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Title page

Isolated basilar artery dissection following blunt trauma challenging the Glasgow coma score: a case report

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Abstract

Blunt cerebrovascular injury is a very rare complication of blunt trauma and a diagnostic challenge. A 14 year old male fell 10 meters sustaining multi system trauma. The atypical Glasgow Coma Score was six with a fully preserved eye component. Initial whole-body CT scanning demonstrated multiple injuries but no obvious brain injury. Trauma management involved non-operative resuscitation and was successful, however profound coma occurred and brain stem reflexes disappeared on day two. Repeat brain CT scan demonstrated multiple cerebral and cerebellar ischemic lesions and no opacification of the vertebral or basilar arteries. Secondary analysis of the first CT scan demonstrated a small focal basilar artery dissection not initially reported.

Our case report highlights an unusual cause of coma after traumatic brain injury where the clinical scenario mimics locked in syndrome. In such circumstances cerebrovascular injury, and in particular traumatic basilar artery dissection, must be actively excluded

Introduction

Cerebrovascular injuries and basilar artery dissections in particular, are rare complications of blunt trauma [1]. As such, they represent a diagnostic challenge for the clinician. Here we report the case of a young man that was transferred to our trauma-center following a fall from a height of 10 meters. An undiagnosed isolated basilar artery dissection at presentation evolved into complete basilar and vertebral occlusion and brain death.

Case

A 14-year-old patient fell from a height of ten meters and was admitted to our traumacenter. **The cause of the fall was clearly identified as a jump in order to escape a police control and witnesses present on scene did not report any preceding signs or symptoms.** Prehospital assessment revealed a Glasgow Coma Scale (GCS) of six on-scene with spontaneous eye opening, graded as eye response of four, no verbal or motor response and a conserved pupillary response to light. Rapid sequence induction, intubation and ongoing sedation were initiated on site. The patient was transferred to our Trauma-Center where initial in hospital management included hemodynamic stabilization and chest tube insertion for massive pneumothorax diagnosed on ultrasound examination. A whole-body computed tomography scan (CT scan) demonstrated multiple injuries including hemopneumothorax, pulmonary contusions, diaphragm rupture, pneumoperitoneum, spleen and kidney lacerations and thoracic spine fractures. There was no obvious structural brain injury. The pneumoperitoneum led to a trauma laparotomy that failed to demonstrate any intra-abdominal lesion. Intracranial pressure was not monitored considering the lack of intracranial injury in the initial scan and a normal transcranial Doppler exam (Pulsatility index (IP) (right IP: 0,9, left IP:1,0). Sedation was lightened and the spine surgery scheduled for the next day.

On day two, despite sedation have been stopped completely, the patient did not regain consciousness. Clinical evaluation demonstrated the absence of pupillary reaction to light,

spontaneous ventilation, corneal, oculocephalic, and gag reflexes. At day two, brain CT scan revealed the appearance of multiple cerebral and cerebellar ischemic lesions (Fig.1) and no opacification of vertebral or basilar arteries. Secondary analysis of the day one CT scan showed the presence of a small focal basilar artery dissection that had been previously overlooked (Fig.2). There was no evidence of vertebral artery dissection, no cranial base fracture and no craniocervical dislocation. Basilar artery recanalization was not attempted given the severity of clinical and radiological signs and brain death was confirmed by electroencephalography on day three.

Discussion

Several factors explain this delayed diagnosis of traumatic basilar artery dissection. Firstly, blunt cerebrovascular trauma is a very rare complication of blunt trauma with a reported incidence of 1% to 9.2% [1-3]. The most frequently reported lesion is vertebral artery dissection, and this lesion is commonly associated with cervical spine fractures [1,3]. Traumatic basilar artery dissections are comparatively rare. Secondly, traumatic basilar artery dissection is often an extension of vertebral artery dissection or due to basilar artery entrapment with a longitudinal clivus fracture [4]. Neither vertebral artery dissection nor clivus fracture were a feature in this case. Thirdly, the suspected underlying mechanism is hyperextension or hyperflexion of the basilar artery induced by the movement of the brain stem during severe traumatic impact. Trauma with sufficient kinetic energy to produce basilar artery dissection will frequently be associated with multiple organ injuries, which make the diagnosis of a small focal basilar artery dissection more challenging on the whole-body CT scan.

