

We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,900

Open access books available

185,000

International authors and editors

200M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com



Liver Trauma

Marco Massani, Luca Bonariol, Bruno Pauletti,
Cesare Ruffolo, Roberta Bonariol,
Ezio Caratozzolo and Nicolo' Bassi

Additional information is available at the end of the chapter

<http://dx.doi.org/10.5772/64543>

Abstract

Objective: The aim of this section is to assess the evolution in the management of liver injuries during the last two decades.

Methods: The authors reviewed the English literature, reporting the wider experiences, and on the basis of the personal experience, they suggest an up to date treatment.

Results: Mortality due to hepatic injury has decreased over the past century from nearly 70% at the beginning of last century to the current level of 6–7%. This could be partially attributed to the improvement of operative techniques, to a lower surgical trend, to the application of damage control laparotomy in very complex liver trauma, and particularly to the diffusion of hepatic angiographic embolization. Scientific evidence has shown that more than 67% of laparotomies performed for blunt liver trauma are not therapeutic and in about 86% of all post-traumatic hepatic lesions the hemorrhage stopped spontaneously at the time of the laparotomy. On the other hand, blunt hepatic trauma can be conservatively treated in 70–90% of hemodynamic stable patients, in the absence of other associated lesions/peritoneal signs or in the case of limited need for transfusions. Thus far, the main decision criteria for a surgical rather than a conservative approach to the management of liver trauma have been the hemodynamic stability and not the American Association for the Surgery of Trauma-Organ Injury Scale (AAST-OIS) grading of the lesion.

Conclusions: Restricting the indications for a conservative treatment of liver traumas solely to the relief of hemodynamic stability seems nowadays to be unreasonable. Criteria for a conservative treatment are as follows: hemodynamic stability, limited need of blood transfusions, and non-associated visceral lesions on CT scan. In the presence of these conditions non-operative approach

can be undertaken, and the cornerstone of this treatment is transarterial angiographic embolization.

Keywords: Liver trauma, embolization, laparotomy, interventional radiology, associated lesions

1. Objective

Liver and spleen are the most frequently injured organs in blunt abdominal trauma. Management of hepatic trauma has nowadays radically changed in comparison with the past, when the chance of spontaneous hemostasis of hepatic lacerations was considered an impossible event, and conservative management appeared dangerous. Therefore, surgery was almost ever the best therapeutic option for the patient with liver injury. In the 1980s–1990s, surgical indication for liver trauma was dictated not only by the need to control hemorrhage or control biliary leak, but also to reveal concomitant unknown hollow viscus injuries. At the beginning of twentieth century, most hepatic injuries had fatal outcome, despite the routinely use of liver pedicle clamping introduced by J. Hogarth Pringle in 1908.

2. Methods

The authors reviewed the English literature, reporting the wider experience in the management of liver trauma and considering their personal experience; during 20 years, they analyzed how liver trauma management has changed in the last two decade.

3. Results

Nowadays, mortality for hepatic injury has decreased significantly as reported in the analysis of data collected by the National Trauma Data Bank (NTDB) (under the aegis of the American College of Surgeons Committee on Trauma NTDB (2008)) from nearly 70% at the beginning of last century to 16.7% between 2000 and 2004 [1]. Current series report a mortality rate of 6–7% for liver trauma [2].

Many factors led to decrease mortality rate, including:

- better knowledge of liver physiopathology,
- non-operative management (NOM) (success rate of 80–90%),
- development of high experience centers in hepatobiliary surgery,
- damage control laparotomy (DCL) in complex liver trauma.

Moreover, new CT scan techniques can provide more detailed images of hepatic lesions, exclude hollow viscus injuries and quantify the amount of blood into peritoneal cavity.

These factors have drastically changed the approach to liver trauma, which is no longer an exclusive dominion of the surgeons.

3.1. Classification

Liver injury is often due to a polytrauma; therefore, its management needs a score to state severity of clinical condition: frequently adopted scales are Revised Trauma Score (RTS) and Injury Severity Score (ISS).

| Liver | Abbreviated Injury Scale Score (AIS) |
|---|--------------------------------------|
| Grade I | 2 |
| <i>Hematoma:</i> sub capsular, < 10% surface area | 2 |
| <i>Laceration :</i> capsular tear, < 1 cm parenchymal depth | |
| Grade II | 2 |
| <i>Hematoma:</i> subcapsular, 10% to 50% surface area, intraparenchymal < 10 cm in diameter | 2 |
| <i>Laceration:</i> capsular tear 1 to 3 cm parenchymal depth, < 10 cm in length | |
| Grade III | 3 |
| <i>Hematoma:</i> subcapsular, > 50% surface area, ruptured subcapsular or parenchymal hematoma; intraparenchymal hematoma >10 cm or expanding | 3 |
| <i>Laceration:</i> >3 cm parenchymal depth | |
| Grade IV | 4 |
| <i>Laceration:</i> parenchymal disruption involving 25% to 75% hepatic lobe or 1 to 3 Coinaud's segments | |
| Grade V | 5 |
| <i>Laceration:</i> parenchymal disruption involving > 75% of hepatic lobe or > 3 Coinaud's segments within a single lobe | 5 |
| <i>Vascular:</i> juxtahepatic venous injuries, ie retrohepatic vena cava/central major hepatic vein | |
| Grade VI | 6 |
| Vascular hepatic avulsion | |

Table 1. Liver Injury classification according to American association for the Surgery of Trauma-Organ Injury Scale.

Revised Trauma Score (RTS) is based on three parameters: blood pressure, breathing, and Glasgow Coma Scale (GCS) each of them is quantified with a level score from 0 to 4. A cumulative score <11 is considered as a severe condition [3].

On the contrary, ISS is a more complex scoring system generally closely related to morbidity, mortality, and length of hospital stay. It is based on Abbreviated Injury Scale (AIS) that assigned a six-grade score for each of six body regions. The score ranges between 1 and 25; thus, the trauma is considered mild for score <9, moderate for 10–14, and severe for 15–24 [4].

Both previous reported score systems are useful for evaluating trauma in its complexity but in order to create an injury severity scale for single organs; in 1987, the American Association for the Surgery of Trauma (AAST) assessed the Organ Injury Scale (OIS), which is now the most frequently adopted [5] (**Table 1**). The scale, first proposed by Moore and revised in 1994, includes six grades of severity.

