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Case Report

Pupil sparing oculomotor nerve paresis after anterior communicating artery aneurysm rupture: False localizing sign or acute microvascular ischemia?

Anirudh Srinivasan, Sivashanmugam Dhandapani, Ajay Kumar[1]

Departments of Neurosurgery and [1]Neuroradiology, PGIMER, Chandigarh, India

E-mail: Anirudh Srinivasan - bmc.anirudh@gmail.com; *Sivashanmugam Dhandapani - ssdhandapani.neurosurg@gmail.com; Ajay Kumar - kumar.ajay@pgimer.edu.in
*Corresponding author

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Abstract

Background: We describe a rare case of solitary pupil sparing oculomotor nerve paresis following rupture of anterior communicating artery (ACom) aneurysm and discuss the pertinent literature. Oculomotor nerve paresis caused by an ACom aneurysm rupture is an uncommon occurrence. Also, partial paresis affecting only fibers of superior division of oculomotor nerve is never reported before.

Case Description: A 55-year-old female, known hypertensive presented 5 days after an episode of acute severe headache, with Glasgow Coma Scale (GCS) of E2V2M5, left ptosis, normal pupils, paraparesis, and computed tomography (CT) scan showed diffuse subarachnoid hemorrhage (SAH). CT angiography revealed ACom aneurysm pointing antero-superiorly toward right. Patient later underwent endovascular coiling of the aneurysm. Subsequently there was partial improvement of ptosis in 3 weeks.

Conclusion: Though pupil sparing oculomotor nerve paresis may not have much localizing value, it helps to understand acute microvascular spasm with potential therapeutic implications.

Key Words: Anterior communicating artery aneurysm, micro vascular spasm, pupil sparing oculomotor paresis, subarachnoid haemorrhage

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INTRODUCTION

Oculomotor nerve paresis associated with subarachnoid hemorrhage is typical of aneurysms located at the junction of the internal carotid artery and posterior communicating artery (PCom), intracavernous part of the internal carotid artery, basilar artery, posterior cerebral artery, and superior cerebellar artery. However, oculomotor nerve paresis caused by an anterior communicating artery (ACom) aneurysm is a very uncommon occurrence. More importantly, partial paresis affecting only the fibers of superior division of third cranial nerve (CN) has never been reported before. Management and the pertinent literature are reviewed along with the probable mechanism of nerve involvement.

CASE REPORT

A 55-year-old female, known hypertensive presented 5 days after an episode of acute severe headache, with

Glasgow Coma Scale (GCS) of E2V2M5, left ptosis, normal pupils [Figure 1], paraparesis, and computed tomography (CT) scan showed diffuse subarachnoid hemorrhage (SAH) [Figure 2]. CT angiography revealed ACom aneurysm pointing antero-superiorly toward right [Figure 3]. Patient underwent endovascular coiling of the aneurysm. On further detailed examination, patient showed partial weakness of left superior rectus. Other extra ocular movements were normal. At 3 weeks follow-up, there was partial improvement of ptosis.

DISCUSSION

Paresis of CN III is associated with ruptured aneurysms in 30% of patients.[6] PCom aneurysms, constituting 11% of ruptured aneurysms,[2,3] are the most common associated aneurysms, followed by ruptured aneurysms of intracavernous internal carotid artery, basilar artery, posterior cerebral artery, or superior cerebellar artery. The degree of paresis is usually dependent on the direction and site of the fundus and the rate of distension of the aneurysmal sac. The amount of subarachnoid blood is, however, a less important factor for contributing to third nerve paresis.[13]

Although there has been six cases of CN III paresis associated with ACom aneurysms,[13,15,16] this is probably the first report of isolated involvement of the superior division of oculomotor nerve with sparing of pupils.

The CN III nucleus comprises a complex of subnuclei located within the midbrain, ventral to the periaqueductal gray matter. The motor neurons of each subnuclei innervate their corresponding ipsilateral extraocular muscles except for the superior rectus subnuclei, which innervate the contralateral superior rectus muscle, and the midline levator palpebrae subnuclei, which innervate the ipsilateral and contralateral levator palpebrae muscles.[12]

The nerve exits the midbrain just medial to the cerebral peduncle as 10 to 15 rootlets merging to form a single nerve, and then it passes between the posterior cerebral and superior cerebellar arteries, traveling adjacent to as well as inferior and lateral to the PCom to arrive at the superolateral aspect of the cavernous sinus.[9] Within the cavernous sinus, CN III is located in the dural fold of its lateral wall. In most cases, the anatomical bifurcation of CN III into a superior and inferior division occurs in the region of the anterior cavernous sinus or superior orbital fissure. The superior division, supplies the superior rectus and the levator palpebrae superioris, and the inferior division, supplies the medial and inferior recti, the inferior oblique, and the presynaptic parasympathetic outflow to the ciliary ganglion (sphincter pupillae muscle and ciliary muscles).[9]

