Traumatic Occlusion of the Middle Cerebral Artery

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Abstract

Traumatic occlusion of the middle cerebral artery (MCA) is very rare compared to occlusion of the internal carotid and basilar artery. Patients with longer latency have lower mortality. Herein, we report two cases of MCA occlusion who deteriorated under observation because of MCA territory infarction. CT angiography revealed occlusion of MCA in M1 segment. Both were subjected to decompressive craniotomy. First patient is ambulatory and has spastic gait. Second patient showed improvement in sensorium and still requires nursing care.

Keywords

- posttraumatic occlusion
- posttraumatic thrombosis
- middle cerebral artery
- decompressive craniectomy

Introduction

Traumatic occlusion of middle cerebral artery (MCA) is rare, more common being occlusion of basilar and internal carotid artery (ICA) and it carries a high morbidity. Recently, we came across two cases of MCA occlusion and because of rarity these are being reported. The patients were managed by decompressive craniectomy and showed improvement on follow-up.

Case reports

Case I

A 26-year-old man fell down from height of 12 feet. He did not lose consciousness. Next day, he had vertigo lasting for few hours. On third day he went to office. On return, he complained of headache and developed left-sided weakness. He was seen in another hospital where computed tomographic (CT) scan of the head revealed infarct in right frontoparietal region (►**Fig. 1a**). His sensorium deteriorated and patient was brought to this hospital. On examination, pulse was 86/minute BP was 110/70 mm Hg. He was drowsy but arousable. Pupils were normal in size and reacted to light briskly. There was upper motor neuron paresis of seventh cranial nerve and hemiplegia on left side. Plantars were extensor. Routine investigations were normal. Computed tomography revealed enlarged infarct with midline displacement to left side (►**Fig. 1b**). Urgent decompressive craniectomy was done. Patient gradually improved. CT angiography done on fifth postoperative day revealed occlusion of right MCA 3 to 4 mm distal to its origin (►**Fig. 1c**). He improved on follow-up and became ambulatory. Cranioplasty was done after 2 months. In postoperative follow-up, the patient has improved clinically with power being grade 3/5 in upper limb and grade 4/5 in lower limb on left side and he walks with spastic gait. CT scan at 6 months showed gliosis in the infracted region.

Case 2

A 37-year-old man fell down from 8 to 9 steps. He had altered sensorium since then. There was no history of bleeding from ear, nose, or throat. He had no vomiting or convulsions. On examination, GCS was E3V1M5. Both pupils were normal sized and normally reacting to light. There was weakness of the left upper limb. Respiration was spontaneous and adequate.

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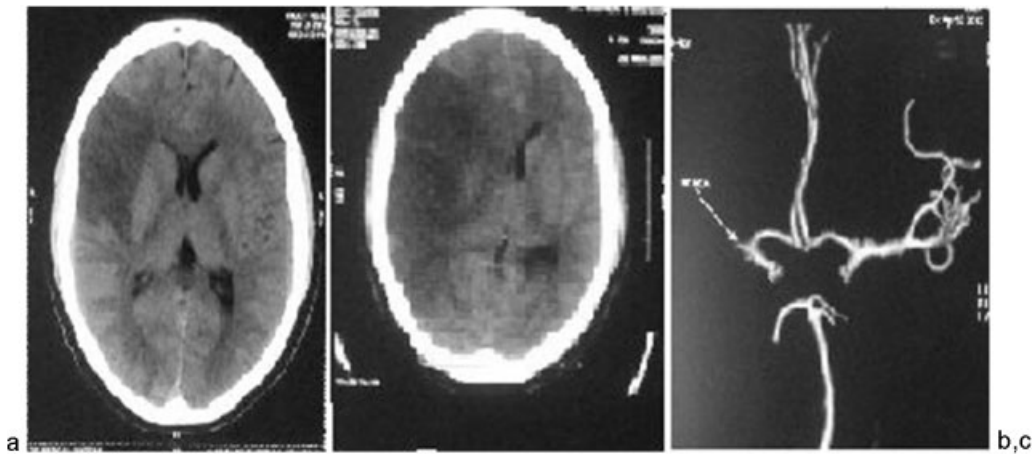

Fig. 1 Case 1: (a) Initial computed tomogram showing infarction in the middle cerebral territory. (b) Increased size of infarction with edema. (c) Computed tomographic angiogram showing occlusion of middle cerebral artery in M1 segment.

CT revealed small contusion in the frontal region and fracture in the right occipital bone. His condition deteriorated next day. Repeat CT revealed acute infarct in the middle cerebral territory (►**Fig. 2a**) and CT angiography revealed occlusion in the MCA in M1 segment (►**Fig. 2b**).

Decompressive craniectomy was done and the patient was managed with antiedema, anticonvulsants, antibiotics, and ventilator support. Tracheostomy was done. Patient improved gradually in sensorium and there was improvement in the motor power on the left side. Tracheostomy tube was removed.

