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Outcomes of abdominal surgery in patients with liver cirrhosis

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Abstract

Patients suffering from liver cirrhosis (LC) frequently require non-hepatic abdominal surgery, even before liver transplantation. LC is an important risk factor itself for surgery, due to the higher than average associated morbidity and mortality. This high surgical risk occurs because of the pathophysiology of liver disease itself and to the presence of contributing factors, such as coagulopathy, poor nutritional status, adaptive immune dysfunction, cirrhotic cardiomyopathy, and renal and pulmonary dysfunction, which all lead to poor outcomes. Careful evaluation of these factors and the degree of liver disease can help to reduce the development of complications both during and after abdominal surgery. In the emergency setting, with the presence of decompensated LC, alcoholic hepatitis, severe/advanced LC, and significant extrahepatic organ dysfunction conservative management is preferred. A multidisciplinary, individualized, and specialized approach can improve outcomes; preoperative optimization after risk stratification and careful management are mandatory before surgery. Laparoscopic techniques can also improve outcomes. We review the impact of LC on surgical outcome in non-hepatic abdominal surgeries required in this cirrhotic population before, during, and after surgery.

Key words: Liver cirrhosis; Outcomes; Coagulopathy; Nutritional status; Abdominal surgery; Adaptive
The prevalence of chronic liver disease is increasing. Patients with liver cirrhosis may be more likely to need non-hepatic abdominal surgery than the non-cirrhotic population. The incidence of non-alcoholic fatty liver disease and non-alcoholic steatohepatitis may increase due to alcohol consumption and the potential high rates of hepatitis C virus (HCV) and hepatitis B virus (HBV) in coming years in European countries. In addition, the obesity epidemic in Western countries, which is associated with metabolic syndrome, is expected to generate a large cohort of patients with non-alcoholic steatohepatitis (NASH) and non-alcoholic fatty liver disease (NAFLD) that may potentially change the etiological pattern of LC.

LC patients may require surgery for abdominal wall hernia, gallstones, peptic ulcer disease, biliary, small bowel, colo-rectal, and pancreatic diseases as well as liver procedures, such as transplantation. Surgery represents an additional source of stress for compromised liver function, and perioperative complications frequently appear in spite of significant advances in surgical and intensive care management. Preoperative risk stratification is difficult due to the limited accuracy of the tools available to assess LC, and, in some cases, due to the absence of diagnosis. The mortality and morbidity risks are associated with the severity of the underlying LC. This is one of the factors that explains the wide variation in outcomes recorded by different studies, in addition to patient characteristics, the surgical approach used, and the levels of expertise of the surgeons, anesthetists, and intensive care unit (ICU) staff. Liver function is usually assessed by the Child-Turcott-Pugh (CTP) and Model for End-Stage Liver Disease (MELD) scores. Early studies found 30 d mortality rates after surgery were 10% in CTP-A, 30% in CTP-B, and 76%-82% in CTP-C, figures that have not significantly changed in more recent assessments. Despite the poor results, advances in the medical management of LC and life expectancy have increased the eligibility of these patients for abdominal surgery. To be able to give definitive recommendations and indications for non-hepatic abdominal surgery in the cirrhotic population, it is important to identify the patients most likely to benefit from it. There is also a need to assess contemporary surgical techniques and the various scoring systems currently in use.

This review summarizes the outcomes of patients with LC undergoing non-hepatic abdominal surgery. Indexed articles in Medline of series of patients with LC who underwent non-hepatic abdominal surgery between 1950 and March 2014 were reviewed using the OVID interface. We aimed to select manuscripts addressing outcome based on the degree of LC assessed with MELD and/or CTP scores. Articles addressing the pathophysiology of cirrhotic patients and the clinical implications in non-hepatic abdominal surgery were selected based on their importance, their date of publication, and the citations of the manuscripts. As for articles describing the different types of surgery in LC patients, the most recent publications were selected in order to preserve comparability between contemporary surgical techniques. For these reasons, the present review does not follow the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) statement.