Ptosis can be present at birth (congenital) or develop later in life (acquired). Ptosis may be due to a myogenic,

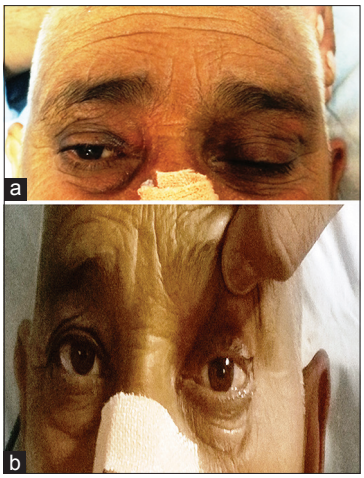

Figure 1: (a) Clinical photograph showing left eye ptosis. (b) Clinical photograph showing normal sized pupils

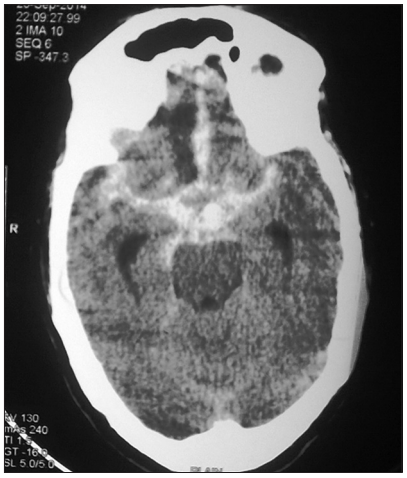

Figure 2: NCCT Head showing diffuse sub arachnoid hemorrhage in the basal cisterns

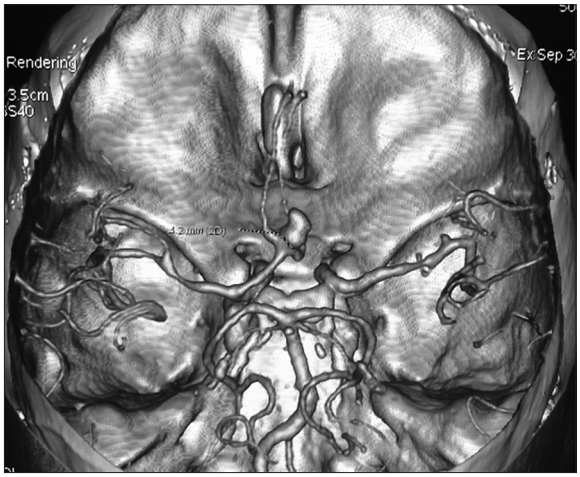

Figure 3: CT Angiography cerebral vessels showing ACom aneurysm

neurogenic, aponeurotic, mechanical, or traumatic cause. Usually, ptosis occurs isolated, but may be associated with various other conditions, like immunological, degenerative,

or hereditary disorders, tumors, or infections. However, these etiologies cannot explain the ptosis, which presented as an acute event for the first time following SAH.

The likely role for the trigemino-oculomotor pathway is to provide inhibition of levator motor neurons during the down-phase of a blink elicited by trigeminal stimulation. However, this cannot explain unilateral ptosis and this has more significance in the corneal reflex physiology.[11] Large areas of cerebral/basal ganglion dysfunction secondary to vasospasm can indeed lead to contralateral ptosis, however, this is associated with focal neurological deficits related to the cortical or basal ganglion dysfunction.

Although the subarachnoid or cisternal portion of CN III is a single structure, based on previous clinical observations, there is a topographic arrangement of the fibers within the nerve but the precise anatomical arrangement in humans has not been specified. The few localization studies performed in animals have demonstrated pupillary fibers to be superficially located in the cisternal portion of CN III.[1,8,10] When the peripheral oculomotor nerve is involved by mass lesion or aneurysm, the pupilloconstricter fibers are usually involved first, followed by palsy of the levator palpebrae, superior rectus, and medial rectus, in order. In contrast, when there is anintramedullary lesion due to microvascular ischemia, the pupilloconstrictor fibers are spared.[14] Hypertension and greater Fisher grade could have predisposed to acute microvascular spasm of perforators to CN III. Though cerebral vasospasm has been the most probable cause of neurological deficits following SAH,[2,3] this has never been reported to occur in perforators to nerves. The perfusion deficits are known to occur at subclinical level not routinely detectable.[4] Other probable mechanisms would be hemorrhagic dissection of the nerve, or premature branching of CN III with smaller superior division more vulnerable to cisternal blood.[5,7]

Though pupil sparing CN III paresis may not have much localizing value, it helps to understand acute microvascular spasm with potential therapeutic implications.

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