous infection from adjacent structures on MRI. Seven had one or more samples sent for culture and sensitivity with *Enterococcus* (surgical swab), *Streptococcus pneumonia* (from CSF) and coagulase positive *Staphylococcus* (ear swab) being cultured. The median antimicrobial course length was 6 weeks (range 2 – 28 weeks). All dogs for which treatment was attempted survived to discharge, with a median hospitalization time of 7 days (range 4-10 days). Four of the seven are still alive at the time of writing (1 lost to follow up; 2 euthanized for other reasons) with all four considered neurologically normal with a successful long term outcome.

Conclusions – Although intracranial empyema in dogs is a rare condition, excellent outcomes are possible in those cases treated appropriately.

Introduction

Intracranial empyema is a neurological emergency that once diagnosed, requires rapid and aggressive intervention that comprises both surgical and medical management. Mechanisms of infection are thought to include hematogenous spread from other foci within the body (e.g septic emboli), contiguous infection from adjacent structures (inner ears, cribriform plate, sinuses and eyes), direct access (trauma, bite wound, iatrogenic (surgery/cerebrospinal fluid (CSF) acquisition)) and migration of foreign bodies or aberrant parasites[1-5]. Infectious agents reported to be implicated in intracranial empyema include *Staphylococcus* spp., *Streptococcus* spp., *Nocardia* spp., *Pasteurella* spp., *Actinomyces* spp., *Fusobacterium* spp., *Bacteroides* spp. And *Peptostreptococcus* spp and occasionally fungal organisms[5-7].

Intracranial empyema is a relatively rare condition, which is reflected in the limited number of reports currently in the veterinary literature, particularly concerning dogs. Although uncommon in people, intracranial empyema has been extensively described in the human medical literature with decompressive surgery and culture considered to be the gold standard in management of human cases[8-10]. Little is known about the clinical presentation, imaging findings and outcome in dogs, potentially stalling diagnosis and subsequent treatment. To the authors' knowledge, this is the first case series specifically evaluating the clinical features of intracranial empyema in dogs from multiple sources with the aim of describing its presentation, diagnostic imaging findings, treatment options and outcome. The aim is that by detailing such cases, we will attain a better understanding of the condition to allow owners to make a more informed decision regarding treatment.

Materials and Methods

Medical records from two referral centers between December 2008 and November 2015 were searched to identify dogs that had been diagnosed with intracranial empyema. To be included in this study, dogs had to fulfill 1 or more of the following 3 inclusion criteria: a magnetic resonance imaging (MRI) scan with space occupying accumulation of extra-axial material suggestive of empyema, a CSF analysis suggestive of empyema, or direct visualization of purulent material during intracranial surgery. Dogs that received surgical, medical, or both forms of treatment were included. Dogs were excluded from the series if medical records or imaging studies were incomplete or unavailable for review or if a final diagnosis of intracranial empyema could not be reached. This study was approved by the clinical ethical research committee board of the Royal Veterinary College RVC (reference number 2016 1526B)

Information retrieved from the medical records included signalment, history, general physical and neurological examination findings, concurrent medical conditions, imaging findings, CSF cytology, culture and sensitivity results, treatment administered, surgical procedure if performed, presence of complications and short term outcome.

All dogs were anesthetized and MRI scans were performed using a 1.5 or 0.4 Tesla scanner (Intera; Philips Medical Systems or Aperto, Hitachi Medical Corporation, Tokyo, Japan) or a CT scan using a 16 multi detector row unit CT scanner (Mx8000 IDT, Philips, Best, the Netherlands). Anesthetic protocols differed among dogs, premedication with methadone (0.2mg/kg IV), anesthetic induction with propofol (1mg/kg IV and then to effect) and maintenance of anesthesia with sevoflurance in oxygen was a frequently used protocol. All imaging series were available for review and comprised a minimum of T2-weighted, T1-weighted and T2-weighted FLAIR sequences and included transverse, sagittal and dorsal

