



A retrospective study of quadrigeminal arachnoid cysts diagnosed by Magnetic Resonance Imaging and Computed Tomography in 26 dogs¹

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ABSTRACT.- Alves L.S., Rodriguez D., Machado V.M.V., Mamprim M.J., Vulcano L.C. & Amorim R.M. 2018. **A retrospective study of quadrigeminal arachnoid cysts diagnosed by Magnetic Resonance Imaging and Computed Tomography in 26 dogs.** *Pesquisa Veterinária Brasileira* 38(2):300-308. Faculdade de Medicina Veterinária e Zootecnia, Universidade Estadual Paulista, Distrito Rubião Junior, s/n, Botucatu, SP 18618-970, Brazil. E-mail: lidianealves.vet@gmail.com

Arachnoid cysts are accumulations of cerebrospinal fluid that occur within the arachnoid membrane due to the splitting or duplication of this structure. The aim of this retrospective study is to report the occurrence of this condition in 26 dogs, as well as to describe the findings of computed tomography imaging and magnetic resonance image scans and to correlate these with the clinical signs. A prevalence of the condition was observed in males, predominantly in the small breeds Miniature Pinscher and Shih Tzu, with age ranging from 2 months to 9 years. The mainly clinical signs observed in these dogs were seizures and cerebellar ataxia. The study concluded that in 17 dogs the quadrigeminal arachnoid cyst was regarded as an incidental finding, in 6 dogs this condition was regarded as the determinant cause of the clinical signs, and in 3 dogs some clinical signs were associated with arachnoid cyst, but they also presented clinical signs involving others sites of intracranial lesion.

INDEX TERMS: Quadrigeminal arachnoid cysts, Magnetic Resonance Imaging, Computed Tomography, dogs, seizures, cerebellar ataxia, hydrocephalus, brain diseases, pathology.

RESUMO.- [Estudo retrospectivo de cistos aracnoides da cisterna quadrigeminal diagnosticados pela ressonância magnética e tomografia computadorizada em 26 cães.]

Cistos aracnoide são acúmulos de líquido cefalorraquidiano que ocorre dentro da membrana aracnoide devido à divisão ou duplicação desta estrutura. O objetivo desse estudo retrospectivo é relatar a ocorrência dessa condição em 26 cães, assim como descrever os aspectos de imagem pela tomografia computadorizada e ressonância magnética e correlacionar com os sinais clínicos. No estudo observamos a prevalência em machos, em raças de pequeno porte, com predomínio de Pinscher miniatura e Shih Tzu, com idade variando de dois meses a nove anos. Os principais sinais neurológicos

observados nestes animais foram crises epiléticas e ataxia cerebelar. Foi possível concluir que em 17 cães o cisto aracnoide quadrigeminal foi considerado um achado incidental, em seis cães essa condição foi considerada a causa determinante dos sinais clínicos, e em três cães alguns sinais estavam associados com o cisto aracnoide, porém esses animais apresentaram sinais clínicos relacionados a outros sítios de lesão intracraniana.

TERMOS DE INDEXAÇÃO: Cistos aracnoides, cisterna quadrigeminal, ressonância magnética, tomografia computadorizada, caninos, convulsões, ataxia cerebelar, hidrocefalia, encefalopatias, patologia.

INTRODUCTION

Arachnoid cysts are characterized by focal division of the leptomeninge which forms a bag or diverticulum within an arachnoid membrane (Rengachary & Watanabe 1981, Galassi et al. 1985, Osborn 1994). They can be classified as primary, which are congenital (Dei-Anang & Voth 1989, Matiasek et al. 2007), that have active secretion of cerebrospinal fluid (CSF) through either the ectopic choroid plexus

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(Dei-Anang & Voth 1989, Osborn 1994), or of the ependymal cells (Osborn 1994); or as secondary, resulting from brain trauma (Rengachary & Watanabe 1981), intracranial infection and hemorrhage of the subarachnoid space (Di Rocco 1996). They may also be defined as communicating or non-communicating depending on the relationship with the subarachnoid space (Rengachary & Watanabe 1981, Osborn 1994).

A diagnosis of the condition can be obtained by computed tomography (CT) (Osborn 1994, Dewey et al. 2007), magnetic resonance imaging (MRI) (Osborn 1994, Duque et al. 2005, Dewey et al. 2007, Matiassek et al. 2007) and ultrasonography (Saito et al. 2001).

On MR images, the quadrigeminal arachnoid cysts present themselves as well-demarcated, extra-axial, cystic-appearing structures located between the occipital lobe of the cerebrum and rostral lobe of the cerebellum (Dewey et al. 2009) that displace or deform the adjacent structures (Osborn 1994) and are isointense to CSF in all sequences (Osborn 1994, Matiassek et al. 2007). These cysts do not enhance with intravenous gadolinium administration (Galassi et al. 1985, Osborn 1994, Vernau et al. 2002, Matiassek et al. 2007), except if they have associated meningoencephalitis (Vernau et al. 2002, Kitagawa et al. 2003, Duque et al. 2005). Occasionally, hemorrhage or high protein content may impair the appearance of an arachnoid cyst on MRI. On CT images the cysts also have features of a sharply defined, extra-axial, non-calcified and non-enhancing lesion. Unless that have present hemorrhage, most of the arachnoid cysts have similar attenuation to CSF (Osborn 1994).

Matiassek et al. (2007) observed three different phenotypes of quadrigeminal cysts in imaging examination: in association with the 3rd ventricle, in association with the 4th ventricle, and a loculated phenotype with relation to the 3rd and 4th ventricle.

