

The enigma of traumatic, behaviourally benign brain stem bleeds : Case report

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INTRODUCTION

With easy availability and the increasing sophistication of imaging, traumatic brain stem bleeds are being increasingly being detected. There is a clinical spectrum of severity of brain stem bleeds that varies from the relatively innocuous to the moribund. The presentation profile depends more on the etiopathogenesis of the bleed than on its quantum and location.

At the higher end of the severity spectrum are Duret haemorrhages¹. Duret induced the brain stem bleeds that bear his name in experimental rats by increasing the intracranial pressure. The aetiology of Duret bleeds has been the subject of debate². Brain stem descend in herniation syndromes resulting in the avulsion of microperforators from a relatively fixed basilar artery is the most likely cause of these bleeds. Venous thrombosis and microhaemorrhages is another postulate.

The spectrum of diffuse axonal injury has been radiologically graded. Microhaemorrhages at the grey white junction induced by differential movement due to the different densities of grey and white matter, corpus callosal bleeds and brain stem bleeds at junctions of relatively mobile and anchored parts of the brain are considered entities of more aptly the markers of increasing inertial stress and diffuse axonal rupture. These patients too have a prolonged obtunded course and guarded neurological outcomes.

The entity of behaviourally benign brain stem bleeds³ possibly indicates a third mechanism of a 'coup' contusion haemorrhage that occurs when the upper brain stem strikes against the clivus. We report the case of a young man with a benign brain stem bleed after a motorcycle accident.

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CASE REPORT

A 21-year-old-male was driving a motorcycle wearing a cloth cap at around 30 Km/h, when he skidded and fell. He had transient loss of consciousness, but remembers being taken to the hospital, seated between two friends on a motorcycle. He was sent home after first aid (immobilisation of an undisplaced wrist fracture with a POP slab). Two days later, he reported to a tertiary care hospital with numbness and weakness of the left side of his body and drooling of saliva from the left corner of his mouth.

Clinically, he was found to have a left sided facial palsy along with left sided upper and lower extremity weakness (MRC grade 4). The left sided plantar was



Fig 1: Photographs taken seven days post trauma demonstrating a mild lower motor neuron facial' in the recovery phase

