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Case Report

Empty Sella Syndrome Following Cranial Trauma in a Dog

Key words

pituitary insufficiency;
dog;
magnetic resonance imaging;
empty sella;
cranial trauma

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Abstract: This report describes a case of empty sella syndrome in a 6-month-old male dog that experienced cranial trauma 4 months prior. Clinical signs included behavioural disturbances such as aggression and growth retardation. Clinical examinations, laboratory tests, and magnetic resonance imaging (MRI) revealed the absence of the pituitary gland in the sella turcica and possible pituitary insufficiency indicated by decreased IGF-1 levels with TSH below the detection limit of the assay. The dog subsequently developed polyuria and intense polydipsia, raising suspicion of central diabetes insipidus. Persistent aggression led the owners to opt for euthanasia. In humans, empty sella syndrome has been associated with hormonal alterations, although it may be asymptomatic. This report underscores the importance of evaluating brain structures via MRI following cranial trauma and paying attention to the functionality of the endocrine system.

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Introduction

The sella turcica is normally fully occupied by the pituitary gland, with the diaphragm separating the gland from the suprasellar cistern. This diaphragm has a small aperture through which the pituitary stalk, its portal vessels, and the trabecular arteries pass, situated on either side of its anterior face. A pathological condition, often discovered incidentally during magnetic resonance imaging (MRI) is known as empty sella syndrome (ESS). This condition arises from the invagination of subarachnoid spaces into the sella turcica (1).

There are two types of ESS to distinguish. The first type arises from a pathological process, which may be tumour-related or due to cranial trauma, leading to pituitary involution and atrophy, with cerebrospinal fluid (CSF) occupying the space, referred to as secondary empty seller syndrome. The second type occurs without any known prior pathological process and is termed primary empty sella syndrome (1).

In human medicine, both types are often found in older patients, with a prevalence of 20% in individuals undergoing MRI for various reasons, and these patients may not necessarily present clinical signs related to pituitary function (2, 3). The objective of our study was to document a case of ESS in a dog, as this event has been infrequently reported and studied in veterinary medicine.

Case presentation

We report the case of a 6-month-old male dog weighing 12 kg, presented by the owners due to concerns about stunted growth. Additionally, they noted behavioural changes characterized by progressive aggression toward the owners and absence of play behaviour. The dog had been adopted at 2 months of age from an animal shelter, where the owners were informed that a door fell over the animal´s head two weeks prior and suffered a cranial trauma. At that time, his behaviour was normal and expected for the puppy age. Starting at 4 months old, they noticed changes in his behaviour (it didn't accept petting and tended to bite when someone tried to touch or pick it up), which gradually worsened to the point of aggression. The owners reported that the dog had grown less compared to the littermates, whom they saw about a month ago, noting that he was approximately half the size of the others.

