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Unprecedented reversible quadriplegia after ruptured anterior communicating artery aneurysm: A case report

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Abstract
Anterior communicating artery (ACoA) aneurysm is a common site for spontaneous subarachnoid hemorrhage. The classical presentation of paraplegia and akinetic mutism is well known. We present a case of ruptured ACoA aneurysm who presented with quadriplegia. We discuss the possible mechanism for this finding. The patient gradually showed improvement in quadriplegia. It is important to be aware of this unusual presentation. Rehabilitation of patient along with treatment of aneurysm is equally important.

Keywords: Subarachnoid hemorrhage, anterior communicating artery aneurysm, bilateral anterior cerebral artery infarcts, quadriplegia

Introduction
The incidence of spontaneous subarachnoid hemorrhage (SAH) is 6 per 100,000 per year, and 85% are due to intracranial saccular aneurysms [1].
Subarachnoid hemorrhage from ruptured anterior communicating artery aneurysm have classical neurological deficit in form of paraplegia and akinetic mutism. We present a case who presented as subarachnoid hemorrhage due to ruptured blister aneurysm of anterior communicating artery and was successfully managed with coiling and flow diverter placement. The cause of quadriplegia in our case remains speculative. The correlation with imaging findings may not be complete. The patient gradually showed improvement in his motor power and language functions.

Case History
Mr. PK, a 46 years old male, presented with history of severe headache, recurrent vomiting, weakness of both lower limbs, generalized seizure one episode and became unconscious after few hours. He went to a hospital. BP was found to be 220/120 mmHg. He was intubated and connected to ventilator. CT brain showed subarachnoid hemorrhage [Figure 1]. CT angiography and Digital subtraction angiography (DSA) showed anterior communicating artery blister aneurysm (2x1.5 mm with 2 mm neck) [Figure 2]. Ommaya reservoir was placed with ventricular drain in right frontal horn through burr hole. He was managed with intravenous nimodepine, antiepileptics, anti hypertensives and other supportive care.
Patient was moved to our hospital on 5 th day as the facility of coiling was not available at the first hospital. Patient was E1 M1Vet when he came to us. There was quadriplegia. He had ommaya reservoir along with external drain connected to reservoir. Patient was continued on supportive treatment. He was not immediately taken for procedure as his Glasgow coma scale was very low. He was managed with intravenous nimodepine and Triple H therapy.
Gradually his sensorium improved over the course of next five days, he continued to have quadriplegia and mutism but he was understanding verbal commands. His horizontal and vertical gaze was full. CT and MRI brain revealed bilateral para sagittal infarcts [Figure 3, 4]. He also underwent MRI cervical spine to rule out any cervical cause for quadriplegia, which was normal [Figure 5]. He was taken up for definitive procedure on 11th day of ictus. Endovascular coiling and flow diverter was inserted in anterior communicating artery (ACOM) aneurysm [Figure 6]. The procedure was uncomplicated. Patient was gradually weaned off from ventilator. His tracheostomy site was also closed.

He gradually regained motor power in his upper limbs and started speaking few words after 2 weeks. His lower limb power also improved to a limited extent. When he was discharged his modified ranking score (mRS) was 4. Regular physiotherapy was advised to patient.

Discussion

The anterior communicating artery (ACoA) is the most common site for both ruptured and unruptured aneurysms, and ACoA aneurysms account for 30%-37% of all intracranial aneurysms. Existing treatment strategies for aneurysms include surgical and endovascular management, both being equal in their efficacy [1-3].The usual presentation of ruptured ACOM aneurysm is headache, vomiting, loss of consciousness. It is associated with paraplegia, behavioural disturbance (akinetic mutism) if ACA territory infarct has occurred.

Quadriplegia is a rare presentation. Quadriplegia has been reported in literature in few cases of bilateral ACA infarcts [4-9]. Bilateral ACA infarction was found in two cases where clinical diagnosis was basilar artery occlusion [8]. Saleh *et al*. has described his case of ACOA aneurysm who presented with quadriplegia and also compared with two previous cases reported in literature who had presented with quadriplegia [4]. RajShekharan *et al*. described a case with bilateral ACA territory artery infarcts which lead to quadriplegia [7]. He demonstrated unpaired ACA territory and presence of azygous ACA in his case.