**PREOPERATIVE MANAGEMENT**

**AND PATHOPHYSIOLOGICAL CONSIDERATIONS OF LC BEFORE SURGERY**

The estimation of liver functional reserve and the identification of coexisting pathophysiological disorders, such as coagulopathy, malnutrition, cardiomyopathy, renal dysfunction, respiratory dysfunction, ascites, and hyponatremia, are key issues in the preoperative evaluation of LC patients scheduled for non-hepatic abdominal surgery. Despite the lack of evidence-based guidelines for the management of these patients, clinical and surgical teams are acquiring experience with LC patients undergoing non-hepatic abdominal surgery, and the body of knowledge of the pathophysiology of LC in the literature is increasing. Therefore, some general recommendations for the care of these patients are in order.
Liver function

The occurrence of portal hypertension (PHT) in LC leads to variceal bleeding, ascites, spontaneous bacterial peritonitis (SBP), and hepatic encephalopathy. Patients with LC are at a higher risk of liver-related complications during surgery due to inappropriate response to surgical stress. Mortality is higher in non-hepatic abdominal surgery in the presence of PHT, which is also an indicator of illness progression [10,15]. The presence of high intraoperative PHT is associated with postoperative mortality due to rebleeding [16]. Preoperative evaluation of liver function by a physician experienced in managing liver disease in patients with confirmed or suspected LC is needed in order to stage the severity [17]. In clinical practice, severity should be assessed by means of both CTP and MELD scores, although MELD is more objective [18]. These scores should be assessed throughout the patient’s admission because rapid deterioration indicates that surgery may not be safe. The value of indocyanine green plasma disappearance rate has been demonstrated in patients with LC undergoing cardiac surgery [19,20] and partial hepatectomy [21], and it may be useful for assessing liver functional reserve in this scenario.

As a general rule, surgery is safe in CTP A patients without PHT and MELD < 10, but its pros and cons should be carefully considered in patients with severe LC [22,23]. Surgery should be avoided if possible in the setting of acute viral hepatitis, alcoholic hepatitis, acute liver failure, acute renal failure, severe coagulopathy, hypoxemia, and/or cardiomyopathy [14].

Regarding the etiology of LC, certain specific considerations need to be addressed before surgery. Due to the stress that surgery represents in patients with autoimmune hepatitis who are receiving or have recently received steroids, increasing the dose is a possible alternative. D-Penicillamine, the chelating agent used to treat Wilson disease, interferes with wound healing and should be reduced 2 wk before surgery and until the wound has healed completely. Alcohol consumption must be discontinued, especially in patients with a history of alcohol abuse, because of its associations with the risk of hepatotoxicity from drugs such as paracetamol, poor wound healing, bleeding, delirium, infections, and alcoholic hepatitis (the last of these being a clear contraindication for elective surgery in those patients) [24-26]. In addition, alcohol abstinence is very difficult, and the majority of patients have superimposed alcoholic hepatitis [26].

Coagulopathy

Coagulopathy is the major concern for surgeons, because both pro- and anti-coagulants present reductions in LC. Thrombocytopenia due to poor nutritional status, hypersplenism and/or bleeding from varices may exacerbate bleeding problems [27,28]. Myelosuppression due to hepatitis C, folate deficiency, and ethanol toxicity can also aggravate thrombopoietin deficiency [27]. However, primary homeostasis may not be defective in LC, and a low platelet count, if not severe, should not be automatically considered as an index of an increased risk of bleeding [20]. Thrombocytopenia of < 50000/µL for moderate risk and 100000/µL for high risk procedures must be corrected by platelet transfusion prior to surgery [30]. Infections, renal failure, malabsorption, and/or malnutrition can adversely affect coagulation in decompensated LC.

Conventional coagulation tests measure only part of the process of thrombin generation. The prothrombin time-derived international normalized ratio (PT-INR) is universally used to assess bleeding risk and prognosis in MELD score and to guide treatment of coagulation disturbances in clinical practice; however, it only measures the activity of procoagulants. Thromboelastography (TEG) provides a better assessment of the degree of coagulopathy and offers information allowing immediate transfusion therapy. It is useful before and during surgery for guiding transfusion therapy [31]. Serum fibrinogen, a key factor in fibrin clot formation, and protein C deficiency may also determine the risk of bleeding in LC [32].

