Post-traumatic cerebellar infarction due to vertebral artery foramina fracture: case report

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Abstract: Posttraumatic cerebral infarction is an uncommon cause of morbidity and mortality and many studies have highlighted that trauma needs to considered as causative factor for cerebellar infarction. We present a case of cerebellar infarction in a 35 year old young patient secondary to vertebral fracture involving the vertebral foramen and vertebral artery injury. CT scan cervical spine showed C2-3 fracture on left side with fracture extending into the left vertebral foramen. A CT scan angiogram could not be performed because of poor neurological status. Possibly the infarction was due to left vertebral artery injury. Without surgical intervention prognosis of these patients remain poor. Prognosis of patients with traumatic cerebellar infarction depends on the neurological status of the patient, intrinsic parenchymal damage and more importantly extrinsic compression of the brainstem by the edematous cerebellar hemispheres.
Key words: Cerebellar infarction, traumatic brain injury, cervical spine injury, vertebral artery injury

Introduction

Posttraumatic cerebral infarction is an uncommon cause of morbidity and mortality in patients with traumatic brain injury. (1-5) Many studies have highlighted that trauma needs to considered as causative factor for cerebellar infarction particularly in young patients. (2, 4-7) We present a case of cerebellar infarction in a young patient secondary to vertebral fracture involving the vertebral foramen and vertebral artery injury.

Case report

A 35 year old gentleman met a road traffic accident while he was trying to overtake another vehicle and driver lost the control and

collided with the vehicle. Drive died on the spot. Details of pre-hospital care were not available. He was brought to the emergency department 25 hours after the accident. The patient was put on cervical collar. His GCS was E1VTM4. Pupils were bilateral 3 mm and non-reactive to light. CT scan brain showed thin left fronto-temporo-parietal acute subdural hematoma with minimal mass effect and midline shift. CT scan also showed left cerebellar infarction. With mass effect and diffuse deep cerebral edema. In addition CT scan cervical spine showed C2-3 fracture on left side with fracture extending into the left vertebral foramen. A CT scan angiogram could not be performed because of poor neurological status. Possibly the infarction was due to left vertebral artery injury. In view of poor neurological status the patient relatives opted for conservative management. Poor prognosis was explained and in spite of all measures the patient could not be revived.