We discuss a case where patient had quadriplegia and mutism for few days. There were bilateral anterior cerebral artery territory infarcts. The parasagittal area of brain controls the leg area, hence paraplegia is expected. Different mechanisms for quadriplegia have been postulated. The possible explanation for quadriplegia is thrombosis of anterior cerebral artery A1 segment or involvement of recurrent artery of Heubner. Recurrent artery of Heubner arises from the lateral surface of A2 just beyond the ACom Art and proceeds laterally parallel to the course of the ipsilateral A1 to supply anterior portions of the caudate, putamen, globus pallidus and internal capsule. There may also be vasospasm of recurrent artery of Heubner along with bilateral ACA arteries. Improvement in motor power of upper limbs after few days point towards vasospasm as being the primary pathology.

Infarction of the ACA territory itself is rare and accounts for only 0.3% to 4.4% of cerebral infarctions reported. Bilateral ACA infarction is even rarer. It has been shown in a study by Kumral *et al*., where 2 out of all 48 patients, had bilateral anterior cerebral artery infarction (10). In his study, cause of ACA territory infarction varied, but quadriplegia not reported in his series. Twenty-seven cases of ACA territory infarction were reported among 1490 cases of cerebral infarction in the Lausanne Stroke Registry, however, there were only two cases of bilateral ACA territory infarction [11].

Ours is a rare case of quadriplegia due to bilateral ACA territory infarcts. Very few cases of similar nature have been reported in literature [4-8]. Such patients require long term rehabilitation and follow up.

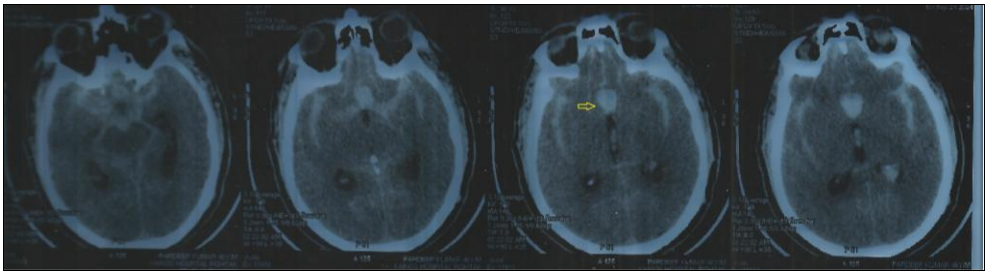

Fig 1: CT image showing diffuse subarachnoid hemorrhage, blood seen in all cisterns and sulci, mainly around site of anterior communicating artery.

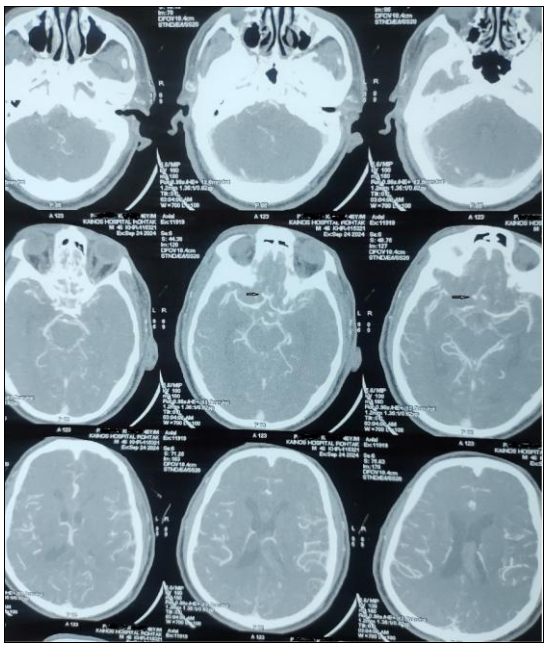

Fig 2: CT angiography shows ruptured anterior communicating artery aneurysm.

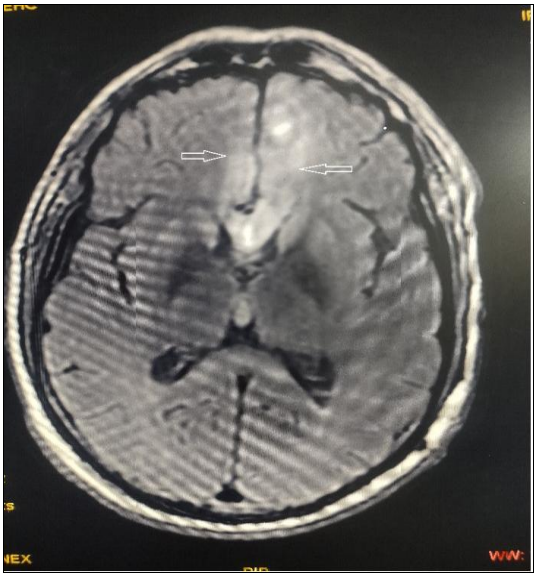

Fig 3: MRI brain axial section showing bilateral parasagittal infarcts, frontal area.

