lying cause of neurological symptoms seen in vitamin B12 deficiency.

The exact mechanism involved in epileptogenesis due to cobalamin deficiency is not clear. It is likely that cerebral neurons with destroyed myelin sheaths are more susceptible to the excitatory effects of glutamate. Cobalamin deficiency may share similarities with multiple sclerosis in this regard.

In conclusion, seizures rarely occur in patients with vitamin B12 deficiency. Serum B12 levels should be checked, especially in those patients who present with other known neuropsychiatric features of vitamin B12 deficiency. Early withdrawal of antiepileptic drugs should be attempted as long-term antiepileptic use is not warranted and may be associated with adverse effects in such cases. Chronic carbamazepine therapy has been found to lower the levels of vitamin B12 and folate.

References


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Superior sagittal sinus and torcular thrombosis in minor head injury

G. S. S. Kumar, A.G. Chacko, M. Joseph
Department of Neurological Sciences, Christian Medical College and Hospital, Vellore - 632004, India.

A 27-year-old man suffered a relatively minor trauma. He developed signs of raised intracranial pressure three days after injury. Investigations revealed superior sagittal sinus and torcular thrombosis.

Key Words: Anticoagulation, Superior sagittal sinus, Head injury.

Introduction

Post-traumatic superior sagittal sinus and torcular thrombosis is rare. Various mechanisms in its pathogenesis have been postulated but mortality and morbidity remain high. We report such a case and discuss the treatment protocol.

Case Report

A 27-year-old man presented a few hours after a head injury and brief loss of consciousness. When admitted he was irritable but had no focal neurological deficits. There was a scalp laceration in the midline occipital region with an underlying linear fracture. The laceration was sutured and he was admitted for observation as the fracture line extended across the sagittal sinus. A plain computed tomogram (CT) of the brain showed a crack fracture of the occipital bone on the right side, which extended to the midline. Rest of the brain was normal (Figure 1). Seventy-two hours after the trauma he developed headache and vomiting and developed bilateral sixth cranial

Figure 1: Plain CT at admission showing fracture of the occipital bone on the right side extending up to the midline. Rest of the brain was normal

Mathew Joseph
Department of Neurological sciences, Christian Medical College and Hospital, Vellore - 632004, India.
nerve pareses. Fundoscopy revealed papilledema. A magnetic resonance angiogram (MRA) showed a block in the anterior third of the superior sagittal sinus and a thrombus partly occluding the confluence of sinuses (Figure 2). The patient was placed on anticonvulsants and anti-edema drugs and heparin with which his symptoms improved over five days. Heparin was then changed to oral anticoagulants and he was discharged when he was asymptomatic.

Discussion

Thrombosis of the superior sagittal sinus (SSST) and torcular is rare and is usually attributed to scalp or skull infections, oral contraception, dehydration, pregnancy, blood dyscrasias and metabolic derangements.1,2 Although post-traumatic SSST has been reported, it still remains an unusual complication of traumatic brain injury. The high mortality rate (40%-80%)3 stresses the need for early diagnosis and treatment.

The diagnosis is based on a high index of clinical suspicion, confirmed by imaging. General symptoms and signs are headache and seizures due to cerebral venous infarcts.4 Buonanno et al.5 reported 11 cases of SSST diagnosed with CT and later confirmed by angiography or autopsy. A reliable finding on CT is the “empty triangle” sign seen on contrast films in the cuts slicing perpendicularly across the posterior aspect of the sinus. The “empty triangle” is due to the presence of an isodense clot within the sinus enclosed by an area of engorged vessels. Less specific findings include small ventricles, gyral enhancement in a distribution of venous infarct and multiple focal bilateral parasagittal hemorrhages.

The pathogenesis of SSST is not well established. Carrie and Jaffe5 state that abnormalities in the clotting mechanism, disturbances in blood flow or damage to the capillary endothelium may predispose and lead to thrombosis. Alteration in coagulation after head injury has been documented supporting this hypothesis.6

Treatment includes maintaining good hydration, and the administration of anti-edema agents like dexamethasone and mannitol. Anticonvulsants are necessary to prevent seizures. There are reports of the successful management of SSST with heparin and urokinase, including the restoration of the patency of the sagittal sinus with continuous urokinase infusion directly into the sinuses via a transfemoral transvenous microcatheter.7,8

References


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Atlantoaxial dislocation in idiopathic cervical dystonia

S. Kanekar

Department of Radiology, PO Box 12, Salmania Medical Complex, Manama-Bahrain.

We report a case of severe cervical spondylosis and atlantoaxial dislocation (AAD) in association with idiopathic cervical dystonia (ICD) in a middle-aged male. To our knowledge, this is the first case of ICD reported in association