The treatment can be performed with medical management with anticonvulsant drugs and corticosteroids (Vernau et al. 1997, Duque et al. 2005) or with surgical treatment such as fenestration (Vernau et al. 1997) and cystoperitoneal shunting (Saito et al. 2001, Dewey et al. 2007).

There are controversies about the clinical significance of arachnoid cyst in dogs and humans and they are considered in many cases to be incidental findings of imaging or *post-mortem* findings (Vernau et al. 1997, Saito et al. 2001, Kitagawa et al. 2003, Duque et al. 2005, Matiassek et al. 2007).

The aim of this retrospective study is to report the occurrence of the quadrigeminal arachnoid cysts in 26 dogs, as well to describe the imaging findings by computed tomography and magnetic resonance image scans and to correlate these with the clinical signs.

MATERIALS AND METHODS

Records of head MRI and CT scans of dogs that were admitted to the Veterinary Neurology Service between February 2007 and July 2015 were reviewed by a radiologist resident and a professor. MRIs were performed with a 0.25 Tesla scanner (Vet-MR Grande, Esaote, Italy), including T1, T2, T2* and fluid-attenuation inversion recovery (FLAIR) sequences in the sagittal, transverse and dorsal planes. CTs were performed with helical equipment SCT-7800TC (Shimadzu; Kyoto, Japan) in the transverse plane, with most of the scans performed in sagittal and dorsal multiplanar reconstruction (MPR).

Images were evaluated using a medical digital imaging system (PACS, Synapse, Fuji Medical System; Tokyo, Japan). The degree of the cerebral and cerebellar compression was calculated from the mid-sagittal planes in 16/17 MRI and 5/9 CT scans as proposed in a previous study by Matiassek et al. (2007).

The diagnosis of quadrigeminal arachnoid cyst was based on the following inclusion criteria: location of cystic-appearing structure between the occipital lobe and cerebellum, fluid-content intensity/attenuation similar to cerebrospinal fluid (CSF), and absence of enhancement after the intravenous contrast-medium administration (0.02ml/kg of gadoversetamide [Optimark®, Mallinckrodt Inc., Raleigh, NC, USA] to MRI; 0.2ml/kg of iopamidol [Iopamiron®300, Patheon Italia S.p.A., Ferentino, Frosinore, Italy] to CT).

Additional information was obtained from the medical records as well as signalment, presenting complaint, neurologic evaluation and blood profile (CBC and serum biochemistry). Cerebrospinal fluid (CSF) analysis was performed in seven dogs. The CSF protein was considered increased if greater than 25mg/dL (cerebellomedullary cistern). It was considered as mild (<50 mg/dL), moderate (<50-200 mg/dL) or severe (>200mg/dL) (Bohn et al., 2006). The total count of nucleated cells (CTCN) was characterized as pleocytosis when >5 nucleated cells per μL (Terlizzi & Platt, 2006). It was classified as primary, if the cell type comprised >85%, and mixed if none of the cell types comprised at least 85% of the nucleated cells. It was considered mild (<25 cells/ μL), moderate (26-100 cells/ μL), or marked (>100 cells/ μL) and was further defined by the predominant cell type in the sample the neutrophilic, eosinophilic, mononuclear or mixed (Freeman & Raskin, 2001).

RESULTS

Altogether, 650 records of canine and feline imaging brain scans were reviewed - 148 MRI images and 502 CT images. Twenty-six dogs were diagnosed with quadrigeminal arachnoid cyst, all of them small breeds weighing less than 10 kg (mean 4.5; SD = 2.83). Breed population consisted of 7 Miniature Pinschers, 7 Shih Tzus, 4 Lhasa Apsos, 2 Poodles, 2 mixed breed and 1 each of Yorkshire Terrier, Miniature Schnauzer, Boston Terrier, and Brazilian Terrier. Fifteen dogs were male and 11 dogs were female. The age at diagnosis ranged from 2 months to 9 years, with a mean of 3.1 years (SD = 2.43).

CBC and serum biochemistry did not reveal significant changes in any of the patients. CSF analysis of two dogs presented changes consistent with inflammatory brain disease showing lymphocytic pleocytosis (case 15) and neutrophilic pleocytosis (case 6). Five dogs presented with normal CSF analysis parameters (cases 10, 12, 14, 16). One CSF analysis could not be completed due to insufficient sample (case 1) that showed increased protein concentration with 81mg/dL (reference range, 0-30mg/dL) (Table 1).

The neurologic exams recorded revealed several clinical signs including generalized tonic-clonic seizures (13), focal seizures (1), cerebellar ataxia (8), hypermetria (3), wide-base stance (2), intention tremor (3), circling (7), compulsive walking (5), head pressing (2), deficit of menace response (8), behavioral changes (7), mental depression (2), conscious proprioceptive deficit (7), proprioceptive ataxia (5), paresis (3), head tilting (4),

