

Sellar Colloid Cyst—An Anatomically Aberrant and Diagnostically Challenging Entity: Report of a Rare Case with Literature Review

Syed Asmat Ali¹ Kavita Gaur^{2,3} Arvind Kumar Srivastava⁴ Ravindra Kumar Saran²

¹Department of Neurosurgery, Jawaharlal Nehru Medical College, Aligarh Muslim University, Aligarh, Uttar Pradesh, India

²Department of Pathology, GB Pant Institute of Postgraduate Medical Education and Research, New Delhi, India

³Department of Pathology, Lady Hardinge Medical College, New Delhi

⁴Department of Neurosurgery, GB Pant Institute of Postgraduate Medical Education and Research and associated Maulana Azad Medical College, New Delhi, India

Address for correspondence Kavita Gaur, MD, DNB, Department of Pathology, Lady Hardinge Medical College, New Delhi 110001, India (e-mail: kavgaaur@gmail.com).

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Abstract

Keywords

- colloid cyst
- pituitary
- magnetic resonance imaging

The sellar colloid cyst is a rare entity anatomically occurring at the intermediate lobe of the pituitary gland. Clinically a cystic sellar lesion with pressure effects usually evokes the suspicion of a pituitary adenoma. We present the case of a middle-aged woman presenting with visual diminution and bilateral optic atrophy, caused by a large sellar lesion, variably intense on magnetic resonance imaging (MRI). The subsequent histologic diagnosis of a colloid cyst was unexpected. This report highlights the subtle intraoperative and diagnostic features key to diagnosing this rarity. We also discuss a practical differential diagnostic approach relevant to the practicing surgeon and review the existing literature.

Introduction

Colloid cyst (CC) is an uncommon non-neoplastic lesion characteristically situated at the third ventricle.¹ The occurrence of this entity at the sella is rare and comprises less than 0.5% of resected sellar lesions.² Diagnostic dilemma with pituitary adenoma and other cystic sellar-suprasellar lesions is likely. Multidisciplinary correlation is essential to nail the correct diagnosis. We present the case of a 50-year-old woman presenting with headache and visual diminution whose imaging findings suggested a pituitary adenoma. Subsequent histopathology revealed the presence of a CC contrary to our preoperative assumption of a pituitary neoplasm. We also discuss possible differential diagnoses based on clinical imaging and pathologic assessment.

Case Report

A 50-year-old woman presented with episodic headache for 3 years. This was moderate in intensity, initially bitemporal gradually becoming holocranial, and relieved with analgesics. She also developed painless progressive diminution of vision in both the eyes for the previous 6 months (especially for the outer halves of both visual fields). There was no history of fever, vomiting, seizures, trauma, or loss of consciousness. No significant past history was elicited. Ocular examination revealed a visual acuity of 6/24 in the left eye and 6/18 in the right eye, with bitemporal hemianopia and bilateral optic atrophy on funduscopy. Neurologic examination was unremarkable. Contrast-enhancing computed tomography (CECT) scan of the head revealed

