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Case Report

Successful balloon dilatation for postoperative caval stenosis caused by primary venorrhaphy for traumatic retro-hepatic caval injury in a three-year-old child: Report of a case

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ABSTRACT

Inferior vena cava injuries are highly lethal. We experienced a case of retrohepatic inferior vena cava injury as a result of blunt trauma in a three-year-old female.

Because the site of bleeding of the IVC was identified, we repaired it with running sutures.

An attempt at primary repair resulted in postoperative narrowing of the vena cava. There was pressure gradient of the right atrium and inferior vena cava, and collateral circulation developed.

Since it was also found that the haemodynamics was unstable, the child underwent another intervention before the stenosis of the IVC was fixed.

To the best of our knowledge, there have been no previous reports of therapeutic radiological intervention for stenosis that developed after treatment of a traumatic IVC injury.

The IVC in the present case recovered enough patency so that the collateral venous flow could be decreased after balloon dilatation angioplasty.

Introduction

Inferior vena cava (IVC) injuries caused by blunt trauma are rare, however, they are usually associated with high mortality, especially in children. The optimal management of patients with IVC injuries requires rapid transportation to tertiary trauma centres, aggressive fluid resuscitation, and prompt surgical repair. The prehospital mortality rate for IVC injuries is as high as 36% [1]. For patients that reach the hospital alive, the overall mortality rates range from 20% to 57% [1].

The repair of IVC injuries itself can also carry significant morbidity and mortality, and the deaths are reported to be commonly caused by intraoperative exsanguination. The survival rates for patients with IVC injuries obviously depend on the location of the injury. Infra-renal caval injury is associated with a mortality rate of 25%, whereas injuries between the renal veins and the hepatic veins are associated with a mortality rate of 41–55% [2]. The death rate surpasses 80% at the level of the hepatic veins or the supra-hepatic vena cava [2]. It is generally accepted that children do not tolerate these severe conditions as well as adults, therefore, the paediatric case reports describing the recovery from these lethal injuries have been limited.

We herein present the case of a three-year-old female with a retro-hepatic IVC (RH-IVC) injury caused by blunt trauma, who had survived this lethal injury by undergoing primary venorrhaphy. In this case, the repaired IVC developed postoperative stenosis. The postoperative balloon dilatation angioplasty after IVC repair is also described.

Case report

A three-year-old, previously healthy female, was transferred to a referring institution after suffering direct abdominal compression by the family car which her mother was slowly backing in her residential parking area. The patient was temporarily unresponsive

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after this accident, and her mother initiated basic life support immediately. On presentation to the emergency department, the presence of severe liver injury was suspected. She was transferred to our hospital 2 h after the accident.

At presentation to our emergency department, the patient was severely ill and her vital signs were unstable. She showed tachypnea, a pulse of 190 beats/min, peripheral coldness, and a prolonged capillary refilling time (more than 3 s). Her blood pressure could not be measured. The physical examinations revealed severe abdominal distension, tenderness and muscle guarding. Laboratory findings revealed severe anaemia (red blood cells 2.39×10^6 μL, Hb 7.3 g/dl), elevated aspartate aminotransferase at 515 IU/L and alanine aminotransferase at 398 IU/L. Severe metabolic acidosis (pH 7.219, BE −9.0 mEq/L, HCO_3 16.7 mEq/L) and coagulopathy (31% prothrombin time) were also observed.

Contrast enhanced computed tomography (CT) demonstrated massive haemorrhage in the abdominal cavity, and poor contrast enhancement with contrast extravasation from the branch of the right hepatic artery in the entire part of the right posterior hepatic lobe (Fig. 1). A collapsed IVC was also observed at the level of retrohepatic region. These findings were suspected to have resulted from the presence of an AAST (American Associations for the Surgery of Trauma) grade IV–V liver injury, and immediate laparotomy via an upper abdominal transverse skin incision was initially performed. Although a damage control surgery was scheduled, the presence of juxta-hepatic venous injuries had not been observed until the exposure of abdominal cavity was obtained.

Massive haemorrhage was encountered following laparotomy and the right posterior hepatic lobe proved to be almost completely disrupted from the residual liver.

This disrupted segment showed discoloration, with a complete tear of both the right posterior hepatic artery and the portal vein (Fig. 2). Therefore, resectional debridement of the segment was selected. However, continuous massive bleeding from the RH-IVC was encountered, immediately after mobilization of right posterior hepatic lobe. Two approximately 1 cm-long RH-IVC tears were then noted to be present in addition to the liver disruption. Fortunately, digital pressure could provide immediate vascular control, and subsequently, intestinal Kelly clamps were placed at both the proximal and distal extents of the tears. After this procedure, good temporary vascular control could be obtained, and complete hemostasis was obtained by using running sutures of 4-0 polypropylene for the RH-IVC tears. After the establishment of hemostasis, resectional debridement for the posterior right hepatic lobe was performed. The total amount of intraoperative haemorrhage was 3550 ml. A total of 1400 ml of packed red cells, 1000 ml of fresh frozen plasma and 200 ml of platelet-rich plasma were transfused during surgery.

