Severe vasculo-biliary injury (D4dpv) following laparoscopic cholecystectomy: case report

Ivelin Takorov¹, Ts. Lukanova¹, I. Vasilevski¹, V. Mihaylov¹, A. Hartova², E. Odisseeva², N. Vladov²

¹HPB and Transplant Surgery; ²Department of Anesthesia and Intensive Care
Military Medical Academy, Sofia, Bulgaria

ABSTRACT

Introduction: Combined vasculo-biliary injuries, including biliary duct, hepatic artery and portal vein, are very rare, but severe complications after cholecystectomy due to their high morbidity and mortality.

Case presentation: We present a D4dpv (according to Hanover-classification of biliary injuries) biliary tract injury in a 41-years-old lady who underwent an elective laparoscopic cholecystectomy with a conversion for a massive bleeding from portal vein. Total transection of the common hepatic duct, sutured portal vein, narrowed up to 25% at the near trifurcation with a thrombus to superior mesenteric vein, ligation of right hepatic artery (existing Sg4 arterial branch, replaced left hepatic artery and opened arterio-portal shunt) were observed on the second postoperative day when she was referred to our HPB centre with diagnosis of portal vein thrombosis. The portal inflow was restored by means of thrombectomy and reconstruction of portal vein, no liver resection was performed. Biliary reconstruction required suture approximation of both hepatic ducts and an end-to-side Roux-en-Y hepatico-jejunostomy. The necrotic lesions in Sg6 and Sg7 gradually converted to cystic lesions on the 30-th postoperative day with no signs of infection. A strict observation is being carried on in order prompt treatment to be issued if necessary.

Conclusion: Prompt diagnosis and individual treatment strategy at a tertiary centre in terms of time and type of surgical procedure are of outmost importance whenever a vasculo-biliary injury is presumed.

Key words: vasculobiliary injury, portal vein thrombosis, cholecystectomy

INTRODUCTION

Bile duct injuries in combination with major vascular injuries — of both portal vein and hepatic artery are extremely rare and cause serious morbidity and mortality. We present a severe D4dpv (according to Hanover classification of biliary injuries)(1) biliary injury that turned out to be a near total transection of the whole hepatoduodenal ligament.

CASE REPORT

An elective laparoscopic cholecystectomy was performed on a 41-years-old lady at a non-specialised general surgical centre. Severe uncontrolled portal vein
bleeding was the reason for conversion and haemostatic suture of the vein. A high-volume ascites’ drainage on the second postoperative day suggested a portal vein thrombosis. The patient was referred to the HPB clinic of Military Medical Academy - Sofia with a laboratory constellation of severe liver dysfunction – aspartate aminotransferase – 818 U/l, alanine aminotransferase – 1769 U/l, totalbilirubin 91μmol/L, direct bilirubin – 70 μmol/L, lactate – 2.3mmol/l, INR – 1.43. The CT scan performed revealed a severe portal and superior mesenteric vein thrombosis, ligation of right hepatic artery, the presence of a Sg4 arterial branch, a replaced left hepatic artery with an opened arterio-portal shunts (figure 1 - a, b; and figure 2 – a,b,c).

The intraoperative finding was of a near-total transection of the hepatoduodenal ligament. The common bile duct was missing – the right and the left ducts were ligated high in the hepatic hilus. The right hepatic artery
was transected and the proximal and distal stumps were ligated, there was an arterial branch to Sg4 and a replaced left hepatic artery, originating from the left gastric artery. The portal vein was sutured and narrowed up to 25 % at the near trifurcation (Sg5/8, Sg6/7 and left portal vein) – a thrombus was detected downwards to the SMV (figure 1, b; figure 3 – a, b; and figure 4). The vascular treatment included thrombectomy, portal vein repair and subsequent continuous intravenous heparin infusion. No liver resection was undertaken. A normal portal inflow was detected with Doppler-ultrasonography intra- and postoperatively (figure 5, a). Biliary reconstruction required suture approximation of both hepatic ducts and an end-to-side Roux-en-Y hepatico-jejunostomy. The necrotic lesions in Sg6 and Sg7 gradually converted to cystic lesions on the 30-th postoperative day (figure 5, b).

DISCUSSION

Vasculobiliary injury (VBI) following cholecystectomy is a rare, but severe complication that requires prompt and adequate treatment. There are still controversial points of therapeutic view that should be considered whenever such a complex case is referred to a tertiary HPB centre – the best treatment strategy and timing of surgical repair.

In the common variety (about 90%) of VBI the right hepatic artery and a bile duct are injured(2). There are multiple reports of liver ischaemia of the right hemiliver in patients with right hepatic artery injury (3, 4, 6, 7.
These patients either developed a type of slow infarction of the right liver that is associated with the abscess formation over several weeks following the injury or they developed ischaemic atrophy. Most patients who develop infarction are treated by right hepatectomy (4, 6, 7, 10, 12). There are no reports of rapid complete infarction requiring emergency right hepatectomy, as is sometimes required when the portal vein is also injured (2). Right hepatic artery VBI results in slow hepatic infarction in about 10% of patients. Repair of the artery is rarely possible and the benefit of doing so has not been clearly demonstrated (2).

The uncommon type of vasculobiliary injury involves a bile duct(s) and the proper hepatic artery, the common hepatic artery, the main portal vein, the right portal vein, or one of these veins as well as a hepatic artery, possibly including the right hepatic artery (3). A recent review (2) identified 25 such patients in the literature. Various degrees of rapid and slow hepatic ischaemia are described with subsequent liver necrosis, abscess formation, acute liver failure or secondary biliary cirrhosis, requiring different surgical approaches as far as liver is considered – from right hepatectomy (4, 9, 14, 16) to liver transplantation (9, 13, 15). Death occurred in about 50% of the patients with rapid ischaemia reported (2, 11).

Wang et al. suggest direct repair or suture of the vein during the early stage (5). If diagnosed later on acute massive thrombus could be treated conservatively with thrombolytic and anticoagulation therapy. Liver transplantation could be estimated as a salvage therapy during the late stage.

The delay in the correct diagnosis and the elapsed period between the time of a concomitant portal vein and bile duct injury and the time of referral are factors that could negatively influence postoperative outcome. A CT angiogram with portal venous imaging should be considered as part of the preoperative evaluation in these patients in order an optimal therapeutic strategy to be assessed. We managed to restore the portal flow to the liver as early as the patient was diagnosed and referred to our centre – on the second postoperative day. The vascular treatment included thrombectomy, portal vein repair and subsequent heparin infusion. This was a successful attempt of preserving the liver parenchyma taking into consideration the anatomical variation and the existing porto-arterial shunts (between the replaced left hepatic artery and left portal branch). Fortunately, the presence of this anatomical variation self-guarded the patient from an acute liver failure and probably a lethal outcome. A sufficient inflow to liver parenchyma prevented rapid hepatic ischemia and no liver resection was undertaken. A month later the CT scan revealed colliquational necrosis, transforming toparenchymal cysts in Sg 6 and Sg 7 with no signs of infection. A close observation of hepatic function and imaging is being carried on in order prompt treatment to be issued if necessary.

**CONCLUSION**

The infrequency of these severe complications cannot determine a valid, well-founded treatment protocol. An early diagnosis should be made whenever a post-cholecystectomy iatrogenic lesion is suspected.
Individual approach based on the expertise of an experienced multidisciplinary team at a tertiary HPB centre probably is the best way of conducting an optimal treatment.

REFERENCES