Painful Isolated Third Nerve Palsy: A Life Saving Diagnosis

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Abstract: Intra cranial aneurysms are the most common cause of isolated oculomotor nerve palsy with pupillary involvement. Aneurysm is suspected, particularly when the patient has a history of sudden severe pain in or around the eye. Oculomotor nerve palsy is a sign of alarm in such cases, as it usually is an initial manifestation of the expansion of this vascular malformation. The rupture of an aneurysm leads to a sudden sub-arachnoid hemorrhage with a high rate of morbidity and mortality. Timely neuro-radiological diagnosis and treatment can prevent this catastrophic outcome. Here, we report a case of 47 year old male patient who presented with acute onset painful isolated third nerve palsy. He was admitted in the emergency department of our hospital with the chief complaint of severe headache which in his words was “this is the worst headache of my life”. MRI was done which was reported to be normal. Magnetic resonance angiogram was done suspecting aneurysm of posterior communicating artery. This revealed a 3mm×5 mm aneurysm of the posterior communicating artery. Patient underwent left parietal craniotomy for clipping of aneurysm and recovered well.

Keywords: Aneurysm, Posterior communicating artery, Magnetic Resonance Angiogram, Isolated 3rd nerve palsy, Craniotomy

INTRODUCTION:
Oculomotor nerve palsy may be congenital or acquired, partial pupil sparing or pupil involving, isolated or accompanied by signs of more extensive neurological involvement. Precise knowledge of its origin and its course, along with accompanying clinical features, help in localising the site of involvement and thus appropriate management. Associated symptoms carry definite importance and patient should be thoroughly asked for headache and periorbital or orbital pain.

In isolated IIIrd nerve palsies, the sudden and painful onset with meningeal signs warrant immediate CT without contrast to look for subarachnoid haemorrhage (SAH), followed by CT with intravenous contrast or Gadolinium MRI to look for aneurysm or any other cause and to know about the extent of the IIIrd nerve involvement, irrespective of the age of the patient. Magnetic resonance angiography (MRA) provides the most sensitive way to detect aneurysm as small as 2-3 mm. However catheter angiography remains the gold standard for the diagnosis of intracranial aneurysm. Basilar and contra lateral circulation should also be studied as 20% patients have multiple aneurysms. Diabetic micro vascular IIIrd nerve palsies are commonly painful and usually pupil sparing.

CASE HISTORY:
A 47 year old man was referred from the medicine department with the chief complaint of headache and “droopy eyelid”. He was non diabetic and non hypertensive. Patient complained of headache since 15 days followed by the drooping of the eyelid five days before consultation in the hospital. He was examined thoroughly. Vision was 6/6 right eye and 6/18 after retracting the upper lid. Colour vision was normal. On examination, he demonstrated complete left sided ptosis, non reactive, dilated pupil, and downward and lateral deviation of the left eye. Fundus was normal.

The patient underwent conventional unenhanced CT scan and MRI Scan of the brain. The study was negative for any mass, bleed, or acute cerebrovascular accident. Patient was advised regular follow up after seeing the MRI Report to be negative. Same night patient again came to the hospital emergency with the chief complaint in his own words as “THE WORST HEADACHE OF MY LIFE”. He was admitted with high clinical suspicion of a vascular emergency. MRI Scan was reviewed thoroughly and magnetic resonance angiogram imaging of the brain was done. This revealed a 3 mm×5 mm aneurysm of the posterior communicating artery. Patient underwent left
parietal craniotomy for clipping of aneurysm and recovered well.

Fig 1: Left Sided Ptosis before Surgery

Fig 2: Recovery after Surgery

Fig 3: Left Sided Ptosis and Lateral Deviation of Left Eye before Surgery
Fig 4: Recovery after Surgery

Fig 5: MRA showing aneurysm of posterior communicating artery
DISCUSSION

Painful third nerve palsy is a well known presenting sign of a posterior communicating artery aneurysm. Intracranial aneurysms are the most common cause of isolated oculomotor nerve palsy with pupillary involvement, particularly when the patient has a history of sudden severe headache.

Pupillary dysfunction with third nerve palsy results from loss of parasympathetic input, which produces a dilated pupil that responds poorly to light. The aneurysm usually arise from the junction of internal carotid and posterior communicating arteries, a location in close proximity to third cranial nerve. Such aneurysm may injure the oculomotor nerve by direct compression, from a small haemorrhage, or at the time of major rupture. Trauma to the oculomotor nerve may occur during aneurysm surgery. Thus non-traumatic third nerve palsy with pupillary involvement, or evidence of progression to pupillary involvement, must be assumed to be secondary to an aneurysm until proven otherwise [1].

