Air Medical Journal xxx (2016) 1–4

Contents lists available at ScienceDirect

Air Medical Journal

journal homepage: http://www.airmedicaljournal.com/

Case Review

A Traumatic Epidural Hematoma in a 15-Year-Old Male

Michelle M. McLean, MD, EMT-P, Sara Adibi, MD, Corey Alvarez, MD, Mohammed Alkhalifah, MBBS, Alyssa Stroud, Jacob Flinkman, MD, Peter V.R. Tilney, DO, EMT-P, FACEP

A 15-year-old male was brought into the emergency department via an air medical helicopter. He was riding his bicycle without a helmet when he was struck by a motorcycle traveling at an unknown rate of speed. Witnesses reported that the patient was thrown from his bike. The distance he traveled was not quantified. He was ambulatory on scene for a brief period. Upon emergency medical service arrival, the patient became less responsive and was only responding to repeated painful stimuli. He continued to become more unresponsive and required assistance with ventilations via a bag mask. Upon flight crew arrival, he was subsequently intubated using rapid sequence induction using standard doses of succinylcholine, etomidate, and vecuronium per the team's out-of-hospital guideline. There were no complications. During the course of transport, he did not receive any additional analgesic or sedative medications because he showed no purposeful movement or significant pain response.

His initial vital signs upon flight crew arrival included blood pressure of 138/80, temperature of 94.4°F, heart rate of 54 beats per minute, respiratory rate of 16 breaths per minute, and SaO_2 of 100%. The physical examination showed an unresponsive, intubated young male with a Glasgow Coma Scale (GCS) of 3T (verbal: 1, eye: 1, and motor: 1). He had multiple abrasions to both sides of the scalp as well as a large hematoma to the frontal and right parietal region without any significant external bleeding. There was no periorbital ecchymosis, battle signs, or hemotympanum noted. There were no facial fractures or deformities appreciated. His pupils were 3 mm bilaterally and minimally reactive to light. The only other significant finding was that his pulses were palpable, but they were noted to be weaker in the lower extremities; he had a 4- to 5-cm laceration to the lateral aspect of the left lower leg with no bony deformities noted. Bleeding was well controlled, and his capillary refill was less than 2 seconds in all extremities. There were notable abrasions on his back in the lumbar paraspinal region, which were more significant on the right versus the left side. No step-off deformities were noted with palpation of the spinous processes. Subsequently, the patient was placed on a long board with spinal immobilization and transferred by air medical helicopter to a level 2 trauma center with pediatric capabilities. During transport, the patient remained unresponsive without alterations in his vital signs.

The air medical crew requested a primary trauma response before arriving at the level 2 trauma center in order to facilitate an appropriate level of resuscitation. The entire trauma team was in the room before the patient's arrival. The advanced trauma life support protocol was initiated. The primary survey was completed, which included a portable chest and pelvis radiograph that were negative for any acute traumatic injury. The endotracheal tube was 2.5 cm above the carina. A bedside focused assessment with sonography for trauma was negative for any evidence of blood in the abdominal cavity. Peripheral access was obtained by paramedics before arrival; in addition, another large-bore intravenous line was established by nursing staff. The secondary survey was conducted, and the patient was removed from the long spinal board. Appropriate trauma laboratory values were obtained including a type and cross match for blood. Additionally, an arterial blood gas revealed the following: pH of 7.24, PCO_2 of 62, and PO_2 of 424. Ventilator settings were manipulated to decrease the fraction of inspired oxygen to 50% with a tidal volume of 350 mL, a respiratory rate of 20, and positive end-expiratory pressure of 5. The respiratory rate was increased to facilitate the reduction of his hypercarbia. Continuous end-tidal carbon dioxide monitoring was initiated to further assist in monitoring his respiratory status.

As the patient was being prepared for further imaging, he showed decorticate movement. Fifty micrograms of fentanyl was provided intravenously. Shortly after this medication administration, his heart rate became bradycardic with a rate of 30. His blood pressure remained stable and normotensive. A discussion ensued as to the etiology of the bradycardia. The ongoing hypothesis was that the dramatic rate drop was secondary to the medication administration and possible parasympathetic stimulation because of movement of the endotracheal tube. His heart rate continued to drop into the 20s. This prompted the team to administer atropine 1 mg intravenously, and his heart rate increased appropriately. There was no concurrent elevation of blood pressure concerning for a full Cushing response. A repeat neurologic examination was completed. His pupils were nonreactive, which was concerning for worsening neurologic injury. He was taken to the computed tomographic (CT) scanner with the trauma team accompanying him.

A CT scan of the patient's head showed a right frontal and temporal epidural hematoma with a mass effect and midline shift (Fig. 1). In addition, there were right frontal and temporal skull fractures (Fig. 2). The neurosurgical staff was then contacted for consultation and management of this

