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CASE REPORT

Companion or pet animals

Choroid plexus carcinoma in a dog—Case report

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Abstract

Obtaining a conclusive definitive diagnosis for an intra-ventricular tumour in a dog can be challenging. In doing so, all differential diagnoses should be considered. Differentiation between choroid plexus tumours (CPTs) (a choroid plexus papilloma, atypical choroid plexus papilloma or choroid plexus carcinoma) and gliomas, including ependymoma, oligodendroglioma and astrocytoma should be considered. Histopathologic features and immunohistochemistry are essential in the differentiation between intra-ventricular tumours. A 4-year-old Boerboel dog presented with a left head tilt and a wide range of nonspecific clinical abnormalities not limited to weight loss, depression and marked bradycardia. MRI proved useful in providing a diagnosis of an intra-ventricular tumour and demonstrated the potential of choroid plexus carcinomas (CPCs) to metastasize. In this case, the histological features displayed malignancy with invasion into the neuroparenchyma, drop metastasis and immunohistochemistry with E-cadherin-positive result that assisted with differentiation of CPC from other tumours.

KEYWORDS

dogs, histopathology, magnetic resonance imaging (MRI), neuropathology, tumours

BACKGROUND

Brain tumours in dogs occur mostly in middle-aged to older dogs with the majority being >5 years of age.[1–5] Intra-ventricular tumours in dogs include mainly those arising from ependymal cells and choroid plexus epithelium.[6,7] Choroid plexus tumours (CPTs) comprise approximately 10% of all primary brain tumours in dogs[5] and MRI can assist in differentiating choroid plexus papillomas (CPPs) and choroid plexus carcinomas (CPCs) that may provide valuable prognostic information.[5] Other differential diagnoses for an intra-ventricular tumour include ependymoma and less likely oligodendroglioma and astrocytoma.[7,8] This knowledge can assist the clinician in considering all possible differential diagnoses for an intra-ventricular tumour in a dog and in deploying the correct modalities to make a definitive diagnosis. Mainstream palliative treatment for brain tumours includes anticonvulsant drugs for tumour-associated structural epilepsy, corticosteroids for peritumoural vasogenic oedema and analgesic therapy.[1,9–11] Surgical resection and fractionated radiotherapy is considered the principal treatment of choice.[11] Chemotherapeutics agents with the ability to penetrate the blood–brain barrier can be considered.[1,12–14] Other therapy options include stereotactic radiosurgery and convention-enhanced delivery.[11] Advancements in MRI and functional neuroimaging will continue to develop the therapeutics of brain tumours.[11] Despite progression in therapeutic options, definitive antemortem diagnosis of brain tumours remains infrequent.[11]

CPTs are intra-ventricular tumours that occur mostly in the lateral and fourth ventricles, with a lower incidence of occurrence in other ventricular locations.[1] To date, there is no accepted CPT classification system for dogs and is currently classified by the WHO human choroid plexus tumour criteria.[1,15] Histopathologic features of carcinomas are based on the presence of desmoplasia, microvascular proliferation, which is rare in CPCs compared with gliomas, and other malignant features such as high mitotic rates and areas of necrosis.[1,16]

Immunohistochemistry with use of Kir7.1 will stain the apical portion of neoplastic choroid plexus cells in the dog.[1,17] Furthermore, pancytokeratin expression is diffuse in CPTs.[1,18] E-cadherin labelling does not differentiate between benign and malignant CPTs and N-cadherin is more commonly localised in CPPs than CPCs.[1,19,20] Immunolabelling for glial fibrillary acidic protein (GFAP), vascular endothelial growth factor and platelet-derived growth factor has also been recorded.[1,16,19]

FIGURE 1 The left head tilt observed in a Boerboel dog

We discuss the important tumours to consider on a differential diagnoses list for an intra-ventricular tumour in a dog. Additionally, we explain how to support the diagnosis of a CPC using MRI, immunohistochemistry (IHC) and morphology to differentiate this tumour from other tumours.

CASE PRESENTATION

A 4-year-old intact male Boerboel dog (body weight, 39 kg) presented with a progressive 8-month history of weight loss, tonic-clonic seizures and a left head tilt. Physical examination revealed nonspecific clinical abnormalities including moderate obtundation and bradycardia with a pulse rate of 44 beats per minute, with a normal range being 60–120 beats/min in large breed dogs,[21] with a pronounced sinus arrythmia. Intermittent inappetence and lethargy, a score of two out of five using the five points body condition scoring system,[22] with a history of a 40% loss of body weight within the past 4 months, was evident. On palpation, the left ear region elicited a pain response. On neurological examination the main abnormalities detected were a left head tilt (Figure 1). All cranial nerve reflexes were within normal limits, and no ataxia was observed.

