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CASE REPORT

Companion or pet animals

Transsphenoidal hypophysectomy as a treatment for Rathke's cleft cyst in a dog

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Abstract

The objective of the study was to describe the clinical, imaging, surgical and histological findings in a dog with Rathke's cleft cyst of the pituitary gland. A 6-year-old, female, neutered Staffordshire bull terrier was presented with an acute onset of abnormal behaviour. Magnetic resonance imaging of the skull showed a pituitary mass of 12.9 mm (height) × 8.8 mm (width) × 10.2 mm (length) with a pituitary height/brain area value of 0.73 (reference <0.31). Magnetic resonance imaging findings were suggestive of pituitary apoplexy or neoplasia. Transsphenoidal hypophysectomy was performed and a cystic mass was removed. Histopathology revealed a Rathke's cleft cyst lined by a layer of pseudo-stratified ciliated columnar epithelial cells and mucin-secreting goblet cells with remnant pituitary tissue with positive immunostaining against adrenocorticotropic hormone, alpha melanocyte and growth hormone in the periphery. Rathke's cleft cyst should be included in the differential diagnosis of pituitary masses in the dog, and transsphenoidal hypophysectomy is an effective treatment.

BACKGROUND

Rathke's pouch arises from the epithelial lining of the cranial end of the craniopharyngeal duct and gives rise to the anterior part of the pituitary. With development of the pouch wall into the anterior lobe, the residual lumen is reduced to a cleft, which usually regresses.[1,2] Failure of closure of the pouch during embryonal development results in a Rathke's cleft cyst (RCC) that is located between the anterior and intermediate lobe of the pituitary gland. Microscopically, they are lined with columnar or cuboidal epithelium with cilia and mucin-producing goblet cells. The cyst can be no more than a discontinuous cystic remnant of microscopic proportion. However, fluid or mucus can accumulate, making the cyst obvious to the naked eye. The content can range from mucoid to serous to cellular, accounting for its mixed appearance on MRI.[2] In humans, 20% of pituitaries contain macroscopically visible RCCs that are thin-walled, uniloculated, fluid-filled structures.[1,2] Almost all contain microscopic cysts, which may remain asymptomatic, but cysts larger than 1 cm may cause symptoms. Symptoms described in human medicine include headaches, disturbances in vision and endocrine dysfunctions such as hypopituitarism, hyperprolactinemia and rarely diabetes insipidus (DI). Surgery is indicated if the patient develops visual disturbances or if the cyst results in hypopituitarism. DI is not a surgical indication, as the clinical signs do not resolve after removal of the cyst. Usually, a transsphenoidal approach is used to drain the cyst. Recurrence after surgery is rare.[2] Adenohypophyseal cells can be found in small numbers. Squamous metaplasia of the lining of the cells may occur and may result in loss of superficial columnar cells and may mimic papillary craniopharyngioma, especially in cases in which there is also evidence of old haemorrhage and inflammatory infiltration.[1]

RCCs are not a common finding in veterinary medicine, only a few case reports describe the radiological and clinical findings[3,4] and the difficulty to differentiate RCC from pituitary apoplexy or neoplasia.[5] As suggested by Hasegawa et al., transsphenoidal hypophysectomy could be an adequate treatment for these patients.[4] The aim of this study is to show the clinical, imaging and histological findings, as well as describe the surgical treatment of an RCC in a dog.

CASE PRESENTATION

A 6-year-old, female, neutered Staffordshire bull terrier was referred to the Utrecht University Small Animal Clinic with a history of acute onset of abnormal behaviour for several days. The dog was reported by the owner to be nervous, jumpy on touch and had loss of hearing. Upon presentation to the referring veterinarian, the clinical signs had resolved and general physical examination and neurological examination showed no abnormalities. The clinical signs could have indicated a

lesion in the central nervous system and the owners elected to pursue further workup, which was performed at the referring veterinarian.