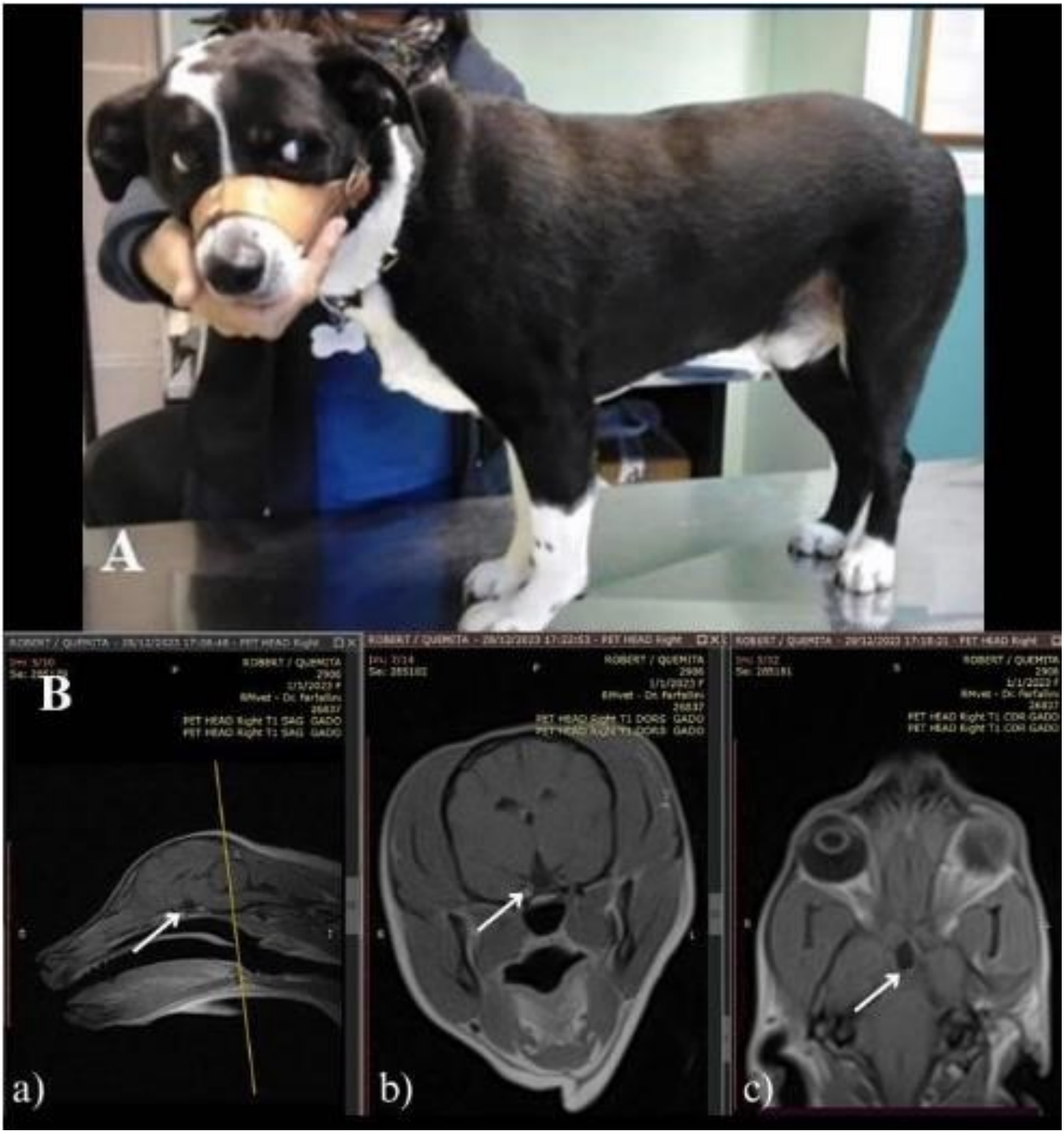

Figure 1: (A) The dog described at first consultation and (B) MRI images of the brain

Given the history of cranial trauma and the potential for brain injury, an MRI was indicated. Additionally, regarding the growth retardation, measurements of TSH, canine-specific free T4, and IGF-1 (insulin-like growth factor) as an estimator of GH concentration (due to the unavailability of a commercial canine GH assay) were performed. The hormones were measured using chemiluminescence immunoassay (Immulite 1000; Siemens, Germany) with kits provided by the manufacturer.

Results

The dog (Figure 1) showed no abnormalities during the general clinical examination, except for his smaller height compared to the littermates. The neurological examination revealed bilateral miosis, a diminished threat reflex, and a decreased response to stimulation of the VII cranial nerve. This led to the suspicion of an intracranial injury and the

subsequent indication for an MRI. The dog was anaesthetized for the study using a propofol protocol (4 mg/kg/IV) without prior induction. The maintenance dose was 0.5 mg/kg if required during the study. The MRI was performed weighting the region with 3-mm-thick slices with a gap of 0 in the axial, coronal, and sagittal planes.

A) Although the animal is still a puppy, the use of a muzzle is indicated due to its aggressive behaviour. B) MRI images of the brain in T1-weighted, cuts every 3 mm, presented four months post head trauma. Mid-sagittal (a), transverse (b) and coronal (c) sections. Arrows indicate the anatomical location of the pituitary gland (sella turcica) where the pituitary is not observed and filling of the sella turcica by cerebrospinal fluid (T-1 images)

The scan was performed using T1-, T2-, and T1-weighted sequences with gadolinium. MRI revealed, as a key and relevant finding, that the pituitary gland was not visible in the sella turcica and was replaced by CSF, leading to a diagnosis of ESS (Figure 1). No other intracranial or brain mass abnormalities (degeneration, ischemia) were observed or described after the application of the paramagnetic contrast agent. It is noteworthy that the cortical subarachnoid spaces show adequate depth, with no evidence of increased intracranial pressure.

The obtained endocrine laboratory results showed TSH concentrations ≤0.030 ng/ml (0.03-0.30), canine-specific free T4: 1.91 ng/dl (0.6-2.5), and IGF-1: 15 ng/dl (100 ± 25). Treatment with growth hormone at a dose of 0.1 IU/kg subcutaneously every 72 hours (HHT 4 UI®, BioSidus-Argentine) (4, 5) was initiated, with a plan to reevaluate the thyroid axis in 30 days, due to the TSH result.

