Posterior Communicating Artery Aneurysm in a 20 Year Old Boy Presenting as Non-Isolated Third Nerve Palsy.

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SUMMARY

Objective: The need for neuroimaging in non-isolated third nerve palsy remains uncertain. Even with associated partial pupillary involvement, the possibility of aneurysm is said to be low. We highlight the need for vigilance for possible life-threatening aneurysms in cases of non-isolated third nerve palsies.

Method: Case report of a 20 years old boy who presented with drooping of the left upper eyelid, double vision and dull headache. Examination showed moderate ptosis, 300 exotropia and limited extraocular movements of the left eye. Quantitative pupillary measurement revealed 1mm anisocoria with decreased left pupillary light response.

Result: A clinical diagnosis of left third and fourth cranial nerve palsies from intracranial space-occupying lesion was made. Computed tomography and computed tomography angiography confirmed left posterior communicating artery aneurysm.

Conclusion: Any degree of pupillary involvement in third nerve palsy, whether isolated or not should warrant neuroimaging in view of the high mortality risk from intracranial aneurysms.

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Key words: third nerve palsy, partial pupillary involvement, aneurysm.

INTRODUCTION

The etiologies of third nerve palsy often present diagnostic challenges for the ophthalmologist, neurologist and neurosurgeon. While some causes of third nerve palsy are relatively benign, others are life threatening especially aneurysm. Missing a cerebral aneurysm which ruptures subsequently carries up to 50% risk of mortality[1]. Neuroanatomic localization and determination of the causative lesion is the key to successful management of third nerve palsy. Isolated third nerve palsies are commonly localized to the subarachnoid space with ischemia, aneurysm, trauma, migraine, tumor and congenital damage as common causes. The presence or absence of pupil involvement helps to further delineate third nerve palsies into surgical and non surgical clinical entities respectively. Acute isolated third nerve palsy with pupil involvement warrants neuroimaging to rule out aneurysm. However, the presence of multiple cranial nerve palsies and apparently normal pupils may mislead the clinician since third nerve palsies due to aneurysm rarely involve other cranial nerves and typically cause dilated, non-reactive pupil1. We present a case of non isolated third nerve palsy with partial pupillary involvement in a 20 year old boy due to posterior communicating artery aneurysm and highlight the need for neuroimaging facilities in tertiary medical centers in Nigeria.

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CASE REPORT

A 20 years old male student presented to our out patient clinic on 27th March, 2009 with a three months history of headache and two months history of drooping of the left upper eyelid. The headache was dull in character and localized to the left side of the head. There was no associated fever, dizziness or vomiting.

Drooping of the left eyelid began one month thereafter. It was present throughout the day and not variable. The drooping was associated with double vision which was worse in right gaze. There was no history of trauma, diabetes or hypertension. Examination showed an anxious young man with normal vital signs. His unaided distance visual acuity was 6/6 right and 6/9 left. Pinhole visual acuity was 6/6 either eye. The left eye had moderate ptosis: margin reflex distance (MRD) 2mm, palpebral fissure height 7mm and upper lid excursion 6mm. Corresponding measurements in the right eye were normal (4mm, 9mm, 15mm). There was 300 exotropia of the left eye. Movement of the left eye was limited to three quarters in dextroversion, dextroelevation, dextrodepression, laevoelevation and laevodepression (Figure 1). Only laevoversion was normal. Diplopia chart showed binocular double vision in all the cardinal positions of gaze except laevoversion. The clearer image was always on the right. There was a subtle anisocoria: right pupil 4mm, left pupil 5mm in dim light. The anisocoria increased in bright light. The left pupil reacted sluggishly to light. The left fundus was normal. The right eye was normal. Intraocular pressures were 16 and 18 millimeters of mercury. Refraction and central visual field examinations were normal.

