

Pituitary Apoplexy Mimicking Pituitary Abscess: Case Report

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Citation

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Abstract

We are reporting an uncommon case of febrile apoplexy of a pituitary macroadenoma eroding into sphenoid sinus.

A 58-year-old female was transferred with confusion, fever, headaches and visual abnormalities. She had CSF pleocytosis and a presumptive diagnosis of meningitis and was on antibiotics.

CT showed a large pituitary mass 21×19×21mm, an increased density on the left of the mass compatible with hemorrhage, a moderate soft tissue density and mild inflammatory change in left sphenoid sinus, erosion of the floor of the sella on the left primarily and right deviation of the midline bony septum of the sphenoid sinus. MRI confirmed these findings.

The pituitary mass was debulked. Pathology revealed scant foci of poorly preserved pituitary adenoma with extensive necrosis, active inflammation and granulation tissue. Cultures were again negative.

Pituitary apoplexy is an important noninfectious entity to keep in mind beside pituitary abscess when a patient presents with fever and acute neurological, visual and endocrine manifestations.

INTRODUCTION

Pituitary apoplexy (PAP), an acute hemorrhagic infarction of the pituitary gland with abrupt neurological impairment, usually occurs in an existing macroadenoma (> 10 mm). It is an uncommon event that takes place in an estimated range of 0.6%₁ (typical symptomatic PAP with significant hemorrhagic and necrotic changes) to 27.7%₂ (only MRI criteria without clinical evidence of apoplexy or blood discovered in specimens of asymptomatic patients) of cases of pituitary adenoma, although the figure is probably closer to 10%₃.

A macroadenoma may extend beyond the confines of the sella turcica, upward toward the optic chiasm and hypothalamus, downward toward the sphenoid sinus and laterally toward the cavernous sinuses. PAP is a big imitator of other intracranial pathological entities. It frequently manifests itself by signs and symptoms that may resemble those of different intracranial pathological processes. Headache, photophobia, stiff neck, and oculomotor palsy may mimic a ruptured intracranial aneurysm, whereas fever, meningismus, and lethargy can appear to indicate bacterial or viral meningitis_{4,5,6}. Periorbital edema from compression of the venous plexus contained in the cavernous sinus paired with defects resulting from encroachment on adjacent cranial nerves can emulate cavernous sinus thrombosis. Unilateral

ptosis and mydriasis, in addition to restriction of adduction, elevation, and downward gaze, may raise consideration of the presence of a posterior communicating artery aneurysm or, if lacking the inclusion of pupillary dilation, diabetic third nerve palsy.

We are reporting an uncommon case of febrile apoplexy of a large pituitary tumor eroding into sphenoid sinus that was managed by our medical team.

CASE

A 58-year-old female was transferred to us from another hospital where she presented with confusion, fever, headaches and visual field defects. She had a presumptive diagnosis of meningitis and was put on Rocephin, Doxycycline, Acyclovir and Bactrim. On physical exam she had third cranial nerve abnormalities but a supple neck. She had a history of diabetes mellitus II and hypertension. A lumbar puncture performed prior to transfer revealed 31 white cells, poly predominant, with elevated glucose and protein levels. Cerebrospinal fluid (CSF) gram stain and cultures had failed to demonstrate any organisms.

Her vitals on transfer were temperature 100.5 °F, pulse 76/minute, respiratory rate 21/minute and blood pressure 147/52 mmHg. Lab analyses showed WBC 6.5 x10⁹ /litre,

PMN 85%, decreased prolactin and TSH levels and normal blood and urine osmolality levels.

We ordered brain CT that showed evidence of a fairly large mass involving the sella and pituitary gland 21×19×21mm. There was increased density on the left of the mass compatible with hemorrhage. The mass impinged upon the optic chiasm as well as the hypothalamus with the displacement of these structures upwards. There was evidence of a moderate soft tissue density and mild inflammatory change involving the left sphenoid sinus (Fig.1A) and there was also some erosion of the floor of the sella on the left primarily. The midline bony septum of the sphenoid sinus was deviated to the right at its lower part (Fig.1B). There was evidence of moderate to marked inflammatory change involving the frontal sinuses, especially on the left. There was also evidence of mild inflammatory change involving the anterior ethmoids on the left as well. MRI demonstrated a large pituitary mass with hemorrhage and necrosis (Fig.2A). It confirmed the frontal, ethmoid and sphenoid sinusitis (Fig.2B).