INVESTIGATIONS

Initial diagnostic workup included bloodwork, collection of cerebrospinal fluid (CSF) and MRI. Complete blood count and chemistry panel were unremarkable, except for a slight increase in red blood cells. Cytologic examination of CSF showed no abnormalities. MRI was performed using a low-field 0.2 Tesla scanner (Airis Mate, Hitachi, Japan), showing a well-defined ovoid pituitary mass located within the suprasellar region (Figure 1). The mass measured 12.9 mm (height) × 8.8 mm (width) × 10.2 mm (length). On T2-weighted and fluid-attenuated inversion recovery (FLAIR) images, the mass was homogeneously hyperintense, and on T1-weighted images it was hypointense compared to the grey matter. After intravenous (IV) contrast administration, there was a thin peripheral rim enhancement of the mass, which remained hypointense at the centre. There was a mass effect with mild compression of the adjacent ventral part of the third ventricle and infundibular recess.

DIFFERENTIAL DIAGNOSIS

Based on the acute onset of clinical signs, in combination with the findings on diagnostic imaging, the differential diagnosis for this suprasellar cystic lesion consisted of neoplasia (endocrine inactive pituitary adenoma, meningioma, lymphoma, granular cell tumour, gliomatosis cerebri or metastasis to the pituitary fossa), pituitary apoplexy, hypophysitis, pituitary abscess or a pituitary cyst. Other neoplasias of this region, such as carcinoma or meningioma, were considered less likely. An active, hormone-producing pituitary adenoma was not considered likely due to the absence of an endocrine syndrome like Cushing's syndrome (hypercortisolism) or hypersomatotropism.

TREATMENT

After diagnosis of the pituitary mass, the dog was referred to the Utrecht University Small Animal Clinic. Despite the fact that clinical signs had resolved at the time of presentation, owners elected for surgical removal of the pituitary mass, as the differential diagnosis included neoplasia. The available low-field MR images were used for surgical planning purposes. Transsphenoidal hypophysectomy was performed as described previously.[6] Briefly, the patient was anaesthetised with midazolam (0.15 mg/kg IV, Aurobindo Pharma, Baarn, Netherlands), methadone (0.25 mg/kg IV, Comfortan, Eurovet Animal Health, Bladel, Netherlands) and atropine (5 µg/kg IV, Centrafarm, Etten-Leur, Netherlands) and propofol (3 mg/kg IV, Fresenius Kabi, Huis Ter Heide, the Netherlands). An endotracheal tube was placed, and anaesthesia was maintained in a closed system using a mixture of isoflurane (IsoFlo, Zoetis, Rotterdam, Netherlands), oxygen and air, as well as continuous-rate infusion of sufentanyl (1 µg/kg/h IV, Hameln Pharma, Hameln, Germany).

LEARNING POINTS/TAKE HOME MESSAGES

- Rathke's cleft cyst, although rare, should be included in the differential diagnosis of dogs with a pituitary mass.
- Magnetic resonance imaging and contrast-enhanced computed tomography are the imaging modalities of choice in patients suspected of Rathke's cleft cyst.
- Histology of a Rathke's cleft cyst is marked by the typical epithelial lining.

Perioperative antibiotic administration consisted of cefazoline (20 mg/kg IV, Kefzol, Eurocept International, Ankeveen, Netherlands) every 90 minutes. After midline incision, the soft palate was retracted laterally with a self-retaining Gelpi retractor. The mucoperiosteum was separated from the sphenoid bone and the hamular processes and the sphenoid ridge were identified to determine the position of the burr hole. Using a high-speed burr, the sphenoid bone was removed at the level of the pituitary fossa. After exposure of the sella turcica, the dura was coagulated using bipolar electrocautery and incised. On initial exploration of the sellar fossa, a firm pituitary mass was encountered. During attempts to remove the mass, it ruptured, and a grey, mucoid content was collected. After drainage of the apparent cyst, a remnant rim of tissue was removed separately. There was no evidence of a well-defined neurohypophysis. The mucoid content and the tissue sample were sent in for histopathology. A swab of the fossa was taken for bacterial culture, and the fossa was closed in a routine manner.[6]