Coagulopathy is one of the factors of LC that can be modified preoperatively with intravenous vitamin K replenishment and cryoprecipitate transfusions in order to maintain a serum fibrinogen level < 100 mg/dL. Preoperative administration of fresh-frozen plasma to correct the INR should be avoided due to its ineffectiveness and its association with volume overload, exacerbation of PHT, risk of infections, and transfusion-related acute lung injury [33,34]. Administration of desmopressin and tranexamic acid should be considered when platelet dysfunction and hyperfibrinolytic state are suspected [35]. This may help survival after surgery and convert CTP C patients into CTP B by improving the coagulopathy [36].

The lack of improvement in PT-INR with the administration of vitamin K may reflect a poor hepatic reserve and outcome. In addition, plasma hemoglobin levels lower than 10 g/dL and higher intraoperative transfusion needs are independent prognostic factors for mortality in these patients [1,18]. These findings are linked with a major deterioration of coagulation and the association of perioperative blood product transfusion with the triggering of the inflammatory response [37].

Immune dysfunction and nutritional status

Infections are an important cause of death during the postoperative period due to an innate and adaptive immune dysfunction in LC, which is associated with the development of liver dysfunction and an enhanced susceptibility to acute inflammatory processes [38,39]. This is also linked with nutritional status: between 50% and 90% of LC patients lack sufficient nutritional reserve and have a poor metabolic state, with inadequate inflammatory and immune responses to surgery [40,41].
Malnutrition in patients undergoing abdominal surgery is associated with a greater risk of postoperative complications and longer hospitalization. Although the evidence is controversial, preoperative nutrition support may play an important role in preventing complications. Nutritional disorders are more severe with alcoholic cirrhosis than with non-alcoholic cirrhosis, which can explain the poorer prognosis of this subgroup of patients when undergoing surgery. A combination of transferrin, prealbumin, and albumin seems an adequate approach for nutritional evaluation in LC. Indeed, hypoalbuminemia has been associated with poor outcome in abdominal and colon cancer interventions in patients with advanced LC.

**Cardiovascular and renal dysfunction**

Preoperative evaluation of cardiovascular dysfunction in LC is crucial. Cardiovascular diseases are a common cause of mortality in LC because of the severity of liver injury, and inflammation is strongly associated with an increased cardiovascular risk and an atherogenic lipid profile. This is especially relevant in NASH/NAFLD chronic liver disease because the etiological risk factors are the same for both LC and cardiovascular disease, even taking into account the expected rise in the incidence of NASH/NAFLD worldwide. A hyperdynamic circulatory state with increased cardiac output in response to splanchnic arterial vasodilatation is an inherent characteristic of LC, leading to heart failure with the progression of liver disease. Cirrhotic cardiomyopathy develops a variety of progressive clinical manifestations and is characterized by diastolic dysfunction and impaired inotropic and chronotropic competence, leading to a suboptimal ventricular contractile response during stressful conditions.

The dynamic assessment of preoperative cardiac function with dobutamine stress echocardiography may play a role in the indication of abdominal surgery and postoperative management in the setting of LC. Cirrhotic cardiomyopathy may also be involved in the pathogenesis of hepatorenal syndrome (HRS) or the development of acute kidney injury (AKI). AKI can lead to a positive fluid balance, resulting in vital organ edema related to cardiac output performance. LC may cause renal dysfunction and HRS; which occurs in conjunction with microcirculatory dysfunction in other organs, including the heart and the peripheral vascular bed. Assessment of preoperative renal function is of paramount importance due to the high influence of AKI on postoperative survival in LC patients. Thus, the identification of high creatinine levels as an independent predictor of complications and mortality in LC patients undergoing surgery was not surprising.

