Clinical Reports

Giant cell arteritis presenting as oculomotor nerve palsy with pupillary dilatation

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Summary: Acute complete oculomotor palsy with headache is a classical presentation of an extrinsic compression most commonly due to a posterior communicating artery aneurysm. We present a patient with such a presentation but with histologically proven giant cell arteritis. This possibility should be considered especially in the elderly to avoid complications and the need for angiography.

Introduction

An acute unilateral oculomotor nerve palsy associated with headache strongly suggests compression by a posterior communicating artery aneurysm. Giant cell arteritis (GCA) can rarely cause ocular palsies with pupillary sparing and preservation of vision. We describe a case of histologically confirmed GCA presenting with a complete third nerve palsy.

Case report

A 74 year old female presented with a 2 week history of throbbing headache and intermittent diplopia on left lateral gaze. On the day of admission she awakened with an exacerbation of her pain with complete right ptosis. There was no past history of diabetes or hypertension.

On examination she was noted to have a pyrexia at 38.1°C and an isolated right third nerve palsy with a large non-reactive pupil (Figure 1). Visual acuity and fundoscopy were normal as was the rest of the general physical examination. The right temporal artery was not pulsatile nor tender.

Routine haematology demonstrated a normocytic normochromic anaemia with mild thrombocytosis and an ESR of 57 mm/first hour. Serum chemistry was normal including glucose estimation on several occasions. Dynamic thin section serial computer tomographic (CT) imaging of the retro-orbital space and parasellar region including the circle of Willis showed no evidence of intracranial aneurysm. Lumbar puncture failed to demonstrate the presence of blood or xanthochromia in the

subarachnoid space. Temporal artery biopsy confirmed the diagnosis of GCA with typical features of severe perivascular cellular reaction, intraluminal thrombosis and loss of the elastic coat.

Eighteen months following admission and commencement of steroid therapy the haematological indices returned to normal. There was partial improvement in ocular motility and ptosis.

Discussion

Pupillary sparing is the hallmark of an ischaemic oculomotor palsy, as seen classically in diabetes, whereas pupillary involvement is taken as an indication of compression commonly from a posterior communicating artery aneurysm. There are few exceptions to the rule in either situation.¹ Dynamic thin-section orbital CT scanning has been shown to exclude a symptomatic aneurysm adequately.² In our patient the negative CT and the normal cerebrospinal fluid study made us quite confident in excluding a symptomatic posterior communicating artery aneurysm as a cause of this patient's complete third nerve palsy. Ophthalmoparesis is a recognized manifestation in GCA. Histologically proven arteritis involving the posterior cerebral arteries (the blood supply to the third nerve) was reported by Fisher.³ Another cause of ocular palsy in GCA is ocular muscle ischaemia which was histologically noted in some patients without involvement of the oculomotor nerves.⁴ In oculomotor palsy in GCA the pupil is usually spared and this is due to the peripheral position of the pupillary fibres in the oculomotor nerve, the brunt of the ischaemia being more

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Figure 1 A complete right oculomotor palsy with pupillary dilatation.

centrally placed within the nerve. In our patient the partial improvement 18 months later would suggest that the involvement of the oculomotor nerve was more severe which accounts for the additional pupillary involvement. In rare reports of pupillary involvement with oculomotor palsy, unlike our case, GCA has not been confirmed histologically or there was associated blindness.⁵

This case illustrates that a painful oculomotor palsy with pupillary dilatation especially in an elderly person is not an immediate indication for carotid angiography. Other causes such as GCA have to be considered as they can cause morbidity and perhaps mortality if they are not recognized and treated early.

References

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