An initial basic workup included a complete blood count, blood smear, urinalysis and fecal evaluation. Haematology revealed a mild lymphocytopaenia, and on serum biochemistry mild hyperglycaemia and mild hypermagnesaemia were present. The faecal evaluation and urinalysis were unremarkable.

The bradycardia was of interest and consequently an echocardiogram and ECG was performed. The patient was hypertensive with a mean systolic blood pressure of 177 mm Hg compared with a normal reference range of 80–120 mm Hg.[21] A vertebral heart score of 10.5, with reference to the normal value range of 9.2–10.2,[23] was found. The bradycardia and sinus arrhythmia were confirmed on ECG and the heart did not display any disease on echocardiography. The findings of hypertension and bradycardia, suspected to be secondary to intracranial hypertension (ICH),[21] and the presence of a left head tilt and left ear region pain indicated the need for MRI.

LEARNING POINTS/TAKE-HOME MESSAGES

- All differential diagnoses, which include choroid plexus tumours and gliomas, should be considered in making a definitive diagnose of an intra-ventricular tumour.
- MRI proved useful in providing a diagnosis of an intra-ventricular tumour and demonstrated the potential of choroid plexus carcinomas (CPCs) to display malignant evidence of drop metastasis.
- CPC may include a histopathologic feature of tumour invasion into the neuroparenchyma.

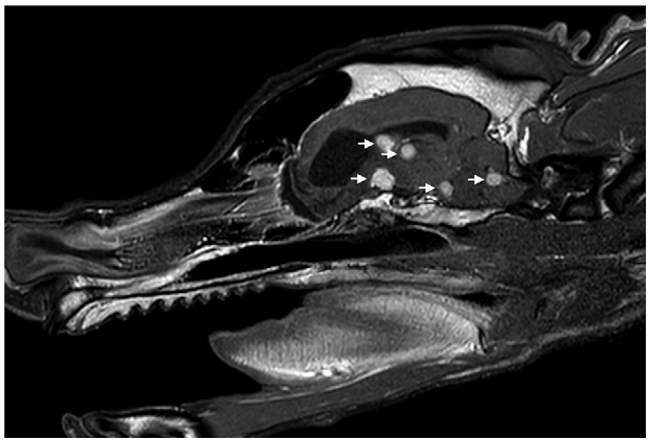

FIGURE 2 Sagittal post-contrast T1W MRI image with marked hyper-intensities showing multiple well-defined masses in the brain indicated by the arrows

INVESTIGATIONS

Magnetic resonance imaging (MRI)

Images were obtained with a Phillips, Achieva 1.5T (dStream) MRI machine. MRI findings of the brain, as shown in Figures 2–6, indicated the presence of multiple intracranial intra-ventricular masses. The MRI sequences included sagittal, dorsal and transverse T2-weighted, pre- and post-contrast sagittal and transverse T1-weighted, transverse FLAIR, transverse diffusion-weighted imaging and apparent diffusion coefficient and transverse T2. Multihance (Gadobenate dimeglumine), manufactured by Bracco, was the gadolinium-based contrast agent that was administered intravenously at a dose of 0.1 mmol/kg. The images demonstrated multiple T2W/FLAIR iso-to-hyperintense, T1W isointense, markedly contrast-enhancing, well-defined masses throughout the brain's ventricular, cisternal and subarachnoid systems. The MRI concluded the presence of multiple intra-ventricular and extra-axial masses associated with the ventricles (lateral, third and fourth), subarachnoid cisterns and a significant amount extension of the lateral ventricles into the olfactory bulb. More specifically

- In the central part of the right lateral ventricle, a bilobed mass of 20 mm × 17 mm × 15 mm in size extends into the dorsal aspect of the third ventricle (Figures 2 and 3).