**Ascites, respiratory dysfunction, and hyponatremia**

Ascites and fluid overload may cause or aggravate pulmonary function due to atelectasis and pulmonary edema. In advanced LC, hepatopulmonary syndrome, portopulmonary hypertension, and hepatic hydrothorax are typical pulmonary complications. The end-expiratory lung volume may fall, leading to impairment in the mechanics of the respiratory system, lung, and chest wall as well as gas-exchange. Thus, initial use of moderate Positive End Expiratory Pressure is advisable to improve oxygenation and compliance without causing adverse effects in the respiratory function.

Hemodynamic changes, such as systemic vasodilation and volume retention in the splanchic bed secondary to PHT, reduce preload and the associated retention of water and sodium in order to compensate for the low effective circulatory volume. These mechanisms explain the development of ascites and the impairment of the kidneys in the elimination of solute-free water. Ultimately, this also explains the development of hyponatremia, which is associated with increased morbidity and mortality in patients with LC. Sodium < 130 mmol/L in non-hepatic abdominal surgery and ascites in general surgery are markers of poor prognosis in LC patients.

Preoperative portal decompression by transjugular intrahepatic portosystemic shunt (TIPS) seems to be a safe procedure for reducing the risk of bleeding in the presence of varices and the risk of ascites even in decompensated LC. Although there is not enough evidence to support its routine use before non- hepatic surgery, it could be considered in patients with significant ascites, extensive abdominal varices, or both. Reducing portal pressure seems to be helpful in decreasing bleeding during surgery.

If possible, these complications need to be screened in candidates for surgery, especially in those with a medical history consistent with LC-related complications, and then medically optimized prior to surgery. Despite their implications for the perioperative course of LC patients undergoing any type of surgery, they have not been adequately addressed in non-hepatic abdominal surgery, especially when compared with other major surgeries, such as cardiac surgery. In Table 1 we summarize recommendations for preoperative evaluation in patients with LC scheduled for non-hepatic abdominal surgery.

**INTRAOPERATIVE MANAGEMENT**

Patient management during surgery is crucial for outcomes in LC. A team-based approach involving hepatologists, surgeons, and anesthetists with experience in treating LC patients is required, ideally at a specialist center.

**Anesthesiology**

Intraoperative management depends more on the anesthesiologist than on the surgeon, because the use of anesthesia tends to cause hepatic decompensation in patients with LC. Fluid management can be difficult since crystalloids (e.g., Hartmann's solution or
**Table 1** Recommendations for evaluation before non-hepatic abdominal surgery in liver cirrhotic patients

