Brainstem hemorrhage can occur as a primary or secondary event in traumatic brain injury (TBI). Secondary brainstem hemorrhage that evolves from raised intracranial pressure and transtentorial herniation is referred to as Duret hemorrhage. Duret hemorrhage following TBI has been considered an irreversible and terminal event. The authors report on the case of a young adult patient with TBI who presented with a low Glasgow Coma Scale score and advanced signs of cerebral herniation. She underwent an urgent decompressive hemicraniectomy for evacuation of an acute epidural hematoma and developed a Duret hemorrhage postoperatively. In accordance with the family’s wishes, aggressive TBI monitoring and treatment in the intensive care unit was continued even though the anticipated outcome was poor. After a lengthy hospital course, the patient improved dramatically and was discharged ambulatory, with good cognitive functioning and a Glasgow Outcome Scale score of 4. Duret hemorrhage secondary to raised intracranial pressure is not always a terminal event, and by itself should not trigger a decision to withdraw care. Aggressive intracranial monitoring and treatment of a Duret hemorrhage arising secondary to cerebral herniation may enable a good recovery in selected patients after severe TBI. (DOI: 10.3171/2008.8.JNS08314)

**Key Words**
- intracranial hypertension
- outcome
- traumatic brain injury
- traumatic brainstem hemorrhage

**Case Report**

**History and Presentation.** This 24-year-old woman was a restrained passenger in a high-speed, rollover motor vehicle accident. Her field GCS score was 3 and she was taken to the hospital after a 10-minute extrication. On arrival at the emergency department, she had a GCS score of 6 (E1, V1, M4), with fixed 6-mm dilated pupils bilaterally, agonal respirations, absent cough and gag reflexes, and a systolic blood pressure of 80 mm Hg. She withdrew all 4 extremities to pain. A hemotympanum was present bilaterally, and fluid drainage, possibly cerebrospinal fluid, was observed from the left external auditory canal. She underwent intubation and fluid resuscitation in the emergency department. A CT scan of her head obtained within 20 minutes of her arrival at the hospital demonstrated a right frontotemporal EDH with mixed density suggestive of hyperacute bleeding (Fig. 1A and B). The EDH was 2.0 cm in maximum diameter and was associated with a mass effect with 1.5 cm of right-to-left midline shift, complete obliteration of the quadrigeminal and ambient cisterns, and tonsillar tissue effacing the cisterna magna. A bone window scan showed multiple fractures of the right temporal bone and skull base.

**Operation.** The patient was taken immediately to the operating room for a right hemicraniectomy and evacua-
Preoperative

Postoperative

Fig. 1. Unenhanced CT images. A: Preoperative scan obtained at admission showing a heterogeneous right frontotemporal EDH (arrows) with mass effect and a right-to-left midline shift. Mixed density signal within the EDH suggests ongoing hyperacute bleeding. B: Image of the rostral brainstem showing complete obliteration of the basal cisterns. C and D: Images obtained 24 hours after decompressive hemicraniectomy revealing good decompression and resolution of the midline shift. There is interval evolution of a small contralateral, left frontal epidural/subdural collection. Image (D) corresponding to the level shown in B revealing a new hemorrhage in the pons (arrow).

Good outcome after Duret hemorrhage

Postoperative Course. An unenhanced CT scan obtained 24 hours after decompressive surgery confirmed good evacuation of the EDH (Fig. 1C). The scan also demonstrated a 1.7 × 1–cm area of increased density in the brainstem, consistent with blood in the pons (Fig. 1D). Computed tomography angiography showed no evidence of communication between the pontine hemorrhage and the basilar artery. An MR image obtained on postoperative Day 3 confirmed the existence of a pontine hemorrhage (Fig. 2) together with preoptic cistern compression and anteroposterior elongation of the midbrain. A diffuse axonal shear injury was evident in the right temporal lobe stem and a small focus was present in the left dorsolateral midbrain extending to the pontomesencephalic junction. There was a paucity of subcortical or corpus callosal shear injury. Ischemic lesions within the left cerebral peduncle and right occipital lobe demarcated severe uncal herniation. Cerebellar ischemia in the territory of the left posterior inferior cerebellar artery was consistent with acute tonsillar herniation.