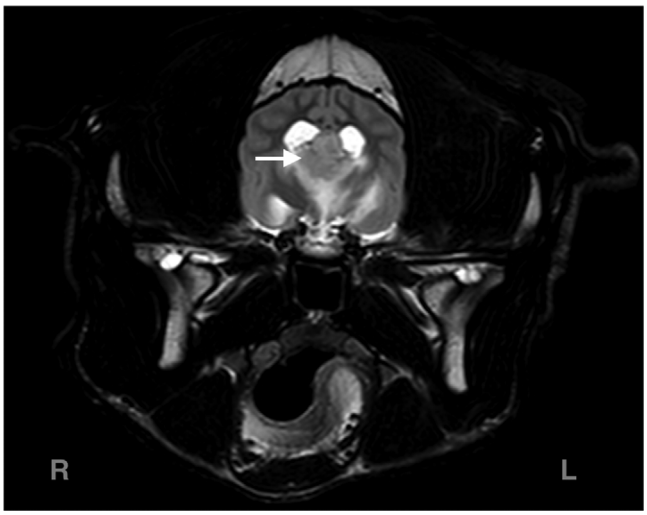

FIGURE 3 Transverse post-contrast T2W MRI image showing a large mass associated with the lateral and third ventricle indicated by the arrow

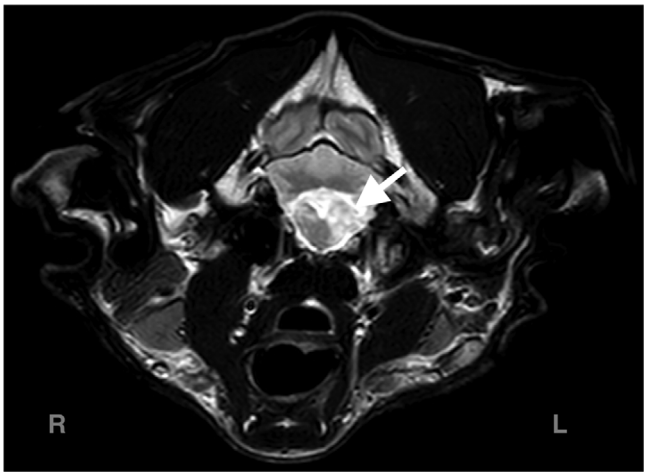

FIGURE 4 Transverse post-contrast T2W MRI image with contrast enhancement in the region of the fourth ventricle and brain stem indicated by the arrow

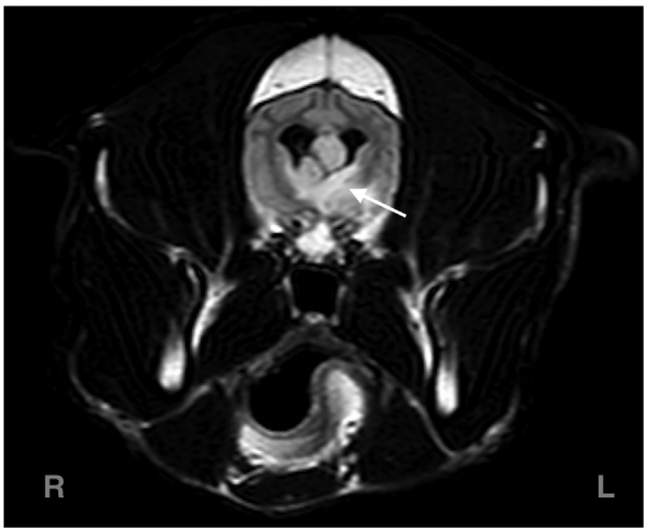

FIGURE 5 Transverse FLAIR image with long TR with hyper-intense perilesional oedema indicated by the arrow

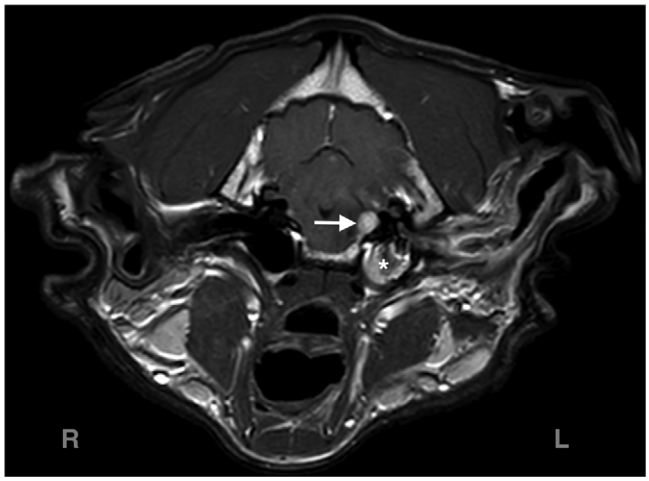

FIGURE 6 Transverse post-contrast T1W MRI image displaying the tumour (arrow) and altered tympanic cavity. Notable enhancing material in the tympanic cavity indicated by the asterisk

- In the left and right lateral ventricle masses of 10 mm × 5 mm × 3 mm and 10 mm × 5 mm × 5 mm in size are located (Figure 2).
- In the third ventricle a focally extensive mass of 20 mm × 15 mm × 5 mm in size is located (Figures 2 and 3).
- In the fourth ventricle, dorsolateral around the brainstem, a focally extensive mass of 15 mm × 10 mm × 5 mm is located (Figures 2 and 4).

