Delayed Development of a Biloma in an Infant Following Hepatic Injury

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ABSTRACT

A case report is presented of an infant who developed a biloma over three months after major hepatic injury and after almost complete healing. A brief literature review is given to highlight unusual features of this case.

Desarrollo Retardado de un Biloma en un Infante Luego de un Daño Hepático

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RESUMEN

Se presenta el caso de un infante que desarrolló un biloma en tres meses, tras sufrir un serio daño hepático y luego de una curación casi completa. Se ofrece una breve revisión de la literatura a fin de destacar las características poco usuales de este caso.

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INTRODUCTION

Most patients who sustain liver injury cease bleeding spontaneously (1, 2). Recognition of this fact, along with the development of better imaging facilities, has led to the increasing acceptance of non-operative management of hepatic injury. Complications of this selective treatment include intrahepatic collections of blood, infected material and bile (bilomas). The latter are rare, since, unlike extrahepatic injury, intrahepatic bile duct lacerations are unusual sequelae to blunt liver trauma and less than 25 cases had been reported as recently as 2002 (3). Their formation has been recognized at intervals ranging from a few days to several weeks after injury.

An infant recently presented with intrahepatic collections of bile that developed over three months after hepatic injury, following almost complete resolution of a severe laceration. This responded to conservative management by percutaneous drainage under ultrasound guidance.

CASE REPORT

A 21-month old male infant was admitted, as an emergency, shortly after having collided with a slowly reversing motor vehicle while riding his bicycle. He was afebrile on presentation, with a tachycardia of 164/min, respiratory rate of 36/min, and a normal blood pressure of 90/50 mm Hg.

A CT scan of the abdomen showed a grade 3 laceration of the right hepatic lobe, measuring 6 x 4 cm, extending from the hilum to periphery (Fig. 1), as well as a 2 x1 cm splenic laceration extending from the hilum to the capsule. There was no evidence of injury to the splenic, hepatic or portal vessels, and no extravasation of contrast. The solid organs, hollow viscera and bony structures were of normal appearance. The child responded to fluid resuscitation and a blood trans-fusion of 250 ml whole blood, equivalent to 23% of his calculated blood volume. Jaundice developed nine days after the injury, associated with a serum bilirubin of 6.3

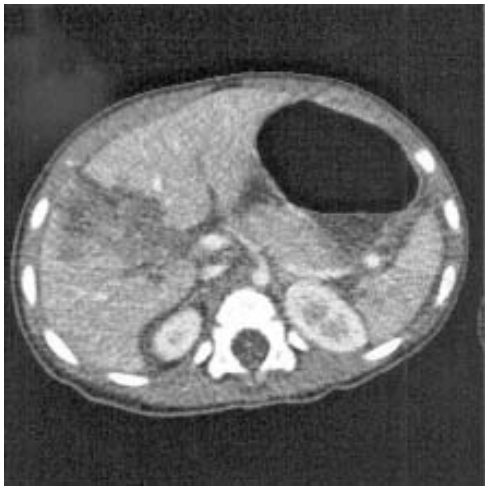

Fig. 1: Axial enhanced CT image through the liver shows a grade 3 hepatic laceration 6 x 4 cm.

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mg/100 ml, comprising a direct level of 1.2 and indirect of 5.1 mg/ 100 ml, but resolved by the end of the second week. He was discharged home19 days after admission.

