Double Posterior Communicating Artery as a cause of Oculomotor Nerve Palsy

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Abstract:
Amongst clinicians the diagnostic approach differs to patients presenting with isolated third cranial nerve palsy. Some issues remain controversial and the management of this condition is a challenge. In a 38-year-old male presenting with pupil-involving oculomotor nerve palsy (internal dysfunction) the cause was found to be a double posterior communicating artery; a very rare cause of third nerve palsy.

Introduction:
Oculomotor nerve palsy can result from lesions located anywhere from the oculomotor nucleus in the midbrain to the termination of the third nerve in the extra-ocular muscles within the orbit (1). It can be an isolated or a non-isolated lesion according to the presence of other neurological deficits and it can be classified into internal dysfunction (pupil-involving) and external dysfunction (pupil-sparing) (2). The diagnosis and treatment of this condition vary according to the age of the patient, the characteristics of the third nerve palsy, and the presence of associated signs and symptoms (1).

Case Report:
A 38-year-old male presented to the Emergency Department of Hamad General Hospital with a history of headache and diplopia of two days duration. There was no history of fever, vomiting, dizziness, weakness, loss of consciousness, or trauma. He was not hypertensive or diabetic and had an unremarkable past medical history. He was conscious and oriented with blood pressure 121/74 mm Hg, pulse rate 72 beats/min, temperature 36.7°C, respiratory rate 14/min. Examination of the chest, heart, and abdomen showed no abnormalities. Complete blood counts, urea and electrolytes, coagulation profile, were all within normal limits. Chest x-ray and ECG were unremarkable.

Examination of the central nervous system revealed partial right ptosis and a dilated right pupil not reacting to light, with normal eye movements in all directions. There was no nystagmus or papilledema. The left eye was normal. The rest of CNS examination, including computed tomography of the brain, was normal.

In order to rule out posterior communicating artery aneurysm the patient was admitted and further investigations showed erythrocyte sedimentation rate (ESR) 1 mm/h, C-reactive protein (CRP) 1 mg/l, ALT 23 u/L, AST 25 u/L, total bilirubin 19 μmol/L, cholesterol 4.94 mmol/l, triglyceride 1.7 mmol/l, homocysteine 11.5 μmol/L, C3 112 mg/dl, C4 17.9 mg/dl, ANA negative, ANCA screening negative, Treponema pallidum antibody negative, protein C clotting 79.1%, protein S clotting 111.4%, and antithrombin III 78%.

MRI of the brain and non-contrast intracranial MRA showed right-sided double posterior communicating artery, no obvious aneurysms in the regions of the Circle of Willis, no arterial occlusion or stenosis (Figs. 1, 2)

Discussion:
Etiologies of oculomotor nerve palsies include a wide variety of pathologies; congenital, traumatic, infectious, inflammatory, and ischemic (the most common cause of pupil-sparing third nerve palsy) but the most dreaded and most common cause of pupil-involving third nerve palsy is compression by intracranial aneurysms (3,4).

Ischemic 3rd nerve lesions typically present with intact pupillary function, probably because of lack of damage to the superficial periphery of the third nerve where the majority of the pupillomotor fibers are thought to pass (5). Even if the pupil is involved impairment is usually incomplete. It is more common in diabetic patients, which is why it is called diabetic third and the pathogenesis is thought to be microvascular.
The most common site of an intracranial aneurysm causing oculomotor nerve palsy is the posterior communicating artery but aneurysms involving the internal carotid artery and the basilar artery are reported also to produce third nerve palsy. The posterior communicating artery originates from the internal carotid artery and is about 8-15 mm in length from the point of origin to the point of union with the posterior cerebral artery.

The mean age of patients with posterior communicating artery aneurysm is 55 years although aneurysms have been reported also in young children and in the elderly. The most common site of aneurysm is the junction between the posterior communicating artery and the internal carotid artery according to Fujiwara et al who found 25 out of 26 aneurysms located at that site.

Management of third nerve palsy depends upon localization of the lesion and determination of the etiology. Clinical features such as pain, speed of onset, and completeness of palsy are not reliable in the diagnosis of either the nature or the location of the cause, pupil involvement is, however, often associated with compressive lesions, so the degree of external and internal dysfunction can direct the type of neuro-imaging needing to be performed.

Imaging studies used to diagnose or exclude an intracranial aneurysm include MRI with MR angiography (MRA), computed tomographic angiography (CTA), and cerebral angiography. Cerebral angiography remains the gold standard test but it is invasive and associated with infrequent but significant risk. Contrast enhanced MRI with MRA has a sensitivity as high as 95%-98% for detection of aneurysms in the setting of third nerve palsy and a specificity of 100%.

In the case reported here the patient presented with headache and dilated non-reacting pupil that could have heralded the presence of a life-threatening posterior communicating artery aneurysm but the MRI with MRA examination showed a double right posterior communicating artery, which is rare and has not been reported before as a cause of third nerve palsy.

Treatment of oculomotor nerve palsy depends upon the cause. For patients with complete external dysfunction (pupil-sparing) most commonly caused by ischemic injury, observation alone is an appropriate option although contrast enhanced brain MRI and MRA should be considered if there is deterioration or no improvement by 6-12 weeks. Patients who do not have a history of diabetes or hypertension and in whom pupil-involving third nerve palsy develops should undergo MRI with MRA imaging initially to exclude the presence of structural lesions compressing the oculomotor nerve.
References