Marked perilesional hyperintensities were noted on the FLAIR sequence with long repetition time (TR) that indicated associated perilesional oedema (Figure 5). Additionally, the left tympanic region was occupied by moderately heterogeneous, predominantly T1W hyperintense material (Figure 6). The wall of the outer ear canal was moderately to severely thickened, with marked stenosis at the terminal third of the external ear canal and elicited heterogeneous contrast enhancement.

The diagnosis of tumour type for primary brain tumours when relying on MRI features has been found to have an accuracy percentage of 70%.[24] The MRI report concluded the main differential diagnosis as a CPC with drop metastases with a resultant guarded prognosis.

Postmortem findings

This patient was subjected to euthanasia with administration of Eutha-naze (sodium pentobarbitone), manufactured by Bayer, intravenously at a dose of 1 mg/kg. Soon thereafter, a postmortem examination was performed to obtain a definitive diagnosis.

Grossly, multiple pale pink to white well-defined, non-encapsulated masses were noted in the brain and olfactory region. These included a mass (20 mm × 17 mm × 15 mm) in the lateral ventricle and a mass (20 mm × 15 mm × 5 mm) in the third ventricle (Figure 7). Additionally, a mass (10 mm × 10 mm × 5 mm) on the right dorsal medulla oblongata (Figure 8) and a 10 mm × 5 mm × 3 mm mass at the

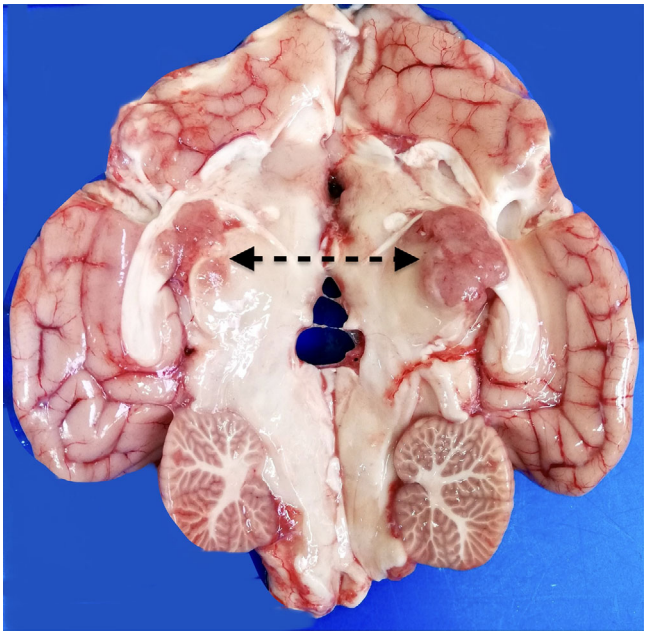

FIGURE 7 Macroscopic postmortem examination of the brain showing a well-defined 20 mm × 17 mm × 15 mm mass that occupies the lateral and third ventricle

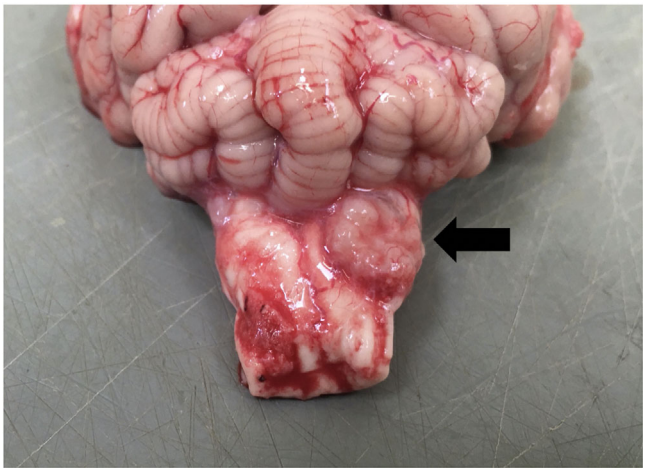

FIGURE 8 Macroscopic postmortem examination of the dorsolateral right side of the brainstem showing a 10 mm × 10 mm × 5 mm mass

