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Case Report

Intrasellar chordoma masquerading as a pituitary neuroendocrine tumor: Illustrative case

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ABSTRACT

Background: Chordomas are rare, locally aggressive neoplasms recognized as derivatives of the notochord vestiges. These tumors typically involve the midline axial skeleton, and intracranial chordomas exhibit proclivity for the spheno-occipital region. However, purely intrasellar occurrences are extremely rare. We report a case of intrasellar chordoma, which masqueraded as a pituitary neuroendocrine tumor.

Case Description: An 87-year-old female presented with an acutely altered mental state after a few-week course of headaches and decreased left vision. Adrenal insufficiency was evident, and magnetic resonance imaging revealed an intrasellar lesion with heterogeneous contrast enhancement and marked T2 hyperintensity. Central adrenal insufficiency due to an intrasellar lesion was suspected. Cortisol replacement was initiated, and transsphenoidal surgery was performed. Anterosuperior displacement of the normal pituitary gland and the absence of the bony dorsum sellae were notable during the procedure. Histological examination led to a diagnosis of conventional chordoma, and upfront adjuvant stereotactic radiosurgery was executed. She has been free from tumor progression for 12 months.

Conclusion: This case and literature review suggested that the pathognomonic features of intrasellar chordoma were heterogeneous contrast enhancement, marked T2 hyperintensity, osteolytic destruction of the dorsum sellae, and anterosuperior displacement of the pituitary gland. Clinical outcomes seemed slightly worse than those of all skull base chordomas, which were the rationale for upfront radiosurgery in our case. Neurosurgeons should include intrasellar chordomas in the differential diagnosis of intrasellar lesions, carefully dissect them from the adjacent critical anatomical structures, and consider upfront radiosurgery to achieve optimal patient outcomes.

Keywords: Endoscopic transnasal surgery, Intrasellar chordoma, Sellar tumor, Skull base chordoma

INTRODUCTION

Chordoma is a rare mesenchymal tumor originating from the remnants of notochord.[21] The tumor is locally aggressive and has a high tendency to recur.[19] As the notochord is a midline structure ultimately giving rise to the vertebrae and basiocciput, chordoma typically involves these structures and is situated extradurally;[4,10] approximately 50% of them develop in the sacrococcygeal region, 35% in the clivus, and 15% in the other vertebrae.[32] Although intracranial chordomas exclusively

arise in the clivus or the craniovertebral junction,[18] they can rarely arise inside the sella turcica. Due to their rarity, the details of intrasellar chordomas remain to be clarified. Based on our recent experience and extensive literature review, we share the clinical course, imaging features, and surgical findings of intrasellar chordomas.

CASE ILLUSTRATION

An 87-year-old female patient with no history of diabetes or other conditions causing blood glucose abnormalities was brought to our emergency department due to an acutely altered mental state after a few-week course of headaches and decreased left vision. Notably, 2 months before admission, she was incidentally found to have a sellar mass [Figure 1a]. Initial workup revealed severe hypoglycemia (17 mg/dL), and the administration of glucose restored consciousness. Given the mild hyponatremia (131 mEq/L), relatively low levels of serum cortisol (13.3 μg/dL) and adrenocorticotropic hormone (39.7 pg/mL) despite the acute sick condition, as well as a known history of sellar mass, central adrenal insufficiency was suspected, and the patient was initiated on hydrocortisone. Further, the endocrinological evaluation revealed hypothyroidism (thyroid-stimulating hormone level, 0.36 μIU/mL; free T4 level, 0.58 ng/dL; and free T3 level, 1.5 pg/mL). Her visual field was significantly narrowed, with mildly decreased visual acuity (20/200) on the left side, although these deteriorations were deemed to be attributable to glaucoma rather than the mass, considering the mild compression of the optic apparatus as well as the atypical pattern of visual field disturbance. Given her senility, she and her family initially refused surgical intervention and imaging surveillance with cortisol replacement was initiated. Two months later, however, she complained of rapid deterioration of her left eye vision and loss of light perception. Repeat magnetic resonance imaging (MRI) revealed rapid mass growth [Figures 1b and c]. Imaging studies demonstrated a T1-isointense, T2-hyperintense, heterogeneously enhanced, and irregularly shaped mass with restricted diffusion in the sellar and suprasellar regions [Figures 1c-f]. Marked bony erosion of the posterior clinoid process and dorsum sellae was noted [Figure 1g]. On ^{18}fluorodeoxyglucose positron-emission tomography, the mass exhibited a maximum standardized uptake value of 6.2 [Figure 1h]. After a thorough discussion with the patient and her family, we decided to proceed with the surgery.

