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A 20-Year-Old-Male with Hemorrhagic Shock

A 20-year-old man was the operator of a motor vehicle that ran off the road, rolled, and struck a telephone pole at a high rate of speed. On arrival of the first responders, the patient was unresponsive with agonal respirations. There was significant damage to the vehicle, and extrication of the patient was required. Given the severity of the patient's condition and the anticipated extrication time, rotor wing critical care transport was dispatched for transport of the patient.

On arrival of the helicopter emergency medical services (HEMS) team, initial vital signs were a blood pressure (BP) of 90/60, a heart rate (HR) of 112 beats/min, and an oxygen saturation of 65% despite assistance with a bag valve mask and supplemental oxygen. The patient had agonal respirations at a rate (RR) of 3 spontaneous breaths/min and decreased breath sounds bilaterally. Ground emergency medical services had already placed a nasopharyngeal airway. The patient was unresponsive with a Glasgow Coma Scale score of 3 (E1 V1 M1) and a blood glucose level of 168 mg/dL. The patient was not moving his arms or legs. He was immobilized on a backboard with a cervical spine collar in place. Intravenous access had been established with a 16-G catheter in the right antecubital fossa.

Despite the respiratory support being provided, the patient was persistently hypoxic with oxygen saturations in the 50s and 60s. Given the patient's inability to protect his airway and failure to oxygenate, the decision was made to perform a rapid sequence intubation. A nasal cannula was placed for augmentation of apneic oxygenation. A 2-person bag valve mask technique was used in an attempt to optimize preoxygenation. The patient's oxygen saturation increased to 90% but could not be improved further. Ketamine and succinylcholine were used for rapid sequence intubation. The patient was intubated on the first attempt by direct laryngoscopy and without further oxygen desaturation from the patient's preintubation baseline. Tube placement was confirmed with continuous end-tidal carbon dioxide waveform capnography. The patient was placed on the transport ventilator, and despite a fraction of inspired oxygen (FiO_2) of 100% and increasing positive end-expiratory pressure, the patient's oxygenation could not be significantly improved. Because of persistent hypoxia, there was concern for pneumothorax, and bilateral needle decompression with 10-G angiocatheters was performed. The patient did have improvement in oxygenation after needle decompression.

The patient was transferred to the HEMS transport cot, at which time a pelvic binder was placed because of concern that the patient could be in hemorrhagic shock because of bleeding in the pelvis. En route to the level I trauma center, the patient's vital signs were as follows: BP = 73/41, HR = 130, O_2 saturation of 98% on 100% FiO_2, RR = 17 and end-tidal carbon dioxide of 34 mm Hg. Because the patient was now severely hypotensive from presumed hemorrhagic shock, aggressive resuscitation was initiated with blood products. During transport to the hospital, the patient received 1 unit of uncross-matched O negative packed red blood cells (pRBCs) and 1 unit of A negative liquid plasma. Because the patient only had 1 point of intravenous access, additional access was obtained with placement of a right humeral intraosseous access. The patient was given 1 g tranexamic acid (TXA) through this access point.

On arrival to the emergency department, the patient's vital signs were as follows: BP = 119/80, HR = 90, O_2 saturation of 90% on 100% FiO_2, and RR = 16. The patient received bilateral chest tubes for evacuation of hemopneumothoraces. The patient again became hypotensive with a BP of 84/50 and HR of 104 beats/min, prompting further resuscitation with an additional 1 U pRBCs and 1 U fresh frozen plasma. His blood pressure improved, increasing to 125/70, and his tachycardia resolved. On tertiary examination, the patient was found to have a right ear laceration with damaged cartilage, which was repaired at bedside. He also had absent rectal tone and priapism, which were suggestive of a spinal cord injury. To identify injuries, cross-sectional imaging of the patient's head, chest, abdomen, and pelvis as well as cervical, thoracic, and lumbar spines was performed. The patient was found to have injuries including pulmonary lacerations with associated pulmonary hemorrhage and bilateral hemothoraces (Fig. 1), residual bilateral pneumothoraces (Fig. 2), traumatic brain injury (TBI) with multiple areas of scattered subarachnoid hemorrhage and associated intraventricular hemorrhage as well as intraparenchymal hemorrhage, and several thoracic spinal fractures with associated paraspinal hematoma (Fig. 3). He was admitted to the surgical intensive care unit for management of his injuries.