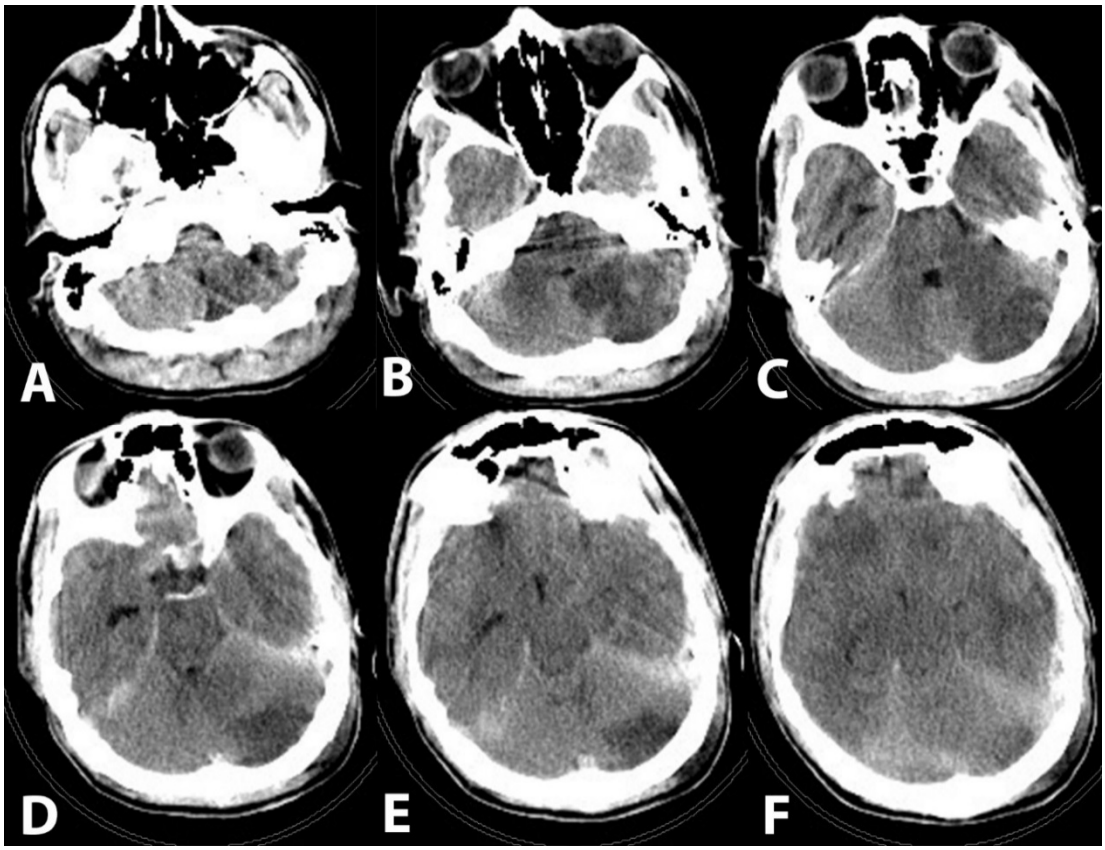

Figure 1

(A) CT scan showing right cerebellar infarction, (B) follow-up CT scan showing infarction, and (C) postoperative scan showing the opened up ventricle

CT scanning reveals large bilateral cerebellar and occipital infarct in the territory of PCAs, SCAs and AICAs with acute hydrocephalus

