

Advances in Diagnosis and Clinical Management Strategies for Liver Trauma

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Abstract: Liver trauma, a common and severe surgical emergency, refers to injuries caused by various external forces acting on the liver. The causes of liver trauma are diverse, with direct external impacts such as traffic accidents, falls from heights, and violent collisions being frequent contributors. Additionally, iatrogenic procedures like improper liver biopsy techniques may also lead to liver trauma. Clinically, symptoms vary depending on the severity and extent of injury. Mild cases might present with localized symptoms like right upper abdominal pain and tenderness, while severe cases could result in massive intra-abdominal bleeding accompanied by shock symptoms, including pallor, rapid heart rate, and hypotension, along with gastrointestinal manifestations such as nausea and vomiting. Treatment approaches differ significantly: For minor injuries, conservative management typically involves bed rest, close monitoring of vital signs, hemostasis, and anti-infection measures. In critical scenarios involving liver rupture or massive hemorrhage, immediate surgical intervention is required to repair liver damage and control bleeding.

Keywords: Liver trauma; Diagnosis; Clinical treatment strategy

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1. Introduction

The etiology of liver trauma encompasses multiple dimensions. Regarding causative factors, traffic accidents, falls from heights, violent conflicts, and industrial accidents may all contribute to liver injuries. In terms of risk factors, excessive alcohol consumption can impair normal liver function, making the liver more vulnerable to injury when subjected to external impacts. Individuals with pre-existing liver conditions often have structural abnormalities or pathological changes in their liver tissue, significantly increasing the risk of liver damage during similar traumatic events ^[1]. Additionally, high-risk occupational groups such as construction workers and miners face elevated risks of liver trauma due to exposure to hazardous working environments.

2. Pathogenesis and pathophysiology

The pathophysiology of liver trauma is centered on a vicious cycle of hemorrhage-inflammation-metabolic failure. Early intervention should focus on hemostasis, anti-shock measures, and infection prevention, while dynamically assessing hepatic regeneration capacity and systemic multi-system functions to halt disease progression and improve prognosis ^[2].

2.1. Acute hemorrhage and circulatory failure

- (1) Vascular rupture: Damage to the hepatic artery (high-pressure system) and portal vein (high blood flow) causes rapid blood loss, leading to hypovolemic shock.
- (2) Coagulation disorders: Hepatocyte injury reduces synthesis of clotting factors (e.g., II, VII, IX, X), exacerbating bleeding tendencies.

2.2. Inflammatory response and microcirculation disorders

- (1) Cytokine release: Pro-inflammatory factors like TNF- α and IL-6 activate neutrophil infiltration, intensifying tissue edema and microthrombus formation.
- (2) Ischemia-reperfusion injury: Reactive oxygen species (ROS) surge after blood flow restoration, further damaging hepatocyte mitochondrial function.

2.3. Bile exudation and secondary injury

Biliary tract injury: Bile leakage into the peritoneal cavity triggers chemical peritonitis, causing severe abdominal pain and increasing bacterial infection risks.

2.4. Metabolic and detoxification failure

- (1) Glucose metabolism disorders: Depletion of hepatic glycogen reserves leads to hypoglycemia, while impaired lactate clearance causes acidosis.
- (2) Detoxification dysfunction: Accumulation of blood ammonia and bilirubin may induce hepatic encephalopathy (consciousness impairment) or jaundice.

2.5. Complication progression

- (1) Infectious complications: Bile leakage or secondary bacterial infections (e.g., *Escherichia coli*) from necrotic tissues may result in liver abscesses or sepsis.
- (2) Delayed bleeding: Pseudoaneurysm or arteriovenous fistula formation, which may rupture and bleed days to weeks after injury.
- (3) Liver failure: Multiple organ failure, such as coagulation disorder and hepatorenal syndrome, occurs when extensive liver necrosis exceeds regeneration capacity.

3. Diagnosis

Open liver injuries are relatively straightforward to diagnose, but clinicians must also consider the possibility of combined thoracoabdominal trauma. Closed injuries presenting with typical hemorrhagic shock and peritoneal irritation signs, when combined with a history of trauma, can be easily identified. However, patients with

multiple injuries, such as unconsciousness from head trauma, multiple fractures complicated by shock, or elderly individuals with delayed responses due to frailty, require heightened vigilance to avoid missed diagnoses. Patients with cirrhosis or liver cancer may experience hepatic rupture from minor trauma, which demands careful evaluation. The determination of whether closed abdominal injuries involve liver damage significantly impacts the need for open surgery, thus requiring high diagnostic accuracy ^[3].