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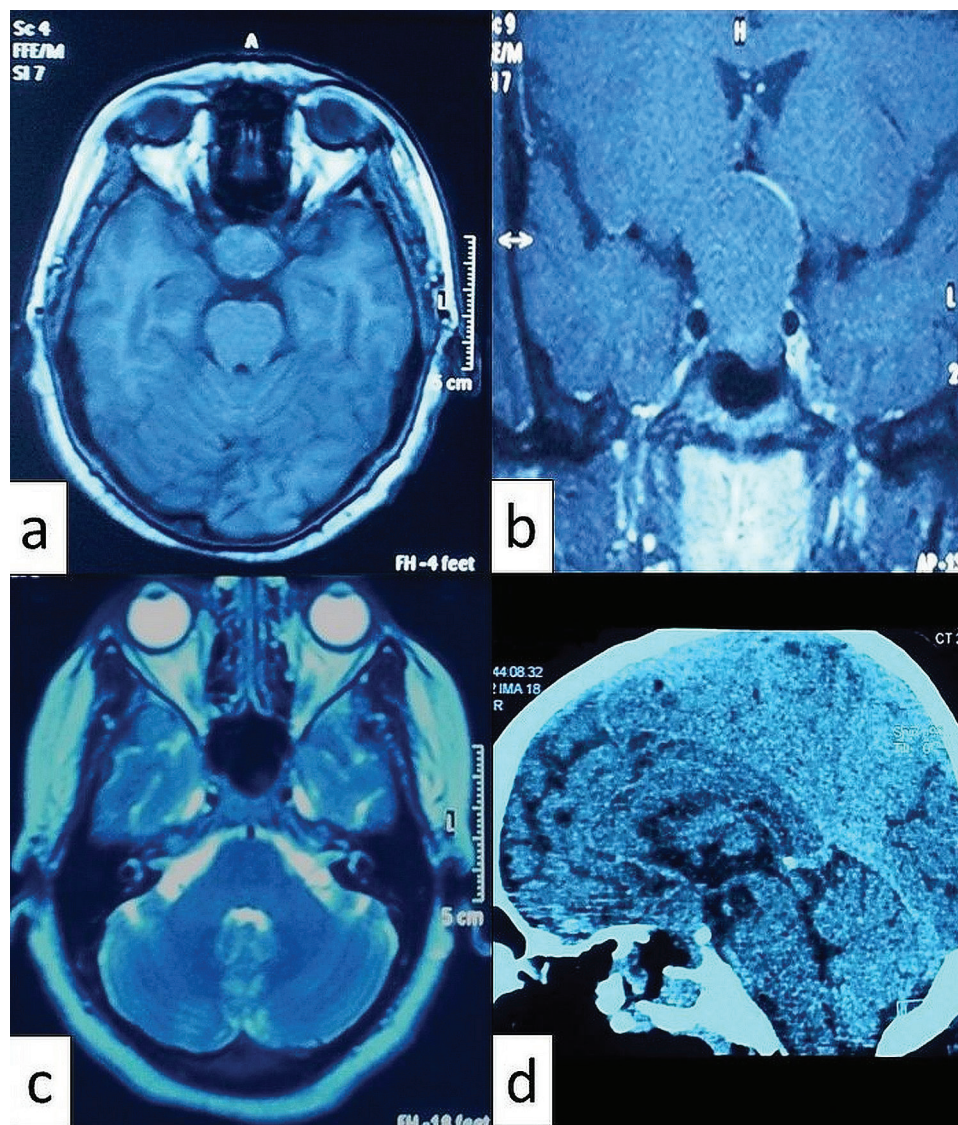


Fig. 1 T1 axial image of MRI of the brain showing an isointense circular lesion abutting the surrounding cistern compressing the optic chiasma. **(b)** T1 contrast (coronal view) of MRI of the brain showing nonenhancing pituitary mass abutting the suprasellar cistern and third ventricle with slight pseudocapsule displaying delayed contrast enhancement. **(c)** T2-weighted image showing isointense well-circumscribed lesion in sellar region. **(d)** Computed tomography of the brain (sagittal view) showing absence of the lesion at 1 year of follow-up.

a homogeneously enhancing hyperdense mass in the sella causing sellar expansion, extending into the suprasellar region. Magnetic resonance imaging (MRI) showed a smooth margined, bilobed mass lesion $3.2 \times 3.1 \times 2.6$ cm in dimension arising from the sella with suprasellar extension compressing the optic chiasm and involving the infundibulum. The lesion was isointense on T1-weighted image (T1WI) and iso-hyperintense on T2-weighted image (T2WI) (**Fig. 1a–c**), hyperintense on fluid attenuation inversion recovery (FLAIR) sequence, and nonenhancing with contrast. Radiologic impression suggested a pituitary macroadenoma. Endocrine evaluation (thyroid function tests, serum cortisol, prolactin, growth hormone [GH], luteinizing hormone [LH], follicle-stimulating hormone [FSH], adrenocorticotrophic hormone [ACTH], insulin-like growth factor 1 [IGF1]) was normal. Tests for assessing the functional status of the posterior pituitary to rule out diabetes insipidus (including fluid deprivation test)

were performed and were unremarkable. A preoperative working diagnosis of nonsecretory pituitary adenoma was made. The lesion was excised via a transnasal, transsphenoidal approach. Perioperatively, the lesion was grayish and contained mucoid material. Histopathologic examination revealed amorphous eosinophilic periodic acid–Schiff (PAS)–positive material with focal fibrous tissue compatible with a CC (**Fig. 2**). Postoperative period was uneventful. Follow-up CT 1 year later revealed complete absence of the lesion and the presence of shrunken pituitary gland at the sella (**Fig. 1d**). The patient is currently doing well and is free of neurologic or endocrinologic deficits.

