



Non-accidental trauma causing inferior vena cava and liver injuries

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ABSTRACT

Child abuse, or non-accidental trauma (NAT), resulting in concomitant hepatic and inferior vena cava (IVC) injuries is exceedingly rare. No cases of survival after physical abuse giving rise to concurrent hepatic and IVC injuries have been reported in the current literature. This disturbing case details the presentation and hospital course of a 3-year old male who sustained grade V hepatic injury and disruption of the IVC secondary to NAT. The patient was referred from another hospital for evaluation and management of altered mental status and evolving hemodynamic instability. Large volume fluid was discovered on abdominal ultrasound, suggestive of hemoperitoneum. Subsequent comprehensive trauma survey raised suspicion for physical child abuse, and computed tomography (CT) scan revealed a grade V hepatic injury with complex IVC disruption. Mass transfusion protocol (MTP) was activated, and the patient underwent immediate damage control laparotomy. Coagulopathy, acidosis, and hypothermia were corrected within 4 hours of surgery. Two subsequent abdominal explorations were performed over the next 36 hours. Despite massive hemorrhage, disseminated intravascular coagulopathy (DIC), and respiratory failure, the patient survived and achieved complete neurologic recovery. The National Trauma Data Bank (NTDB) was queried for similar hepatic and caval injuries after child abuse, and only one patient was found who did not survive.

1. Introduction

Child abuse is a pervasive societal problem that often presents with delay and occult, life-threatening injuries. One of the rarest and most life-threatening injury patterns is hepatic laceration combined with IVC disruption.

2. Case report

A three-year old male was brought to an outside hospital after reportedly collapsing at home and having two seizures, which Emergency Medical Services witnessed. Born healthy at term, the child had normal past medical history and development. At the other hospital, the patient was reported to be post-ictal but awake and interacting appropriately. The patient was accepted as a Medical Transfer to our pediatric tertiary care center.

2.1. Emergency Department course

Upon arrival in the Emergency Department at our pediatric tertiary care center, the patient was hypothermic, with a blood pressure of approximately 120/70 mm Hg and pulse of 120 beats per minute (bpm). Hemoglobin and hematocrit were 9.5 g/dL and 30%, but the child had a severe lactic acidemia (pH of 7, lactate 11 mmol/L).

Thirty minutes after his arrival, the child became hypotensive and tachycardic, with blood pressure and heart rate of approximately 60/30 mm Hg and 160 bpm, respectively. Subsequent intravenous (IV) bolus of 2 L of normal saline did not raise the patient's blood pressure; an epinephrine drip was initiated. Repeat blood gas demonstrated declining hematocrit (17%) and normalizing pH. The patient's acute hypotension and newly distended abdomen prompted an immediate Focused Assessment with Sonography in Trauma (FAST) exam, which revealed large volume intraperitoneal fluid. Laboratory findings and hemodynamic lability were indicative of hypovolemic shock secondary

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to suspected internal hemorrhage. At this time, the patient was converted from a Medical Transfer to a Level 1 Trauma. Coagulation profile and hepatic function tests later confirmed a consumptive coagulopathy and liver injury, yielding International Normalized Ratio of 9.8 and Partial Thromboplastin Time of 158.4 seconds, with significantly elevated AST (1617 U/L) and ALT (994 U/L).

Mass transfusion protocol (MTP) was activated, resuscitating the child with fresh frozen plasma (FFP), packed red blood cells (pRBCs), and platelets (PLTs). The Pediatric Trauma Service was called immediately for further evaluation and management.

In the resuscitation/trauma bay, the patient received 2 units of trauma-designated blood and was pre-treated with ketamine and rocuronium preceding intubation. The patient's blood pressure stabilized at 121/105 mm Hg, but he remained tachycardic and tachypneic with an oxygen saturation of 94% and temperature 97.7 °F. Primary trauma survey revealed blood in the oropharynx and nasopharynx, petechiae on the buttocks, scattered healing bruises on the extremities, and Glasgow Coma Score (GCS) of 6 with impaired pupillary light reflex on the right. No obvious signs of external abdominal trauma were detected, but the patient's abdomen was now firm and distended.