3.1. Clinical manifestations

Subcapsular hemorrhage and/or liver parenchymal contusion primarily present with hepatic pain and hepatomegaly, with minimal peritoneal irritation signs. The pain gradually subsides as vital signs stabilize. In cases of high-tension subcapsular hematomas, delayed acute abdominal pain and internal bleeding may occur. True rupture is characterized by massive internal bleeding, potentially manifesting as biliary peritonitis with right upper quadrant pain radiating to the right chest and shoulder. The peritonitis progresses from the right upper quadrant to involve the entire abdomen. Severe hemorrhage and biliary peritonitis may occur with major vessel tears, leading to early shock. Some patients with liver trauma may experience hematemesis or melena when blood from damaged liver tissue enters the duodenum via bile ducts.

3.2. Ancillary examinations

3.2.1. Laboratory tests

- (1) Blood tests show leukocytosis with progressive decreases in red blood cell count, hemoglobin, and hematocrit levels.
- (2) Coagulation parameters (prothrombin time, activated partial thromboplastin time) may be prolonged and international normalized ratio (INR) elevated due to impaired liver synthesis of clotting factors.
- (3) Liver function tests reveal significantly elevated ALT and AST levels during hepatocyte injury, particularly after blunt trauma. Elevated bilirubin levels suggest cholestasis or biliary tract injury, especially when total bilirubin and direct bilirubin are high. Renal function tests (blood urea nitrogen and creatinine) may indicate pre-renal renal insufficiency caused by hypovolemia or subsequent multi-organ failure.
- (4) Blood gas analysis shows increased lactate levels, indicating tissue hypoperfusion, metabolic acidosis in shock, or hypovolemic conditions. Blood gas analysis, including pH, BE (alkaline residue), and HCO₃, can reflect metabolic status and help assess the severity of shock.
- (5) Inflammatory markers such as CRP and PCT may be used to monitor infection complications like secondary hepatic abscess or peritonitis.

3.2.2. Imaging examinations ultrasound

- (1) Rapid assessment of intra-abdominal hemorrhage is commonly used for emergency initial screening.
Indications: Preliminary screening for hemodynamically unstable patients.
Advantages: Fast, non-invasive, repeatable, with 85% sensitivity in detecting free fluid in the abdominal cavity.
Limitations: Cannot clearly grade liver injury, may miss minor bleeding or retroperitoneal injuries.
- (2) CT: Contrast-enhanced CT scans (gold standard) can detail liver injury extent, active bleeding, hematomas, cholesteatoma, and assess other abdominal organ injuries.
Diagnostic value: Injury grading: Clarifies AAST classification, identifies depth of liver laceration,

hematoma range, and vascular damage.

Complication screening: Cholesteatoma, pseudoaneurysm, hepatic abscess.

- (3) Angiography: Used for suspected active bleeding or vascular injury, allowing simultaneous embolization therapy.

Indications: CT-predicted active bleeding or suspected vascular injury (e.g., hepatic artery pseudoaneurysm).

Therapeutic value: Simultaneous embolization (gelatin sponge, coil) achieves > 90% hemostasis success rate.

- (4) MRI: Less frequently used but useful for specific conditions like biliary tract injury.

Indications: Suspected bile duct injury (e.g., biliary leakage) or unclear CT findings.

Advantages: Non-radiation, clear visualization of biliary tree structure (MRCP), intrahepatic hematoma evolution (subacute phase).

Limitations: The procedure takes longer and is not suitable for emergency or critically ill patients.

- (5) Diagnostic peritoneal puncture or lavage: For patients with hemodynamic instability, this method can rapidly confirm the presence of intra-abdominal hemorrhage.

Indications: When FAST is negative but clinical suspicion of peritoneal bleeding is high, or when imaging is unavailable.

Positive criteria: Extracting > 10 mL non-clotted blood or detecting red blood cells > 100,000/mm³ in lavage fluid.

Limitations: Cannot pinpoint the source of bleeding, and may yield false negatives due to localized peritumoral hematoma.