During surgery, a transnasal corridor was created using endoscopic visualization. The mass had destroyed the sellar floor. After opening the dura in a U-shaped fashion, the mass was piecemeal removed. It was soft, grayish, and gelatinous, compressing the normal pituitary gland to the left [Figure 2a]

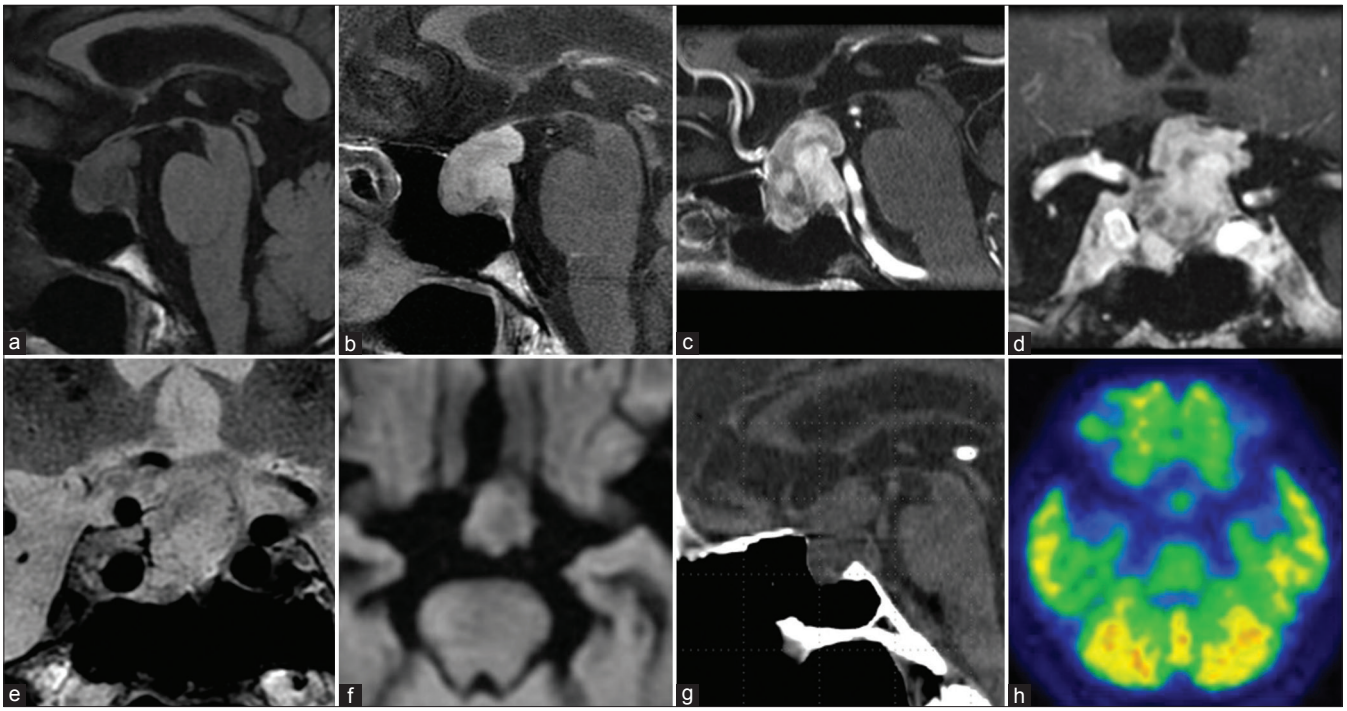

Figure 1: (a) Magnetic resonance imaging reveals an intrasellar lesion extending to the suprasellar region. (b) The lesion exhibits an avid contrast enhancement. (c) The mass grows rapidly in a short period and reaches 28 mm in size. (d) It compresses the optic chiasma and left optic nerve and invades the right cavernous sinus. (e) Marked T2 high intensity is characteristic. (f) Mild diffusion restriction is observed. (g) The dorsum sellae shows osteolysis and a bony protuberance is observed at the sellar floor. (h) Fluorodeoxyglucose positron-emission tomography reveals uptake.

and invading the right cavernous sinus [Figure 2b]. Notably, the bony dorsum sellae was absent, and the sellar side of the dural membrane was almost completely torn by the mass [Figure 2c]. A bony prominence was observed on the left side of the sellar floor [Figure 2c], which also lost its covering dura. The bony prominence was removed using a high-speed drill to access the tumor behind it. Finally, the suprasellar mass was removed while preserving the surrounding dural membrane and diaphragm. Although intraoperative cerebrospinal fluid leakage was not apparent, we decided to reinforce the diaphragm in