# Traumatic thrombosis of internal carotid artery

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Abstract: 27 year old soldier sustained closed head injury in a road accident. He was unconscious for about 6hr.When seen at our hospital his GCS was 15/15; normal pupil but he had left hemiplegia. Head CT scan showed a low attenuation lesion in right fronto-parietal region suggestive of infarct. A cerebral angiogram revealed right internal carotid occlusion. He was treated with unsuccessful thrombectomy and STA-MCA by pass, anti-platelet agents and improved partially. Problems of traumatic ICA thrombosis in the setting of head injury are discussed.

Keywords: Head injury, Traumatic internal carotid thrombosis.

## INTRODUCTION

Majority of extra-cranial traumatic injuries to internal carotid artery (ICA) are caused by direct trauma from penetrating injuries especially from firearms<sup>1,2</sup>. There is a small subset of injuries of extra-cranial ICA injuries caused by blunt trauma and some of these occur in the setting of head trauma. The incidence of carotid injury in association with head injury is reported to be between 0.05to 0.5%<sup>3,4</sup>. These injuries are generally missed on initial examination unless one is aware and vigilant. We are reporting a case ICA thrombosis associated with head injury.

#### CASE REPORT

A 27-year-old soldier met with road traffic accident, following which he was unconscious for 4-6 hours, on regaining consciousness noticed weakness of left half of his body. He was initially treated in a private hospital and shifted to our hospital next day. On examination at our centre he was found to be haemodynamically stable. Neurological examinations revealed GCS score of 15/15, normal pupillary size and reflexes, left hemiplegia (power 0/5), left extensor plantar but no sensory deficit. He also had abrasions on the left side of forehead. There was no neck injury or bruit. His haematological and biochemical investigations were normal. A CT scan and MRI of the head revealed a low attenuation lesion in the peripheral distribution of right middle cerebral artery

(Figs 1 & 2)). A four vessel cerebral angiogram showed occlusion of right ICA (Fig 3). He under went open thrombectomy by our vascular surgeon, however the distal occlusion could not be opened up as seen on intraand postoperative angiograms (Fig 4). In view of persisting hemiplegia, small peripheral infarct of right of MCA a PET scan was done which showed low perfusion in right MCA territory. He was finally taken up for right superficial temporal-middle cerebral artery (STA–MCA) anastomosis, following which there was some improvement in his hemiplegia. However, postoperative angiogram did not show patency of STA-MCA anastamosis. In addition to surgical interventions he was given disprin 150mg and clopidgrel 75 mg once a day.



Fig 1: CT showing infarct in the region of middle cerebral artery

Fig 2: MRI showing infarct in the region of middle cerebral artery

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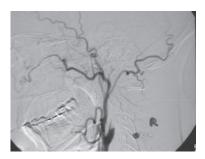


Fig 3: DSA showing occlusion of the internal carotid artery (common carotid artery injection)

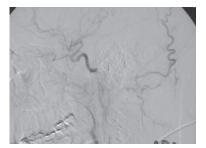


Fig 4: DSA showing non-visualisation of right internal carotid artery

# **DISCUSSION**

Incidence of carotid artery injury in the setting of head injury is very low and can be missed in the setting of cranio-cerebral trauma. Yamada et al could collect only 52 cases in a review of literature<sup>5</sup>. Because of this low incidence there is lack of awareness that results in delay in the diagnosis. Usually there are signs and symptoms such as transient ischaemic attacks (TIA)/ reversible ischaemic neurological deficit (RIND)/ hemiparesis / hemisensory loss/ hemianopsia, suggesting ischaemia of ICA but in the setting of head injury these symptoms may not be there and neurological deficit can be easily be attributed to the head injury. When ischaemia is severe, neurological deficit is disproportionate to the level of consciousness, which was also the case in our patient2. High index of suspicion (hyperextension-hyper flexion mechanism of injury, basilar skull fracture, cervical spine injury, mid face fracture, mandibular fracture, diffuse axonal injury and neck seat belt sign) clinches early diagnosis or directs efforts for early diagnosis and treatment<sup>4</sup>. The suggested mechanism of injury is hyper extension and rotation of neck which results in stretching and compression ICA against lateral mass C1 or C2 vertebra that causes either tear in intima or haematoma in the media 6, 7,8 of arterial wall and subsequent

thrombosis and occlusion. Angiography is gold standard for diagnosis but CT scan, MRI are complimentary and will so the extent of infarct. MRA, CT angiography can also be useful in showing the stenosis/occlusion/tear of intima or haematoma in the wall of ICA9. PET scan of brain will show the area of hypo-perfusion which can be more than the area of infarct as was seen in our case as well.

Anticoagulation is mandatory for blunt carotid injuries to reduce the chances of stroke, provided there is no contraindication and patients are promptly diagnosed before occlusion 10. Once occlusion has occurred surgical intervention becomes imperative. Surgical management means thrombectomy or embolectomy which needs to done expeditiously<sup>11, 12, 13</sup>. In our patient due to late arrival, it was done late and complete clearance of thrombus could not be achieved. Augmentation of cerebral circulation by emergency EC –IC bypass is done through STA-MCA anastamosis or saphenous vein graft and this requires experience. Though the need to restore the cerebral circulation is always urgent there is invariable delay in execution of surgical procedure as happened in our patient as well. This report also high lights rare occurrence of blunt carotid injury in the setting of closed head injury, which is usually missed due to lack of awareness for early diagnosis and treatment or reaches late for any worthwhile therapeutic intervention and results in avoidable morbidity and mortality<sup>14</sup>.

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