Based on Moore classification, Mirvis scale was designed on CT scan findings (**Table 2**) [6]. OIS-AAST grading system provides a descriptive value of hepatic injury, but its utility in clinical practice is somehow limited, because it does not correlate with specific treatment of the hepatic trauma.

Recently, the National Trauma Data Bank (NTDB) [2] under the aegis of the American College for Surgeons Committee on Trauma tried to give a clinical value to the AAST-OIS scale.

The NTDB V. 5.0 included 1,130,093 patients from 405 centers in USA between 2000 and 2004 with liver, kidney, and spleen trauma, isolated or associated with other lesions. Data analysis showed that mortality rate increased as well as grade of lesions: for patients with liver trauma, grade 1–2 associated with other lesions was reported a mortality of 12.7%, 15% for grade 3, 27.9% for grade 4, 64.8 % for grade 5, and 94.9% for grade 6.

Overall mortality was 16.7%, and 80–90% of patients with hepatic and kidney trauma had undergone NOM. Nevertheless, operative rate increased as well as grading from 29.2 to 37.2%.

| Grade | |
|-------|---|
| 1 | Capsular avulsion, superficial laceration (s) <1cm deep, subcapsular hematoma <1 cm maximal thickness, periportal blood tracking only |
| 2 | Lacerations (s) 1–3 cm deep, central/subcapsular hematoma (s) 1–3 cm in diameter |
| 3 | Lacerations (s) > 3 cm deep, , central/subcapsular hematoma (s) >3 cm in diameter |
| 4 | Massive central/subcapsular hematoma (s) >10 cm in diameter, lobar tissue destruction (maceration) or devascularization |
| 5 | Bilobar tissue destruction (maceration) or devascularization |

Table 2. Mirvis classification of liver trauma (Mirvis).

3.2. Non-operative Management (NOM)

Since 1990, management of liver trauma has become more and more conservative, particularly due to studies on pediatric population. Karp et al. reported 17 pediatric cases with blunt hepatic trauma successfully managed without surgical intervention. Moreover, he described four stages of damaged liver healing: (1) blood reabsorbtion within the first 2 weeks, (2) coalescence of the laceration, (3) size reduction, and (4) restitutio ad integrum in a period of 3–4 months [7]. Furthermore, reviewing CT scans, he revealed that 20–67% of laparotomies for trauma were not therapeutic and in 50–85% of hepatic lacerations, bleeding has already stopped at the moment of surgical exploration [8].

Results from pediatric experience gained considerable popularity also in adult trauma management, and around 1995–1996 first reports about NOM appeared in literature, even if they concern small series and not uniform population. However, success rate of conservative treatment has been reported between 17 and 60%.

In a prospective study by Malhotra et al., a population of 661 patients with blunt trauma was compared to other two patient cohorts of previous years (1985–1990 and 1993–1994); starting from this analysis, a new “paradigm shift” of liver trauma management in non-operative approach was provided [9]. Success rate of conservative management was 92.5%, associated with a low rate of intra-abdominal infections, low need of blood transfusions, and shorter hospital stay. Failure of NOM was due to hepatic-related causes in only 48% of cases.

The early studies about NOM were performed on patients with low grade lesions (I-II AAST-OIS) and reported a success rate of conservative management around 70–90% [10]. More recent experience showed good results also in stable patients with grade 4–5 liver injuries.

A recent analysis of NTDB V. 5.0 (between 2000 and 2004) [2] that tried to validate the AAST-OIS, showed that only 14% of patients with liver trauma needs surgical operation, and in case of isolated hepatic trauma, success rate of NOM was, respectively, 91.5% for grade 1–2 lesions, 79% for grade 3, 72.8% for grade 4, and 62.6% for grade 5.

The same results were described by Hurtuk et al. [11], reporting a success rate of 76.6, 69.3, and 62.3%, respectively, for grades 3, 4, and 5 lesions, and by Polanco et al. [12] in recent review of 3627 patients with blunt liver trauma and AAST-OIS score ≥ 4 , reporting a success rate of NOM $>90\%$.

On the contrary, Kozar et al. [13] reported a success rate of 40% in grade 4 injuries and only 4% in grade 5.

Finally, in multicentre retrospective study including 393 patients with blunt liver trauma AAST-OIS grade 4–5, the authors confirmed good results with NOM even in complex liver injuries. Two-thirds of patients underwent conservative treatment with a success rate of 91.3% [14].

Therefore, the lesson from these studies is that NOM is considered advisable even for severe hepatic injuries, assuming that hemodynamic stability is verified (**Figures 1 and 2**).

It should be noted that even if the shift towards non-operative management drastically improved survival of patients with hepatic trauma, its high success rate does not release surgeon from close supervision, especially in patients with complex liver injuries. Richardson [1] reported that mortality for liver trauma decreased from 19 to 9% in a 25 years lapse and liver directly related death from 12 to 5% not only for the introduction of non-operative management of grade 4–5 liver trauma, but also due to improvement in surgical techniques and use of damage control laparotomy.

The most important selection criteria for a conservative approach to patients with hepatic trauma are in summary:

- hemodynamic stability (blood pressure ≥ 90 mm Hg and good response after fluid resuscitation),
- limited need of blood transfusions (≤ 4 UI/24 h),
- non-associated visceral lesions on CT scan.

In the presence of these conditions non-operative approach can be undertaken, and the cornerstone of this treatment is transarterial angiographic embolization.

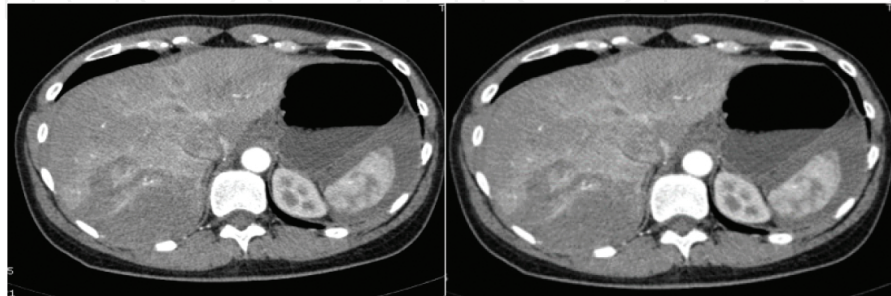


Figure 1. Female, 49 years old, cyclist hit by a truck. She was hemodynamically stable, blood pressure 110/80 mmHg, RTS 12, hemoglobin 12 gr/dl, GCS 15. CT scan of abdomen and chest was performed at admission with evidence of pneumothorax and multiple lacerations in the right lobe of the liver, Mirvis scale grade 3–4, with hemoperitoneum, but not vascular blush. She was not transfused and conservatively treated. Discharged after 15 days in good health.