Discussion

Cystic sellar lesions invariably present a diagnostic and therapeutic challenge for the neurosurgeon. Such cysts may be classified variously as functional/nonfunctional (on the basis

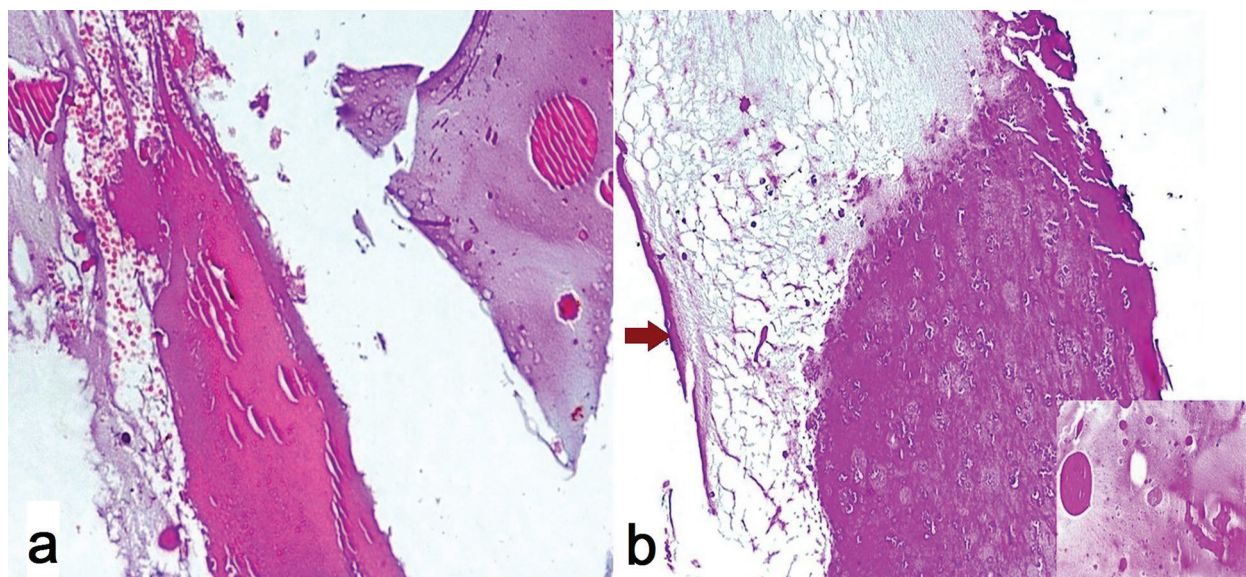


Fig. 2 Photomicrograph of the resected cyst histologically displaying amorphous eosinophilic colloid like material (H&E, 200×) (**b**) Focal fibrous tissue (arrow) devoid of epithelium forms the lining (PAS, 200×). The colloid-like material is periodic acid–Schiff–positive (inset) (PAS, 400×).

of hormonal secretion), symptomatic/nonsymptomatic, and as neoplastic (pituitary adenoma and craniopharyngioma) or non-neoplastic pars intermedia, Rathke's cleft, arachnoid, and miscellaneous.^{3–5}

The most common location of CC is the anterior part of the third ventricle.¹ Other sites are the brainstem, cerebellum, pia mater, and fourth ventricle.⁴ CC in the sellar region is rare as seen in this case where it was detected at the pars intermedia. Descriptions of sellar CC are extremely rare in the medical literature, with only a few published works describing clinical presentation.^{4–7} The exact pathogenesis of this entity lacks clarity. Possible theories include degenerative processes and/or vascular insults.⁵ Sellar CC unlike similar cysts seen at the third ventricle are devoid of epithelium and are considered by some as pseudocysts.⁸

Presentation at aberrant anatomical locations often delays diagnosis. Hence, a combined approach including CT, MRI, and histopathology is favored to diagnose these lesions. Cystic lesions in the sellar region may evoke a number of diagnostic possibilities, including Rathke's cleft cyst, cystic pituitary adenoma, craniopharyngioma, arachnoid cyst, and epidermoid cyst (►Table 1).⁹ On CT, most sellar cysts are hypodense except the CC that is usually hyperdense as seen in our case and is nonenhancing on contrast.⁴ Findings on MRI are variable. On T1WI one-half of the cases are hyperintense, and the rest are iso- or hypointense. On T2WI, most CCs show iso- to hypointensity whereas FLAIR sequences reveal increased signal intensity. This variation is attributed to paramagnetic effects of metal ions in the cyst fluid, variable protein content, and hemorrhage.^{1,10} Our case was isointense on T1, iso-hyperintense on T2, and hyperintense on FLAIR. Though hyperintensity on T2 is not usually seen in the CC, other T2 hyperintense lesions such as Rathke's cyst,

arachnoid, epidermoid cysts, and cystic pituitary adenomas have a characteristic histology, as outlined in ►Table 1. These histologic features were absent in this case, which showed colloid-like material on microscopy.

