

Damage Control Resuscitation of a Patient With Traumatic Aortic Rupture: A Case Report

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Traumatic aortic rupture (TAR) is a highly fatal injury mechanism resulting from blunt deceleration forces against the descending aorta. The mechanism of TAR is directly attributed to the aorta suffering damage by indirect shearing forces. The descending aorta remains fixed to the posterior chest wall, while the heart and ascending aorta are exerted forward, thus causing the intimal tear. A characteristic triad presents as increased blood pressure in the upper extremities, decreased blood pressure in the lower extremities, and a wid-

ened mediastinum on radiography. Early recognition of signs and symptoms of the mechanism of injury is key to initiating early damage control surgery and ultimately decreasing morbidity and mortality. This case report describes the intraoperative management of an elderly female patient with TAR following a motor vehicle collision in a remote location in rural Pennsylvania.

Keywords: Critical event, damage control resuscitation, traumatic rupture.

Dr R. Adams Cowley, known as the father of trauma medicine, is quoted as saying “there is a golden hour between life and death. If you are critically injured, you have less than 60 minutes to survive. You might not die right then; it may be three days or two weeks later—but something has happened in your body that is irreparable.”¹ In the United States, trauma is the leading cause of death for individuals up to 45 years of age and is the fourth leading cause of death overall.² Hemorrhage accounts for 30% to 40% of all trauma deaths and mortality is widely attributed to uncontrolled hemorrhage from preventable coagulopathy.³ The golden hour serves as the practice foundation of civilian and military emergency medical services (EMS) and trauma systems around the world.

Among the leading causes of death in trauma, traumatic aortic rupture (TAR) is a highly fatal injury mechanism resulting from blunt deceleration forces with an immediate mortality rate of 85% of those who reach the emergency department (ED).⁴ An estimated 7,500 to 8,000 cases of TAR occur each year in the United States and only 1,000 to 1,500 of these individuals survive to hospital arrival.⁵ Of those who arrive at an ED, 23% present with cardiac arrest on arrival.⁶ Motor vehicle accidents are the leading cause of TAR (50%), followed by falls from higher than 3 m (10 ft), pedestrians struck by motor vehicles, and severe crush injuries. With an equal proportion of deaths resulting from complete and partial aortic transections, the overall mortality rate is 59%.⁷ Surgery is the only definitive treatment of this catastrophic injury complex.

In most cases, TAR results in immediate death in the prehospital setting well before surgical intervention is possible. Because of the time-sensitive nature of the

disease process, prehospital providers are extensively trained in the rapid assessment and early recognition of signs and symptoms related to the TAR injury complex. In rural settings, air medical evacuation is often necessary to maintain the golden hour doctrine. Amid the forward evolution of early hemorrhage control, many air medical programs are outfitted with antifibrinolytic agents, such as tranexamic acid, and have become increasingly equipped with packed red blood cells (PRBCs) and fresh frozen plasma (FFP) to administer in the setting of refractory hemorrhagic shock.

Tranexamic acid is a synthetic derivative of lysine that inhibits fibrinolysis by competitively inhibiting the lysine binding sites on plasminogen. This mechanism prevents conversion of plasminogen to plasmin leading to clot breakdown.⁸ Comparatively, tranexamic acid is 10 times more potent than aminocaproic acid.⁹ Forming the systemic integration of tranexamic acid into the prehospital environment, the CRASH-2 (Clinical Randomization of an Antifibrinolytic in Significant Hemorrhage-2) trial evaluated the effects of tranexamic acid on mortality and transfusion requirements in 20,211 adult trauma patients, in 274 hospitals throughout 40 countries.¹⁰ The global study found that early administration of tranexamic acid safely reduced the risk of mortality by 20% in patients experiencing hemorrhaging trauma when the medication was administered within the first 3 hours of vascular insult.¹⁰ Tranexamic acid is administered as a 1-g intravenous (IV) loading dose over 10 minutes, followed by a 1-g IV infusion over 8 hours. Often because of rapid transport, prehospital initiation of the infusion is often not possible in the constraints of time. In these situations, the infusion should be started in the ED for maximum efficacy.