After surgery, the patient recovered uneventfully and was monitored and treated in the intensive care unit according to hypophysectomy protocol.[7] Direct postoperative care consisted of amoxicillin-clavulanic acid (12.5 mg/kg IV every 12 hours, Augmentin, GlaxoSmithKline, Amersfoort, Netherlands), metoclopramide (0.15 mg/kg IV every 8 hours, Le Vet Beheer, Oudewater, Netherlands), omeprazole (1 mg/kg orally every 24 hours, Aurobindo Pharma, Baarn, Netherlands), carprofen (2 mg/kg IV every 12 hours, AST Beheer, Oudewater, Netherlands) and buprenorphine (10 µg/kg IV every 6 hours, Buprecare, Animalcare, York, UK). Hormone supplementation consisted of L-thyroxin (15 µg/kg orally every 12 hours, Forthyron, Eurovet Animal Health, Bladel, Netherlands), hydrocortisone acetate (1 mg/kg orally every 12 hours, Teva Nederland, Haarlem, Netherlands) and desmopressin (1 gtt every 8 hours, Minrin, Ferring, Hoofddorp, Netherlands). Preoperative adrenocorticotropic hormone (ACTH) was low (9 pg/ml) and remained low within hours after surgery (Figure 2). Postoperative bloodwork showed no abnormalities in potassium and sodium concentrations. The dog was discharged 3 days after surgery.

The dosage of hydrocortisone acetate was tapered in 3 weeks to a lifelong maintenance dose of 0.25 mg/kg once daily. L-thyroxin administration was continued and regularly checked with bloodwork, T4 remained within normal limits during the check-up period. Desmopressin was discontinued at 3 weeks after surgery. However, this resulted in clinical

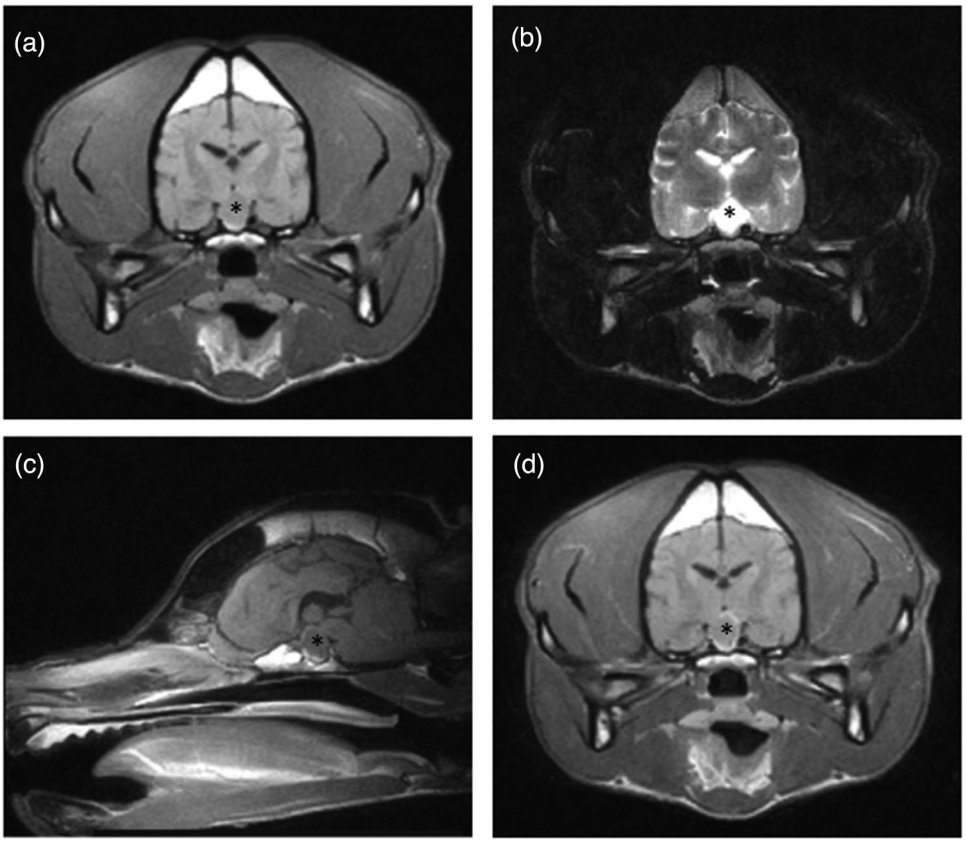

FIGURE 1 Magnetic resonance (MR) imaging in a 6-year-old Staffordshire bull terrier with a Rathke's cleft cyst of the pituitary gland. T1-weighted (a) and T2-weighted (b) transverse MR images of the skull showing isointensity (a) and hyperintensity (b) of the pituitary mass. Post-contrast T1-weighted image, showing rim enhancement of the pituitary mass on sagittal (c) and transverse (d) MR images. *Pituitary gland

