Recovery From Duret Hemorrhage: A Rare Complication After Craniotomy

—Case Report—

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Abstract

A 44-year-old female presented with Duret hemorrhage due to transtentorial herniation by extradural hematoma as a complication after craniotomy for treatment of spontaneous middle cranial fossa cerebrospinal fluid leakage through the oval window. Brain computed tomography revealed linear hemorrhage in the midbrain and the rostral pons. She awoke after 2 weeks in a coma, despite showing ocular bobbing and bilateral intranuclear ophthalmoplegia. She was discharged from the hospital with minimal neurological defects. Duret hemorrhage is usually fatal, but this case shows that early surgical decompression is the most important factor to avoid the worst sequelae.

Key words: brain stem injury, craniotomy, computed tomography, Duret hemorrhage

Introduction

Extradural hematoma after craniotomy may occur as a postoperative complication, but rarely results in brain stem hemorrhage.⁵⁾ Secondary brain stem hemorrhage, also known as Duret hemorrhage, is one sign of transtentorial herniation and is thought to imply a poor prognosis.⁴⁾ We describe a patient who survived Duret hemorrhage after craniotomy associated with severe brain-stem dysfunction.

Case Report

A 44-year-old female was hospitalized in February 1997, with a 1-year history of intermittent cerebrospinal fluid (CSF) spontaneous rhinorrhea after a minor head injury. Physical examination on admission revealed left CSF rhinorrhea. Neurological examination found no abnormalities. Coronal computed tomography (CT) demonstrated an osseous defect in the left middle cranial fossa (Fig. 1). She underwent external lumbar drainage, but the fistula persisted after 6 days.

She was treated via the epidural approach through a left frontotemporal craniotomy. Encephalic tissue was present inside the osseous defect between the

Fig. 1 left: Computed tomography (CT) scan showing the ostium, with a liquid level, in the left middle cranial fossa. right: CT scan (bone window) clearly showing the ostium in the left middle cranial fossa extending into the sphenoid sinus.

foramen ovale and the foramen spinosum, which was resected to treat the encephalocele. The hole was closed with a galeal pericranial flap using biologic bond and the dural laceration in the same region was sutured. She was then transferred to the neurological intensive care unit (ICU).

She was awake and alert on the next day in the

Fig. 2 Computed tomography scan showing a left frontotemporal extradural and subgaleal hematoma, with obliteration of the suprasellar and other cisterns.

morning with normal neurological findings except for slight numbness in distribution of the left maxillary nerve. However, as soon as the subgaleal drainage tube was removed, arterial hemorrhage began from its orifice, which was sutured. After 30 minutes she began to develop headache and vomiting, followed by decreased level of consciousness. Her left pupil was larger with absent light reflex. She was intubated and CT showed a left large subgaleal hematoma and an extradural hematoma, accompanied by remarkable midline shift (Fig. 2). The hematoma was evacuated about 30 minutes after the diagnosis, and a fiberoptic intracranial pressure (ICP) monitor (Camino Laboratories, San Diego, Calif., U.S.A.) was placed in the subdural space. The epidural drainage was left in place to maintain the operative region without CSF pressure and help cure the fistula.

Postoperatively, she had a normal ICP value. We believe that the probable causes of the hematoma formation were a fast fall in CSF pressure and detachment of the dura mater in the operated area. The patient presented with deterioration of consciousness and she became comatose with anisocoria. Although she demonstrated ocular bobbing and bilateral internuclear ophthalmoplegia, which lasted for about 3 days, her consciousness recovered gradually. Repeat CT revealed a linear hemorrhagic lesion in the midbrain and the upper pons (Fig. 3). She was discharged from the hospital

Fig. 3 Computed tomography scan 24 hours after evacuation of the hematoma showing a linear high density area in the midbrain, suggesting hemorrhagic brain stem injury. The suprasellar cistern has reappeared.

with a slightly confused mental state.

Discussion

The diagnosis of traumatic brain stem injury (BSI) was based on the clinical presentation of a patient or postmortem autopsy before the introduction of CT. The frequency of BSI is high in autopsy cases (49.5%) or when histological techniques are used (88–100%), but is low in most large CT studies at less than 5%.^{6,7)} The large discrepancy between CT and autopsy series can be explained by the greater severity of injury in the autopsy group, and by the high false-negative rate of CT. CT has significantly improved the diagnosis of acute head injury, including BSI, but lesions in the brain stem are not always easy to detect. Magnetic resonance (MR) imaging is particularly useful to visualize slight lesions, especially in the posterior cranial fossa. CT detected 30.8% of acute BSI lesions, whereas T₁- and T₂-weighted MR imaging visualized 100% of these lesions.⁶⁾ The frequency of BSI was 41.4% in nonfatal cases, much higher than previously reported. However, the use of MR imaging is limited in the acute stage of head injury.

Traumatic BSI can be classified into primary and secondary.^{4,6,9)} The most common type of primary BSI is diffuse axonal injury. Secondary BSI may be hemorrhagic or ischemic, and focal or systemic. Hemorrhagic BSI may result from compression of the brain stem by transtentorial herniation due to

increased ICP. This may produce caudal displacement of the upper brain stem and distortion of the direct perforating branches of the posterior cerebral arteries in the interpeduncular cistern and the paramedian branches of the basilar artery, resulting in a hemorrhagic lesion,⁶⁾ which is called a Duret hemorrhage. Duret²⁾ originally described multiple small hemorrhages mainly surrounding the floor of the fourth ventricle, but the eponym Duret hemorrhage is generally used for centrally located midline hemorrhagic lesions in the tegmentum of the rostral pons and midbrain. The frequency of Duret hemorrhage is 5.6% in BSI cases.⁶⁾ The majority of lesions of primary BSI are located in the dorsal and dorsolateral aspects of the brain stem, whereas the lesions of secondary BSI usually occur in the midline, paramedian, and ventral aspects in the tegmentum of the midbrain and rostral pons.^{1,6,9)}

Extradural hematoma is a common postoperative complication, but large hematoma that requires evacuation is rare. A large series of 1055 intracranial operations included 168 extradural hematomas (15.9%), but only four (0.38%) localized under the operative sites required operation.

BSI due to Duret hemorrhage is considered to be fatal and is rarely detected by CT or even MR imaging. Our patient survived an incident Duret hemorrhage, despite presenting with ocular bobbing, which is also a sign of poor prognosis.^{3,8)} BSI in our patient was due to a large extradural hematoma as a rare complication after craniotomy. Our patient survived with minimal sequelae because of strict observation of the neurological symptoms and signs during the postoperative course in the neurological ICU, resulting in early diagnosis and removal of the hematoma.

We conclude that ICP should be managed postoperatively in patients with CSF leakages. Rigorous closed neurological observation is required with the patient in the ICU for at least 7 days, and a subdural or extradural transducer should be

inserted to measure the ICP. Any changes in ICP resulting in minimal or apparently minimal neurological focal signs (better observed in the ICU) should be followed by immediate CT for early identification of any hematoma formation. Early treatment will decrease the chance of a bad outcome due to brain stem lesion.

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