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A Case Report of Symptomatic Rathke's Cleft Cyst

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ABSTRACT

Background: Rathke's cleft cyst (RCC) is a not-so-common pituitary developmental disorder, it is a benign growth found on the pituitary gland in the brain, specifically a fluid-filled cyst in the posterior portion of the anterior pituitary gland. Symptomatic cysts are very rare. With only few cases have been documented in the literature. **Aim of the work:** this study aimed to assess the effect of corticosteroids on patients with symptomatic Rathke's cleft cyst. **Methodology:** we reported a rare case of a 21 years old male patient, who was referred from a primary health center to the endocrine clinic with the complain of decreased visual acuity, blurred vision, and headache. On examination, there were no signs of puberty, no secondary sexual hair, truncal and generalized obesity. The fundus examination showed bilateral temporal pallor, with pale disc and papilledema. On the basis of symptoms and examination of the patient we suspected a lesion in the brain obstructing the optic pathway and therefore suggested MRI. **Conclusion:** this case led us to conclude that Rathke's cleft cysts should be considered in the differential diagnosis of patients who present with adrenal insufficiency and that the cysts can be reduced by glucocorticoids.

Keywords: pituitary adenoma; Rathke's cleft cyst; magnetic resonance imaging.

INTRODUCTION

Rathke's cleft cyst is a benign congenital fluid filled cyst that is found between the anterior and posterior parts of the pituitary gland [(1)]. Symptomatic Rathke's cleft cyst is an extremely rare condition with only a few cases published in review literatures. Most of the cysts are usually asymptomatic and found only on autopsy findings. Symptomatic cysts can trigger hormonal symptoms, visual disturbances and headache [(2)]. Here, we reported a rare case of a patient with a symptomatic rathke's cleft cyst.

PATIENTS AND METHODS

A 21 years old male patient was referred from his school to the Primary Health Center with decreased visual acuity and blurred vision for two months. The family physician then referred the patient to the hospital for further investigations. Patient came to the endocrine clinic and more detailed history revealed that the patient has frontotemporoparietal headache, throbbing in nature intermittent and not relieved on taking medication. He has mild bilateral papilledema and visual filed defect. The patient was admitted to the male medical ward for more investigation.

The study was done after approval of ethical board of Umm Al Qura university.

RESULTS

Examination showed that the patient was conscious, alert, oriented, afebrile, pulse rate 57/min, blood pressure 110\75 mm Hg, height 150 CM and weight 70 kg. Patient's BMI was 31.1. There were no signs of puberty, no secondary sexual hair, truncal and generalized obesity. The cardiovascular system and the respiratory system examination were within normal limits. The abdomen was soft, no lump or organomegaly, bowel sounds were present. Central nervous system examination showed no neck rigidity; pupil bilateral reacting to light and plantar bilateral flexion. Examination of vision showed that the patient had decreased peripheral visual field, the fundus examination showed bilateral temporal pallor, with pale disc and papilledema. On the basis of symptoms and examination of the patient we suspected a lesion in the brain obstructing the optic pathway and therefore suggested MRI.

MRI brain study revealed enlargement of the sella with huge sellar and suprasellar region being of high signal intensity in T1\FLAIR, isointense in T2w1 part from some heterogenicity at the poster inferior aspect, the suprasellar component is measuring 2.72 * 2.02 CM and the sellar component is measuring 2.73*2.5CM, the whole mass was measuring 3.83CM in craniocaudal dimension, no evidence of hemorrhagic component in gradient echo, no evidence of fat suppression on fat suppresses sequences, no evidence of definite enhancement after IV contrast. This mass was exerting mass effect on the optic chiasm with superior displacement as well as on the cavernous sinus with lateral displacement of the internal carotid artery, however with normal caliber and signal intensity, inferior mass effect on sphenoid sinus and clivus. The pituitary gland and stalk were not demonstrated, compressed by the above mentioned mass. The final impression: sellar and suprasellar mass as above described with MRI findings suggesting retake's cleft cyst.

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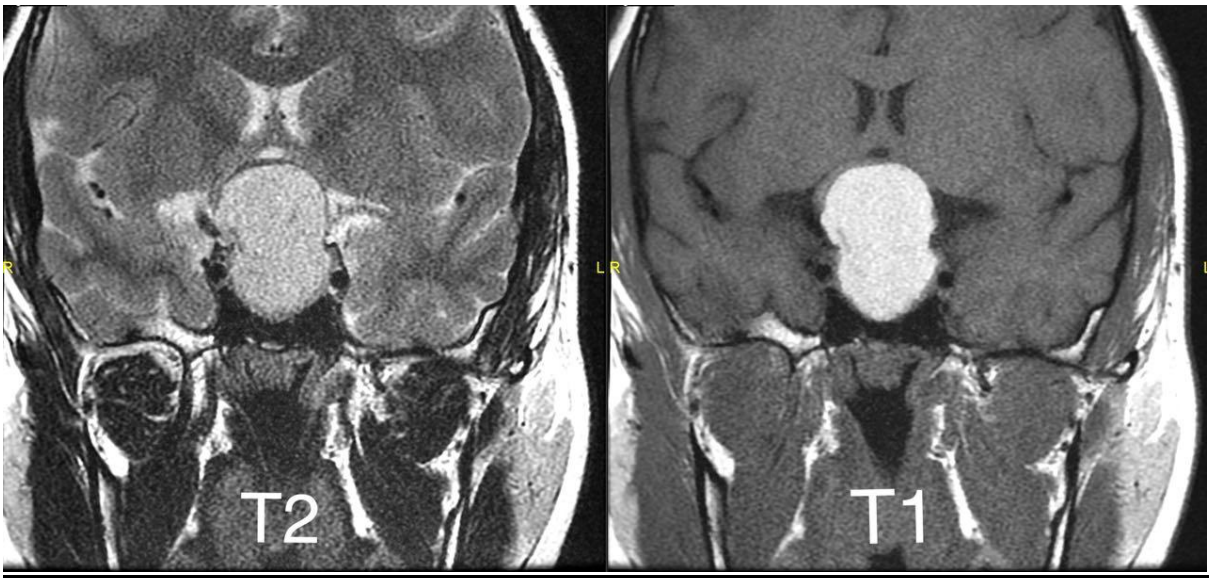