images, with T1-weighted images acquired before and after IV administration of gadolinium contrast agent (0.1 mmol/kg gadoterate meglumine, Dotarem ®; Guerbet, Milton Keynes, UK or 0.05 mmol/kg gadobenate dimeglumine, Multihance ®; Bracco, Milan, Italy). All imaging studies were reviewed for diagnostic accuracy by two board certified neurologists (SDD and IP) and a residency trained radiologist (SG) and only those cases with imaging features consistent with intracranial empyema were included in the study. These features include the presence of extra-axial material that is T1 weighted hypointense, T2 weighted hyperintense with peripheral or heterogenous contrast enhancement together with contrast enhancing meninges.

CSF analysis was performed in selected cases (cisternal: total nucleated cell count ((TNCC) <5 cells/µl)), total protein (TP) 25mg/dl)). Owners were informed of the findings and the treatment options were discussed with a board-certified veterinary neurologist or resident in a veterinary neurology training program. The final decision for medical or surgical treatment or euthanasia was made by the owner of each dog.

Short-term outcome was defined as the period immediately following intervention up to the point of discharge and re-examination visits 4-6 weeks following treatment. Long-term outcome was obtained initially via telephone conversation with the referring veterinary surgeons. For dogs that were deceased, date and cause of death, as well as the last documented neurological status were recorded. Conforming to hospital ethics and welfare committee guidelines, only owners of dogs that were confirmed alive at the time of data collection were subsequently contacted. Owners were posted a letter with study details and a standardized questionnaire that had been reviewed and approved by the hospital ethics and welfare committee. Telephone interviews were conducted using the questionnaire, which included

questions covering the patients' quality of life, neurological status, any long term medication and overall response to treatment.

Results

Nine dogs with a median age of 3.5 years (range 4 months-12.5 years) were included in this study (Table 1). Four dogs were male (one neutered) and five were female (two neutered). Breeds included Labrador Retriever (n = 1), King Charles Spaniel (n = 2), Cocker Spaniel (n = 1), Jack Russell Terrier (n = 1), Golden Retriever (n = 1), Hungarian Viszla (n = 1) Bassett Hound (n = 1) and a cross breed (n = 1). All dogs presented with an acute onset of progressive clinical signs and seven of the nine dogs had been placed on antibiotics prior to referral. Despite seven of these dogs receiving empirical antibiosis prior to presentation, no improvement was seen and their signs continued to progress. Duration of clinical signs varied from 1 to 4 days (median 3 days, mean 2.8 days). General physical examination revealed abnormalities in six dogs, including a heart murmur (n=3 dogs), chronic dermatitis/otitis externa (n=1), submandibular lymphomegaly (n=2), pain on opening of the jaw (n=2) and pyrexia (n=2 dogs). Indications of a potential primary source of infection were observed in six dogs. This included unilateral exophthalmos (n=3), craniofacial wounds (n=2) and otitis externa (n=1).

Neurological examination revealed abnormalities in seven dogs, which included obtundation (n=5), postural reaction deficits (n=5), and cranial nerve deficits (n=5). Two of these dogs had experienced generalized tonic-clonic seizures prior to presentation. The two dogs without neurological deficits presented for further evaluation of unilateral exophthalmos.

Six dogs had complete blood cell counts performed on admission, two of which were

unremarkable, with abnormalities including neutrophilia (n=2), lymphocytosis (n=1) and mild anemia (n=1). Biochemistry was also performed in six dogs with no abnormalities identified. Three dogs did not have complete blood cell counts or biochemistry performed at the time of admission but did have venous blood gas analysis and blood smear evaluation performed with no abnormalities detected. Urinalysis was performed in one dog which revealed moderate hematuria and proteinuria with a negative bacterial culture. Thoracic radiography (n=1) and abdominal ultrasonography (n=1) were unremarkable. Echocardiography was performed in the three dogs with heart murmurs, two of which were consistent with mitral valve disease and the third with a congenital defect affecting the aortic valve. One dog had an ocular ultrasound which revealed signs consistent with retrobulbar cellulitis. Two dogs had ophthalmic examinations by board certified ophthalmologists with one having age related nuclear sclerosis and cataracts and the other having conjunctival hyperemia, exophthalmos and third eyelid protrusion.