Figure 1. Anterior Computed Tomography Image Revealing Right Neck Hematoma due to Shoulder Belt Restraint

Surgical intervention is a definitive need beyond the early recognition and treatment of hemorrhage. Referred to as *damage control surgery* (DCS), the approach involves exploratory laparotomy by the surgical team, with prioritization given to short-term physiologic repair over anatomical reconstruction in the hemodynamically compromised patient. *Damage control resuscitation* (DCR) is a term used for the concurrent management of blood volume replacement in an effort to prevent the lethal triad of hypothermia, acidosis, and coagulopathy. These 2 methods are carried out simultaneously by the surgical and anesthesia care teams in an effort to maximize survivability and reduce patient morbidity and mortality. Indications for DCS are physiologic in nature and include clinically significant hemorrhage requiring massive transfusion (> 10 units of PRBC), severe metabolic acidosis (pH < 7.30), hypothermia (temperature < 35°C), operative time > 90 minutes, coagulopathy, or lactate value > 5 mmol/L.¹¹ The case summary will examine the collaborative approach made by both teams in an effort to provide DCS and DCR for a hemodynamically compromised adult trauma patient.

Case Summary

- **Prehospital Management.** A 65-year-old woman, weighing approximately 90 kg, arrived at a rural level I trauma center in central Pennsylvania via helicopter following a 27-minute flight from the scene of a head-on motor vehicle collision. The trauma victim was the restrained front-seat passenger in a modern sedan, traveling at a

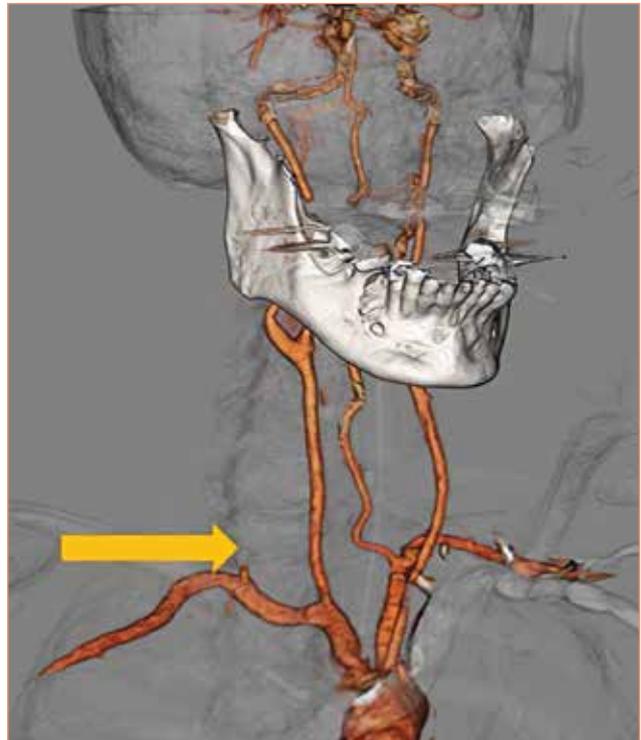


Figure 2. 3-Dimensional Angiogram Revealing Right Vertebral Artery Dissection and Cessation of Blood Flow

high rate of speed, with substantial frontal damage and compartment intrusion. There was 1 confirmed fatality at the scene and 2 additional critically injured patients transported to the same trauma facility. The flight crew reported to the trauma care team that the patient was found anxious, combative, and hypotensive by EMS providers on the ground. Immediate volume resuscitation of 700 mL of crystalloid was initiated during the rescue operation. Following a rapid extrication, the patient was immobilized to a long spine board, and pelvic binder was placed because of instability.

On flight team arrival, the patient exhibited severe ecchymosis across her neck and chest. The bruising pattern extended from the right clavicle to the left midclavicular region with notable swelling, presumably from deceleration forces of the seatbelt. Once secured in the helicopter, she complained of acute dyspnea and became increasingly combative.

Rapid sequence induction was performed by the flight team with 27 mg of etomidate and 140 mg of succinylcholine. Placement of a 7.0-mm endotracheal tube was placed under direct laryngoscopy without complication. Intraosseous needles were established in both the left humerus and right tibia. During transport, a decreased oxygen saturation, elevated peak airway pressures, and diminished bilateral lung sounds were suggestive of tension pneumothoraces. The flight team performed chest needle decompression of the left and right pleural spaces, with notable improvement in oxygenation and

ventilation. While adhering to protocol, the flight team administered a 1-g IV bolus of tranexamic acid because of refractory hypotension with a high index of suspicion for internal hemorrhage.