Table 1. Clinical summary of 26 dogs with quadrigeminal arachnoid cysts

Cão	Signalment	Presenting complaint	Neurolocalization	CSF analysis
1	3 year old, F, Shih Tzu, 8.6kg	Tonic-clonic seizures	Bilateral cerebral cortex	Moderate increase of protein concentration; mild pleocytosis. Cytology did not perform due insufficient sample
2	6 year old, M, Shih Tzu, 5.6kg	Tonic-clonic seizures, central blindness	Bilateral occipital cortex	-
3	4 year old, M, Shih Tzu, 5.7kg	Behavioral changes	Brainstem, temporal cortex	-
4	2 year old, M, Shih Tzu, 1.5kg	Neck pain, parataxia, paraparesis, proprioceptive deficit of hindlimbs	Spinal cord C1-C5	-
5	1 year old, F, Pinscher, 1.8kg	Proprioceptive ataxia, head tilting, gait disturbance with rigid gait	Cerebellum, spinal cord C1-C5	-
6	6 year old, F, Poodle, 2.3 kg	Cerebellar ataxia, hypermetria, wide stance	Cerebellum, spinal cord C1-C5	Moderate increased of protein concentration; moderate mixed pleocytosis
7	2 year old, F, mixed breed, 3.4kg	Gait disturbance with compulsive gait, pleurothotonus to left, hemiparesis to right, central blindness to left, tonic-clonic seizures, circling to left, proprioceptive ataxia	Left cerebral cortex (frontal, parietal, occipital)	-
8	3 year old, F, Boston Terrier, 9.6kg	Tonic-clonic seizures	Cerebral cortex	-
9	7 month old, M, mixed breed, 8.8kg	Circling, central blindness, compulsive walking, ventrolateral strabismus	Occipital and frontal cortex	-
10	1.6 year old, M, Schnawzer, 5.3kg	Tonic-clonic seizures, focal seizures	Cerebral cortex (frontal, temporal, occipital)	No increase of protein concentration; no pleocytosis
11	1.6 year old, M, Lhasa Apso, 7.2kg	Tonic-clonic seizures	Cerebral cortex (frontal, temporal, occipital)	-
12	5.3 year old, F, Lhasa Apso, 5.9kg	Hypermetria, intention tremor, cerebellar ataxia, proprioceptive deficit, wide base, tonic-clonic seizures	Pontine-cerebellar	No increase of protein concentration; no pleocytosis
13	5 month old, F, Shih Tzu, 1.8kg	Head pressing, circling, behavioral changes, central blindness	Cerebelar cortex	-
14	3 year old, F, Pinscher, 2.6kg	Tonic-clonic seizures	Cerebral cortex (frontal, temporal occipital)	Mild increase of protein concentration; no pleocytosis
15	1 year old, M, Pinscher, 4.2kg	Behavioral changes, circling to right, compulsive walking, tonic-clonic seizures	Cerebral cortex (frontal, temporal occipital)	Moderate increase of protein concentration; moderate mixed pleocytosis
16	2 year old, F, Lhasa Apso, 7.5kg	Tonic-clonic seizures	Cerebral cortex (frontal, temporal occipital)	No increase of protein concentration; marked mixed pleocytosis
17	3 year old, M, Shi Tzu, 7 kg	Behavioral changes, tonic-clonic seizures, paraparesis, parataxia, parestesis, central blindness	Cerebral cortex (fontal, parietal, occipital)	-
18	2 month old, M, Shih Tzu, 1.2kg	Tetrataxia, proprioceptive deficit, compulsive walking, head tilting, central blindness	Fronto-parietal cortex, central vestibular, system optic nerve or optic chiasm	-
19	2 year old, M, Pinscher, 1.4kg	Tetrataxia, proprioceptive deficit, behavioral changes, circling, central blindness, depression, head tilting, focal seizures	Cerebral cortex, central vestibular system	-
20	7 year old, M, Yorkshire, 5.4kg	Intention tremor, proprioceptive, behavioral changes, hypermetria	Cerebral cortex (frontal, parietal), cerebellum	-
21	7.6 year old, M, Pinscher, 2.2kg	Head tilting	Peripheral vestibular system	-
22	8 month old, F, Brazilian Terrier, 1.4kg	Cerebellar ataxia, tetraparesis, depression	Cerebral cortex (frontal, parietal), cerebellum	-
23	3 year old, M, Lhasa Apso, 5kg	Tonic-clonic seizures, intention tremor, cerebellar ataxia	Cerebral cortex (frontal, temporal, occipital), cerebellum	-
24	8 month old, M, Pinscher, 2.3kg	Tonic-clonic seizures, head pressing, circling	Cerebral cortex	-
25	9 year old, F, Poodle, 9kg	Cerebellar ataxia	Cerebellum	-
26	2 month old, M, Shih Tzu, 0.3kg	Circling, gait disturbance with compulsive gait, peripheral blindness, behavioral changes, proprioceptive deficit, cerebellar ataxia mmm	Cerebral cortex (frontal, temporal, parietal), cerebellum	-

positional strabismus (1), pleurothotonus (1), parestesis (1), and neck pain (1) (Table 1).

MRI showed a well-demarcated, extra-axial, cystic-appearing structure in various sizes located between the ventrocaudal border of the occipital lobes and the dorso-rostral border of the cerebellum, hypointense on T1, hyperintense on T2, and suppression on FLAIR images. The signal intensity of the cyst content was similar to CSF and non-contrast enhancing with gadolinium administration. These structures were observed on transverse planes in a triangular-like shape between the occipital lobes and the cerebellum (Fig.1). On CT images the structure showed fluid attenuation similar to CSF and no enhancement after contrast-medium administration (Fig.2).

