

CASE REPORT

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Pediatric liver trauma from a bicycle handlebar managed with interventional radiology: a case report

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Abstract

Blunt abdominal trauma in pediatric patients can result in significant internal organ injury due to their smaller body mass and less protective musculature. We report the case of a 12-year-old female who sustained a Grade IV liver injury with hemoperitoneum following a bicycle handlebar impact. She presented with hemodynamic instability and active arterial bleeding confirmed on arterial phase contrast-enhanced computed tomography of the abdomen and pelvis and angiography. Timely imaging and multidisciplinary coordination led to successful management via transarterial embolization (TAE) of the segment VIII hepatic artery. The patient recovered well and was discharged on the 11th day. This case highlights the importance of early recognition, imaging, and interventional management in pediatric hepatic trauma.

Keywords: Bicycle handlebar; Interventional Transarterial Embolization; Multidisciplinary coordination; Pediatric blunt abdominal trauma

INTRODUCTION

Pediatric blunt abdominal trauma poses unique challenges due to anatomical and physiological differences from adults.¹ The role of Interventional Radiology in pediatric trauma is less defined.¹ The liver is one of the most commonly injured organs, and high-grade injuries can lead to life-threatening hemorrhage. While conservative management is preferred in stable patients, transarterial embolization (TAE) has emerged as a critical intervention in cases of active arterial bleeding despite blood transfusion.² This case illustrates the successful use of TAE in a child with Grade IV liver injury, emphasizing the role of multidisciplinary care.

CASE REPORT

A 12-year-old female studying in grade VII was referred to our tertiary care center from a provincial hospital with complaints of right upper quadrant abdominal pain lasting five hours following a bicycle accident. She had sustained a direct impact on her right lower chest from the bicycle handlebar after falling onto a gravel road. After resting at home for approximately 30 minutes, her pain intensified, prompting evaluation at a nearby hospital and subsequent referral. On arrival, her primary survey revealed a patent airway without cervical spine tenderness. She was tachypneic with a respiratory rate of 26 breaths per minute and maintained an oxygen saturation of 99% on room air. Bilateral breath sounds were clear. She was tachycardic with a pulse rate of 148 beats per minute and hypotensive with a blood pressure of 90/60 mmHg. Capillary refill time was delayed at more than 3 seconds. Neurologically, she was alert with a Glasgow Coma Scale score of 15/15 and had round, regular, reactive pupils bilaterally. Her random blood sugar was elevated at 256 mg/dL, and no focal neurological deficits were noted. Physical examination revealed tenderness over the right hypochondriac and epigastric regions. A crescent-shaped bruise measuring 3 x 3 cm was present over the right lower chest, consistent with the impression of a handlebar. Her body temperature was within normal limits. An Extended-Focused Assessment with Sonography for Trauma (E-FAST) scan performed at the referring hospital showed a large heterogeneous intraparenchymal collection (~200 ml) in the right lobe of the liver, suggestive of hepatic laceration with intraparenchymal hematoma. Additionally, a crescentic hypoechoic subcapsular collection (~28 ml) was noted on the surface of the right lobe, consistent with a subcapsular hematoma. Minimal hemoperitoneum was also observed.

Two large-bore intravenous cannulas were inserted, and resuscitation was initiated with intravenous crystalloid (Ringer's lactate) at a dose of 20 ml/kg. All preoperative investigations, including blood cross-matching, were completed. The case was promptly communicated to the pediatric surgery unit, and the patient was shifted for contrast-enhanced computed tomography (CECT) of the abdomen and pelvis. The CECT revealed multiple

hepatic lacerations and contusions with foci of contrast extravasation, consistent with an American Association for the Surgery of Trauma (AAST) Grade IV liver injury (Figure 1 and 2).