a multilayered fashion since the diaphragm was already thin, and we were considering postoperative radiotherapy for this patient. The sellar defects were reconstructed using in- and on-lay fascial grafts with abdominal fat pieces. The dura covering the dorsum sellae was sutured with a 4–0 STRATAFIX Spiral (Ethicon, Johnson and Johnson, NJ, US) in a non-watertight fashion and was covered with pedicled sphenoid mucosa.

Postoperative imaging revealed no residual masses [Figures 3a and b]. Her visual acuity improved significantly (20/40). Histological examination revealed the presence of physaliphorous cells with a background of myxoid stroma [Figure 3c] and positive immunostaining for brachyury [Figure 3d], leading to a diagnosis of conventional chordoma. Although the Ki-67 index was not extremely high (3%), the patient was treated with upfront adjuvant stereotactic radiosurgery (20 Gy in a single fraction), given the previous rapid progression and possible minor tumor remnants around the right carotid artery. She has been free from tumor progression for 12 months.

DISCUSSION

In this study, we report a case of progressive intrasellar chordoma masquerading as a pituitary neuroendocrine tumor. Heterogeneous contrast enhancement marked T2 hyperintensity, osteolytic destruction of the dorsum sellae, and anterosuperior displacement of the pituitary gland were characteristic. We performed a literature search and quantitative analysis to verify if these features are characteristic of this rare entity. A literature search