The patient's neurological examination worsened. The anticipated dismal prognosis was discussed with the family, and the organ donor network was made aware of the patient. As a result of the decompressive hemicraniectomy, however, she did not progress to brain death, and beginning on hospital Day 6, her neurological examination slowly began to improve. At 2 weeks she was localizing on the right side and underwent tracheostomy and percutaneous gastrostomy. Her hospital course included treatment for meningitis, gram-negative sepsis, and operative repair of a left humeral fracture. At 1 month postinjury, she was tracking and obeying simple commands and was transferred out of the intensive care unit. At 3 months, she was verbal.
Duret hemorrhage in the setting of cerebral herniation after a severe TBI has been considered a terminal brainstem event with a high incidence of death or persistent vegetative outcome. Secondary brainstem hemorrhages are found in only a small percentage of patients who die of TBIs, and in radiological series, Duret hemorrhages have been estimated to represent only 5.6% of all brainstem injuries. Prospective studies of mortality rate and outcome in patients with radiologically proven Duret hemorrhage are lacking. Despite this, the finding of a Duret hemorrhage on neuroimaging studies has been considered such an ominous finding that it has often provided the rationale to stop or withdraw care. As a consequence, this injury has become associated with a high mortality rate, which de facto has endorsed withdrawal of care as the accepted management practice for these injuries. Overall, early withdrawal of care circumvents a true estimate of mortality rate and outcome for Duret lesions. As our experience in the present study demonstrates, aggressive surgical treatment and continued intensive neurocritical care can enable a good outcome in some patients with Duret hemorrhage even when the early neurological examination would suggest that the situation is hopeless. Duret hemorrhages that evolve secondary to transtentorial herniation are distinct in their distribution and pathophysiology from primary brainstem hemorrhages that occur at the time of impact. Primary brainstem injuries have a predilection to involve the dorsolateral aspects of the midbrain, and the majority of these injuries are associated with diffuse axonal shear injuries. In contrast, secondary Duret brainstem hemorrhages typically occur at the midline of the rostral pons and the ventral tegmentum of the midbrain. These lesions are often accompanied by the pathological hallmarks of raised ICP and cerebral herniation, including lesions in the contralateral cerebral peduncle and occipital lobe infarction in the distribution of the posterior cerebral artery. Duret hemorrhage secondary to downward transtentorial herniation may occur within 30 minutes of injury; therefore, differentiating primary from secondary brainstem hemorrhage can be difficult. The absence of brainstem injury on the initial CT scan in our patient indicated that the pontine hemorrhage was not a primary injury. On MR images, the classic median location within the rostral pons, the minimal signal abnormality in the dorsolateral midbrain, and the paucity of additional diffuse axonal shear injury, together with associated findings of signal changes in the left cerebral peduncle, occipital lobe, and cerebellum indicative of uncal and tonsillar herniation, provided evidence that the brainstem hemorrhage in our patient was a secondary Duret hemorrhage.

The sudden downward movement of the brainstem against a relatively fixed and immobile basilar artery is a key step in the pathogenesis of a Duret hemorrhage. This assertion is supported by studies of raised ICP induced by inflation of intracranial balloons in canine models. As the brainstem moves downward, stretching and disruption of paramedian pontine perforating arteries may directly lead to hemorrhaging. During herniation, the upper brainstem buckles and elongates in the anteroposterior direction, stretching midline perforating vessels further. Veins may be compressed more easily than arteries, and hemorrhagic transformation of an area of venous ischemia may also contribute to Duret hemorrhage. In addition, surgical decompression may promote Duret hemorrhage as part of a reperfusion injury. The CT and MR imaging studies obtained in our patient revealed anteroposterior elongation of the midbrain, compression of the pons, obliteration of the prepontine cisterns, and tonsillar displacement into the foramen magnum that together were compatible with downward displacement and distortion of the brainstem leading to the Duret hemorrhage. The good outcome in this case is at variance with a mechanism involving stretching and mechanical disruption of the pontine perforating vessels and is more compatible with one of venous or arterial ischemia and reperfusion injury after surgical decompression.
The novel point of this case is not that the patient survived and made a good recovery from her initial GCS score of 3, but rather that this recovery occurred after traumatic cerebral herniation so advanced that it precipitated a Duret hemorrhage. A few exceptional cases of good outcomes after traumatic Duret hemorrhage have been reported, but these accounts predate the advent of CT neuroimaging. In the nontraumatic setting, good outcome has been reported in 2 cases of Duret hemorrhage precipitated by the insertion of a lumbar drain and after rapid correction of severe hyponatremia. Our patient and her serial neuroimaging studies confirm a secondary Duret brainstem hemorrhage and uniquely document a case of good recovery following cerebral herniation with Duret hemorrhage following severe TBI.

The good outcome in our patient may be attributable to a number of factors. Given the short time from admission CT scan to surgical evacuation, it is possible that the resulting Duret hemorrhage arose from less forceful pressure changes and downward herniation ongoing after the decompression. The relative absence of a primary brainstem injury may also have been a survival advantage, as the mortality rate for patients with transtentorial herniation is 2–3 times higher when accompanied by primary brainstem injury. Moreover, although large, the hemorrhage in our patient was 1.7 cm in diameter. Centered in the ventral pons, it may not have reached the threshold size at which it would damage the fibers of the pontine and medullary reticular formations situated in the dorsal pontine tegmentum. Indeed, spontaneous pontine hemorrhage unrelated to cerebral herniation has a heterogeneous outcome profile, and is adversely related to the paramedian location and hematoma diameter > 2 cm.

This case illustrates that a good outcome can be achieved following aggressive TBI care even in patients with low admission GCS scores, ongoing deep coma, and neuroimaging findings consistent with a Duret hemorrhage. It is difficult to predict what percentage of patients with Duret hemorrhages might survive with the more widespread institution of continued, aggressive TBI care in these difficult cases. Careful patient selection is critical to guard against persistent vegetative outcomes. Biomarkers, prognostic metabolic signatures on neuroimaging, and sensitive and specific neurophysiological tests are needed to identify comatose patients who are likely to benefit from prolonged, aggressive treatment for TBI.

Disclaimer

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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