Pediatric brainstem hemorrhages after traumatic brain injury

Report of 2 cases

ALEXANDRA D. BEIER, D.O.,1 AND PETER B. DIRKS, M.D., PH.D.2

1Division of Pediatric Neurological Surgery, University of Florida Health Jacksonville, Florida; and
2Division of Neurosurgery, Department of Surgery, Hospital for Sick Children, University of Toronto, Ontario, Canada

Traumatic brain injuries afflict a large number of pediatric patients. The most severe injuries lead to increased intracranial pressure and herniation, with resultant changes in the brainstem. Traumatic brainstem hemorrhages have previously been associated with poor neurological outcome and fatality. However, this report discusses 2 pediatric patients who sustained severe head trauma with subsequent brainstem hemorrhages, and yet experienced good neurological outcome; the possible mechanism is described.

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KEY WORDS • Duret hemorrhage • brainstem hemorrhage • decompressive craniectomy • traumatic brain injury • trauma

Case Reports

Case 1

History and Examination. A 5-year-old male pedestrian was hit by a motor vehicle. The emergency medical services (EMS) crew arrived, stabilized the patient, and documented a Glasgow Coma Scale (GCS) score of 12. During transfer, the patient waxed and waned in mental status. On arrival to the trauma center, his GCS score declined to 8 and subsequently to 3. His pupils remained reactive. He was resuscitated, and paralytics and sedatives were given for intubation. The patient became bradycardic and was given atropine. A CT scan revealed a 4-mm left subdural hematoma (SDH) with areas of hyperacute blood, causing significant mass effect and 7 mm of midline shift. The basal cisterns were obliterated, and there were bilateral temporal bone fractures, extending across the skull base (Fig. 1A and B). Mannitol was given and the patient was taken to the operating room on an emergency basis for subdural evacuation and decompressive craniectomy. The time that elapsed between the incident and initiation of the operative procedure was 2.5 hours.

Operation and Postoperative Course. Intraoperative-
ly it was noted that his left temporal bone fracture had lacerated a distal M_1 branch, which was controlled. His cranial flap was left off, a subdural ICP monitor was inserted, and the patient was intubated and taken to the pediatric intensive care unit. Over the next few hours, he started following commands. Nine hours postoperatively he was following commands briskly in all 4 extremities. Postoperative CT scans showed a left craniectomy with evacuation of SDH, with decreased cerebral edema and decreased effacement of the basal cisterns. There was also increased density in the ventral pons and midbrain pontine junction consistent with hemorrhage (Fig. 1C). The patient was extubated on postoperative Day 2 and suffered a left sixth cranial nerve palsy, presumably from increased ICP, and a partial seventh cranial nerve palsy due to his skull base fracture. He never experienced elevated ICP postoperatively, and the monitor was discontinued on postoperative Day 2. An MR image revealed further evidence of his transtentorial herniation, with pontine ischemic changes (Fig. 1D).

The patient continued to recover well, and was discharged to rehabilitation 11 days after his injury. He was alert, awake, with language intact, without focal motor deficits, but with residual left sixth and seventh cranial nerve palsies. His cranial flap was replaced 6 weeks later. He was clinically evaluated 2 weeks after the cranioplasty, and he was found to be without any deficit besides the resolving cranial nerve palsies (Glasgow Outcome Scale [GOS] 5).

Case 2

History and Examination. A 14-year-old boy fell down 20 stairs and had immediate loss of consciousness. The EMS workers stabilized and intubated the patient. It was documented that the patient was decerebrate posturing and that he had a right dilated pupil. He was taken to an outside hospital, where a CT scan revealed a 7-mm acute right SDH with 8.5 mm of midline shift and a basilar skull fracture. His mesencephalic cisterns were compressed, but there was no evidence of hemorrhage in the brainstem (Fig. 2A and B). He was airlifted to the trauma center, and on arrival he had a fixed, dilated right pupil, a nonreactive 3-mm left pupil, and a GCS score of 4. Mannitol and seizure prophylaxis were given, and he was taken immediately to the operating room.

Operation and Postoperative Course. The patient underwent a right subdural evacuation, decompressive craniectomy, and placement of an external ventricular drain. The time from EMS assessment to surgical incision was 3 hours and 20 minutes. He remained intubated postoperatively and his pupillary reactivity returned. A postoperative CT scan showed evacuation of SDH and
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resolution of midline shift, with a pontine hemorrhage and small subgaleal hematoma (Fig. 2C). An MRI study obtained on postoperative Day 1 revealed cerebral and cerebellar hemorrhagic contusions and right-sided diffusion changes, Duret hemorrhage, and enlarged right subgaleal hematoma (Fig. 2D). On postoperative Day 2 the patient had ICP issues and a tense flap due to the subgaleal hematoma; therefore he returned to the operating room for evacuation of the hematoma and placement of subgaleal drain. His ICP improved, and 48 hours after the second surgery he was weaned from sedation and paralytic agents. He was hemiparetic on the left and withdrawing on the right side, but was not following commands. He underwent tracheostomy and gastrostomy tube placement. On postinjury Day 10 he had spontaneous movement of the left side, and by postinjury Day 16 he was speaking in sentences and playing board games.