Figure 1

Figure 1: Brain CT, Transverse and Coronal Views

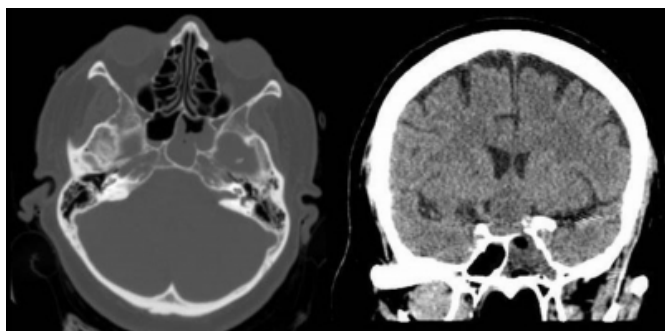
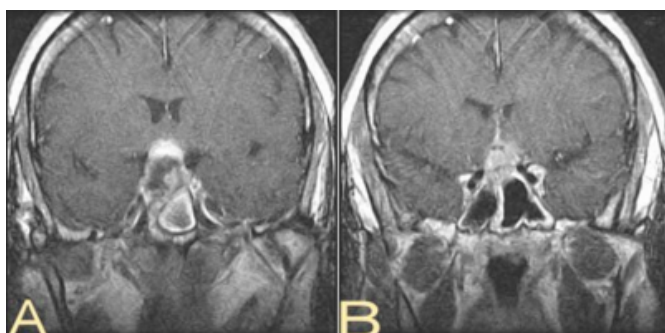


Figure 2

Figure 2: Brain MRI, Coronal View



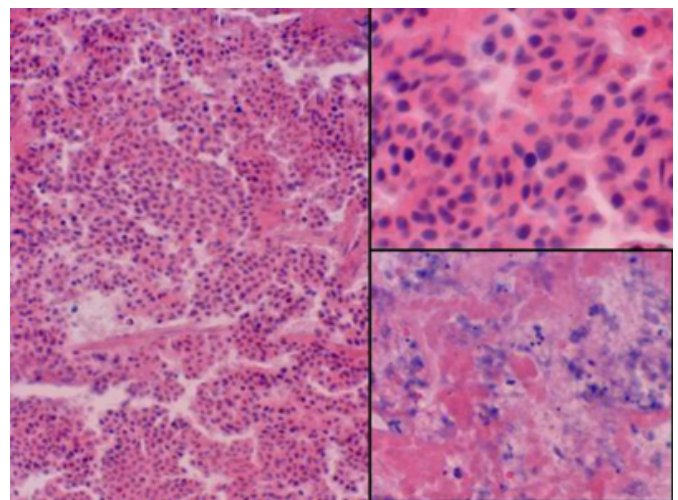
After thorough discussion, it was decided to proceed with operative intervention. The patient underwent transsphenoid resection of the pituitary lesion. There was evidence of

bleeding in the left lateral pituitary area and erosion into the left sphenoid sinus. The pituitary appeared to be grossly necrotic. A substantial mass was removed and sent for pathologic expertise.

Pathologic study showed scant foci of poorly preserved pituitary adenoma with extensive necrosis, active inflammation and granulation tissue. Microscopic examination revealed solid islands of tumor cells with round to oval nuclei and moderately abundant eosinophilic cytoplasm (Fig.3). Nuclear pleomorphism was minimal and mitoses were virtually absent (Inset A). Most of the tumor showed poor preservation and extensive necrosis was noted (Inset B). Culture yielded no anaerobes, fungi or acid-fast bacilli.

Figure 3

Figure 3: Pathologic View



Vancomycin was given intraoperatively and the insulin dose was reduced because of her decreased calory intake. The panhypopituitaric patient was started on Synthroid and Hydrocortisone.

Based on the patient's clinical presentation, imaging studies and tissue histopathology she was diagnosed with an infarction type of PAp presenting with a meningoencephalitis picture. The patient's general condition and vision rapidly improved in the post operative setting.

