Letters to Editor

Cerebral venous sinus thrombosis in closed head trauma: A call to look beyond fractures and hematomas!

Dear Editor,

Development of cerebral venous sinus thrombosis (CVST) secondary to closed head injury with or without skull fractures or intracranial hematomas is being increasingly reported in the recent literature.[1-3] Venous sinus thrombosis has been shown to be one of the unsuspected causes of delayed intracerebral hemorrhage (ICH) following head trauma.[2,4] In view of enormous global burden of head trauma cases, particularly in the developing countries, few recognized cases perhaps represent the tip of the iceberg of this potentially treatable complication. Early imaging-based diagnosis is of paramount importance to prevent the grave consequences of venous hypertension in the settings of co-existing cerebral injuries masking the clinical diagnostic clues.

Most cases of acute posttraumatic CVST are associated with skull fracture occurring close to a dural sinus; however, delayed sinus thrombosis can occur even in the absence of skull fracture.[4,5] The acute CVST in blunt trauma is associated with skull fractures; the risk is highest when a fracture extends to a dural sinus or jugular bulb. Fractures of the petrous temporal bone have the highest chance of injury to the transverse sinuses, sigmoid sinuses, and jugular bulbs, whereas association of occipital bone fractures is higher with thrombosis of the superior sagittal sinus.[5] Matsushige et al. showed that the most commonly involved sinus is posterior part of the superior sagittal sinus; however, sigmoid sinus thrombosis is the most common site according to Daligic et al.[6] Even minor trauma has been reported to cause venous sinus thrombosis in children.[1]

The pathogenesis of cranial trauma related CVST has not been well-established yet. Various postulated hypotheses include skull fractures or intracranial hematomas causing thrombosis by direct compression of the sinus, endothelial injury within the sinus leading to the activation of the coagulation cascade, intramural hemorrhages due to rupture of small sinusoids, extension of the thrombus from injured emissary veins and compression of the sinuses from intracranial edema.[1]

Imaging plays an important role in early diagnosis of CVST as clinical manifestations are varied and nonspecific. Posttraumatic CVST can be easily overlooked due to the low index of suspicion, unawareness of its association with head injury, and difficulty in diagnosing the venous thrombosis on noncontrast computed tomography (NCCT).

Furthermore, initial imaging may be normal as CVST can follow a subacute course occurring after few days or weeks after the head injury.[2,7]

NCCT is usually the first imaging investigation. The radiological signs of CVST can be divided into two categories, direct and indirect. The direct signs enable the demonstration of the intravenous thrombus on imaging, whereas the indirect signs reflect ischemic or vascular changes due to venous outflow obstruction. Delta sign, cord/dense vein sign on NCCT, and empty delta sign on contrast CT studies are the characteristic signs. Magnetic resonance (MR) imaging with MR venography (MRV) has nearly replaced by invasive cerebral angiography and conventional CT; however, CT venography is a reliable investigation and can be performed in the emergency settings without any additional delay when initial NCCT is equivocal or suggestive of venous thrombosis.[1,7]

Therefore, it is very important to include careful evaluation of cerebral venous sinuses in the diagnostic checklist when looking at admission CT in all cranial trauma cases, particularly in patients with fractures or hematomas in close proximity to venous sinuses e.g., basilar skull fractures, which are associated with transverse/sigmoid sinus thrombosis.[2,4] Similarly, not all extra-axial hyperdense hemorrhagic collections are subdural hematomas when located in the posterior fossa and/or along the interhemispheric fissure, and deserve careful scrutiny to exclude or confirm associated with dural sinus injury. Second, the persistence or emergence of raised intracranial pressure features in delayed or subacute period e.g., persistent headache, seizure, papilledema, etc., with CT showing unexplained cerebral edema or delayed ICH, should be promptly investigated with appropriate vascular imaging (CT venography/MRV). There is some concern about the use of anticoagulants in head trauma patients. Hence, the role of hyperosmolar therapies (mannitol and hypertonic saline) has been advocated as first-line treatment to lower intracranial pressure. If there is further worsening of the clinical condition, systemic anticoagulation, surgical decompression or endovascular treatments such as chemical thrombolysis or mechanical thrombectomy can be tried.[2]
38

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Conflicts of interest
There are no conflicts of interest.

Mandeep Singh Ghuman, Pravin Salunke¹, Sushanta K. Sahoo¹, Shabdeep Kaur²

Departments of Neuroradiology and ¹Neurosurgery, Post Graduate Institute of Medical Education and Research, Chandigarh, ²Department of Radiology, Indira Gandhi Medical College, Shimla, Himachal Pradesh, India
E-mail: dr.msghuman@gmail.com

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