Transient Oculomotor Nerve Palsy After Subarachnoid Hemorrhage Due To Middle Cerebral Artery Aneurysm

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Citation

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Abstract

Oculomotor nerve (ON) palsy after subarachnoid hemorrhage due to middle cerebral artery (MCA) aneurysm is a rare entity with very few reported cases in literature. A 55-year-old male presented with subarachnoid hemorrhage due to ruptured MCA aneurysm on left side with partial left (ON) palsy. The patient had no clinical or radiological feature of raised intracranial pressure. The patient underwent endovascular coiling following, which there was complete recovery within two days. The involvement of ON in MCA aneurysm and such rapid recovery has not been reported before.

INTRODUCTION

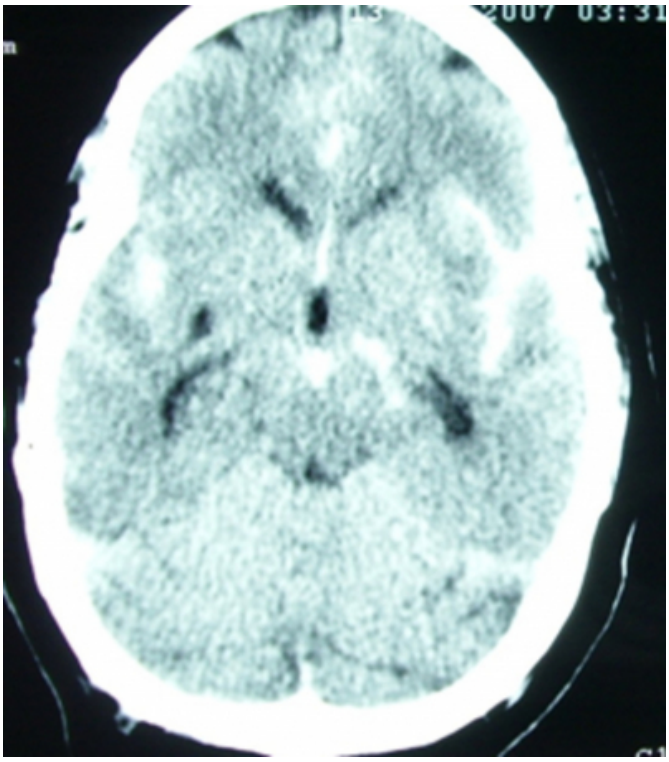
Oculomotor nerve (ON) palsy following subarachnoid hemorrhage (SAH) is usually due to posterior communicating artery (pcom) aneurysm or uncommonly due to basilar artery - superior cerebral artery aneurysm. Rare aneurysms to cause ON palsy are, those arising from posterior inferior cerebral artery, distal anterior cerebral artery, anterior communicating artery, anterior choroidal artery and even rarer is middle cerebral artery (MCA). [1, 2,3,4,5]. Subarachnoid hemorrhage due to any cause can lead to ON palsy in association with cerebral herniation [5]. The involvement of ON can be partial or complete depending of various parameters like duration, severity and site of compression. [6].

CASE REPORT

A 58- year- old male presented with sudden onset of headache and vomiting. CT scan revealed presence of subarachnoid hemorrhage and there were clots seen in left crural, interpeduncular and left ambient cistern and there was no evidence of herniation. (Figure 1) .