Figure 1. Arterial Phase of CECT Abdomen & Pelvis showing extravasation of contrast in the arterial phase of CT (brown arrow), which is situated in the segment VIII in the subcapsular region (brown arrow)

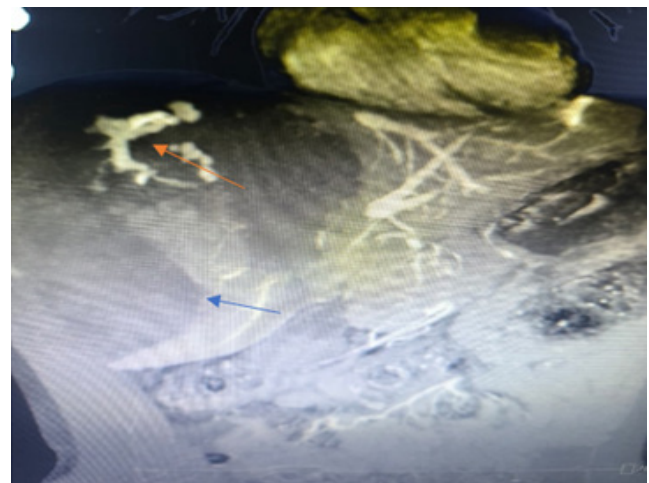


Figure 2. Showing Venous Mip Image of CECT Abdomen & Pelvis showing Extravasation of blood (brown arrow) and large subcapsular hematoma; compressing liver parenchyma (blue arrow).

Blood resuscitation was initiated in the emergency department following a significant drop in hemoglobin from 11.2 g/dL to 5.7 g/dL. The patient received one pint of whole blood during initial stabilization. A multidisciplinary discussion was held between the pediatric surgery team, interventional radiology, and the patient's family. Subsequently, the case was transferred to the Biplane Catheterization Laboratory, involving teams from Interventional Radiology, Emergency Medicine, Anesthesia and Critical Care, Pediatric Surgery, and the Pediatric Intensive Care Unit.

The procedure commenced under general anesthesia at 2:15 AM, with blood transfusion initiated via a central venous line. An intra-arterial line was placed for invasive blood pressure monitoring. During the procedure, the

patient received an additional 1 pint of whole blood and 1 pint of fresh-frozen plasma.

The patient underwent angiography (Figure 3) followed by angioembolization of the segment VIII branch of the hepatic artery using polyvinyl alcohol (PVA) particles measuring 300–500 microns. (Figure 4(A) and 4(B)). Following the procedure, she was transferred to the Pediatric Intensive Care Unit (PICU) under intubated status for continued monitoring and supportive care.

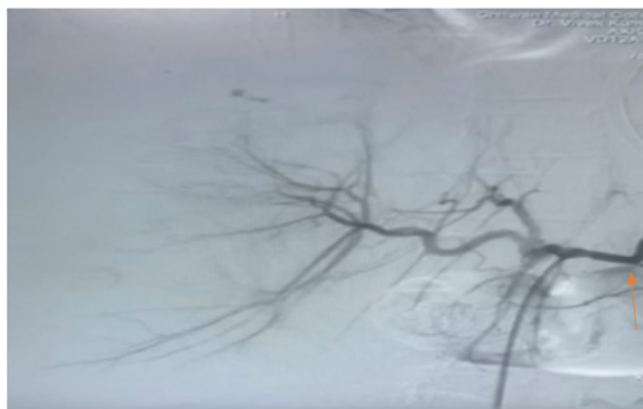


Figure 3. Showing angiographic evaluation showing the celiac trunk (brown arrow)