A diagnosis of left third and fourth cranial nerve palsies with partial pupil-involvement due to intracranial space-occupying lesion was made and urgent computed tomography (CT) of the head requested from Memfys Hospital for Neurosurgery, Enugu. Cranial CT without contrast revealed a rounded lesion on the left side of circle of Willis very suggestive of posterior communicating artery aneurysm and computed tomography angiography (CTA) was advised. CTA performed on ceretom 8-slice using lopamidol with post-CTA processing and multiplanar reconstructions revealed a left internal carotid

artery aneurysm around the origin of the posterior communicating artery with no other aneurysms or lesions evident (Figure 2). A diagnosis of left posterior communicating artery aneurysm was therefore made and he was referred back to Memfys Hospital for Neurosurgery. Unfortunately, he could not undergo surgery because of financial constraints. He was seen last on 17th June, 2009 with less painful headache, reduced ptosis (MRD 3mm) with other clinical features static. We could not contemplate strabismus surgery yet because the patient had not undergone neurosurgery.

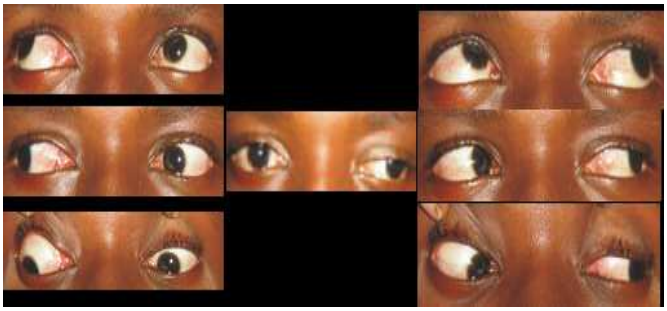

Figure 1: Eye movements into the cardinal positions of gaze

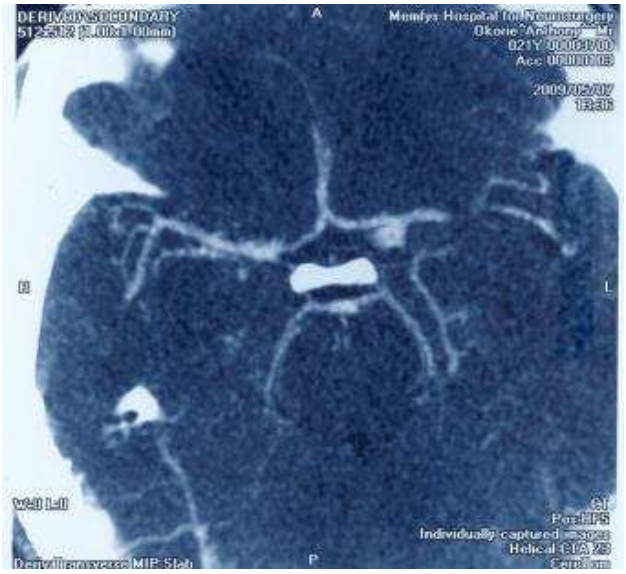

Figure 2: CTA showing left posterior communicating artery aneurysm.

DISCUSSION

The third cranial nerve supplies motor efferents to the levator palpebrae superioris and four extraocular muscles (medial rectus, superior rectus, inferior rectus, and inferior oblique). It also carries presynaptic parasympathetic outflow to the sphincter pupillae and ciliary muscle. Patients with third nerve palsy, therefore typically present with drooping of the eyelid, double vision, squint and photophobia. Our patient presented with unilateral acquired ptosis which may be caused by several factors including myasthenia gravis. However, the associated non-variable binocular diplopia from ophthalmoplegia involving the medial rectus, superior rectus, inferior rectus and inferior oblique of the left eye implied the ptosis was from left third nerve palsy. The limited left dextrodepression implied additional fourth nerve palsy. Our patient therefore had non-isolated third nerve palsy with the fourth cranial nerve as a fellow traveler. This feature typically localizes the third nerve palsy to the cavernous sinus since the fourth cranial nerve lies closely below the third cranial nerve in the lateral wall of the cavernous sinus[2]. We rejected this misleading evidence because we considered other subtle findings more relevant (mild anisocoria, dull headache).

