

A CRANIOPHARYNGIOMA IN A SEVEN-YEAR-OLD DOG

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ABSTRACT

A seven-year-old male Border Collie was presented with a history of lethargy, episodic circling, incoordination and polydypsia. Physical examination revealed depression, obesity and bradycardia. A neurological examination indicated the possible presence of a space-occupying lesion in the brain. Results of the clinical investigation revealed hyposthenuria, sinus bradycardia and increased concentration of protein in the cerebrospinal fluid. A computerised axial tomography scan revealed a mass in the region of the hypophysis. The dog was euthanased and a post mortem examination confirmed the presence of a craniopharyngioma.

Key words: Dog, computerised axial tomography scan, craniopharyngioma.

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INTRODUCTION

A review of the literature on brain tumours^{4,6,8,11,12,17} and pituitary gland tumours^{2,3,7,14} revealed that craniopharyngiomas are extremely rare^{7-10,13,15}.

Craniopharyngiomas are benign tumours that are derived from epithelial remnants of the oropharyngeal ectoderm of the craniopharyngeal duct (Rathke's pouch)^{2,3}. Compared to all other types of pituitary neoplasms, craniopharyngiomas occur in younger dogs and they are present in either suprasellar or infrasellar locations³. Craniopharyngiomas in young dogs are often large and grow along the ventral aspect of the brain, where they may incorporate several cranial nerves. In addition, they extend dorsally into the hypothalamus and thalamus³.

The clinical signs of this type of pituitary tumour are often a result of combined hormonal derangements. A lack of pituitary tropic hormone secretion

results in atrophy and subnormal functioning of the adrenal cortex and thyroid³. Dwarfism, due to subnormal secretion of somatotropin, prior to the closure of growth plates, and gonadal atrophy may also be features of this tumour³. Large tumours may also result in diabetes insipidus with polyuria, polydypsia and hyposthenuric urine. This is due to these tumours interfering with the synthesis and release of antidiuretic hormone^{3,15}. Eigenmann⁷ reported a case of panhypopituitarism with growth hormone deficiency, secondary hypothyroidism and secondary hypoadrenocorticism associated with diabetes insipidus in an adult dog, that had a suprasellar tumour suspected to be a craniopharyngioma. Cranial nerve deficits and other neurological syndromes may be caused by these tumours extending into the hypothalamus and compressing surrounding brain tissue³.

Craniopharyngiomas consist of alternating solid and cystic areas. The solid areas are composed of nests of epithelial cells (cuboidal, columnar or squamous) with focal areas of mineralisation. The cystic spaces are lined by columnar or squamous epithelial cells and contain keratin debris and colloid^{2,3,8,10}.

To the best of our knowledge this is the first reported case of a craniopharyngioma in an old dog.

CASE REPORT

A 7-year-old male Border Collie, body mass 20 kg, was presented with a history of lethargy, episodic circling and incoordination of 2 months duration. However, the patient had shown polyphagia and polydypsia over a longer period. The abnormal findings on physical examination were moderate obesity, a dull dry haircoat, bradycardia (heart rate 64 beats min⁻¹) and moderate depression. A complete neurological examination revealed depression, a slightly incoordinated gait with a tendency to circle to either side. Cranial nerve examination revealed no abnormalities. The forelimb and hindlimb reflexes were slightly exaggerated indicating an upper motor neuron lesion, while the attitudinal and postural reactions indicated a proprioception deficit in all 4 limbs. The neurological examination indicated a lesion in the brain. Investigative procedures included a bloodsmear, haematology, chemical pathology, urinalysis (specific gravity 1,007) faecal analysis, radioimmunoassays (cortisol 118 nmol l⁻¹, T4 (total) 25 nmol l⁻¹) electrocardiography (sinus bradycardia; heart rate 64 beats min⁻¹) cerebrospinal fluid analysis (protein 0,85 g l⁻¹, total nucleated cell count very low) and a computerised tomography scan (CAT).

Analysis of these results revealed hyposthenuria, a high concentration of protein in the cerebrospinal fluid (CSF) and sinus bradycardia. The CAT scan (Somatom 2) (Fig. 1) confirmed the presence of a mass in the region of the pituitary gland, causing pressure on the hypothalamus. Contrast studies with iopamidol (Jopomiron, Berlimed) slightly enhanced the outline of the mass. Because of the likelihood of the mass in the brain being a tumour, and the progressive nature of the condition, the owners requested euthanasia.

A complete post mortem examination was performed. Selected sections of various tissues as well as the lesion in the brain, were taken in 10% formalin for histopathological examination. The sections were routinely processed, cut and stained with haematoxylin and eosin (H & E).

A single lesion was seen in the brain. This extended as a focal cystic lesion from the hypophyseal stalk to the hypothalamic region. It was fluctuating, thin-walled, about 2 cm in diameter, and

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Fig. 1: Contrast-enhanced transverse CAT scan of the dog with a craniopharyngioma. A mass (black arrow) can be seen in the region of the hypophysis and hypothalamus

contained a pus-like, fluid content. After fixation, the content oozed out as the brain was sectioned to process specimens for histological purposes.

The histological lesions adjacent to the cyst were manifested by scant malacia and focal haemorrhage. The latter area was also infiltrated by scattered gitter cells, and focal large aggregates of haemosiderin-laden macrophages. The cyst wall was lined by an epithelium thrown into uneven folds producing cystic spaces containing amorphous cellular detritus and cholesterol clefts. A few of the spaces contained small calcified masses resembling corpora amylacia. The lining epithelium varied in appearance. It consisted mostly of undifferentiated columnar epithelium which in areas appeared to be compressed into a cuboidal to squamous epithelium. Occasionally there was an abrupt transition from undifferentiated columnar epithelium to a ciliated columnar epithelium in which the cytoplasm was eosinophilic. In the interstitial spaces, small aggregates of lymphocytes and plasma cells occurred. Large ovoid cells with a centrally-placed nucleus and ample granular eosinophilic cytoplasm occurred singly or in small groups in the supporting connective tissue framework.