On admission, an extraventricular drain was placed for intracranial pressure monitoring. The patient's hospital course was complicated by severe acute respiratory distress syndrome, requiring aggressive ventilator support. Chest tubes were able to be removed on hospital day 5 with no residual pneumothorax or hemothorax. He had his spinal column injuries repaired on hospital day 12. He was unable to be completely weaned from ventilator support and had a tracheostomy and percutaneous endoscopic gastrostomy on hospital day 16. He was discharged from the hospital to a long-term care facility on hospital day 18. His neurologic

examination at discharge was a Glasgow Coma Scale score of 9T (E3 V2 M4).

Discussion

Trauma Epidemiology

Trauma-associated injury is the leading cause of death for individuals under the age of 46.[1] Many of these patients die from hemorrhage within the first 2 to 3 hours after injury.[2-4] TBI is also a highly prevalent cause of mortality in patients with traumatic injury.[5] Because patients of severe trauma can have multiple injuries, their resuscitation and care require a thoughtful approach.

TXA

The ability of TXA to reduce mortality after traumatic injury has been documented in 2 large clinical trials.[6,7] The benefit in mortality was shown with administration of the drug within 3 hours of initial injury, and increased mortality from bleeding is noted with administration greater than 3 hours after injury.[8] Because treatment is time sensitive with a defined treatment window beyond which the drug may be harmful, the prehospital setting may be the preferred location for the initiation of treatment. One study examined the time from injury to TXA administration as well as the time from injury to delivery to a trauma center for HEMS.[9] The authors report that many of the patients treated with TXA arrived at the accepting hospital within 10 minutes of or beyond the 3-hour treatment window. Thus, by delivering TXA in the prehospital setting, they were able to capture patients who otherwise would have fallen outside of the appropriate treatment time frame. A review of the newest literature to help guide the use of TXA has recently been published,[10] and a brief review of some commonly accepted indications include the following: 1) age $\geq$ 16 years, 2) evidence of significant blunt or penetrating trauma based on history and/or physical examination, 3) $\leq$ 3 hours since initial injury, and 4) evidence of hemodynamic instability as indicated by systolic blood pressure $\leq$ 90 mm Hg and/or HR $\geq$ 110 beats/min.

Resuscitation

One of the more frequently discussed topics in the critical care transport and trauma literature is the appropriate resuscitation of the trauma patient. Terms such as permissive hypotension, damage control resuscitation, hypotensive resuscitation, and hemostatic resuscitation are used to connote a resuscitation strategy that is cognizant of the potential adverse effects of high-volume fluid resuscitation strategies in hemorrhaging trauma patients. Fluid restriction before operative management for the patient with penetrating torso trauma has been shown to improve survival.[11] However, the physiologic mechanism for this clinical phenomenon is unproven. The response of each individual to traumatic injury is dependent on injury type and severity as well as individual physiology. Consequently, there is likely no single resuscitative strategy that is appropriate for all patients with traumatic injury. There are many factors to consider when resuscitating a trauma patient.[12] Important considerations include mechanism of injury (blunt, penetrating, crush, or burn), is there active bleeding and can it be controlled in the prehospital setting, is there evidence of hemodynamic instability or collapse (hypotension, tachycardia, cool extremities, prolonged capillary refill, or palpable pulse), presence of TBI,

Figure 1. Pulmonary laceration with associated hemothoraces after bilateral tube thoracostomy evacuation.

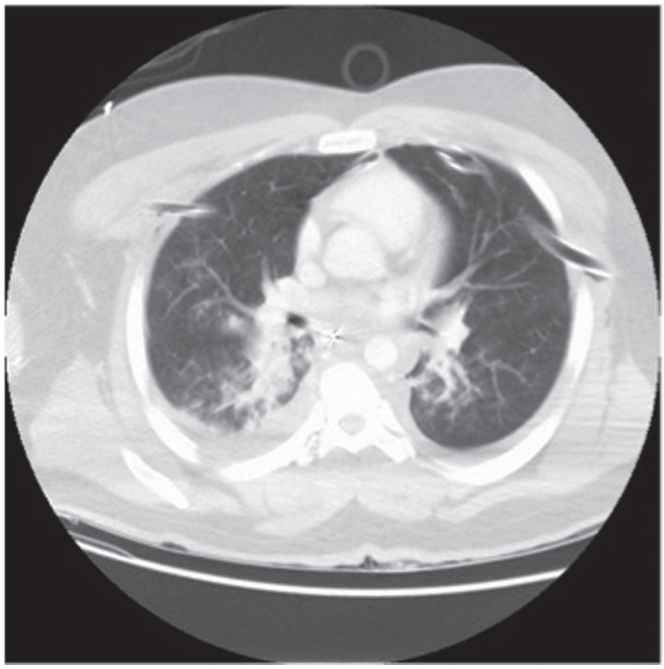

Figure 2. Small, residual bilateral apical pneumothoraces after tube thoracostomy evacuation.