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General Surgery

Unilateral Pupil Sparing Third Nerve Paresis Associated With Ruptured Anterior Communicating Artery Aneurysm-A Case Report.

Amit Lamba	Senior Resident, Department of Neurosurgery, Government Medical College, Trivandrum
Sourabh Kumar Jain	Senior Resident, Department of Neurosurgery, Government Medical College, Trivandrum
Anil Peethambaran	Professor, Department of Neurosurgery, Government Medical College, Trivandrum
Saurabh Sharma	Senior Resident, Department Of Neurosurgery, Government Medical College, Trivandrum
Milesh Nagar	Senior Resident, Department Of Neurosurgery, Government Medical College, Trivandrum

ABSTRACT Oculomotor nerve paresis (ONP) with subarachnoid hemorrhage (SAH) occurs usually when oculomotor nerve is compressed by growing or budding of posterior communicating artery (PcoA) aneurysm. Midbrain injury, increased intracranial pressure (ICP), or uncal herniation may also cause it.2 We report herein a rare case of ONP associated with SAH which was caused by anterior communicating artery aneurysm rupture.3 A 63 year old female patient presented with multiple episodes of vomiting and sudden onset severe headache for the past 4 days. She was found to have anterior communicating artery aneurysm rupture leading to WFNS grade 1 SAH. She also complained of diplopia and ptosis and was found to have left third nerve palsy. Remaining clinical examination findings were normal.

KEYWORDS : Oculomotor nerve paresis, Anterior communicating artery aneurysm, Subarachnoid hemorrhage

Introduction

Unilateral occulomotor nerve paresis associated with subarachnoid hemorrhage (SAH) is usually caused by aneurysm of ipsilateral internal carotid- posterior communicating artery junction aneurysm or less frequently by basilar artery aneurysm. Unilateral third cranial nerve palsy as a result of a ruptured anterior communicating artery aneurysm is very rare because A ComA aneurysms are not in the vicinity of the oculomotor nerve.[1]

There are several possible pathological causes of oculomotor nerve paresis, including midbrain bleeding or ischemia, ischemia of the nerve itself, tumors, trauma, neuritis, meningitis, and intracranial aneurysm.

A number of mechanisms of a third nerve palsy in patients with an intracranial aneurysm

had been classified by Fox:

A. Direct peripheral causes include, 1) local pressure by the aneurysm, and 2) hemorrhagic dissection of the nerve.
B. Direct central causes include bleeding into midbrain parenchyma or direct pressure of a large basilar artery on the nucleus.
C. Indirect central causes include increased ICP and vasospasm
D. Indirect peripheral causes include increased ICP (from clot, edema, and hydrocephalus) Causing uncal herniation.[1]

Case Report

A 63-year-old woman was admitted to our hospital due to frequent episodes of vomiting and sudden onset of severe headache without any H/O loss of consciousness. Also, no history of trauma, diabetes mellitus, hypertension, heart disease, stroke, or cancer. On examination pt. was E4V4M6, pupil 1.5 mm B/L reactive with left third nerve paresis with no other neurological deficit. CT brain was evaluated that exhibited diffuse SAH involving interhemispheric fissure, basal cistern, B/L sylvian fissure and B/L frontal cortical sulci as seen in Figure 1.

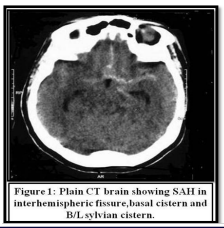

Figure 1: Plain CT brain showing SAH in interhemispheric fissure,basal cistern and B/L sylvian cistern.

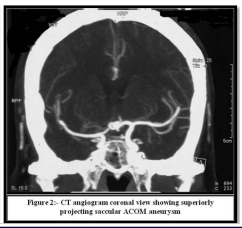

Figure 2:- CT angiogram coronal view showing superiorly projecting saccular ACOM aneurysm

CT angiogram was done which showed saccular anterior communicating artery aneurysm with no other vascular abnormality anywhere else as seen in Figure 2,3&4 from coronal and sagittal view. Left pterional craniotomy with clipping of aneurysm done next day. Intraoperatively aneurysm was saccular shaped and projected posteriorly. Left sylvian fissure exhibited clotted blood around aneurysm. Occulomotor nerve appeared intact in carotid cistern. Post operative period was uneventful. Left third nerve paresis persisted. Patient was discharged on 7th post operative day and currently under OPD follow up with partial improvement of ptosis.