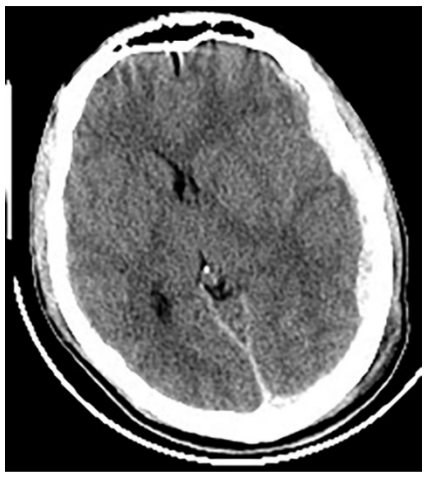

Figure 2

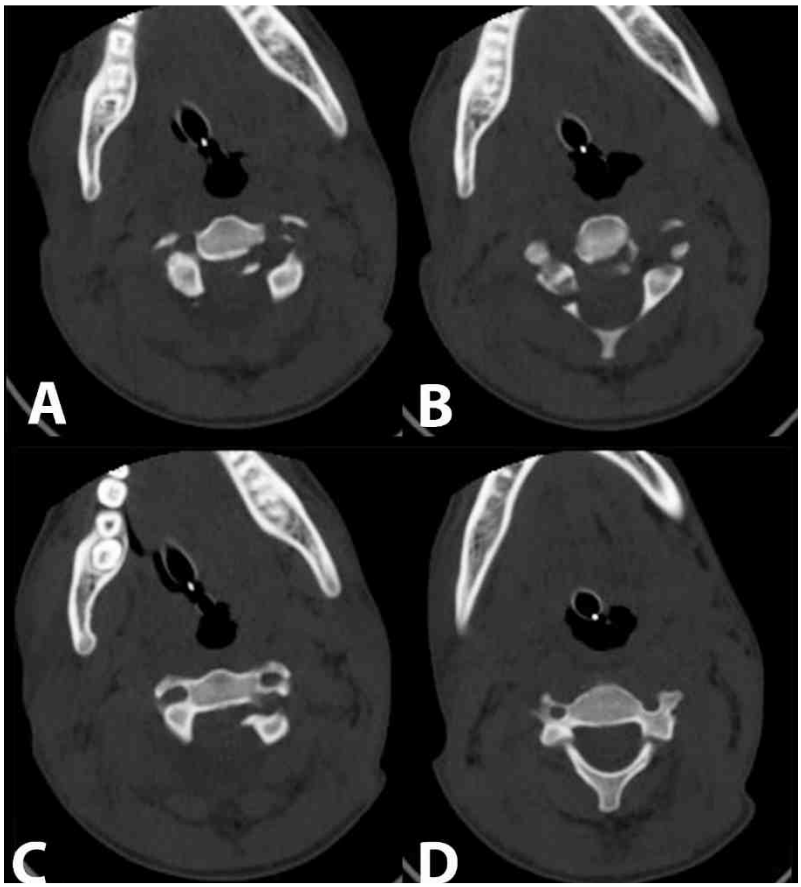

Figure 3

Discussion

A number of mechanisms have been described to explain the cerebellar infarction following head injury. These include dissections (with progressive thrombosis and vascular occlusion) or vertebrobasilar spasm, embolization, and systemic hypoperfusion compromising the vascular supply to the cerebellum, (2, 5, 8-10) local trauma severe enough to deform the overlying occipital bone and causing injury to the cerebellar cortical artery thus leading to the cerebellar infarction. (2, 4) Once the infarcts sets in than it leads to cerebellar edema and compression of the fourth ventricle and brain stem responsible for neurological deterioration and if not intervened early than this can be fatal. (11) Clinical features of the cerebellar infarction are similar to the intrinsic cerebellar lesions and depend on the size of the lesions, any associated compression of the fourth ventricle and brain stem and extent of other associated intracranial lesions. (12-16) In early stages there may be headache, dizziness, nausea, vomiting, loss of balance, signs of truncal and appendicular ataxia, nystagmus, and dysarthria. (12-14) However, if the lesion is large enough there may altered level of consciousness, ataxic respirations, extensor plantar responses, posturing, or flaccidity, impaired oculocephalic responses, decreased or absent corneal responses, and impaired or absent pupillary responses. (12, 14-17) In majority of the cases of traumatic brain injury CT scan brain with bone window is the investigations of choice and can show cerebellar infarction as a focal hypodense area (with or without evidence of fourth ventricular compression) (18); however we need to remember that in early stages ischemic changes and presence of cerebellar infarction can be missed. (1, 5) Where there is high index of suspicion an MRI of the brain shall provide greater details of cerebellar infarction, details of brain stem compression and presence of any associated hydrocephalus. (16, 18, 19) Conventional digital subtraction angiography is the gold standard to diagnose injury to the neck vessels but may not be feasible in emergency situation. (1) Same holds true for magnetic resonance imaging and magnetic resonance angiography, it can demonstrate the vascular pathology but will be difficult to perform in emergency situation like head injury. (10) To detect the injury to the neck vessels Doppler can be used as a screening investigations, however it will be difficult to interpret the vertebra-basilar system. (20) In a patient with head injury now a day's computed tomography angiography (CTA) is recommended a noninvasive, highly specific, and sensitive imaging modality to rule out vascular injuries. (21)

The management of post-traumatic cerebellar infarction is controversial and it is directed to reduce the intracranial pressure i.e. diversion of CSF (external ventricular drain) to control hydrocephalus and/or decompression of the posterior fossa to reduce the mass effect on brain stem. (1, 5, 13, 14, 17, 22) Many authors advocate that surgical decompression should be performed first to reduce the mass effect and if the clinical features continue to persist or there is deterioration in neurological status a CSF

diversion procedure can be performed. (5, 11, 23-27) Management of the hydrocephalus with external ventricular drainage alone without posterior fossa decompression will not help to reduce the mass effect from the brain stem and shall be carrying the inherent risk of upward herniation. (2) There is a need to emphasize here that medical management (steroids, mannitol and hyperventilation) to reduce the intracranial pressure are usually ineffective in these cases. (22, 28)

Conclusion

Prognosis of patients with traumatic cerebellar infarction depends on the neurological status of the patient, intrinsic parenchymal damage and more importantly extrinsic compression of the brainstem by the edematous cerebellar hemispheres. (13, 22, 27) For traumatic cerebellar infarction, surgical intervention is the mainstay of treatment. (13, 14, 18, 29) Without surgical intervention prognosis of these patients remain poor. (1, 13)

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