At out-patient follow-up, with serial ultrasonography, rapid resolution of the splenic haematoma was noted. Five weeks after the injury, ultrasonography also showed a significant reduction in size of the hepatic injury to 2.5 x 1.0 cm (Fig 2). However, two months later (*ie* three months after the

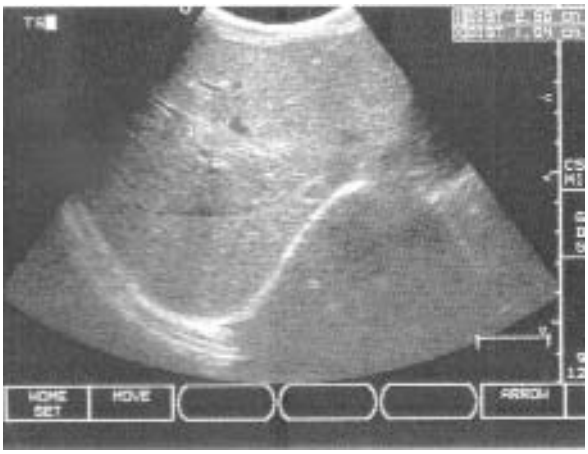

Fig. 2: On ultrasonography five weeks after presentation, there is a marked reduction in size of the laceration which is reduced to 2.5 x 1.0 cm.

initial injury) he presented with a six-day history of abdominal pain and anorexia, and had vomited three times prior to the onset of symptoms. He had had signs of an upper respiratory tract infection for one week, along with pyrexia of three days duration. A course of amoxicillin had produced no improvement.

His vital signs at this admission included a tachycardia of 150/min, temperature of 37.5ºC, blood pressure of 100/70 mm Hg and a respiratory rate of 40/min. There was gross distension of the right upper abdominal quadrant with hepatomegaly. Decreased air entry was noted in the right chest. He had maintained a haemoglobin level of 11.6G/100 ml, haematocrit of 34.5% and normal white cell count of 11.7 x 10^3/c mm. A plain radiograph confirmed a right pleural effusion and a raised right hemidiaphragm. The CT scan on this occasion demonstrated dominant fluid density thin walled subcapsular lesions along the lateral and inferior surfaces of the right hepatic lobe, measuring 13.2 x 7.5 x 7.4 cm superiorly, and 11.6 x 8.2 x 5.3 cm inferiorly. Ultrasound imaging showed abnormal fluid collections beneath the capsule of lateral and inferior surfaces of the right hepatic lobe (Fig. 3).

Percutaneous ultrasound guided puncture was carried out to allow the insertion of a pigtail catheter. About 1700 ml of bilious fluid drained over the next few days, but repeat ultrasound imaging one week after this admission showed persistent loculated fluid collections with estimated volumes of 330 and 240 ml. Another pigtail catheter was inserted percutaneously *via* the right flank after disruption

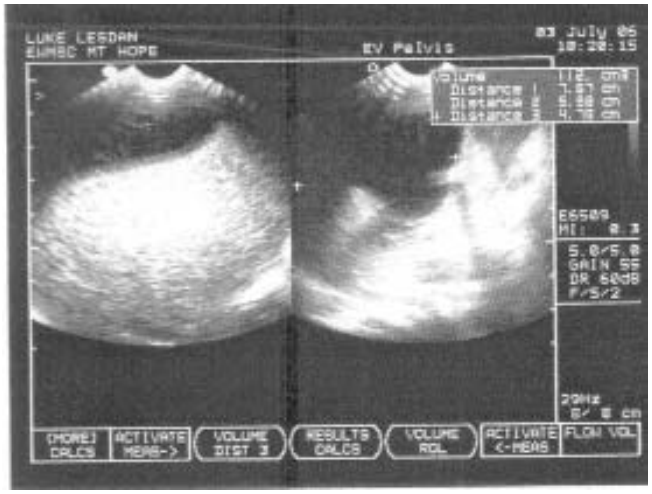

Fig. 3: Repeat ultrasound imaging just over three months after initial presentation shows abnormal fluid collections beneath the capsule of lateral and inferior surfaces of the right hepatic lobe.

of the septum between the collections. Ultrasonography five days later showed a significant decrease in size of the residual fluid collection to 7.7 x 5.9 x 4.8 cm.

A total of 1320 ml of bilious fluid was collected over the subsequent five days, the total drainage being replaced intravenously by an equivalent volume of normal saline. He was discharged home following cessation of drainage five weeks after re-admission.

