

Are your MRI contrast agents cost-effective?

Learn more about generic Gadolinium-Based Contrast Agents.



**FRESENIUS
KABI**

caring for life

AJNR

Hyperprolactinemia: an unusual manifestation of suprasellar cystic lesions.

W W Woodruff, Jr, E R Heinz, W T Djang and D Voorhees

AJNR Am J Neuroradiol 1987, 8 (1) 113-116

<http://www.ajnr.org/content/8/1/113>

This information is current as
of April 19, 2024.

Hyperprolactinemia: An Unusual Manifestation of Suprasellar Cystic Lesions

William W. Woodruff, Jr.^{1,2}
 E. Ralph Heinz¹
 William T. Djang¹
 Diana Voorhees¹

Two patients with suprasellar cysts and hyperprolactinemia are described. These lesions were diagnosed by CT metrizamide cisternography. Suprasellar cysts are a rare cause of pathologic hyperprolactinemia, which most commonly results from pituitary adenomas. Tissue diagnosis revealed suprasellar arachnoid cysts in the first patient and Rathke's cleft cyst in the second. The differential diagnosis of suprasellar cysts is presented, and distinguishing radiographic characteristics are discussed.

Pathologic hyperprolactinemia with resultant amenorrhea and galactorrhea is most commonly the result of a prolactin-secreting pituitary adenoma. We have recently seen two patients with elevated serum prolactin levels who did not have pituitary adenomas but who did have CT evidence of suprasellar cysts.

Materials and Methods

Two women, ages 20 and 36 years old, both with hyperprolactinemia, were evaluated by CT. The patients were imaged on either a GE 8800 or a Siemens Somatom 2 scanner. Both were scanned twice, with initial scans obtained after intravenous contrast administration (50 cc bolus 60% Renografin and rapid drip 200 cc 43% Conray). At a later date, CT metrizamide cisternography (CTMC) was performed on both patients. Five ml of 170 mg% metrizamide was instilled into the subarachnoid space. The patients were placed in a head-down position for 2–4 min to allow cephalad flow of metrizamide into the head before scanning was conducted. Axial and coronal images of 1.5 mm through the sella and suprasellar cistern were obtained in both patients after metrizamide administration.

Results

Case 1. This woman presented in 1979 at age 20 with a 6-month history of amenorrhea, galactorrhea, and headaches. Eighteen months earlier she had an uneventful labor and delivery, and she began taking oral contraceptives for a period of 1 year. The patient remained amenorrheic with a serum prolactin level elevated to 147.6 ng/ml (normal 25 ng/ml). The thyroid-stimulating hormone level was 4.7 μ U/ml (normal 0–10 μ U/ml) and the follicle-stimulating hormone level was 10.8 mlu/ml (normal approximately 10–25 mlu/ml contingent upon the phase of the cycle). Plain radiographs revealed enlargement of the sella turcica, which measured 17 \times 11 mm. CT from an outside hospital at that time reportedly demonstrated hypodensity within the sella, thought to represent an empty sella.

The patient presented 3 years later with a chief complaint of increasing frontal headaches. CTMC was performed and demonstrated a non-CSF-communicating suprasellar cyst (Fig. 1). The findings were thought to be most compatible with suprasellar arachnoid cyst. The patient subsequently underwent right subfrontal craniotomy and cyst decompression. The diagnosis of arachnoid cyst was established. The most recent serum prolactin level (18 months after surgery) was 21.5

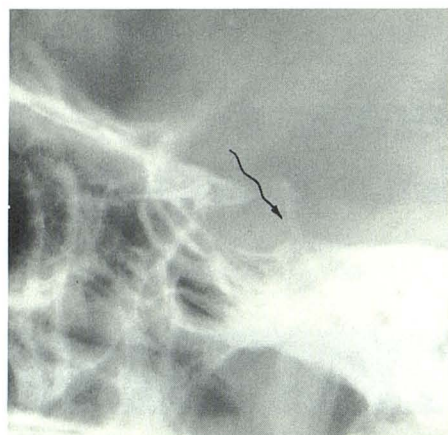
Received March 19, 1986; accepted after revision July 16, 1986.

¹Department of Radiology, Duke University Medical Center, Durham, NC 27710.

