

# Anesthetic Management of a Complex Pediatric Trauma Patient

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*Trauma is a leading cause of mortality for pediatric patients worldwide. An increase in pediatric trauma cases occurs during summer because of a change in schedule and an increased risk with recreational activities. This case report presents the anesthetic care and management of a 15-year-old female who was involved in a high-speed rollover motor vehicle accident. In this case, multiple emerging therapies*

*were combined with long-standing treatments. As a result, the patient who had a complicated intraoperative course survived multiple injuries and cardiac arrest without any neurologic insult.*

**Keywords:** Cardiac arrest, hepatic injury, massive transfusion, pediatric trauma, tranexamic acid.

Intraoperative management of pediatric trauma patients is multifaceted. Injuries must be quickly identified and addressed. Pediatric patients maintain normal hemodynamic parameters but may quickly deteriorate.<sup>1</sup> As a result, if signs of instability are present, the patient is generally in profound shock. Intra-abdominal trauma presents an ideal situation for a hemorrhage to progress undetected, and a significant blood volume may be lost.

Early volume replacement has long been the standard treatment for intraoperative bleeding.<sup>2</sup> As trauma care has evolved, research has led to new therapies and techniques. These include massive transfusion protocols (MTP), recombinant factor VIIa, and tranexamic acid (TXA). Current world events have led the military to evaluate these methods in real world conflicts, which have large numbers of acute trauma patients. This has resulted in changes to the standards of practice in civilian trauma care.

## Case Summary

A 15-year-old female, with an estimated weight of 70 kg, presented to the emergency department of a level-1 trauma center. Because of the distant location in a neighboring state, the patient was transported by air after a high-speed rollover motor vehicle accident. The patient was restrained by airbag deployment; however, there was significant damage to the vehicle with intrusion into the passenger compartment. Two vehicle occupants were ejected and expired at the scene. Responders found the patient unresponsive with a Glasgow Coma Scale of –3; she was extricated with a cervical collar and placed on a long spine board. The patient was intubated with rapid sequence intubation by responders prior to transport. She was hemodynamically unstable en route, with suspected head injuries.

Upon arrival in the resuscitation bay, the waiting trauma team evaluated her. Placement of a 7.0-mm (internal diameter) endotracheal tube was confirmed with bilateral breath sounds and the presence of colorimetric end-tidal carbon dioxide. She remained on full spinal precautions. One intravenous line was in place with a 1-L infusion of lactated Ringer's. The primary survey was completed and revealed coarse rhonchi, but equal bilateral breath sounds; a stable pelvis; an open wound on the posterior scalp with a suspected, depressed skull fracture; ecchymoses on the face and head; and abrasions to the right arm, face, and scalp. An initial focused assessment sonography in trauma (FAST) exam revealed trace fluid in the lower pelvis. Laboratory studies were ordered and included the following: complete blood count (CBC), basic metabolic panel (BMP), type and cross (T&C), lactate, and coagulation studies. A portable chest radiograph revealed that the endotracheal tube was in a good position, minimal bilateral hemothoraces, and pulmonary contusions. A pelvic radiograph revealed no abnormalities. Pupils were 2 mm and minimally reactive. Her vital signs are shown in Table 1. During assessment, the patient remained hemodynamically unstable in spite of fluid resuscitation and the administration of 2 units of O Rh-negative red blood cells (RBC). A repeat FAST exam was done that showed an increase in the previously revealed pelvic fluid and new fluid surrounding the liver. At this point, the decision was made by the trauma surgeon to defer computerized tomography, to initiate massive transfusion protocol, and to take the patient to the operating room for an emergency exploratory laparotomy.

In the operative suite, the patient's abdomen was prepped and draped by the surgical staff while anesthesia obtained additional vascular access. A 16-gauge catheter was inserted into the right arm and an 18-gauge into the

	Blood pressure	Heart rate	Respiratory rate	Oxygen saturation (%)
Hospital arrival	83/48 mm Hg	140 beats/min	16 breaths/min (Controlled)	100
Anesthesia start	60/40 mm Hg	115 beats/min	12 breaths/min (Controlled)	100
Postoperatively	95/40 mm Hg	110 beats/min	18 breaths/min (Controlled)	90