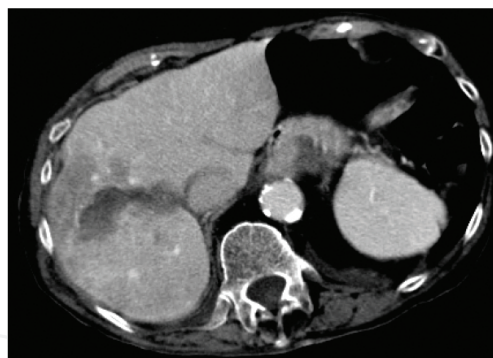


Figure 2. Female, 91 years old, with chest trauma following accidentally fall in her house. Ct scan showed deep laceration in the central part of the liver, Mirvis scale grade 3. The patient was conservatively treated with success and discharged after 13 days.

3.2.1. *Transarterial angiographic embolization (TAE)*

The introduction in clinical practice of angiographic embolization, based on CT scan finding of contrast blush, is the factor that most contributed to improvement in survival.

Arterial hemorrhage differs from venous bleeding because it cannot be stopped by simple manual compression or by packing. Thus, use of angiographic embolization has increased both in NOM and after damage control laparotomy.

A recent review of our experience concerning the treatment of grade 3–4 hepatic trauma revealed that since 2005, thanks to the introduction of a new, high quality multislice CT scanner and to greater improvement in angiographic embolization expertise, the success rate of NOM has significantly increased compared to the past, with lower conversion to laparotomy (6% vs 62%) and operative rate (52% vs 22%) [15].

Fang et al. [16] divided the CT finding of contrast blush into three groups:

- free extravasation into peritoneal cavity;
- intraparenchymal bleeding with concomitant hemoperitoneum;
- intraparenchymal contrast blush without hemoperitoneum.

Intraperitoneal contrast extravasation, in presence of hemodynamic stability, is considered a strong indication for early angiographic embolization, and it is, however, an independent predictive factor for the need of operative treatment.

It is quite controversial whether to perform angiographic embolization in hemodynamically stable patients presenting intraparenchymal blush rather than submit them to close observation, performing angiographic embolization only in cases of remarkable signs of bleeding.

Moreover, contrast blush detected at CT scan is not always confirmed by subsequent angiography. This is another interesting aspect because it is still debated whether to continue with embolization anyway, taking the risk of hepatic ischemia, rather than stop the procedure and start a close monitoring as soon as the first signs of hemorrhage appear. A recent study by Alarhayem et al. [17] reported 68 patients with Ct scan evidence of contrast blush; 22 (33%) of them had no sign of contrast pooling at subsequent angiography and therefore did not undergo embolization. The re-bleeding rate in this group was 32%, significantly higher than what observed in embolized patients. According to the authors, patients should undergo angioembolization procedures when clear CT scan signs of contrast blush are visible.

The same results were reported by Hagiwara et al. [18] comparing CT scan to angiography in detecting contrast extravasation.

The Eastern Association for the Surgery of trauma guidelines assign a level 2 recommendation for angioembolization in hemodynamically stable patients with contrast blush evidence on abdominal CT scan [19] (**Figures 3 and 4a–c**).

A challenging issue proposed by some authors is the use of angioembolization in high-grade hepatic injury found radiologic, despite the finding of contrast blush.

Poletti et al. [20] compared CT scan to angiography in 72 patients with blunt hepatic trauma. The author took into consideration hepatic injury, with or without major veins involvement (the first group had a bleeding risk 3.5 times higher). According to the authors' experience, angiography should routinely be performed in all patients with grade 4 liver trauma when hepatic major veins are involved.

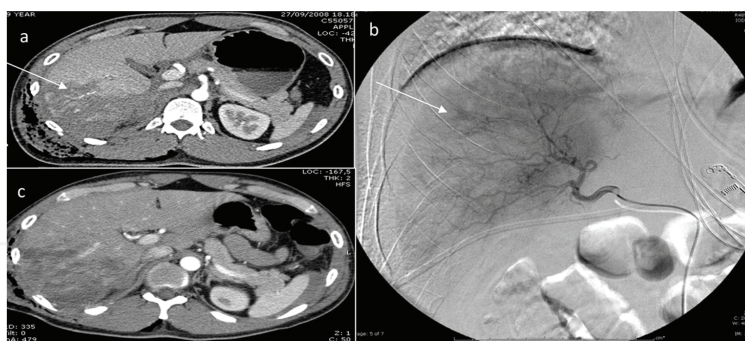


Figure 3. Male, 29 years old, admitted in emergency department for abdominal and chest trauma following fall with paragliding. GCS 15, RTS 12, hemodynamically stable. (a) Ct scan showed deep lacerations in the right liver, large hemoperitoneum and extraparenchymal vascular blush. (b) The patient underwent angiography with evidence of blush in the lateral segments of the liver and selective embolization was performed with gelfoam. (c) CT scan at 48 h. The hospital stay was uneventful and the patient was not transfused.