• **Emergency Department Management.** In the trauma bay of the ED, findings of a rapid assessment revealed an unresponsive patient with hemodynamic instability in extremis. The 7.0-mm endotracheal tube was assessed at 22 cm and confirmed for proper placement with a chest radiograph. Her neurologic status revealed a Glasgow Coma Scale score of 6 (eye opening, 1; verbal response, 1; and motor response, 4) with respect to chemical sedation and paralysis administered to facilitate prehospital endotracheal intubation. The ED staff administered ketamine, 50 mg IV, to the patient for amnestic and analgesic management. Further examination findings revealed an increasingly large right-sided hematoma of the neck, extending into the clavicular region and highly indicative of a shoulder belt injury (Figures 1 and 2). Although heavily diminished, lung sounds were assessed to be present in bilateral fields. Widespread abdominal bruising with marked mottling extending into the extremities was noted as an ominous sign to the trauma care team. Results of a focused assessment with sonography for trauma (FAST) examination of the intraabdominal cavity revealed significant hemorrhage suggestive of vascular disruption. Pelvic instability was secured with a pelvic stabilization device (Traumatic Pelvic Orthotic Device [T-POD], Pyng Medical). A large open avulsion injury of the left lower extremity with mild oozing was also noted. The right lower extremity pulse was palpable; however, there was absence of the left femoral pulse with no distal Doppler sounds present.

On identification of hemodynamic instability, the ED team immediately consulted a vascular surgeon. Bilateral chest tubes were placed as indicated by radiographically confirmed hemopneumothoraces. A central venous catheter was placed in the left femoral vein for rapid volume administration as well as a right femoral arterial line for continuous blood pressure monitoring. An immediate recognition of blood pressure variance was noted between the noninvasive cuff placed on the right upper extremity and the right femoral arterial line, with the lower extremity experiencing a decrease greater than 20 mm Hg systolic blood pressure (SBP) compared with the upper extremity. A 1-g infusion of tranexamic acid was administered, and massive blood transfusion was initiated. Following head, chest, and abdominal computed tomography (CT), the trauma and vascular surgery teams elected to perform DCS and obtain an endovascular aortogram out of concern for TAR and underlying injuries consistent with small-bowel disruption and splenic laceration. The anesthesia care team was immediately notified, and the operating room was prepared within 20 minutes of patient arrival.

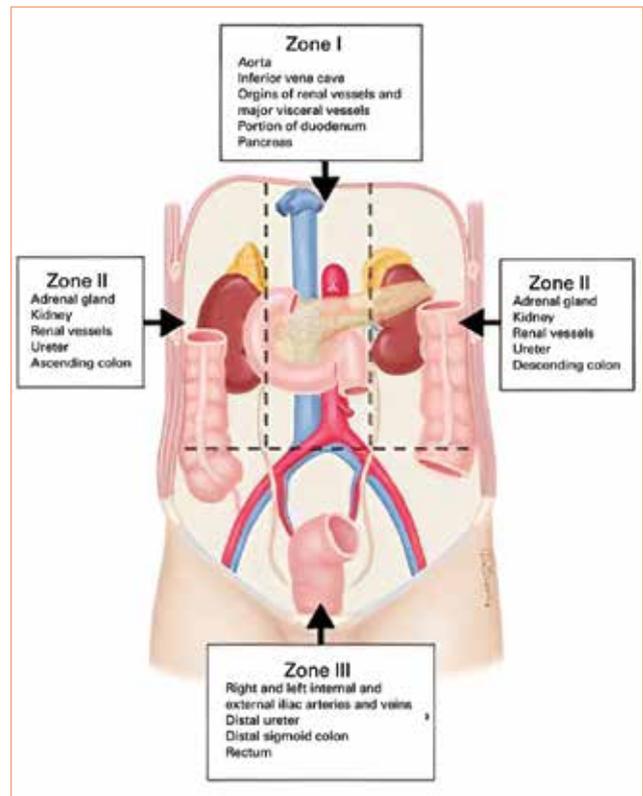


Figure 3. Illustration of Zones of Retroperitoneum

^a Zone 2 was packed with hemostatic gauze during this case. (Reproduced with permission from Mandell SP. Overview of the diagnosis and initial management of traumatic retroperitoneal injury. UpToDate. Copyright 2018 UpToDate Inc. https://www.uptodate.com/contents/overview-of-the-diagnosis-and-initial-management-of-traumatic-retroperitoneal-injury?search=Overview%20of%20the%20diagnosis%20and%20initial%20management%20of%20traumatic%20retroperitoneal%20injury&source=search_result&selectedTitle=1~150&usage_type=default&display_rank=1. Accessed December 1, 2019.)

• **Intraoperative Management.** On the patient's arrival in the operating room, the findings of a secondary trauma assessment revealed increased periumbilical and flank bruising, ashen skin color, and widespread mottling. Pulse oximetry, electrocardiography, and end-tidal carbon dioxide monitors were applied. An additional 50-mg dose of IV ketamine and 2 mg of IV lorazepam were injected via a peripheral IV catheter. Isoflurane at a 0.5 minimum alveolar concentration with a fraction of inspired oxygen of 100% was administered to provide amnesia while maintaining hemodynamic stability. An additional 14-gauge peripheral IV catheter was placed for warmed fluid and blood administration. A 2-g IV dose of cefazolin was administered. Massive blood transfusion was continued via institution protocol in an effort to maintain SBP above 90 mm Hg and preserve body core temperature above 36.0°C.

