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Case Report

Experience in Hepatic Artery Embolisation in Paediatric Hepatic Blunt Trauma

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Abstract Non-operative management is the current mainstay of treatment for hepatic blunt trauma unless haemodynamic instability is present. Evidence for hepatic artery embolisation (HAE) for ongoing bleeding in hepatic trauma is well established in adults but there are only several case reports in the paediatric population. We herein report the first paediatric case of isolated grade IV hepatic injury successfully managed with primary HAE at our institution.

Key words Blunt trauma; Hepatic artery embolisation; Hepatic trauma

Introduction

Hepatic trauma is mostly managed conservatively with intensive monitoring and non-operative management nowadays (NOM). Abdominal computerised tomography (CT) scan is a reliable tool to detect ongoing arterial bleeding in high grade hepatic trauma as evidenced by active contrast extravasation.[1,2] In a patient with relatively stable haemodynamics, it is justified to choose hepatic artery embolisation (HAE) over exploratory laparotomy to control ongoing bleeding.[3] HAE is a common adjunct, either primary or postoperative, in adult trauma series but only few reports are available in the paediatric literature.[4-6] We herein present our experience with primary HAE in an isolated American Association for the Surgery of Trauma (AAST) grade IV hepatic blunt trauma.

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Case Presentation

A 7-year-old boy suffered from bicycle handlebar injury to right upper abdomen. He presented to the casualty for persistent abdominal pain after the injury. On presentation, he was haemodynamically stable with an initial haemoglobin of 12.9 g/dL. Focused assessment with sonography for trauma scan was negative. Urgent CT scan with contrast of the abdomen revealed a grade IV hepatic injury according to the liver injury scale of the AAST without active contrast extravasation. He was then admitted to the paediatric intensive care unit for close monitoring. NOM was adopted as initial management approach. He was haemodynamically stable until post-injury day 3 with his haemoglobin dropped to 8 g/dL. On physical examination, persistent right upper quadrant dull pain without sign of ascites or fluid collection. The anemia was only transiently responsive to blood product transfusion. Contrast CT was repeated which showed interval enlargement of acute right subcapsular haematoma with new liver parenchymal haematoma, suspicious of pseudoaneurysm formation over right lobe of liver without any evidence of bile leak or intra-abdominal collection (Figure 1a).

In view of repeated requirement for blood transfusion, HAE of right hepatic artery with Gelfoam was performed (Figures 1b and 1c). Due to the large liver haematoma involvement of the right lobe, embolisation of right hepatic artery was decided instead of supra-selective approach.

A brush of contrast extravasation was seen on pre-procedure angiogram but then disappeared after emobolistion. He did not require further blood products transfusion with a static haemoglobin level of about 12g/dL (Figure 2) after HAE. Diet was resumed gradually and he was transferred to general paediatric surgical ward 3 days later. Empirical antibiotic was prescribed in view of a sizable haematoma. There was a transient derangement of liver parenchymal enzymes after HAE but it soon resolved. No major complication was encountered after the procedure. Follow-up ultrasound study 9 days after the procedure confirmed size of liver haematoma remained static with no perihepatic collection suggestive of bile leak. The patient remained stable and was discharged 2 weeks after the injury with the advice on avoidance from intensive physical activity. One month later he was able to resume his normal daily activity. The follow-up USG showed a gradually resolving haematoma with normal biliary ductal configuration and homogenous liver echotexture. In addition, serial haemoglobin monitoring showed a static level while his liver function tests were completely normalised by 2 months after the injury.

Discussions

The current worldwide standard of care in managing blunt liver injury is toward non-operative strategy whenever

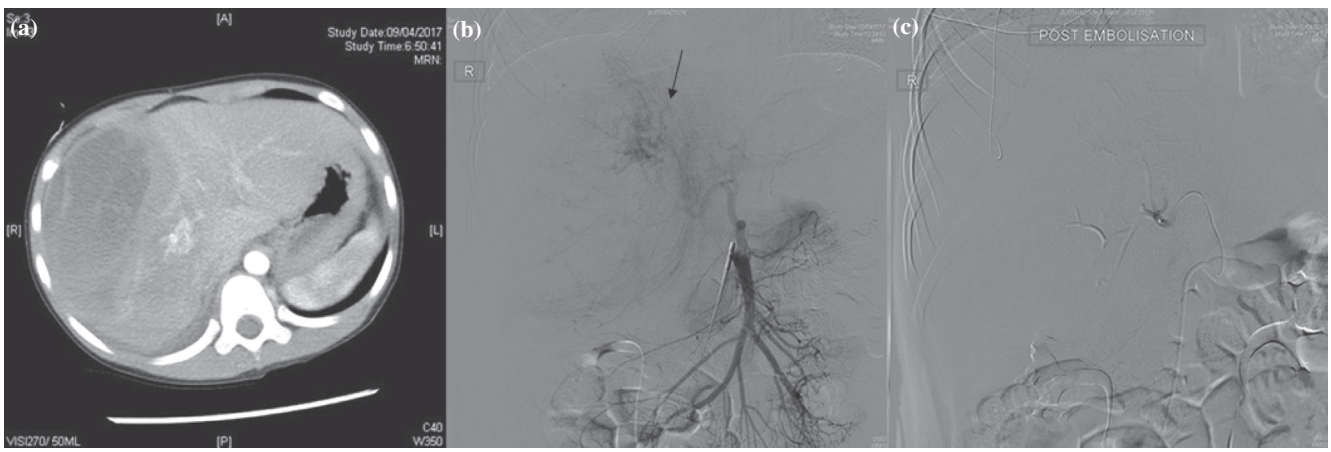

Figure 1 (a) Huge subcapsular haematoma over right lobe displacing the hepatic vessels in pre-procedural CT imaging. (b) Brush of contrast was noted in pre-procedure angiogram as shown by the arrow. (c) Post-HAE angiogram showing no contrast extravasation.