Other brain abnormalities were observed such as hydrocephaly, of which variations were observed ranging from discrete to severe in 20 dogs, with three obstructive hydrocephalus (cases 1, 5, 9) and two hydrocephalus *ex vacuo* (cases 6, 7). Other changes were consistent with inflammatory brain disease in five dogs, including meningoencephalitis (cases 2, 6), cortical (cases 8, 15) and interstitial edema (case 7), anomalous/congenital disease in 15 dogs, including hydrocephalus (cases 4, 5, 9-14, 17-22, 24, 26),

syringohydromyelia (cases 5, 9), agenesis of the corpus callosum and lissencephaly (case 17), atlanto-occipital malformation and branchial cysts (case 4), degenerative disease, including cerebellar atrophy (case 25), and neoplasia in a dog with a tumor on lateral cerebellar pontine angle involving the fourth ventricle (case 1) (Table 2). Only three CT and MRI scans did not present abnormalities beyond the quadrigeminal cyst (cases 3, 16, 23).

Three phenotypes of the quadrigeminal arachnoid cyst were easily distinguished on MRI and CT images of this study, including on CT transverse planes for which it was not possible to perform MPR mid-sagittal images (Fig.3). A predominance of phenotype was noted in association with the fourth ventricle (11 images) followed by association with the third ventricle (9 images) and loculated with the third and fourth ventricles (6 images). Four loculated phenotypes presented with equal relation to the third and fourth ventricle, while the other two presented with phenotype loculated with a higher relation to the fourth ventricle than to the third ventricle.

The degree of cerebral and cerebellar compression was calculated from the mid-sagittal planes in 16/17 MRI scans (Fig.4 A and B), which obtained ranging from 0-24.2% (mean, 6.5%; SD = 9.2)

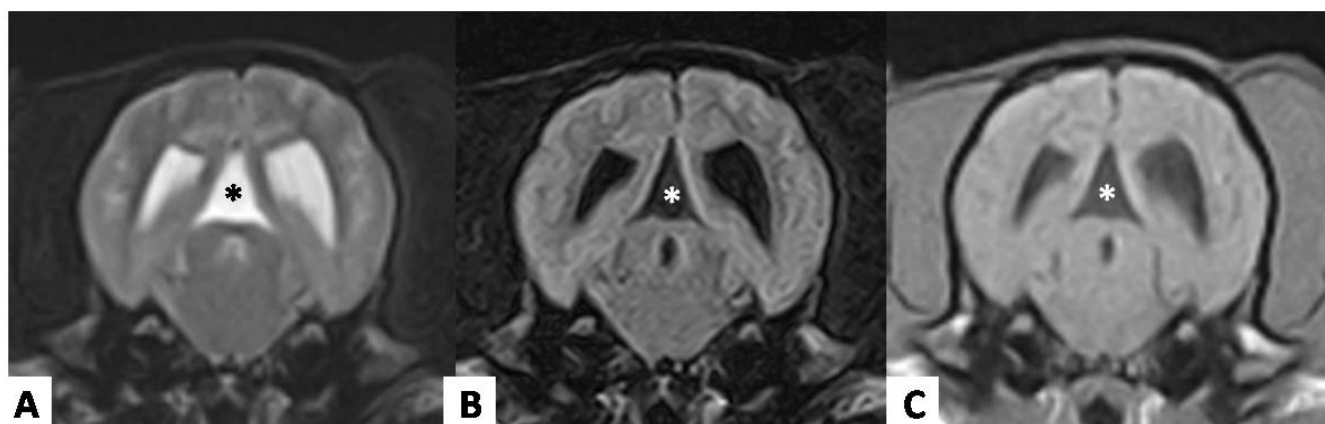


Fig.1. (A) Transverse MRI of a 3-year-old female Boston Terrier with 9.6 kg presenting seizures weekly, agitation and behavior abnormal. Note the triangular-like shape of the structure (*) between occipital lobes showing the same signal intensity than CSF on T2-weighted, and (B) FLAIR with no contrast enhancement showed on T1-weighted (C).

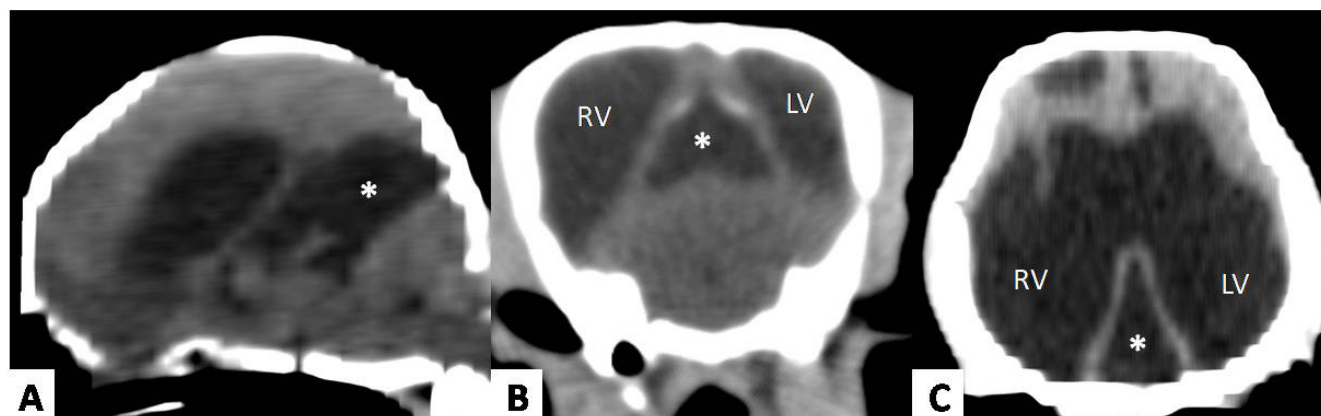
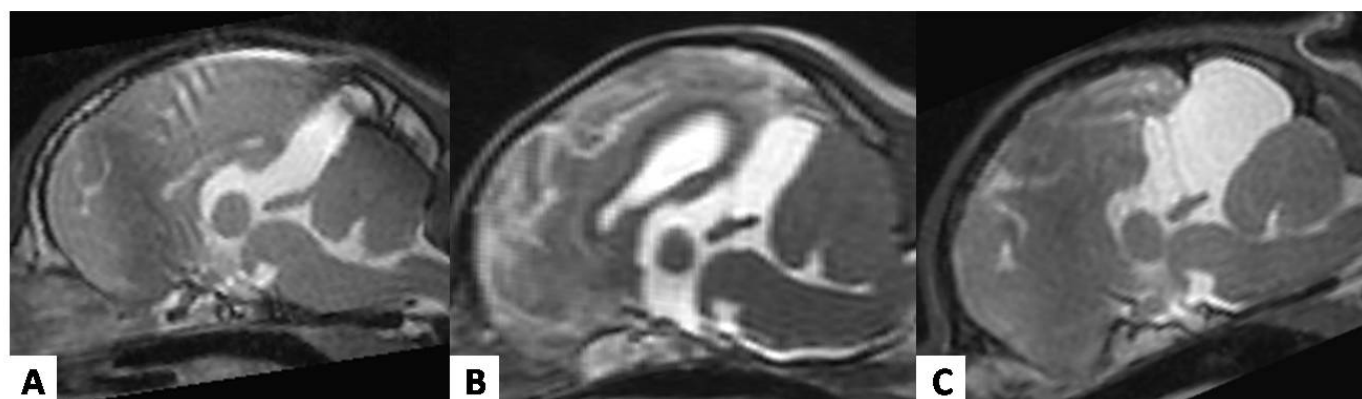