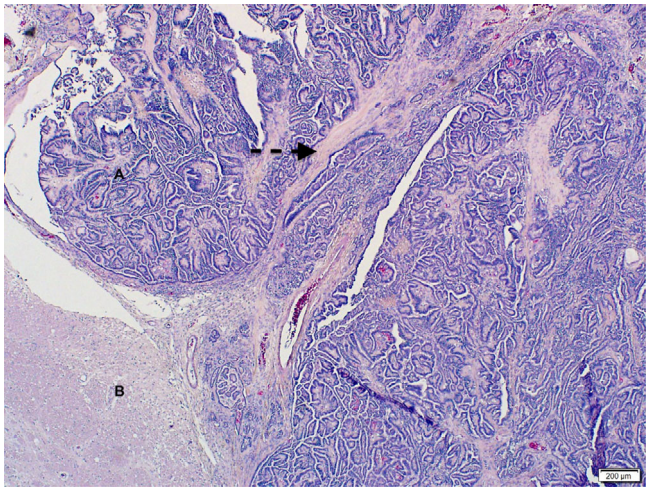

FIGURE 9 Histopathology section with haematoxylin and eosin (H&E) stain showing the invasion of the tumour associated with the lateral ventricle with papillary-like architecture (A) invading the neuroparenchyma (B). Desmoplasia that is associated with the tumour is indicated by the arrow: Obj × 4

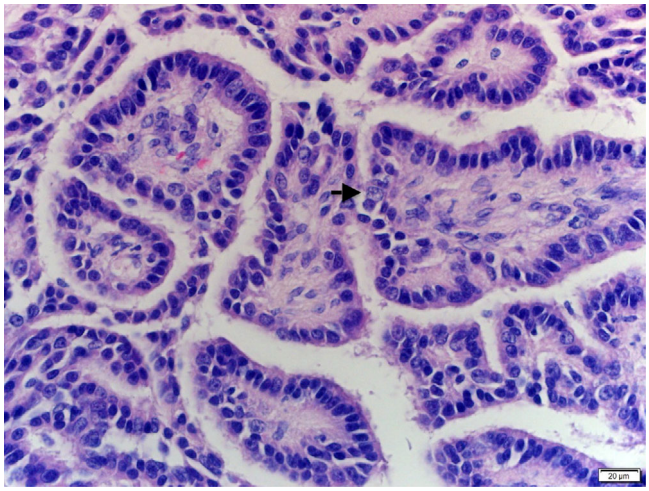

FIGURE 10 Histopathology section with H&E stain of the tumour associated with the lateral ventricle showing mitosis indicated by the arrow and cellular atypia. Obj × 40. H&E, haematoxylin and eosin

caudal margin of the cerebellum were noted. Macroscopically, the entire spinal cord showed no obvious masses.

Multiple tumour tissue samples, with the largest sample being 1 cm^3, were fixed in 10% buffered formalin for 6 days before trimming for histopathology processing. The samples were pooled in a tight seal container and stored at room temperature. Histopathologic sections were prepared and stained on-site.

Histopathology findings

Haematoxylin and eosin (H&E) staining, displayed in Figures 9 and 10, showed that the multiple brain neoplasms observed macroscopically were histologically identical and consisted of proliferating cuboidal to columnar neoplastic epithelial cells that formed papillary projections interspersed by coarse fibrovascular stroma. Desmoplasia within areas of tumour infiltration into the neuroparenchyma was noted. The neoplastic cells were predominantly arranged in a single layer; however, occasional multi-layered areas (up to three layers thick) and small sheets replacing papillary projections were also noted. Neoplastic cell nuclei generally displayed moderate anisokaryosis. The mitotic count is defined as counting the number of mitotic figures in 10 consecutive high-power fields (hpf).[25] In this case, five mitoses were counted in 10 consecutive hpf at × 400 magnification. Multifocally, areas of intra-tumoural necrosis and neoplastic cell infiltration into the adjacent brain parenchyma were also noted.

Available IHC markers that were applied included pancytokeratin, GFAP and E-cadherin. In this case, IHC immunolabelling was negative for pancytokeratin. The GFAP marker displayed no stained stellate cellular processes and evidently no signs of gliosis could be seen. E-cadherin yielded a positive result (Figure 11).

