Case Report

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Magnetic Resonance Imaging Features and Management of a Large Symptomatic Rathke Cleft Cyst: A Case Report

Anaesthesia Section

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ABSTRACT

The Rathke pouch is a permanent remnant of the embryonic buccal cavity that, in normal conditions, disappears following the establishment of the anterior pituitary. Pathogenesis of cystic sellar and suprasellar lesions, referred to as pars intermedia or Rathke cleft cysts, results from failure of pars intermedia closure. The cysts are usually small, symptomless, and incidentally discovered, with diagnosis strongly dependent on magnetic resonance imaging. We report a case of a 51-year-old postmenopausal female patient with a six-day history of headache, weakness, and progressive worsening of vision for two years. She developed leukocytosis on laboratory findings, while endocrine parameters were normal. Magnetic resonance imaging showed a massive extra-axial non-enhancing cystic lesion in the sellar, suprasellar, and parasellar region that was hypointense on T1-weighted images and hyperintense on T2-weighted and fluid-attenuated inversion recovery sequences with mass effect upon the optic chiasm, hypothalamus, and thalamus. On these radiological findings, Rathke cleft cyst was diagnosed. The patient was admitted for left pterional craniotomy with complete surgical excision of the cyst. Post-operative course was uneventful, and she was discharged on the fourth day on a regimen of regular follow-up. This case emphasises the clinical importance of detecting symptomatic Rathke cleft cysts, which, while rare, can produce progressive loss of vision and neurological findings, if considerable. It emphasises the importance of thorough radiological evaluation in diagnosis, defines surgical treatment as the therapy of choice for symptomatic lesions, and illustrates a favourable outcome after prompt intervention.

Keywords: Headache, Hypopituitarism, Pituitary neoplasms, Rathke pouch, Sella turcica

CASE PRESENTATION

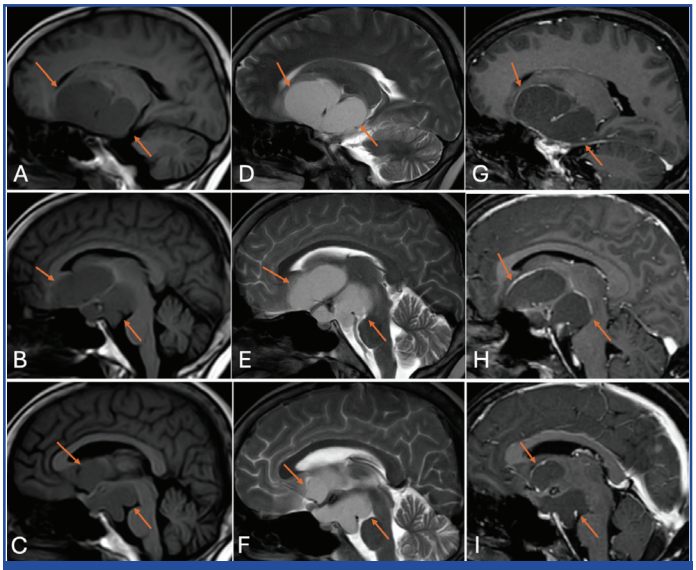
A 51-year-old postmenopausal female came to the medicine outpatient department with complaints of headache, generalised weakness, and fever for the past 6 days. The headache was described as dull aching in nature, of moderate intensity, continuous in character, without any clear aggravating or relieving factors. It was predominantly localised to the frontal region and did not show significant variation with posture or time of day. She did not report associated nausea, vomiting, drowsiness, or altered mental status. She complained of progressive diminishing of vision for the past 2 years. She was the mother of a single child, delivered vaginally, with no significant family history and no known systemic illness. On physical examination, she was febrile with an increased pulse rate. The patient's growth was regular and consistent with the mid-parental height. A Central Nervous System (CNS) examination was done, and it revealed an absence of neck rigidity. Higher mental functions, cranial nerves (except vision-related findings), motor system, sensory system, and cerebellar examination were within normal limits. There were no focal neurological deficits. On ophthalmic examination, the peripheral vision was reduced with bilateral pupils reacting to light. Fundoscopy did not reveal papilloedema or retinal haemorrhages. The laboratory parameters showed an increase in the total leucocyte count with no endocrine hormone abnormalities, as shown in [Table/Fig-1].

Magnetic Resonance Imaging (MRI) of the brain with contrast revealed an extra-axial non-enhancing altered signal intensity lesion in the sellar, suprasellar, and parasellar region appearing hypo intense on T1 weighted imaging (T1WI), hyper intense on T2 weighted imaging (T2WI) and Fluid-Attenuated Inversion Recovery (FLAIR) with no diffusion restriction on Diffusion-Weighted Imaging (DWI) causing effacement of the suprasellar cistern and mass effect on the brainstem, optic chiasm, hypothalamus, and thalamus with a midline shift towards the right as shown in [Table/Fig-2] and [Table/Fig-3].