The owners reported that the dog began experiencing polyuria and polydipsia three weeks after the tests. This development raised suspicion of central diabetes insipidus, which can occur following cranial trauma (6). A complete urinalysis was indicated as a first step. The urine density was 1.005 in relation to polyuria-polydipsia, being the only relevant finding. The rest of the urinalysis showed no abnormalities. Owners were asked to collect urine samples for 5 consecutive days to evaluate urine specific gravity, the results obtained for each day were between 1.001-1.006. A water deprivation test followed by desmopressin administration was indicated to confirm the diagnosis of a deficiency in antidiuretic hormone (ADH) secretion. Due to this new situation, and the continued aggression of the dog, the owners decided on euthanasia.

Discussion and conclusions

In human medicine, both GH deficiency and diabetes insipidus have been associated with empty sella syndrome in some patients, although this syndrome may be asymptomatic (2, 7, 8). In veterinary medicine, Burgener et al (9) described one case of hyperadrenocorticism associated with empty sella turcica. Later, in a study conducted by Konar et al. (10) analysing 370 MRIs in dogs they found that the pituitary gland was small or absent in 3% of cases, representing a total of 11 dogs, of which only one presented an endocrine disorder corresponding to hyperfunction of the adrenal axis. The authors noted that endocrine function tests were not conducted in most dogs, which were examined for neurological disorders, with ESS being an incidental finding in MRI. Thus, Konar et al. concluded that the entity of ESS also occurs in dogs as it does in humans, and pituitary hormones should be evaluated in the presence of this incidental finding, as animals are referred for consultation due to other disorders affecting the brain.

In the reported case, the dog had brought in due to growth retardation following cranial trauma and presented progressive aggression after the traumatic event. It cannot be confirmed nor ruled out that the behavioural changes were caused by the empty sella turcica or by the endocrinological alterations found; however, it could indeed be related to the trauma suffered. Although the MRI did not reveal brain lesions, neuronal or neurotransmitter lesions cannot be ruled out (11). In the reported case, and given the short time elapsed until euthanasia, behavioural studies were not performed.

Unlike the observations made by Burgerer et al (9) and Konar et et (10), in this case, the pituitary function was assessed regarding GH secretion (indirectly via IGF-1) and TSH due to the growth delay. The finding of the decreased IGF-1 concentrations indicates GH secretion insufficiency, clinically manifesting as reduced growth. While TSH was at its detection cut-off, the normal T4L level would rule out a failure in thyrotropin secretion. Our findings are consistent with what was reported by Murtagh et al (5) in a similar case. The later development of polydipsia and polyuria, with a urinary density of 1.005, is indicative of ADH deficiency. This deficiency may result from injury to the pituitary stalk, leading to impaired delivery and secretion of ADH (6).

Due to the owner's decision, confirmatory diagnostic tests for diabetes insipidus could not be performed, nor could other pituitary hormones be evaluated.

Based on the observations in this case, it strongly suggests that the growth retardation, along with marked polydipsia and polyuria and changes in behaviour developed following cranial injury. This aligns with previous research indicating a link between cranial injury and the development of ESS (5, 6).

It is suggestive that it also affected GH secretion, leading to the observed lack of growth. Furthermore, the development of diabetes insipidus is associated with cranial trauma, regardless of its concomitance with ESS (6, 11).

In veterinary medicine, this syndrome has been poorly documented and reported. Therefore, it is important that following cranial trauma, an MRI evaluation of brain structures is conducted, with due attention to the sella turcica, as well as appropriate consideration of clinical signs indicating hormonal deficiencies.

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Sindrom praznega turškega sedla po poškodbi lobanje pri psu

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Izvleček: Poročilo opisuje primer sindroma praznega turškega sedla pri 6-mesečnem psu, ki je 4 mesece prej utrpel poškodbo lobanje. Klinični znaki so vključevali vedenjske motnje, kot sta agresivnost in upočasnjena rast. Klinični pregledi, laboratorijske preiskave in magnetna resonanca (MR) so pokazali odsotnost hipofize v turškem sedlu in možno insuficienco hipofize, na kar je kazalo zmanjšanje ravni IGF-1 s TSH pod mejo zaznavnosti testa. Pes je nato razvil poliurijo in intenzivno polidipsijo, kar je vzbudilo sum na centralni diabetes insipidus. Zaradi trajajoče agresivnosti so se lastniki odločili za evtanazijo. Pri ljudeh je sindroma praznega turškega sedla povezan s hormonskimi spremembami, čeprav lahko poteka brez simptomov. Poročilo poudarja pomembnost ocenjevanja možganskih struktur z MR po poškodbi lobanje in pozornosti, ki jo je treba posvetiti delovanju endokrinega sistema.

Ključne besede: hipofizna insuficienca; pes; magnetna resonanca; prazno turško sedlo; poškodba lobanje