Midline frontal depressed skull fracture with venous infarct

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Cerebral venous thrombosis (CVT) is a condition where thrombosis of the cortical or deep cerebral veins or venous sinuses occurs resulting in obstruction to the venous outflow and known to occur due to variety of reasons. The causes of CVT include hypercoagulable states, dehydration, intracranial infection, congenital cyanotic heart disease, pregnancy, oral contraceptive pills usage, malignancies and rarely trauma¹⁻³.

A 52 year old female patient was admitted to our neurosurgical unit 10 days after being allegedly assaulted with a "lathi" (wooden rod, an Indian weapon). She was brought by her relatives with complaints of headache and abnormal behavior for last 2 days. She had no history of loss of consciousness or seizures. On neurological examination she was conscious but irritable. She was responding to verbal commands but was not fully oriented to time, place and person. She did not have any focal motor or sensory deficit. Her pupils were bilaterally equal and reacting well to light. Neurological examination of her cranial nerves was within normal limits. Her CT Brain revealed a depressed fracture in the frontal region in the midline and the depressed bone fragment was causing compression of the anterior third of the superior sagittal sinus. Right frontal lobe showed area of hypodensity suggesting associated venous infarct (Figs 1 and 2). As the depressed fragment was involving the anterior two third of the sinus a conservative management approach was adopted.

Cerebral sinovenous thrombosis (CVT) after head injury is relatively uncommon accounting for up to 4% of CVT cases and is usually seen following penetrating head injury⁴. Compression of the sinus by adjacent structures with progressive thrombosis or direct trauma

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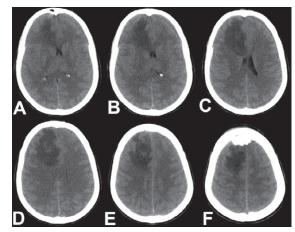


Fig 1: CT scan brain (plain) showing diffuse hypodensity in right frontal lobe mainly involving the white matter (note- the lesion is not confined to any arterial territory)

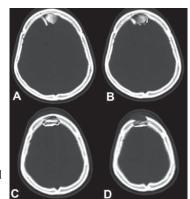


Fig 2: CT scan brain (bone window) showing midline frontal depressed fracture

to the endothelium of the sinus has been suggested as possible causative mechanism. There are however reports documenting that a mild closed head injury, even without skull fracture crossing over the sinus, can cause CVT⁵. In our case, the depressed fractured segment in the midline over superior sagittal sinus caused the obstruction to the venous flow resulting in cortical venous infarct.

Clinical presentation of cerebral venous thrombosis varies widely and progression and severity of the symptoms depends on the degree of thrombosis and presence of collateral draining system. Headache is the

most common and predominant symptom and is seen in more than three- fourth patients⁶. Focal or generalized seizures followed by hemiparesis, aphasia, hemianopia, or other focal neurologic dysfunction may be present. Neuroimaging (CT, MR imaging) suggest an ischemic lesion with a margin that does not match arterial distribution (as in present case). Although cranial CT and MR may visualize the thrombosed sinus, MR venography and if necessary conventional angiography should be performed to evaluate the venous system completely⁷.

Treatment options for cerebral sinus venous thrombosis includes standard or low molecular weight heparin followed by long term oral anticoagulation8, direct thrombolysis by endovascular local infusion of urokinase or tPA9, mechanical thrombolysis using microballoon percutaneous transluminal angioplasty10 and direct thrombectomy11. Although anticoagulation is considered to be safe even in cases with hemorrhagic infarct, its use in patients with trauma is controversial because of the risk of increased intracranial bleeding. Role of surgery in these cases is limited to decompressive craniectomy in cases where the intracranial pressure is very high. In cases with depressed fracture overlying the venous sinus elevation is not indicated, however if a major sinus occlusion results in raised ICP with deteriorating neurological status, an urgent elevation of the depressed segment and sinus repair with restoration of blood flow has been recommended 12.

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