

Pediatric Pituitary Neuroendocrine Tumor With Extensive Necrosis: A Case Report

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Abstract

We describe an unusual presentation of a pediatric pituitary neuroendocrine tumor (PitNET). A 17-year-old previously healthy male presented to the emergency department with altered mental status, lethargy, nausea, vomiting, fever, and tachycardia. Head imaging incidentally demonstrated a large expansile sellar/suprasellar cystic and solid mass displacing the optic chiasm, invading bilaterally into the cavernous sinuses, and encasing the internal carotid arteries. He developed decreased vision of the left eye on hospital day two and underwent urgent endoscopic endonasal transsphenoidal resection of the mass. Pathology demonstrated a PitNET with extensive necrosis, lactotroph differentiation, and changes suggestive of superimposed tumoral infection. His postoperative course was complicated by panhypopituitarism, chronic diabetes insipidus, and a cerebrospinal fluid leak managed with lumbar drainage. His presenting symptoms resolved completely, with no tumor recurrence at two-month follow-up.

The initial clinical manifestation in this case was suggestive of adrenal crisis secondary to pituitary hypofunction and secondary adrenal insufficiency, attributed to significant tumor compression and pituitary necrosis, and surgical decompression was performed for new visual loss.

Categories: Pediatric Surgery, Pediatrics, Neurosurgery

Keywords: adrenal crisis, endoscopic endonasal transsphenoidal resection, nonfunctioning pituitary adenoma, pediatric endocrinology, pediatric neurological surgery, pediatric neurosurgery, pitnet, pituitary necrosis, pituitary neuroendocrine tumor, secondary adrenal insufficiency

Introduction

Pediatric pituitary neuroendocrine tumors (PitNETs) are extremely rare neoplasms of the pituitary gland in children and adolescents that account for less than 3% of pediatric brain tumors. Only 2% to 6% of surgically treated pituitary adenomas occur in the pediatric population [1-3]. A higher incidence is present in females, particularly due to the female predominance of prolactinomas [2,4,5]. PitNETs with hormone hypersecretion or pituitary compression carry a particularly negative potential in growing children and adolescents, who depend on this balance of hormones for appropriate development [1].

The majority of presenting symptoms include headache, visual impairment, menstrual irregularity, and those associated with specific hormone hypersecretion or pituitary hyposecretion [1-3,5,6]. Prolactinomas may present with delayed puberty, gynecomastia, and galactorrhea; corticotroph tumors with Cushing disease, rapid weight gain, acne, hirsutism, and moon facies; and somatotroph tumors with precocious growth, acromegaly, and gigantism [2,5]. Nonfunctioning adenomas (NFAs) are more often associated with impairment in pituitary and endocrine function, including arrested growth, hypothyroidism, apoplexy, pubertal delay, hyperprolactinemia, and secondary adrenal insufficiency [2,7]. Corticotrophic deficiency, commonly associated with secondary adrenal insufficiency, and apoplexy are important to recognize early, as these conditions can precipitate serious hemodynamic instability and life-threatening decompensation [7,8]. This is especially important in patients with NFAs, who do not present with the classic symptoms of hormone hypersecretion and may therefore present later in their disease course.

Here we present an instructive case of an adolescent male who presented with uncharacteristic hemodynamic symptoms and was incidentally found to have a PitNET on imaging.

Case Presentation

A 17-year-old previously healthy male was brought in by ambulance to an outside hospital and subsequently transferred to our institution for altered mental status, lethargy, nausea, and vomiting. On arrival he was unresponsive, febrile, tachycardic, and hyponatremic. He was immediately started on supportive measures and empiric antibiotics for sepsis rule-out, as well as high-dose corticosteroids for concern for adrenal insufficiency.

Following the finding of a pituitary mass on head computed tomography (CT) at the outside hospital, brain

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magnetic resonance imaging (MRI) with and without contrast using pituitary and tumor protocol demonstrated a large expansile sellar/suprasellar cystic and solid mass with marked superior displacement of the optic chiasm, extending bilaterally into the cavernous sinuses with encasement of the bilateral cavernous internal carotid arteries. Imaging also demonstrated erosion of the sellar floor with extension of the mass into the sphenoid sinus (Figures 1, 2). M g hormone (TSH), free thyroxine (free T4), luteinizing hormone (LH), adrenocorticotrophic hormone (ACTH), and prolactin (PRL), while levels of follicle-stimulating hormone (FSH), cortisol, growth hormone, and insulin-like growth factor 1 (IGF-1) were within normal limits. On hospital day two, the patient had improvement in mental status but developed decreased vision in his left eye.