DISCUSSION

The anterior pituitary gland is perfused by its portal venous system, which passes down the hypophyseal stalk. This unusual vascular supply likely contributes to the frequency of PAp. Some postulate that a gradual enlarging pituitary tumor becomes impacted at the diaphragmatic notch,

compressing and distorting the hypophyseal stalk and its vascular supply. This deprives the anterior pituitary gland and the tumor itself of vascular supply, apoplectically causing ischemia and subsequent necrosis. Another theory stipulates that rapid expansion of the tumor outstrips its vascular supply, resulting in ischemia and necrosis. This explanation is doubtful, since most tumors that undergo apoplexy are slow growing.

The pituitary gland is contained in the sella turcica, which is a midline depression in the sphenoid bone. Inferiorly, the sella turcica has a thin floor of cortical bone, below which lies the air-containing sphenoid sinus. Inferior extension of an adenoma causes either remodeling of the floor of the sella turcica or frank erosion through the floor into the sphenoid sinus. It may be difficult if not impossible to determine whether the inferiorly extending adenoma is eroding through the bone of the floor or merely remodeling it, because the floor of the sella turcica, formed from thin cortical bone, is difficult to appreciate. There is no appreciable contrast interface on MRI between dark cortical bone and the dark air-containing sinus. Inferior extension is seen as asymmetric downward protrusion of a soft tissue mass from the inferior aspect of the gland.

PAP is usually manifested by sudden onset of headache, visual symptoms, altered mental status, and hormonal dysfunction. The visual symptoms include visual acuity, visual field impairment or ocular motility dysfunction from involvement of the optic nerve, chiasm or the cranial nerves traversing the cavernous sinus, respectively.

Clinical unawareness of the presence of a preexisting pituitary mass often renders apoplexy an overlooked possibility; consequently, the diagnosis is often delayed and the clinical signs are mistaken as produced by a different cause. Fever, meningismus, and lethargy can be signs of encephal meningitis and if visual field defects are added pituitary abscess (PAb) should be included in the differential diagnosis. The nonspecific nature and variability of signs and symptoms further confound expeditious establishment of an appropriate diagnosis.

PAb secondary to an adenoma is rare. To date, only 20 cases have been reported. Pituitary tumors become susceptible to infection because of impaired circulation, areas of necrosis and local immunological impairment. It remains very difficult to diagnose it preoperatively or before autopsy.

A PAb usually develops over a few months. The mass effect

may cause headache and visual loss. The loss of vision due to extension above the level of the diaphragma sellae is usually gradual, but it may be as acute as in PAP. Endocrine deficiency may or may not be present, depending on the duration of the disease and the degree of compression of the pituitary. The development of diabetes insipidus may help differentiate a PAb from a PAP. Diabetes insipidus only develops in 10% of pituitary adenomas but commonly occurs in the evolution of PAb and metastatic tumors of the pituitary¹⁰. The presence of combined anterior and posterior pituitary disturbance in the absence a hypothalamic lesion should also arouse the suspicion of an abscess.

CSF findings in PAP may mimic those in meningitis, and the presence of inflammatory changes in the CSF is well documented in the literature¹¹. CSF examination may reveal elevated numbers of leukocytes and/or polymorphonuclear cells with negative microbiologic studies. Pleocytosis can be a difficult diagnostic dilemma in patients who present with a febrile form of PAP¹². CT and MRI help solve the problem.

The culture in our case was negative as should be in PAP but could also be the result of the use of antibiotics as would PAb proponents say.

MRI is the most sensitive imaging study for evaluating the pituitary gland. In our case, it visualized necrosis and hemorrhage characteristic of PAP.

PAP remains a severe complication of pituitary adenomas; serious endocrine sequelae and fatal outcomes are frequent. Endocrine insufficiencies can require lifelong hormonal replacement. The natural history of this patient's pituitary macroadenoma eroding into the left sphenoid sinus led to apoplexy and panhypopituitarism. In this case, the only possible operative intervention was transsphenoid drainage.

CONCLUSION

PAP is an important noninfectious entity to keep in mind beside PAb when a patient presents with fever and acute neurological, visual and endocrine manifestations. This case illustrates the difficulty of preoperative diagnosis of PAP presenting with fever and altered mental status and the consequences of missing the right diagnosis. Early suspicion and treatment of PAP are indispensable if significant morbidity and mortality are to be prevented.

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