Case Report

Pituitary apoplexy presenting with isolated third nerve palsy

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We report a 61-year-old gentleman who presented with sudden onset of worsening headache, diplopia and blurring of vision on the left eye. Examination showed presence of relative afferent papillary defect, with visual acuity of 1/60 in the affected eye. Additionally, there was third nerve palsy with pupil involvement. An urgent MRI of the brain imaging confirmed hemorrhagic pituitary tumor with upward compression onto optic chiasma and the upper part of cavernous sinus. Emergency transphenoidal decompression resulted in significant improvement of neurological signs and vision. (Rawal Med J 201;40: 122-124).

Keywords: Pituitary apoplexy, third nerve palsy, transphenoidal decompression.

INTRODUCTION

Pituitary apoplexy is a sudden neurological or endocrine dysfunction arising from acute infarction or hemorrhagic of a pre-existing pituitary mass. Sudden expansion of the sellar contents lead to compression of the adjacent structures, resulting in typical symptoms of headache, focal neurological signs, altered mental status or multiple hormonal deficiencies. The progression of this condition varies from acute onset with rapid deterioration, or evolving subacute and commonly non-specific symptoms. The occulomotor nerve is the first nerve at the superior part of the cavernous sinus, and isolated involvement is rarely seen. Isolated third nerve impairment may be an important sign, which points to vision and life threatening pathology. Prompt diagnosis is life- saving.

CASE REPORT

A 61-year-old gentleman presented with three days history of severe headache, drooping left eyelid, blurring of vision and diplopia. He denied history of trauma prior to the complaints. There was no history of loss of consciousness, altered mental status, seizure, neck stiffness or body weakness. He had hypertension and was on treatment. Visual acuity was 1/60 with obvious left eye ptosis, and ipsilateral limitation of adduction, elevation and downward eye movement (Figure 1). Anisocoria was also noted with left dilated pupil and sluggish reaction to light. Anterior segment examination was normal but the optic disc was pale with mild blurring of the disc margin. Other cranial nerves were unaffected.

There was bitemporal hemianopia and left inferonasal quadrantanopia on visual field test (Figure 2). An urgent MRI showed a hemorrhagic mass in pituitary fossa which compressed the optic chiasma superiorly (figure 3). Hematological investigation revealed low serum cortisol, thyroid stimulating hormone and testosterone level. Systemic hydrocortisone was initiated prior to surgical emergency transphenoidal decompression and excision of the pituitary tumour.

Figure 1. Extraocular movement of left eye was restricted on elevation, adduction and depression.

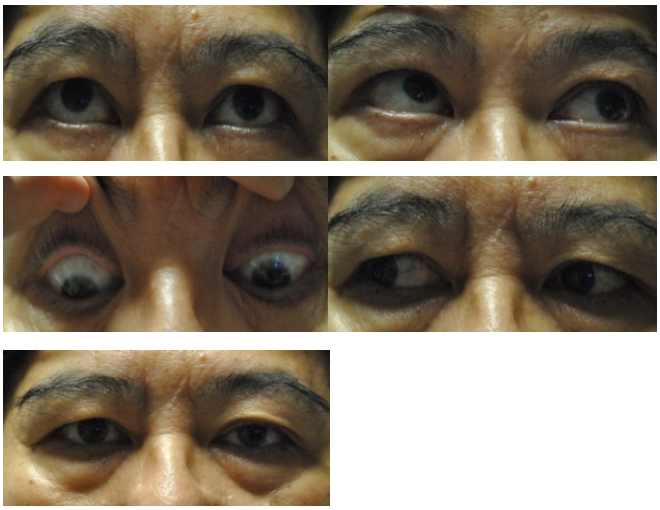


Figure 2. MRI scan of the brain showing a mass in pituitary fossa with signs of hemorrhage and upward compression of the optic chiasma.

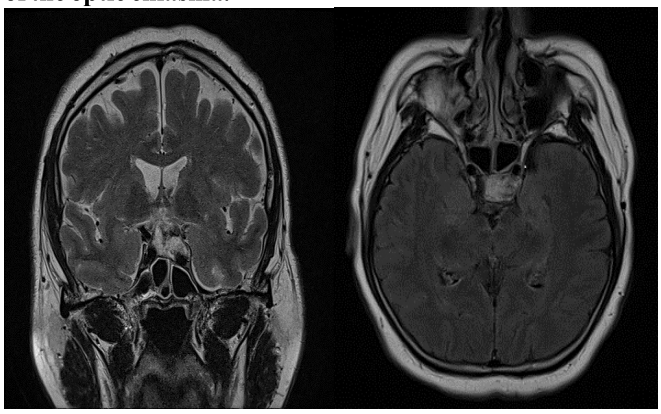
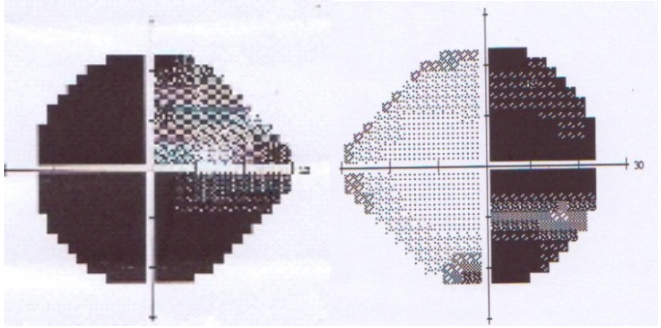
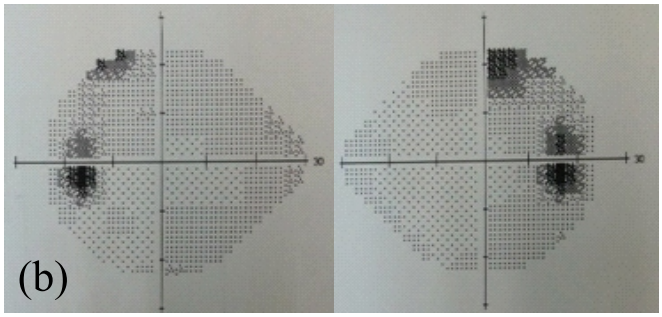