**Table 1.** Vital Signs

left arm with difficulty because of intravascular depletion. A 20-gauge left radial arterial line was also inserted, while 3 units of emergency release type O Rh-negative blood was administered. The laboratory results are shown in Table 2. Initial vasopressor support was accomplished through multiple intravenous (IV) boluses of phenylephrine, 100 µg, and ephedrine, 5 mg. With delayed hemodynamic improvement, boluses of vasopressin, 1 unit IV, were also administered. Crystalloid administration was restricted to avoid hemodilution. Volume replacement was accomplished with 750 mL of 5% albumin. An 18-French orogastric tube was inserted with return of normal gastric contents. Massive transfusion protocol was continued with blood products administered roughly in a ratio of 1 RBC to 1 fresh frozen plasma (FFP), with 1 unit of platelets (PLT) given for every 10 units of blood product (FFP/RBC). Additionally, the patient received midazolam, 2 mg IV; calcium chloride, 1 gm IV; and scopolamine, 0.4 mg IV.

Prior to incision, there was a delay in obtaining additional blood products, requiring additional colloid and medication support. The patient received an additional 500 mL of 5% albumin IV and 500 mL of hydroxyethyl starch IV. Sodium bicarbonate, 100 mEq, and rocuronium, 50 mg, were also given IV. The patient was briefly hemodynamically stabilized, and sevoflurane inhalation was administered at 0.4%.

Upon opening the abdomen, gross hemoperitoneum was apparent. Laparotomy sponges were packed into the abdomen to obtain hemorrhage control. The abdomen was then evaluated by quadrant with the right upper quadrant identified as the source of the bleeding. The liver was examined and had substantial injury to segments 6, 7, and 8. With the identified hepatic injury and impaired coagulation of the patient, cryoprecipitate, 2 units; desmopressin, 2.1 µg; and recombinant human coagulation factor VIIa, 6,300 µg (90 µg/kg), were administered IV. The patient received several more grams of calcium chloride and 100 mEq of sodium bicarbonate. Vasopressors (ephedrine, Neo-Synephrine, and vasopressin) were also administered. An epinephrine infusion was initiated and titrated in an attempt to maintain a systolic blood pressure of 90 mm Hg.

With continued hypotension, the abdomen was reopened to identify the venous bleeding from the injured liver. The liver was repacked in an attempt to control the bleeding; however, ST-segment depression developed in

all electrocardiogram (ECG) leads. The patient's condition deteriorated, and ventricular fibrillation (VF) was identified on the monitor. External cardiac compressions (CPR) were initiated while the defibrillator was charged. An asynchronous shock of 200 J was delivered, and CPR was resumed. Epinephrine, 1 mg, was bolused IV, but the patient remained in VF. A subsequent shock was delivered at 300 J without any effect. CPR was resumed, and amiodarone, 300 mg, was administered with an IV bolus. A third shock at 360 J was delivered, CPR was resumed, and the rhythm converted to sinus tachycardia.

After the bleeding was controlled, gel foam was applied to the surface of the liver, the abdomen was closed, and a wound vacuum was applied. With closure, the patient again deteriorated, experiencing cardiac arrest with ventricular tachycardia (VT) identified on the monitor. CPR was initiated, and the patient was defibrillated at 360 J. The abdomen was reopened, and a retractor was placed to visualize a continued hemorrhage from the liver. The hepatic artery and portal vein were clamped (Pringle maneuver) with bleeding cessation. Because of continued coagulopathy, a 10-mg/kg bolus of TXA was administered IV, followed by an infusion at 1 mg/kg/h.

During the 3-hour case, the patient had received a substantial volume of blood products, and concern developed for transfusion-related acute lung injury (TRALI) versus non-cardiogenic pulmonary edema. Protective strategies included an increase in positive end-expiratory pressure (PEEP) to 8 mm Hg and maintenance of ventilation with decreased tidal volumes with an increased rate. Urine production had remained stable, greater than 1 mL/kg during the case, but with the significant volume of fluid resuscitation, it was felt that the patient was volume overloaded. Pink, frothy fluid was suctioned from the endotracheal tube, and furosemide, 20 mg, was administered IV. With the existing pulmonary contusions, oxygenation became increasingly difficult, as her SpO<sub>2</sub> decreased into the upper 80s. An additional 20-mg dose of furosemide was administered IV with a subsequent increase in urine output.

After the patient was stabilized, the abdomen was packed with an ABThera dressing that was applied to a vacuum. At this point, the patient had received 40 units of RBCs, 28 units of FFPs, 6 units of PLTs, and 2 units of cryoprecipitate. The estimated blood loss was 5 L.

