

Cerebral abscess in dog - a Case report*

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ABSTRACT. Cosme J.C., Silva M.A., Santos R.P., Andrade Júnior P.S.C. & Nunes L.C. **Cerebral abscess in dog - a Case report.** [Abscesso cerebral em cão - Relato de caso.] *Revista Brasileira de Medicina Veterinária*, 37(1):15-19, 2015. Departamento de Medicina Veterinária, Centro de Ciências Agrárias, Universidade Federal do Espírito Santo, Alto Universitário, s/nº Cx Postal 16, Guararema, Alegre, ES 29500-000, Brasil. E-mail: louisiane.nunes@ufes.br; louisianecn@yahoo.com.br

Cerebral abscess is an affection of the nervous system with rare manifestation in dogs, caused by proliferation of pyogenic bacteria. The aim of this work was to describe the clinical and pathological findings in a case of cerebral abscess in dog, as well as to discuss the adopted treatment procedures. A 48-day old, male labrador dog was examined at the veterinary hospital of the Federal University of Espírito Santo (UFES), exhibiting a lesion caused by trauma on the frontal left region of the skull. The animal presented signs of ataxia and apathy, and received antibiotic and anti-inflammatory treatment. The animal died 30 days after consultation. Among other macroscopic findings, necroscopic examination revealed cutaneous scabby lesion on the frontal left region of the skull, cerebellar herniation, and increase of the left hemiencephalon. Microscopical analyses showed neutrophilic leptomeningitis of brain, cerebellum and cord, neuropil vacuolation, and neutrophilic infiltrate in the perivascular Virchow-Robin spaces of the brain. *Staphylococcus* sp. was evidenced by microbiological isolation. The non-specificity of the clinical signs in cases of cerebral abscess may hinder the establishment of an early diagnosis, thus reducing the treatment and survival chances of the animal.

KEY WORDS. Necrosis, inflammation, brain, canine, *Staphylococcus* sp.

RESUMO. Abscesso cerebral é uma afecção do sistema nervoso causada pela proliferação de bactérias patogênicas, com manifestação rara em cães. O objetivo deste trabalho foi descrever os achados clínicos e patológicos de um caso de abscesso cerebral em cão, bem como discutir os procedimentos de tratamento adotados. Aos 48 dias de idade, um cão labrador macho foi examinado no Hospital Ve-

terinário da Universidade Federal do Espírito Santo (UFES), apresentando uma lesão causada por trauma na região frontal esquerda do crânio. O animal apresentava sinais de ataxia e apatia, e recebeu tratamento com antibióticos e anti-inflamatórios. O animal morreu 30 dias após a consulta. Entre outros achados macroscópicos, o exame necroscópico revelou lesão subcutânea na região frontal esquer-

*Received on October 19, 2012.

Accepted for publication on February 9, 2014.

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da do crânio, herniação do cerebelo e aumento do hemiencéfalo esquerdo. Análises microscópicas mostraram leptomeningite neutrofílica de cérebro, cerebelo e medula, vacuolização de neurópilo, e infiltrado neutrofílico nos espaços de Virchow-Robin do cérebro. *Staphylococcus* sp. foi evidenciado pelo isolamento microbiológico. A não especificidade dos sinais clínicos em casos de abscesso cerebral pode dificultar o estabelecimento de um diagnóstico precoce, reduzindo assim as chances de tratamento e sobrevivência do animal.

PALAVRAS-CHAVE. Necrose, inflamação, cérebro, canino, *Staphylococcus* sp.

INTRODUCTION

Cerebral abscesses result from the proliferation of pyogenic bacteria, and consist of a neoformed cavity filled with pus (Jubb et al. 2007), which is nearly always secondary to a suppuration focus in another part of the body (Ratnaike et al. 2011). Pyogenic infections of the central nervous system (CNS) may arise from the blood circulatory system, by settling into wounds, or by direct extension to the brain from contiguous structures (Colgrove & Migaki 1976), all considered endogenous pathways (Arlotti et al. 2010).

Infections may arise from contiguous structures, including otitis media, paranasal sinusitis, mastoiditis, and dental infections, or through hematogenous dissemination from a remote source, such as infectious endocarditis, congenital cyanotic cardiopathy, and lung infections. The origin of infection can also be exogenous (Arlotti et al. 2010), as occurs in penetrating skull trauma or neurosurgical procedures (Erdogan & Cansever 2008).

The evolution of a cerebral abscess can be divided in four stages: early cerebritis (1st to 4th days), late cerebritis (4th to 10th days), early capsule formation (11th to 14th days), and late capsule formation (14th day) (Erdogan & Cansever 2008).