• **Damage Control Surgery.** Although the trauma and vascular surgery teams worked in collaboration, with many of the collective interventions concurrent

with one another, it is best to describe each approach as individual. Beginning with DCS, an open exploratory laparotomy was performed. An extensive peritoneal sac rupture was identified, with disruption of the visceral cavity and contents infiltrating the subcutaneous tissue of the abdominal wall. Zone 2 of the paracolic gutters was packed with hemostatic gauze. Hemorrhage from the left superior epigastric artery was identified and ligated. An expanding hematoma was noted in the midportion of the small bowel. The devascularized sigmoid colon was resected and the mesenteric artery clamped. Zone 1 retroperitoneum, distal abdominal aorta, and the inferior vena cava (IVC) were exposed, and the primary source of exsanguination was located at the bifurcation of the iliac arteries (Figure 3). Because of the presence of widespread vascular disruption, the decision was made to infuse blood products and volume replacement through a 14-gauge peripheral IV catheter as opposed to the central venous catheter in the femoral vein to avoid potential extravasation into the abdomen. With continued massive blood transfusion, intraoperative cell salvage was not used because of gross bowel contamination, for which the patient was administered 1 g of cefoxitin IV.

- **Vascular Surgery.** Vascular intervention began with left brachial artery exposure, introduction of the catheter into the abdominal aorta, and aortoiliogram. An abrupt disruption of the proximal left common iliac artery was observed at the iliac bifurcation. With active exsanguination of blood volume occurring, a sheath was inserted into the left common femoral artery in preparation for balloon catheter insertion. A balloon catheter (Coda, Cook Medical) was inserted via the left femoral artery sheath and an endovascular balloon was inflated to occlude above the level of the aortic injury (Figure 4). Bilateral iliac arteries were clamped. At this time, hemostasis and hemodynamic stability were achieved. Primary repair of the aortic injury was performed. The occlusive balloon was then deflated.

Following the autotransfusion of lower extremity blood volume and deflation of the endovascular balloon, a rupture of a secondary, midabdominal infrarenal aortic tear was identified at the level of the inferior mesenteric artery. The endovascular balloon was advanced proximal to the injury and reinflated. Despite primary repair of the site, ligation of the inferior mesenteric artery, and ongoing volume resuscitation, the patient's blood pressure remained tenuous. It was promptly discovered that an IVC tear at the right gonadal vein bifurcation was also present, thus requiring additional primary repair and ligation.

Before achieving control of the disrupted aorta, the patient was started on infusions of phenylephrine (50-200 $\mu\text{g}/\text{min}$) and norepinephrine (2-20 $\mu\text{g}/\text{min}$). Results of serial arterial blood gas analyses revealed worsening metabolic acidosis. An IV infusion of vasopressin was initiated (2.4 U/h). Intravenous sodium bicarbonate



Figure 4. Balloon Catheter Placed Superior to Iliac Aorta via Left Femoral Artery and Inflated to Occlude Distal Hemorrhage

and IV calcium chloride were administered as indicated. Following initial aortic repair and deflation of the endovascular balloon, the SBP decreased sharply. At this time, a secondary tear of the infrarenal aorta was identified as a source of hemorrhage. The endovascular balloon was reinflated and SBP was maintained with aggressive volume resuscitation and vasopressor administration. Following primary repair of the second identified aortic injury, the endovascular balloon was deflated and the SBP continued to decrease by 50 to 60 mm Hg. The balloon was reinflated and the aortic clamp replaced. Further exposure revealed an injury to the IVC, which was promptly repaired. It was then determined that the patient would need to be further volume resuscitated and blood pressure maintained at a level that would compensate for the predicted 50 to 60 mm Hg decrease in blood pressure that had been exhibited multiple times with the previous release of the balloon or aortic clamp. An additional 1 L of 5% albumin was administered, and vasopressor therapy was increased to achieve an SBP of 160 mm Hg. The endovascular balloon was deflated and

aortic clamp released. As predicted, the SBP decreased by 50 to 60 mm Hg but was sustained within a range of 90 to 100 mm Hg following release. The DCS continued; zones 1 and 2 of the retroperitoneal space were packed with hemostatic gauze, and the abdominal fascia was kept open with a wound vacuum in place. Hemodynamic stability was maintained for the remainder of the intraoperative course.