Figure 1

Figure 1: CT scan showing presence of subarachnoid hemorrhage with clots in left crural and ambient cistern



The patient reported to us the next day and was having significant relief in headache and on examination he was in WFNS (world federation of neurological surgeons) grade I. His only neurological deficit was the presence of left

oculomotor partial palsy. He had ptosis, pupillary dilatation not reacting to direct or consensual light and had paresis of adduction. The patient underwent digital subtraction angiogram which revealed presence of left middle cerebral artery aneurysm, a lobule of which was pointing medially (Figure 2)

Figure 2

Figure 2: Angiogram showing left middle cerebral artery aneurysm

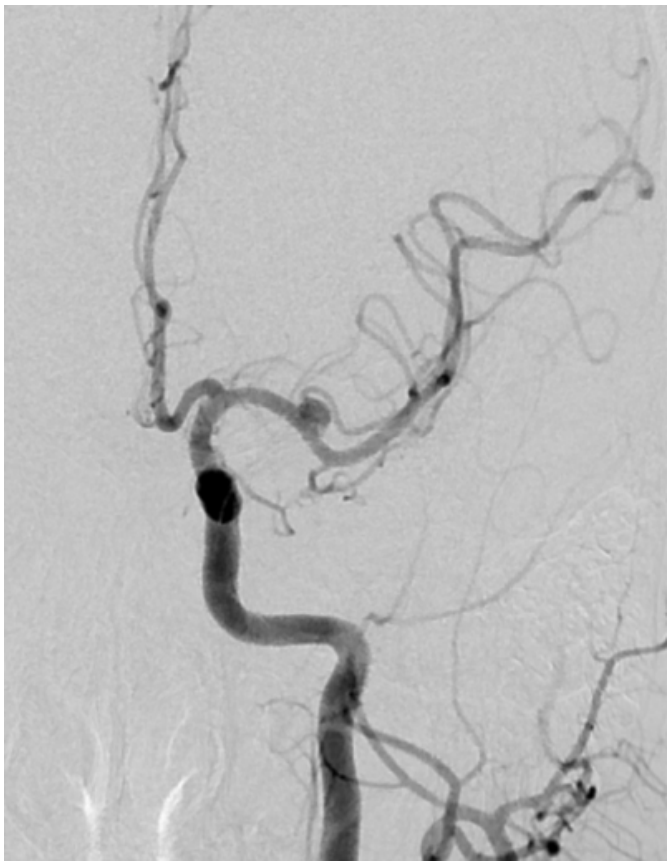
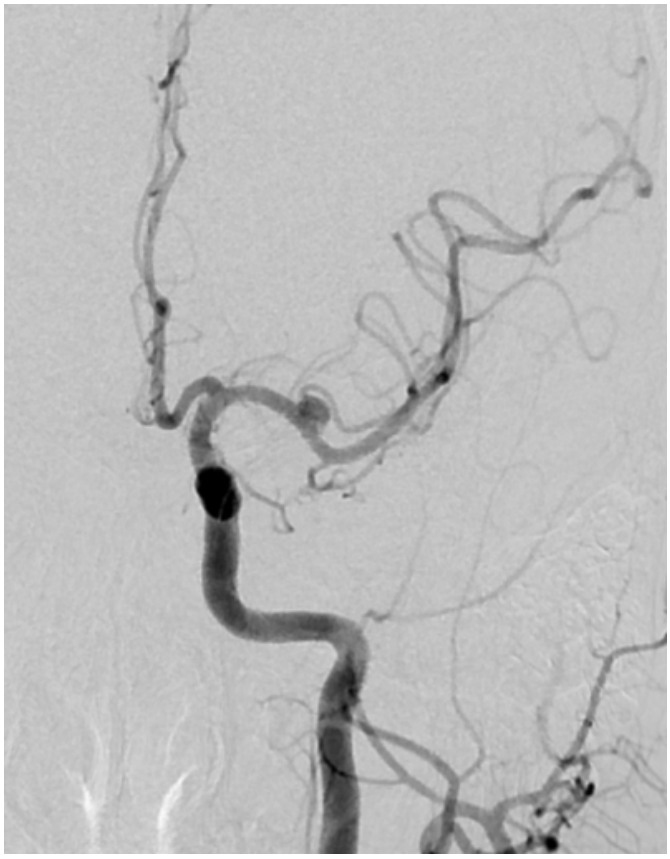