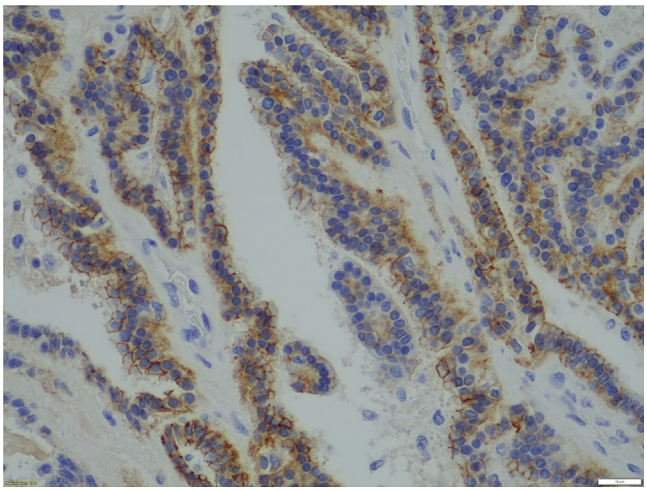

FIGURE 11 immunohistochemistry (IHC) immunolabelling for E-cadherin expression was positive: Obj. × 40

DIFFERENTIAL DIAGNOSIS

A conclusive diagnosis for an intra-ventricular tumour in a dog relies heavily on morphologic features and IHC[26,27] and in doing so, all differential diagnoses should be considered. Differentiation between CPTs (a choroid plexus papilloma, atypical choroid plexus papilloma or choroid plexus carcinoma) and gliomas, including ependymoma, oligodendroglioma and astrocytoma should be considered.

Human choroid plexus tumour classification is based on the WHO classification and is adopted in veterinary medicine.[6,15] The WHO classification system has been designed to group-specific tumour subsets according to their morphology, molecular features and their clinical outcome.[15] These tumours are classified as grade I CPP, grade II atypical CPP and grade III CPC.[6] In this classification system, a diagnosis of grade III CPC is based on the presence of at least four of the following six criteria: five or more mitotic figures in 10 consecutive hpf, nuclear atypia, multi-layering of epithelium, increased cell density, loss of papillary pattern with solid cell sheets and/or multifocal areas of necrosis. The presence of all six histological features described for the WHO histological grading of tumours of CPTs in humans[15] confirmed the suspicion of a grade III CPC rather than a grade I CPP or a grade II atypical CPP.

CPP has morphologic features that include monomorphic nuclei with low mitotic activity and atypical CPP have high increased mitotic activity with nuclear pleomorphism and blurring of the papillary pattern.[15]

The evident rare occurrence of ventricular involvement CPCs generally shows multiple areas of invasion with vacuolation and desmoplasia of the adjacent neuroparenchyma.[27] Features of CPC found in our case showed evidence of invasion into the neuroparenchyma and desmoplasia as described in the literature.[27]

Differentiation of a CPC from a papillary variant of ependymoma was based on the typical HE features in combination with IHC. Ependymomas in dogs have rosettes and pseudorosettes, can express GFAP and Olig2 immunolabelling is rare.[7]

It can be challenging to differentiate between a CPC and papillary ependymoma.[6] The most common location of an ependymal tumour is in the lateral ventricle as opposed to a CPT with the most common location being the fourth ventricle.[7,28] The GFAP stain provided no evidence of stellate projections, indicating gliosis, in this case. The negative cytokeratin result was contrary to what is described for CPTs in human literature[29] where positive cytokeratin is noted in virtually all CPTs regardless of whether they are benign or malignant. In the veterinary literature, the same premise was adopted for dogs where cytokeratin was used to support a diagnosis of CPTs. However, it has been found that this IHC is unreliable in dogs and only labels a low number of these tumours.[19] As an alternative, E-cadherin and β-catenin, also used in human CPTs, have proven to be more reliable in dogs to support a diagnosis of a choroid plexus tumour.[19] β-catenin was not available; however, E-cadherin was available and yielded positive immunolabelling of the neoplastic cells. N-cadherin and β-catenin have also been described as useful in differentiating between ependymal and choroid plexus neoplasms.[27,30]

Oligodendroglioma and astrocytoma should be considered, even though less likely, in the differential diagnosis of intraventricular tumours in dogs.[7,8] Cerebral oligodendroglioma in dogs can clinically and grossly present as intra-ventricular tumours and is important to consider as these tumours are typically not included as a differential diagnosis for cerebral ventricular tumours.[8,31] Uncommon manifestations of oligodendroglioma have been associated with the ventricular system and thus should be considered a differential diagnosis for an intra-ventricular tumour.[8,32,33] Additionally, although there is no veterinary literature that describes a true intraventricular oligodendrogliomas; breakthrough of the ventricular lining and invasion of the ventricular spaces, occurring as intra-ventricular tumours with involvement of the adjacent white matter, has been reported.[6,8,28,34]