<table>
<thead>
<tr>
<th>Elements to evaluate</th>
<th>Recommended tests</th>
<th>Recommended action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Homeostasis</td>
<td>Thrombo-cytopenia: Platelet number and function by means of thromboelastography</td>
<td>Preoperative transfusion if: Platelet &gt; 50000/µL → moderate risk procedures. Platelet &gt; 100000/µL → high risk procedures. Consider desmopressin (300 µg intranasal) if uremia or altered platelet function in thromboelastography</td>
</tr>
<tr>
<td></td>
<td>Coagulopathy: PT-INR; thromboelastography, Serum fibrinogen; Thromboelastography</td>
<td>IV replenishment of vitamin K (&gt;10 mg OD during 3 d). Cryoprecipitate if serum fibrinogen ≥ 100 mg/dL. Consider Tranexamic acid (10 mg/kg TD during 2-7 d)</td>
</tr>
<tr>
<td>Liver function</td>
<td>PTH</td>
<td>Consider the less invasive surgical treatment or avoid surgery if severe PTH. Consider TIPS. Antibiotic prophylaxis or treatment. Sodium restriction and diuretics (careful monitoring of renal function avoiding hyponatremia).</td>
</tr>
<tr>
<td>Ascites</td>
<td>Diagnostic ascitic tap; check diuretics response</td>
<td>Consider prophylactic treatment (i.e., β-blockers, variceal banding) based on risk of bleeding. Vitamin B1 in alcohols. Optimize protein and caloric intake (higher requirements than normal individuals). Administer antibiotic prophylaxis if suspected concurrent infections (Other than SBP).</td>
</tr>
<tr>
<td>Immune function</td>
<td>Malnutrition, hypoalbuminemia: White blood cells count; Nutritional biomarkers: Albumin, Pre-albumin, transferrin; muscle wasting</td>
<td>Insulin infusion</td>
</tr>
<tr>
<td>Nutritional status</td>
<td>Glucose intolerance: Laboratory testing</td>
<td>Consider close invasive monitoring and hemodynamic strategy in order to preserve normal cardiac function and avoid organ hypoperfusion (especially liver and kidney).</td>
</tr>
<tr>
<td>Cardiac function</td>
<td>Cardiomyopathy: Dobutamine stress; echocardiography</td>
<td>Consider close invasive monitoring and hemodynamic strategy in order to preserve normal cardiac function and avoid organ hypoperfusion (especially liver and kidney).</td>
</tr>
<tr>
<td>Renal function</td>
<td>Renal dysfunction; Hepatorenal syndrome: Serum creatinine; Glomerular filtration rate; Evaluate normal Blood Pressure and cardiac performance</td>
<td>Consider β-blockers in perioperative period. Avoid dehydration if possible before surgery. Avoid positive fluid balance during perioperative course (if hemodynamics allow that).</td>
</tr>
<tr>
<td>Pulmonary function</td>
<td>Hydrothorax; HPS; PPH: Chest-X ray; Electrocardiogram and echocardiography; Spirometry</td>
<td>Optimize pulmonary function: Discard high arterial pulmonary pressure. Discard pleural effusion/thoracentesis if necessary. If HPS/PPH evaluate appropriate therapy (i.e., IV epoprostenol, sildenafil).</td>
</tr>
<tr>
<td>CNS</td>
<td>HE</td>
<td>Use of lactulose despite absence of HE if medical past history or PTH. Treat or avoid potential triggers of HE (i.e., diuretics, infections, constipations, CNS depressants, azotemia, uremia, hyponatremia).</td>
</tr>
</tbody>
</table>

PT-INR: Prothrombin time-derived international normalized ratio; IV: Intravenous; PTH: Portal Hypertension; US: Ultrasound; TIPS: Transjugular intrahepatic portosystemic shunt; SBP: Spontaneous bacterial peritonitis; HPS: Hepatopulmonary syndrome; PPH: Portopulmonary hypertension; CNS: Central nervous system; HE: Hepatic encephalopathy.

Saline (0.9%) may worsen ascites and peripheral edema and have little effect over intravascular volumes\(^ {66} \). Indeed, intravascular volume may be depleted, even in the setting of extravascular volume overload\(^ {14} \). Thus, it seems more appropriate to provide fluid support in the form of a volume expander if necessary, prioritizing fluid restriction. Blood products should be used routinely, and TEG can achieve optimal monitoring of coagulopathy. The increased physiological demand that surgery represents in patients with identified cirrhotic cardiomyopathy can lead to heart failure. Minimization of large fluctuations in preload and afterload are recommended in order to improve outcomes. The avoidance of heart failure during the perioperative period has important prognostic implications\(^ {66} \). It is important to note that most of these patients are currently receiving treatment with β-blockers\(^ {67} \), which may ultimately reduce the cardiac response under stressful conditions. Thus, a closer monitoring of cardiac performance and the prompt use of inotropes and vasoconstrictors are mandatory in order to avoid low cardiac output and intraoperative hypotension.

Perioperative antibiotic prophylaxis should be given in the presence of ascites to prevent postoperative SBP or bacteremia secondary to SBP, which may occur during the procedure. The most frequent microbiological causes, such as gram-negative bacteria, must be covered\(^ {68} \). Although quinolones are the most frequently used antibiotic for SBP prophylaxis, an individualized approach is needed due to the emergence of multidrug-resistant agents\(^ {69} \).