Discussion

Intracranial vascular injury after head injury is a rare occurrence. In adults, it may be in the form of traumatic aneurysm, arterial dissection, or diffuse vasospasm.[1,2] Occlusion of MCA following blunt head trauma is rare cause of brain infarction. Majority of the cases occur following minor head injury without even loss of consciousness and our first patient also had no loss of consciousness. The time duration for the development of symptoms of occlusion vary from few minutes to days.[3,4] Patients usually presented with headache, contralateral weakness decreased consciousness, and aphasia. A definitive diagnosis of MCA occlusion cannot be made based on clinical features alone. In the recent years, CT has played a great role in the evaluation of the patients with head trauma. Early scans may be normal and subsequent scan may show infarction in the vascular territory.[5] We also had a similar observation in our case 2. CT angiography or routine angiography usually reveals the site of lesion clearly; though they may not differentiate the various pathological processes leading to the arterial occlusion.

Several mechanisms of pathogenesis of MCA occlusion have been propounded. Head injury may result in the separation of the intima from the media and outer layers. Intramural hematoma forms which results in the narrowing

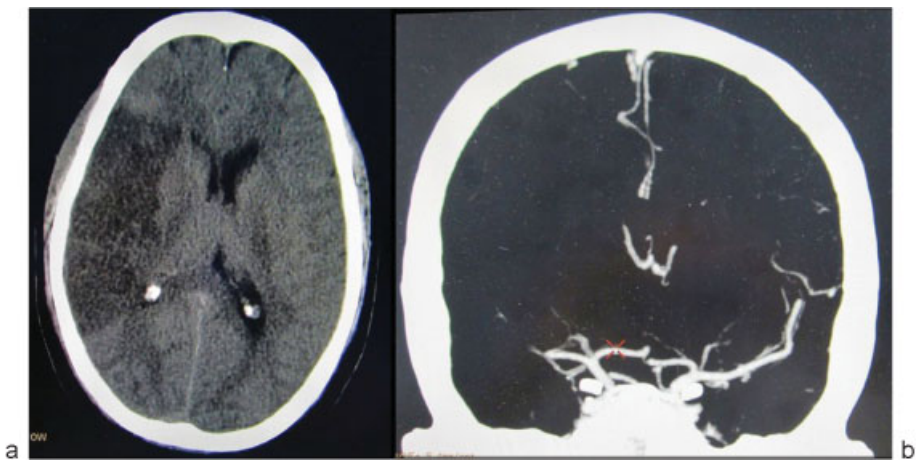

Fig. 2 Case 2: (a) Computed tomography (CT) showing infarct in the middle cerebral artery territory. (b) CT angiogram showing complete occlusion of middle cerebral artery in M1 segment.

and occlusion of the artery. The evidence of this has been found in the autopsied cases.[3] MCA may get pressed against lesser wing of sphenoid and it may affect the intimal charge leading to platelet accumulation and clot formation which may be facilitated by the decreased blood flow through the injured site. Sedzimir postulated the development of spasm of the supraclinoid portion of ICA caused by the stretching or compression by the neighboring bony structures.[6] It may spread upward to involve the MCA and give illusion of thrombosis. Another theory advanced for the occlusion has been embolus from the cervical portion of the ICA. Thrombus may form in neck in ICA due to direct contusion or stretching of the vessel by hyperextension, especially in boxers.[6] In our first patient because of the tapering appearance of the proximal patent MCA, it is suggestive of dissection rather than embolus or primary thrombosis. We had to resort to decompression of the brain because of the mass effect and continued antiplatelet aggregation therapy with aspirin. Treatment consists of steroids and vasodilators.[4,7] Mortality of the condition is high 33.33%.[4] Short duration of latency especially less than 24 hours carries mortality of 58.3%, whereas latency more than 24 hours has low mortality of 15.3%.[8]

There is no consensus for the treatment of traumatic middle cerebral occlusion. High index of suspicion of MCA occlusion should be the practice in patients with changes in the level of consciousness or lateralizing neurological findings unexplained by initial CT scans and may necessitate CT, CT angiography, magnetic resonance angiography, or conventional angiography in the suspected cases. Currently practiced guidelines are in the form of anticoagulants, antiplatelets, or stenting of the occluded site if there is dissection of MCA. If patient develops large infarct in the whole territory of MCA, then decompressive craniectomy is required. In total occlusion of MCA with large infarct, angioplasty or stenting should not be preferred as it causes reperfusion hemorrhage in the brain.[9]

Anticoagulation is preferred to antiplatelets when there is severe stenosis, arterial occlusion, or pseudoaneurysm, whereas antiplatelet therapy is preferred in cases of large infarcts, intracranial dissections, high risk of bleeding, or inadequate collateral circulation.[10] We had to undertake surgical decompression because of the mass effect due to large infarct.

Conflicts of Interest
The authors have nothing to declare.

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