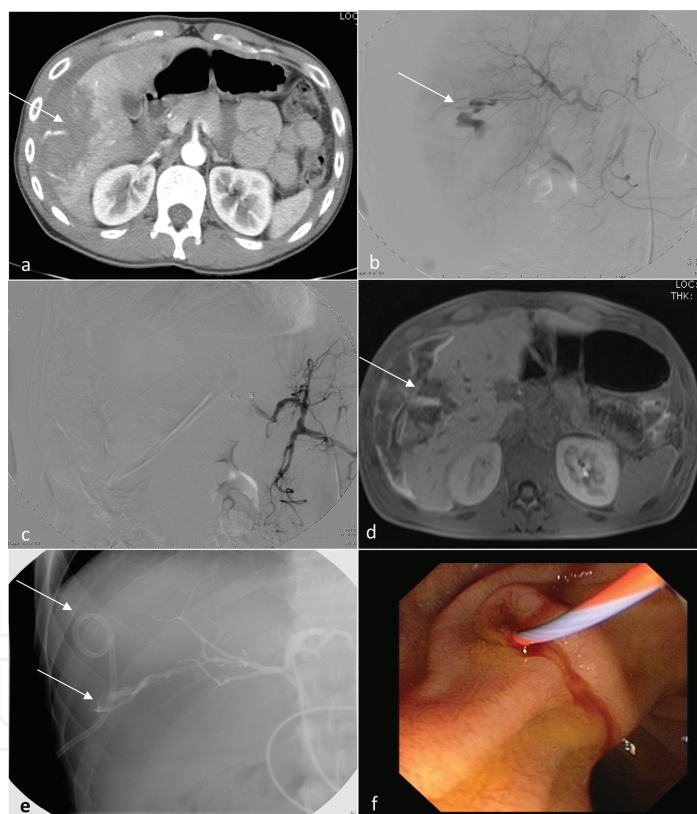


Figure 4. Carpenter, 35 years old hit by a piece of wood in his abdomen. At admission CT scan (a) revealed large hemoperitoneum due to deep lacerations in 5-6 segments of the liver with vascular blush. He underwent angiography (b) that confirmed the blush originating from branch of the right hepatic artery for the fifth segment. The patient was embolized with gelfoam in the distal part of the right hepatic artery and metal coils. No evidence of blush at the end of the procedure (c). In the following days the patients remained always stable, but he developed jaundice. An MRI (d) was performed that showed deep non-bleeding laceration in the lateral segments of the liver, large perihepatic biliary collection with biliary fistula due to traumatic disruption of the dilated biliary ducts inside the laceration (e). The fluid collection was drained by percutaneous insertion of pig-tail (e), and the biliary fistula healed after ERCP and stent insertion (f).

Carrillo [21] reported a 83% efficacy rate in arterial hemorrhage control in liver trauma, whereas Sirivrikoz [22] demonstrated that angiographic embolization is an independent survival factor (AOR 0.48, 95% CI; 36–63, $p < 0.01$) in a series of 6042 patients with isolated grade 4–5 hepatic trauma (years 2002–2011, NTDB. multivariate analysis). In another multicenter study [14], TAE was administered to the two-thirds of patients with grade 4–5 hepatic trauma, with a 91.3% success rate; a quarter of them had undergone angioembolization.

Angioembolization efficacy in hepatic trauma has so far widely been confirmed. Nevertheless, this procedure is not free from life-threatening complications. Hepatic necrosis, gallbladder ischemia, biliary fistulas, and hepatic abscess have been well described in the literature, with a morbidity rate that varies from 29 to 80% [23]. In 2003, Mohr et al. [24] reported a considerably high incidence of complications in patients with hepatic trauma treated by embolization, with a gallbladder infarction rate of 15%.

Letoublon et al. described a 70% morbidity rate in 23 patients treated with TAE [25]. According to the author, however, complications are not exclusively caused by the procedure itself, but also a consequence of complexity of liver trauma.

Dabbs [26] reported a post-TAE hepatic necrosis rate of 42%. TAE morbidity rate is variable and complications are more often caused by the severity of liver trauma than by the procedure itself, which should definitely be performed in selected patients in order to avoid specific hepatic complications.

From 1993 to 2015, 222 patients with hepatic trauma were referred to our Institution; 43% of them have been submitted to NOM. In our experience, we noticed that after 2005, with the introduction of a new, high-quality multislice CT scan and the improvement in angiographic arterial embolization procedures, 70% of patients with grade ≥ 3 AAST-OIS hepatic trauma underwent NOM with a success rate of 91%. Seventeen (26%) hemodynamically stable patients underwent TAE for contrast extravasation revealed at CT scan. Three patients with severe hepatic trauma underwent TAE even in the absence of bleeding at CT scan, but confirmed at angiographic images. One patient underwent embolization after damage control laparotomy. Two patients were submitted to laparotomy for gallbladder infarction and coleperitoneum. Efficacy, morbidity and mortality rate of TAE were 100, 53 and 18%, respectively. Just in one case, a complication (gallbladder infarction) was directly caused by the procedure.

3.2.2. *Prognostic factors*

The success of conservative management in blunt liver trauma is related to the ability to predict the need for surgical intervention before deterioration of patients' clinical conditions. Conservative approach may sometimes lead to late hemodynamic instability and the need of an emergent laparotomy, with increased surgical risks; In fact, operations performed in critical conditions when patient's parameters have worsened is certainly more challenging.

Some reports provide that negative prognostic factors at admission are blood pressure < 110 mmHg and base deficit of 4 [26, 27]. Almost 25% of patients with liver trauma conservatively treated, need a surgical intervention for complications or NOM failure and in the 3.1–6.7% of cases there is the possibility to ignore concomitant hollow viscus injuries [28]. Fur-

thermore, failure of NOM is associated with increased mortality, particularly in high-grade trauma [12]. This implies that a critical approach and intensive surveillance for patients with hepatic lesions grade ≥ 3 –4 conservatively treated is mandatory, especially if hypotensive at admission and with high ISS.

According to Malhotra et al. [9], bad hemodynamic parameters at admission, high ISS, large hemoperitoneum, and vascular blush on CT scan represent risk factors for NOM failure, even if none of them have a predictive value in statistical terms.

Polanco et al. [12] reported others risk factors as age (OR 1.02), male gender (OR 1.73), high ISS (OR 1.02), low GCS, and low blood pressure at admission (OR 2.07).

A recent systematic review of the literature considered eight prospective studies including 410 patients. Failure of NOM was observed in 9.5% of cases, mortality rate was 4%, and 26 prognostic factors were identified, even if only six of them had a statistical relevance: blood pressure, fluid resuscitation, blood transfusion, the presence of peritoneal signs, ISS and intra-abdominal-associated lesions. Grading of hepatic trauma was not considered a risk factor for NOM failure [29].

In the absence of specific criteria to certainly predict which patients with blunt hepatic trauma would develop hemodynamic instability or complications after NOM, Fang et al. [30] identified some CT scan findings that can predict the need of surgical operation:

- intraperitoneal contrast blush, hemoperitoneum involving six abdominal compartments,
- hepatic trauma involving more than two segments,
- high-grade Mirvis scale,
- porta hepatis involvement, hepatic lacerations depth >6 cm.

Among them, only intraperitoneal contrast blush, and hemoperitoneum involving six abdominal compartments result predictive factors at multivariate analysis.

