Pituitary apoplexy presenting with bilateral oculomotor nerve palsy

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DESCRIPTION

An 82-year-old man with a history of pituitary macroadenoma, was admitted for sudden onset binocular diplopia and headache. Physical examination showed complete ptosis of both eyes ([video 1](#)). He had bilateral oculomotor nerve palsy and right abducens nerve palsy ([videos 2 and 3](#)). The horizontal vestibulo-ocular reflex was impaired. The pupillary reflex was also impaired ([video 4](#)). Visual field was normal and limb power was full. Serum sodium level was 112 mmol/L (N 136–145) and 9:00 cortisol

Video 4 Pupillary reflex was impaired.

Video 1 Complete ptosis of both eyes.

Video 2 Bilateral oculomotor nerve palsy and right abducens nerve palsy.

Video 3 Bilateral oculomotor nerve palsy and right abducens nerve palsy.

level was 57 nmol/L (N 171–536). Thyroid function test was normal. MRI of the brain revealed a pituitary macroadenoma with haemorrhage abutting bilateral cavernous sinuses and optic chiasm ([figures 1 and 2](#)). Trans-sphenoidal total excision of pituitary tumour was urgently performed; the oculomotor nerve palsy improved partially after the operation.

Pituitary apoplexy presenting with bilateral oculomotor nerve palsy is rare.^{1 2} Pituitary apoplexy is a potentially life-threatening disorder characterised by sudden onset of headache, visual disturbances, ophthalmoplaegia and impairment of consciousness. It is caused by an infarct or haemorrhage of the pituitary gland.³ Acute secondary adrenal insufficiency, which is the major cause of mortality, is seen in about two-thirds of patients with pituitary apoplexy. Hypocortisolaemia increases vasopressin release from the posterior

Figure 1 MRI T2W axial image shows heterogeneously hyperintense pituitary mass expanding the sella turcica, which is suggestive of subacute haemorrhage (arrow). T2W, T2-weighted.

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Figure 2 MRI T1W coronal image with gadolinium contrast shows heterogeneously contrast-enhanced pituitary mass with suprasellar extension abutting the optic chiasm and bilateral cavernous sinuses (arrow). T1W, T1-weighted.

pituitary and has an inhibitory effect on water excretion, contributing to fluid and electrolyte disturbances.³ Urgent corticosteroid replacement with hydrocortisone 100–200 mg intravenous bolus followed by continuous intravenous infusion of 2–4 mg/h is needed in patients with suspected hypoadrenalism. Prompt correction of electrolyte and fluid disturbances is life-saving. Neurosurgical decompression should be considered in patients with severe visual impairment.³ Long-term hormonal replacement therapy, including corticosteroids, desmopressin and sex hormones, is needed in about 80% of patients after

pituitary apoplexy. Long-term follow-up with hormonal evaluation and eye assessment is required in this group of patients.³

Learning points

- ▶ Pituitary apoplexy presenting with bilateral oculomotor nerve palsy is rare.
- ▶ Acute secondary adrenal insufficiency, which is the major cause of mortality, is seen in about two-thirds of patients with pituitary apoplexy. Urgent replacement of corticosteroids, and correction of electrolyte and fluid disturbances are life-saving.
- ▶ Neurosurgical decompression should be considered in patients with severe visual impairment.

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