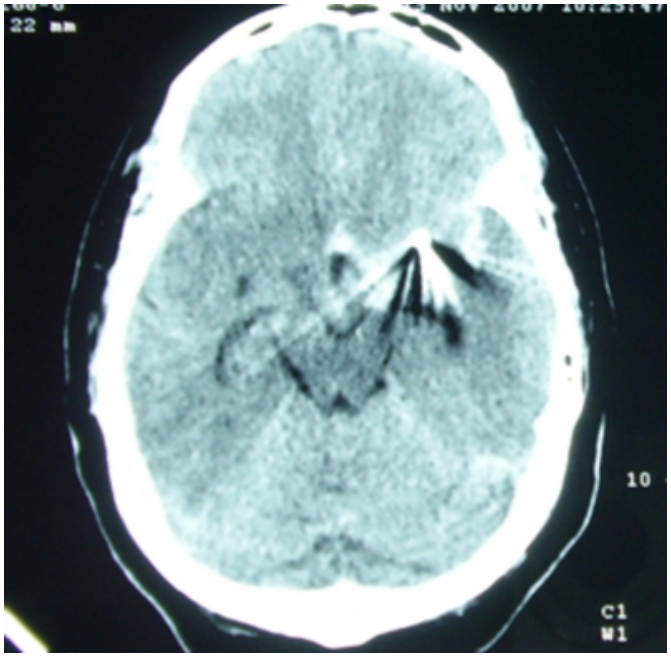
Figure 3



The patient underwent endovascular coiling of the aneurysm. During the procedure, patient was administered intra-arterial nimodipine and he was started on hypervolemic therapy. He was receiving dexamethasone 16 mg for 24 hours along with nimodipine 60 mg 4 hourly. The blood pressure was maintained around 160 mm (Hg systolic). The next day ptosis reverted to normal followed by pupillary and ocular movements. The patient had complete normal nerve functions within 48 hours of intervention. CT scan done 48 hours after coiling showed complete resolution of perimesencephalic blood clots. (Figure3).

Figure 4

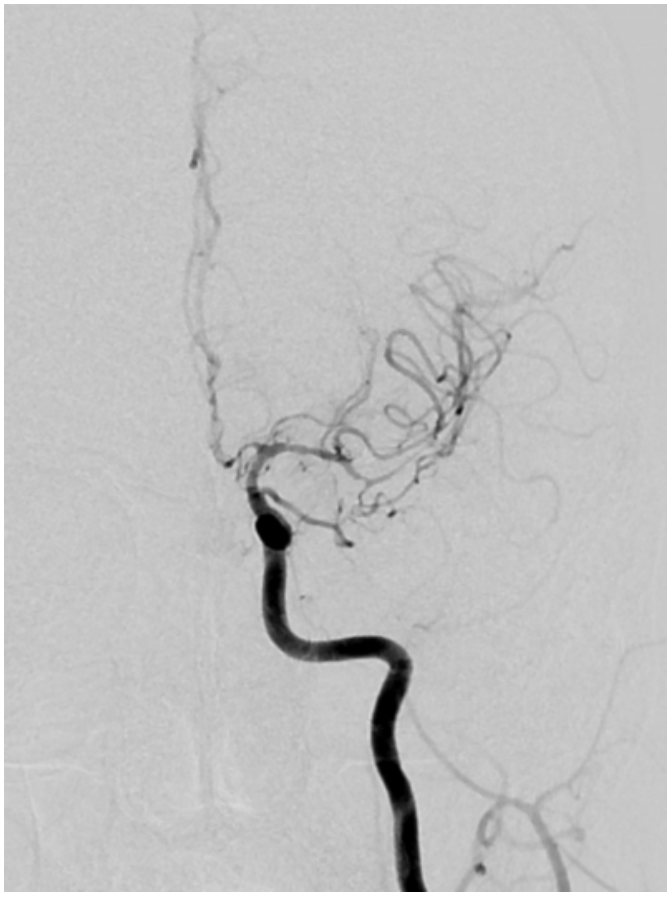
Figure 3: Post coiling CT scan showing resolution of perimesencephalic clots and coil is also seen



