

Choroid plexus papilloma in a dog surviving for 15 months after diagnosis with symptomatic therapy

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ABSTRACT. A 4-year-old female French bulldog presented with a 6-month history of right-sided head tilt and acute onset ataxia. Magnetic resonance imaging (MRI) showed a large mass lesion at the cerebellomedullary pontine angle. The dog was able to stand and walk after beginning symptomatic therapy with prednisolone, acetazolamide and glycerin. Magnetic resonance imaging 10 months after the first examination indicated slight expansion of the tumor. The dog was able to walk with continuous symptomatic therapy for 15 months until death, although the head tilt persisted. On postmortem examination, the gross tumor was slightly larger than when seen on the second MRI scan and was histopathologically diagnosed as a choroid plexus papilloma.

KEY WORDS: canine, cerebellomedullary pontine angle, choroid plexus papilloma

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Choroid plexus tumors account for 7-13% of all primary brain tumors in dogs [4, 5, 10, 11] and are histologically classified as choroid plexus carcinoma (CPC) or choroid plexus papilloma (CPP) [9, 14, 16]. CPC is a locally invasive tumor and more than half metastasize to distant sites in the brain, whereas CPP rarely shows local invasion [14]. Although the histological and diagnostic imaging features of canine CPP have been well documented [5, 9, 14], there are few reports of the long-term clinical course of CPP that include periodic changes in the diagnostic images [7]. We describe a dog with CPP that survived for 15 months after detection using magnetic resonance imaging (MRI) with symptomatic therapy, during which minimal tumor growth was seen.

A 4-year-old intact female French bulldog, weighing 7 kg, presented with a 6-month history of right-sided head tilt and acute onset ataxia. On physical examination, horizontal nystagmus was seen, and the dog could not stand or walk, even when supported. Postural reactions were absent in all four limbs, and patellar reflexes were exaggerated. No abnormalities were noted on blood examination and thoracic radiography. Transverse MRIs of the brain revealed a mass lesion at the left cerebellomedullary pontine angle occupying the left half of the pontine and cranial medulla oblongata (Fig. 1). The mass was well-circumscribed and heterogeneously hypointense on T1-weighted images (T1WI), hyperintense on T2-weighted images (T2WI) and strongly enhanced with a

heterogeneous pattern on T1WI after intravenous administration of gadolinium contrast. Both the lateral and third ventricles were mildly dilated. Based on these findings, the dog was diagnosed as having a primary brain tumor.

The dog was able to stand and walk with resolution of the nystagmus within three days of beginning treatment with prednisolone (3 mg/kg/day, orally (PO)) and a 10% glycerin/5% fructose mixture (Glyceol[®], Chugai Pharmaceutical, Tokyo, Japan) at a dose of 5 ml/kg, intravenously, q 12 hr. Prednisolone (1.5 mg/kg/day) and acetazolamide (5 mg/kg, PO, q 12 hr) were continued. Hydroxyurea (30 mg/kg, PO, every other day) was administered for the first 30 days, but the owner declined further anti-tumor therapy. MRI 10 months after the first presentation indicated slight expansion of the tumor (Fig. 2B). Transient deterioration with difficulty standing and eating occurred 2, 10 and 13 months after the first presentation, and each time, these symptoms improved within a few days of administering glycerin and a higher dose of prednisolone (3 mg/kg/day). Fifteen months after the first presentation, the dog developed coma and died despite intensive treatment.

On postmortem examination, a large dark red papillary mass was detected at the left side of the cerebellomedullary pontine angle occupying almost two-thirds of the medulla oblongata (Fig. 2C). Histopathologically, the tumor consisted of papillae lined by a single layer of columnar or cuboidal epithelium supported by vascular connective tissue stroma (Fig. 3). The tumor did not invade, but did compress adjacent neural tissue where mild edema and hemorrhage were observed. Based on these findings, the tumor was diagnosed as a CPP.

According to a recent report that included a case series and literature review [14], the incidence of CPP in 123 dogs with

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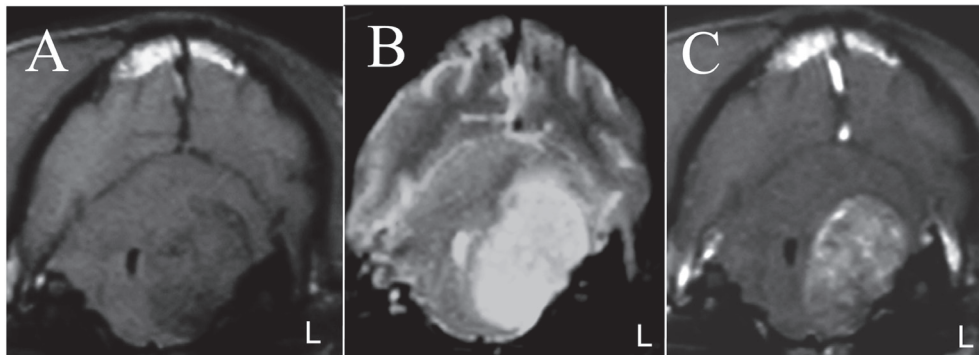


Fig. 1. Transverse magnetic resonance images at the level of the pons; (A) T1-weighted image, (B) T2-weighted image and (C) enhanced T1-weighted image.