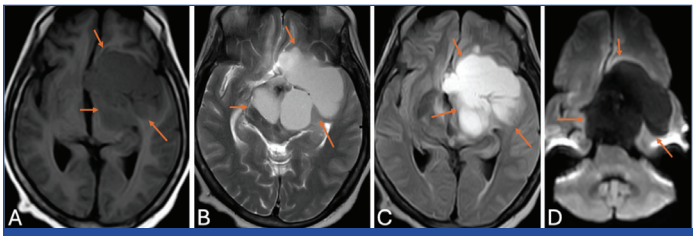
The lesion appeared lobulated in shape with a smooth outline. No intracystic nodule, which is often considered a characteristic finding of Rathke cleft cyst, was observed in this case. The lesion showed no post-contrast enhancement, consistent with a cystic nature, and the compression effects were evident on the adjacent optic pathways, hypothalamus, and thalamus. A diagnosis of Rathke's cleft cyst was made. Based on radiological features, differentials considered at the time of diagnosis included craniopharyngioma, arachnoid cyst, and epidermoid cyst; however, the absence of

Investigation (Units)	Patient Value	Reference Range
Haemoglobin (g/dL)	10.4	12.1–15.2
MCHC (g/dL)	32.8	32–36
MCV (fL)	86.1	80–100
MCH (pg)	29.1	27–33
Total RBC count (cells/µL)	4.7	4.3–6.2
Total WBC count (cells/µL)	19,600	4,000–11,000
Total Platelet count (cells/µL)	1,57,000	1,50,000–4,00,000
FSH (mIU/mL)	56	25.8–134.8
LH (IU/L)	40.5	14.2–52.3
Cortisol (µg/dL)	5.3	5–25
T3 (nmol/L)	1.1	0.9–2.8
T4 (µg/dL)	5.9	5–12
TSH (mIU/L)	0.5	0.4–4
INR (Ratio)	1.0	<1.2
Urea (mg/dL)	18	5–20
Creatinine (mg/dL)	0.8	0.6–1.1

[Table/Fig-1]: Laboratory parameters of the patient with normal reference range
MCHC: Mean Corpuscular Haemoglobin Concentration; MCV: Mean Corpuscular Volume; MCH: Mean Corpuscular Haemoglobin; RBC: Red Blood Cell; WBC: White Blood Cell; FSH: Follicle Stimulating Hormone; LH: Luteinising Hormone; T3: Triiodothyronine; T4: Thyroxine; TSH: Thyroid Stimulating Hormone; INR: International Normalised Ratio



[Table/Fig-2]: Sequential magnetic resonance imaging sagittal sections of brain T1-weighted imaging (A, B, C), T2 weighted imaging (D, E, F), and T1-weighted contrast sequence (G, H, I) showing extra-axial non-enhancing lesion in the sellar, suprasellar and parasellar region appearing hypointense on T1WI and hyperintense on T2WI causing effacement of suprasellar cistern suggesting Rathke's cleft cyst (orange arrows).



[Table/Fig-3]: Magnetic resonance imaging axial sections of brain T1-weighted imaging (A), T2-weighted imaging (B), fluid-attenuated inversion recovery sequence (C) and diffusion-weighted imaging (D) showing extra-axial lesion in the sellar, suprasellar and parasellar region appearing hypointense on T1WI and hyperintense on T2WI and FLAIR with no diffusion restriction on DWI causing effacement of suprasellar cistern suggesting Rathke's cleft cyst (orange arrows).

calcifications, solid enhancing components, and diffusion restriction helped to favor Rathke cleft cyst. The endoscopic endonasal approach to drain the cyst, although minimally invasive, was not considered an option in this case because of significant suprasellar extension. Hence, a left pterional craniotomy was performed, and the cystic lesion was excised completely. The patient had an uneventful postoperative course and was discharged 4 days after the surgery with advice to follow up after 7 days for suture removal or earlier if any complaints developed. Postoperatively, the patient reported partial improvement in vision, with stabilisation of peripheral visual field defects, while endocrine function remained intact without any new hormonal deficits. Follow-up MRI is planned to monitor for recurrence, which remains a possibility in a subset of cases. Histopathological examination of the excised specimen confirmed Rathke cleft cyst lined by cuboidal epithelium with mucoid content, consistent with its developmental origin. Prognosis is generally favorable after complete excision, with low recurrence rates when adequate cyst wall removal is achieved.

DISCUSSION

The pituitary gland begins to develop in the 4th week of foetal life. The neurohypophysis, or the posterior part, develops from the neuroectoderm and the adenohypophysis, or the anterior part, develops from the buccal mucosa (oral ectoderm) of the embryo. Buccal ectoderm gives a superior evagination towards neuroectoderm, called the Rathke pouch, which is developed from the hypophyseal placode that is formed by the thickening of cells of the oral ectoderm. Simultaneously, a downward evagination is provided by neuroectoderm, which forms the posterior lobe of the pituitary gland. By six to eight weeks, the Rathke pouch gets separated and compresses at the base of the bilobed structure. Incomplete closure of the Rathke pouch leads to the development of the pars intermedia cysts, or Rathke cleft cysts [1,2].