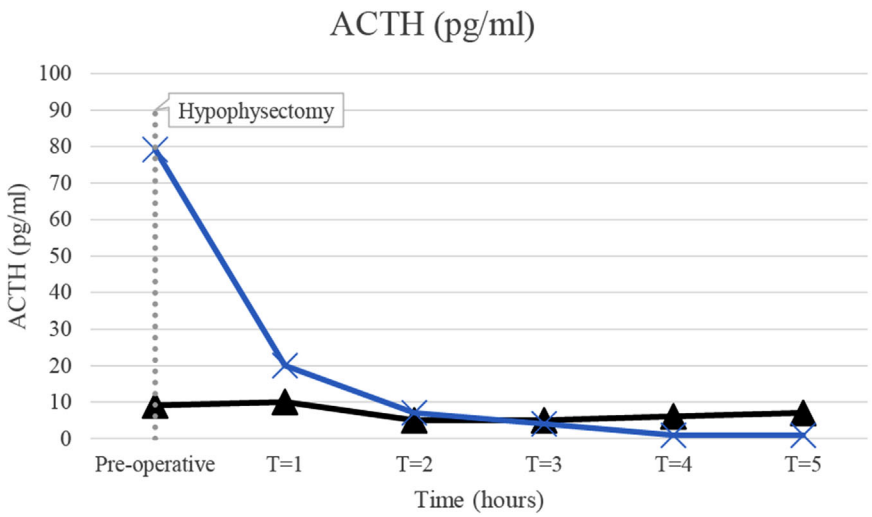

FIGURE 2 Plasma adrenocorticotropin hormone (ACTH) concentrations before and 1–5 hours after hypophysectomy (HX, moment marked with dashed line). The dog with the non-functional Rathke's cleft cyst of the present case report (▲) is compared with a typical patient with a corticotroph pituitary adenoma causing Cushing's disease that underwent HX (X)

signs of polyuria/polydipsia and therefore desmopressin was restarted and maintained during follow-up.

OUTCOME AND FOLLOW-UP

Histopathologic examination of the wall of the cyst and its content was performed. Haematoxylin and eosin (HE) staining of the tissue surrounding the cystic cavity showed fragmented adenohypophysis, with focally an epithelial lining of the largest fragment (Figure 3a). This lining consisted of a pseudo-stratified columnar epithelium that varied in height and contained cilia at the surface (Figure 3b). Within the epithelial lining, also scattered mucin-producing goblet cells were present. The underlying adenohypophysis contained a mixed population of acidophilic and basophilic cells, with fewer chromophobes. Immunohistochemistry was performed for ACTH, alpha melanocyte (α-MSH)-stimulating hormone and growth hormone as described previously.[8] The epithelial cells lining the cyst were negative on immunohistochemistry for these proteins. The content of the cystic mass consisted mainly of mucoid material that was positive in the periodic