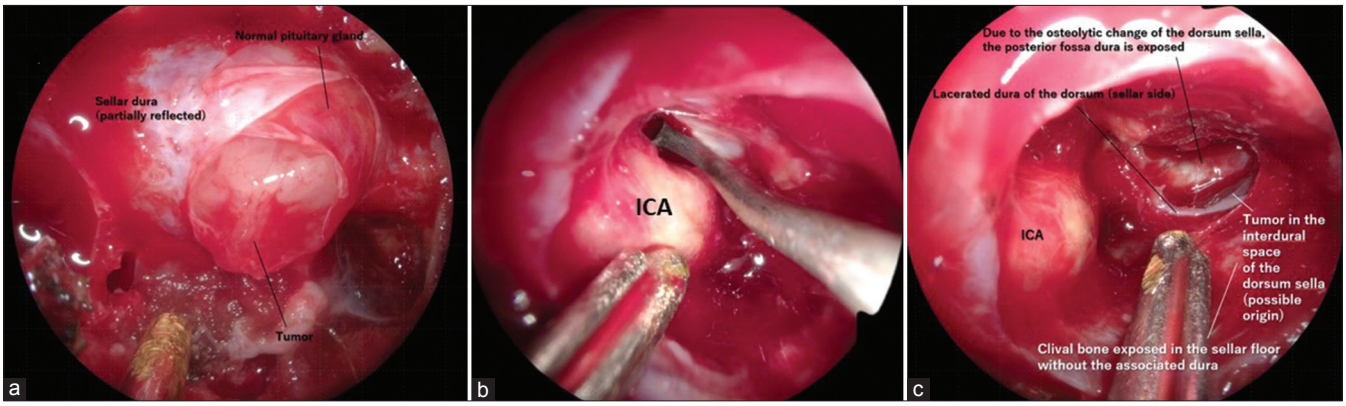

Figure 2: The tumor is soft and grayish and has a gelatinous texture. (a) The normal pituitary gland is displaced at the anterosuperior direction. (b) The tumor is invading the right cavernous sinus and is encasing the right internal carotid artery. (c) The tumor is resected in a piecemeal fashion, and the right internal carotid artery, clival bone, and dorsum sellae are exposed. The dura covering the dorsum sellae is lacerated by the tumor invasion, and the clival bone is exposed without the associated dura. Due to the osteolytic change in the dorsum sellae, the posterior fossa dura is directly exposed. ICA: Internal carotid artery.

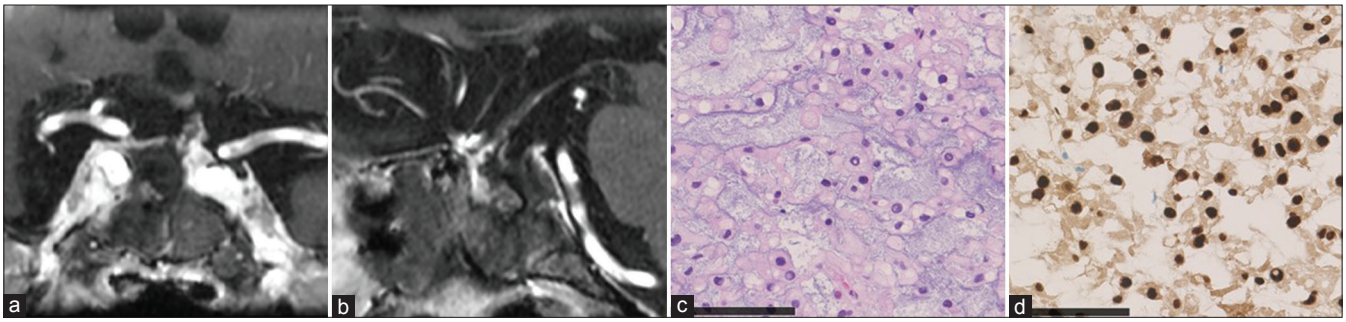

Figure 3: (a) A gross total resection is achieved. (b) The optic nerve and chiasma are relieved from compression. Histopathological evaluation is compatible with the diagnosis of chordoma. (c) The tumor cells are arranged in nests and cords embedded in a mucoid stroma. (d) These cells exhibit brachyury positivity by immunohistochemistry.

yielded 26 patients with intrasellar chordomas among 23 articles.[2,3,6,7,11,14,15,17,20,21,23-25,26,28,31-34] All articles were case reports. Including our patient, a total of 27 patients were included in the quantitative analysis.

Demographic data

The median patient age was 57 years (range, 10–87 years; standard deviation, 15 years). There were 16 males (59%) and 11 females (41%), suggesting a slight male predilection (male-to-female ratio, 1.5:1). These findings are almost identical to the other skull base chordomas, which also exhibit a peak incidence between 50 and 60 years of age with a slight male preponderance.[16] Our case was atypical for its advanced age and gender.