Pain and sedative management regimens need to be minimized and adapted according to the degree of liver disease and the alterations to the metabolism.
caused by these anesthetic drugs. Portal blood flow is reduced as a result of PHT, and anesthetic agents may reduce hepatic blood flow by 30%-50% as a result of impaired autoregulation. In addition, myocardial depression and vasodilatation are frequent in most anesthetic agents\cite{70}. Agents with minimal hepatic metabolism (< 0.2%), such as isoflurane, desflurane, and sevoflurane together with nitrous oxide, are an appropriate choice for the operating room, along with propofol as a narcotic agent\cite{71,72}. These agents also cause fewer disturbances in hepatic arterial blood flow than others\cite{73}. Atracurium and cisatracurium are preferred as muscle relaxants since they are metabolized independently from the liver; vecuronium and rocuronium, on the other hand, are metabolized exclusively by the liver and must be avoided\cite{74,75}.

Some authors argue that epidural anesthesia should be avoided in LC patients due to the complications derived from the coagulopathy\cite{88}. However, epidural analgesia has many benefits, not only in terms of pain control during and after surgery, but also in terms of reducing pulmonary, cardiovascular, thromboembolic, and gastrointestinal complications and enhancing the recovery of gut function after abdominal surgery\cite{76}. In our opinion, epidural anesthesia may be safe in the absence of abnormal coagulation and/or platelet count, especially if evaluated preoperatively by means of TEG.

**Surgical considerations**

Surgery should ideally be elective, due to the higher than average risk associated with emergency surgery in cirrhotic patients\cite{1,7-11}. Careful tissue handling and meticulous surgical technique are vital to prevent major bleeding during surgery. Morbidity and mortality rates vary greatly depending on the severity of the cirrhosis, the presence of PHT, complications of LC, and the nature of the surgical procedure.

The incidence of gall stones and hernia is higher in LC than in the non-cirrhotic population due to the increased intra-abdominal pressure. Cholecystectomy and hernia repair surgery are the most frequently described surgeries in LC, and modified laparoscopic techniques have been proposed in these cases in order to minimize morbidity (Table 2)\cite{77-82}. The use of laparoscopy allows non-exposure of viscera and restricts electrolytic and protein losses\cite{79}. From the technical point of view, the use of coagulation instruments like the harmonic scalpel causes less postoperative pain and lower morbidity\cite{83,84}. The use of laparoscopic general surgery is also supported by the lower morbidity rates, lower bleeding complications, shorter operating time and hospital stay (despite the higher conversion rates to open surgery during the procedure, especially in the case of emergency surgery), and fewer bleeding-related complications compared to non-cirrhotic patients\cite{77,85,86}. The laparoscopic approach also helps to reduce morbidities such as surgical site infection and hemorrhage\cite{87}. A wide range of interventions can be performed laparoscopically in LC patients: splenectomies, colectomies, Nissen fundoplication, Heller’s myotomy, gastric bypass, radical nephrectomy\cite{88}, appendectomies\cite{89}, suture closure, and placement of an omental patch for treatment of a perforated gastric ulcer\cite{90}.

Although, in general, outcomes are poorer in LC patients than in the non-LC population in terms of morbidity and mortality, mortality rates are quite similar in certain surgeries thanks to the technical advances and experience gained in recent years (Table 3)\cite{10,77-79,81-87,89,91-101}. One example is abdominal wall surgery. Elective repair of umbilical hernia in patients with LC and ascites is indicated due to the poor outcomes with conservative management\cite{102-104}, and, even in advanced LC, inguinal hernia repair obtains similar outcomes to those recorded in the non-LC.