The patient went to rehabilitation, and at 10 weeks after injury his tracheostomy had been discontinued and he was speaking in full sentences. His cranial flap was replaced 5 months postoperatively, and at 1-year follow-up his hemiparesis has resolved, with only mild residual cognitive deficits (GOS 5).

Discussion

Traumatic brainstem hemorrhages have routinely been divided into primary and secondary etiologies. The differing etiologies for primary traumatic brainstem hemorrhages have been summarized by Hashimoto et al. The 4 mechanisms described are as follows: 1) diffuse axonal injury from acceleration/deceleration; 2) shear strain at the midbrain as a result of the tentorium; 3) injury to neurovascular structures due to edge of tentorium; and 4) hyperextension of the cervical vertebrae injuring the lower brainstem. In distinction, secondary traumatic brainstem hemorrhages result from intracranial hypertension. Secondary hemorrhages also present in a delayed fashion, whereas primary hemorrhages are evident on initial imaging.

The classically described secondary hemorrhages are termed “Duret” hemorrhages and have been long associated with Henri Duret. Although he actually described hemorrhages located along the floor of the fourth ventricle, his name has become the eponym for brainstem hemorrhages secondary to elevated ICP. The first to describe these brainstem hemorrhages pathologically was Attwater in 1911. He identified the hemorrhages on necropsy, and hypothesized that they were due to a large intracranial hemorrhage that resulted in an increase in ICP. The overall mechanism that is agreed upon is that ICP rises so significantly that it results in transtentorial herniation that displaces the brainstem inferiorly, resulting in brainstem hemorrhages.

It has been debated whether the source of bleeding within the brainstem is arterial or venous. With the arterial physiology, the brainstem is displaced inferiorly while the basilar artery remains fixed, thus shearing small perforating vessels, with resultant hemorrhage. The venous hypothesis specifies that the hemorrhages are caused by compression of the rostral brainstem draining veins, with resultant venous stasis and hemorrhage. In 1965, Klintworth described that the occurrence of secondary brainstem hemorrhages corresponded with alterations in systolic blood pressure. Also, the hemorrhages were dependent on the volume and rate of expansion of the intracranial lesion. Interestingly, the improvement of ICP during a particular period of physiological compensation actually accentuated the hemorrhages and was often critical to their production. Therefore they could be regarded as iatrogenic due to the treatment of the elevated ICP. However, regardless of the cause, Duret hemorrhages are known to portend a poor prognosis and may even discourage continuing care due to the grim outlook.

Decompressive craniectomies are typically performed for 2 indications: a mass lesion with resultant edema, or due to refractory elevated ICP. With mass lesions, there is significant literature regarding the timing of surgery for acute SDHs that states that if evacuation is indicated, it should be performed in a timely fashion. Jaganathan et al. reviewed 23 pediatric decompressive craniectomies for elevated ICP and stated that the timing of a craniectomy is best determined with continuous ICP monitoring, serial follow-up imaging, and assessment of the patient’s condition. However, in TBIs, close attention needs to be paid to the indirect signs of elevated ICP and these need to be acted on swiftly.

Although most studies attribute fatality to secondary traumatic brainstem hemorrhages, there have been sparse reports of good recovery. One case report discusses a brainstem hemorrhage after electrolyte disturbances in an adult, whereas 4 papers report on adults with brainstem hemorrhages after evacuation of mass lesions who had good neurological recovery. The time to evacuation of the mass lesion in the aforementioned cases is unknown. However there is a growing number of patients with these “fatal” brainstem lesions who have good outcomes; therefore there has to be a commonality that allows a certain subset of patients to do well after significant brainstem compromise. As evidenced by our 2 cases, a younger, more plastic brain may also play a role; however, we also think that timely evacuation leads to less downward herniation on the brainstem and perhaps an improved outcome. Therefore, traumatic brainstem hemorrhages should not be presumed to be uniformly fatal.

Conclusions

Good neurological recovery after traumatic brainstem hemorrhages has not been previously described in the pediatric population. We think that timely decompression led to these outcomes. One could theorize that the shorter the duration of the transtentorial herniation, the better likelihood of a good outcome, regardless of the postoperative brainstem hemorrhage, which could actually be due to the rapid decompression. Also, a factor that must be considered is the inherent plasticity of the pediatric brain. We strongly believe that the case for intervening in a severely neurologically compromised child should be individualized, and immediate decompression shouldn’t be used in all cases of TBI. However, if a brainstem hemorrhage develops after intervening, all hope should not be lost. Good neurological prognosis is not the norm, but it is a feasible outcome.
Disclosure

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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