²Present address: Wilford Hall USAF, Department of Radiology/SGHQORD, Lackland Air Force Base, San Antonio, TX 78236-5300. Address reprint requests to W. W. Woodruff, Jr.

AJNR 8:113–116, January/February 1987
 0195–6108/87/0801–0113

© American Society of Neuroradiology



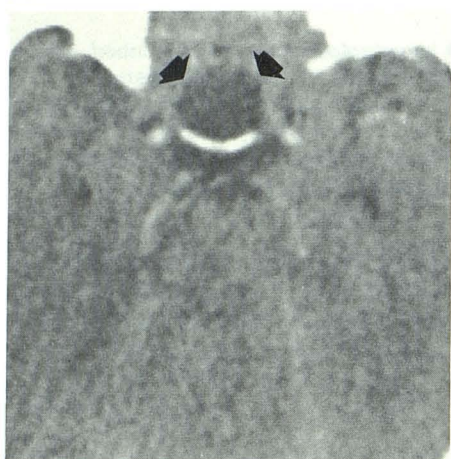
A

Fig. 1.—Case 1. Suprasellar arachnoid cyst.

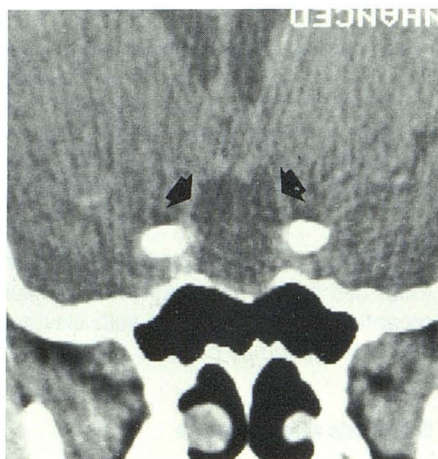
A, Lateral skull radiograph reveals mild enlargement of sella with demineralization of dorsum (arrow).

B, C, Axial and coronal IV-enhanced CT reveals nonenhancing low-density mass (arrows) in sella, which extends into suprasellar cistern. Hypophyseal stalk is not visible.

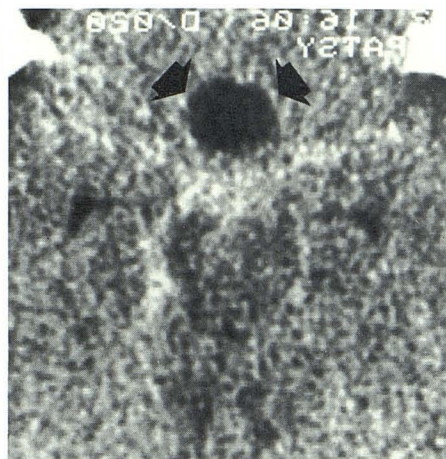
D, CT metrizamide cisternography reveals no communication between suprasellar cystic mass (arrows) and CSF cisterns.



B



C



D

ng/ml. The patient is now having normal menses, without galactorrhea.

Case 2. This woman presented in 1981 at age 36 with an 8-month history of amenorrhea, galactorrhea, and headache. The serum prolactin level on first evaluation was 64.4 ng/ml. Additional serum hormone levels from the adenohypophysis were not obtained. CT performed at an outside hospital 6 months later reportedly demonstrated a large, mixed-density lesion within the sella and suprasellar cistern. No additional diagnostic tests were performed at that time.

The patient was treated conservatively and followed for 3 years, when visual acuity in the temporal field of her left eye began to deteriorate. The serum prolactin level was now 87.5 ng/ml. CTMC was performed and showed a non-CSF-communicating suprasellar cyst (Fig. 2).

The patient subsequently underwent a right frontal craniotomy and excision of a suprasellar cyst. A diagnosis of Rathke's cleft cyst was established on histopathology. A postoperative serum prolactin level was not obtained; however, normal menses began 3 months after surgery.

Discussion

Prolactin is a hormone produced and released by the anterior lobe of the pituitary gland under hypothalamic control. The hypothalamus has a stimulatory effect on all the hormones released by the adenohypophysis with the exception of prolactin [1]. It has an inhibitory effect on prolactin production and secretion. It is theorized that this inhibitory effect is mediated by a yet-to-be-isolated prolactin-inhibitory factor. The relationship between the hypothalamus and the production of prolactin is complicated by the fact that under certain physiologic conditions, such as during lactation, a signal is sent to the hypothalamus from the stimulated mammary nipple to increase the production of prolactin. This presumably is mediated by prolactin-releasing hormone. The physiologic effect of prolactin is to increase the secretion of milk by the mammary glandular tissue.

