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Surgery in patients with advanced liver cirrhosis: a Pandora's box

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ABSTRACT

Advanced liver cirrhosis is a relative contraindication for abdominal surgery, as such patients are likely to develop post-operative complications and mortalities. We describe two patients with liver cirrhosis who developed postoperative decompensation and expired after undergoing non-abdominal surgery. We highlight that even non-abdominal surgery could incur high mortality in patients with Child's class B or C liver cirrhosis. Surgery should be avoided in such patients unless it is absolutely necessary. If the procedure is essential and life-saving, the patient should be co-managed by a team of surgeon, anaesthestist and hepatologist. A full evaluation of the baseline liver status, preoperative optimisation, and close postoperative monitoring are required to reduce the risk of decompensation and improve survival.

Keywords: chronic hepatitis B, hepatic encephalopathy, liver failure, postoperative complications

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INTRODUCTION

Patients with liver cirrhosis are known to be at high risk of morbidity and mortality following abdominal surgery. In general, surgery is well-tolerated in patients with Child's class A cirrhosis, permissible with preoperative preparation in patients with Child's class B cirrhosis (except those undergoing extensive hepatic resection or cardiac surgery), but at high risk in patients with Child's class C cirrhosis[1]. However, decompensation can occur even after non-abdominal surgery, and the balance between benefit of surgery and risk of postoperative decompensation may be difficult to assess in some cases. We describe two patients with liver cirrhosis who developed postoperative decompensation and expired after undergoing non-abdominal surgery.

CASE REPORTS

Case One

A 57-year-old male had a two-year history of decompensated liver cirrhosis from chronic hepatitis B, complicated by splenomegaly, oesophageal varices and ascites. He sustained a Garden's grade three right neck of femur fracture after a fall. Preoperatively, his bilirubin, serum albumin, and prothrombin time (PT) were at 71 (normal <30) uM, 22 (normal >35)g/L, and 17.4 (normal <13) seconds, respectively. He was managed with oral spironolactone for his ascites and he had no history of hepatic encephalopathy. His preoperative Child-Pugh score was 12 (hence, Child's class C). He was initially managed with insertion of cancellous screw fixation of the neck of femur fracture under general anaesthesia, and he made an uneventful recovery postoperatively.

However, after the first surgery, the patient complained of persistent right hip pain. Hip radiograph and bone scintiscan suggested non-union. The patient subsequently underwent an elective left Moore's hemiarthroplasty six months later. Unfortunately, he developed decompensation on the first postoperative day, with new onset of hepatic encephalopathy, worsening of ascites, and deterioration of laboratory markers with gradual elevation of bilirubin and PT to 100 uM and 22 seconds, respectively. Subsequently, he developed acute renal failure, nocosominal pneumonia and worsening liver function. Despite maximum supportive therapy in the intensive care unit, including haemofiltration, ventilatory support, liver dialysis with extra-corporeal albumin dialysis by the Molecular Adsorbent Recirculating System (Teraklin AG, Rostock, Germany), his condition deteriorated and he died on the 22nd postoperative day.

Case Two

A 54-year-old woman had a past history of diabetes mellitus and cryptogenic cirrhosis. She was admitted for left shin swelling and tenderness of one week duration. Physical examination revealed left shin

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Table I. Child-Pugh classification.

	Score		
Components	1	2	3
Bilirubin, uM	<34	34-51	>51
Albumin, g/L	>35	28-35	<28
PT prolongation (sec)	<4	4-6	>6
INR	<1.7	1.7-2.3	>2.3
Ascites	None	Mild to moderate	Severe
Encephalopathy	None	Grades I or II	Grades III or IV

Child's class A, 5-6 points; class B, 7-9 points; class C, 10-15 points.

cellulitis with abscess and pyomyonecrosis. She did not have prior history of hepatic encephalopathy or ascites. Her baseline bilirubin, albumin, and PT were 64 uM, 19 g/L, and 14.6 seconds, respectively, and her Child-Pugh score was 9 (hence, Child's class B).

Surgical debridement with drainage of abscess was performed under general anaesthesia, but the patient's liver function deteriorated on the first post-operative day, with new onset of grade two hepatic encephalopathy, and the PT rising to 19.7 seconds. Pus culture from shin abscess grew *Staphylococcus aureus*. With hepatology support, including treatment of infection with the appropriate antibiotics, and administration of vitamin K, laxatives, and albumin, her mental status improved and ascites were controlled. However, her shin infection did not improve and she underwent a second surgical debridement under general anaesthesia eight days later. Pus culture from second debridement grew *Pseudomonas aeruginosa*, and her antibiotics were changed accordingly.