4. Treatment

4.1. Traditional surgical approaches

Traditional surgical methods play a crucial role in managing liver trauma, primarily including liver suturing, hepatic resection, and packing procedures. Liver suturing stands as one of the most commonly used techniques, particularly suitable for patients with parenchymal lacerations that do not involve major blood vessels or bile ducts. Studies indicate its adoption rate reaches 51.2% in liver injury cases, especially right lobe injuries. Hepatic resection is indicated for severe parenchymal damage or uncontrollable bleeding, though surgeons must carefully balance surgical invasiveness against postoperative liver compensatory capacity. Packing procedures (such as perihilar packing) are typically employed for hemodynamically unstable patients to control bleeding and buy time for subsequent treatment, though they carry higher complication risks like infection and rebleeding. Additionally, hepatic artery ligation may be considered for specific cases but carries potential long-term complications such as pseudoaneurysms or collateral circulation formation leading to biliary bleeding. The primary advantage of surgical intervention lies in rapid hemorrhage control, which proves critical for hemodynamically unstable or high-grade (AAST IV-V) liver trauma patients. For instance, in penetrating liver injuries with active bleeding, hepatic artery ligation or packing can significantly reduce early mortality rates (22.7%). However, these procedures involve substantial surgical trauma, increased postoperative complications (including hepatic necrosis [16%], bile leakage [1.5%-4.5%], and infection risks), and extended hospital stays (averaging 11 days).

Furthermore, obesity-related higher surgical intervention rates may correlate with anatomical complexity

and slower postoperative recovery. Non-surgical management (NOM) demonstrates success rates of 85%-99% in stable patients, though surgery remains the irreplaceable salvage intervention, particularly when complicated by multi-organ injuries or vascular abnormalities such as hepatic arteriovenous fistulas, requiring multidisciplinary collaboration ^[4].

4.2 Interventional

Therapy Endovascular therapy plays a pivotal role in managing liver trauma and complications, with transarterial embolization (TAE) being a core technique. Clinical evidence shows TAE demonstrates significant efficacy in emergency scenarios including traumatic hepatic artery tears, pseudoaneurysm rupture, and spontaneous hepatic rupture associated with HELLP syndrome. For post-liver transplant pseudoaneurysm rupture, combined embolization with restorative balloon occlusion (REBOA) serves as a transitional treatment, although some patients still face prognostic risks due to multiple organ failure. The core advantage of interventional therapy lies in its minimally invasive nature, avoiding the trauma and prolonged recovery associated with traditional open surgery. For instance, TAE can be performed via percutaneous femoral artery puncture, making it particularly suitable for hemodynamically unstable emergency cases.

However, limitations must be acknowledged:

- (1) The technical threshold is high, requiring interventional radiologists to master vascular anatomy and catheterization skills proficiently, with significant challenges in managing complex scenarios like multi-vessel hemorrhage or combined portal vein thrombosis.
- (2) Certain patients may experience complications such as postembolism hepatic ischemia leading to liver dysfunction or biliary ischemic injury. Furthermore, long-term efficacy is influenced by underlying conditions, such as the higher recurrence risk in patients with malignant tumors following pseudoaneurysm embolization.
- (3) Interventional therapy often requires multidisciplinary collaboration (e.g., trauma surgery and transplant teams) to develop stepwise treatment plans ^[5,6]. Overall, interventional therapy remains a crucial option for hepatic vascular lesions, but strict patient-specific risk assessment is essential.

4.3 Non-surgical strategies

Nonoperative management (NOM) has become the standard treatment for hemodynamically stable blunt liver injuries, particularly for injuries graded I-III according to the American Society of Trauma Surgeons (AAST) classification. Indications include: hemodynamic stability (systolic blood pressure > 90 mmHg), absence of peritoneal irritation signs, and no other emergency laparotomy complications (e.g., bowel perforation) ^[7]. Multiple studies demonstrate that even high-grade (IV-V) liver injuries can be safely managed through NOM under strict monitoring, achieving success rates exceeding 85%. Contraindications include persistent hemodynamic instability, intra-abdominal organ injuries requiring surgical intervention, and imaging evidence of active contrast agent extravasation (indicating arterial bleeding). The core advantage of NOM lies in avoiding surgical trauma and related complications (e.g., infection, adhesive intestinal obstruction), especially reducing secondary trauma in patients with multiple injuries. Angioembolization (AE), a crucial adjunct to neurointerventional surgery (NOM), effectively controls arterial hemorrhage. However, it carries a 43% complication rate, including hepatic infarction, biliary ischemia, and abscess formation. Delayed bleeding, the primary risk of NOM, typically occurs within 72 hours post-injury and may result from incomplete embolization or open collateral circulation. Additionally, NOM requires intensive monitoring (e.g., continuous CT scans and laboratory tests), potentially prolonging hospital

stays. Novel therapeutic approaches like melatonin, which modulates mitochondrial-endothelial contact (MAMs) to reduce post-traumatic liver injury, offer new adjunctive treatment options^[8]. Overall, successful NOM relies on multidisciplinary collaboration (trauma surgery, interventional radiology, ICU) while balancing its dual effects of reducing surgical risks and preventing delayed complications.