Figure 3 (a) and (b). Humphrey visual field test.



(Left eye) (Right eye)

Pre-operation: bitemporal hemianopia with left inferonasal quadrantanopia



(Left eye) (Right eye)

Intraoperatively, sella turcica was approached via transphenoidal route and blood clot was evacuated. The patient recovered well and at one month follow-up, his left visual acuity improved to 6/6, with recovery of ptosis and extraocular movement. The pupil size returned to normal with almost comparable reaction to light with the contralateral right eye. His ocular condition remained well and stable during his follow up in eye clinic a year after operation. Currently, he is on maintenance hydrocortisone, thyroid hormone and testosterone replacement therapy. His vision remained good extraocular muscle movements were back to normal.

DISCUSSION

Pituitary apoplexy occurs from spontaneous hemorrhage or infarction of pre- existing pituitary adenoma. The rate of hemorrhagic infarction may be as high as 15 percent.[1] The adjacent structures at risk of collateral damage from this condition include the optic chiasma and cavernous sinus with its contents. Sudden expansion of the sellar contents from fluid or blood extravasations causes compression of the immediate surrounding structures, resulting in neurological deficits. The non-specific symptom of headache arises from meningeal stretch due to increase in the intracranial pressure. In severe cases, this condition may lead to permanent blindness, coma and death. Compression of occulomotor, trochlear, trigeminal and abducens nerves in the affected cavernous sinus can occur. Consequently, this results in various degree of ophthalmoplegia, usually involving multiple cranial nerves due to their proximities at the wall or within the cavernous sinus.[2] Isolated cranial nerve involvement, particularly, the third nerve as a presenting sign of pituitary apoplexy is rare.[3,4,5,7] Anatomically, the oculomotor nerve traverses the lateral wall of cavernous sinus and lies superiorly to trochlear and trigeminal nerves. It is approximately lies at the same level of the pituitary gland horizontally. The blood supply of the intracavernous part of third cranial nerve is derived from the inferior cavernous sinus artery. This fact explains the focal neurological signs of anisocoria, ptosis and limitations in extraocular muscle movements corresponding to oculomotor nerve palsy in conditions affecting the lateral wall of cavernous sinus. In particular, lateral expansion of the sellar contents from edema or hemorrhage of pituitary tissue in pituitary apoplexy results in similar clinical presentation.[6] Hence, there is a close relationship between the blood supply of the intra-cavernous third cranial nerve and pituitary gland.[8]

Close proximity of other cranial nerves result in neurological deficit corresponding to the impairment of cranial nerves IV and V. It is quite rare, therefore, for pituitary apoplexy to only present with isolated third cranial nerve palsy. A possible explanation is compromise of the blood supply to the intra-cavernous third cranial nerve due to expansion of sellar contents which attributes to acute isolated third cranial nerve palsy in pituitary apoplexy.[7] In our case, the most likely mechanism is direct compression of the third cranial nerve at the superior part of the lateral wall of the cavernous sinus which was supported by the MRI findings.

The visual outcome in pituitary apoplexy patients is good in surgical decompression and conservative group. Bill et al have reported that the recovery of visual acuity was 88%, visual field defect was 92% and ophthalmoplegia was 100% post surgical decompression.[9] Timing of the surgical intervention is an important factor for visual prognosis. Early surgical intervention within one week after clinical presentation and diagnosis was recommended in order to achieve full recovery of vision.[10]

Almost all patients need a certain amount of hormone replacement therapy following an episode of pituitary apoplexy. The recurrence rate of pituitary tumor post pituitary apoplexy is still unclear.[11] Thus, all patients should have a proper endocrine and neuro-ophthalmology follow up after pituitary apoplexy, with annual MRI imaging of the brain to detect any recurrence. Visual field should be performed to aid the neurosurgeon in identifying any recurrence or new change to the primary lesion. Regular monitoring based on hematological parameters is important to ensure that hormone level is within normal range, and patient may continue with daily activities without hindrance.

Isolated oculomotor palsy with pupil involvement is usually attributed to posterior communicating artery aneurysm. Our case highlights that pituitatry apoplexy may also come with similar presentations, with additional visual loss due to the close proximity with optic chiasm. Early diagnosis and prompt intervention is crucial to ensure good clinical outcome,[7] as shown in this case.

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