Cerebral Infarction in a Child after Moderate Brain Trauma: A Case Report

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Abstract

Intracranial hematomas and contusions are common entity after traumatic brain injury in children and young population. However, cerebral infarction after traumatic head injury is a rare entity with frequency ranging from 1.9 to 10.4%. We herein report a case of cerebral infarction in right anterior cerebral artery (ACA) and parts of middle cerebral artery (MCA) territory after moderate head injury in a 14-year-old boy, in whom computed tomographic (CT) scan of the brain was performed 3.5 hours after injury, which showed specks of pneumocephalus in sellar suprasellar region, and 12 hours after injury showed right ACA territory infarct and evolving infarct in right MCA territory. Bony injury included right orbital roof and right sphenoid wing linear undisplaced fractures. Possible mechanism for development of infarction is right internal carotid artery (ICA) dissection by fractured bone fragments and/or thrombosis. The patient developed hemiplegia on second day after trauma. Intracranial pressure (ICP) monitoring was done, which was suggestive of raised ICP. The patient underwent right fronto-temporo-parieto craniotomy and decompression (tissue sent for histopathologic analysis) and augmentative duraplasty with bone flap replacement on fifth day after trauma. Histopathology was suggestive of cerebral infarct with hemorrhage. The patient was improved at follow-up, 10 months after trauma after rehabilitation, and physiotherapy to modified Rankin scale (mRS) score 3. Hence delayed CT scan plays a vital role in detecting developing posttraumatic infarcts, and rigorous rehabilitation care is necessary for clinical improvement.

Keywords
- infarct
- traumatic
- computed tomographic scan
- modified Rankin scale
- rehabilitation

Introduction

Intracranial hematomas, contusions are common after traumatic brain injury (TBI) in children and young population. However, cerebral infarction after traumatic head injury is a rare entity with frequency ranging from 1.9 to 10.4%.[1-6] Diagnosis of this pathology in the pediatric population is usually difficult.[7,8] Such pathologies can be associated with hematologic alterations and vascular lesions.[9] Here, we present a case of boy who suffered from a right anterior cerebral artery (ACA) and parts of middle cerebral artery (MCA) territory infarction after moderate TBI. Possible mechanism for development of infarction is right internal carotid artery (ICA) dissection by fractured bone fragments and/or thrombosis. This case highlights importance of delayed computed tomographic (CT) scan in detecting developing infarcts after head injury as well as importance of rigorous rehabilitation for clinical improvement.

Case Report

A 14-year-old boy presented to emergency department due to head trauma after road traffic accident, a pedestrian hit by a tractor. The patient presented with the complaint of loss

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of consciousness for 2 hours, one episode of vomiting, and left ear bleed. On examination, he was in altered sensorium with Glasgow coma scale (GCS) score 9 (E2M5V2), right pupil was not reacting and larger than left, left-sided paucity of movements, and he was hemodynamically stable. CT scan of the brain was performed 3.5 hours after injury showed specks of pneumocephalus in the sellar and suprasellar region with right sphenoid wing fracture and right orbital roof fracture (►**Fig. 1A**, **1B**).

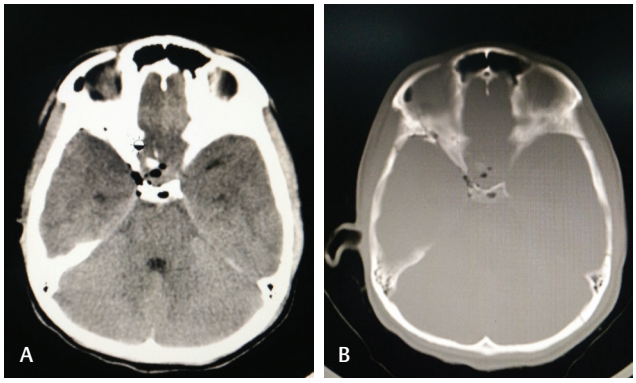

Fig. 1 (**A**) CT brain plain 3.5 hours after trauma. (**B**) CT brain plain 3.5 hours after trauma (bone window). CT, computed tomography.

The patient was managed on antiedema measures, and CT scan repeated 12 hours after trauma (►**Fig. 2A**, **2B**) showed right ACA territory hypodensities. CT scan performed 3 days after trauma showed right ACA and parts of right MCA territory infarcts. Left-sided limb weakness progressed to hemiplegia. Magnetic resonance imaging (MRI, fluid attenuation inversion recovery [FLAIR], diffusion-weighted imaging [DWI] sequences) with contrast done 4 days after trauma showed well-developed infarcts in the right ACA territory as well as right MCA territory supplying basal ganglia and frontal lobe regions with edema and midline shift to left approximately 7 mm (►**Fig. 3A**). Magnetic resonance angiography (MRA) with time-of-flight (TOF) sequence showed nonvisualization of the right ICA, right ACA, and right MCA (►**Fig. 3B**). Collaterals from right posterior cerebral artery (PCA) and posterior communicating artery (PCOM) were seen supplying right MCA territory.