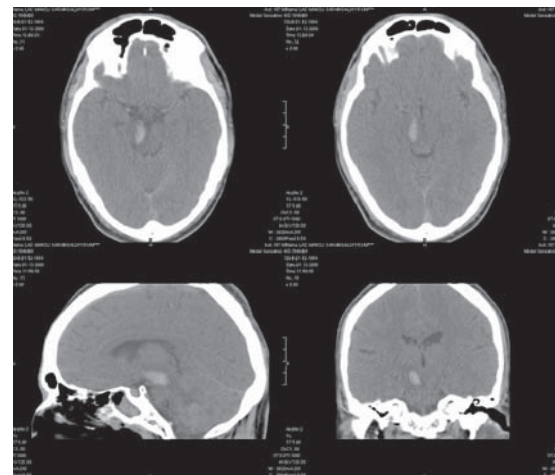


Fig 2 : CT images showing the location of bleed

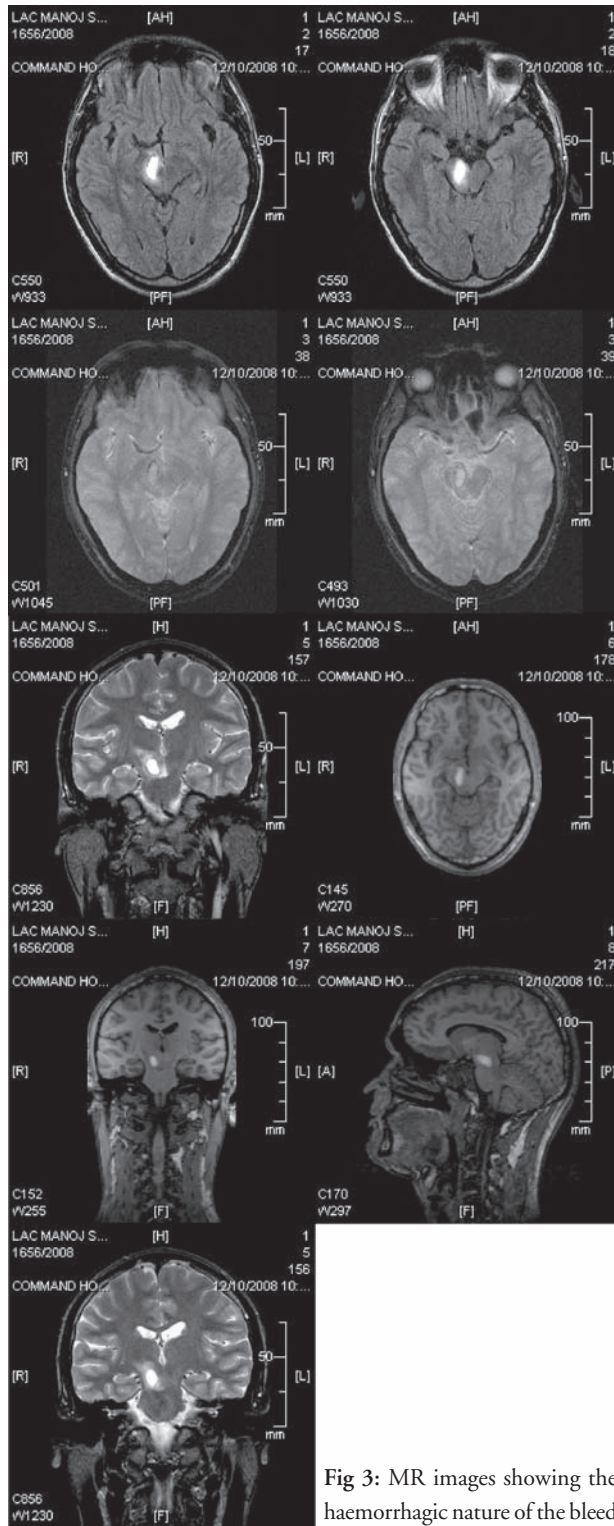


Fig 3: MR images showing the haemorrhagic nature of the bleed

extensor. He was evaluated with a CT scan of his head and later MRI was carried out. The images showed a right-sided midbrain hematoma. Patient's clinical deficits progressively improved over the next few weeks. Facial

palsy recovered completely, with the hemiparesis and extensor plantar too recovering within six weeks of injury.

DISCUSSION

Traumatic brain stem bleeds are often assumed to indicate a severe traumatic brain insult, often with a guarded outcome. Brain stem bleeds however vary in clinical spectrum from the benign (as in the above reported case), to the grievous⁴. The biomechanics of the injury and the aetiology of the haemorrhage are the decisive factors in prognostication⁵. The worst outcomes occur with the classical Duret haemorrhages which are a consequence of microperforator ruptures due to herniation syndromes consequent to intracranial supratentorial hypertension due commonly to hematomas or cerebral edema⁶. These hematomas are often post-mortem findings.

Brain stem hemorrhages as part of the diffuse axonal injury complex occur in high-speed motor vehicular accidents and are also harbingers of poor outcome. Isolated brain stem bleeds in traumatic brain injury however may occur in the scenario of less severe impact as in the above reported case. Outcomes in these 'impact' bleeds are often excellent. The entity of benign brainstem bleeds has been described by Bhatoe, who reported a series of nine cases with favourable outcomes⁷. General awareness of this entity is important in prognostication. The grave prognosis associated with Duret's haemorrhages has resulted in a nihilistic approach to the treatment of brain stem bleeds. All traumatic brain stem hemorrhages however do not share the same sordid outcome⁸. The case presented above is an example of a case with a benign brain stem bleed.

The pathogenesis of the brain stem bleed is possibly the most important factor in prognostication. The classical Duret's haemorrhage is considered to occur when mid brain descent (herniation), stretches the perforators arising from the basilar artery resulting in their rupture⁹.

CONCLUSION

Primary brain stem bleeds also occur as apart of the diffuse axonal injury complex when the inertial stress of acceleration/deceleration compounded with impact result in multiple axonal ruptures and disruption of the microvasculature resulting in the micro-bleeds which might expand. The diffuse axonal injury complex is often associated with prolonged coma and a delayed and incomplete recovery. Brain stem bleeds in diffuse axonal

injury complex have been graded as Grade iii (most severe). Yet the morbidity and outcome of DAI depends on the quantum of axonal ruptures. Microvascular ruptures can occur in relative isolation with inertial stress. Isolated traumatic brain stem bleeds and indeed limited inertial intra cerebral bleeds may be behaviourally benign.

It is proposed that brain stem bleeds in traumatic brain injury be grouped into:

- a. Primary brain stem bleeds resulting from a combination of inertial injury with impact injury to the midbrain at the apex of clivus. This constitutes the entity of behaviourally benign brain stem bleeds.
- b. High-speed inertial stresses with diffuse axonal ruptures and microbleeds associated with astroglial 'stars' at the grey white junction, corpus callosum and brain stem.
- c. Duret bleeds resulting from downward mid brain herniation and perforator rupture.

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