These are mostly incidentally detected non-neoplastic cystic lesions found in the suprasellar or sellar region. The peak incidence of detection is in the 3rd to 5th decade, with a female predominance. The Rathke cleft cysts do not cause any symptoms when small, but they might cause visual disturbances, headaches, and pituitary dysfunction in large sizes [3].

The diagnosis is made on imaging, with MRI being the imaging investigation of choice [4].

Lateral skull radiographs are of little use, which might provide a nonspecific sign of sellar enlargement. A Computed Tomography (CT) scan shows the presence of a unicameral, low attenuated, homogenous, non-calcified lesion in the midline with non-enhancing walls. Occasionally, the cyst can be of mixed attenuation with small curvilinear wall calcifications and wall enhancement [5]. MRI shows a unicameral lesion in the sellar region, which appears hypointense to the surrounding fluid on T2WI and hypointense on T1WI. The cyst may show high signal intensity on T1WI in case of high protein content of the cyst [6].

The treatment depends on the size of the cystic lesion. No treatment is required for asymptomatic small-sized lesions, and they are advised to have a regular MRI follow-up. Surgical resection is necessary for the symptomatic large-sized cysts. The location of vessels plays a valuable role in deciding the site for surgical resection [7,8].

The developmental differential diagnosis of the Rathke cleft cyst includes epidermoid cyst, which shows diffusion restriction on DWI; arachnoid cysts and ependymal cysts, which follow Cerebrospinal Fluid (CSF) intensity signals on all MRI sequences; dermoid cyst, which usually has a fatty signal and solid components; transsphenoidal meningocoele, which is characterised by a bony defect, and internal porencephalic cyst, which shows communication with the ventricle. The neoplastic differentials include pilocytic astrocytoma and pilomyxoid astrocytoma, which have a solid component within and show post-contrast enhancement [9-12]. To contextualise the findings of our patient, we compared her presentation, imaging features, and management outcomes with other published cases of Rathke's cleft cyst, as summarised in [Table/Fig-4] [13-15].

Case	Clinical Features	Examination & Endocrine	MRI Findings	Management & Outcome
Present case (51/F)	Headache (dull, continuous, 6 days), weakness, fever; progressive vision loss (2 years); blurring with bright lights (6 months)	Peripheral vision reduced, pupils reactive, no papilledema; no focal deficits; baseline hormones normal	Sellar–suprasellar–parasellar cyst; T1 hypo intense, T2/FLAIR hyper intense; non-enhancing, no DWI restriction; no intracystic nodule; smooth outline; mass effect on optic chiasm, hypothalamus, thalamus; midline shift	Left pterional craniotomy, cyst excision; Rathke's Cleft Cyst (RCC) confirmed histologically (cuboidal epithelium, mucoid content); uneventful recovery, partial visual improvement, endocrine intact; follow-up MRI advised
Naik VD and Thakore NR [13] (40/F)	Throbbing frontotemporoparietal headache (3 months, refractory), blurring of vision, giddiness with lights, amenorrhoea (5 months)	Decreased peripheral visual fields, perimetry constriction, hyperprolactinaemia, central hypothyroidism, hypocortisolism	Well-defined sellar cyst with suprasellar extension; T1 isointense, T2 hyper intense; rim enhancement; optic chiasm compression	Medical management: hydrocortisone, cabergoline, thyroxine, calcium/vit D; no surgery reported; outcome not detailed

Yang C et al., [14] (62/M)	Severe pulsatile frontal headache (6 months, worsened), visual acuity deficit (R eye), polyuria, polydipsia, slight consciousness disturbance	Severe hyponatremia, panhypopituitarism, hyperprolactinaemia; no CNS infection signs	Sellar–suprasellar cyst (~18–20 mm); iso-/hypo intense T1, iso-/hyper intense T2; rim enhancement mimicking pituitary abscess	Trans-sphenoidal surgery; yellow-brown material drained, no pus; histology confirmed RCC (cuboidal epithelium, no inflammatory cells); hormone replacement (hydrocortisone, levothyroxine, desmopressin); recovery with improved headache & vision; continued desmopressin, long-term hormone replacement; no recurrence at 12 months
Chaudhary M et al., [15] (25/F)	Headache, bitemporal hemianopia, progressive visual disturbance	Bitemporal field defect, baseline pituitary hormones normal	Sellar cyst with suprasellar extension; T1 hypo intense, T2 hyper intense; no enhancement; smooth outline; optic chiasm compression	Conservative management with serial imaging and endocrine follow-up; spontaneous regression at 18 months; no recurrence at 2 years; vision improved

[Table/Fig-4]: Comparative summary of clinical features, examination findings, radiological characteristics, and management outcomes of Rathke's cleft cyst cases

CONCLUSION(S)

Rathke cleft cysts are congenital developmental lesions of the sellar and suprasellar region, which are asymptomatic until they are large. This case demonstrates an unusually enlarged Rathke pouch cyst involving the sellar, suprasellar and parasellar region with a mass effect on the adjacent structures.

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