All dogs had advanced imaging performed within 24 hours of presentation. Eight of the nine cases underwent MRI of the head with one case undergoing cranial CT. MRI revealed the presence of extra-axial material, which appeared T1weighted hypointense (n=6) or isointense (n=2) and T2 weighted hyperintense (n=8) to grey matter. On fluid attenuation inversion recovery images (FLAIR) the material had a heterogenous intensity (n=5) or was hyperintense (n=3) to grey matter. In two cases the material was extensive and compressing half of the cerebral cortex, with the remaining cases having material compressing one or more of the frontal lobes (n=2), the parietal lobe (n=1), the olfactory lobe (n=2), the cerebellum (n=2), the brainstem (n=1), the thalamus (n=1) and the temporal lobe (n=1), with the location often dictated by the source of infection. In seven of the eight cases the material appeared subdural in nature as it was crescent in shape and crossing suture lines. Perilesional edema was identified

in four of the eight cases. A degree of osteomyelitis was identified in five cases, affecting the frontal bone (n=3), the temporomandibular joint and calvarium (n=1) and the sphenoid bone (n=1). The material was contrast enhancing with peripheral (rim) enhancement (n=5) or heterogenous contrast enhancement (n=3). Meningeal enhancement was also evident in all cases and was predominantly dural in nature (n=8) with some dogs also having a degree of leptomeningeal enhancement as well (n=5). Five of the eight cases had evidence of mass effect in the form of midline shift (n=5), ventricular compression (n= 3), foramen magnum herniation (n=2) and caudal transtentorial herniation (n=1). (Figures 1 and 2). The neuroanatomical localization following examination of forebrain (n=5) and central vestibular syndrome (n=2) was consistent with the lesion location on MRI in all cases. In the two dogs that had a history of generalized seizure activity, the empyema identified on MRI was affecting the right side of the cerebrum in one dog and the left thalamus and left temporal lobe in the other dog.

CSF analysis was performed in two cases and the first revealed a pleocytosis (35% neutrophils; 40% monocytes/macrophages) with a TNCC of 260 cells/µl and total protein of 59 mg/dl. The cytology revealed occasional degenerate neutrophils, rarely containing very small coccoid structures and thin rod like structures. The structures identified were not convincing enough to definitively confirm intracellular bacteria and unfortunately this sample was not sent for culture. The second case did not have sufficient sample for a total nucleated cell count but did have a protein of 475 mg/dl and cytology consistent with a neutrophilic pleocytosis. The neutrophils varied from mature to variably karyolitic, frequently having phagocytosed the bacteria which were mature small diplococcoid in nature. Culture of the latter revealed a moderate growth of *Streptococcus pneumonia*.

Following the suspected diagnosis of intracranial empyema, six of the cases had surgical intervention either in the form of a single craniectomy (n=4), multiple small craniectomies (n=1) or a total ear canal ablation and lateral bulla osteotomy (n=1). Of those dogs that had intracranial surgery, two had a transfrontal approach and three a rostrotentorial approach, with the mean surgery time being 136 minutes (range:110-185mins). A durotomy was performed, which was followed by extensive lavage with saline before closure. A cranioplasty was not performed in any of the cases (Figure 3). Purulent fluid was seen and sampled for bacterial culture in all surgically treated cases. Of the 5 dogs that underwent a craniectomy, a neurological decline was not seen in any following the surgery; 2 remained neurologically normal, 2 remained neurologically static, and 1 experienced an immediate neurological improvement following surgery.

Of the remaining three cases, one was medically managed in the form of intravenous antibiosis administration and supportive care. Enrofloxacin and amoxicillin clavulanic acid were administered intravenously for 10 days before changing to oral enrofloxacin and amoxicillin clavulanic acid. This medically managed case had not received any antibiotics prior to referral and the decision to medically manage this case was owner driven, as they did not want any further invasive diagnostic or treatment modalities. The remaining 2 were euthanized immediately after diagnosis without treatment attempted. Of those dogs that underwent treatment (either medical or surgical), 4 of 7 had treatment initiated within 24 hours of presentation, 2 of 7 had treatment initiated within 36 hours of presentation, and 1 of 7 had treatment initiated within 48 hours of presentation.