Fig. 2: Low magnification of the wall of the craniopharyngioma showing the papillary arrangement, cholesterol clefts and accompanying inflammatory reaction (HE, x40)

DISCUSSION

The tumour in the brain was diagnosed as a craniopharyngioma. The diagnostic features were consistent with what has previously been described in the literature^{2,3,8,10}. Because these tumours are congenital tumours due to maldevelopment, this tumour must have been present since birth. This dog was asymptomatic until it reached 7 years of age.

All the abnormal clinical, neurological and laboratory findings can be related to the presence of the hypophyseal tumour. The pituitary gland had not been destroyed, as was evident at post mortem as well as on radioimmunoassays for cortisol and thyroxine, where these hormone con-

centrations were found to be within normal limits. There were also no growth defects or other clinical signs of growth hormone deficiency. The growth of the tumour into the hypothalamus resulted in central diabetes insipidus. Polyphagia with resultant obesity has previously been described in dogs with pituitary neoplasms³, however, the reason for this is not known. It is also possible that the tumour affected the functioning of the osmoreceptors in the hypothalamus resulting in polydipsia with a secondary polyuria. Elevated concentrations of protein in the cerebrospinal fluid frequently occurs in association with brain tumours⁶, as was the situation in this case. Sinus bradycardia is also a recognised entity in patients with space-occupying lesions in the brain^{6,16}.

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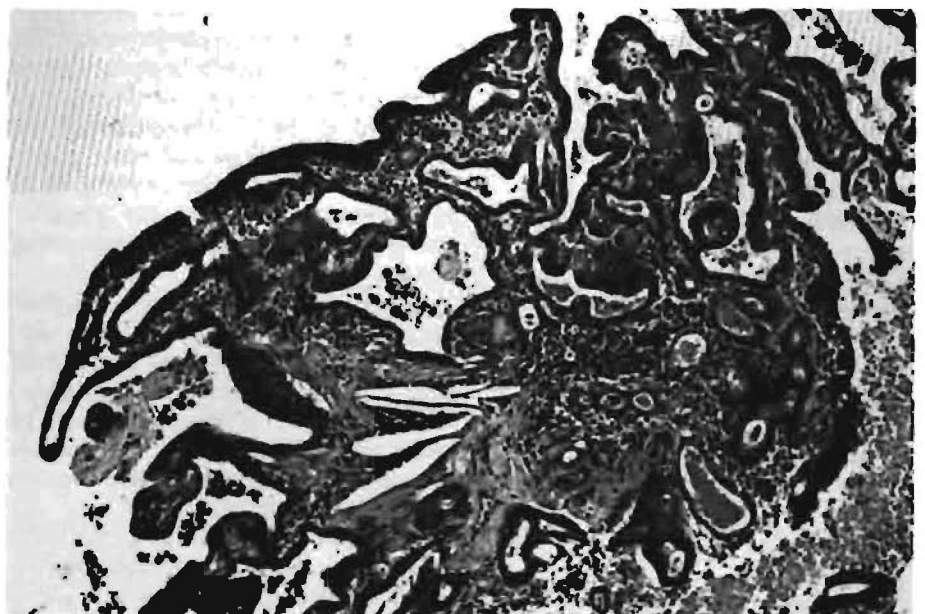
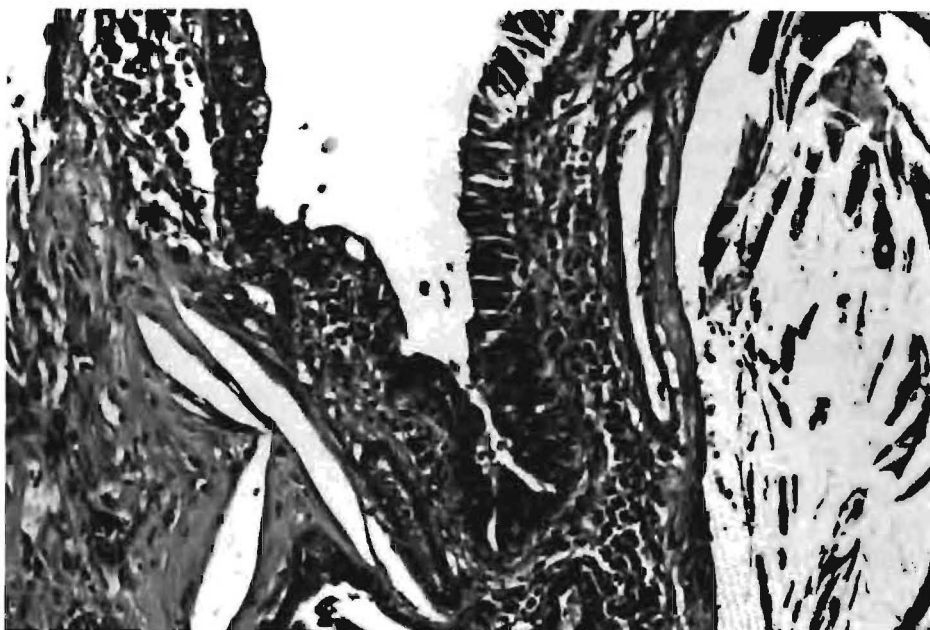


Fig. 3: High magnification of the craniopharyngioma showing the transition from non-ciliated to ciliated epithelium. Also note the inflammatory response and the accumulated, lipid-rich debris below the neoplastic epithelium (HE x400)



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