While an attempt was made to administer blood products in a 1:1:1 ratio, limited availability resulted in

	Hemoglobin	Hematocrit	Platelet count	Partial thromboplastin time	International normalized ratio	Calcium	Lactate
Hospital arrival	6.8	21.3	258	58	2.04	6.1	4.3
Intraoperative status	9.1	26.5	110	57	1.07	9.9	5.8
Postoperative status	14.6	41.3	72	38.9	1.18	10.5	9.7

**Table 2.** Laboratory Results

a discrepancy. The patient's hemoglobin and hematocrit postprocedure were normalized; however, the platelet count remained low necessitating the administration of additional platelets in the pediatric intensive care unit (PICU). The blood pressure remained stable while the patient was administered an epinephrine infusion. The TXA infusion was also continued in the PICU.

In the PICU, the patient's oxygen saturation remained 88%-92%. Neurosurgery was consulted because of the suspected skull fracture and head injury. The decision was made to insert an intracranial pressure (ICP) monitoring device. The initial ICP was 7 mm Hg, alleviating the concern to conduct a CT scan on the patient until she was further stabilized. The patient was placed on a propofol infusion for continued sedation. The epinephrine drip was changed to a norepinephrine drip with a decrease in heart rate and maintenance of perfusion. A subclavian central venous catheter was inserted by the trauma service. The patient's pulmonary status continued to improve overnight, while her oxygen saturation levels remained at 95%-100%. A 12-lead ECG was conducted that showed T-wave inversion in the inferolateral leads. Postoperative diagnoses included liver laceration, splenic laceration, renal injury, and bilateral pulmonary contusions. The patient's postoperative course was complicated by a pleural effusion, which was treated with a pigtail catheter with improvement. She did necessitate several trips to the operating room for abdominal closure and was discharged home on postoperative day 24, when she was neurologically intact.

## Discussion

Uncontrolled hemorrhage is a leading cause of death from trauma. It generally occurs because of a combination of surgical and coagulopathic bleeding. Coagulopathy in trauma is multifactorial, involving hemodilution, hypothermia, consumption of clotting factors, and metabolic derangements.<sup>3</sup> Two major conditions that often present with severe coagulopathy and trauma are acidosis and hypothermia, both of which have a profound impact on coagulation and clot stability.

In the event of unsuccessful reversal of coagulopathy through traditional means, administration of factor VIIa recombinant (rFVIIa) has been used to control traumatic bleeding.<sup>4</sup> Factor VIIa is indicated for hemorrhage resulting from the inhibition of factors VII and IX that are

found in patients with hemophilia A or B. Hemorrhage control is accomplished through activation of the extrinsic pathway of the coagulation cascade. The activation results in the formation of a complex with tissue factor (TF) at the site of injury, thus activating factors IX and X. Subsequently, factor Xa then converts prothrombin to thrombin, forming a hemostatic plug by converting fibrinogen to fibrin, ending in local hemostasis.

The medication is dosed at 90 µg/kg that can be administered every 2 h until hemostasis is achieved. The half-life of rFVIIa is approximately 2 h. In cases of severe hemorrhage, the interval for maintenance doses is 3–6 h. The most significant adverse event is the potential for thromboembolism; this has been associated with disseminated intravascular coagulation (DIC), crush injury, atherosclerotic disease, and septicemia. Monitoring of coagulation factors has not been found to predict the effectiveness of rFVIIa therapy. Concern must be given to the increased cost associated with the administration of rFVIIa that may also decrease its facility availability.

Desmopressin (DDAVP) was first used to treat patients with hemophilia A and von Willebrand's disease (vWF) in 1977.<sup>5</sup> Originally studied in Italy, it was found that desmopressin could raise the plasma levels of factor VIII and vWF without the need for blood products that could be extremely beneficial, so the World Health Organization (WHO) added it to the list of essential medications. It was quickly recognized that desmopressin could be utilized beyond the bleeding disorders for which it was originally intended. These uses included the platelet function defects that are commonly associated with chronic liver and kidney diseases.<sup>6</sup>

In spite of longstanding clinical use, the mechanisms of action are still not completely understood.<sup>7</sup> Increases in factor VIII and vWF are found to occur in healthy and deficient patients. DDAVP shortens the prolonged activated partial thromboplastin time and bleeding time. It is thought these effects occur as a result of increases in factor VIII and vWF, which accelerate primary hemostasis. It also has no effect on platelet count or aggregation but improves platelet adhesion to vessel walls. Another short-lived effect is the release of tissue plasminogen activator, which in turn generates plasmin. However, it does not produce fibrinolysis; thus, it is often necessary to inhibit fibrinolysis when DDAVP is used. Recommended dosing is 0.3 µg/kg that may be adminis-

tered IV, subcutaneously (SQ), or intranasally. Benefits of DDAVP are reduced cost, increased availability, diversity of usage, and ability to meet religious requests.