Figure 1: coronal section of brain.

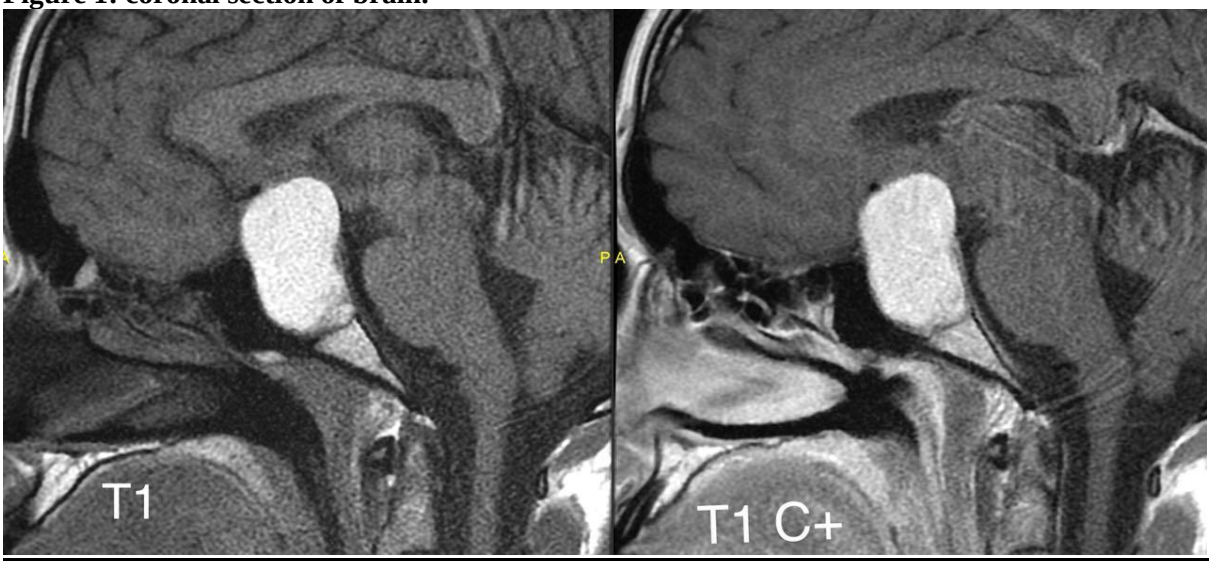

Figure 2: Sagittal section of brain.

Blood investigation showed the following: haemoglobin 11.81, white blood cells 5.944, red blood cells 5.02, CRE 1, Glucose Random 87, Bilirubin (Total) 0.6, Urea 13, Sodium 138, Potassium 3.6, Cortisol 66.73, Prolactin 344.1, Free T4 13.57, Free T3 4.56, Tsh 4.41, Fsh 2.45, Lh 0.321

By radiological investigations the ultrasonography of the scrotum showed the left testis to be slightly decrease in size (right testis measures around 3.3*1.5*2.5 cm – left testis measures 2.8*1.4*2.5cm). The echo texture of the testis appears to be homogenous with respected vascularization, no notable focal lesions seen or suspicious calcification- no notable hydrocele or abnormal dilated vein. Chest x-ray and ECG were normal.The echocardiography revealed a mild left ventricular dysfunction, dilated left atrial and pulmonary hypertension

Management

We started dexamethasone injection 20 mg/mL TID for 3 days during the hospital stay, then the patient was discharged on dexamethasone tablet 4 MG BID, Thyroxin 50 MCG OD, Cabergoline tablet 1 MG twice a week. After 7 months of follow-up the Dexamethasone improved the symptoms, and the cyst spontaneously shrank.

The patient's visual deficit remained unchanged, whereas the results of the hormonal assays were normal. This case led us to conclude that Rathke's

cleft cysts should be considered in the differential diagnosis of patients who present with adrenal insufficiency and that the cysts can be reduced by glucocorticoids.

DISCUSSION

Voelker *et al.* [3] stated that the most common theory about the origin of RCCs is that the cysts are derived from true remnants of the embryological Rathke's pouch. Symptomatic RCCs vary in presentation. In a study of 11 symptomatic patients by **Rao *et al.*** [3] eight patients initially had visual symptoms. In a study of **Eguchi *et al.*** [4] visual symptoms occurred in 47% of patients. Signs and symptoms included reduced visual acuity, optic atrophy, visual field defects and a chiasmatic syndrome.When a non-enhancing cyst-like structure is demonstrated on imaging, the possibility of a coexisting Rathke's cleft cyst should be considered.

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