The rule of the pupil which states that when an aneurysm causes a third nerve palsy, the iris sphincter will be impaired and the affected pupil dilated and sluggishly reactive to light is invaluable in localizing third nerve palsies[3]. The rule applies to isolated third nerve palsies. Consequently, isolated third nerve palsy with pupillary involvement warrants neuroimaging to rule out aneurysm[3]. The rule is however silent in non-isolated third nerve palsy[1]. It was therefore not applicable in our patient. Moreover, our patient had only a subtle anisocoria (1mm) which could be easily missed without quantitative pupillary examination. While complete pupillary dysfunction (fixed and dilated) indicates a high risk of aneurysm, the risk of aneurysm in partial pupillary dysfunction (relative pupil-sparring) is low[4].

The decision to undergo neuroimaging in our patent was therefore challenging since current evidence is that patients with complete external dysfunction third nerve palsy but partial internal dysfunction (reactive but sluggish pupil, minimal anisocoria) are more likely to have diabetic, ischemic third nerve palsy than an aneurysm1. The primary associated symptom in our patient was headache. Expanding but unruptured aneurysms typically cause headache[5]. While severe headache, neck stiffness and other meningeal signs should raise the suspicion of aneurysm, the use of less severe pain (as in our patient) in differentiating aneurysmal from ischaemic third never palsy is unclear and lack of pain does not exclude aneurysm[6].

Aneurysms are more common in females, with male female ratio 1:1.37. They are more common after the age of fifty in females but occur earlier in males[7]. Our patient was a male, 20 years old which is an uncommon age for the occurrence of aneurysm[8]. Though the risk of aneurysm was uncertain in our patient, the decision to perform CT/CTA was strengthened by the subtle clinical features and non-availability of Magnetic Resonance Imaging (MRI). The results underscore the usefulness of these techniques in preventing deaths from undiagnosed aneurysms[9]. It is noteworthy that due to the absence of any of these facilities in our center, our patient had to travel (on three different occasions) a distance of 152Km to Enugu for the investigations.

CONCLUSION

We recommend that clinicians should maintain a high index of suspicion for aneurysms in cases of third nerve palsy. The finding of any degree of pupil involvement, whether the third nerve palsy is isolated or not should warn the clinician of possible life-threatening disease and warrants neuroimaging. The ministry of health should urgently provide CT/MRI facilities in our center with requisite manpower to reduce the mortality from these neurosurgical diseases.

REFERENCES

1. Lee A. G., Hayman L. A., Brazis P. W. The evaluation of isolated third nerve palsy revisited: An update on the evolving role of magnetic resonance, computed tomography and catheter angiography. *Surv ophthalmol* 2002; **47(2):** 137–157.
2. Yanovitch T., Buckley E. Diagnosis and management of third nerve palsy. Curr Opin ophthalmol 2007; **18:** 373–378.
3. Jacobson D. M., Trobe J. D. The emerging role of magnetic resonance angiography in the management of patients with third cranial nerve palsy. *Am J ophthalmol* 1999; **128:** 94–96.
4. Jacobson D. M. Pupil involvement in patients with diabetes-associated oculomotor nerve palsy. *Arch ophthalmol* 1998; **116:** 723–727.
5. Ferguson G. G., Drake C. G. Carotid-ophthalmic aneurysms: Visual abnormalities in 32 patients and the results of treatment. *Surg Neurol* 1981; **16:** 1–8.
6. Witham T. F., Kaufmann A. M. Unruptured cerebral aneurysm producing a thunderclap headache. *Am J Emerg Med* 2000; **18:** 88–90.
7. Soni S. R. Aneurysms of the posterior communicating artery and oculomotor paresis. *J Neurol Neurosurg Psychiatry* 1974; **37:** 475–484.
8. Ida M., Kurisu Y., Yamashita M. Magnetic resonance angiography of ruptured aneurysms in acute subarachnoid hemorrhage. *Am J Neuroradiol* 1997; **18:** 1025–1032.
9. Goddard A. J. P., Tan G., Becker J. Computed tomography angiography for the detection and characterization of intracranial aneurysms: current status. Clinical Radiology 2005; **60:** 1221–1236.