Her wound infection subsequently improved and she underwent skin grafting, her third surgery, three weeks after her second wound debridement. Unfortunately, she developed grade two hepatic encephalopathy five days after the wound grafting. And despite maximum liver support including vitamin K, lactulose, antibiotics, and albumin infusion, her encephalopathy worsened and she was subsequently transferred to intensive care unit for haemodynamic support and ventilatory support. She died ten days after the skin grafting operation.

DISCUSSION

The Child-Pugh classification helps evaluate status of liver function, and comprises two clinical findings and three laboratory findings (Table I). The serum bilirubin, albumin levels, and PT represent the metabolic and synthetic functions of the liver. Ascites and hepatic encephalopathy are related to the degree of portal hypertension and portal-systemic shunting, both reflecting severity of underlying liver cirrhosis. In other words, a patient's Child-Pugh score indicates his/her residual liver function. Although the Child-Pugh classification was originally derived to predict mortality post portal-system shunt surgery, it has also been used for prognostication for postoperative complications after non-shunt surgery among cirrhotics[2].

Garrison et al[3] evaluated 100 consecutive cirrhotic patients undergoing abdominal surgery, and described that the mortality rates were 10% in Child's class A, 31% in class B, and 76% in class C, when the patients were segmented according to their Child's class. Another study by Mansour et al[4] in a group of 92 cirrhotic patients undergoing abdominal surgery showed similar results, with mortality rate being 10%, 30%, and 82% in patients classified as Child's classes A, B, and C, respectively. Studies have shown that among patients with cirrhosis, regardless of their Child-Pugh status, those presenting with abdominal surgical problems are at great risk of having postoperative complications that can lead to death. Doberneck et al[5] described that mortality rate was significantly increased by gastrointestinal – related operations to 27.6 %, compared to the overall mortality of 19.6%. Wound dehiscence, liver failure, peritonitis, sepsis, pneumonia and bleeding are among the most common postoperative complications and causes of death found in these patients.

Mechanism of decompensation post-surgery is thought to be multi-factorial. Friedman[9] pointed out that a cirrhotic liver is particularly susceptible to the haemodynamic changes that accompany anaesthesia and surgery. Anaesthesia causes a moderate reduction in hepatic arterial blood flow and hepatic oxygen uptake. The greater the degree of haemorrhage or haemodynamic instability with surgery, the greater the fall in hepatic blood flow and chance of ischaemic injury. Other perioperative factors like sepsis, hypotension, and blood loss precipitated by coagulopathy may also aggravate the risk of postoperative decompensation.

In patient 1, the perioperative blood loss during the hemiarthroplasty was 1.5 L, which could have been the triggering factor for anaemia and acute renal failure, as well as subsequent acute decompensation after surgery. In retrospect, conservative treatment by traction and analgesics, rather than open hemiarthroplasty for his non-

union, might have prevented liver failure and death. Patient 2 had already decompensated once after the first surgical debridement, indicating high risk of further decompensation after further surgery. However, as her shin abscess and pyomyonecrosis did not improve with medical therapy, further surgeries were indicated.

Liver cirrhosis can be the end-result of a diverse set of aetiologies, including chronic viral hepatitis B and C, autoimmune hepatitis, and non-alcoholic steatohepatitis (NASH) syndrome. Management of cirrhosis would include suppressing underlying necroinflammatory activities as well as managing complications from portal hypertension[6]. In patients with chronic hepatitis B related decompensated cirrhosis, nucleoside or nucleotide anaologues such as lamivudine and adefovir dipivoxil have been shown to reduce hepatic necroinflammatory activity, with the potential of reversing hepatic fibrosis and improving their liver reserve[7]. Similarly, corticosteroids had been shown to reverse cirrhosis in patients with autoimmune hepatitis[8]. However, the majority of patients with decompensated cirrhosis from other causes would not benefit from definitive therapy against their underlying aetiologies, and the main theme of management would focus on managing complications from cirrhosis.

If surgery is essential and life-saving, preoperative optimisation should be instituted immediately to reduce the risk of development of postoperative complications. It is important to identify the presence of cirrhosis, jaundice, ascites, or encephalopathy, to obtain a complete biochemical assessment of liver function, and, if necessary, evaluate the cause of liver disease[9]. It should be noted that NASH syndrome is being increasingly recognised as an important cause of chronic liver disease and cirrhosis, and yet diagnosis often depends on clinical association of the metabolic syndrome, such as obesity and insulin resistance, and exclusion of alternative causes[10]. Therefore, it is important for the surgeon and anaesthetist involved to be able to recognise this syndrome prior to the surgery.