Clinical and radiographic presentations

Visual defects, eye movement disorders, and hypopituitarism were the predominant presenting signs and symptoms in 59%, 56%, and 52% of patients, respectively. This, in conjunction with the presence of a sellar mass, is thought to be the main rationale why intrasellar chordomas are frequently misdiagnosed as pituitary neuroendocrine tumors before surgery.[26]

Detailed information on the imaging features, either descriptively or as actual images, was provided in 27 patients (100%). Among the 26 patients with sufficient CT findings, osteolytic changes in the dorsum sellae were documented in 24 patients (92%), and bony prominence at the sellar floor was observed in 3 patients (11%). Among the 15 patients for whom MRI findings were presented, remarkable T2 hyperintensity was documented in 11 patients (73%), and heterogeneous gadolinium enhancement was observed in 15 patients (100%). Marked T2 hyperintensity, which is believed to be due to the high fluid content of the chordoma, is a hallmark of skull base chordomas.[8,27] Displacement patterns of the pituitary gland were described in nine patients; 8 (89%) of the nine patients showed anterior or superior displacement, and only 1 (11%) showed posterior displacement. Displacement of the pituitary gland theoretically reflects the tumor's epicenter and direction of growth. Given that anterosuperior displacement is rarely observed in pituitary neuroendocrine tumors, it may be of potential importance for differentiation.[21] Thus, heterogeneous contrast enhancement marked T2 hyperintensity, osteolytic destruction of the dorsum sellae, and anterosuperior displacement of the pituitary gland were all characteristic and would be the key findings for discerning intrasellar chordomas from pituitary neuroendocrine tumors.

Intervention and outcome

Transsphenoidal surgery was performed in 24 patients (89%), whereas 2 (7%) underwent craniotomy and 1 did not undergo surgery, in whom the diagnosis was incidentally made by autopsy (4%). Among the 26 patients who underwent surgery, gross total resection, subtotal resection, and partial resection were achieved in 14 (54%), 7 (27%), and 5 (19%) patients, respectively. Adjuvant radiotherapy was performed in 12 patients (44%); among the six patients in whom the detailed modalities of radiotherapy were described, particle beam radiotherapy was used in 1, conventional fractionated radiotherapy in 4, and stereotactic radiosurgery in 1. Neurological recovery was described in 10 patients (37%), and endocrinological recovery was observed in 3 patients (11%).

We managed the present case with hormone replacement and gross total resection in conjunction with upfront adjuvant stereotactic radiosurgery of the tumor bed. Transsphenoidal surgery is the mainstay treatment for intrasellar chordoma and has been performed in 92% of the reported cases. Although marginal resection is ideal,[35] the deep location of the ventral skull base adjacent to critical neurovascular structures and the infiltrative nature of the tumor often limit the extent of resection. In fact, cavernous sinus invasion was present in our patient and 80% of reported cases. Intradural extension, which was confirmed in 20% of the cases, can also be an obstacle to surgical cure. Given these locally aggressive features, the addition of high-dose local radiation therapy would be a reasonable option.[33] Although clear advantages of upfront radiotherapy over salvage radiotherapy remain to be elucidated, we performed upfront radiosurgery given the preoperative progressive course and difficulty with repeat surgery due to the patient's senility. To replicate our strategy, surgeons should carefully dissect the tumor from the optic pathway to prevent radiation-induced optic neuropathy.

Postoperative course

Sufficient postoperative follow-up data were available in 15 patients. The median postoperative follow-up period was 6 months (range, 1–72 months; standard deviation, 23 months). Recurrence was observed in four patients (33%). The 1-year and 5-year progression-free survival (PFS) rates were 62% and 41%, respectively. The 1-year and 5-year overall survival (OS) rates were 89% and 67%, respectively [Figures 4a and b]. These seemed slightly worse than those of all skull base chordomas (5-year PFS, 67%; 5-year OS, 58%).[5] Ahmed *et al*. recently analyzed pooled data on intrasellar chordoma using the surveillance, epidemiology, and results database and reported a 5-year OS of 60%.[1] Although biases cannot be excluded due to the small patient number, these formidable outcomes were the rationale for upfront radiosurgery in our case. We do not have a good explanation for the worse prognosis of