<p>| Table 2 Modifications in operative laparoscopic techniques in non-hepatic abdominal surgery in liver cirrhosis who underwent cholecystectomy and hernia repair |
|---------------------------------|---------------------------------|---------------------------------|</p>
<table>
<thead>
<tr>
<th>Ref.</th>
<th>Modified technique</th>
<th>Objective and advantage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Laparoscopic cholecystectomy</td>
<td>Use of open technique using Hassan’s trocar</td>
<td>Prevent inadvertent puncture of umbilical varix</td>
</tr>
<tr>
<td>Fried et al\cite{75}, 1999</td>
<td>Placement of the trocar in the right paramedian position</td>
<td>Facilitate laparoscopic technique and prevent complications in cases of severe gallbladder inflammation</td>
</tr>
<tr>
<td>Shiff et al\cite{79}, 2005</td>
<td>Use of additional ports</td>
<td></td>
</tr>
<tr>
<td>Clark et al\cite{79}, 2001</td>
<td>Performance of retrograde cholecystectomy</td>
<td></td>
</tr>
<tr>
<td>Clark et al\cite{79}, 2001</td>
<td>Modified subtotal cholecystectomy</td>
<td></td>
</tr>
<tr>
<td>Palanivelu et al\cite{79}, 2006</td>
<td>Mechanical compression from introduced surgical sponges (i.e., oxidized cellulose)</td>
<td>Facilitate haemostasis</td>
</tr>
<tr>
<td>Friet et al\cite{79}, 1999</td>
<td>Application of ultrasonic energy via harmonic scalpel</td>
<td></td>
</tr>
<tr>
<td>Use of argon beam coagulator through an operative port</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Laparoscopic hernia repair</td>
<td>Minimally invasive and tension-free laparoscopic technique</td>
<td>Prevent inadvertent puncture of collateral veins</td>
</tr>
<tr>
<td>Belli et al\cite{79}, 2006</td>
<td>Dual mesh prosthesis: fixation of mesh in a preperitoneal space</td>
<td>Prevent recurrence rates and mesh migration</td>
</tr>
<tr>
<td>McAlister et al\cite{79}, 2003</td>
<td>Sterile fashion of mesh insertion</td>
<td>Prevent recurrence rates and mesh migration</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Prevent wound infections</td>
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</tbody>
</table>
population and improves quality of life.\[81,82,100-110\]

**POSTOPERATIVE MANAGEMENT**

Postoperative management should ideally be performed in the ICU, at least during the first 24 h, especially in CTP B and C. It is important to monitor for the complications that may be expected depending on the degree of LC and type of surgery. As discussed above, postoperative management should be based on the pathophysiological characteristics of LC. Physicians should focus on fluid management, vigilance, and prophylaxis of infections (especially surgical site infections); opioids and sedatives should be used cautiously. The inadequate administration of normal saline can lead to the development of ascites. Salt restriction with both intravenous fluid and oral intake is mandatory in order to prevent the postoperative occurrence of AKI, ascites, and/or hepatic encephalopathy.\[111,112\]. Early introduction of lactulose should prevent encephalopathy. In the presence of fast decomposition of liver function, physicians should first rule out infection.\[113\]. The dosage of analgesics for pain control should be based on the degree of liver dysfunction because the drug metabolism of simple analgesics, such as paracetamol, non-steroidal, and opioid analgesics, may be impaired. If opioids are necessary, fentanyl is preferred because it does not produce active metabolites; however, it may accumulate in the fat tissue for several days and is cleared through the liver.\[114\]

**CONCLUSION**

Patients with LC undergoing non-hepatic abdominal surgery are at an increased risk of poor outcome. Emergency surgery should be avoided if possible, and conservative management is preferred in the presence of decompensated LC, alcoholic hepatitis, severe/advanced LC, and significant extrahepatic organ dysfunction. A multidisciplinary team approach involving surgeons, anesthesiologists, intensivists, and gastroenterologist/hepatologists together with specialized hospital staff with experience in the perioperative management of those patients can improve outcomes. Since there are no formal guidelines and few randomized controlled trials have been performed, the pathophysiological characteristics of LC mean that an individualized approach to the care of the patients is essential. Preoperative optimization after risk stratification is mandatory before surgery. Laparoscopic techniques may improve outcomes in those patients, and recent advances in cholecystectomy, abdominal wall surgery, and appendectomy have reduced mortality to levels similar to those found in the non-LC population. Future prospective randomized studies are needed to assess the effect of preoperative TIPS, to compare elective surgery vs conservative management, and to compare preoperative assessment with MELD and CTP scores for specific surgical procedures. These studies should also assess the efficacy of new approaches, especially before surgery, in order to establish formal guidelines for the management of patients with LC undergoing non-hepatic abdominal surgery.

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