Pituitary adenomas are the most common cause of pathologically elevated serum prolactin levels. The cases presented are most unusual in that no primary pituitary glandular abnor-

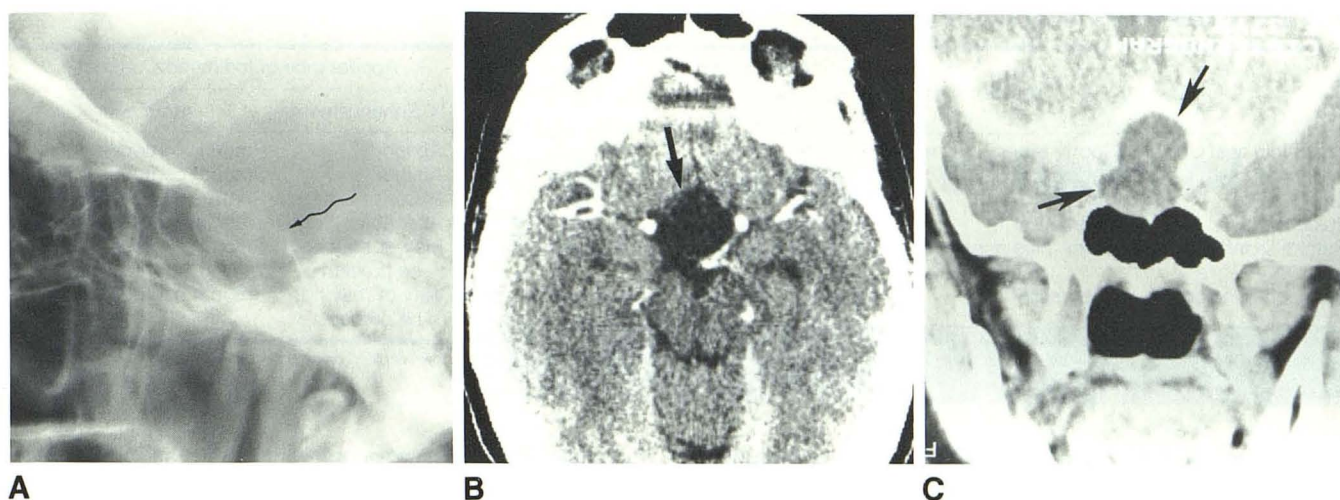


Fig. 2.—Case 2. Rathke's cleft cyst.

A, Lateral skull radiograph reveals marked thinning of dorsum sellae (arrow).

B, Axial IV-contrast CT reveals CSF-density lesion (arrow) in suprasellar cistern.

C, Axial and coronal CT metrizamide cisternography reveals non-CSF-communicating cyst (arrows) filling suprasellar cistern.

malady was present in either patient. However, pituitary glandular dysfunction was caused by a suprasellar cystic lesion in each case. At our institution, the laboratory normal serum prolactin level is 25 ng/ml. When serum prolactin levels approach 50 ng/ml our endocrinologists become concerned about structural lesions adjacent to the infundibulum interfering with transport of prolactin-inhibitory factor to the adenohypophysis. Serum prolactin levels greater than 100 ng/ml are most commonly related to prolactin-secreting pituitary adenomas.

In a report by Korsgaard et al. [2], patients with nonpituitary origin suprasellar tumors were evaluated for endocrinologic dysfunction. Two separate patient populations were identified: group A, with tumors intrinsic to the hypothalamus-pituitary axis—i.e., hypothalamus gliomas and craniopharyngiomas, and group B, with neoplasms extrinsic to the hypothalamus-pituitary axis—i.e., meningiomas, optic nerve gliomas, arachnoid cysts, and epidermoids. Endocrinologic abnormalities were present more commonly in patients with intrinsic neoplasms. Also, size being equal, intrinsic neoplasms more frequently lead to pituitary dysfunction than do extrinsic lesions. Though not included in Korsgaard's series, a suprasellar aneurysm also has been reported to have caused reversible panhypopituitarism and hyperprolactinemia [3].

