ABSTRACT

Pituitary apoplexy is an endocrinological emergency, usually occurring in a pituitary adenoma and is caused by sudden onset hemorrhage and ischemic necrosis of the pituitary gland. Sudden-onset headache along with vomiting is the usual presenting feature. Signs of meningism, however, occur late and are not a characteristic feature. These may be associated with other neurological features like disturbances of consciousness, vertigo, and hemiparesis. Ocular features include marked reduction in the visual acuity with bitemporal hemianopia, diplopia, and ophthalmoplegia due to oculomotor nerve palsies. Acute pituitary apoplexy is unpredictable and should be considered in any patient with abrupt neuro-ophtalmological deterioration associated with headache. We report a unique case of pituitary apoplexy with bilateral total ophthalmoplegia having complete oculomotor nerve palsy in one eye and pupil-sparing oculomotor nerve palsy in the other.

Keywords: Bilateral ophthalmoplegia, Occulomotor nerve palsy, Pituitary adenoma, Pituitary apoplexy.

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INTRODUCTION

Pituitary apoplexy is a rare endocrine emergency that can occur due to infarction or hemorrhage in pituitary gland. This disorder most commonly occurs in a pituitary adenoma. Some well-recognized risk factors are hypertension, medications, major surgeries, coagulopathies, head injury, radiation, or dynamic testing of the pituitary. But it may also occur without any precipitating risk factors. Both neurological and endocrinological signs and symptoms are usually present and ocular manifestations may be the presenting features. The clinical presentation can mimic optic neuritis, ruptured arterial aneurysms, carotico-cavernous fistula, and giant cell arteritis. Headache due to raised intracranial pressure is the commonest complaint (63–100% cases), followed by visual deficits (40–100%), cranial nerve palsies, and vomiting. Here, we report a case of pituitary apoplexy with bilateral total ophthalmoplegia having complete oculomotor nerve palsy in one eye and pupil-sparing oculomotor nerve palsy in the other.

CASE REPORT

A 52-year-old woman presented to our outpatient department with a 4-day history of acute onset severe headache localized in the frontal and occipital areas, swelling over both eyes and inability to open the eyes. She was conscious and oriented. On examination, her temperature was 39°C, pulse was 84 beats per minute, and blood pressure was 126/88 mm Hg. Ocular examination revealed vision of finger counting at 3 m in both eyes. She had a chin up head posture and the movements of extraocular muscles were restricted in all gazes in both the eyes except levoversion which was present in the left eye. Exodeviation was seen in the left eye (Fig. 1). However, she did not complain of diplopia. Anterior segment examination revealed edema over eyes, upper and lower lids, bilateral ptosis and 6 mm round, nonreacting pupil of the left eye (Fig. 2). Rest of the anterior segment was normal. Corneal sensations were present in both the eyes. Fundoscopy...
revealed healthy optic disk with clear disk margins and cup disk ratio of 0.3:1 with normal macula and periphery in both eyes. Visual fields could not be assessed as the patient was not cooperative. No other focal neurological signs were present. Differential leukocytic count was done, which showed 50% neutrophils and 42% lymphocytes. Random blood sugar was 118 mg/dL. A noncontrast computed tomography of the brain was reported to have a $2.2 \times 1.7$ cm sized hyperdense mass in the region of sella turcica extending above the suprasellar region and bulging above the cavernous sinus on both sides. The mass showed Hounsfield unit values of +71, suggestive of bleed within the mass (Fig. 3). Magnetic resonance imaging (MRI) of the brain using T1, T2, and Fluid Attenuated Inversion Recovery (FLAIR) sequences (Figs 4 to 6) showed $2.6 \times 1.8 \times 2.4$ cm sized T2 hyperintense and T1 heterointense lesion within the sella causing ballooning of the sella and extending into the suprasellar cistern causing displacement of both the internal carotid arteries and elevation of the optic chiasm. Bony erosion of the inferior aspect of sella was noted. Peripheral enhancement on T1 postcontrast with a central hypointensity was noted in the left cavernous sinus, which was suggestive of left cavernous sinus thrombosis (Fig. 7).
Bulging of the lateral wall of cavernous sinus was noted, more on the right side. A diagnosis of pituitary apoplexy within a pituitary macroadenoma was made based on the radiological findings. After discussion with neurosurgical colleagues, she was started on injection hydrocortisone 100 mg 8 hourly. Prolactin levels, growth hormone levels, and thyroid profile tests were advised. Patient went to another hospital thereafter for further treatment.