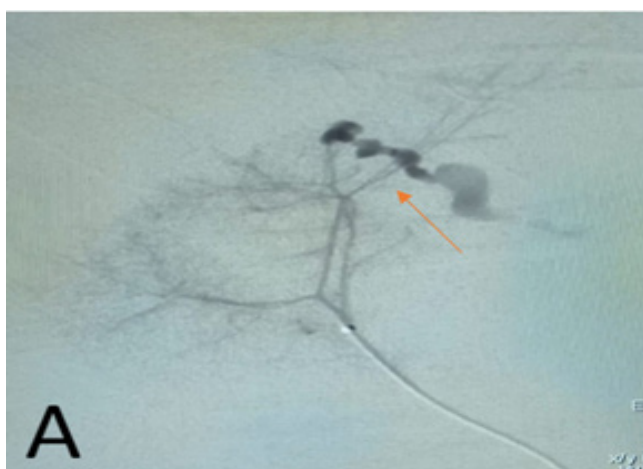


Figure 4(A). Pre-angioembolization; showing active bleeding from hepatic artery (brown arrow)

The patient initially required ventilator support, blood product transfusions, and noradrenaline infusion to maintain hemodynamics. Transfusions included 2 pints of fresh frozen plasma (FFP), 1 pint of packed cell volume (PCV), and 1 pint of platelet-rich plasma (PRP) (Table 1). A pigtail catheter was inserted on the 2nd Post-Operative Day (POD) and drained significant intra-abdominal blood (900ml). By the third day, she was extubated, noradrenaline discontinued, and her neurological status improved (GCS 13/15, later 15/15). Respiratory support was gradually weaned, invasive lines and tubes were removed, and oral intake was resumed. On 7th POD she was shifted from ICU to HDU, then to the pediatric surgery ward on 9th POD. Imaging showed a perihepatic hematoma with early liquefaction, but no major progression, and the pelvic drain was removed.

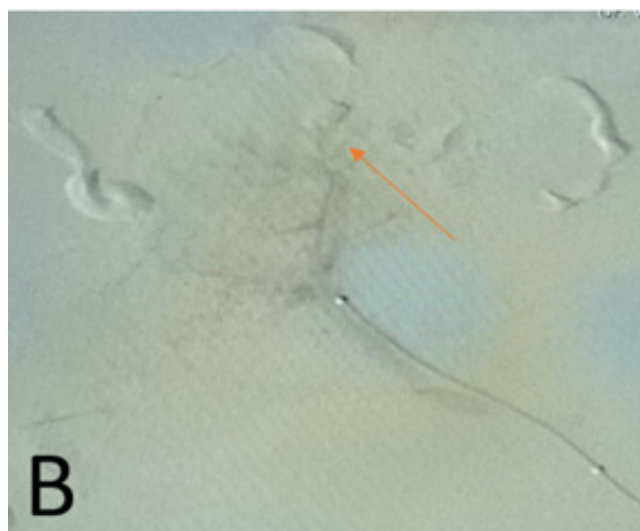


Figure 4(B). Post-angioembolization, no active bleeding (brown arrow)

She tolerated a normal diet, remained hemodynamically stable, and was discharged on oral medications by day 11.

Table 2 summarizes the serial laboratory investigations during the hospital course. On Day 1, the patient presented with severe anemia (hemoglobin 5.7 g/dL, PCV 18.2%) and leukocytosis with neutrophilia, accompanied by thrombocytopenia. Over the following days, hemoglobin and hematocrit showed gradual improvement, reaching normal levels by Day 9 (Hb 13.9 g/dL, PCV 42.3%).

Seven days after discharge, the patient returned to the Pediatric Surgery OPD, accompanied by her mother, wearing a bright, cheerful smile (Figure 5). On follow-up abdominal imaging, a large subcapsular hematoma was noted in the right hepatic lobe, without evidence of ascites. The hematoma was left to resolve spontaneously without active intervention.



