



A CASE OF MIDDLE CEREBRAL ARTERY INFARCT FOLLOWING UNEVENTFUL CLIPPING OF ANTERIOR COMMUNICATING ARTERY ANEURYSM

Neurosurgery

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ABSTRACT

Background: Aneurysms of the anterior communicating artery are the most frequently encountered intracerebral aneurysms in routine neurosurgical practice. Management of intracerebral aneurysm involves aneurysm clipping or endovascular coil aneurysm embolisation. However, to the best of our knowledge, there is no reported case of an isolated ipsilateral middle cerebral artery infarction following clipping of anterior communicating artery aneurysm.

Case Description: A 65-year-old female with hypertension presented with a history of giddiness, fall and altered sensorium. The patient had a Glasgow Coma Scale (GCS) score of 12, and further investigation of the magnetic resonance imaging on suspicion of stroke revealed subarachnoid haemorrhage in the bilateral parietal sulcus, left Sylvian fissure, left ambient and quadrigeminal cisterns with intra-ventricular extension. The patient was subsequently referred for neurosurgery consultation. Computed tomography (CT) angiogram confirmed the presence of a ruptured anterior communicating artery aneurysm. The neurological assessment showed reduced responsiveness to verbal commands, with a Hunt and Hess score of 3. The patient underwent uneventful clipping of the aneurysm. Postoperatively, the patient did not wake up from anaesthesia and had persistently elevated blood pressure and right-sided hemiplegia. On the third day of postoperative care, a CT head scan revealed a left middle cerebral artery infarction with a midline shift. Decompressive craniotomy was performed, and following this procedure, the patient improved in sensorium with residual right-sided hemiplegia.

Conclusion: This case report presents an undetected new micro embolism or postoperative cerebral vasospasm as possible causes for the development of cerebral infarct in patients with subarachnoid haemorrhage following an aneurysm rupture.

KEYWORDS

Anterior communicating artery aneurysm; Middle cerebral artery infarct; Cerebral vasospasm

INTRODUCTION

Anterior cerebral artery aneurysms are one of the most commonly observed intra-cerebral aneurysms involved in subarachnoid haemorrhage (SAH). They most frequently involve the anterior communicating artery and are associated with a wide range of complications due to the blood vessel architecture and its encompassing anatomical structures.^[5,8] The treatment of choice for anterior communicating artery aneurysm includes microsurgical management with clipping of the aneurysm or endovascular coil embolisation of the aneurysm.^[10]

Although there have been ongoing advances in the surgical management of intracerebral aneurysms, there are still a significant number of complications that occur in the patients who undergo treatment.^[9] This case report presents an instance of an isolated ipsilateral middle cerebral artery (MCA) infarction following clipping of the anterior communicating artery aneurysm.

Case Report

A 65-year-old female was brought to the hospital with the complaints of giddiness and history of fall one week prior to admission. She had altered sensorium that was one day in duration. The patient had hypertension and was non-adherent to the medication for one week of duration. On examination, the patient's blood pressure (BP) was 200/110 mmHg, she had a Glasgow Coma Scale (GCS) score of 12 (E3V3M6) and she was moving all four limbs.

Control of the accelerated hypertension was initiated, and a magnetic resonance imaging (MRI) scan was ordered by the physician due to suspicion of stroke. The MRI revealed SAH in bilateral parietal sulcus, left Sylvian fissure, left ambient and quadrigeminal cisterns with intra-ventricular extension. Following this finding, the patient was referred for neurosurgical consultation. On evaluation, the patient was drowsy, moving all four limbs with bilateral reactive pupils but reduced responsiveness to verbal commands. She had a Hunt and Hess Score of 3 and a positive Babinski sign on the right side.

A computed tomography (CT) cerebral angiogram revealed a saccular aneurysm, 3.1 x 2.7 mm in size that arose from the anterior communicating artery. It was directed anterosuperiorly and right A1 artery was hypoplastic. Carotid and vertebral arteries were normal.

Echocardiogram showed no vegetation or valvular abnormalities and an ejection fraction of 60%.

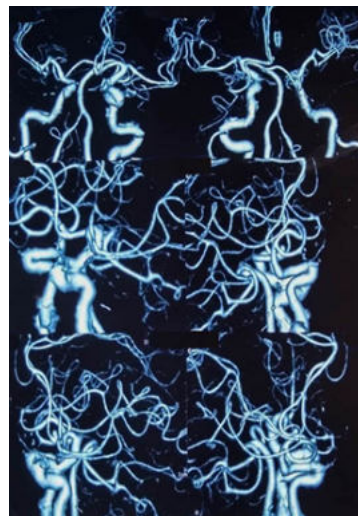


Figure 1: Computed tomography cerebral angiogram showing saccular aneurysm of 3.1 x 2.7mm arising from anterior communicating artery

The patient underwent a left pterional craniotomy and aneurysm clipping surgery. The operating procedure was straightforward and uneventful. After clipping of the aneurysm, all five vessels, including bilateral A1, A2 and anterior communicating artery, were visualised and found to be patent by intraoperative Doppler. Throughout the procedure, the left MCA was not exposed.