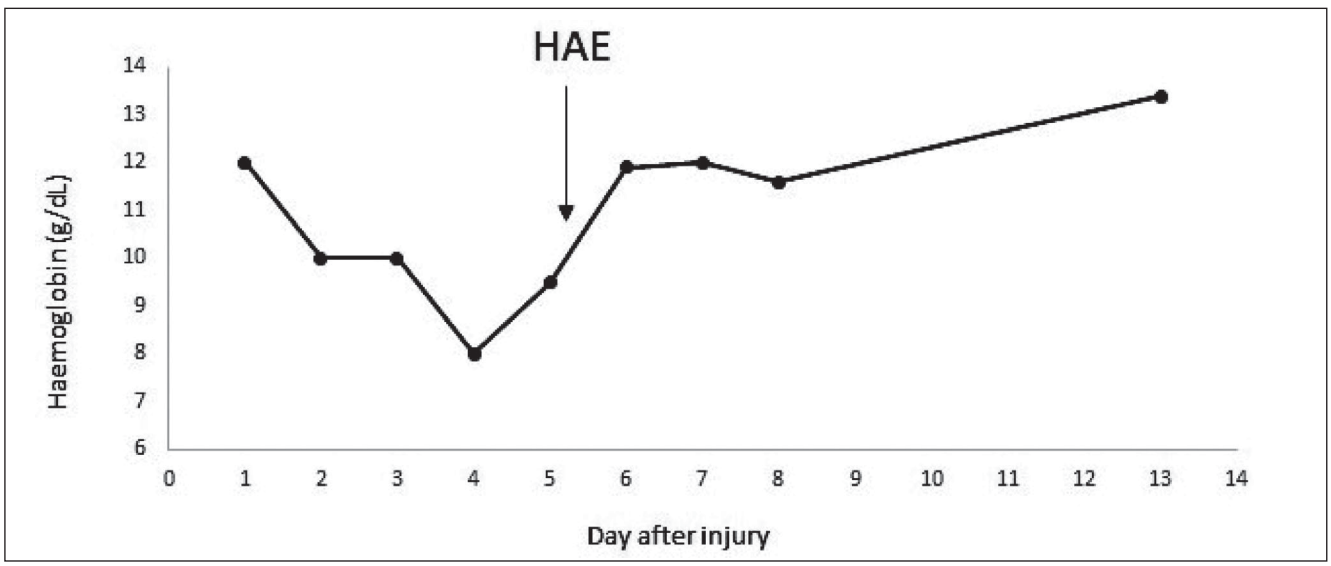

Figure 2 Haemoglobin trend before and after HAE. No further blood transfusion is required after HAE.

possible. Traditional guideline for emergency laparotomy in blunt liver trauma includes transfusion requirement of more than 40 mL/kg or clinical judgement of haemodynamic instability.[1,2] Cessation of hepatic bleeding from venous origin is often accomplished by mechanical pressure such as perihepatic packing or direct suturing of liver parenchyma. On the contrary, bleeding from arterial origin may not be adequately controlled by mechanical pressure; and that is the basis for the implementation of HAE. There are 2 roles of HAE in the management of hepatic trauma (1) as a part of non-operative treatment if angiogram or CT showed active bleeding and (2) when there is evidence of inadequate haemostasis after laparotomy. The potential complications of HAE include puncture site complication, thrombo-embolic event, intra-abdominal compartment syndrome and the most serious event will be hepatic necrosis.[3]

Our institution is a tertiary trauma centre for paediatric population. From 1998 till now, there are 146 paediatric trauma cases admitted to our unit with 52 patients suffering from solid organ injuries. Among the 52 patients, 43% had hepatic injury and only one patient required emergency laparotomy in view of ongoing hepatic bleeding and shock. Our patient was haemodynamically stable on admission but slowly deteriorated after initial resuscitation. In view of major morbidity of laparotomy and his relatively stable condition, he was considered as a candidate for HAE. Our reported case is the first patient in our trauma series to undergo HAE and no major complication was encountered.

The reason of conversion from a conservative strategy to HAE was due to the maintained low haemoglobin level which signified an ongoing bleeding though not to the extent of shock. The criteria adopting selective HAE includes (1) active contrast extravasation on CT scan, (2) decrease in haemoglobin level, (3) transient episodes of hypotension which is responsive to plasma volume expansion, (4) absence of peritonism. However, the timing of implementing HAE is controversial and the decision mainly depends on the attending surgeons after serial clinical assessment and the availability of expertise.

The successful hepatic angioembolisation in our case allowed us to avoid a high morbidity laparotomy and gave new insight in trauma care. Due to paucity of data in the literature, further experience is essential before drawing a clear recommendation and protocol for HAE in the management of hepatic blunt trauma in paediatric population.

Declaration of Interest

All the authors report no conflicts of interest.

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