3.2.3. *NOM failure and complications*

Approximately 25–27% of hepatic trauma conservatively treated needs a surgical intervention for complication or late hemorrhage [31]. Therefore, non-operative management of hepatic trauma should be considered not only in terms of mortality but also in terms of morbidity: Overall morbidity is about 0–7% but can be higher than 14% in high-grade lesions. Kozar et al. [32] analyzed 699 patients with blunt liver trauma grade 3–5. About 65% of them was conservatively treated, and they reported an overall morbidity of 14%. Complications including bleeding, bile leaks, infections, and abdominal compartment syndrome (ACS) were reported in 5% of grade 3 lesions, 22% of grade 4, and 52% of grade 5. ACS and bleeding were the most common complications during the first days, while after the third day, patients presented biliary and infectious complications more frequently. At multivariate analysis, grading, and transfusion number within 24 h were predictive factors for complications occurrence.

Management of complication after NOM, recognized on CT scan, should be multimodal, especially in case of biliary accidents as biloma, biliary fistula, and biliary peritonitis. Such kind of problems occur in 3% of cases, particularly in grade 4 lesions, and represent one-third of liver-related complications (**Figure 4d–e**) [27].

Small bilomas usually undergo radiologic observation, and in case of increasing in size, a percutaneous drainage placed under ultrasonography control may be required (**Figure 4e**). ERCP and stenting are the most common techniques in case of major biliary fistula (**Figure 4f**). In case of biliary peritonitis with systemic signs of inflammatory reaction, a laparotomy or laparoscopy with multiple drainages placement and hepatic resectional debridement may be necessary. Bleeding complications usually occur within 72 h after trauma and can be successfully controlled with angiographic embolization in most of cases, even in sometimes a surgical intervention is mandatory.

3.2.4. Follow-up in NOM

It is by now well demonstrated that patients with complex liver trauma must be carefully monitored in Intensive Care Unit (ICU).

On the contrary, the indication to radiologic follow-up during hospital stay and after discharge is still controversial. In a multicentre study, Patcher et al. [33] assert that there is no indication for CT scan follow-up for patients with grade 1–3 liver trauma. The same results are reported by Carrillo et al. [34] while they suggest a radiologic observation for patients with severe hepatic trauma (grades 4–5) when precise conditions (hemodynamic abnormalities, abdominal pain, drop of hemoglobin level, and increase of liver function tests) are revealed.

It is well known that healing time of hepatic injuries, as shown by CT scan observation, is 4–12 weeks; therefore, these patients may resume their physical activity after at least 4 weeks in case of grade 1–3 injuries and after 8 weeks for grade 4–5 injuries.

Cox et al. [35] showed that at CT scan control, 86% of patients presents no modification in liver lesions within the first week after trauma. An improvement was seen between 7 and 10 days after injury and an almost complete healing after 3 months.

3.3. Operative treatment

In the era of interventional radiology, non-operative treatment has become the most used approach in liver blunt trauma. Nevertheless, up to 35% of patients with complex hepatic injury (12–15% of all hepatic injuries) still needs surgical treatment [23, 36–38].

Forty years ago, all patients with liver injury underwent surgical exploration, because liver parenchyma was considered unsuitable for spontaneous hemostasis, and mortality rate exceeded 80% [36, 39].

Starting from the second half of the last century, the introduction of NOM and use of arterial embolization and the improvement of surgical strategies and surgeons ability have radically changed prognosis of these patients [40].

Indeed, the main indications for early surgical exploration in blunt abdominal trauma are by now hemodynamic instability with hemoperitoneum and the presence of peritonitis (in most of cases, it concerns grade 4 and 5 lesions). Patient with low blood pressure, not responding to fluid resuscitation should undergo urgent laparotomy [23, 41, 42]. In this context, well known that the choice of the correct strategy is essential to influence the final outcome of the patient. Critical decision-making, profound knowledge of liver anatomy, and surgical expertise in liver surgery are crucial points, and for these reasons, the operation should be managed by a dedicated liver team [36, 37, 41].

A correct approach must be systematic (**Figure 5**) and follows some “gold rules”: use simple maneuvers, pack and compress the liver for damage control, stop the operation when patient is stable, and postpone adjunctive procedure to a second look [39, 41, 43]. This is the concept of “damage control surgery,” introduced by Stone at al. in 1980s and promulgated by many authors in numerous large group studies [38, 39, 41, 42].

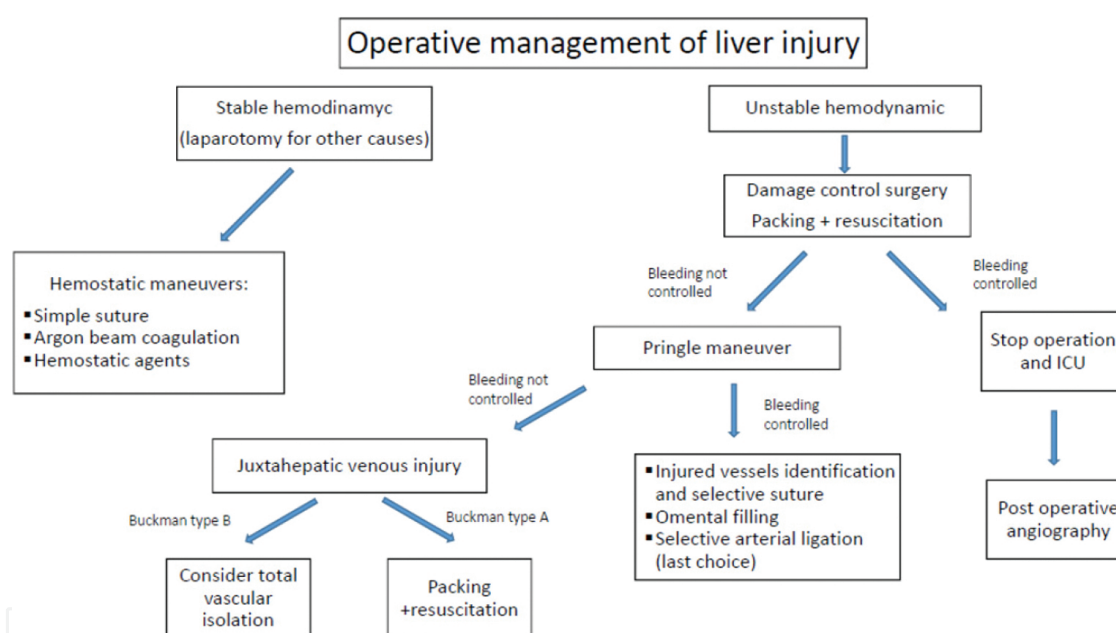


Figure 5. Flowchart for operative management of liver trauma.

Laparotomy should nearly always start with a midline incision with adequate exposure of the upper abdomen. In case of a complex right lobe injury or vena cava lesion, a right extension of the incision may be necessary. Rarely, a sternotomy is needed to control a lesion of the inferior vena cava (IVC) [37, 43].

