# Pictorial CME

## Cerebral Infarction following Head Injury

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**Case 1 :**

A 12 year old boy presented to usin an unconscious state following a road traffic accident. He regained consciousness the next day and was noted to have weakness of right upper limb and right lowerlimb. On examination, the patient was conscious, pupils 3 mm in size, equally reacting to light on bothsides and extra ocular movements were normal. Right upper motor neuron facial palsy was present. There was right upper and lower limb weakness of MRC grade 3 . Bilateral extensor plantarwas noted. Magnetic Resonance Imaging(MRI) brain showed T1 hypointensity, T2 & FLAIR hyperintensity with restricted diffusion in DWI and ADC map over bilateral basal ganglia suggestive of bilateral basal ganglionic infarcts (Fig 1). Magnetic Resonance Angiography (MRA) showed normal study. Routine blood investigations includingblood biochemistry, haematological profile,cardiac evaluation including echocardiogram were normal. Diagnosis of cerebral infarction following head injury was made. Patient was treated with antiedema measures, physiotherapy and supportive measures. Patient is improving.

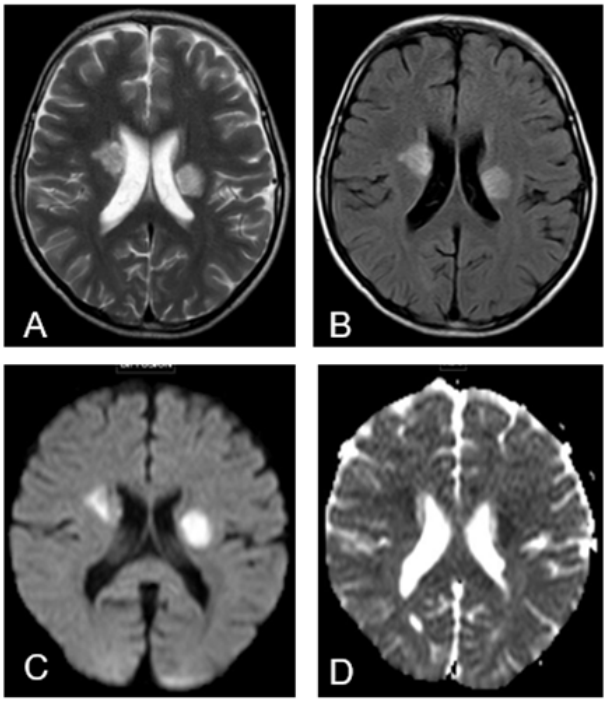


Fig 1 — MRI-T2 AXIAL (A) and FLAIR axial (B) images showing hyperintensity with restricted diffusion in DWI(C) and ADC (D) map over bilateral basal gangliasuggestive of acute infarct

**Case 2 :**

29 years old, previously healthy male was brought to the hospital in a state of unconsciousness following a road traffic accident. 3 days later, patient developed drooping of right eyelid. On examination, patient was consciousand had complete right third cranial nerve palsy with 5mm pupil size. There was no weakness of limbs. Plantars were flexor bilaterally. Magnetic Resonance Imaging(MRI)brain showed T1 hypointensity, T2 & FlAIR hyperintensity with restricted diffusion in DWI and ADC map over splenium of corpus callosum and right side thalamus suggestive of acute infarcts in splenium of corpus callosum and right thalamus

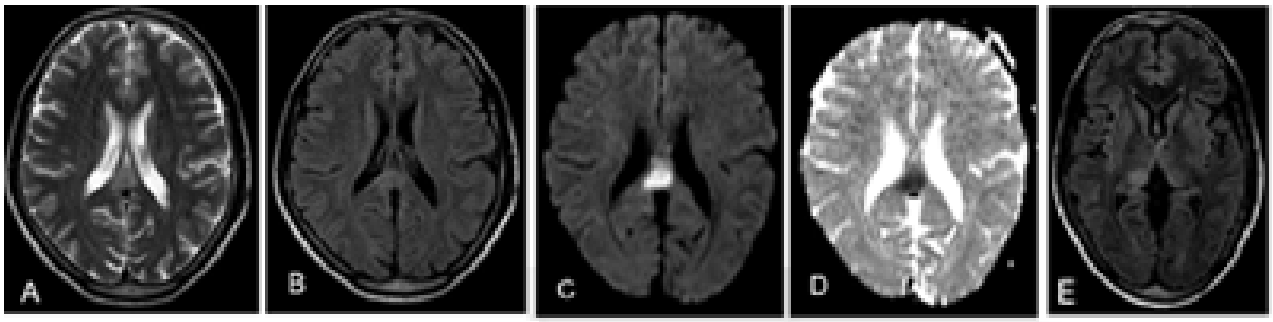


Fig 2 — MRI- T2 AXIAL (A) and FLAIR axial (B) images showing hyperintensity with restricted diffusion in DWI(C) and ADC (D) map over splenium of corpus callosum suggestive of acute infarct and MRI-FLAIR axial (E) shows hyperintensity in right thalamus

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(Fig 2). Magnetic Resonance Angiography (MRA) showed normal study. Routine blood investigations including blood biochemistry, haematological profile, cardiac evaluation including echocardiogram were normal. Diagnosis of

cerebral infarction following head injury was made. Patient was treated with antiedema measures, physiotherapy and supportive measures. Patient is improving.

Cerebral infarction following head injury occurs in less than 2 percent of all strokes. Other possible causes should always be excluded before making the diagnosis of post traumatic stroke. Imaging of brain and cerebral vessels must be done to look for lesions and dissection. Arterial dissection, thrombosis and vasospasm are the suggested pathogenic mechanisms of post traumatic infarcts[1]. Brain displacement across dural surface accounts for post traumatic infarction. Occipital infarctions are more common as posterior cerebral artery is being compressed by the temporal lobe against the tentorium.

Basal ganglia is supplied by lenticulate striate branches of middle cerebral artery. The angle between the middle cerebral artery and lenticulate striate artery is more acute and since these are functional end arteries, stretching and distortion of the angle of perforating branches during trauma leads to damage to the vessel and reduces blood flow. Less frequently, lenticulostriate,thalamoperforating and choroidal arteries are compressed against the skull base resulting in basal ganglionic infarction[2]. Corpus callosal infarcts are not common due to the rich blood supply from three arterial systems. Splenium is vulnerable than genu and body of corpus callosam.

The possible mechanismby which infarction occurred incase no 1 is due to distortionof lenticulostriate branches of MCA on both sides producing basal ganglionic infarcts and compression of right posterior cerebral artery in Case 2 causing right thalamic and splenial infarcts. The complete 3rd cranial nerve palsy on right side in Case 2 is due to ipsilateral paramedian thalamic infarct. The midbrain and thalamus have a similar vascular supply. Some individuals may have vascular variations also. Oculomotor nerve palsy is a common symptom of a midbrain infarction, but rare cases of a paramedian thalamic infarction causing third-nerve palsy without a definite lesion on the brainstem, have been reported. Even though there was no definite midbrain lesion in this case, the third nerve palsy can be partly attributable to the involvement of the oculomotor nuclei or fascicle, which is usually associated with thalamic lesion extension[3].

## Conclusion :

Post traumatic infarctions are rare. They are closely related to anatomical peculiarities of brain skull base in children. Hospital admission, careful observation and early DWI MRI should be considered in patients having neurological deficits.

### References


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3 Thurtell MJ, Halmagyi GM — Complete ophthalmoplegia: an unusual sign of bilateral paramedian midbrain-thalamic infarction. *Stroke* 2008; **39:** 1355-7.