In total, the patient had suffered a blunt dissection of the aortic bifurcation, infrarenal aorta, and IVC as well as a vertebral artery dissection, peritoneal sac rupture, grade 4 splenic laceration, diaphragmatic rupture, and mesenteric artery tear. The end-case blood loss was grossly immeasurable but estimated to be approximately 4,000 mL. Crystalloid and colloid replacement were as follows: plasmalyte, 6 L; albumin, 5%, 2 L; PRBCs, 39 units; FFP, 29 units; platelets, 10 units; cryoprecipitate, 2 units; and factor IX, 5,450 units. (Table 1 shows a timeline of critical events.)

• **Postoperative Management.** Ongoing resuscitation continued in the trauma intensive care unit (ICU), where rapid development of acute respiratory distress syndrome and massive blood loss from the abdominal wound vacuum became the primary focus for management. Despite a slight correction of hypothermia, coagulopathy, and metabolic derangement, maintaining oxygenation and hemodynamic stability was a major challenge. Extracorporeal membrane oxygenation was not considered because of widespread vascular disruption. Ultimately, the widespread destruction of the abdominal wall was deemed surgically irreparable and that continued hemorrhage would be imminent (Figure 5). The patient's family was informed that because of the extensive abdominal wall injury and continued hemodynamic instability, the patient would not survive. At the request of the family, resuscitative efforts were stopped. The patient was declared dead 12 hours after hospital admission.

Discussion

Hypothermia, acidosis, and coagulopathy are collectively known as the lethal triad in the trauma population and serve as the secondary injury process following the initial insult. Although the death of this patient was ultimately attributed to uncontrollable hemorrhage due to mass vascular and abdominal wall insult, the lethal triad is a precursor to a complex and progressive cascade.

• **Hypothermia.** Hypothermia induces a multitude of physiologic effects on coagulation, which includes altering platelet function and reducing fibrin enzyme kinetics. Because of widespread cellular damage in the presence of metabolic acidosis, the trauma classification of hypothermia differs from that of traditional normal values (Table 2). When compounded with metabolic acidosis, thrombin and fibrinogen synthesis are inhibited, leading to a potential deficit in fibrinogen stores and further

reducing efficacy of the intrinsic mechanisms to form a clot. Disseminated intravascular coagulation poses a major threat as clotting mechanisms are rapidly depleted in an effort to restore hemostasis. In a recent study, external factors contributing to hypocoagulable state were explored in an in vitro analysis of the lethal triad in trauma. Whole blood samples were taken from 5 healthy volunteers and introduced to respective conditions of hypothermia and metabolic acidosis. Standard coagulation testing, thromboelastometric analysis, and differentiated platelet mapping were performed on the samples. Untreated blood samples obtained at baseline served as a physiologic reference. Following a 33% hemodilution to simulate intravascular conditions during resuscitation, the exposure of blood samples to hypothermia and metabolic acidosis combined led to a one-third reduction in hemoglobin, hematocrit, platelets, and fibrinogen as well as increased prothrombin time.¹²

Hypothermia management of the patient in this case report was difficult because of a multitude of factors. The patient's temperature was continuously monitored via an esophageal probe. Continuous blood loss, visceral exposure, and continuous contact with metal retractors led to a combination of radiation, convection, conduction, and evaporation heat losses. The patient's head was wrapped with a warm blanket, and between critical events, the surgical field was kept covered by sterile towels to preserve the core body temperature. Crystalloid and colloid resuscitation was administered via IV warming devices; however, this excluded platelets, cryoprecipitate, and factor IX administration. There was a malfunction in the heated rapid infuser; therefore, this device was not used. Although crystalloid and colloid were still infused via a fluid warmer, the downfall was that all blood products required a dedicated provider to ensure administration under pressure infusers. In an effort to combat conduction loss, an underbody heating device was used while set to 42°C for maximum heat production.

• **Metabolic Acidosis.** A state of metabolic acidosis begins in the presence of inadequate tissue perfusion due to acute hemorrhage, which results in cellular metabolism converting from aerobic to anaerobic, thus increasing the production of lactate and reducing pH. Metabolic acidosis impairs the coagulation cascade. In a study evaluating the effect of temperature and pH on the activity of factor VIIa in hypothermic and acidotic patients, it was found that platelets were reduced by 90% when the pH was reduced from 7.4 to 7.0.¹³ Hess et al¹⁴ reviewed the reasons for development of acute coagulopathy in trauma. The factors identified included the following: (1) endothelial tissue damage, (2) tissue inflammation via the thrombomodulin-protein C pathway, (3) tissue ischemia, (4) hemodilution resulting from volume resuscitation, (5) hypothermia, and (6) acidosis.