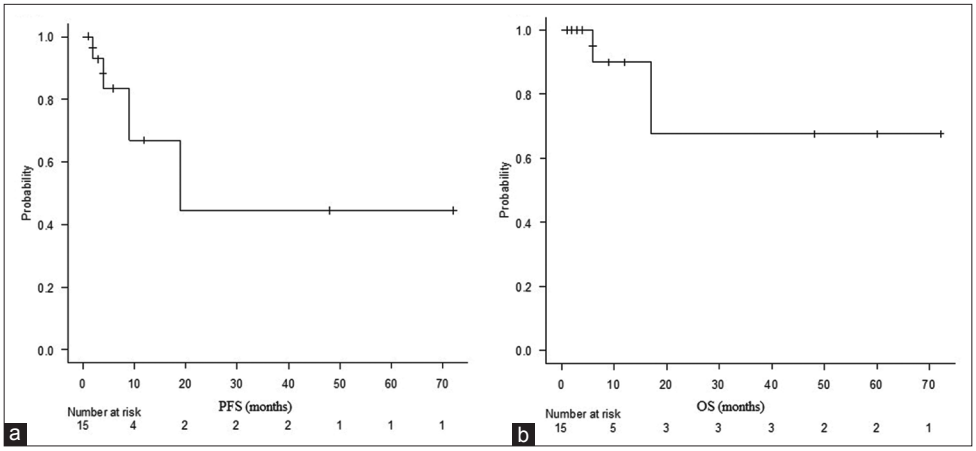

Figure 4: (a) The Kaplan–Meier curve is depicted regarding the progression-free survival and (b) overall survival. OS: Overall survival, PFS: Progression-free survival.

intrasellar chordomas; however, frequent preoperative visual deficits may be responsible, as reported in a previous meta-analysis on all skull base chordomas,[35] which may limit the daily activities and accelerate the deterioration of disabilities.

Pathogenesis

Few studies have addressed the tumorigenesis of intrasellar chordomas. From an embryological standpoint, chordomas are thought to originate from relics of notochordal tissues.[16] Because the cranial end of the notochord is situated in the basiocciput during normal development, skull base chordomas are predominantly located in the clivus and craniovertebral junction. In the meantime, several developmental studies demonstrate that the notochord may extend beyond the basiocciput, reaching just posterior to the hypophyseal fossa within the basisphenoid, which is around the dorsum sellae.[12,22,29] Moreover, studies on canalis basilaris medianus or a possible remnant of cephalic portions of the notochord revealed that there are several variations of the course of the notochord, with one of them reaching the dorsum sellae.[9,13,30] With all things considered, we hypothesize that intrasellar chordoma is extremely rare because it arises in a limited number of patients in whom the notochord reached the dorsum sellar during the normal development, failed to involute completely, and exhibited neoplastic progression later in life. Osteolytic changes in the dorsum sellae appear to imply a tumor origin, whereas an unusual bony prominence at the sellar floor suggests that an ossification center, or notochord, reached the level of the sellar floor.

Limitation

This study has several limitations that should be considered when interpreting the results. First, the retrospective nature and small number of patients with a relatively short follow-up period did not fully elucidate the prognosis of intrasellar chordomas. Second, literature-based data analyses may be susceptible to reporting bias. Third, not all imaging data for the literature cases were available, and the percentage of each imaging feature may have been underestimated. Despite these shortcomings, due to the rarity of intrasellar chordomas, a single-center study is unrealistic, and our method is the best available way to unveil their clinical and radiologic features.

CONCLUSION

The clinical manifestations of intrasellar chordomas resemble those of pituitary neuroendocrine tumors. Heterogeneous contrast enhancement, marked T2 hyperintensity, osteolytic destruction of the dorsum sellae, and anterosuperior displacement of the pituitary gland are the pathognomonic features. Neurosurgeons should consider intrasellar chordomas in the differential diagnosis of intrasellar lesions.

Ethical approval

The Institutional Review Board approval is not required.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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