Fourthly, as is often the case when trauma patients present with multiple injuries, initial resuscitation and/or emergency surgery impeded thorough clinical assessments. Indeed, an

initial GCS graded as 6 associated with normal pupil size and eye response were exclusively interpreted as a consequence of the traumatic brain injury. In the context of TBI, a low motor component is often interpreted by clinicians as a threatening sign, whereas in fact a GCS of six with spontaneous eye opening but no verbal or motor response is very unusual in trauma patients [5]. This discrepancy should have been considered as a quadriplegia mimicking a locked in syndrome and should have raised suspicion earlier regarding the possibility of basilar artery dissection. Further investigations for cerebrovascular lesions should ideally have been performed on day one. **An early identification of basilar dissection could have led to an attempt of endovascular repair (basilar artery stenting) and possibly to a better outcome.**

Conclusion

This case highlights the diagnostic challenge of basilar artery dissection in the context of traumatic brain injury. Practitioners should pay particular attention to initial Glasgow Coma Scale and to each specific sub-component. The diagnosis of basilar artery abnormality should be considered when the presentation includes an unusually low GCS with preserved eye opening.

Author contributions

MJD and DBM authors wrote the manuscript and created the figure. All authors critically reviewed and agreed to the submission of the final manuscript.

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Figure 1: Control (day two) unenhanced axial CT images show large bilateral hypoattenuating areas (asterisks) consistent with cerebral (A) and cerebellar (B) infarctions.

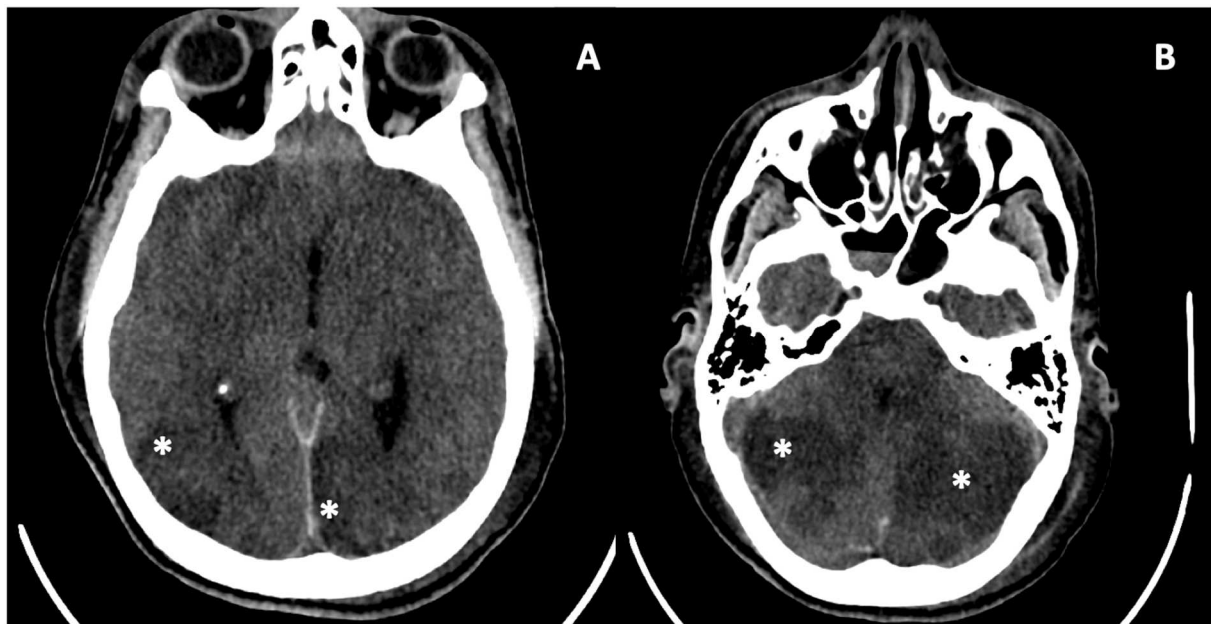


Figure 2: A: Initial unenhanced sagittal CT image shows spontaneous focal hyper-attenuation of basilar artery (arrow) consistent with acute occlusion. B: contrast enhanced (biphasic injection protocol) coronal CT-MIP reconstruction (6mm) confirms the focal occlusion of the distal portion of the basilar artery (arrow).