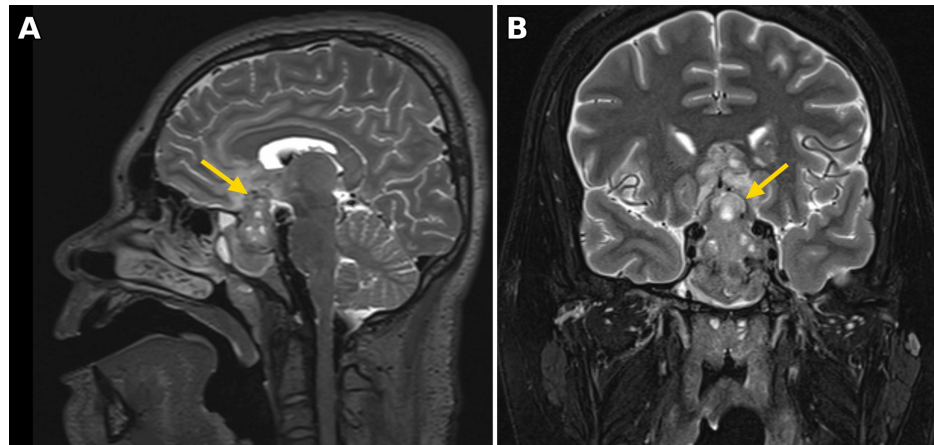


FIGURE 1: Preoperative MRI brain with contrast

T2-weighted sagittal (A) and coronal (B) sections demonstrating the large expansile sellar-suprasellar cystic and solid mass (arrows) with superior displacement of the optic chiasm. MRI: magnetic resonance imaging



FIGURE 2: Maxillofacial CT with contrast

Axial section redemonstrating the large expansile sellar/suprasellar mass (arrow) expanding the clivus with extension into the sphenoid sinus. CT: computed tomography

The patient was taken to the operating room on hospital day three for endoscopic endonasal resection of the sellar-suprasellar tumor with a combined neurosurgery and ear, nose, and throat (ENT) team. The ENT team obtained exposure through an endonasal approach with posterior septectomy, wide sphenoidotomy, and bilateral ethmoidectomies. Once exposure was complete, a bimanual two-surgeon technique was used for tumor resection. The tumor was found to have eroded into the sella. A micro-Doppler probe was used to confirm midline dural exposure. The dura was opened medial to the carotid arteries laterally with a knife and the opening was extended with scissors in a cruciate fashion. The tumor, which appeared as a whitish fibrous mass, and its capsule were subsequently identified and removed inferiorly and superiorly. A minimal tumor rim was left on the carotid arteries laterally. Copious irrigation and a gelatin-thrombin hemostatic matrix (FloSeal; Baxter International Inc., Deerfield, IL, USA) were applied. The ENT team then proceeded with closure. No intraoperative cerebrospinal fluid (CSF) leak was encountered and there was minimal blood loss.

The postoperative course was complicated by chronic diabetes insipidus and panhypopituitarism. Repeat brain MRI with and without contrast demonstrated postsurgical changes related to transsphenoidal resection of the large cystic sellar mass, with significantly decreased associated mass effect on the optic chiasm (Figure 3). Pathology demonstrated a PitNET with extensive necrosis, lactotroph differentiation on immunohistochemistry (IHC), and changes suggestive of superimposed tumoral infection. Postoperatively, the patient was found to have improvement in vision in his left eye and complete resolution of his initial presenting symptoms. He was discharged on hospital day 14 on aspirin, desmopressin, hydrocortisone, and levothyroxine.

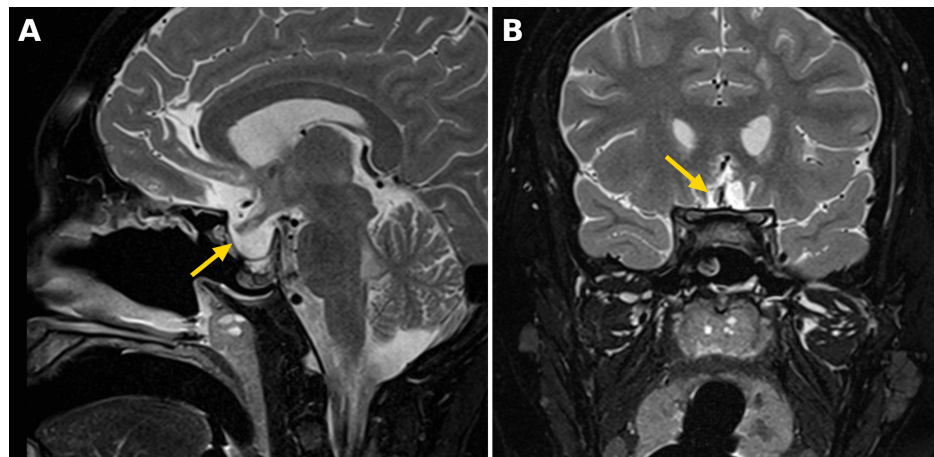


FIGURE 3: Postoperative MRI brain with contrast

T2-weighted sagittal (A) and coronal (B) sections in the immediate postoperative period demonstrating postsurgical changes (arrows) related to transsphenoidal resection of the large cystic sellar/suprasellar mass, with significantly decreased associated mass effect on the optic chiasm. MRI: magnetic resonance imaging