DISCUSSION

Pituitary apoplexy was first described by Bailey in an acromegalic patient. The term “pituitary apoplexy” was coined years later by Brougham et al. It is a potentially life-threatening disorder occurring due to acute ischemic infarction or hemorrhage within the pituitary gland, usually within a pituitary tumor. Most cases of pituitary apoplexy present in the fifth or sixth decade with a slight male preponderance ranging from 1.1 to 2.25:1.0. The exact pathogenesis of pituitary apoplexy is still unclear. Various theories have been put forward regarding the pathophysiology of pituitary apoplexy. One theory is that rapid tumor growth may outstrip arterial supply, thereby causing ischemic necrosis and hemorrhage. The size of the adenoma appears to be a major factor, but even microadenomas can bleed. Another theory is that the tumor growing inside the narrow space situated between the pituitary stalk and diaphragm sellae leads to constriction of the thin vascular network and finally ischemia, necrosis, and hemorrhage on the anterior lobe and tumor tissue. But the adenoma is supplied by the inferior hypophyseal artery and the compression of the superior hypophyseal artery and its branches against the diaphragm sellae could lead to ischemia of the anterior pituitary. Also the hemorrhage could be the result of invasion of the vessel wall by the tumor cells causing rupture of the vessel wall. Out of

![Fig. 6: Fluid attenuated inversion recovery images](image1)

![Fig. 7: T2 postcontrast images showing enhancement of the lesion](image2)
the many risk factors causing pituitary apoplexy, hypertension is the commonest (26%) predisposing factor. The earliest and most common presentation of pituitary apoplexy is headache (up to 100%), followed by ocular palsies in 70% patients. Headache is usually retro-orbital or bifrontal. Ocular motor nerve palsies and trigeminal dysfunction usually occur due to involvement of the cavernous sinus by the erosion of thin sellar wall. Third, fourth, and sixth cranial nerves are commonly involved in case of pituitary apoplexy. Decreased visual acuity and visual field defects, specifically bitemporal hemianopia, are seen in nearly 75% of the patients and are caused by upward enlargement of the intrasellar contents, leading to optic chiasmal compression. Extravasation of blood or necrotic tissue into the subarachnoid space can cause meningism, resulting in fever, photophobia, and altered consciousness level.

Management of pituitary apoplexy is controversial. In some cases, a conservative approach with administration of high-dose corticosteroids along with hormone replacement therapy is indicated, while in others early trans-sphenoidal surgical decompression is advocated. Prognosis of pituitary tumor apoplexy has improved with declining morbidity and mortality because of early diagnosis, better therapeutic support, use of glucocorticoids, and refinement of surgical and postoperative techniques. Urgent surgical decompression is indicated when the patient has severe neuro-ophtalmic signs with sudden visual loss, diminished level of consciousness, or hypothalamic disturbance. On the contrary, stable patients having no visual field defects, neuro-ophtalmic signs, or mental status changes are, however, managed conservatively with a regular evaluation of electrolyte and fluid balance. In adults, hydrocortisone 100 mg intramuscular (IM) bolus, followed by 50 to 100 mg 6 hourly by IM injection or 100 to 200 mg as an intravenous (IV) bolus followed by 2 to 4 mg per hour by continuous IV infusion can be used. Surgical decompression is indicated if there is no improvement seen after 24 to 48 hours of treatment. In patients undergoing surgical decompression improvement is seen within 1 week of the surgery.

Diabetes insipidus is the commonest presentation in case of a pituitary adenoma. However, in our patient, there were no urinary complaints. Also, the signs of meningism, which is a usual presentation in a case of pituitary apoplexy, were not seen in this case. The appearance of symptoms was insidious in onset without any predisposing or any precipitating event. Bilateral ophthalmoplegia can be attributed to the cavernous sinus involvement. However, the affection of cranial nerves was different in both the eyes, with the involvement of third and sixth cranial nerve in one eye and only the third cranial nerve in the other. Also one eye presented with total third nerve palsy, while the other had pupil sparing third nerve involvement.

CONCLUSION

Pituitary apoplexy is an uncommon and underdiagnosed complication of pituitary adenomas having varied clinical presentations. It can mimic a wide spectrum of clinical conditions due to its myriad signs and symptoms. The constellation of signs must alert the clinician to the possibility of pituitary tumor apoplexy. Timely diagnosis by imaging studies and early treatment can help decrease the morbidity and mortality.

REFERENCES