Painful oculomotor nerve palsy with pupillary involvement may result from posteriorly draining, low-flow carotid –cavernous sinus fistula. Tumours and others compressive lesions, such as ectactic posterior cerebral or basilar artery vessels, can stretch or compress the oculomotor nerve in the interpeduncular fossa. Intrinsic lesions of the oculomotor nerve, such as schwannomas or cavernous angiomas, can also produce an acute or progressive oculomotor nerve paresis.

Vascular diseases, particularly diabetes mellitus, often produce an oculomotor nerve palsy that spares the pupil. All patients should therefore have blood measurement, urinalysis and plasma glucose estimation. In most cases, spontaneous recovery occurs within 3 months. Diabetic third nerve palsy is often associated with periorbital pain and may occasionally be the presenting feature of diabetes.

Apart from aneurysms, pituitary tumours can cause third nerve palsy by several mechanisms. It may occur slowly secondary to mechanical compression against the interclinoid ligament, or by compression and invasion of the cavernous sinus by the tumour. Secondly it may occur rapidly, associated with headache due to compressive effects or by compromise of the vascular supply to the nerve itself [2].

The nuclear complex of the third (IIIrd) nerve lies in midbrain at the level of superior colliculus, ventral to sylvian aqueduct, straddling the vertical midline [3, 4]. Each target muscle has its own sub nucleus. Although the anatomical division of the third
craniocavernous nerve occurs in the region of the anterior cavernous sinus or superior orbital fissure, there is a topographical arrangement of the motor fibers within the cisternal portion of the nerve. The clinical evaluation of a patient with a third cranial nerve palsy requires an understanding of the regional neuroanatomy and topographical organization of the nerve [5].

In a study, the causes of ophthalmoplegia in 24 consecutive patients with neurologically isolated, relative pupil-sparing third nerve palsy included, infarction in 10 patients, compression by tumors or aneurysms in 10 patients, and miscellaneous disorders in four patients. There was no significant difference in the proportion of patients with pain, degree of external ophthalmoplegia, or degree of internal ophthalmoplegia between the groups with infarction or mass lesions. Therefore, screening for mass lesions using neuroimaging is indicated in patients with this presentation of third nerve palsy [6].

Radiographic correlation with clinical symptoms was frequently not possible before the advent of magnetic resonance (MR) imaging. MR imaging has proved invaluable in confirming the site of the lesion in many patients with cranial nerve III palsy, because it allows the observer to scrutinize the entire course of the oculomotor nerve and surrounding structures. A characteristic appearance of the nerve is noted in different pathophysiologic processes. A previous study [7] has described the enhancement of the cisternal segment of the oculomotor nerve as a neurora diologic finding most often related to lepto meningeal inflammation or neoplastic infiltration of the nerve.

The clinical dictum that pupil sparing in oculomotor nerve palsy predicts an extra axial ischemic lesion while pupil involvement predicts an extra axial compressive lesion has some important exceptions. Two case reports and a review of the literature disclose that pupil sparing occurs in a small proportion of intra axial and compressive subarachnoid oculomotor nerve lesions and in a large proportion of compressive cavernous sinus oculomotor nerve lesions. Careful examination is also necessary to separate examples of apparent pupil sparing in cases of aberrant regeneration and in some instances of cavernous sinus compression [8].

If aneurysm is found to be the cause of 3rd nerve palsy then emergency craniotomy and clipping of aneurysm is indicated, to prevent rupture of the aneurysm and subsequent intracranial haemorrhage.

Conventional unenhanced CT scan and MRI have high false-negative rates and therefore cannot exclude the diagnosis. Patients must therefore undergo magnetic resonance angiogram or CT angiography, which have sensitivity up to 97%. Early intervention can improve return of neural function and avoid a catastrophic rupture, which carries an unacceptable rate of morbidity and mortality. Definitive treatment includes surgical clipping or endovascular coil embolization.

**CONCLUSION**

Intra cranial aneurysms are the most common cause of isolated oculomotor nerve palsy with pupillary involvement. Magnetic resonance angiography (MRA) provides the most sensitive way to detect aneurysm as small as 2-3 mm. Craniotomy for clipping of aneurysm is the definitive treatment in such cases.

**REFERENCES**