One of our reported cases was an arachnoid cyst. Though considered an extrinsic lesion under Korsgaard's classification, there was associated pituitary axis dysfunction. This association was corroborated by a dramatic fall in serum prolactin levels from a preoperative level of 147.6 ng/ml to a postoperative value of 21.5 ng/ml.

Suprasellar arachnoid cysts are either congenital [4] or acquired [5]. The former are commonly thought to be a result of maldevelopment of the membrane of Lilliequist, thus per-

mitting the development of an arachnoid-lined diverticulum, extending anteriorly from the interpeduncular cistern. It is theorized that this diverticulum may become isolated from the CSF once it acquires sufficient size to occlude its neck [6]. Acquired suprasellar arachnoid cysts are the result of adhesive arachnoiditis usually secondary to basilar meningitis or subarachnoid hemorrhage.

Suprasellar arachnoid cysts are not generally associated with hyperprolactinemia resulting in amenorrhea and galactorrhea. However, the patient in case 1 displayed both amenorrhea and galactorrhea. Gentry et al. [7] recently reviewed the literature and presented in detail eight patients with arachnoid cysts. One of these (case 2) did present with galactorrhea, although the prolactin level was not stated. Sansregret et al. [4] reported a case of arachnoid cyst in a patient with amenorrhea and retarded skeletal growth. However, shortly after diagnosis was established by iodoventriculography, the patient died unexpectedly. On histologic examination, sarcoid was present and involved the infundibulum, neurohypophysis, and third ventricle. Involvement of the hypothalamic-pituitary axis by this noncaseating granulomatous process most probably accounted for the glandular dysfunction.

Patients with arachnoid cysts may present with signs and symptoms of hydrocephalus. This is especially likely if the cyst is located in the suprasellar or quadrigeminal plate cisterns. When suprasellar in location, the expanding cysts may cause a partial or complete obstruction of the anterior third ventricle or the foramina of Monro [7].

The diagnosis of Rathke's cleft cyst was established by histopathology in the second patient. Rathke's cleft cyst is a nonneoplastic remnant of the Rathke's pouch. Rathke's pouch arises from the foregut and ultimately extends cranially to become the craniopharyngeal duct [8]. The anterior portion of the pouch forms the adenohypophysis and the pars tu-

TABLE 1: Common Sellar/Suprasellar Cystic Lesions

	Location	Attenuation Relative to CSF	Calcification	Appearance of Iodinated Contrast	
				IV Enhancement	Intrathecal
Arachnoid cyst	Suprasellar	Equal to	Absent	Absent*	Smooth contour
Ependymal cyst	Suprasellar	Equal to	Absent	Absent	Smooth contour
Empty sella	Sella	Equal to	Absent	Absent	Contrast fills sella turcica
Epidermoid	Suprasellar	Less than or equal to	Absent	Absent*	Irregular contour
Rathke's cleft cyst	Sellar (plus suprasellar)	Greater than or equal to	Absent	May or may not be present	Smooth contour
Craniopharyngioma	Suprasellar	Greater than or equal to	May or may not be present	May or may not be present	Smooth contour

* Increased density may surround the lesion because of compression of normal adjacent brain, not because of actual breakdown of blood-brain barrier.

beralis, the posterior portion forms the pars intermedia. Rathke's cleft cyst is derived from the central portion of the Rathke's pouch, at the junction of the pars intermedia and the adenohypophysis, and is lined with a single layer of columnar to cuboidal secretory cells [9]. This lesion could be considered intrinsic to the hypothalamic-pituitary axis, utilizing Korsgaard's criteria.

Rathke's cleft cyst will produce symptoms when it achieves sufficient size to compress the hypothalamic-pituitary axis or optic chiasm. Obstructive hydrocephalus may occur if the mass enlarges enough to obstruct the CSF pathways. Signs and symptoms of hydrocephalus, visual field defects, hypopituitarism, diabetes insipidus, and aseptic meningitis have been reported in association with Rathke's cleft cyst [8-11].

Rarely has hyperprolactinemia in association with Rathke's cleft cyst been reported. In a report of three cases, Okamoto et al. [12] described a patient (case 3) with hyperprolactinemia. The 36-year-old woman presented with a 2-year history of amenorrhea, galactorrhea, and visual symptoms. The serum prolactin level was 45 ng/ml.