Oligodendrogliomas have histologic features that compose of closely packed sheets of round to oval neoplastic cells supported by a fine fibrovascular stroma and multiple accumulations of mucinous-like material.[8] The immunopositivity for GFAP and Olig2 is highly supportive of a glioma.[7,35,36,]Oligodendroglioma has been found to be uniformly immunoreactive for Olig2 and negative for neuron-specific enolase, neurofilament and GFAP.[8]

Astrocytomas with ventricular involvement can rarely occur in dogs and IHC is an essential tool to confirm diagnosis.[7] Histologically, astrocytomas is composed of polygonal to elongate cells arranged in streams and bundles with evident eosinophilic cytoplasm and oval to elongated nuclei. IHC for these neoplastic cells tend to have strong cytoplasmic immunolabelling for GFAP and strong nuclear immunolabelling for Olig2.[7]

The definitive diagnosis in this case was based on typical histological features, the invasion of neuroparenchyma, and a positive E-cadherin IHC result.

Lastly, paradoxical vestibular syndrome (PVS) could explain the left head tilt as a result of an extra-axial mass on the right caudal cerebellar peduncles.[37]

TREATMENT

Treatment of CPTs in humans relies on surgery and radiation.[15] Surgical tumour resection and fractionated

radiotherapy is considered the principal treatment of choice in dogs.[11,38] Notably, consistent evidence-based data about surgical outcome is lacking and the inclusion of surgery or biopsy sampling should be made on individual case basis.[38] The benefits of surgical resection of brain tumours include immediate improvement in neurological status due to relief of mass effect and ICH.[1,38] Intra-operative ultrasonography provides a reliable and widely available tool for intra-operative localization.[38] Subtotal tumour resection may improve response to adjuvant radiation and chemotherapeutic protocols.[38] In addition, surgical resection enables histopathological diagnosis of the tumour.[1] However, the acute perioperative mortality rate is 11% and nearly 50% of dogs experience acute adverse effects associated with intracranial surgery.[1,39–41] The most frequent causes of morbidity include aspiration pneumonia, intracranial hemorrhage or infarction, pneumocephalus, medically refractory provoked seizures, neurological disability, electrolyte and osmotic disturbances and thermoregulatory dysfunction.[1,39,40] Approximately 10% of brain tumour cases treated with radiation therapy can experience treatment-related mortality or adverse effects.[40] Other therapy options include stereotactic radiosurgery and convention-enhanced delivery.[11] Chemotherapeutics agents with the ability to penetrate the blood–brain barrier can be considered, such as alkylating agents or antimetabolite hydroxyurea.[1,12–14]

Dogs are often subjected to euthanasia, as in this case, or spontaneous death.[27,42] The MRI report concluded that the prognosis for this CPC was guarded. Conservative treatment was considered, but due to the progressive stage of the tumour and the protracted history of weight loss, the owner elected euthanasia.

OUTCOME AND FOLLOW-UP

None.

DISCUSSION

CPTs are uncommon neoplasms that arise from intraventricular choroid plexus epithelium, accounting for 10% of all primary central nervous system tumours in dogs.[6,16] CPTs occur in middle-aged dogs[6] and breeds at increased risk of brain tumours include the Boxer and Golden Retrievers.[27] This case demonstrates that other breeds, such as Boerboel,[43] can also be affected.

Similar to the patient in this case, clinical presentation in patients with CPTs can demonstrate marked variation and may include ataxia, tetraparesis, blindness, depression, seizures, head tilt, tremors, neck pain, aggression and urinary incontinence.[27] Dogs with brain neoplasia may also present with nonspecific clinical signs such as inappetence, weight loss and lethargy.[1] Notably, the multifocal intracranial masses, together with the secondary changes caused by oedema and inflammation, could arguably have accounted for the multitude of clinical signs observed. The bradycardia and hypertension is suggested to be secondary to ICH caused by the masses.[21] Increased ventricular pressure, which these masses can cause, results in reversed flow of cerebrospinal fluid flow back into the neuroparenchyma.[21] Autoregulation mechanisms are often impaired in patients with intracranial disease and when exhausted, ICH leads to a decreased cerebrospinal fluid flow, cerebral ischaemia and carbon dioxide accumulation.[21] Consequently, this stimulates the release of catecholamines that result in systemic vasoconstriction and increased cardiac output.[21] This hypertensive state causes a vagally mediated bradycardia.[21] Furthermore, the left head tilt that was observed (Figure 1) together with MRI and postmortem findings of the 10 mm × 5 mm × 3 mm mass on the dorsolateral right side of the brainstem in the region of the cerebellar peduncles (Figures 2, 4 and 8) is suggestive of PVS.[37] The term PVS is used to describe neurological signs in which the head tilt and loss of balance is contralateral to the side of the brain lesion, typically involving the cerebellar peduncle.[37] Morse specifically, afferent fibres enter the caudal cerebellar peduncle from five sources, which includes the vestibulocerebellar tract.[44] The most common causes for PVS in dogs have been found to be infarcts, inflammation and neoplasia.[37,45]