At out-patient follow-up, four months after the injury, ultrasonography showed almost complete resolution of the superior subcapsular fluid collection (Fig 4). The collection along the inferior surface had completely resolved.

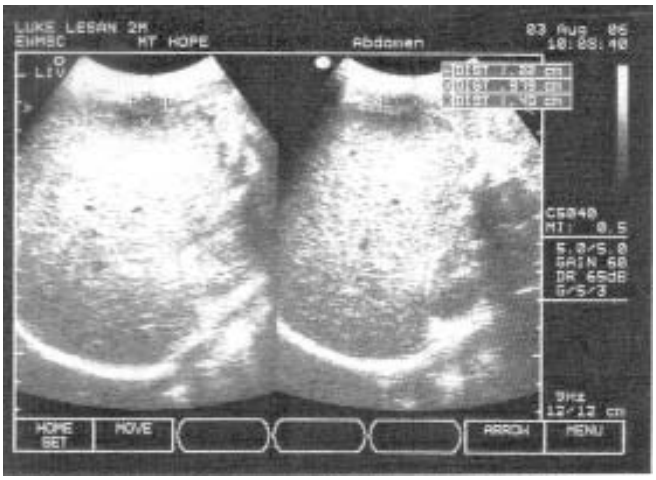

Fig. 4: Four months after initial presentation, there is almost complete sonographic resolution of the superior subcapsular collection.

DISCUSSION

The realization that the majority of laparotomies for blunt abdominal trauma were non-therapeutic paved the way for the evolution of non-operative methods of treatment. Reported advantages of the conservative approach include a

reduction in mortality, a lower rate of complications, fewer transfusion requirements and shorter in-hospital stays (4).

Suggested criteria for non-interventional management include haemodynamic stability, absent peritoneal signs and no significant associated intra- or retro-peritoneal injuries on CT scanning (5). The most critical of these factors is haemodynamic stability and laparotomy is still required for children who present with massive or continuing bleeding requiring transfusion of more than half the estimated blood volume (or 40 ml/kg of whole blood) (6). The presented infant remained clinically stable after the initial transfusion of just over 20% of his blood volume, despite the presence of a concomitant splenic injury, and the continuation of conservative management was thought appropriate.

Early access to CT scanning has been identified as crucial to this approach, since extensive intraperitoneal blood and contrast extravasation on CT scan may predict potential clinical failures (7). Computed tomography is recognized to be more sensitive for hepatic injury than ultrasound, and also helps in identifying concomitant intestinal and retroperitoneal injury, but both methods of evaluation, being complementary both in the initial assessment and ongoing evaluation (8), were applied to this infant. Patients with uncomplicated blunt liver trauma can generally be discharged home after a week or so, but restriction of activity for three to six weeks is commonly advised and contact sports are generally not recommended for two to three months (9). Actual sonographic evidence of resolution of hepatic injury may take as long as one to three months (10) and was well advanced five weeks after injury in this child.

Post-traumatic bilomas have been rarely described in children and are still the subject of isolated case reports (11). The CT or ultrasound guided needle aspiration and drainage have been established as effective therapeutic tools, even in the presence of a documented intrahepatic biliary fistula (12), and were effective in this case.

Bile leaks are known to be capable of clinical manifestation after a latent period, but no report was found of the development of a biloma more than eight weeks after injury. The delay of more than three months in this infant, after apparent almost complete resolution of the initial injury, is unique. There was no history of further trauma, but recrudescence of intrahepatic injury may have followed the onset of an upper respiratory tract infection, associated with repetitive coughing leading to spasmodic increases in intra-abdominal pressure acting on an incompletely healed injury. It is otherwise difficult to explain how the pronounced upper quadrant distension apparent at the second admission could have long escaped the notice of his mother, as well as of the prescribing primary care physician. This report is presented to highlight the importance of prolonged follow-up with serial imaging after conservative management of severe hepatic injury.

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