Preoperative optimisation includes correcting coagulopathy, ascites, hepatic encephalopathy, malnutrition, and administering antibiotics prophylaxis. Coagulopathy can be corrected by administration of vitamin K, infusions of fresh frozen plasma and platelets prior to surgery in order to reduce blood loss. Ascites should be managed with diuretics and paracentesis with simultaneous administration of albumin to minimise worsening of renal function, and antibiotic prophylaxis to prevent subacute bacterial peritonitis. Hepatic encephalopathy is usually treated with lactulose to enhance excretion of ammonia and ammonia-producing bacteria[11]. It has been shown that converting Child's C patient to Child's B preoperatively improved survival after surgery[12]. Unfortunately, in an emergency situation like for patient 2, there is no time or opportunity to optimise patients adequately before surgery.

Postoperative complications often include renal failure, hepatic encephalopathy, coagulopathy, and sepsis, and patients should be monitored closely for these complications after the successful surgery. If medical therapies fail, then liver dialysis with extra-corporeal albumin dialysis by the Molecular Adsorbent Recirculating System (Teraklin AG, Rostock, Germany), could be considered. Although the extra-corporeal albumin dialysis is expensive (costing about S$6,500 per session), not widely available, and could only act as a bridge to recovery, it helps removes both water-soluble and albumin-bound toxins and metabolites, and could help detoxify and support liver function while awaiting spontaneous recovery, as in patient 1[13].

A point to note is that previous tolerance to surgery does not necessarily indicate that the patient is fit for subsequent operations. Patient 1 underwent open reduction and internal fixation without significant postoperative complications, however he succumbed to liver failure after the hemiarthroplasty. Patient 2 recovered from decompensation after her first surgical debridement, and recovered well after her second debridement, yet she decompensated acutely after her skin graft surgery. We suggest that cirrhotic patients should be evaluated thoroughly with the surgeon, anaesthetist, and hepatologist, before each operation, and any derangement be corrected accordingly.

In conclusion, patients with liver cirrhosis, especially with Child's B or C cirrhosis, are at high risk of decompensation after surgery, even for non-abdominal surgery. Potential benefits from surgery have to be weighed against the potential risk of decompensation. Cirrhotic patients undergoing either elective or emergency operations should be co-managed with the surgeon, anaesthetist, and hepatologist for maximum optimisation before surgery and for close monitoring after surgery.

REFERENCES

1. Stone HH. Preoperative and postoperative care. Surg Clin North Am 1977; 57:409-19.
2. Pugh RN, Murray-Lyon IM, Dawson JL, Pietroni MC, Williams R. Transection of the oesophagus for bleeding oesophageal varices. Br J Surg 1973; 60:646-9.

3. Garrison RN, Cryer HM, Howard DA, Polk HC Jr. Clarification of risk factors for abdominal operations in patients with hepatic cirrhosis. Ann Surg 1984; 199:648-55.
4. Mansour A, Watson W, Shayani V, Pickleman J. Abdominal operations in patients with cirrhosis: Still a major surgical challenge. Surgery 1997; 122:730-6.
5. Doberneck RC, Sterling Jr. WA, Allison DC. Morbidity and mortality after operation in nonbleeding cirrhotic patients. Am J Surg 1983; 146:306-9.
6. Lubel JS, Angus PW. Modern management of portal hypertension. Intern Med J 2005; 35:45-9.
7. Wai CT, Lok ASF. Treatment of hepatitis. Br J Gastroenterol 2002; 37:771-8.
8. Dufour JF, DeLellis R, Kaplan MM. Reversibility of hepatic fibrosis in autoimmune hepatitis. Ann Intern Med 1997; 127:981-5.
9. Friedman LS. The risk of surgery in patients with liver disease. Hepatology 1999; 29:1617-23.
10. Sass DA, Chang P, Chopra KB. Nonalcoholic fatty liver disease: a clinical review. Dig Dis Sci 2005; 50:171-80.
11. Wiklund RA. Preoperative preparation of patients with advanced liver disease. Crit Care Med 2004; 32(4 Suppl):S106-15.
12. D'Albuquerque LA, de Miranda MP, Genzini T, Copstein JL, de Oliveira e Silva A. Laparoscopic cholecystectomy in cirrhotic patients. Surg Laparosc Endosc 1995; 5:272-6.
13. Tan HK. Molecular adsorbent recirculating system (MARS). Ann Acad Med Singapore 2004; 33:329-35.