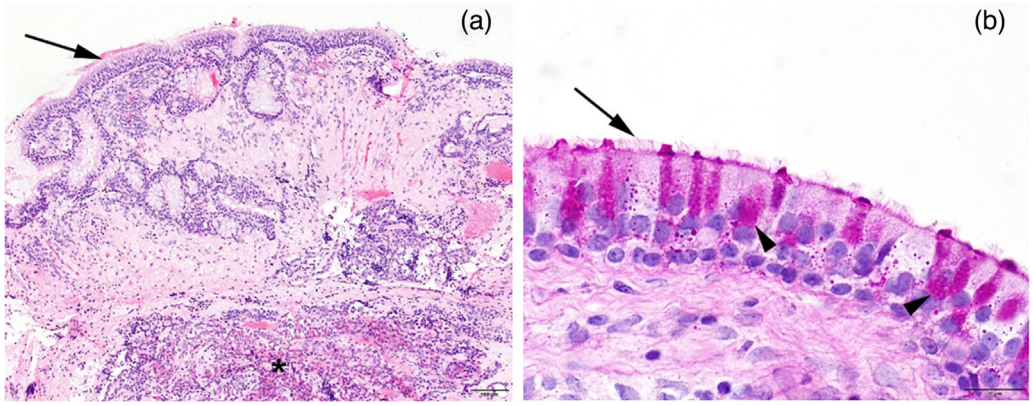

FIGURE 3 Histopathology of the pituitary cystic mass. (a) Part of the wall of the removed cyst lined with pseudo-stratified columnar epithelium (arrow). Note the presence of pre-existing adenohypophysis in the lower section (asterisk). Haematoxylin and eosin stain, obj. 10×. (b) Detailed view of the epithelial lining revealing ciliated epithelial cells (arrow) and mucin-producing goblet cells (arrowheads). Periodic acid–Schiff stain, obj. 80×

acid–Schiff stain with scattered small ribbons of epithelial cells, few neutrophils, macrophages and erythrocytes. These histopathological findings are consistent with a cyst originating from Rathke's pouch. No evidence of neoplasia was found. Bacterial culture of the sellar fossa was negative.

Urine cortisol creatinine ratios (UCCR) were determined 1 month after hypophysectomy and were 0.3 and 0.5×10^{-6} on 2 consecutive days. Reference value for UCCR is $<10 \times 10^{-6}$.[9]

Survival of the dog up until the time of writing this report was 1410 days. The dog was without recurrent clinical signs until 1136 days after surgery, when it developed a short episode of abnormal behaviour. This consisted of episodes of lethargy, not recognising the owner and nervous behaviour. The patient was presented at this time and diagnostics included bloodwork, urinalysis and computed tomography (CT). Bloodwork revealed no abnormalities in T4 value. UCCR was repeated and was 0.2×10^{-6}, consistent with absence of pituitary tissue. CT (Siemens Somatom Definition AS, Siemens Healthcare) of the skull (Figure 4) was performed and showed a bony defect in the basisphenoid bone compatible with the previously performed transsphenoidal hypophysectomy. Contrast enhancement with iobitridol (2 ml/kg, Xenetix 350 mg I/ml, Guerbet, Villepinte, France) showed an empty sella and no evidence of a pituitary adenohypophysis or cystic mass except for a small (<2.5 mm) contrast enhancing structure, suggestive of blood vessels and/or scar tissue in this area. The remaining bony and soft tissue structures were unremarkable. Clinical signs resolved without further intervention within a few days.

DISCUSSION

The present case reports the successful treatment of RCC by hypophysectomy in a Staffordshire bull terrier. Since 1993, when hypophysectomy was reintroduced in Utrecht for treatment of corticotroph adenomas causing Cushing's disease, other pituitary disorders that were eligible for hypophysectomy were also encountered and/or described in the veterinary literature.[10,11] Besides corticotroph adenomas, the list of pituitary disorders that may or have been treated by hypophysectomy include non-functional adenomas, hypophysitis,[12] prolactinoma,[13] somatotroph adenoma[14] and pituitary apoplexy.[5]

Pituitary disorders in dogs can largely be classified as functionally endocrine active or inactive disorders. The pituitary corticotroph adenoma is the most common endocrine active disorder leading to excess secretion of ACTH (Cushing's disease) and hypercortisolism (Cushing's syndrome). Other endocrine active pituitary neoplasms include prolactinoma[13] (leading to hyperprolactinemia) and somatotroph adenoma[15] (leading to hypersomatotropism). Treatment success in endocrine active pituitary adenomas can be evaluated by measuring the systemic level of the target hormones. Endocrine inactive pituitary disorders do not lead to an endocrine syndrome and usually cause clinical neurological signs due to the mass effect. These masses may also lead to hypopituitarism or diabetes insipidus, but this is usually not immediately clinically evident.