The initial maneuvers are essential and are directed to control bleeding and allow resuscitation and must be simple and determined [37]. The liver should be manually compressed pushing both the lobe in their natural position and toward the diaphragm. This procedure can stop venous bleeding and allows intra-operative resuscitation. This is a crucial phase, because a delay in control of hemorrhage could lead to the “triad of death”: hypothermia, acidosis, and coagulopathy; this is a high-risk condition for patient death. For this reason, it is recommended

to use damage control surgery as a routine procedure and not as the last desperate option when other maneuvers for bleeding control have failed and the situation is deteriorating [37–39, 42].

An early damage control leads to survival of up to 67% of patients, versus only 2% when used as an extreme ratio [36].

Surgical strategy of damage control includes packing technique: Folded laparotomy packs are inserted around the liver, pushing it posteriorly against diaphragmatic surface; some authors suggested to take down the falciform ligament, even if it is a debatable maneuver (**Figure 6**) [23]. Packing must be performed correctly, because a wrong pads positioning could worsen hemorrhage: it is important not to place pads within the liver injury and avoid to pack so tightly that inferior vena cava could be compressed and venous return impaired [23, 36, 37, 43].



Figure 6. CT scan after damage control surgery.

If these maneuvers prove to be effective, the operation should be stopped and the patient taken to intensive care unit, where a close observation can be performed.

Perihepatic packing can completely control low pressure bleeding, originated from portal and hepatic veins, but it is not resolute for arterial bleeding. For this reasons, many authors suggest the use of postoperative angiography and eventual selective embolization after damage control surgery because more than 50% of patients demonstrate bleeding at postoperative radiographic control. Even if prospective studies are needed, arterial embolization should be incorporated in the management of bleeding after perihepatic packing [38, 42, 43]. Intra-operative selective ligation of hepatic artery should be avoided for the high risk of parenchymal and gallbladder necrosis and reserved for selective cases of massive uncontrollable bleeding [38, 40, 43].

Once the physiologic status of the patients has been restored, packing removal can be considered. The optimal timing of pads removal is still controversial, but it is evident that if performed before 24 h, risk of re-bleeding is very high. On the other hand, packing removal after 72 h increases risk of sepsis; even if not yet well defined, 48 h seem to be the best time for second look [36, 37, 40].

If bleeding persists after damage control procedures, Pringle maneuver should be applied placing an atraumatic clamp, or preferably a double vessel loop, around the liver pedicle. This can control bleeding from hepatic artery or portal vein; if hemorrhage persists after hilar clamp, a juxtahepatic venous injury should be suspected [37].

Pringle maneuver must be intermittent, clamping the porta hepatis for 10–15 min and taking it off for 5 min, to avoid parenchymal ischemic suffering and postoperative liver failure [43].

Once bleeding is controlled, injured vessels and bile ducts must be identified and sutured. It is suitable to avoid deep rough suture, especially in profound lesions, for the high risk of main vessel ligations, abscess and hematomas/bilomas formations, and late bleeding. It is advisable to identify the injured structures and selectively repair them; to allow this maneuver, it could be necessary to extend the parenchymal lesion using the finger fracture technique or sometimes a stapling device [37, 43].

To control the hemorrhage, Stone and Lamb proposed the insertion of a viable omental pedicle into the parenchymal injury. It seems to have an effective hemostatic effect, filling the “dead space” with vascularized tissue, introducing peritoneal macrophages into a potential area of sepsis, and providing stromal cell-derived factor 1-alpha which may recruits chemokine receptor cells vital for healing [36, 38].

An issue that in the last years raised great discussions is the role of liver resection in the management of hepatic trauma. Advances in operative techniques lead to decrease mortality for liver resection from 80% in 1900 to less than 20% [37, 38]. Moreover, anatomic resection of an injured segment definitively controls bleeding and potential bile leak and removes devitalized tissue; however, its employment is still controversial.

Polanco et al. reported in a great series of liver resection for liver trauma a mortality lower than 9% and morbidity lower than 30% [39, 44].

Some authors assert that the majority of liver resections performed for hepatic trauma can be considered as resectional debridements of destroyed parenchyma rather than formal hepatic resection [39].

Therefore, simple maneuvers with packing and damage control are the treatment of choice, and the recourse to liver resections must be considered when simple methods fail to successfully control parenchymal bleeding and should, however, be performed by expert liver surgeons.

Finally, hepatic resection can be evaluated at re-exploration to remove injured or necrotic parenchyma as definitive treatment [37, 39, 43].

Juxtahepatic venous injury (grade 5 lesions) represents the most challenging and most deadly form of liver trauma, even if it is a rare event. The patients may be stable and, in rare cases, treated non-operatively, but 92% of them needs urgent operation. The mortality rate described is between 33 and 81%, mainly due to the surgical inaccessible location of the retrohepatic vena cava or hepatic veins [37, 45].

Buckman et al. [46] described two types of iuxtahepatic lesions: Type A includes lesions of the intraparenchymal tract of hepatic veins, usually associated with tissue disruption, most often involving the central posterior portion of the liver. This is the most common lesion and hemorrhage is well controlled in most of cases with perihepatic packing or simple suturing. It is not advisable to mobilize the liver and expose hepatic veins to perform a direct repair of the vessels, for the high risk of bleeding worsening [36, 38, 43, 46].

The second pattern, type B, is represented by extra-parenchymal venous lesion, and it is frequently associated to disruption of suspensory ligaments of the liver or diaphragm. It often leads to an uncontained hemorrhage and may require total vascular isolation by clamping inferior vena cava above and under the liver. In this case, mobilization of the liver is mandatory, and it may be necessary to extend incisions cutting the diaphragm to achieve a correct exposure of the injured vessel. Venovenous bypass or atriocava shunt are not advisable in the management of these injuries [36–38, 46].

Liver transplantation may be an option in rare cases when there is a lesion of the main portal triad structure that cannot be repaired (grade 6 lesion), or in the presence of an uncontrollable bleeding despite the use of all the procedures described above. A further indication can be postoperative liver acute failure [36, 41, 43].

3.4. Operative treatment complications

Complications of surgical treatment of liver trauma include parenchymal necrosis with hepatic abscess, sepsis, re-bleeding, and bile leakage [41, 47].

Re-starting of hemorrhage should possibly be treated with angiography and arterial embolization, rather than an adjunctive surgical intervention.