Figure 5. Patient on follow-up

Table 1. showing the course of hospital stay

Day	Event
1 st (after procedure)	The patient was managed on ventilator support in PC-BiPAP mode, with FiO ₂ titrated according to oxygenation status. Transfusions included 2 pints of fresh frozen plasma (FFP), 1 pint of packed cell volume (PCV), and 1 pint of platelet-rich plasma (PRP). She experienced three febrile spikes (101.5°F, 100.8°F, and 100.6°F), which were managed with intravenous fluids, IV antibiotics, and supportive care. Noradrenaline infusion was initiated at 1 ml/hour and titrated to maintain a mean arterial pressure (MAP) of 60–65 mmHg.
2 nd	A 10 Fr pigtail catheter was inserted in the right iliac fossa (RIF) into the peritoneal cavity, draining approximately 900 ml of blood.
3 rd	The patient was extubated and placed on CPAP with 10 liters of oxygen. Noradrenaline was discontinued, and the intra-arterial line was removed. Glasgow Coma Scale (GCS) was 13/15 (E3V4M6).
4 th	CPAP was discontinued at 10:00 AM. Oxygen saturation was maintained on room air. Oral sips were allowed.
5 th	GCS improved to 15/15 (E4V5M6). Foley catheter and nasogastric (NG) tube were removed.
6 th	GCS remained 15/15. Liquid diet was initiated, and the central venous pressure (CVP) line was removed.
7 th	The patient was shifted to the pediatric high-dependency unit (HDU). She was maintained on a soft diet, and intravenous fluids were discontinued.
8 th	Ultrasound revealed a perihepatic hematoma with early liquefaction; no significant change in size. Pelvic drain output was < 20ml/hour. The pigtail drain was removed.
9 th	The patient was transferred to the pediatric surgery ward. No new clinical concerns were noted.
10 th	She remained under observation in the pediatric ward and tolerated a normal diet.
11 th	The patient was hemodynamically stable and discharged on oral medications.

DISCUSSION

This case involves a 12-year-old female with a Grade IV liver injury and hemoperitoneum following blunt abdominal trauma caused by a bicycle handlebar. Pediatric patients are particularly vulnerable to such injuries due to their relatively smaller body mass and less protective musculature.³ In a blunt abdominal trauma patient, an elevated blood glucose level reflects the stress response triggered by catecholamines and steroid release.⁴ The patient's blood glucose level was elevated to 241 mg/dL at initial presentation, reflecting a significant stress response. This rise may serve as an early marker of trauma severity, often linked to initial blood loss.⁵ As noted in the Advanced Trauma Life Support (ATLS), the energy from blunt trauma is transmitted over a smaller surface area in children, resulting in greater force per unit area and an increased risk of internal organ damage.³

Initial imaging revealed active contrast extravasation in the arterial phase of a CT scan, localized to segment VIII of the liver in the subcapsular region, suggestive of hepatic arterial bleeding. Angiography confirmed active bleeding from the segment VIII branch of the hepatic artery. Given the patient's hemodynamic instability and significant drop in hemoglobin, urgent angiography followed by angioembolization was performed.

According to the American Pediatric Surgical Association guidelines, therapeutic embolization is indicated in pediatric patients with liver or splenic injuries who show signs of ongoing bleeding despite transfusion of blood products.² In this case, two pints of whole blood and one pint of fresh frozen plasma were transfused prior to the procedure.

There are three potential sources of bleeding in hepatic trauma: the hepatic arterial system, the portal venous system, and the hepatic veins.⁶ Hepatic arterial bleeding may occur into the biliary tree, hepatic parenchyma, or peritoneal cavity.⁷ Unlike venous bleeding, which can often be controlled with mechanical measures such as perihepatic

packing or direct suturing, arterial bleeding is typically more forceful and resistant to such interventions.^{7,8} In unstable patients, hepatic artery embolization is a preferred technique to achieve hemostasis. The success rate of angioembolization varies, with a technical success rate of 94% and a clinical success rate of 68% to 87%.⁶

The decision to proceed with embolization in this case was based on multiple factors: active contrast extravasation on CT, hemodynamic instability, and a “brush” of contrast leakage on angiogram, which resolved following embolization.² Hepatic angioembolization can be performed safely to arrest hepatic bleeding when specialist interventional radiology services are available.⁶

The patient was managed in the Pediatric Intensive Care Unit for six days, followed by transfer to the Pediatric High Dependency Unit. She was discharged on the 11th day, hemodynamically stable.