The clinical syndrome may be caused by factors involved in the organism-host interaction; abscess number, size and distribution; specific brain structures involved; and the disturbance of contiguous anatomical regions involving cisterns, ventricles, and dural venous sinus (Muzumdar et al. 2011).

Cerebral abscess is a lesion of complicated diagnosis and treatment, owing to the difficulty in obtaining specific clinical signs (Kielian 2004). It still represents a serious condition of the CNS, in spite of advances in neuroimaging, neurosurgery, microbiological techniques, and availability of new antibiotics, as related by Muzumdar et al. (2011). These

authors also affirmed that the lesion represents a direct interaction between virulence of the offensive microorganism and host immune response. Treatment success in cerebral abscesses requires surgery, adequate antibiotic therapy, and eradication of the primary source. However, many controversial issues concerning the management of this severe infection remain unsolved (Arlotti et al. 2010).

Occurrence of cerebral abscesses is very rare in domestic dogs (Munana 1996), probably due to the rich blood supply to the CNS by the hematoencephalic barrier, in particular to the grey matter, which prevents the establishment of bacteria (Kaplan 1985).

Further, it is important to comprehend the pathophysiology of cerebral abscesses, since clinical signs are not specific and, as a consequence, often diagnosed only during necropsy (Hirooka et al. 2007).

In view of the rare nature of this condition, as well as the difficulties of its diagnosis, the aim of this work was to describe the macro- and microscopic findings of a canine case of cerebral abscess.

HISTORIC

At the veterinary hospital of the Federal University of Espírito Santo (UFES), a 48-day old, male labrador dog was attended for vaccination and deworming. During the clinical examination of the animal, a lesion was observed in the frontal left side of the skull, which had been caused by trauma, according to report of the owner. Vaccination was performed, and lesion cleaning with physiological solution and application of a topic antibiotic spray (rifampicin) twice a day was prescribed.

One month after the first appointment, the dog was again attended for continuation of the vaccination protocol. However, the animal presented signs of apathy, ataxia, and volume increase in the left portion of the skull. Further, the animal showed tensed abdomen and pasty diarrhea. Canine distemper was suspected, and as differential diagnoses were indicated intoxication, skull fracture, cranial edema, encephalitis, or meningitis. In order to facilitate the diagnosis, complete hemogram and radiographic examination were carried out. The diagnosis established was cranial trauma in the frontal left region, edema, encephalitis, and meningitis. The diagnosis of canine distemper was discarded.

The animal was treated with 7.5 mg/Kg doxycycline every 12 hours (BID); 0.5 mg/Kg dexamethasone BID; 1 g/Kg mannitol; 25 mg/Kg dipyron every eight hours (TID); 2 mg/Kg tramadol TID; and 20 mg/Kg ceftriaxone BID. Despite this treatment, the animal died two days after consultation.

The corpse was transported to the Animal Pathology department for necropsy. Fragments of various organs and the whole encephalon were collected. The material was fixed in 10% formaldehyde, subjected to routine histopathological processing, and stained with hemato-

xylin-eosin (HE). Encephalic material was collected for microbiological culture.

External necroscopic examination revealed volume increase in the frontal left region with firm consistency, with presence of scabby cutaneous lesion. Opening of the thoracic and abdominal cavities and in situ examination of the organs revealed lung collapse and hepatic congestion.

During disarticulation of the head at the atlanto-occipital region, cerebellar herniation was observed, as well as dense cephaloraquidian liquid with increased volume, showing greenish color. Skin removal from the cranial region revealed granulation tissue under the cutaneous lesion, adhered to the left portion of the frontal bone. Encephalon exposition showed adherence and thickening of the leptomeninges, purulent secretion, and accentuated volume increase of the left hemiencephalon. Exudative lesions were mainly observed in the frontal, temporal, and parietal lobes (Figure 1 and Figure 2). The right hemiencephalon was compressed. Microbiological examination of the encephalon revealed presence of *Staphylococcus* sp.

Microscopic brain examination presented accentuated neutrophilic leptomeningitis, as well as necrosis with liquefaction delimited by a fibrous tissue capsule, which extended from the frontal to the parietal lobe. In the necrotic centers, a number of neutrophils and cell debris were observed. Intense neuropil vacuolization and neutrophilic infiltrate in the perivascular Virchow-Robin spaces were also detected. Further, cerebellum and spinal cord showed diffuse neutrophilic leptomeningitis, and neutrophil infiltrate was observed in the cord in the interior of the spinal canal.