Computed tomography (CT) of the head was unrevealing. CT of the abdomen and pelvis visualized multiple liver lacerations, including a large intrahepatic laceration of the right lobe which extended to the suprahepatic IVC (Fig. 1A and B). In addition to evidence of massive hemoperitoneum and shock bowel, CT demonstrated a healing left clavicular fracture and healing bilateral fractures of ribs five through seven. Diffuse, bilateral ground glass opacities consistent with Acute Respiratory Distress Syndrome (ARDS) were appreciated further on CT.

The patient was admitted temporarily to the Pediatric Intensive Care Unit (PICU) for better resuscitation in the context of abusive, blunt-force abdominal trauma yielding complex hepatic and IVC lacerations. Blood products administered pre-operatively in the PICU included 1 unit of pRBCs and 1 unit of FFP.

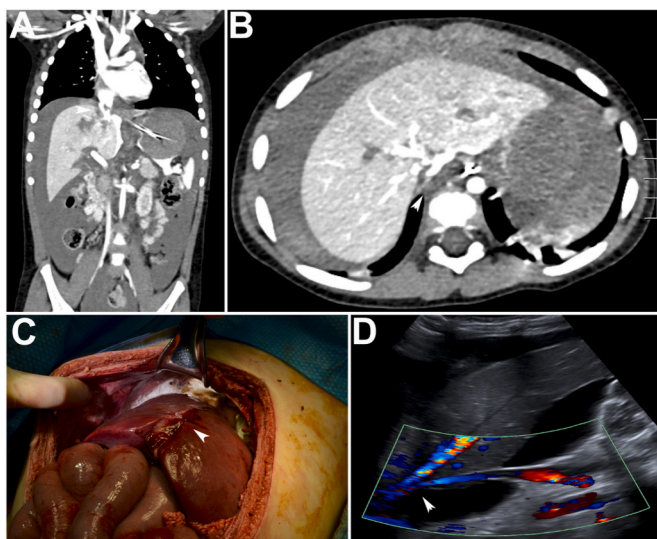


Fig. 1. A-D. (A, B) Computed tomography at presentation shows disruption of the hepatic vein confluence with the inferior vena cava (arrow heads). Note extensive hemoperitoneum and retroperitoneal hematoma. (C) Intraoperative photograph at time of definitive abdominal closure ~36 hours after admission. Arrowhead denotes laceration along line of Cantlie. Final packing with dense oxidized regenerated cellulose. (D) Ultrasound one week after abdominal closure shows patency of hepatic vein confluence with inferior vena cava and no extravasation.

2.2. Damage control laparotomy

After receiving volume resuscitation, the child was taken emergently to the operating room (OR) for damage control laparotomy and placement of central venous access. Upon opening the abdomen, it was confirmed that the child was actively and rapidly bleeding into the peritoneum without any evidence of clotting. Laparotomy sponges were packed into all four quadrants to slow intraperitoneal hemorrhage, then removed one at a time, starting sequentially with the lower quadrants, followed by the left upper quadrant. Bleeding from the pelvis, gastrointestinal tract, and spleen was excluded, and no signs of mesenteric or bowel ischemia were appreciated. A large anterior liver laceration was visualized, although this site was bleeding minimally. Attempts to remove packing from the right upper quadrant (RUQ) were foiled by profuse bleeding from superior/posterior liver laceration, which could not be visualized clearly. Extension of the incision and attempted visualization of the suprahepatic IVC was deferred due to continued hemorrhage causing poor visualization and hemodynamic instability of the patient. The hemorrhage from the RUQ was relatively controlled with packing, but the patient was becoming increasingly hypotensive, coagulopathic, hypothermic, and acidotic. The decision was made to leave thirteen sponges packed around the liver, place an Abthera wound vac connected to suction, and leave the abdomen open until the patient could be stabilized for second-look surgery. Estimated blood loss (EBL) for the operation was 2000 mL and the patient was transfused 6 units of pRBCs, 3 units of FFP and 1 unit of superpacked PLTs.

The patient was returned to the PICU, where coagulopathy, acidosis, and hypothermia were corrected within 4 h of surgery. Resuscitative interventions over the next 15 h included transfusion of 10 units of pRBCs, 6 units of FFP, 3 units of PLTs, 1 packet of cryoprecipitate, multiple boluses of normal saline, IV Vitamin K, and inotropic and vasopressor support with epinephrine drip. The patient was observed to have multiple myoclonic movements and was initially unresponsive in the PICU, but his level of consciousness improved throughout the day, eventually requiring sedation.