This is a single-patient case, so findings cannot be generalized. Follow-up was short, and long-term hepatic outcomes were not assessed. Pediatric embolization was technically challenging due to small vessel size and higher complication risk. Radiation exposure from CT and angiography was not evaluated. The management required advanced interventional radiology and multidisciplinary support, which may not be available in all centers.

CONCLUSION

Transarterial embolization is a vital tool in the management of high-grade hepatic injuries in pediatric trauma, especially when accompanied by hemodynamic instability and active arterial bleeding. Early imaging, prompt resuscitation, and multidisciplinary collaboration are key to improving outcomes. This case exemplifies the effectiveness of TAE in pediatric liver trauma and reinforces the importance of integrated trauma care. Her journey reminds us that with coordinated care and clinical vigilance, even the most serious injuries can be met with hope, healing, and strength.

Table 2. Laboratory Investigation of the patient during hospital stay

Laboratory Investigation	Day 1 (05/14)	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 9 (05/22)
Hemoglobin (gm/dl)	11.2 (6 pm) 5.7 (9 pm)	7.9 (6 am) 9.3 (11 am) 7.6 (6 pm)	7.3 (7 am) 7.1 (11 am) 7.1 (8 pm)	9.8 (2 am) 10.6 (9 am)	13.7 (9 am) 12.4 (6 pm)	12.2 (2 am) 12.5 (6 am) 12.6 (2 pm)	13.3	13.9
PCV (%)	32.3 (6 pm) 18.2 (9 pm)	24.4 (6 am) 29.3 (11 am) 23.5 (6 pm)	20.7 (7 am) 22.4 (11 am) 20.6 (8 pm)	30.3 (2 am) 32.3 (9 am)	42.3 (9 am) 38.6 (6 pm)	37.4 (2 am) 35.8 (6 am) 38.9 (2 pm)	38.6	42.3
WBC (/cumm)	17590	14,070 (6 am) 11,780 (11 am)	8600	10320	12790	11630		11000
N/L (%)	80/13	67/23 (6 am) 71.9/19.8 (11 am)	80/13	83.2/8.5	85.5/6.3	77/17		56/34
Platelets (/cumm)	226000	88,000 (6 am) 105,000 (11 am)	86000	100000	120000	115000		228000
Blood Sugar (mg/dL)	RBS:241							
Urea (mg/dL)	37	32	43 (1 am) 42 (7 am)	51	36			
Creatinine (mg/dL)	0.54	0.52	0.51 (1 am) 0.47 (7 am)	0.58	0.46			
Sodium (mmol/L)	142	137.2	139 (1 am) 139 (7 am)	141	139.1			
Potassium(mmol/L)	3.65	4.69	3.55 (1 am) 3.1 (7 am)	3.89	4.84			
CRP (mg/L)		33						
Bilirubin Total (mg/dL)	0.5	0.7	1.6 (1 am) 1.3 (7 am)		1			2
Bilirubin Direct (mg/dL)	0.2	0.2	0.5 (1 am) 0.4 (7 am)		0.3			0.9
SGPT (IU/L)	967	3429	3618 (1 am) 3330 (7 am)		1953			510
SGOT (IU/L)	934	4581	6240 (1 am) 4962 (7 am)		810			84
Alkaline Phosphatase (U/L)	354	219	208 (1 am) 228 (7 am)		276			193
Total Protein (gm/dl)	5.8	4.4	4.6 (1 am) 4.7 (7 am)		5			5
Albumin (gm/dl)	4	2.5	2.9 (1 am) 2.9 (7 am)		2.7			3.5
Serology	Non-reactive							
Calcium (mg/dL)			8					
PT (sec)	17.6	20.5	20.6	18.4				18.7
INR	1.37	1.61	1.62	1.44				1.46

DECLARATIONS**Conflict of Interest**

None.

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Consent

Written Informed consent for publication of this case report was obtained from the patient's mother, as the patient was 12 years old.

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