Seven cases had one or more samples sent off for culture and sensitivity from surgery (n=5), fine needle aspirate (FNA) of an intra-oral lesion (n=1), CSF (n=1) or an ear swab (n=1).

From these samples, five returned with no bacterial isolates, one returned with *Enterococcus* (surgical swab), one with *Streptococcus pneumonia* (from CSF) and one with coagulase positive *Staphylococcus* (ear swab). Seven of the nine dogs received antibiotics prior to referral which included the two dogs with positive cultures. Four of the five dogs that had surgical samples taken for culture were already on antibiotics, and the one that hadn't received any antibiotics returned with no bacterial isolates.

All cases received antibiotics with seven cases being on at least two different types of antibiotics (Table 1). Antibiotics were initially given intravenously and subsequently changed to oral administration. Antibiotics were administered intravenously for between 2 and 10 days (mean 5 days, median 4 days) before being changed to oral administration. The median antimicrobial course length was 6 weeks (range 2 – 28 weeks). Some dogs received a short course (48 hours) of intravenous corticosteroids (n=2 (0.1-0.3mg/kg IV; Colvason; Norbrook 0.2%w/v)). Administration of intravenous mannitol was considered on an individual basis with bolus administration in case of suspected increased intracranial pressure. A total of four dogs were administered mannitol (n=4 (0.3-0.5g/kg IV)), with three receiving a single dose during the surgical procedure and the fourth dog receiving a dose during surgery and 48hours and 72 hours post-operatively. The latter was administered the extra doses of mannitol due to concerns that the dog was showing clinical signs suggestive of increased intracranial pressure. All four dogs that received mannitol had evidence of increased intracranial pressure on their MRI scans.

Seven out of the nine dogs survived to discharge, with the median time hospitalized being 7 days (range 4-10 days), with the one case medically managed being hospitalized for 10 days. One dog developed a corneal ulcer whilst hospitalized, which was medically managed with topical medication to which it responded to well. A neurological improvement was seen

within 24 hours (n=5), 48 hours (n=1) and within 72 hours (n=1) following intervention. At the time of discharge, three of the dogs were neurologically normal and the other four, whilst still showing neurological deficits (cranial nerve deficits n=3; postural deficits n=2; head tilt n=1), had all shown a neurological improvement. The two that did not survive to discharge were euthanized at the time of diagnosis without treatment attempted. One dog was euthanized under general anesthetic following the MRI scan with no reason given as to why treatment wasn't pursued, despite treatment being offered. The second dog was euthanized as the owners did not want to proceed because of an uncertain prognosis together with a lack of guarantee the dog would regain a good quality of life. Whilst the two euthanized dogs were neurologically abnormal at the time of presentation (similar to the other dogs in this series), nothing could be identified on their MRI scans or clinical notes that would have given a suggestion that these two dogs were precluded from having a similar outcome as the other dogs in this series if treatment was attempted.

Given the external signs evident on clinical examination, together with the advanced imaging findings, the routes of infection were suspected to be intra-oral (n=2), retro-bulbar (n=3), external trauma (n=2) and otogenic (n=2). For the two cases suspected to be intra-oral in nature, lesions were identified in the mouth when both patients were induced for anesthesia. Subsequent MRI revealed one to have an abscess involving the temporomandibular joint extending intra-cranially through the skull and the other revealed signs consistent of contiguous infection extending to the sphenoid bone and in towards the left temporal lobe at the level of the thalamus. One of the otogenic cases had underwent a total ear canal ablation and lateral bulla osteotomy 6 months prior to presentation on the same side as the empyema.