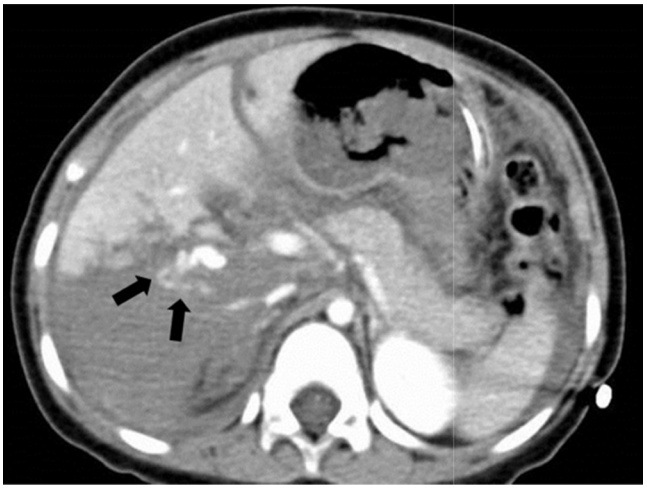

Fig. 1. A computed tomography scan showed massive haemorrhage in the abdominal cavity and poor contrast enhancement, with contrast extravasation from the branch of the right hepatic artery in the entire regions of the right posterior hepatic lobe.

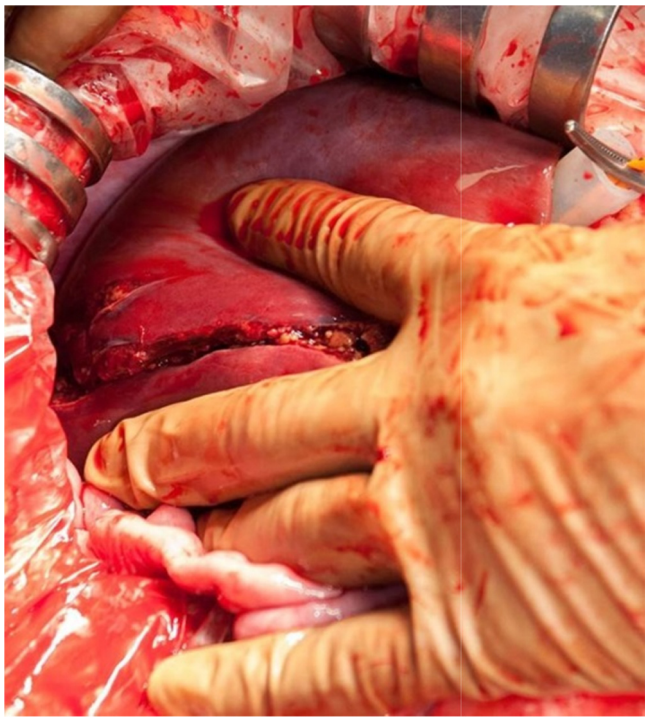

Fig. 2. The right posterior hepatic segment was already dissected and discoloured dark red.

After surgery, the patient received meticulous cardiopulmonary support in the ICU, and her vital signs could be maintained. On the first postoperative day, oliguria and severe abrupt oedema of both lower extremities and the lower trunk emerged. An ultrasound examination demonstrated the inevitable stenosis of the RH-IVC, and this oedema was considered to have resulted from the primary repair for the RH-IVC. After the administration of a diuretic agent, the oliguria and oedema gradually resolved over 2 days. Contrast enhanced CT on the 22nd postoperative day also demonstrated stenosis of the RH-IVC and the dilatation of the azygos and hemiazygos vein. Although the formation of a thrombus was not confirmed at that time, the administration of low dose aspirin (75 mg/day) was initiated to decrease the probability of future thromboembolism. After that, the postoperative course was uneventful, and the patient was discharged on the 25th postoperative day.

After discharge, she occasionally complained of hot flashes at the lower trunk following bathing. Furthermore, she developed distinct venous dilatation of her abdominal surface and minor bruising which also caused the skin to become red. These changes were suspected to have resulted from the development of collateral drainage veins following the stenosis of the RH-IVC. To estimate the degree of the stenosis and the development of collateral veins, an IVC venogram (IVC-gram) was performed in 9 months after the surgery.

The IVC-gram at the level just distal to the stenosis demonstrated the patency of the IVC. The minimum diameter of RH-IVC stenosis proved to be only 2 mm (Fig. 3). However, another IVC-gram at the level distal to the bilateral renal veins revealed that most of the injected contrast material finally drained to both