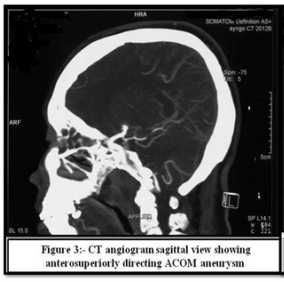

Figure 3:- CT angiogram sagittal view showing anterosuperiorly directing ACOM aneurysm

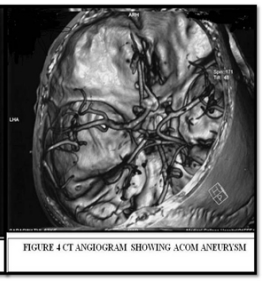

FIGURE 4 CT ANGIOGRAM SHOWING ACOM ANEURYSM

Discussion

ONP is one of the manifestations of intracranial aneurysms and SAH, and IC-PcoA aneurysm is the most common cause of ONP (34-56%) [4,5]. However, it is also possible that the rupture of distant aneurysms or anomalous vessel may cause ONP. Aiba and Fukuda[6] reported anomaly of basilar arteries, posterior cerebral arteries, and superior cerebellar arteries as well as posterior cerebral artery and superior cerebellar artery inversion might cause ONP.

Kurokawa et al.[7] described that the anatomical relationship between the exit of the PcoA and the entry of the oculomotor nerve into the cavernous sinus that would usually result in superomedial compression of the nerve by the aneurysm. Typical ONP in this situation may manifest as unilateral pupil dilation, ptosis, incomplete or partial extra ocular palsies, and oculomotor synkinesis.

ONP is usually recovered very slowly, ranging from a few weeks to months[8,9,10]. Chen et al.[8] reported that the time to complete resolution of ONP takes about 6 months. Furthermore, early treatment might contribute to early recovery. Indeed, Leivo et al.[9] recommended early treatment for aneurysm-induced third nerve palsy, preferably within 3 days, to avoid functionally and cosmetically unwanted disability.

The Present case of third nerve paresis caused by ACOM aneurysm is very rare. Clinically our patient presented with pupil sparing third nerve palsy which is not common with ICA aneurysm or trauma. Pupil sparing third nerve palsy is mostly associated with extra axial micro vascular ischemia like in diabetic patients because the parasym pathetic fibers are located in the periphery of oculomotor nerve. However, our patient had no history of diabetes. Acute subdural hematoma, compressive cavernous sinus lesion, midbrain lesion, cerebrovascular accident and compression of nerve at root exit zone by aneurysm or tumor may cause oculomotor nerve palsy. But no such lesion was detected radiologically. Present case of pupil sparing oculomotor nerve palsy appears to be caused by micro vascular spasm to oculomotor nerve perforators. The oculomotor nerve (third cranial nerve) supplies motor innervation for the superior rectus, medial rectus, inferior rectus, inferior oblique, and levator palpebrae superioris muscles and also parasympathetic input to the pupillary constrictor and ciliary muscles. We attribute that apart from the pupillary constrictor and ciliary muscle remaining muscles were involved leading to paresis. Vasospasm is prolonged cerebral arterial constriction caused by vascular smooth muscle contraction.. Cell bodies reside in the midbrain in a nuclear mass straddling the vertical midline. Oculomotor nerve paresis may be in intraocular or extraocular. Extraocular nerve palsy usually occurs in the presence of compressive lesion whereas intra ocular or true pupillary sparing occurs in vascular ischemia of the nerve.

True pupillary sparing oculomotor nerve paresis implies that each of the extraocular muscles innervated by the oculomotor nerve is involved to some extent, but the pupil remains of normal size and reactivity. The cause of most isolated pupil-sparing third-nerve palsies is believed to be microvascular ischemia, frequently associated with diabetes mellitus age more than 50 years, hypertension, dyslip idaemia , smoking,atherosclerosis, arteritis and vasospasm following SAH. The explanation for this may be anatomic in that the peripherally located pupillary fibers may receive more collateral blood than the main nerve trunk. Though cerebral vasospasm has been the most probable cause of neurological deficits following SAH, this has never been reported to occur in perforators to nerves. The perfusion deficits are known to occur at subclinical level not routinely detectable. Other probable mechanisms would be hemorrhagic dissection of the nerve.

In a case reported by White et al in 2007 a 46 year old male patient presenred with headache, emesis, and an isolated left third nerve palsy-the remainder of his exam was normal. A head CT showed diffuse subarachnoid hemorrhage that was later attributed to an ACOM aneurysm as determined by angiography. Following a successful clipping, the patient experienced a delayed, transient, monocular visual loss. Upon follow-up, his oculomotor palsy had completely resolved.[11]

In another case that was reported by Justin et al in 2016, An isolated cranial nerve-III palsy was reported as a rare clinical finding in a patient with perimesencephalic subarachnoid hemorrhage. In this unusual case, the patient presented with complete cranial nerve-III palsy including ptosis and pupillary involvement. Initial studies revealed subarachnoid hemorrhage in the perimesencephalic, prepontine, and interpeduncular cisterns. Angiographic studies were negative for an intracranial aneurysm. The patient's neurological deficits improved with no residual deficits on follow-up several months after initial presentation. This report further adds a case of isolated cranial nerve-III palsy as a rare initial presentation of this type of bleeding, adding to the limited body of the literature.[12]

Conclusion

ONP induced by a ruptured anterior communicating artery aneurysm is very rare, and it would better be kept in mind that the ONP with SAH may occur by the rupture of distant intracranial aneurysms.

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