If bile leakage and biloma are present, less invasive procedure as ERCP or radiologic drainage is the first choice, unless it may occur a peritonitis for choleperitoneum that requires re-laparotomy [41].

Parenchymal necrosis is a less frequent complication and occurs when a destroyed parenchyma is left in situ after damage control, but can be related to a deep suture closing important vessels, or to hepatic artery ligation/embolization. It can be treated non-operatively, but sometimes, an abscess can develop, and a percutaneous or surgical drainage is necessary [41, 47].

4. Conclusions

Scientific evidence has shown that more than 67% of laparotomies performed for blunt liver trauma are not therapeutic and in about 86% of all post-traumatic hepatic lesions the hemorrhage stopped spontaneously at the time of the laparotomy itself. By contrast, blunt hepatic trauma can be conservatively treated in 70–90% of hemodynamic stable patients, in the absence of other associated lesions/peritoneal signs or in the case of limited need for transfusions. Thus far, the main decision criteria for a surgical rather than a conservative approach to the management of liver trauma have been the hemodynamic stability and not the American Association for the Surgery of Trauma–Organ Injury Scale (AAST-OIS) grading of the lesion. Restricting the indications for a conservative treatment of liver traumas solely to the relief of hemodynamic stability seems nowadays to be unreasonable. Criteria for a conservative treatment are as follows: hemodynamic stability, limited need of blood transfusions, and non-associated visceral lesions on CT scan. In the presence of these conditions, non-operative approach can be undertaken, and the cornerstone of this treatment is transarterial angiographic embolization. Patients with complex liver trauma after stabilization should be sent to a referring, specialized center where a dedicated team is present. Nowadays, non-operative treatment is the gold standard for most of the patients with blunt liver injuries and the role of interventional radiologist is more and more determinant in the outcome of these patients.

Author details

Marco Massani*, Luca Bonariol, Bruno Pauletti, Cesare Ruffolo, Roberta Bonariol, Ezio Caratozzolo and Nicolo' Bassi

*Address all correspondence to: mmassani@ulss.tv.it

IV Department of Surgery, Center for HPB Surgery, Treviso Regional Hospital, Treviso, Italy

References

- [1] Tinkoff G, Esposito TJ, Reed J, et al. American Association for the Surgery of Trauma Organ Injury Scale I: spleen, liver, and kidney, validation based on the national trauma Data Bank. *J Am College of Surgeon*. 2009; 207: 646–655.
- [2] Richardson JD. Changes in the management of injuries to the liver and spleen. *J Am Coll Surg*. 2005; 200: 648–669.
- [3] Joosse P, Soedarmo S, Luitse JS, Ponsen KJ. Trauma outcome analysis of a Jakarta University using the TRISS method: validation and limitation in comparison with the

major trauma outcome study. Trauma and Injury Severity Score. J Trauma. 2001; 51(1): 134–40.

- [4] Baker SP, O'Neill B, Haddon W, Long WB. The injury severity score: a method for describing patients with multiple injuries and evaluating emergency care. J Trauma 1974; 14(3): 187–196.
- [5] Moore EE, Cogbill TH, Jurkovich GJ, Shackford SR, Malangoni MA, Champion HR. OIS: spleen and liver (1994 revision). J Trauma 1995; 38: 323–324.
- [6] Mirvis SE, Whitley NO, Vainwrigth JR, et al. Blunt hepatic trauma in adults: CT based classification and correlation with prognosis and treatment. Radiology. 1989; 171: 27–32.
- [7] Karp MP, Cooney DR, Pros GA, et al. The nonoperative management of pediatric hepatic trauma. J Pediatr Surg 1983; 18: 512–518.
- [8] Pachter HL, Hofstetter SR. The current status of non operative management of adult blunt hepatic injuries. Am J Surg 1995; 169: 442–454.
- [9] Malhotra AK, Fabian TC, Croce MA, Gavin TJ, et al. Blunt Hepatic Injury: a paradigm shift from operative to non operative management in the 1990s. Ann Surg. 2000; 231(6): 804–813.
- [10] Richardson JD, Franklin GA, Lukan JK, Carrillo EH, et al. Evolution in the management of hepatic trauma: a 25-year perspective. Ann Surg. 2000; 232(3): 324–330.
- [11] Hurtuk M, Reed RL, Esposito TJ, Davis KA, Luchette FA. Trauma surgeons practice what they preach: the NTDB story on solid organ injury management. J Trauma. 2006; 61(2):243–54; discussion 254–5.
- [12] Patricio MP, Brown JB, Pujana JC, et al. The swinging pendulum: a National perspective of non operative management in severe blunt liver injury. J Trauma Acute Care Surg. 2013; 75(4): 590–595.
- [13] Kozar RA, Moore FA, Niles SE, et al. Complications of non operative management of high-grade blunt hepatic injuries. J Trauma. 2005; 59(5): 1066–1071.
- [14] Van der Wilden GM, Velhamos GC, Emhoff T, Brancato S, Adams C, Georgakis G, Jacobs L, Gross R, Agarwal S, Burke P, et al. Successful non operative management of the most severe blunt liver incurie: a multicenter study of the research consortium of new England centers for Trauma. JAMA Surg. 2012; 147: 423–429.
- [15] Bonariol L, Massani M, Caratozzolo E, Ruffolo C, et al. Management of grade III IV blunt liver traumas: a comparative, observational study. Minerva Chirurgica. 2015; 70(1): 7–15.
- [16] Fang JF, Chen RJ, Wong YC, et al. Classification and treatment of pooling of contrast material in computed tomography scan of blunt hepatic injury. J Trauma. 2000; 49:1083–1088.

