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Thalamic infarction in pituitary apoplexy syndrome

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DESCRIPTION

A 36-year-old man presented with drooping of both eyelids and diminution of vision in both eyes for 7 days, preceded by fever and headache for 15 days. He had diplopia, accentuated in looking forward. He did not complain of difficulty in mastication, impaired swallowing, weakness in any limb or fluctuations in symptoms. The patient's history was negative for head trauma, anticoagulants and other drugs or endocrinopathy. A general physical survey revealed bilateral gynaecomastia. He had bilateral vision loss, visual acuity being 6/24 in right eye and 6/36 in left eye. Right pupil was dilated with sluggish reaction and left pupil was mildly dilated with impaired reaction. The patient had bilateral ptosis which was more marked in left eye. On primary gaze, left eye was abducted and externally rotated (figure 1). Adduction and upward movements of both eyeballs were restricted (figure 2). Thus, he had a partial right oculomotor (third

cranial nerve) palsy and a complete left oculomotor palsy. Rest of the neurological examination was unremarkable. Routine hematological and biochemical parameters were normal. Hormonal assay revealed growth hormone, 0.06 ng/ml (normal range 2.1–17.7); thyroid stimulating hormone, 21.83 μ IU/ml (0.35–5.5); T3, 31 ng/dl (60–200); T4, 1.5 μ g/dl (4.5–12) and serum prolactin, 3.71 ng/ml (normal 2.1–17.7). MRI cranium revealed heterogeneous-enhancing extraxial space occupying lesion in sellar region with suprasellar extension diagnostic of pituitary macroadenoma (figure 3). Other findings were acute infarct in left thalamus and adjoining midbrain (figure 4). Thus, a diagnosis of pituitary apoplexy with thalamic and mid-brain infarction was made. He was treated with steroids and an emergent transphenoidal decompression surgery for pituitary macroedema. Histopathology of the resected tumour showed large areas of ischaemic necrosis with round to polygonal tumour cells and dispersed chromatin

Figure 1. Patient photograph revealing ptosis, more marked in left eye. On primary gaze, left eye is abducted and externally rotated.

Figure 2. Patient image showing restriction in adduction and supraduction.

Figure 3. T1 MRI contrast image revealing mass in sellar and suprasellar region with mixed hypointense and hyperintense signals and rim enhancement.

consistent with diagnosis of pituitary adenoma (figure 5). In follow-up patient showed remarkable clinical improvement.

Pituitary apoplexy syndrome occurs due to infarction or haemorrhage in pituitary gland or a pre-existing adenoma and presents with vision loss, ophthalmoplegia, impaired mentation, meningismus and endocrinal abnormalities.¹

Figure 4. T2 fluid attenuated inversion recovery showing infarct in left thalamic region.

Figure 5. Histopathology showed large areas of ischaemic necrosis with round to polygonal tumour cells and dispersed chromatin consistent with diagnosis of pituitary adenoma.

A concurrent cerebrovascular episode with a stroke is a rare occurrence. Previously described case reports have mentioned pituitary apoplexy-associated strokes in internal or middle cerebral artery territory presenting with hemiparesis. However, isolated thalamic stroke has not been yet described. According to existing literature, stroke in such cases has been described to occur due to one of the following mechanisms²⁻³: (1) compression of intracavernous portion of internal carotid artery due to expanding pituitary adenoma or a haemorrhage within it; (2) vasospasm caused by factors released from haemorrhagic or necrotic material. Thalamic infarction in our patient was probably due to vasospasm secondary to factors released from haemorrhagic or necrotic material from pituitary. However, exact explanation usually remains elusive in such cases.

Learning points

- ▶ Pituitary apoplexy syndrome although rare is a life-threatening neurological disorder of emergent nature.
- ▶ Thalamic infarction has not been reported previously in the literature.
- ▶ So any patient presenting with bilateral ptosis, vision loss and impaired mentation which can occur in posterior territory stroke should also be examined for pituitary apoplexy syndrome.

Competing interests None.

Patient consent Obtained.

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