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Delayed Fatal Bleeding after Non-operative Management of High Grade Liver Injury

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Abstract

Most liver injuries after blunt trauma abdomen are currently treated by non-operative management (NOPM)1-3. Delayed haemorrhage necessitating operative intervention is one of the main complication following NOPM3. We report the case of a young male who suffered delayed fatal exsanguination after NOPM of a high grade liver injury and review the available literature regarding management of high grade liver trauma.

Keywords: High grade liver Injury; Non-operative management; Bleeding

3 Key Messages: Delayed bleeding after non-operative management of liver trauma remains the main complication necessitating urgent lifesaving intervention. Follow-up imaging of the liver prior to dismissal from the hospital may help to triage patients prone to complications.

Introduction

Even high grade liver injuries in a haemodynamically stable patient are currently treated by NOPM [1-3]. With improvements in imaging techniques and the availability of angio-embolisation, surgical management of liver trauma is limited to damage control surgery and management of complications following NOPM. Delayed bleeding from the liver is one of the main complication following NOPM3 which necessitates urgent surgical intervention. If appropriate resuscitative measures and urgent intervention to control the bleeding are not carried out, the outcome is usually fatal. We report the case of a young male who suffered delayed fatal exsanguination after NOPM of a grade four liver injury.

Case History

A twenty two year old male presented to the hospital with history of motor vehicular accident. He was hemodynamically stable at initial presentation. Evaluation of injuries showed fractures of multiple long bones (Left tibia, left Femur, Clavicle and Mandible). A focused assessment with sonography for trauma scan revealed evidence of free fluid in the right upper quadrant. After initial trauma survey and resuscitation, the patient was taken up for a triphasic CT scan of the abdomen. This revealed an American Association for the Surgery of Trauma [4] (Table 1) grade four liver laceration, with laceration to the caudate lobe (Figure 1).

There was no contrast blush identified although there was significant free fluid around the liver. Since the patient remained haemodynamically stable, he was elected for NOPM of the liver injury. The patient remained stable both haemodynamically (normal pulse rate and blood pressure) and by laboratory parameters (no fall in haemoglobin) throughout the stay (Table 2). He was taken up for fracture fixation on post trauma day (PTD) seven. The patient tolerated the operative procedure well. Follow up ultrasonography of the liver laceration showed a progressive decrease in the size of the liver hematoma and patient was dismissed form the hospital on PTD fifteeen.

He presented for routine follow-up eight days after dismissal (PTD twenty one). On presentation, he complained of vague abdominal discomfort that started during travel to the hospital, which rapidly progressed in severity. On evaluation he was found to be in shock with a distended abdomen. Before resuscitative efforts could be initiated, he suffered asystole. Cardio-pulmonary and volume resuscitation were started and after return of pulse, he was taken up for an emergency laparotomy. Intra-operatively there was three liters of old blood and two liters of fresh clot in the inferior surface of the liver. This was evacuated and the abdomen was packed with plan of delayed reexploration. Six hours post laparotomy, the patient again had a drop in blood pressure with a concomitant drop in Haemoglobin. An emergency interventional angiogram demonstrated a right posterior sectoral arterial bleed that was coiled (Figure 2).

Grade*	Type of Injury	Description of injury
I	Hematoma	Subcapsular, <10% surface area
	Laceration	Capsular tear, <1cm parenchymal depth
II	Hematoma	Subcapsular, 10% to 50% surface area Intraparenchymal <10 cm in diameter
	Laceration	Capsular tear 1-3 parenchymal depth, <10 cm in length
III	Hematoma	Subcapsular, >50% surface area or ruptured Intraparenchymal hematoma > 10 cm or expanding
	Laceration	>3 cm parenchymal depth
IV	Laceration	Parenchymal disruption involving 25% to 75% of hepatic lobe or 1-3 Couinaud's segments
V	Laceration	Parenchymal disruption involving >75% of hepatic lobe or >3 Couinaud's segments within a single lobe
	Vascular	Juxtahepatic venous injuries; i.e, retrohepatic vena cava or central major hepatic veins
VI	Vascular	Hepatic avulsion

*Advance one grade for multiple injuries up to grade III

Table 1: The American Association for the Surgery of Trauma Organ Injury Severity Scoring scale. Liver Injury Scale (1994 Revision) [4].

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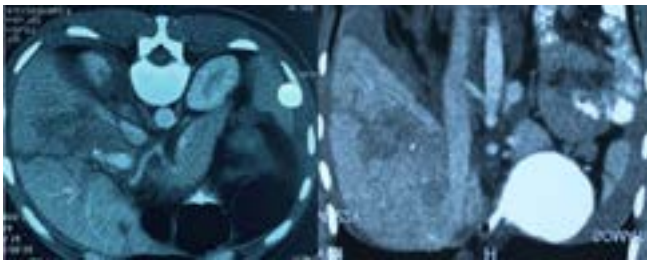

Figure 1: Computed tomographic images demonstrating a grade 4 laceration to the liver parenchyma. There was no contrast blush visible. After non-operative management of the laceration, repeat imaging by ultrasonography had demonstrated no increase in the size of the hematoma.