TXA is a synthetic derivative of the amino acid, lysine, which inhibits fibrinolysis by blocking the lysine-binding sites on plasminogen.<sup>8</sup> Because the hemostatic responses to surgery and trauma are similar, TXA might reduce mortality secondary bleeding in trauma patients. The Clinical Randomisation of an Antifibrinolytic in Significant Haemorrhage 2 (CRASH-2) study assessed the effects of the early administration of a short course of TXA on death, vascular occlusive events, and the receipt of blood transfusions in trauma patients with or at risk of significant hemorrhage.<sup>9</sup> The results showed that the early administration of TXA to trauma patients with or at risk of significant bleeding reduces the risk of death from hemorrhage with no apparent increase in fatal or nonfatal vascular occlusive events. All-cause mortality was significantly reduced with TXA.

Comparatively, TXA has a relatively low cost, ease of administration, and a low side effect profile.<sup>10</sup> The results of the CRASH-2 study indicated that the drug reduces the risk of death from bleeding in trauma patients. The method through which the medication reduces bleeding is unclear. The blood transfusion requirements for the TXA and placebo groups were similar, and notwithstanding the survival bias, the mortality benefit might have been attributable to an effect of TXA on an issue other than acute traumatic coagulopathy. What is clear is the requirement for early administration, less than 3 h after an injury. The study clearly revealed an increased mortality if TXA was administered more than 8 h after an injury.

A question as to which patients should receive TXA exists.<sup>11</sup> Thus, a fault of the study included the following: it was conducted primarily in low- to moderate-income countries that have a decreased availability of blood products. Patients in the United States commonly have access to FFP and other products as a first-line therapy. The evidence demonstrates early administration is more advantageous than late. As a result, prehospital administration may be the most appropriate time for therapy.

In addition to confusion regarding patient selection and the exact mechanism of action, there are several other discrepancies regarding TXA. These include dosage and administration schedule. There are various doses ranging from a one-time 20 mg/kg dose<sup>12</sup> to the CRASH-2 dosing of 1 g IV over 10 min, followed by a subsequent infusion of 1 g over 8 h. The advantages of TXA include its low cost, lack of need for refrigeration, and decreased transfusion reaction risk.

Massive transfusion, which has been defined as the administration of 10 or more blood products, has a demonstrated role in the management of traumatic hemorrhage. Accompanying this definition is a standing protocol found in facilities to allow access to large volumes

of blood products to ease rapid administration to critical patients.<sup>13</sup> Massive transfusion is associated with hemostatic and metabolic consequences. An additional component of a massive transfusion is the recognition that the early administration of blood products in a ratio that approximates whole blood is beneficial for the patient. With implementation of massive transfusion protocols, the provider must also consider several issues including patient's volume status, tissue oxygenation, hemorrhage control, and coagulation abnormalities. Large-volume blood product administration also affects potassium, ionized calcium, and acid-base balance.

Clinical research has demonstrated that the administration of blood products in a 1:1:1 ratio (RBC:FFP:PLT) is the most appropriate for the early resuscitation of patients with severe hemorrhage.<sup>14</sup> Early mortality from trauma is primarily a result of head injury or hemorrhage that becomes more significant because of coagulopathy.<sup>15</sup> Late trauma mortality is secondary to multiorgan failure. Massive transfusion has been found to contribute to coagulopathy through the dilution of coagulation factors. Surgery to arrest a hemorrhage is the primary intervention, but early blood product replacement has demonstrated efficacy.

## Conclusion

Trauma is a leading worldwide cause of pediatric mortality. Recent developments that impact the anesthetic management of trauma patients include massive transfusion protocols, recombinant factor VII, and TXA. While the exact role of these new developments has not yet been fully explored, it appears prudent to consider and implement these strategies and to include TXA early during the resuscitation of a bleeding trauma patient.

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