1067-991X/$36.00

http://dx.doi.org/10.1016/j.amj.2016.04.013

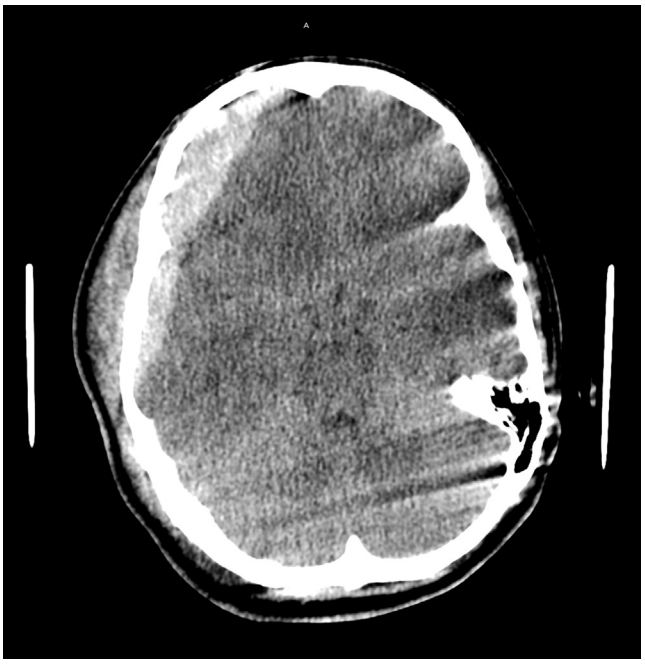

Figure 1. A CT scan of the head showing a right frontal and temporal epidural hematoma with a mass effect and midline shift.

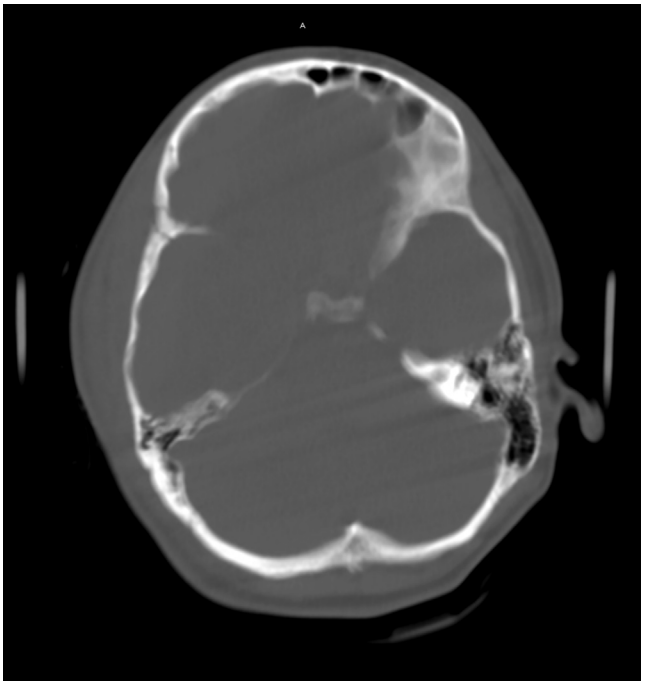

Figure 2. CT head bone windows showing right frontal and temporal skull fractures.

traumatic brain injury. The neurosurgeon believed that the patient would benefit from being taken to the operating room (OR) for a craniotomy and hematoma evacuation. During the surgery, approximately 150 mL blood was immediately evacuated, and a drain was left in place. Other CT imaging studies were reviewed by the treating physicians before the patient was taken to the OR and were negative for any additional acute injuries. The patient's laboratory values were unremarkable. Serial hemoglobin and hematocrit measurements were monitored. Additionally, a repeat arterial blood gas showed an alkalosis with a pH of 7.54; ventilator settings were adjusted by decreasing the respiratory rate to 16. A propofol drip was initiated for sedation. The patient was admitted to the pediatric intensive care unit (ICU) for the remainder of his recovery.

Discussion

Of the many neurosurgical emergencies, epidural hematomas account for approximately 3% to 8%, making them the most common serious head injury.[1] Epidural hematomas are less common in the pediatric population, comprising only 2% to 5% of pediatric head injuries.[2,3] It is considered an acute emergency, and unless they are promptly diagnosed and surgically treated, these injuries typically have a poor outcome without intervention. The mortality in children postepidural hematoma has been shown to be as high as 17%. In order to understand the presentation of a patient with an epidural bleed, it is necessary to understand the basic anatomy and physiology.

A cranial epidural hematoma is an irregular collection of blood between the bone of the skull (termed the *calvarium*) and the protective layer surrounding the brain (termed the *meninges*). Although the skull is designed to protect the softer brain from external insults, the meninges help maintain a suitable environment for the central nervous system by providing a lubricated contact surface for this tissue. This prevents microtrauma that would otherwise occur with even slight movement. The meninges are also responsible for maintaining a closed pressure system separate from the rest of the body. This prevents cerebrospinal fluid from leaving the closed nervous system while harmful substances are restricted from entering. If this closed system is compromised in any way (eg, by skull fracture or by mass effect with subsequent compression), nervous system function may rapidly deteriorate.[4]

The outermost layer of the meninges surrounding the brain and spinal cord is termed the *dura mater*, or simply, the *dura*. In the cranium, the dura adheres directly to the calvarium, creating a potential space.[5,6] Adherence between the dura and the skull is greatest at the sutures, or the "edges" of the bone, which combine to make up the skull. This is where the bones are actively producing new cells for growth. The adherence of dura to bone increases with age; therefore, epidural hematomas are primarily discovered in the younger population.[1,4] There is also a tight adherence of the dura to the calvarium during the neonatal period, thus making epidural hematomas more rare in this population as well.[2] Both arteries and venous sinuses traverse the meninges and calvarium. Disruption to the integrity of these vessels results in the accumulation of blood between the skull and the dura. Epidural hematomas can be caused by many insults including, but not limited to, infection, arteriovenous malformation, venous sinus insufficiency, metastases to the skull, and other rare bone or vessel disorders.[7]