Of the seven cases that survived to discharge, we attained long term follow up for six (Table 1). Four of the six were still alive at the time of writing with all four being neurologically normal and free of clinical signs according to the owners and the referring veterinary surgeons. None of the dogs were on any long term treatment nor experienced recurrence of clinical signs. The two remaining dogs were euthanized for unrelated reasons. One was euthanized 3 years following surgery for gastrointestinal disease and the other was euthanized 17 months following diagnosis due to congestive heart failure. At the time of death, both cases were neurologically normal other than one dog retaining a mild head tilt according to the referring veterinary surgeons.

Discussion

Intracranial empyema is a neurological emergency that once diagnosed, requires rapid and aggressive intervention. For an owner, making decisions regarding treatment can be challenging given the expensive and invasive nature of surgical intervention and the uncertainty of the prognosis. To the authors knowledge, this is the first case series in the veterinary literature evaluating the clinical presentation, diagnostic findings, treatment and outcome of dogs diagnosed with intracranial empyema.

This case series highlights the differing presentations and underlying causes of intracranial empyema in dogs. The presumed sources of infection in this case series comprised otogenic, traumatic, retrobulbar and intra-oral extensions of infection. Interestingly, whilst sinusitis, otitis and skull trauma are amongst the most common sources of infection for human subdural empyema, the most common current etiology is subsequent to a neurosurgical procedure such as surgical evacuation of a subdural haemorrhage[8,11] It is possible with the

continued advancement of veterinary neurology coupled with increasingly performed intracranial surgery, there is the potential to see intracranial empyema more commonly as a complication following veterinary neurosurgical procedures.

Despite these variations within a particular condition, a consistent finding was the acute and progressive nature of the clinical signs. A previous study investigating otogenic intracranial infection categorized the onset of neurologic dysfunction into acute (1-48 hours), subacute (3-7 days) or chronic (> 7days), with the majority of the patients in that series having a chronic onset[2]. Elsewhere in the veterinary literature, the onset of neurological signs in cases with intracranial empyema have varied with some being acute, subacute or chronic in nature[1, 25]. In our series, the duration of clinical signs varied from 1 to 4 days (median 3 days, mean 2.8 days). If we were to categorize similarly to the previous study, then 4/9 would be classed as acute and 5/9 would be subacute. It is possible that certain origins of empyema are associated with an acute onset of neurological signs. This hypothesis however needs further investigation in future studies. Another striking finding of this case series was despite the severity of the presenting clinical signs, the majority of those cases in which treatment was attempted resulted in a good outcome. Only one of the cases presented in this series was medically managed, the majority being surgically treated. Although being presented with a dog with intracranial empyema can be overwhelming and stressful, our findings suggest that rapid and aggressive intervention can result in a successful outcome.

Although intracranial empyema is a manifestation of an infectious disease, general physical examination findings and ancillary diagnostics, such as a complete blood count, biochemistry, urinalysis, echocardiography, and abdominal and thoracic imaging did not reveal clear indications of a more generalized infectious process. For all dogs in which treatment was

attempted, one or more samples were submitted for bacterial culture and sensitivity testing. However only three of a total of eight samples returned culture positive, one each from a surgical swab, ear swab and CSF sample. Previous literature in the subject has suggested that culturing from CSF is not normally rewarding and CSF analysis itself is often not specific for bacterial abscessation[7,12,13]. Whilst the ear swab and CSF positive cultures were from the same dog, it was considered unlikely the ear swab isolate was the cause of the intracranial empyema. The fact that different isolates were cultured should not be considered surprising given that in dogs with otitis media, cultured samples from the horizontal ear canal and the middle ear have been reported to demonstrate different results in 89.5% of ears[24]. Culture directly from the surgical site was positive in just one of the five cases that had sampling from that site in this case series, and this particular case was already receiving antibiotics. A possible explanation for this could be due to the fact that the majority (7/9) of cases were placed on antibiotics prior to referral, which has previously been discussed in the literature[6,13]. Blood culture was not attempted in any of these dogs but could potentially be considered as an additional ancillary diagnostic in dogs with intracranial empyema given its use in other neurological disorders such as discospondylitis and spinal epidural empyema[22,23].