On the fifth day the patient deteriorated in sensorium and had dysphasia and left hemiparesis. Urgent angiogram was done which revealed presence of vasospasm. (Figure 4)

Figure 5

Figure 4: Presence of vasospasm on check angiogram , coil is also seen in the aneurysm



Intra-arterial nimodipine was administered and vasospasm got relieved. The weakness gradually improved and his dysphasia improved. After three weeks patient is able to walk independently and able to verbalize with mild difficulty.

DISCUSSION

In absence of herniation syndrome, ON palsy can be caused by varied etiologies like ischemia of nerve due to disease like diabetes, direct trauma to nerve, neuritis, meningitis, intracranial aneurysms, midbrain hematoma or infarcts.[7,8,9,10,11]. The clinical spectrum of ON involvement due to aneurysm is varied. The syndrome is very well described for the Pcom aneurysms due to their frequency. Orbital pain or headache usually proceeds the involvement of ON. The disturbance in ON function may be partial or complete. [3,6,12] There may be only anisocoria, ptosis with or without pupil involvement or total opthalmoplegia depending on site, severity and duration of involvement of ON. Impairment of ON function in SAH can be because of

any or multiple of following factors; direct compression of ON by aneurysm, irritation effect of blood and vasospasm leading to ischemia of nerve, or cerebral herniation.[13,14,15] In our patient since aneurysm was arising from MCA away from the ON hence it is not the possible cause of involvement. In the absence of clinical or radiological features of raised ICP vasospasm and ischemia can be one of the possible explanations of ON palsy in our patient as he had clots seen in left crural, interpeduncular and left ambient cistern. Another possibility is that after aneurysm rupture jet of blood might have been directed medially as we had clots in left sided cisterns causing direct injury to ON.

Recovery of function after intervention: surgery or endovascular has been reported [15,16]. The recovery of function is first seen in ptosis followed by extra ocular muscle function and pupillary abnormalities [13,15,16,17]. In an important series on the subject, Laun A found regression of ON palsy in 39.2% of patients within 6months and in remaining deficits was permanent [18]. In Sakurai, series regression was seen in 75% of the patient [19].

Resolution of ON palsy following surgery is relatively easy to understand in case of compression of nerve by aneurysm. However, improvement in function following endovascular intervention is not easily understood. Birchall et al suggested decrease in pulsatility within aneurysmal sac following coiling could cause improvement. [15] Some studies have reported decrease in cerebral edema adjoining aneurysms after endovascular coiling leading to improvement. [15,20,21]. In our case both possibilities seen to be unlikely as the aneurysm was away from ON. The possible explanation can be use of intra arterial nimodipine during the procedure leading to decrease in spasm. The recovery could have been aided by good hydration aided by intravenous steroids given to the patient.

CONCLUSION

MCA aneurysms are rare cause of Oculomotor nerve palsy in absence of herniation syndromes. These probably result from transient vasospasm or direct injury due to jet of blood. Complete recovery may occur in such cases by conservative management without a need for surgical intervention.