At the two-week follow-up visit, the patient presented with rhinorrhea and a salty taste in the back of his throat, consistent with a CSF leak. He was admitted and treated with antibiotics and lumbar drain placement. Endoscopic examination of the nasal cavity and skull base demonstrated an intact nasoseptal flap with a low anterior caudal septal perforation and dry, exposed septal cartilage without intact mucosa. The lumbar drain was removed on day five, the rhinorrhea resolved, and he was discharged on hospital day 13.

At the two-month follow-up visit, the patient was doing well without CSF leakage, headaches, or fever, and vision in the left eye continued to improve. Repeat brain MRI did not demonstrate recurrence of the tumor. The patient was continued on the above medications with the addition of testosterone cream.

Discussion

Pediatric neoplasms of the pituitary gland are extremely rare, with an annual incidence of one per 100,000 in children aged 10 to 19 years [4]. Only 10% of pituitary neoplasms occur in children and adolescents, with 80% to 90% of these masses consisting of craniopharyngiomas and only 3% consisting of PitNETs [8]. Further, in children under the age of 20 years, only 3% of PitNETs are nonfunctioning [4,8]. Median age at diagnosis is 15 years, while NFAs are exceptionally rare in those under the age of 16 years [1-5].

PitNETs can be subcategorized into prolactinomas, corticotroph, somatotroph, gonadotroph, and nonfunctioning tumors. Among surgically treated pediatric patients, corticotroph tumors are the most common PitNET, followed by prolactinomas, somatotroph tumors, and nonfunctioning neoplasms [3]. Corticotroph tumors and prolactinomas have a higher female predominance, while gonadotroph tumors and NFAs are more common in males [3]. Overall, in pediatric patients, up to 90% of PitNETs are functioning [1,3]. This is in contrast to adult PitNETs, which are nonfunctioning in up to one-third of patients [6]. The decreased prevalence of NFAs in pediatric patients may be attributable to the fact that NFAs are slow-growing tumors that may not grow large enough to induce symptoms, which requires approximately 80% to 95% compromise of the pituitary gland or optic chiasm. Symptomatic NFAs are therefore less likely to present in children and adolescents [5,8]. Our patient's pituitary laboratory studies did not demonstrate a predominantly elevated hormone level, consistent with a clinically nonfunctioning PitNET despite lactotroph differentiation on IHC. Rather, our patient presented with mass effect compromising the pituitary gland and optic chiasm, given decreased levels of multiple pituitary hormones, imaging findings of superior displacement of the optic chiasm consistent with his visual deficits, and findings of significant necrosis demonstrated on pathology.

The most common symptoms of PitNETs include headache, visual impairment, menstrual irregularity, and findings consistent with specific hormone excess or pituitary hypofunction [1-3,5,6]. PitNETs rarely present with focal neurological deficits. Visual dysfunction is common in adults with NFAs but occurs in fewer than 10% of pediatric patients with the same pathology [5,7].

Compared with adults, PitNETs grow at a more rapid rate and are generally more aggressive, larger, and more invasive, with more frequent involvement of adjacent parasellar and suprasellar structures, as seen in our patient [4,6]. NFAs in particular are more aggressive in pediatric patients than in adults. For NFAs that do not present with symptoms of hormone excess, invasiveness of the tumor can result in visual impairment

and headache due to tumor size and compression of adjacent structures [2]. Younger children whose tumors have not grown large enough to induce symptoms of mass effect may present late in their disease course [2,4]. Our patient did not have any preceding signs of headache or visual impairment prior to his hospital presentation. Rather, he presented with altered mental status, lethargy, tachycardia, and fever, findings consistent with adrenal crisis versus sepsis. His decreased levels of multiple pituitary hormones, likely secondary to tumor compression, as well as improvement in hemodynamic symptoms after high-dose corticosteroid administration, support hormonal deficiency capable of precipitating adrenal crisis.