DISCUSSION

CNS abscesses are rare in domestic dogs (Munana 1996), but considered common alterations in young production animals (Barros et al. 2006). The periosteum and the dura mater function as an effi-

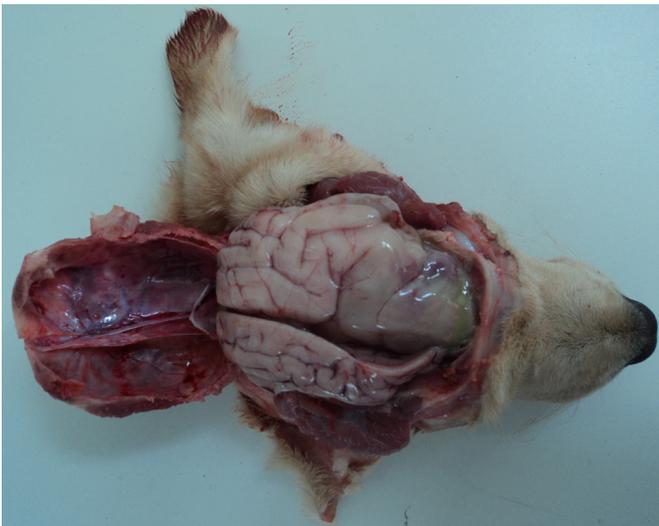


Figure 1. Superior projection of brain abscess of dog involving the lobes frontal, temporal and parietal.



Figure 2. Frontal projection of brain abscess of dog fixed in formalin solution revealing necrotic content.

cient barrier to protect brain and spinal cord from direct penetration of infections. However, susceptibility of the nervous tissue increases when active pyogenic processes are in course at contiguous tissues (Graça et al. 2011).

Kielian (2004) related that cerebral abscesses can originate from a penetrating brain trauma, developing as a response to parenchymal infection by pyogenic bacteria. Suspicion of abscess caused by trauma was also cited by Cornelisse et al. (2001), who accompanied a foal presenting abrasion on the left side of the head, and fracture of the parietal bone overlying the cranial vault, which resulted in acute ataxia and depression. Similar case was observed in this study, which reported a dog developing cerebral abscess due to cranial trauma, with cutaneous lesion in the frontal left region.

Cerebral abscesses can also directly or indirectly result from infections of endogenous origin (Arlotti et al. 2010), extending from foci at the paranasal sinuses, middle ear, and teeth, in addition to affections in other organs that disseminate hematogenously (Smith et al. 2007).

The clinical signs of ataxia found in the reported animal were similar to those described by Smith et al. (2007). These authors suggested that the most likely explanation for the neurological deficiencies resulting from cerebral abscesses may be brain and cerebellum herniation, caused by prosencephalon spaces being occupied by lesions, as well as possible multifocal CNS disease. Cerebellar herniation was also observed in this study, being due to the expansive character of the abscess. However, unilateral encephalon increase with involvement of the left lobes and compression of the right hemiencephalon were observed.

In the animal evaluated in this study, diagnostic

suspicions involved canine distemper, exogenous intoxication, cranial trauma, cranial edema, encephalitis, and meningitis. According to Smith et al. (2007) in cases of ataxia, differential diagnosis should include granulomatous meningoencephalitis, neoplasias, traumas, encephalitis, and vascular encephalopathies.

Early, precise cerebral abscess diagnosis, surgical intervention (Prasad et al. 2006), and efficacious long-term antibiotic treatment were shown to substantially reduce mortality and neurological sequelae from intracranial pathological processes (Cornelisse et al. 2001).

Three possibilities are available for cerebral abscess treatment: clinical intervention, abscess aspiration, or complete excision. The adequate option is selected according to factors such as scoring at the Karnofsky performance scale; primary infection; predisposing conditions; and abscess number, size, localization, and stage. Current cerebral abscess therapies generally include a combination of clinical and surgical interventions. In this context, antibiotics play a critical role in the management of cerebral abscesses, since they can be applied isolatedly or in association with surgical intervention (Erdogan & Cansever 2008).

In this case report, the established diagnoses were edema, encephalitis, and meningitis, and treatment included use of broad-spectrum antibiotics, and anti-inflammatory drugs. Probable reason for treatment failure was that the cerebral abscess, as well as the involved etiological agent, was not timely identified. It is worth noticing that the cranial fracture was only confirmed 30 days after the first clinical appointment, when the animal returned showing neurological symptoms.