Pituitary imaging is indicated following a preliminary diagnosis of a pituitary disorder based on the patient's history, physical examination and laboratory findings. A pituitary lesion is suspected if there is a hyper- or hyposecretion of one or more of the pituitary hormones. High-field MRI is the preferred pituitary imaging modality in humans for anatomic definition of the lesions, the pituitary and its surrounding structures, unless there are contraindications such as certain (metallic) implants. In these patients, CT is used with pre- and post-contrast images for evaluation of the pituitary gland.[16] In veterinary medicine, MRI and CT are both used for pituitary gland imaging, although CT is used more often, as this method is slightly more cost-effective, faster, and more readily available. High-field MRI is preferred over low-field MRI, to obtain more accurate preoperative measurements, as spatial resolution is directly related to magnetic field strength. In patients with hypercortisolism, CT facilitates imaging of the thorax for pulmonary metastasis or ventilatory issues, and assessment of the abdominal cavity to evaluate the adrenal glands.[17] Also, when pituitary surgery is indicated, contrast-enhanced CT provides excellent visualisation of the pituitary tumour in relation to the bony landmarks for the transsphenoidal approach. In cases of neurological complaints of central origin, when a pituitary lesion is not primarily suspected, such as in the patient in this case report, MRI is the imaging modality of choice. Whereas CT offers much better overview of skull bone details, MRI gives more information about the soft tissues within the cranium. For instance, acute haemorrhage of

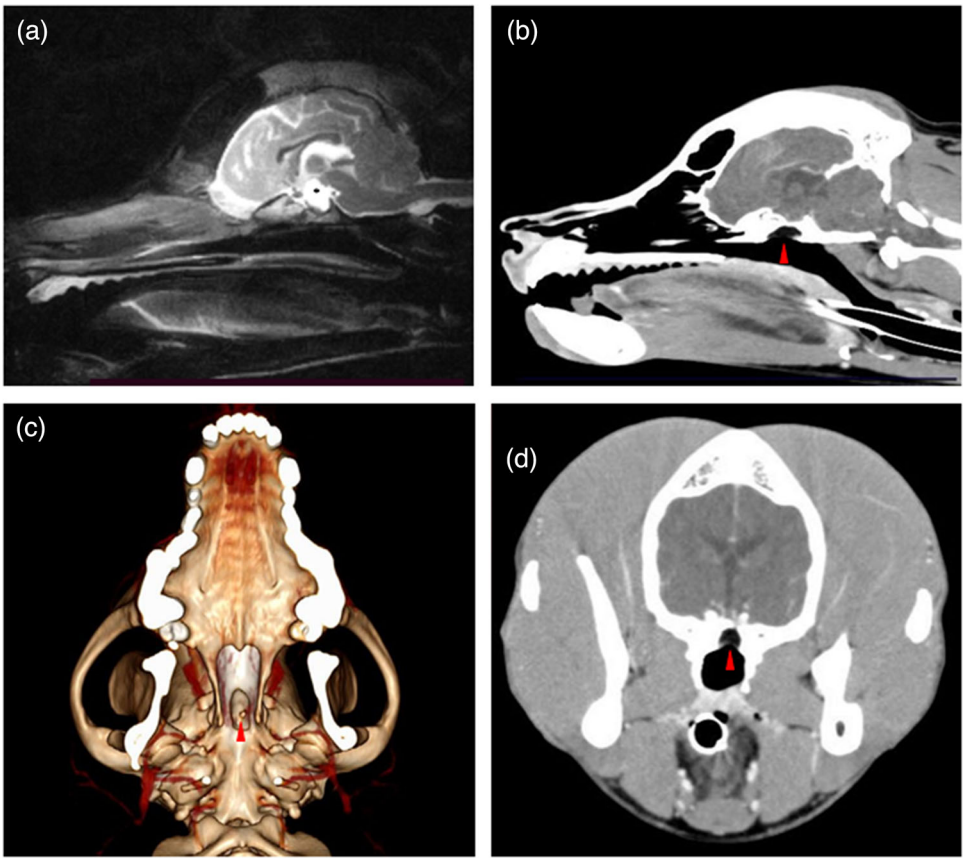

FIGURE 4 Preoperative sagittal T2-weighted magnetic resonance imaging (MRI) (a) and 3-year postoperative sagittal contrast-enhanced computed tomography (CT) (b). Rathke's cleft cyst (*) visible on MRI (a) is absent on CT (b,d). Rathke's cleft cyst was removed by the transsphenoidal approach indicated by the red arrowhead on the sagittal CT image (b), the three-dimensional bone reconstruction of the postoperative CT showing a ventral view of the skull (c) and on the transverse CT image (d)

a pituitary mass (e.g., pituitary apoplexy) can be visualised by T2* sequences. Other changes such as peritumoral oedema can be appreciated on MRI as well. In the present case, low-field MRI was performed at another institution and the MRI resolution was considered of sufficient quality to assess both the pituitary lesion and the bone surgical landmarks; however, this remains at the discretion of the performing pituitary surgeon.