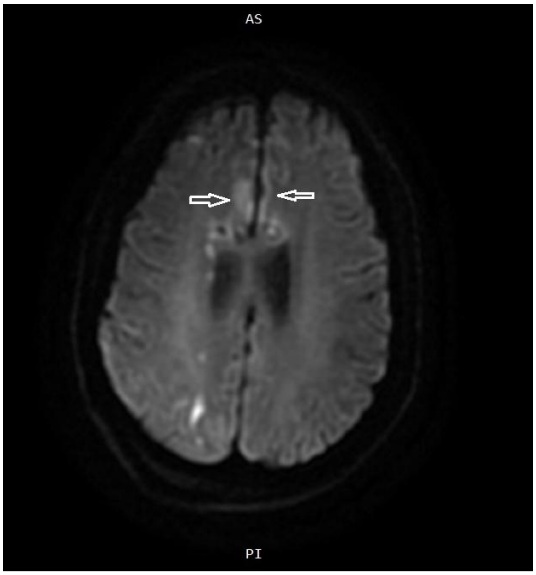

Fig 4: MRI brain, diffusion weighted images showing diffusion restriction bilateral parasagittal area.

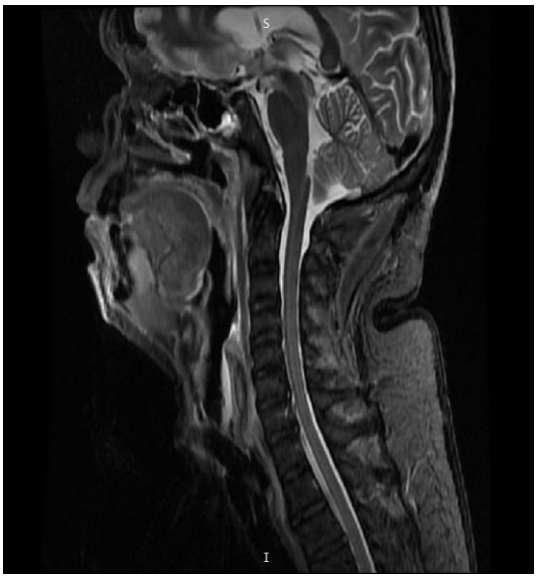

Fig 5: MRI cervical spine STIR sagittal image, normal.

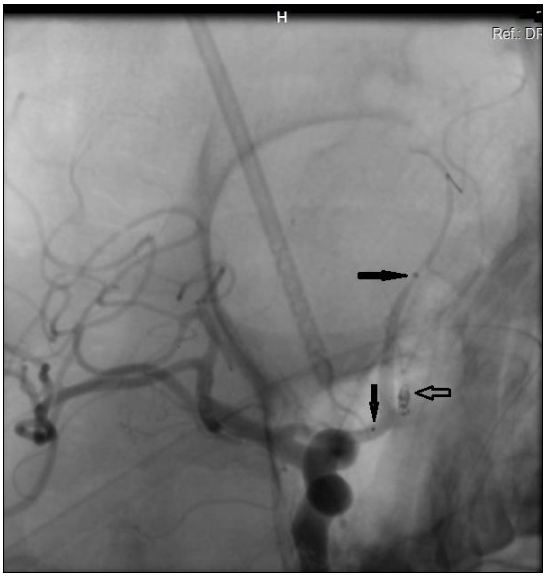

Fig 6: DSA showing coil in ACOM aneurysm and surrounding flow diverter. Solid arrow show two ends of flow diverter, hollow arrow shows coil in aneurysm sac.

Conclusions

Our case represents rare presentation of quadriplegia in a case of bilateral anterior cerebral artery infarcts due to thrombosis/vasospasm of bilateral ACA and recurrent heubner artery. The prognosis and duration of recovery may be variable depending upon reversibility of vasospasm.

Acknowledgement

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Conflict of Interest

None

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Informed Consent

Written consent was obtained from patient for publication.

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