5. Complications

5.1. Early posttraumatic liver complications (Within 72 hours post-injury)

Early post-traumatic liver complications are primarily characterized by acute hemorrhage, infection, and organ dysfunction. Hemorrhagic shock is the most lethal complication due to the liver's high blood supply. Tissue tears of grade III or higher or major vascular injuries can cause blood loss exceeding 2000 mL, requiring emergency surgical hemostasis or interventional embolization. Bile leakage occurs in 10–25% of cases, typically caused by ruptured intrahepatic bile ducts. Bile entering the peritoneal cavity may induce chemical peritonitis, presenting with severe abdominal pain, peritoneal irritation signs, and elevated bilirubin levels in bloody ascites. Infectious complications include liver abscesses (5%-10% incidence) and peritoneal infections, with significantly increased risks during open injuries or combined intestinal ruptures. Coagulation disorders result from reduced synthesis of clotting factors (II, VII, IX, X) due to hepatocyte damage, exacerbated by transfusion-induced dilutional coagulopathy. Adjacent organ injuries often involve diaphragmatic rupture, right-sided hemothorax/pneumothorax, or renal contusions, requiring imaging evaluation. This stage demands close monitoring of vital signs, timely fluid resuscitation, broad-spectrum antibiotics, and vigilance for delayed bleeding^[9].

5.2. Late posttraumatic liver complications (2-24 months post-injury)

Late-stage complications are frequently associated with abnormal wound healing, biliary system injury, and hemodynamic changes. Traumatic biliary strictures may cause obstructive jaundice due to scar contraction, requiring ERCP or biliary-enteric anastomosis. Gallbladder tumors manifest as right upper quadrant masses with elevated alkaline phosphatase levels, treatable by percutaneous drainage or surgical resection. Portal hypertension, caused by injury to the hepatic or main portal vein, leads to esophageal and gastric varices with ascites, requiring TIPS (Transjugular Intrahepatic Shunt) or shunt surgery. Hepatic pseudoaneurysms exhibit characteristic “rapid entry and exit” enhancement in contrast-enhanced CT scans during the arterial phase, with a rupture risk as high as 50%, necessitating interventional embolization therapy. Liver failure predominantly occurs in patients with pre-existing liver disease or hepatic resection exceeding 50%, manifesting as jaundice, hepatic encephalopathy, and worsening coagulation parameters. Additionally, some patients develop chronic abdominal pain associated with perihepatic adhesions, localized peritonitis, or traumatic neuropathy. Advanced complications often require multidisciplinary collaboration, combined with imaging follow-ups (ultrasound/CT/MRCP) and liver function monitoring. Timely intervention can significantly improve prognosis^[10,11].

6. Conclusion

Current treatment challenges primarily focus on optimizing individualized decision-making. For instance, while Non-Operational Management (NOM) has become the standard protocol for low-grade liver injury, its application in high-grade injuries remains controversial. Studies indicate that patients with high-grade injuries

may experience complications (such as delayed bleeding and biliary fistula) at 9.7% after NOM, compared to 45.5% in surgical groups. Additionally, interventional therapies like arterial embolization, while effective in controlling bleeding, require advanced techniques and need more evidence regarding long-term outcomes for intrahepatic bile duct injuries. Emerging technologies such as mesenchymal stem cell-derived exosomes (MSC-exos) show potential in reducing hepatic injury inflammation through gut microbiota regulation and metabolites (e.g., 6-methylnicotinamide and glutathione) in animal experiments, though clinical translation requires further validation.

Future research should prioritize developing precision risk stratification tools (integrating injury grading, hemodynamic parameters, and biomarkers) and innovating minimally invasive techniques. For example, the “suspension technique” in robot-assisted hepatectomy can reduce parenchymal traction injuries, while novel hemostatic materials (e.g., extracellular matrix-modified gelatin sponge) demonstrate superior biocompatibility and regenerative capacity in animal models compared to traditional gelatin sponges. For extreme cases (e.g., hepatic artery rupture or post-transplant pseudotumor), optimizing multidisciplinary strategies (including interventional embolization, injury control surgery, and liver transplantation) will further enhance treatment success rates. In general, the treatment of liver trauma is moving towards individualization, minimally invasive and multimodal integration, but more prospective studies are needed to balance efficacy and safety.

Disclosure statement

The authors declare no conflict of interest.

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