These data corroborate with those of Arlotti et al. (2010), who affirmed that the use of antibiotics is mostly initiated before determination of the infecting organism and the sensibility to the active ingredient, being then continuously used, either empirically or based on the sensibility profile of the infecting organism over a long time span.

The rational use of antibiotics depends on the microorganism causing cerebral abscess at a certain spot. However, isolation of the causing agents, especially anaerobic pathogens, has always presented technical difficulties (Prasad et al. 2006). In this study, bacterial presence in the cephaloraquidian liquid was only verified during necroscopic examination.

Data show higher mortality rate in patients treated with corticosteroids. However, the use of anti-

-inflammatory drugs is recommended in patients with significant perilesional edema diagnosed radiologically (Mampalam & Rosenblum 1988). Owing to the fact that the reported dog presented cerebral edema, the use of corticosteroids was thus also indicated.

Erdogan & Cansever (2008) related that clinical treatment alone can only be considered when the patient does not present adequate conditions for surgical intervention, according to the following criteria: whether lesions are multiple, smaller than 1.5 cm in diameter, located in critical areas, or whether concomitant infections exist, such as meningitis or ependymitis. In this context, though diagnosis was only established upon necroscopic examination, it is possible to derive that the dog evaluated in this study was not in appropriate conditions to undergo surgical intervention, seeing that the extension of the cerebral abscess involved the frontal, parietal, and temporal left lobes, in addition to accentuated existence of leptomeningitis.

Surgical treatment was also considered unfeasible due to the absence of previous diagnosis. According to Ratnaïke et al. (2011), utilization of computed tomography technique for cerebral abscess diagnosis has contributed to improve many aspects of lesion management, by enabling earlier detection, localization, and orientation during the surgical procedure. These authors also related that chances of success in surgical procedures without computed tomography decrease significantly.

Kielian (2004) cited that some sequelae can be observed after infection, such as substitution of the abscess area with fibrous scar, loss of cerebral tissue by surgical excision, as well as abscess rupture with consequent death.

According to experimental development carried out in mice, cerebral abscess formation can be divided in three components: initial edema and glial activation period; intermediate phase of neovascularization and fibronectin deposition; and final phase of collagen deposition and progressive fibrosis (Flaris & Hickey 1992). The microscopical findings observed in the present animal corresponded to the initial and intermediary stages of abscess formation. Neutrophilic leptomeningitis was also observed in cerebral abscess in a four-year-old mare (Hanche-Olsen et al. 2012). Leptomeningitis and diffuse purulent/suppurative inflammation reaching from the brain to the spinal cord have already been described in other species, including cattle (Barros et al. 2006).

Abscess formation in the frontal region, obser-

ved in this dog, is in accordance with report of Muzumdar et al. (2011), who affirmed that most abscesses occur in the frontal, temporal and, posterior fossa regions.

The presence of pyogenic bacteria, such as *Staphylococcus* sp., as found in this case, were also related by Jubb et al. (2007), who mentioned the presence of *Streptococcus* sp., *Staphylococcus* sp., *Brucella* sp., *Pasteurella* sp., *Bacteroides* sp., and *Fusobacterium* sp. in cerebral abscesses of cats and dogs. However, Smith et al. (2007) found bacteria characterized as actinomycetes, with suggestions of *Novocardia* sp. or *Actinomyces* sp., in a cerebral abscess of dog caused by hematogenous infection. In human patients immunely weakened by HIV infection, subjected to organ transplantation, chemotherapy, or steroid use, the most commonly identified agents in cases of abscess are *Staphylococcus* spp., *Streptococcus* spp., *Pseudomonas* spp., and *Toxoplasma* spp. (Young & Mcgwire 2005). In this report, microbiological examination detected *Staphylococcus* sp. as the lesion-causing agent.

Immunosuppression and comorbidities, initial neurological state, and intraventricular rupture are important factors that influence patient outcome (Erdogan & Cansever 2008). The fact that the animal evaluated in this study was very young and recently vaccinated may have led to immunosuppression, which may have contributed to abscess progression.

CONCLUSION

The data revealed that cerebral abscess in dogs, though rare in this species, shows a number of differential diagnoses. This way, clinical diagnosis is cumbersome, and mostly only established during necroscopic examination.

Mortality reduction in dogs with cerebral abscess can be achieved with early diagnosis, surgical intervention, and efficacious antibiotic treatment. Moreover, late diagnosis hinders patient treatment, and the survival possibilities become reduced with

disease progress. However, early isolation of the etiological agents is not always feasible. The choice of adequate treatment varies mainly according to factors such as abscess number, size, localization, and stage.

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