Diagnostic considerations when dealing with a cystic mass in the suprasellar cistern include suprasellar arachnoid cyst, ependymal cyst, empty sella, epidermoid, Rathke's cleft cyst, and craniopharyngioma. Intravenous and intrathecal iodinated contrast substances may be used to further characterize these lesions. Angiography is rarely useful in differentiating these suprasellar cystic lesions. A vascular blush may rarely be observed with craniopharyngiomas. MR imaging may prove useful in the diagnosis of these suprasellar cystic lesions by comparing their signal characteristics to the adjacent CSF [13].

Distinguishing characteristics of these common suprasellar cystic lesions are presented in Table 1. Suprasellar arachnoid cyst and Rathke's cleft cyst are generally indistinguishable radiologically, provided the latter does not enhance. The peripheral enhancement that may occur in Rathke's cleft cyst may be secondary either to inflammation or squamous metaplasia [12]. Ependymal cysts are extremely rare. When they occur in the suprasellar region, they are almost indistinguishable from arachnoid cysts on CT. Gentry et al. [7] distinguish them by expansion of the basal cisterns, which is more likely to occur in arachnoid cysts. The empty sella may be identified if finely collimated (1.5 mm) coronal images are obtained and an infundibulum extending into the sella is identified. Craniopharyngiomas are generally the most heterogeneous of all

suprasellar lesions, usually containing both cystic and calcified components. Contrast enhancement generally occurs in the solid portions of the craniopharyngioma. Epidermoids may exhibit hypodensity referable to CSF and an irregular surface contour that may be well defined by CTMC [14].

Patients with hyperprolactinemia are commonly referred to radiology for evaluation of pituitary adenomas. We have presented two patients with elevated serum prolactin levels as a result of suprasellar cystic lesions, not of adenomas.

REFERENCES

1. Guyton A. *Textbook of medical physiology*, 6th ed. Philadelphia: Saunders, 1981
2. Korsgaard O, Lindholm J, Rasmussen P. Endocrine function in patients with suprasellar and hypothalamic tumours. *Acta Endocrinol (Copenh)* 1976;83:1-8
3. Verbalis J, Nelson P, Robinson A. Reversible panhypopituitarism caused by a suprasellar aneurysm: the contribution of mass effect to pituitary dysfunction. *Neurosurgery* 1982;10:604-611
4. Sansregret A, Ledoux R, Duplantis F, Lamoureux C, Chapdelaine A, Leblanc P. Suprasellar subarachnoid cysts: radioclinical features. *AJR* 1969;105:291-297
5. Berkmen Y, Brucher J, Salmon J. Congenital arachnoid cysts. *AJR* 1969;105:298-304
6. Binitie O, Williams B, Case CP. A suprasellar subarachnoid pouch: aetiological considerations. *J Neurol Neurosurg Psychiatry* 1984;47:1066-1074
7. Gentry L, Menezes A, Turski P, Smoker W, Cornell S, Ramirez I. Suprasellar arachnoid cysts: 2. Evaluation of CSF dynamics. *AJNR* 1986;7:87-96
8. Byrd S, Winter J, Takahashi M, Joyce P. Symptomatic Rathke's cleft cyst demonstrated on computed tomography. *J Comput Assist Tomogr* 1980;4:411-414
9. Kojima T, Waga S. Symptomatic Rathke's cleft cyst in an old patient. *Acta Neurochir (Wien)* 1984;71:113-120
10. Steinberg G, Koenig G, Golden J. Symptomatic Rathke's cleft cyst: report of 2 cases. *J Neurosurg* 1982;56:290-295
11. Rout D, Das L, Rao V, Radhakrishnan V. Symptomatic Rathke's cleft cyst. *Surg Neurol* 1983;19:42-45
12. Okamoto S, Handa H, Yamashita J, Ishikawa M, Nagasawa S. Computed tomography in intra and suprasellar epithelial cysts (symptomatic Rathke cleft cysts). *AJNR* 1985;6:515-519
13. Lee B, Deck M. Sellar and juxtasellar lesion detection with MR. *Radiology* 1985;157:143-147
14. Hiratuska H, Okada K, Matsunaga M, Tanaka K, Fukai N, Inaba Y. Diagnosis of epidermoid cysts by metrizamide CT cisternography. *Neuroradiology* 1984;26:153-155