Fig.2. (A) CT images of a 2-year-old male Pinscher Miniature with 1.4 kg presenting ataxia and circling to the right. Note the cyst-like structure (*) of fluid attenuation between occipital lobes and cerebellum extending from the third ventricle on the sagittal plane. (B) This cyst-like structure (*) can be seen between occipital lobes in a triangular-like shape on transverse, and (C) dorsal planes. This dog had severe hydrocephalus. LV = left ventricle; RV = right ventricle.

Table 2. MRI and CT findings, percentual of forebrain and cerebellar compression and phenotype of quadrigeminal arachnoid cyst in 26 dogs

Cão	Diagnostic imaging modality	Other findings	Forebrain compression*	Cerebellar compression*	Phenotype
			(%)	(%)	
1	MRI	Neoplasia (choroid plexus tumor of the lateral cerebellar pontine angle with moderate obstructive hydrocephalus)	18.9	0	3rd ventricle
2	MRI	Inflammatory/infectious (meningoencephalitis)	24.2	45.2	3rd and 4th ventricle
3	MRI	No abnormalities	20.2	18.6	4th ventricle
4	MRI	Congenital/anomalous (hydrocephalus, atlanto-occipital malformation, branchial cysts)	0	32.3	4th ventricle
5	MRI	Congenital/anomalous (moderate obstructive hydrocephalus, syringohydromyelia)	0	20.2	3rd and 4th ventricle
6	MRI	Inflammatory/infectious (hydrocephalus <i>ex vacuo</i>)	0	15.7	4th ventricle
7	MRI	Inflammatory/infectious (interstitial edema focal in left hemisphere with hydrocephalus <i>ex vacuo</i> to the left)	0	27.6	4th ventricle
8	MRI	Inflammatory (cortical edema with moderate ventriculomegaly)	0	20.6	3rd ventricle
9	MRI	Congenital/anomalous (severe obstructive hydrocephalus with syringohydromyelia, diverticle of 4th ventricle, compression of pons)	-	-	3rd ventricle
10	MRI	Anomalous (moderate ventriculomegaly)	12.8	0	3rd ventricle
11	MRI	Congenital/anomalous (asymmetrical hydrocephalus to the left)	0	53.3	4th ventricle
12	MRI	Anomalous (moderate ventriculomegaly)	8.5	0	3rd ventricle
13	MRI	Congenital/anomalous (severe hydrocephalus)	0	40.9	4th ventricle
14	MRI	Anomalous (moderate ventriculomegaly)	0	12.7	4th ventricle
15	MRI	Inflammatory (cortical edema and moderate hydrocephalus)	0	0	4th ventricle
16	MRI	No abnormalities	0	0	3rd and 4th ventricle
17	MRI	Congenital/anomalous (agenesis of corpus callosum, partial lissencephaly)	19.0	0	3rd ventricle
18	CT	Congenital/anomalous (severe hydrocephaly)	0	55.7	4th ventricle
19	CT	Congenital/anomalous (severe hydrocephalus)	15.8	42.2	3rd and 4th ventricle
20	CT	Anomalous (moderate asymmetrical ventriculomegaly)	14.2	0	3rd ventricle
21	CT	Anomalous (moderate asymmetrical ventriculomegaly)	16.9	36	4th ventricle
22	CT	Congenital/anomalous (severe hydrocephalus)	0	0	3rd and 4th ventricle
23	CT	No abnormalities	-	-	4th ventricle
24	CT	Anomalous (hydrocephalus)	-	-	3rd ventricle
25	CT	Degenerative (cerebellar atrophy)	-	-	3rd ventricle
26	CT	Congenital/anomalous (severe hydrocephalus)	-	-	3rd and 4th ventricle

**Fig.3.** Mid-sagittal T2-weighted MRI showing the 3 different phenotypes of quadrigeminal arachnoid cyst: (A) in association with the 3rd ventricle, (B) in association with the 4th ventricle, and (C) loculated phenotype to the 3rd and 4th ventricle.