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References

1. Fujiwara S, Fujii K, Nishio S, Matsushima T, Fukui M: Oculomotor nerve palsy in patients with cerebral aneurysms. Neurosurg Rev 1989; 12:123-132
2. Laun A, Tonn JC: Cranial nerve lesions following subarachnoid hemorrhage and aneurysm of the circle of Wills. Neurosurg Rev1998; 11:137-141
3. Arle JE, Abrahams JM, Zager EL, Taylor C, Galetta SL: Pupil-sparing third nerve palsy with preoperative improvement from posterior communicating artery aneurysm. Surg Neurol 2002; 57:423-427
4. Suzuki J, Mizoi K, Sato T: Disturbances of ocular movement due to cerebral aneurym-Based upon the experience in 1000 directly operated patients, in Samii M, Jannetta PJ (Eds): The cranial Nerves. Berlin, Spring-Verlag.1981: 229-236
5. Toyotaka Aiba, Masafumi Fukuda: Unilateral Oculomotor nerve paresis associated with anterior communicating rupture. Neurol Med Chir (Tokyo) 2005; 45: 143-147
6. Y Kurokawa, E Ishizaki, K Inaba. Incomplete Oculomotor nerve palsy caused by an unruptured internal carotid-anterior choroidal artery aneurysm. Neurol Med Chir. (Tokyo) 2005; 45: 143-147
7. Biousse V, Newman NJ: Third nerve palsies. Semin Neurol 2000; 20:55-74
8. Kasner SE,Liu GT,Galetta SL: Neuroophtalmologic aspects of aneurysm. Neuroimaging clin N Am 1997;7:679-692
9. Rnowden SA, Harris KM, Hourihan MD: Isolated atraumatic third nerve palsy: Clinical features and imaging techniques. Br J radiol 1993; 66:1111-1117
10. Richards BW, Jones FR, Younge BR: Causes and prognosis in 4278 cases of paralysis of the Oculomotor, trochlear and abducens cranial nerves. Am J Opthalmol 1992;113: 489-496
11. Abrar A Wani , Anil Dhar , AU Ramzan , AH Kirmani , AR Bhatt . Isolated third nerve palsy due to mesencephalic hematoma. Indian journal of neurotrauma 2007;4 (1) 53-54
12. Good EF: Ptosis as the sole manifestation of compression of the Oculomotor nerve by an aneurysm of the posterior communicating artery. J clin Neuroophthalmol 1990; 10:59-61
13. Giombini S, Ferraresi S, Pluchino F. Reversal of oculomotor disorders after intracranial aneurysm surgery. Acta Neurochir 1991;112:19-24
14. Daniel Birchall, Makhan S. Khangure , William McAuliffe: Resolution of Third Nerve Paresis after Endovascular Management of Aneurysms of the Posterior Communicating Artery. Am J of Neuroradiol 1999; 20:411-413
15. Hyland HH, Barnett HJ. The pathogenesis of cranial nerve palsies associated with intracranial aneurysms. Proc R Soc Med 1954;47:141-146
16. Chen PR, Amin-Hanjani S, Albuquerque FC, Mc Dougall C, Zabramski JM,Spetzler RF. Outcome of Oculomotor nerve palsy from posterior communicating artery aneurysms: comparison of clipping and coiling. Neurosurg 2006; 58(6):1040-6;
17. Soni SR. Aneurysms of the posterior communicating artery and Oculomotor paresis. J Neurol Neurosurg Psychiatry 1974; 37:475-484
18. Kyriakides T, Aziz TZ, Torrens MJ. Postoperative recovery of third nerve palsy due to posterior communicating aneurysms. Br J Neurosurg 1989; 3:109-112
19. Albrecht Laun, Jorg-Christian Tonn: Cranial nerve lesions following subarachnoid hemorrhage and aneurysm of

circle of Willis. Neurosurg Rev 1998; 11: 137-141
20. Sakurai Y, Enokida, J Suzuki: Oculomotor palsy caused by aneurysm at internal carotid-posterior communicating artery junction and its prognosis following intracranial surgery. Suzuki J (ed): cerebral aneurysms. Neuron 1979, Tokyo
21. Strother CM, Eldevik P, Kikuchi Y, et al. Thrombus formation and structure and the evolution of mass effect of intracranial aneurysms treated by balloon embolization: emphasis on MR findings. AJNR Am J Neuroradiol 1989; 10:787-796
22. Halbach VV, Higashida RT, Dowd CF, et al. The efficacy of endovascular aneurysm occlusion in alleviating neurological deficits produced by mass effect. J Neurosurg 1994; 80:659-666

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