Appropriate surgical aftercare is a key component in order to successfully treat dogs with intracranial empyema. Most surgically treated dogs underwent a craniectomy, after which not only the basic principles of post-operative aftercare were adhered to but specific recommendations of care following intra-cranial surgery were considered. The use of corticosteroids in patients with intracranial empyema remains a contentious issue, demonstrated by the fact that only two of the nine dogs in this case series were administered corticosteroids. Corticosteroids have been administered to the majority of reported cases in the veterinary literature[2,25,26]. It is thought antibacterials administered to these patients induce

cellular destruction of bacteria and subsequent lipopolysaccharide liberation, leading to the initiation of a cascade of inflammatory events[14,15]. However, It is suggested that dexamethasone early in the treatment of bacterial meningitis, decreases the inflammatory response associated with the release of bacterial cell material[16,17]. In the human literature, the results are inconclusive, with previous reports supporting the use of short term anti-inflammatory doses of corticosteroids in people with bacterial meningitis, as they are beneficial in lowering intracranial pressure and reducing CNS inflammation[27]. However a recent systematic meta-analysis examining the use of adjunctive dexamethasone in patients with bacterial meningitis failed to suggest a clear benefit[28].

It remains unclear how long dogs with intracranial empyema should be kept on antibiotics for. In the absence of positive bacterial culture and sensitivity results, a broad spectrum, bactericidal antimicrobial with the ability to cross the blood-brain barrier and reach adequate concentrations in the central nervous system should be considered the ideal choice[12,13]. Whilst the majority of patients in this case series were placed on two different antibiotics, amoxicillin clavulanic acid was the most commonly used. Whilst this is a broad spectrum antimicrobial, it does not cross the blood-brain barrier effectively. It is however not completely clear how important the ability to cross the blood-brain barrier truly is in patients with intracranial empyema. It can indeed be assumed that animals with such severe brain pathology do no longer have an intact blood-brain barrier, allowing penetration of antimicrobials that might otherwise be restricted[10,18]. A further consideration is the fact that the majority of the empyema in this case series was epidural or subdural in nature and hence outside the blood-brain barrier. Enhancement following intravenous contrast administration was a consistent finding on MRI, further illustrating the questionable importance of blood-brain barrier penetration for antibiotics administered in patients with intracranial empyema. Whilst there is

no consensus on the length of antimicrobial therapy for these patients, demonstrated here by the wide range of 2-28 weeks, it is generally accepted that antibiosis should be initially administered intravenously followed by an oral course long term[19].

The median age of dogs in this case series was 2 years, with four of the nine dogs being less than 12 months old. This is consistent with the human literature where the majority of patients with intracranial empyema are between 10-20 years old[20,21]. Whether these dogs have a more immature immune system or are more prone to traumatic episodes is unclear. Although this finding could indeed suggest a predisposition for younger animals to suffer from intracranial empyema, a larger sample size would be needed to evaluate this hypothesis.

Whilst the MRI characteristics of intracranial empyema have previously been described[1,2,5], this is the first case series focusing on intracranial empyema in dogs from multiple different sources of infection. Generally, the empyema was subdural (occasionally epidural) in nature, T1 weighted hypointense and T2 weighted hyperintense with peripheral or heterogenous contrast enhancement. Evidence of concurrent osteomyelitis and a degree of mass effect was identified in individual cases. These MRI characteristics when considered together with the history and clinical signs of the patient can be considered suggestive of intracranial empyema. In differentiating subdural empyema from a subdural hematoma, you might expect the latter to potentially be more isointense to brain parenchyma on T2 weighted images, lack a contrast enhancing rim and one might also expect the presence of signal void on T2* weighted images[26].