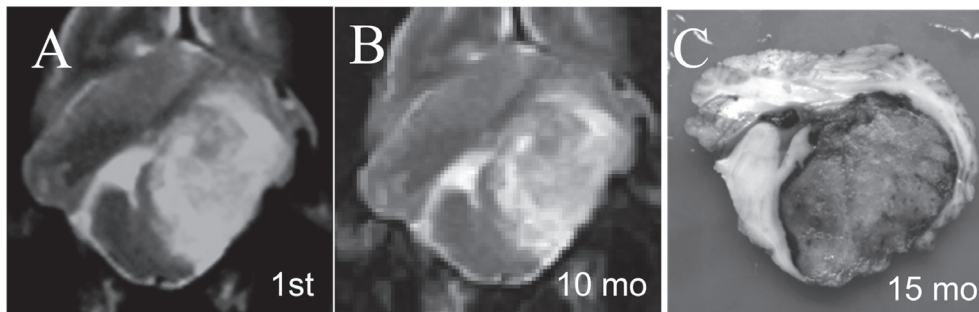


Fig. 2. Transverse T2-weighted magnetic resonance images, A: first series, B: 10 months after diagnosis and gross lesion (C) at the level of the fourth ventricle.

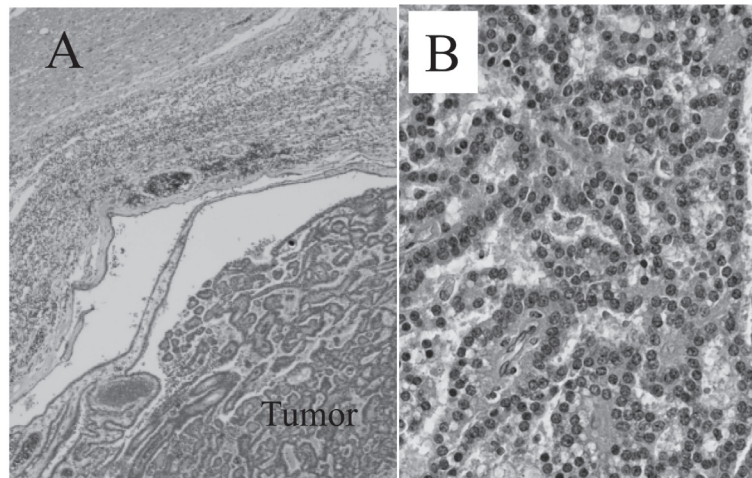


Fig. 3. Photomicrograph showing the histopathological features of the neoplastic lesion. (A) The tumor did not invade, but did compress adjacent neural tissue (HE stain, $\times 40$). (B) The tumor consisted of papillae lined by a single layer of columnar or cuboidal epithelium (HE stain, $\times 400$).

choroids plexus tumors was 60% (74 dogs), and involvement of the fourth ventricle was seen in 35 of the 74 CPP cases (47%). The median age of dogs with CPP and CPC

was 5 and 7 years, respectively. Central vestibular syndrome is the most common clinical symptom in canine CPP of the fourth ventricle [2, 3, 13, 16]. Right-sided head tilt resulting

from the left-sided tumor seen in the dog in our report is known as paradoxical vestibular disease, occasionally seen in canine CPP of the fourth ventricle [2, 3, 16].

The MRI features seen in our case, including a well-circumscribed mass that was hyperintense on T2WI and strongly enhanced by contrast medium on T1WI, were consistent with previous reports of canine CPP [5, 14]. Contrast enhancement in CPP is thought to be associated with lack of a restrictive vascular barrier at the choroids plexus [5]. Ventriculomegaly is also common (78%), but a hypointense pattern on T1WI is not typical (33%) in canine CPP [14]. Differentiating CPP from CPC on MRI can be difficult, but intraventricular or subarachnoid metastases occasionally seen in CPC are not evident in CPP [14].

Reported survival time after examination in 13 dogs with seven CPPs [2, 12, 16] or six CPCs [8, 13, 15, 16] not receiving surgery or radiation was less than 2 months. However, in most of these cases, the exact lengths of time with clinical symptoms were not described. A 25-month survival time after cytoreductive surgery for CPP has been reported in one dog [7], suggesting a slow growth of CPP as seen in our case. The use of hydroxyurea reportedly led to partial remission of a meningioma in one dog [12], but it is uncertain whether this drug had the same effect in our case. However, symptomatic therapy using corticosteroid and diuretics was useful in maintaining the dog's improved status. The tumor had not invaded, but had markedly compressed the medulla oblongata with peritumoral edema and ventriculomegaly; therefore, these drugs might contribute to reducing intracranial pressure and edema.

Although limited reports suggest a grave prognosis in dogs with CPP, prolonging survival using primary radiation therapy has been suggested [1, 11]. Relatively long survival in two CPP cases treated with surgery (25 months) [7] or surgery plus radiation (14 months) [6] is documented, although two cases of death because of surgery are also reported [4]. Symptomatic therapy may, in some CPP cases, contribute to prolonged survival even in brainstem-compressed cases.

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