- [17] Alarhayem AQ, Myers JG, Dent D, Lamus D, Lopera J, et al. "Blush at first sight": significance of computed tomographic and angiographic discrepancy in patients with blunt abdominal trauma. *Am J Surg*. 2015; 210: 1104–1111.
- [18] Hagiwara A, Murata A, Matsuda T, Matsuda H. The efficacy and limitations of transarterial embolization for severe hepatic injury. *J Trauma*. 2002; 52(6): 1091–1096.
- [19] Stassen NA, Bhullar I, Cheng JD, et al. Nonoperative management of blunt hepatic injury: an Eastern Association for the Surgery of trauma practice management guideline. *J Trauma Acute Care Surg*. 2012; 73(5 Suppl. 4): S288–S293.
- [20] Poletti PA, Mirvis SE, Shanmuganathan K, et al. CT criteria for management of blunt liver trauma: correlation with angiographic and surgical findings. *Emerg Radiol*. 2000; 216(2): 418–427.
- [21] Carillo E, Spain D, Wohltmann D, et al. Interventional techniques are useful adjuncts in non operative management of hepatic injury. *J Trauma*. 1999; 46: 619–622.
- [22] Sivrikoz E, Teixeira PG, Resnick S, Inaba K, et al. Angiointervention: an independent predictor of survival in high grade blunt liver injuries. *Am J Surg*. 2015; 209(4): 742–746.
- [23] Ward J, Alarcon L, Peitzman AB. Management of blunt liver injuries: what is new. *Eur J Trauma*. 2015; 41: 229–237.
- [24] Mohr AM, Lavery RF, Barone A, et al. Angiographic embolization for liver injuries: low mortality, high morbidity. *J Trauma*. 2003; 55: 1077–1081.
- [25] Letoublon C, Morra I, Chen Y, et al. Hepatic arterial embolization in the management of blunt hepatic trauma: indications and complications. *J Trauma Injury Infect Crit Care*. 2011; 70(5): 1032–1037.
- [26] Dabbs DN, Stein DM, Scalea TM. Major hepatic necrosis: a common complication after angioembolization for treatment of high grade liver injuries. *J Trauma*. 2009; 66: 621–627.
- [27] Kozar RA, Moore FA, Moore EE, et al. Western Trauma Association critical decision in Trauma: Non operative management of adult blunt hepatic Trauma. *J Trauma Injury Infect Crit Care*. 2009; 67(6): 1144–1149.
- [28] Swaid F, Peleg K, Alfici R, Matter I, et al. Concomitant hollow viscus injuries in patients with blunt hepatic and splenic injuries; an analysis of a National Trauma Registry database. *Injury*. 2014; 45: 1409–1412.
- [29] Boese CK, Hackl M, Muller P, Ruchholtz S, Michael F, Lechler P. Nonoperative management of blunt hepatic trauma: a systematic review. *J Trauma Acute Care Surg*. 2015; 79(4): 654–660.

- [30] Fang JF, Wong YC, Lin BC, Hsu YP, Chen MF. The CT risk factors for the need of operative treatment in initially hemodynamically stable patients after blunt hepatic trauma. *J Trauma*. 2006; 61: 547–554.
- [31] Peitzman AB, Ferrada P, Puyana JC. Nonoperative management of blunt abdominal trauma: have we gone too far. *Surg Infect*. 2009; 10(5): 427–433.
- [32] Kozar RA, Moore FA, Cothren C, Moore EE, et al. Risk factors for hepatic morbidity following non operative management. *Arch Surg*. 2006; 141: 451–459.
- [33] Patcher HL, Knudson MM, Ross S, Hoyt D, Cogbill T. Status of non operative management of blunt liver trauma in 1995: a multicenter experience with 404 patients. *J Trauma*. 1996; 40: 31–38.
- [34] Carrillo EH, Wohltmann C, Richardson JD, Polk HC Jr. Evolution in the treatment of complex blunt liver injuries. *Curr Probl Surg*. 2001; 38: 1–60.
- [35] Cox JC, Fabian TC, Maish GO 3rd, Bee TK, Pritchard FE, Russ SE, Grieger D, Winestone MI, Zarzaur BL Jr, Croce MA, et al. Routine follow-up imaging is unnecessary in the management of blunt hepatic injury. *J Trauma*. 2005; 59(5): 1175–1180.
- [36] Pachter HL. Prometheus bound: evolution in the management of hepatic trauma – from myth to reality. *Trauma Acute Care Surg*. 2012; 72(2): 321–329.
- [37] Piper GL, Peitzman AB. Current management of hepatic trauma. *Surg Clin North Am*. 2010; 90(4): 775–785.
- [38] Kozar RA, Feliciano DV, Moore EE, Moore FA, Cocanour CS, West MA, Davis JW, McIntyre RC Jr. Western Trauma Association/critical decisions in trauma: operative management of adult blunt hepatic trauma. *J Trauma*. 2011; 71(1): 1–5.
- [39] Polanco P, Leon S, Pineda J, Puyana JC, Ochoa JB, Alarcon L, Harbrecht BG, Geller D, Peitzman AB. Hepatic resection in the management of complex injury to the liver. *J Trauma*. 2008; 65(6): 1264–1269; discussion 1269–70. doi: 10.1097/TA.0b013e3181904749.
- [40] Lin BC, Fang JF, Chen RJ, Wong YC, Hsu YP. Surgical management and outcome of blunt major liver injuries: experience of damage control laparotomy with perihepatic packing in one trauma centre. *Injury*. 2014; 45(1): 122–127.
- [41] Li Petri S, Gruttadauria S, Pagano D, Echeverri GJ, Di Francesco F, Cintonino D, Spada M, Gridelli B. Surgical management of complex liver trauma: a single liver transplant center experience. *Am Surg*. 2012; 78(1): 20–25.
- [42] Asensio JA, Petrone P, García-Núñez L, Kimbrell B, Kuncir E. Multidisciplinary approach for the management of complex hepatic injuries AAST-OIS grades IV-V: a prospective study. *Scand J Surg*. 2007; 96(3): 214–220.
- [43] Peitzman AB, Marsh JW. Advanced operative techniques in the management of complex liver injury. *J Trauma Acute Care Surg*. 2012; 73(3): 765–770.

- [44] Asensio JA, Roldán G, Petrone P, Rojo E, Tillou A, Kuncir E, Demetriades D, Velmahos G, Murray J, Shoemaker WC, Berne TV, Chan L. Operative management and outcomes in 103 AAST-OIS grades IV and V complex hepatic injuries: trauma surgeons still need to operate, but angioembolization helps. *J Trauma*. 2003; 54(4): 647–653.
- [45] Prichayudh S, Sirinawin C, Sriussadaporn S, Pak-art R, Kritayakirana K, Samorn P, Sriussadaporn S. Management of liver injuries: predictors for the need of operation and damage control surgery. *Injury*. 2014; 45(9): 1373–1377.
- [46] Buckman RF Jr, Miraliakbari R, Badellino MM. Juxtahepatic venous injuries: a critical review of reported management strategies. *J Trauma*. 2000; 48(5): 978–984.
- [47] MacKenzie S, Kortbeek JB, Mulloy R, Hameed SM. Recent experiences with a multi-disciplinary approach to complex hepatic trauma. *Injury*. 2004; 35(9): 869–877.