This study is obviously limited by its retrospective nature, which restricted standardized patient assessment and treatment. Standardization of patient care was further limited by the

heterogeneous nature of included cases. Despite including animals over a 7-year period from two busy referral centers, only 9 dogs could be included. Although this could be considered a limitation of the study, this finding is most likely a reflection of the rare nature of intracranial empyema in dogs. Despite the small number and heterogeneous character of included cases, several clinically important conclusions can be drawn from this study. Intracranial empyema in dogs is rare, but can present as a neurological emergency that requires rapid and aggressive treatment. Affected dogs can present neurologically normal and the majority of patients do not demonstrate evidence of systemic disease on general physical examination or ancillary diagnostics. Treatment of intracranial empyema seems to be associated with excellent outcomes and a rapid recovery. Further studies are needed to evaluate the most appropriate type of surgical aftercare, type and duration of antibiotic treatment.

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Figure legends

Figure 1: MRI of a 7 year male entire Jack Russell Terrier with subdural empyema. The images show a T2weighted (A), T1weighted (B), T2weighted FLAIR (C) and T1weighted after IV administration of gadolinium based contrast (D) transverse images of the brain at the level of the thalamus. There is accumulation of extra-axial, crescent shaped, T2weighted hyperintense, T1weighted hypointense material compressing the left cerebral hemisphere (asterisk). The material does not suppress on FLAIR (C) and following gadolinium administration there is a peripheral enhancement of the material (D) and both dural and leptomeningeal diffuse contrast enhancement. There is a degree of midline shift and compression of the lateral ventricle (white arrow).