Throughout the duration of this case report, mass

Test or intervention	Time (PM) and surgical intervention								
	3:03 Arrival to OR suite	3:33 Exploratory laparotomy	4:09 Aortic clamp placed	6:13 Aortic clamp released	6:14	6:15 Aortic clamp replaced	6:30 Before clamp release	6:32 Aortic clamp released	7:37 ICU admission
Vital signs									
Heart rate/min	124	125	124	120	NA	111	105	104	105
Noninvasive BP, right arm, mm Hg	92/40 (57)	80/41 (54)	107/58 (72)	66/38 (47)	NA	126/68 (92)	162/84 (110)	96/79 (85)	88/54 (65)
Invasive BP, right side of groin, mm Hg	72/38 (49)	64/40 (48)	88/58 (68)	40/30 (33)	NA	101/55 (71)	158/82 (106)	92/57 (70)	86/52 (63)
SpO ₂ , %	93	93	95	100	NA	100	100	100	100
Temperature, °C	35.0	35.2	34.8	31.2	NA	31.4	32.5	32.5	33.0
Arterial blood gas									
pH	7.0	7.1	7.2	NA	7.1	NA	NA	NA	7.2
pCO ₂ , mm Hg	55.3	50.8	37.0	NA	55.3	NA	NA	NA	47.5
pO ₂ , mm Hg	130.1	113.0	71.3	NA	165.0	NA	NA	NA	88.0
Bicarbonate, mEq/L	14.2	14.4	14.4	NA	17.6	NA	NA	NA	14.8
Base deficit, mEq/L	15.1	15.3	12.1	NA	10.3	NA	NA	NA	14.0
Oxygen saturation, %	95.4	95.8	92.5	NA	94.0	NA	NA	NA	93.0
Whole blood profile									
Hemoglobin, g/dL	5.8	6.0	7.4	NA	7.6	NA	NA	NA	8.6
Sodium, mEq/L	135	137	145	NA	149	NA	NA	NA	153
Potassium, mEq/L	4.1	4.2	3.6	NA	3.5	NA	NA	NA	3.6
Ionized calcium, mEq/L	0.6	0.8	0.7	NA	0.8	NA	NA	NA	1.0
Glucose, mg/dL	376	345	259	NA	247	NA	NA	NA	209
Lactic acid, mmol/L	15.3	14.6	7.6	NA	11.9	NA	NA	NA	11.4

Anesthetic intervention	Ongoing massive blood transfusion and volume replacement
50 mg ketamine IV, 2 mg lorazepam IV, isoflurane at 0.5 MAC, 1 g calcium chloride, 2 g cefazolin IV	Phenylephrine infusion (50-200 µg/min), norepinephrine infusion (2-20 µg/min), 1,000 mL 5% albumin, 100 mEq/L sodium bicarbonate IV, 1 g calcium chloride IV
Insulin infusion (2 U/h), 100 mEq/L sodium bicarbonate IV, 1 g calcium chloride, 1 g cefoxitin IV	1,000 mL 5% albumin, 1 g calcium chloride IV
Vasopressin infusion (2.4 U/h), 100 mEq/L sodium bicarbonate IV, 1 g calcium chloride IV	

Table 1. Timeline of Critical Events

Abbreviations: BP, blood pressure; ICU, intensive care unit; IV, intravenous; MAC, minimum alveolar concentration; NA, not available; OR, operating room; SpO₂, oxygen saturation measured by pulse oximetry.

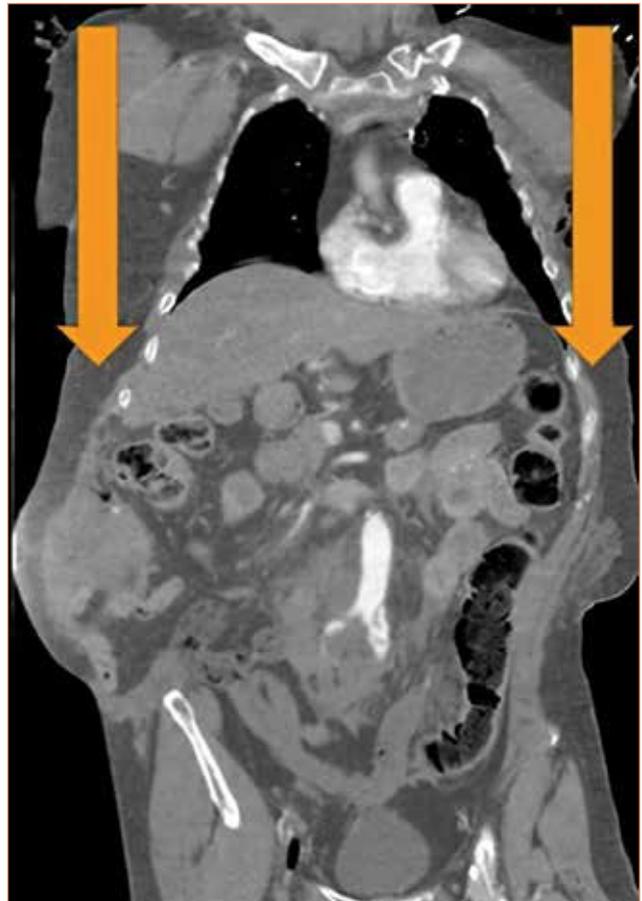


Figure 5. Anterior Coronal View of Irreparable Abdominal Wall Disruption Leading to Continued Postoperative Hemorrhage