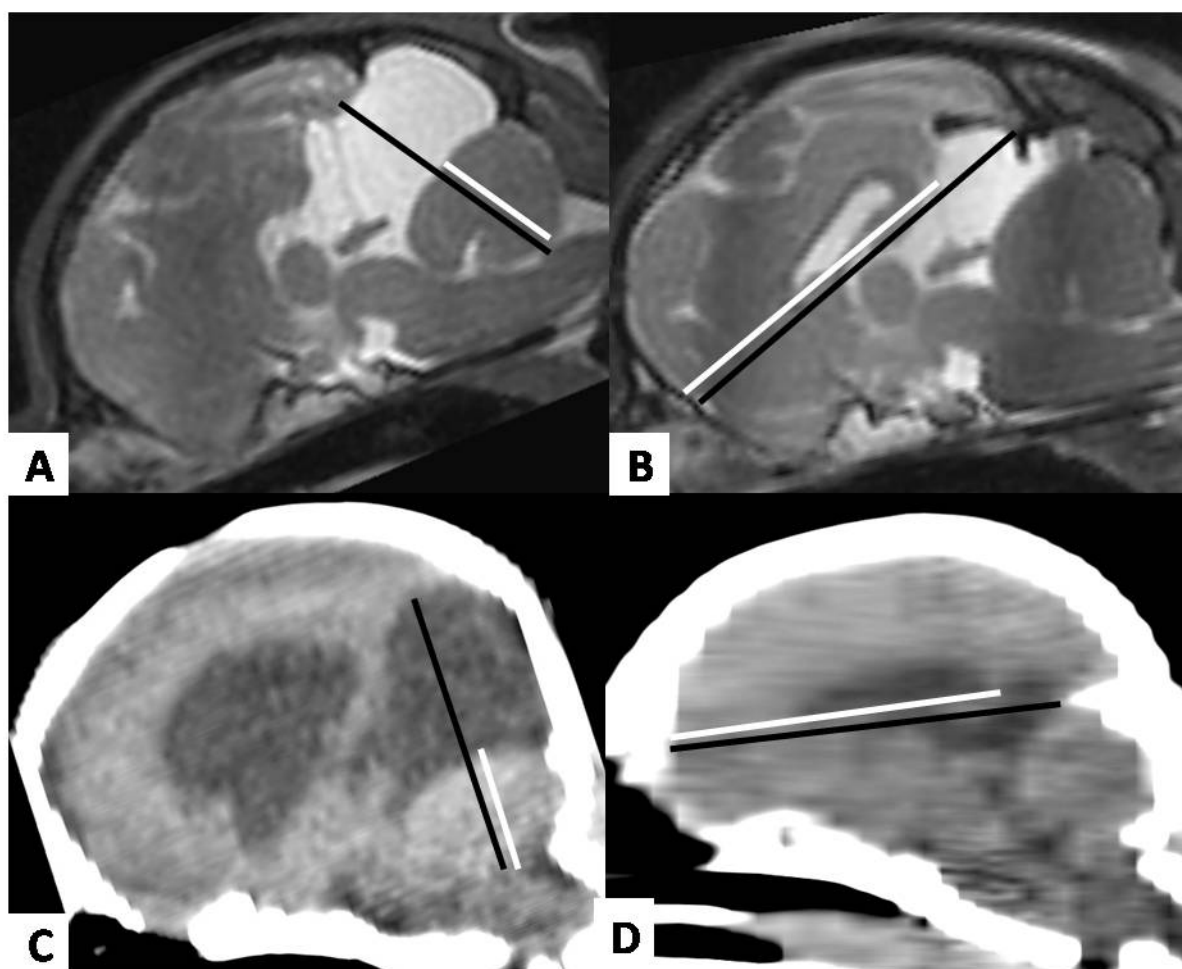


Fig.4. (A,B) Measurement of the compression degree of the cerebral and cerebellar parenchyma by mid-sagittal T2-weighted MRI, and (C,D) mid-sagittal CT images. The black line of (A) and (C) represents the expected diameter of the cerebellum and the measured length is set at 100%; the white line represents the actual diameter of the cerebellum. The black line of (B) and (D) is drawn for the expected longitudinal dimension of the forebrain and the measured length is set at 100%; the white line represents the actual longitudinal dimension of the forebrain.

of occipital lobe compression and from 0-53.3% (mean, 17.9%; SD = 17.8) of cerebellum compression. The same calculation was made in 5/9 CT scans (Fig.4C,D), as in another four CT scans it was not possible to perform the MPR in sagittal planes. The degree of compression of the occipital lobes ranged from 0-16.9% (mean, 9.4%; SD = 8.6) and the compression of the cerebellum ranged from 0-55.7% (mean, 26.8%; SD = 25.5%). Altogether, 21 CT and MRI scans were measured obtaining a mean of 7.2% (SD = 8.9) of occipital lobes compression and a mean of 20.0% (SD = 19.6) of cerebellum compression.

DISCUSSION

There are controversies regarding the quadrigeminal arachnoid cyst in literature about its clinical significance. There have been around 57 reported cases of intracranial arachnoid cyst in dogs (Vernau et al. 1997, Koie et al. 2000, Saito et al. 2001, Vernau et al. 2002, Kitagawa et al. 2003, Duque et al. 2005, Dewey et al. 2007, Matiassek et al. 2007, Reed et al. 2009,

Bernardino et al. 2015) and more than half of these cases were deemed to be an incidental finding.