This case's findings correlated with previously described findings that state that CPTs often occupy more than one anatomical division of the brain, which may cause neurological abnormalities suggestive of multifocal disease.[2] The most common intracranial sites for CPCs, arranged from highest to lowest incidence, are in the fourth, third and lateral ventricles.[6] Others concluded that CPTs most commonly occur in the lateral and fourth ventricles.[1] In this case, there was involvement of the fourth, third and lateral ventricles. Our case varied from previous literature stating that tumours in dogs rarely involve more than one ventricle simultaneously.[6]

MRI findings can be useful in the tentative diagnosis of CPTs and suggested evidence of malignancy to support the diagnosis of a carcinoma.[5] The MRI findings showed that the presence of metastasis was more likely to be a carcinoma.[5] Metastases to the subarachnoid space or within the intraventricular dissemination are important features of CPCs and are easily detected by MRI.[6] CPCs have been reported to have a higher tendency to metastasize by a mechanism known as drop metastases as seen in this patient.[1] In this case, there was evidence of extra-axial drop metastases, with histopathology proving relation to the intraventricular tumours. Additionally, CPCs can be challenging to diagnose, because one of the features of dissemination via cerebrospinal fluid can cause them to be confused with metastatic carcinomas.[27]

Cerebrospinal fluid (CSF) analysis could have provided complementary information to the MRI and could assist in the prioritization of differential diagnoses.[1] If CSF analysis were to be performed it should be done after advance imaging for accurate evaluation.[1] Furthermore, patients with ICH carry greater risk when performing CSF tap and in this case CSF analysis was not performed.

The postmortem examination was consistent with previous literature that describes the tendency of CPCs to demonstrate widespread metastasis to the subarachnoid spaces and the cerebellum.[6] The MRI findings revealed additional metastasis to the olfactory area, previously only found in one out of five cases of primary intracranial neoplasia.[1]

Histopathological examination must contain at least four out of six histological features mentioned previously for the tumour to be classified, according to the WHO, as a grade

III CPC.[6,15] These six criteria were all observed in this case. The WHO classification and grading system for CPC does not include invasion of the neuroparenchyma adjacent to the tumour as an accepted grading criteria; however, this finding in dogs is a common feature of CPC.[6]

Immunohistochemistry for cytokeratin, GFAP, E-cadherin, N-cadherin and β-catenin has been described to be useful, yet inconsistent and with low specificity, to support a diagnosis of CPT.[27,30] Positive result of pancytokeratin stain in CPTs has been variable.[27] In this case, E-cadherin was the most useful and showed a positive result (Figure 11). Kir7.1 has been described to be a reliable antibody for diagnosis of CPCs.[27] This stain's specificity can differentiate CPTs from other primary or metastatic brain tumours, including metastatic carcinomas.[30] Kir7.1 was unable to be sourced but would be recommended based on literature.

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CONFLICT OF INTEREST

The authors have no conflicts of interest to declare.

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ETHICS STATEMENT

The authors declare that ethical research was conducted. The authors consciously assure that for the manuscript is the authors' own original work, which has not been previously published elsewhere and is not currently being considered for publication elsewhere. The paper reflects the authors' own research and analysis in a truthful and complete manner. Furthermore, the paper properly credits the meaningful contributions of co-authors and co-researchers and the results are appropriately placed in the context of prior and existing research. All sources used are properly disclosed. Lastly, all authors have been personally and actively involved in substantial work leading to the manuscript and will take public responsibility for its content.

AUTHOR CONTRIBUTION

Primary case involvement: AA de Witt, JP Schoeman and M Lewis. Drafting of the article: AA de Witt. Critical revision of the article: AA de Witt, M Lewis and JP Schoeman.

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