Post-surgery, the patient did not wake up from anaesthesia, and a persistently high BP of 200/110 mmHg was noted. Nitroglycerine (NTG) and increasing dose of labetalol failed to control the BP. Postoperative CT scan revealed operation site changes, with a midline shift to the right. There was no evidence of infarction. The patient was intubated and BP was brought under control after 6-h NTG infusion.

The patient remained unconscious and showed localized pain with right-sided hemiplegia. As the recurrent artery of Heubner was visualised intraoperatively, vasospasm of the same artery was suspected to explain the deficit.

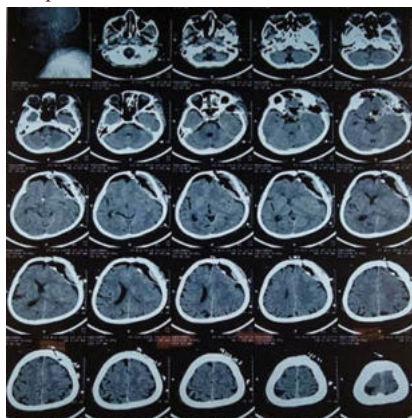


Figure 2: Postoperative computed tomography showing mass effect on left lateral ventricle and midline shift of 8mm to right

A CT head scan on the third postoperative day indicated a left MCA infarct with mass effect. However, CT angiography showed normally perfused left MCA and bilateral normal appearing internal carotid artery (ICA) and common carotid artery (CCA). A decompressive craniotomy with a tracheostomy was performed, which improved the sensorium of the patient. She regained consciousness over the next few days with residual right-sided hemiplegia.

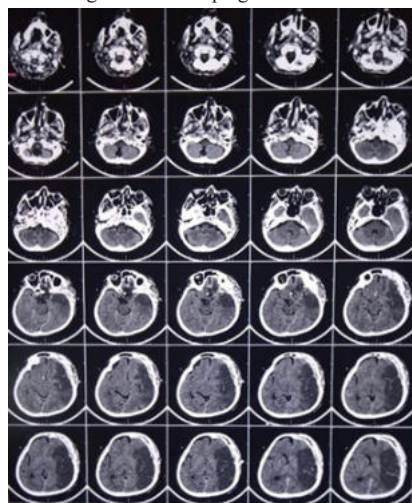


Figure 3: Computed tomography head scan done on the third postoperative day showing a left MCA infarct with mass effect

DISCUSSION

This case report reviews an unusual incidence of an isolated ipsilateral MCA infarction following clipping of the anterior communicating artery aneurysm in a hypertensive patient. The possible cause of the complication in such cases cannot be determined by the present case.

The anterior communicating artery lies under the frontal lobes and connects the anterior cerebral arteries. Thus, it interlinks and completes the anterior circulation of the brain.^[2] Aneurysms of the anterior communicating artery are very commonly observed in neurosurgical practice. It is well known that these aneurysms have a high risk of rupture, and most of the patients present with SAH following aneurysm rupture. The treatment of choice for intracerebral aneurysm includes surgical clipping or endovascular coiling of the aneurysm. Patients with SAH have several complications both perioperative and postoperative; these include cerebral vasospasm, hydrocephalus.^[3]

The possible pathophysiology in this patient with ipsilateral MCA infarction following the clipping of anterior communication artery aneurysm includes a new embolus that arose from an undetected

atheroma in the carotid arteries. However, in the present case, neither the preoperative nor postoperative CT angiogram showed atheroma in the ICA or CCA. Preoperative echocardiogram showed no evidence of any valvular abnormalities or vegetation. The other probable cause of the complication in this patient could be due to cerebral vasospasm of the left MCA. However, in the present procedure, the left MCA was not exposed during the surgery, and it is not possible that it underwent vasospasm due to mechanical manipulation. Nevertheless, there is ample evidence in the literature that shows patients with SAH following a ruptured aneurysm who undergo treatment have a higher risk of cerebral artery vasospasm. SAH causes progressive narrowing of the lumen of arteries, which ultimately reduces the cerebral blood flow and leads to infarct in patients.^[7] Studies demonstrated that cerebral vasospasm that leads to an infarct may occur as early as the third day of postoperative care or as a delayed complication.^[1,6]

Apart from the role of the natural course of aneurysm rupture in deciding the patient's outcome, surgical management of the aneurysm also played a significant role in postoperative outcome. Studies revealed that the risk of cerebral infarction with endovascular coiling is reduced compared to clipping of the aneurysm.^[4]

CONCLUSION

We reviewed a case of an elderly hypertensive female patient who presented with SAH following rupture of an anterior communicating artery aneurysm. The patient developed an isolated ipsilateral infarct of the left MCA following clipping of the anterior communicating artery aneurysm. This report illustrates that new or previously undetected microemboli and postoperative cerebral artery vasospasm could be possible causes of subsequent complication in such patients and should be carefully considered in postoperative management.

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