In humans, the incidence of intracranial arachnoid cysts is 1% of all non-traumatic mass lesions (Robertson et al. 1989), with 50-60% located in the middle fossa (Rengachary & Watanabe 1981, Robertson et al. 1989, Yamakawa et al. 1991, Wang et al. 1998), mainly unilateral (Wang et al. 1998), and 10% in the posterior fossa (Robertson et al. 1989).

The prevalence of this condition is low in dogs, representing about 0.7% of intracranial lesions (Matiassek et al. 2007). Nevertheless, in the present study this percentage presented higher with 4% of total brain examinations evaluated. All arachnoid cysts presented on quadrigeminal cistern, as well as in most of the case reports in the literature (Vernau et al. 1997, 2002, Saito et al. 2001, Kitagawa et al. 2003, Duque et al. 2005, Dewey et al. 2007, Matiassek et al. 2007). Only two cases report the arachnoid cyst occurring at the cerebellomedullary angle (Vernau et al. 1997) and in the pineal region (Koie et al. 2000). There are only three reports of Persian cats presenting intracranial arachnoid cyst located in caudal fossa at level

of the quadrigeminal cistern (Lowrie et al. 2009, Reed et al. 2009) and cerebellar pontine (Milner et al. 1996), and a report of a European cat with supratentorial arachnoid cyst located mainly beneath the piriform lobe (Taroni et al. 2015). However, in our study, neither record of feline imaging brain scans presented this condition. There also exists a report case of a chimpanzee with intracranial arachnoid cyst in the occipital convexity (Miyabe-Nishiwaki et al. 2014).

Arachnoid cyst is present in small breed and brachycephalic dogs (Matiasek et al. 2007), as seen in this study that the affected dogs weighed less than 10 kg. Among the dog breeds previously reported were Shih Tzu, Yorkshire Terrier, Pug, Lhasa Apso, Chihuahua, Bulldog, Staffordshire Bull Terrier, Pekingese, Jack Russel, Cavalier King Charles Spaniel, West Highland White Terrier, Cairn Terrier, Maltese, Pomeranian, Bichon Frisé, Beagle and Miniature Schnauzer (Vernau et al. 1997, 2002, Kitagawa et al. 2003, Duque et al. 2005, Dewey et al. 2007, Matiasek et al. 2007). However, the breeds most prevalent in this study were Miniature Pinscher and Shih Tzu (both representing 26.9%). Dogs with arachnoid cyst ranged in age from 2 months to 10 years and a greater incidence was found in males (Saito et al. 2001, Kitagawa et al. 2003, Duque et al. 2005, Matiasek et al. 2007, Reed et al. 2009). Similarly, more than half of the population of this retrospective study was male (57.7%), with an age range from 2 months to 9 years.

In humans, the posterior fossa clinical signs consist of headache, seizures, ataxia, vomiting, cerebellar disorders, mild cognitive impairment, and focal neurological signs (Wang et al. 1998, Hanieh et al. 1988, Robertson et al. 1989, Cincu et al. 2007). Commonly, the clinical signs were associated with obstructive hydrocephalus (Hanieh et al. 1988, Cincu et al. 2007). Macrocephaly and psychomotor retardation have been reported in young patients (Galassi et al. 1985, Wang et al. 1998). Dementia, ataxia, hemiparesis and gait disturbance have been reported as signs among the elderly (Yamakawa et al. 1991).

Clinical signs more often described in animals were seizures, head tilt, ataxia, circling, paresis, dementia, and deficit of menace response (Vernau et al. 1997, 2002, Kitagawa et al. 2003, Duque et al. 2005, Saito et al. 2001, Reed et al. 2009). Interestingly, all these clinical signs were observed in the animals of this study with a predominance of seizures (54%), followed by cerebellar ataxia (31%) and deficit of menace response (31%).

Arachnoid cysts consist of a thin-walled, well-demarcated, fluid-filled cavity that has a triangular cross-section. On image evaluations the fluid is isodense to CSF in CT, the cysts have a very similar signal intensity to CSF in all sequences on MRI and the capsule does not frequently take up contrast (Lang & Seiler 2010). There were no other features on CT or MRI exams beyond those described in the literature.

Measurement of the degree of parenchymal compression of the occipital lobes and the cerebellum was proposed in a previous study using MRI on mid-sagittal T2- and T1-weighted images (Matiasek et al. 2007). Likewise, the measurement was performed in both MRI and CT images. Although in only 1/17 MRI scan it was not possible to perform this measurement due the extensive dilatation of the ventricular system loosing the anatomical parameters (case 9) and also it was not possible to perform the measurement in 4/9 CT scans due to technical problems. Therefore, 21 MRI and CT

scans had the degree of compression of the occipital lobe and the cerebellum measured.

The occipital lobe is responsible for processing visual information, so when there is damage in this region the main clinical sign observed is visual impairment or central blindness, represented by a deficit of menace response, with intact pupillary reflexes (Lorenz et al. 2010). Seizures can occur when there is compression of the cerebral cortex including in the occipital lobe (Jaggy & Spiess 2010).

In this study, these were the most common clinical signs with a total of 18 dogs, being 10 dogs presenting seizures, 4 presenting deficit of menace response, and 4 presenting both clinical signals (Table 1). The degree of occipital lobe compression in the dogs that presented clinical signs ranged from 0 to 24.2% (mean, 6.4%; SD = 9.2). Nevertheless, for 8 of these 18 dogs (cases 7, 8, 11, 13, 14, 15, 16, 18) the CT and MRI scans revealed 0% of occipital lobe compression, consisting of four dogs (cases 11, 13, 14, 18) with hydrocephalus, three (cases 7, 8, 15) with cortical inflammatory disease and only one (case 16) with no abnormality on MRI beyond the quadrigeminal cyst.