Success after pituitary surgery is evaluated by following resolution of clinical signs, measurements of blood and hormone values and pituitary imaging. In patients with an ACTH-producing adenoma, hormone measurements such as blood ACTH levels and UCCRs can accurately document the successful removal of the adenoma, and thus confirm remission of hypercortisolism. However, if there is no overproduction of hormones before surgery, and especially when there is hypopituitarism due to a pituitary mass lesion, it is difficult to assess with certainty that there is no recurrence of the pituitary mass after surgery by measuring hormone values only. Low UCCRs are an indication of remission of a corticotroph adenoma, but cannot give certainty about complete resection of a pituitary mass in a case like an RCC. Basal plasma ACTH concentration in the present case was already low before surgery and remained low after surgery. In RCC, preoperative low plasma ACTH concentration may have been the result of hyposecretion of the remnant adenohypophyseal tissue, but no baseline UCCR was available before surgery to measure functional ACTH secretion from remnant pituitary tissue. In comparison, high basal plasma ACTH in a case of Cushing's disease before hypophysectomy should decrease to near zero values shortly after successful hypophysectomy (due to the short half-life of endogenous plasma ACTH), as can be seen in Figure 2.[18,19] However, very low UCCRs are consistent with complete removal of the corticotroph adenoma but are not evidence of complete removal of the pituitary gland.

In pituitary disorders that have no endocrine function, like RCC, hormonal assessment to evaluate surgical success is not conclusive, especially when the pituitary mass has caused pituitary insufficiency. Apart from resolution of neurological signs, pituitary imaging is required to evaluate surgical success. Postoperative imaging at several months or years after hypophysectomy can be performed using high-field MRI or CT. Compared to MRI, CT is more informative as it allows for visualisation of the transsphenoidal approach through the skull base. In addition, IV contrast enhancement facilitates in the evaluation of the soft tissues within the pituitary fossa. The presence of a contrast enhancing area within the fossa can be compatible with regrowth of the pituitary mass or cyst but can also be evidence of neovascularisation from the hypothalamus.[20] A study in rats showed that, after hypophysectomy, the fibres of the magnocellular neurons regenerate, resulting in the establishment of new neurohemal connections. On imaging, this is seen as a posterior pituitary-like structure. The visualisation of a small hyperintense structure during contrast-enhanced CT at 3 years after surgery in our case is compatible with neovascularisation from the

hypothalamus.[19,20] Although a posterior pituitary-like structure is able to maintain a basal release of vasopressin from the hypothalamus, an adequate response to hyperosmotic stimulus may be insufficient and necessitates continued supplementation with desmopressin, which was most likely the situation in our case.[15] Whether dependency of desmopressin is permanent in this dog remains to be seen; however, the chances of full recovery become less as time passes.

In conclusion, RCC in the dog should be added to the list of pituitary disorders causing a mass effect. In this case, it was successfully treated by transsphenoidal hypophysectomy with subsequent lifelong hormone substitution with cortisone, thyroxine and desmopressin. However, the correlation between the pituitary cyst and clinical signs remains unclear as clinical signs resolved before surgery, and another episode of abnormal behaviour occurred later in absence of the cystic mass.

CONFLICTS OF INTEREST

The authors declare they have no conflicts of interest.

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This research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

ETHICS STATEMENT

The authors confirm that the ethical policies of the journal, as noted on the journal's author guidelines page, have been adhered to. No ethical approval was required as the patient was client-owned. We demonstrated a high standard (best practice) of veterinary care and had informed client consent.

AUTHOR CONTRIBUTIONS

All authors contributed to conception of study, study design, acquisition of data and data analysis and interpretation. All authors also drafted, revised and approved the submitted manuscript.

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