PTD	0	1	4	7	10	15
Blood Pressure (mm Hg)	100/40	126/64	120/74	116/60	124/80	128/76
Hemoglobin (mg/dl)	7.6	9.2	11.2	10.8	11	10.2

Table 2: Showing the haemodynamic status of the patient during hospitalisation. The patient remained clinically and hemodynamically stable throughout hospitalisation. He underwent fracture fixation on PTD 7 and was discharged on PTD 15 with a stable hemoglobin level. No blood transfusions were given after initial resuscitation.

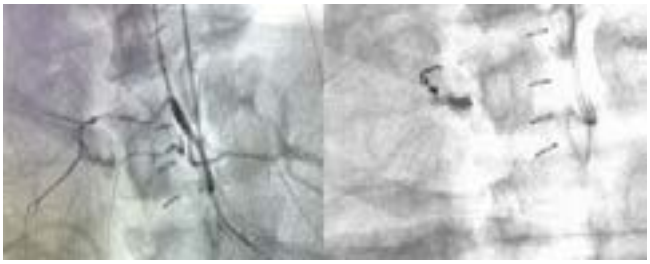

Figure 2: Angiographic images showing active bleeding (Arrow) from the right posterior sectoral artery. The artery was coiled and a fresh angiogram showed complete control and no further bleeding.

The patient improved hemodyanamically following this and responded to resuscitation with an improved urine output. But twelve hours post embolization the patient rebled and despite ongoing resuscitative efforts and plans for repeat angio-embolisation, expired.

Discussion

The management of hemodynamically stable Liver trauma patient has evolved over the past two decades from aggressive surgical intervention to NOPM [5]. With improvements in imaging techniques, NOPM remains the main stay of management of a patient even with major liver trauma. The haemodynamic stability of the patient is what dictates management, rather than the grade of liver injury. NOPM involves a period of intensive monitoring with restriction of mobility of the patient. Serial monitoring of haematocrit and vital signs of the patient is mandatory to diagnose bleeding early so that NOPM can be abandoned, although the frequency of these examinations is not standardized [6]. But critics have pointed out that the excessive use of NOPM, even for high-grade liver injuries, leads to complications like early or late haemorrhage, pseudo aneurysm, arterio-venous fistulae, biliary fistula, etc which necessitate operative intervention later [7,8]. This has led to formulation of protocols requiring re-imaging of the liver at repeated intervals to look for development of complications so that therapeutic measures can be instituted. Given the varied timeline for healing of liver trauma, the duration of treatment varies for every patient. Tiberio et al. [9] monitored Liver trauma patients with serial imaging and found the median healing time for a Grade I hematoma to be 6 days, 16 days for Grade II and 108 days for Grade III hematoma. For lacerations, the median healing time was found to be 29 days for Grade II, 34 days for Grade III, and 78 days for Grade IV. But there are no clear indicators available for duration of immobilisation of the patient, treatment and hospital stay although studies have shown that early mobilisation does not lead to an increase in the incidence of delayed bleeding [10] and that dismissal from the hospital can be based solely on clinical criteria of a normal abdominal examination and stable hemoglobin, irrespective of the grade of liver injury [11]. But delayed bleeding and hemodynamic instability remain the main complication after NOPM in adults and upto 20-63% of patients managed by NOPM (especially high grade injuries) require urgent intervention [12]. Most patients who develop delayed bleeding are too unstable for definitive procedures and hence are candidates for damage control surgery. This is usually complimented by endovascular control of the bleeding, resuscitation and follow-up definitive procedure. Repeated angiographies may be needed to treat re-bleeds following this approach [13,14]. The source of bleeding is usually a pseudo-aneurysm of the artery, although bleeding from the portal vein, hepatic veins and the inferior vena cava have also been reported [15]. Our patient had delayed bleeding from the right posterior sectoral artery as seen during angiography. As there was no pseudoaneurysm detected on previous imaging studies, and since the patient travelled by road to the hospital, the possibility of dislodgement of a pre-existing clot cannot be ruled out. Unfortunately, repeated follow-up imaging by ultrasonography during his previous hospitalization had not demonstrated any features that could have predicted the bleeding. Despite ongoing resuscitative efforts, the patient succumbed to re-bleeding. This underscores the importance of accurate grading of severity by imaging, especially when opting for NOPM and also the importance of follow-up imaging by CT scan or MRI to circumvent the operator dependent limitation of Ultrasonography.

Conclusion

NOPM of high-grade liver trauma remains controversial. The decision for early intervention depends on the haemodynamic stability of the patient and on the expertise available in the hospital. Delayed bleeding remains the main complication necessitating urgent lifesaving intervention. Follow-up imaging of the liver prior to dismissal from the hospital may help to triage patients prone to complications.

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