2.3. Re-exploration

Approximately 15 h after damage control laparotomy, the patient returned to the OR for planned abdominal washout, repacking, and wound-vac change. The remaining thirteen laparotomy sponges in the RUQ were soaked with saline and removed slowly and cautiously from around the liver. However, gentle traction on the liver led to bleeding, which appeared to be originating from the superior portion, as observed in initial damage control laparotomy. Bleeding was controlled by immediate application of direct pressure and repacking with Fibrillar, Surgicel, and sponges around the liver. The patient's wound vac was replaced at this time. Intraoperatively, he required 2 units of pRBCs, 1 unit of FFP, 1 unit of PLTs, 1 packet of cryoprecipitate, 900mL of crystalloid fluids, and intermittent epinephrine drip. His bowel appeared well-perfused throughout.

The child was transported to the PICU with plans to return to the OR the following day. Over the course of the next 24 h, he required continued epinephrine support and received additional IV crystalloid fluids, 3 units of FFP, and 2 units of PLTs.

2.4. Third exploratory laparotomy

After 17 h, the patient was returned to the OR where laparotomy sponges were removed without concurrent bleeding. During this last operation, the thirteen laparotomy sponges were removed carefully, one by one, until the last one remaining was up over segment eight of the liver. This final laparotomy sponge was removed gently and with great care so as not to disturb the oxidized cellulose between the

sponge and the IVC. Further packing was applied around the IVC and the superior aspect of the liver (Fig. 1C).

Repair of the suprahepatic IVC was not performed because no evidence of ongoing bleeding was encountered. Visualization and repair of this patient's retrohepatic injury would have required mobilization of the entire liver, possibly entering the chest, and may have required extracorporeal bypass to repair. No evidence of hepatic congestion or impaired venous return was evident. The risks of mobilizing the healing liver with controlled bleeding at this point seemed to outweigh the potential benefits of pursuing suprahepatic control of the IVC; therefore, the decision was made to preserve the clotting that had already occurred by proceeding with definitive abdominal closure. Hemodynamic stability and hemostasis were confirmed before the abdomen was irrigated with warm saline and closed without tension or wound vac replacement. Primary fascial closure was achieved. The patient had minimal blood loss during this operation and maintained hemodynamic stability throughout.

2.5. Post-operative course and outcomes

After surgery, the patient returned to the PICU where he required continued ventilatory support and epinephrine drip for approximately 24 hours. Chest x-ray indicating right pleural effusion and fluid warranted gentle diuresis with furosemide to prevent further respiratory failure secondary to hemorrhagic shock. Over the course of the day, the child was tolerating continuous positive airway pressure with pressure support (CPAP/PS) trials and was able to be switched to nasal canula.

Approximately 48 hours after surgery, magnetic resonance imaging (MRI) of the brain showed a small infarct of the right parietal lobe. Abdominal distension increased and caused a decline in renal function and respiratory status. Ultrasound performed four days after final abdominal closure revealed a patent IVC and large volume ascites (Fig. 1D). The child's distention and respiratory status improved with paracentesis, which removed 1.6 L of serosanguinous fluid and led to marked improvement in the patient's renal function and respiratory status. The patient required intermittent epinephrine support for three days following abdominal closure.

Neurologically, the patient exhibited communication vulnerability and impairment along with minimal use of expressive language. Improvement in each of these deficits progressed upon re-evaluation of the patient the three days later. The patient was discharged from the hospital two weeks after initial presentation and was found to have made a full neurologic recovery at one-month follow-up. At this appointment, the patient had no signs of scleral icterus or jaundice and was reported to be eating and having pigmented stools daily.

3. Discussion

Grade V liver injuries are exceptionally rare in pediatric blunt NAT. These injuries are often associated with high-speed deceleration from other mechanisms and are not commonly encountered following blunt abdominal trauma in children. The life-threatening nature of the injury and highly time-sensitive nature of surgical management make this injury challenging to treat. An insidious, delayed presentation of a Grade V liver injury made early detection and intervention more challenging in this case. Favorable outcome was even less likely in this case because hepatic injury was not initially suspected or evident at the outside hospital from which the child was transferred. Even at a comprehensive Level 1 Pediatric Trauma Center, the odds were stacked against his survival.