Six of the eighteen dogs that presented seizures and/or deficit of menace response had others brain abnormalities on CT and MRI, associated with some degree of the occipital lobe compression caused by quadrigeminal arachnoid cysts. Three dogs (cases 10, 12, 19) presented hydrocephalus, one dog (case 17) presented lissencephaly and agenesis of the callosum corpus, one (case 2) presented meningoencephalitis, and one (case 1) presented tumor on cerebellar pontine angle.

In four dogs that presented those clinical signs, the degree of occipital lobe compression could not be measured due to technical problems, with three dogs (cases 9, 24, 26) presenting hydrocephalus and one dog (case 23) having no abnormalities on CT images but with a cyst shown with mass-effect.

The cerebellum functions as a coordinator of movements that originate in the upper motor neuron system and serves to help maintain equilibrium, posture, body support against gravity, and eye movements (Lorenz et al. 2010). In general, cerebellar disease causes severe ataxia, dysmetria (hypermetria), intention tremors, wide-legged stance (Jaggy & Spiess 2010), and sometimes vestibular signs (paradoxical vestibulopathy) (Lorenz et al. 2010). The same clinical signs were present in 9 dogs (cases 6, 12, 18-20, 22, 23, 25, 26) affected, of whom 6 dogs had a degree of cerebellum compression ranging from 0 to 55.7% (mean, 18.9%; SD = 24.4). Most of these cases presented CT and MRI scans with hydrocephalus (cases 12, 18-20, 22, 26) and one case presented cerebellar atrophy (case 25). Only one (case 23) presented no abnormalities beyond the quadrigeminal arachnoid cyst.

Among some clinical signs that were not correlated to local compression of the occipital lobe and the cerebellum were compulsive walking (frontal lobe, internal capsule, basal nuclei), paresis (frontal lobe, internal capsule, basal nuclei, brainstem, spinal cord, peripheral nerves), behavioral changes, as head pressing (limbic system, frontal or temporal cortex), head tilt and positional strabismus (vestibular system), mental depression (cerebrum, brainstem), circling and pleurothotonus (frontoparietal lobe, rostral thalamic) (Lorenz et al. 2010), proprioceptive ataxia (parietal lobe, thalamus, brainstem, spinal cord and peripheral nerves) (Jaggy & Spiess 2010).

Meanwhile, seizures occur when there is compression of the cerebrum. Nevertheless, some of these signs may be associated with hydrocephalus, as behavior changes, loss of vision and gait disturbance (Lorenz et al, 2010). In our study, 20 dogs that were diagnosed with hydrocephalus ranging from discrete to severe, 15 dogs presented the clinical signs described above, but two dogs (cases 7, 15) had clinical signs consistent with multifocal disease observed on MRI as inflammatory brain disease. One dog (case 3) presented only clinical signs related with behavioral disturbance and its MRI showed no abnormalities beyond the quadrigeminal arachnoid cyst.

Even on CT scans that could not perform MPR mid-sagittal it was possible to identify the phenotype due its extension from third and/or mesencephalic aqueduct dorso-rostrally to the fourth ventricle. In 14 cases it was observed that the phenotype of the arachnoid cyst had correlation with the compression site. Five cases were observed that showed the phenotype of the arachnoid cyst was correlated with forebrain compression; 7 cases with phenotype in association to the fourth ventricle were correlated with cerebellar compression; and 2 cases with phenotype in association to the third and fourth were correlated with both forebrain and cerebellar compression. However, the clinical manifestation of these dogs indicated lesions in others sites of the brain not related with compression caused by quadrigeminal arachnoid cysts.

Clinical treatment based in seizure management with anticonvulsant and corticosteroids of those dogs improved the clinical signs, but the follow-up was in a short period or was not followed-up in some patients. Cystoperitoneal shunting may be an effective treatment method for dogs clinically affected with intracranial arachnoid cyst (Dewey et al. 2007). A dog of this retrospective study was submitted to ventricle-peritoneal shunting which improved the clinical signs, such as seizures.

Based on the clinical signs, CSF analyses and other abnormalities on CT and/or MRI scans, 17 dogs (cases 1, 4-7, 9, 13, 15-25) had quadrigeminal arachnoid cyst regarded as an incidental finding. In 6 dogs (cases 2, 10-12, 14, 23) the findings were related with quadrigeminal arachnoid cyst. However, in the 3 remain dogs (cases 6, 8, 26), the findings were not associated exclusively with quadrigeminal arachnoid cyst, suggesting others sites of brain lesion.

According to Matiassek et al. (2007), occipital lobe compression greater than 14% is uniformly associated with clinical signs. However in our study, in the six dogs that presented findings related to quadrigeminal arachnoid cyst, the mean of degree of compression of the occipital lobe was 9.1% (SD = 10). The cases in which the quadrigeminal arachnoid cyst was considered to be incidental had a considerable degree of cerebellum compression ranging from 15.7% to 55.7%.

In this retrospective study, it was concluded that the quadrigeminal arachnoid cyst is an incidental finding mainly when clinical signs presented by the patient do not associate with compression of the occipital lobe, cerebellum or with an increase of intracranial pressure. When there are clinical signs related to compression caused by the arachnoid cysts, ruling out other intracranial diseases, they can be regarded as the determinant cause, making therapeutic or surgical treatment necessary.

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