transfusion and fluid resuscitation was ongoing from the time the patient arrived in the ED until her death in the ICU. *Massive transfusion* is defined as a blood transfusion of more than 10 units of PRBCs in less than 24 hours or 4 units within 1 hour.¹⁵ As evidenced by current guidelines for Advanced Trauma Life Support (ATLS) and military medicine, the administration of PRBCs, FFP, and platelets should be maintained at a 1:1:1 ratio. Current recommendations suggest that reduced crystalloid use and massive transfusion protocols with whole blood (or 1:1:1 administration in place of whole blood) have proven benefits in improving trauma outcomes.¹⁵ Furthermore, it is suggested that the benefit of preventing early hemorrhagic death far outweighs the risk of transfusion-related acute lung injury from massive whole blood transfusion. However, whole blood use in a civilian hospital is rare, and the availability of blood products to maintain a 1:1:1 ratio can vary by institution. A risk-vs-benefit analysis must also be taken into consideration. During this case, the ratio was not consistent with this recommendation because of the institution's massive transfusion protocol, blood product availability, and risk-vs-benefit analysis from the hematologist overseeing the dispersion of the

Degree of hypothermia	Traditional classification (°C)	Trauma classification (°C)
Mild	32-35	34-36
Moderate	28-32	32-34
Severe	20-28	< 32
Profound	14-20	—
Deep	< 14	—

Table 2. Comparison of Degrees of Hypothermia Between Traditional Classification and Trauma Classification

blood products. Ideally, a 1:1:1 or 1:1:2 approach would have been maintained for this patient to be consistent with current ATLS recommendations, but in the setting of the catastrophic injury complex, the authors do not believe that this negatively affected the patient's likelihood of survival.

• **Coagulopathy.** Acute traumatic coagulopathy is a systemic phenomenon present in 25% to 35% of patients who have sustained a severe injury with moderate to severe hemorrhage on arrival at the ED. Acute traumatic coagulopathy is associated with higher transfusion requirements, longer hospitalization, prolonged mechanical ventilation, and greater incidence of multiorgan dysfunction. A prolonged prothrombin time (PT) and/or activated partial thromboplastin time (aPTT) before resuscitation is associated with a 300% to 400% increase in mortality compared with those without coagulopathy. Death is 8 times more likely within the first 24 hours after injury in those with substantial coagulopathy.^{14,16-18}

In addition to whole blood administration, Rossaint et al⁹ advocated for the use of fibrinogen concentrate and/or prothrombin complex concentrates because they found increased efficacy in reducing mortality by achieving a target fibrinogen level of 1.5 to 2.0 g/L. Fibrinogen is an integral protein used during hemostasis by strengthening clot formation, and its metabolism is often accelerated by acidosis secondary to hemorrhage. This derangement ultimately accelerates fibrinogen breakdown. Furthermore, hypothermia inhibits fibrinogen synthesis.¹⁹ Often overlooked, fibrinogen is among the first of the blood components to reach suboptimal levels during acute traumatic coagulopathy.²⁰ Current recommendations suggest that fibrinogen should be administered during a massive transfusion protocol (50 mg/kg) for every 6 units of PRBCs. Standard laboratory results and thromboelastogram analysis should be part of the coagulopathy management.^{21,22} Prothrombin complex concentrate is a concentrate of vitamin K–dependent clotting factors (II, IX, and X) indicated to treat hemophilia B and to reverse the effect of vitamin K antagonists. Experimental data indicate that prothrombin complex concentrates may be effective in reversing trauma-induced coagulopathy; however, there is some risk associated with the administration of prothrombin complex concentrates, including thromboembolic events leading to acute myocardial infarction, pulmonary embolism, or cerebrovascular accident.²³

The lethal triad of this patient began on initial injury in the prehospital setting. Vascular endothelial disruption, acute hemorrhage, and environmental exposure were all factors leading to the onset of hypothermia, acidosis, and coagulopathy. The administration of tranexamic acid by the flight team was the first step of many in the prevention of worsening coagulopathy. Intraoperative monitoring of platelets, PT, aPTT, fibrinogen, and a thromboelastogram resulted in prompt management of worsening coagulopathies, with the administration of platelets, cryoprecipitate, and factor IX. As previously stated, whole blood administration or a 1:1:1 ratio of blood products would have been ideal but was not feasible for this case.