Our institution is an American College of Surgeons (ACS)-verified Level 1 Pediatric Trauma Center, with twenty-four-hour coverage by pediatric surgeons and expedient availability of surgical subspecialists [2]. In addition to having a pediatric surgeon on-call 24 h per day,

this institution also has a dedicated pediatric trauma team. The factors that ultimately led to the successful resuscitation and surgical management of this patient include but are not limited to OR availability, established pediatric MTP, and institutional solid organ injury protocols. The institution's evidence-based solid organ injury protocols are based off of recommendations from the American Pediatric Surgical Association (APSA) and include specific liver injury protocols.

The steps taken in the first twenty-four to 48 h to allow this child to overcome his life-threatening injury began with the exceptional clinical instincts and immediate action of the emergency medicine physicians who initiated his care. When the patient arrived hypothermic and subsequently became hypotensive, no time was wasted in initiating resuscitative and diagnostic efforts, beginning with a FAST exam. Once intraperitoneal fluid was visualized on FAST exam, immediate conversion to Level 1 Pediatric Trauma appropriately elevated the resources and personnel dedicated to the care of this patient. Additionally, the weight based MTP in place at this institution was activated without delay.

The pediatric surgeon on-call with the pediatric trauma team took the patient to the OR immediately. Not only was the short time to the OR an important factor in stabilizing the patient, but the damage control approach taken in the operating room ultimately saved his life. One study cited a 51.3% mortality rate among adult patients with Grade IV/V liver injury undergoing early damage control laparotomy with perihepatic packing [3]. As compared to the 67–80% [4–12] mortality rates cited in literature for operative management of the same injury, damage control laparotomy has been widely-accepted as the superior surgical approach for Grade V liver injury with exsanguination from the IVC. Factors predictive of mortality in the adult patient population include lower GCS score, lower systolic blood pressure, lower pH, higher revised trauma score (RTS), and time from scene to ICU admission. The association between shorter time from ER to OR and lower mortality approaches statistical significance ($p = 0.08$) and likely would have demonstrated statistical significance had the sample size been larger [3].

Ultimately, it was the seamless cooperation of skilled interdisciplinary teams that allowed for the success of these life-saving measures. Pediatric Emergency Medicine, Pediatric Trauma Surgery, Pediatric Anesthesia, and Pediatric Critical Care teams exemplified the value of expedient, but precise communication and dynamic engagement within an established trauma-response system. These teams prioritized fast and effective resuscitation in preparation for damage-control surgical approach and longer-term stabilization at the ICU level.

Having the appropriate resources, protocols, and interdisciplinary teams in place to care for a critically ill child overnight allowed this child to survive against all odds with exceptional outcome. Despite massive hemorrhage and an initial GCS of 6, the patient achieved outstanding neurologic outcome. Even with extreme hypovolemic shock and coagulopathy, the child's bowels remained adequately perfused throughout and his ARDS resolved in time, allowing for him to be extubated. This child's grave condition called for seamless teamwork and collaboration within this medical community. All possible resources were exhausted in the unified mission to keep this young patient alive. He has proven through his remarkable outcome that every effort should be made to control intraperitoneal hemorrhage from Grade V liver injury in a child.

To understand the frequency and associated mortality with this constellation of hepatic and caval injuries after child abuse, we chose to query the NTDB [1]. This national and comprehensive trauma database archived 22,489 incidences of child abuse in patients <19 years of age from 2010 to 2017, among whom 326 had liver injuries and a 4.9% mortality rate. Only one child was registered in the NTDB who sustained concurrent hepatic and IVC injuries. She did not survive.

4. Conclusion

Child abuse resulting in concomitant hepatic and IVC injuries is exceedingly rare. Excellent cardiopulmonary and neurologic outcome is possible in a pediatric patient in hypovolemic shock complicated by DIC and ARDS secondary to blunt NAT to the abdomen. Damage control interventions and resuscitation protocols are indicated to achieve best survival with favorable neurologic outcome.

Patient consent

Consent to publish the case report was not obtained. This report does not contain any personal information that could lead to the identification of the patient.

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Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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