Findings of pituitary hypofunction that can complicate NFAs include secondary adrenal insufficiency and hypothyroidism, which are often associated with deficiencies in growth hormone, FSH, and LH [7]. In adults, the prevalence of secondary adrenal insufficiency in patients with NFAs ranges between 17% and 62% [7]. Oshino et al. further demonstrated that 38.7% of adult patients with optic chiasm compression had secondary adrenal insufficiency, and that lateral extension on the less-extending side of the tumor is the strongest risk factor for developing secondary adrenal insufficiency, with a high prevalence in tumors that occupy the sellar space and compress both cavernous sinus walls without invasion [7]. While the cause of our patient's initial symptoms remains incompletely defined, the presence of optic chiasm compression together with bilateral invasion into the cavernous sinus walls encasing the bilateral internal carotid arteries, indicating high tumor mass effect, may suggest secondary adrenal insufficiency as a contributor to his presenting symptoms. Mass effect was likely further aggravated by the cystic changes in our patient's neoplasm, as evidenced by his imaging, while the significant necrosis documented on pathology likely contributed to his pituitary hypofunction. Tumors such as this patient's, with cystic changes or apoplexy, are documented to cause visual impairment and pituitary hypofunction due to compression of the optic chiasm and pituitary gland [3]. The extensive invasiveness of our patient's tumor represents a late presentation with more serious symptomatology, likely due to the absence of typical warning symptoms or signs of hormone excess. Our patient developed visual impairment only during hospitalization, consistent with superior displacement of the optic chiasm. It is therefore important to recognize uncharacteristic and potentially life-threatening findings as possible presentations of PitNETs that require emergent resection for decompression.

PitNETs are diagnosed with brain MRI using thin 1- to 3-mm slices through the sella [4,5]. These tumors fill and may expand the sellar space or invade the adjacent cavernous sinuses. Tumors with hemorrhage or necrosis may also demonstrate internal heterogeneity, as seen in the imaging findings of our patient [5]. Other diagnostic workup includes baseline pituitary hormonal evaluation and ophthalmologic assessment with visual field and visual acuity testing [4,5].

The gold standard treatment for PitNETs is transsphenoidal resection in most cases [1,4]. Indications for immediate surgical intervention include symptoms of mass effect such as acute visual loss or cranial nerve palsy. Extensive optic chiasm compression in the setting of NFAs is another indication for early surgery, as seen in our patient [7].

Postoperative complications are more prevalent in pediatric patients than in adults, given the more invasive nature of PitNETs in children and adolescents [1]. Pediatric patients also have higher rates of pituitary insufficiency following surgery, which can have dramatic impacts on growth, development, and fertility. Other common complications include permanent visual dysfunction, chronic diabetes insipidus, and postoperative CSF leak; chronic pharmacological hormone replacement is required in approximately 25% of cases postoperatively [5]. Our patient had both preoperative and postoperative hypopituitarism, more pronounced following surgery and accompanied by chronic diabetes insipidus, which was supplemented and well controlled with hormonal therapy. Given that our patient is post-pubescent, the impacts of panhypopituitarism are less likely to affect growth velocity, and supplementation with growth hormone was therefore not deemed necessary. CSF leak occurs in approximately 3% to 8% of pediatric transsphenoidal resection cases, with increased risk in the setting of suprasellar extension, as seen in our patient, intraoperative CSF leak, or prior treatment with surgery, radiation, or dopamine agonists. Treatment typically consists of lumbar drainage, acetazolamide, and repair with either an abdominal fat graft or a nasoseptal flap [5]. Our patient's CSF leak was adequately treated with a lumbar drain for a brief period without recurrence.

This report has limitations inherent to its retrospective design. The timing of the cortisol measurement relative to corticosteroid administration is not reported and no confirmatory adrenal testing is presented, so adrenal crisis remains a suspected rather than confirmed diagnosis, and concurrent sepsis was not excluded. The low prolactin level despite lactotroph differentiation on IHC most likely reflects extensive tumor necrosis, although the hook effect cannot be excluded in this report. Superimposed tumoral infection was suggested histologically, and microbiological findings are not reported. Formal visual field results are not reported, and follow-up was limited to two months.

Conclusions

PitNETs are rare neoplasms in children and adolescents and are especially impactful in this age group secondary to endocrine dysfunction and its effects on growth, development, and fertility. Furthermore, PitNETs, and the nonfunctioning subset in particular, are more invasive in pediatric patients, resulting in

more serious presentations. While most patients initially present with symptoms of mass effect or hormone hypersecretion, pituitary hypofunction can induce dangerous hemodynamic instability, including tachycardia, fever, nausea, and vomiting associated with adrenal insufficiency, and this may be the only clinical manifestation. It is therefore important to recognize this symptomatology in otherwise previously asymptomatic patients and to treat urgently, as these signs may indicate significant mass effect and the need for decompression.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Acquisition, analysis, or interpretation of data: Homayoun Ghaseminezhad, Jessica Sawaya, Tanya Minasian, Miguel Lopez-Gonzalez

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Supervision: Tanya Minasian

Disclosures

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