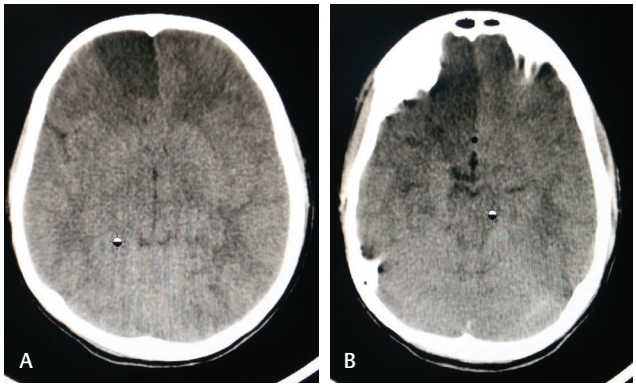

Fig. 2 (**A**) Right ACA territory infarct. (**B**) Right MCA supplying basal ganglion region infarcts. ACA, anterior cerebral artery; MCA, middle cerebral artery.

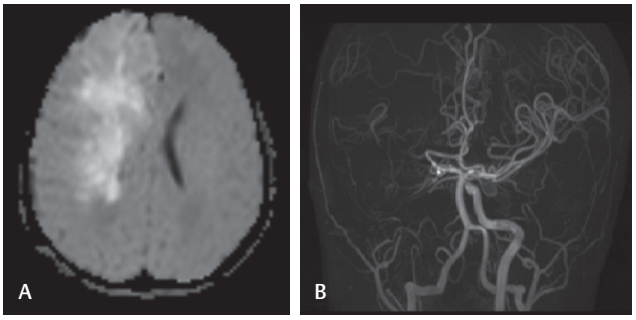

Fig. 3 (**A**) Magnetic resonance imaging (diffusion-weighted image) with contrast done 4 days after trauma showed well-developed infarcts in right ACA territory as well as right MCA territory supplying basal ganglia and frontal lobe regions. (**B**) MRA showing nonvisualization of the right ICA, right ACA, and right MCA. ACA, anterior cerebral artery; ICA, internal carotid artery; MCA, middle cerebral artery.

The patient was put on intracranial pressure (ICP) monitory through external ventricular drain. ICP was raised. The patient underwent right fronto-temporo-parieto craniotomy and decompression fifth day after trauma. Intraoperatively diseased brain was pale and bulging with edema present, diseased tissue sent for histopathologic analysis, and bone flap was loosely replaced to counteract raised ICP. Postoperative CT scan showed well-developed right ACA and part of MCA territory infarct. The patient was put in intensive care unit (ICU) for elective ventilation. He was gradually weaned off and extubated on ninth postoperative day. Histopathology report of surgical specimen was suggestive of cerebral infarct with hemorrhage without any evidence of thrombosed veins. At discharge, the patient had GCS of 8T (E3M5VT) and left hemiplegia. He was advised to undergo regular physiotherapy and general nursing care. At follow-up, 10 months after injury, the patient's left-sided weakness improved (mRS score 3), and he was able to lift left upper limb and lower limb against gravity.

Discussion

The secondary effects of TBI might be more critical than the primary injuries. Cerebral infarction after brain trauma has also been recognized as a potential secondary injury though less common than other secondary injuries such as cerebral edema, posttraumatic hydrocephalus, and vasospasms. In this case, we tried to highlight clinical presentation; importance of CT scan specifically delayed CT and need of rehabilitation in posttraumatic cerebral infarct.

Most patients suffering from mild head injuries achieved good recovery and needed supportive care. However, a small number of these patients underwent subsequent neurologic impairment due to raised ICP due to edema or by the presence of an intracranial expansive mass such as developing intracranial hematoma. Therefore, the patient must stay in the hospital approximately 6 hours to be clinically monitored.[10] Patients with moderate head injury defined as GCS less than 13 require more meticulous observation. Bae et al suggested that increasing age, GCS at admission, and brain herniation were risk factors for posttraumatic cerebral infarction (PTCI).[11] Early recognition of these factors and

prompt treatment may prevent PTCI, or at least minimize fatal consequences.[11]

Various mechanisms for development of PTCI are described in literature such as mechanical shift and subsequent transtentorial or subfalcine herniation and development of infarction, blunt vascular injury, and vascular injury by bony fracture fragment especially skull base fractures.[1,4] CT scan plays a vital role in detecting herniation, skull base fractures, and developing infarcts. In this case, the young male patient developed right ACA territory and part of MCA territory infarct after a road traffic accident. Delayed CT scan showed developing infarct correlating with the patient's left-sided weakness. Clinical presentation, serial CT scans, MRI, and MRA of the brain showed right ACA and part of right MCA territory infarction. Mechanism of injury can be posttraumatic right ICA dissection and/or thromboses. The patient was operated on with craniotomy and biopsy of lesion with loosely replacing bone flaps. He underwent physiotherapy and rehabilitation. During follow-up, his left-sided hemiplegia improved. Hence, meticulous observation for neurologic deterioration, serial CT scans, diagnosis, and management of raised ICP as well as long-term rehabilitation therapy are vital in a case of PTCI.

Conflict of Interest

None.

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