• **Emerging Trends in Trauma.** Resuscitation of trauma patients has historically involved volume resuscitation and surgical intervention. The US conflicts in the Middle East and Southeast Asia have exposed modern military medicine to more than 18 years of forward surgical trauma care. This has resulted in an unprecedented amount of research and emerging trends in civilian trauma care. As related to this case study, there exists the possibility that fatal traumatic injuries will eventually become easily repaired with early and deliberate intervention.

Controlled hypothermia has been investigated as a potential treatment for patients experiencing traumatic cardiac arrest. An emerging concept that has surfaced in recent years is “suspended state animation”—a lifesaving modality involving the immediate preservation of the heart and brain under a hypothermic state during exsanguination-induced cardiac arrest with delayed resuscitation.¹³ Although most studies have used canine and bovine models, UPMC Presbyterian Hospital in Pittsburgh, Pennsylvania, has initiated trials in humans. Known as emergency preservation and resuscitation for cardiac arrest from trauma, this modality involves intentionally exsanguinating blood volume from an individual with uncontrolled lethal hemorrhage, via a large aortic cannula and into an extracorporeal reservoir. The blood volume is replaced with cold saline to induce a deep hypothermic state (10°C), which provides valuable time for the surgical team to repair the bleeding source. Following repair, the patient is rewarmed to 37°C while being resuscitated with blood products. The population cohort comprised patients who sustained penetrating wounds leading to cardiac arrest and of whom had only

a 7% chance of being resuscitated after losing approximately 50% blood volume.²⁴ Although the study remains in progress, the concept is promising for the management of otherwise lethal trauma injuries.

Rapidly gaining popularity, resuscitative endovascular balloon occlusion of the aorta (REBOA) has been shown to be successful in quickly mitigating exsanguination from traumatic noncompressible torso hemorrhage by occluding the aorta. The occlusive balloon catheter is rapidly deployed via a femoral sheath and placed in 1 of 3 zones to prevent hemorrhage distal to the balloon. Inflation of the endovascular balloon occludes the region proximal to the site of vascular injury and maintains central aortic pressure in the presence of hemorrhagic shock. Because of the ability to rapidly mitigate the source of hemorrhage, endovascular occlusion has improved efficacy in reducing mortality rates compared with open thoracotomy.²⁵ This method is not limited to vascular surgeons. It is being increasingly used by emergency physicians, intensivists, prehospital care providers, and military special operations surgical teams around the world. The US Air Force Special Operations Surgical Team published a case study in which they revealed the first known use of REBOA in an out-of-hospital combat environment.²⁶ In a 2-month period, 4 patients presented with penetrating torso trauma, hemoperitoneum, and hemodynamic instability in extremis. Handheld ultrasound guidance was used to insert the REBOA catheters, which were inflated in the aorta. This technique resulted in a 100% survival rate while achieving transport of patients to the next level of care in a hemodynamically stable condition.^{26,27}

As evidenced in this case report, balloon occlusion of the distal aorta was integral in maintaining hemostasis and central aortic pressure in this acutely hemorrhaging patient. In contrast to the method in which emergency resuscitative REBOA is deployed after immediate recognition of acute internal hemorrhage, the balloon used in this case was inserted during the surgical phase. As the concept of REBOA is further integrated into civilian practice, the authors hope to see an evolved culture where this lifesaving technique is performed expeditiously in the prehospital or ED setting, thus limiting hemorrhage and further progression of the lethal triad.

Conclusion

Traumatic aortic rupture is an extremely fatal injury complex in trauma medicine, especially when compounded with the lethal triad of hypothermia, metabolic acidosis, and coagulopathy. Prompt recognition of hemodynamic instability in extremis in the setting of thoracoabdominal trauma begins in the prehospital setting and extends into the ED with an immediate surgical consult. Rapid triage, based on mechanism of injury and underlying signs and symptoms, can lead to early surgical intervention and minimize the primary and secondary

injuries associated with TAR. Despite a remote accident scene, the quick team approach provided the patient in this care report with optimum conditions for a chance at injury repair and survivability, although she ultimately died of uncontrollable hemorrhage due to mass vascular and abdominal wall injury. As emerging trends continue to evolve and become mainstream for rural hospital settings, the trauma disease process will undoubtedly become less lethal. Until then, the authors encourage fellow anesthesia providers to remain vigilant of the lethal triad while understanding the pathophysiology and management of hypothermia, acidosis, and coagulation.

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