Clinical presentation of the sellar CC is insidious, and patients are often asymptomatic. However, large-sized cysts are likely to cause compression symptoms. Endocrinopathies may be a sequela of large cysts, a feature not seen in this case. Our patient, however, presented with visual diminution and bilateral optic atrophy presumably due to pressure effects.

Surgical treatment of CCs is warranted for symptomatic lesions causing compression induced endocrinologic and neurologic disturbances. The approach of choice is transnasal transsphenoidal with cyst decompression.⁴ Glistening mucoid material in the resected cyst may alert the surgeon of this possibility. Excellent outcomes have been seen as in this case. Recurrence is rare in the context of this entity.¹

Conclusion

The sellar CC is an uncommon entity. Cystic pituitary lesions must be thoroughly evaluated pre-, peri- and postoperatively to arrive at a correct diagnosis. Variable MRI findings may confound the clinician.

In resource-constrained settings, the surgeon's perioperative observations with good histopathologic assessment can minimize diagnostic delay. CC may be suspected intraoperatively based on the hint of mucoid material. Though confirmation is provided by histopathology, the astute surgeon should be aware of this entity, its gross presentation, and diagnostic challenges it poses.

Table 1 The imaging-pathologic differential diagnosis of cystic sellar lesions^{9,11,12}

Differential diagnosis	Usual age at presentation (decade)	Sex predilection	Usual CT findings	Common MRI findings (T1, T2, FLAIR)	Perioperative findings	Histopathologic findings
Colloid cyst	3rd to 4th	M = F	Unilocular hyperdense cyst	T1: Variable but commonly isointense to hyperintense T2: Low signal, iso to hypointense (depending upon protein content) FLAIR: Low signal similar to CSF No contrast enhancement	Mucoid material in cyst	Sellar lesions may be devoid of epithelium, periodic acid-Schiff-positive colloid-like material seen
Rathke's cyst	2nd to 4th	F > M	Homogenous hypodense cyst	T1: Hyper- or hypointense T2: Hyperintense No contrast enhancement except thin peripheral enhancement Intracystic nodule is indicative	Mucoid/rarely serous fluid	Simple/pseudostratified columnar epithelium with goblet cells, squamous metaplasia may be seen in the lining
Cystic pituitary adenoma	3rd, 6th	F > M	Hypodense cyst with surrounding wall enhancement	T1: Isointense to hypointense T2: Hyperintense with wall enhancement FLAIR: Isointense Sometime mural nodule found	Pituitary gland not seen separately, intratumoral bleeding seen	Cystic change in a monomorphous proliferation of cells resembling normal pituitary cells but devoid of acinar arrangement
Cystic craniopharyngioma	Bimodal: 1st peak at 1st decade, 2nd at 5th decade	No sex predilection	More extrasellar extension, hypodense with peripheral calcification	T1: Iso- to hyperintense T2: Variable Enhancement of wall noted	Machine oil with hard calcified capsule	Stratified squamous with wet keratin and cholesterol clefts and calcospherules in the adamantinoid type. pseudopapillae may be seen in the papillary type
Arachnoid cyst	4th to 5th decade	No sex predilection	Hypodense lesion with no enhancement	T1: Hypointense T2: Hyperintense No enhancement No restriction on DWI No solid component	Clear watery fluid	Cyst wall comprising fibrous connective tissue, lined by ovoid meningotheial cells
Epidermoid cyst	Middle aged	M > F	Hypodense lesion with no enhancement	T1: Hypointense T2: Hyperintense No enhancement No restriction on DWI	Pultaceous material	Keratin flakes, squamous epithelium with keratohyaline layer

Abbreviations: CSF, cerebrospinal fluid; CT, computed tomography; DWI, diffusion-weighted imaging; F, female; FLAIR, fluid attenuation inversion recovery; M, male; MRI, magnetic resonance imaging.

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Contribution of Each Author

1. Syed Asmat Ali: Collection and analysis of data, literature review, drafting, and final approval of the manuscript.
2. Kavita Gaur: Collection and analysis of data, literature review, image preparation, drafting, and final approval of the manuscript.
3. Arvind Kumar Srivastava: Concept of work, clinical management, providing intellectual content of critical importance, editing, and final approval of the manuscript.

4. Ravindra Kumar Saran: Concept of work, histopathologic analysis, providing intellectual content of critical importance, editing, and final approval of the manuscript.

Declaration

The manuscript has been read and approved by all the authors, the requirements for authorship as stated earlier in this document have been met, and each author believes that the manuscript represents honest work.

Conflicts of Interest

None.

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