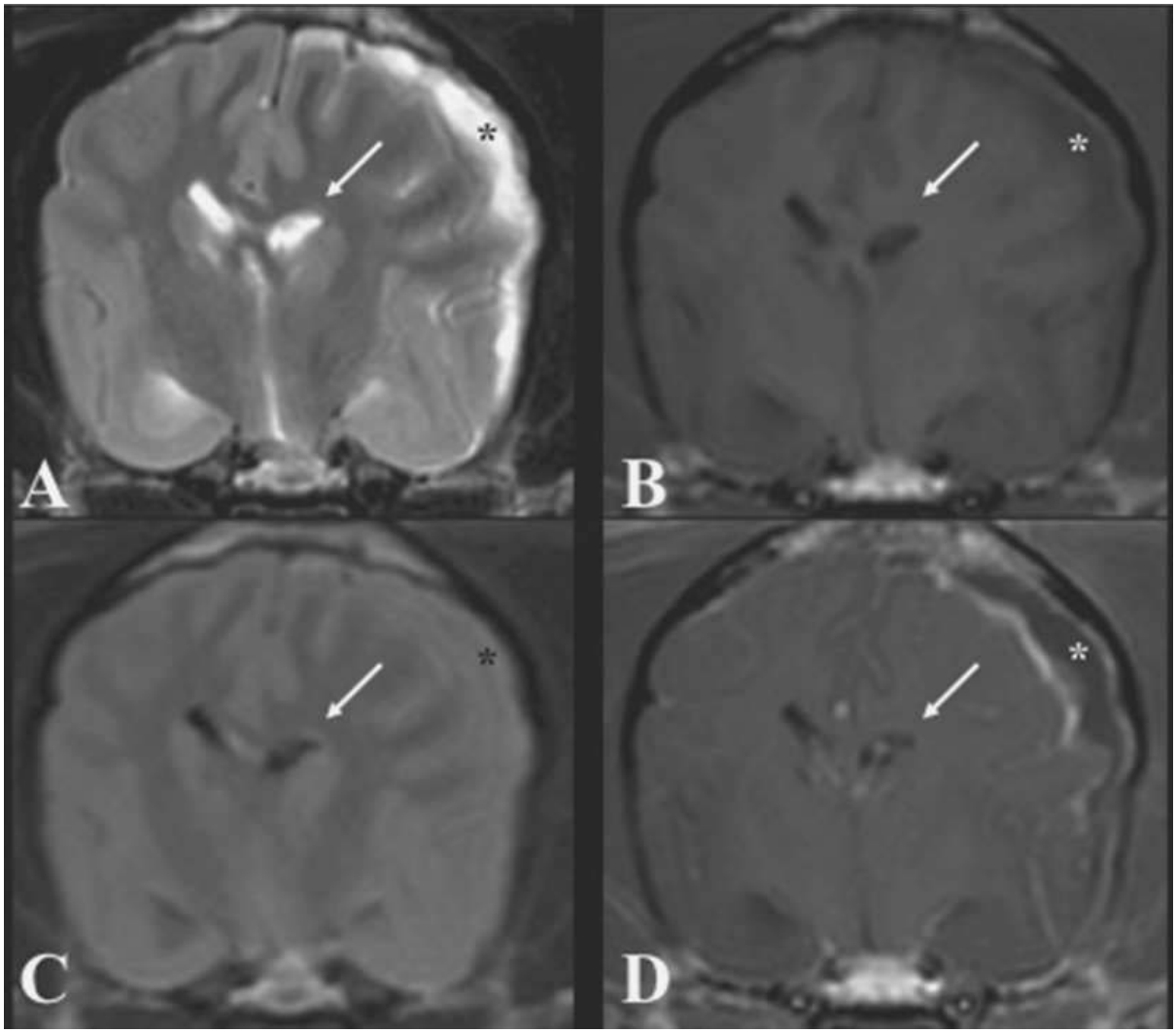

Figure 2: Transverse T2weighted (A), transverse T1weighted after IV administration of gadolinium based contrast (B), dorsal T1weighted (C) and dorsal T1weighted after IV administration of gadolinium based contrast (d) MR images of a 6-month old crossbreed with intracranial empyema. Accumulation of extra-axial, lentiform, T2weighted hyperintense, T1weighted hypointense material adjacent to the frontal and parietal lobes on the right hand side (white arrow). Following gadolinium there is a peripheral enhancement of the material (B and C) and there is a degree of midline shift to the left. There is also a degree of osteomyelitis (A and B: asterisk) with T2 weighted hyperintense thickening of the periorbital and retrobulbar tissues of the right eye. The right frontal sinus also contains hyperintense material together with a degree of mucosal thickening.

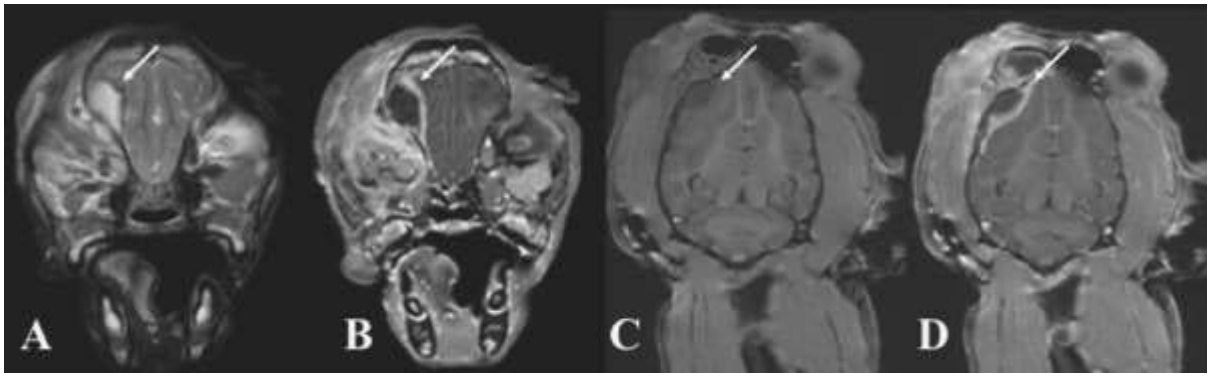

Figure 3: Rostrotentorial craniectomy in a 6 month old female entire Labrador retriever with subdural empyema secondary to a retrobulbar infection. A) Following the craniectomy, the intact dura is visible but appears discolored (white arrow). B) The dura has been incised and the empyema is visible in situ (asterisk). C) Following an intraoperative swab for culture and sensitivity, the empyema is removed and flushed with copious amounts of saline, with normal brain parenchyma now visible (white arrow).