Management of pseudo-aneurysm in the hepatic artery after laparoscopic cholecystectomy

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ABSTRACT

INTRODUCTION Symptomatic hepatic-artery pseudoaneurysm (HAP) after bile-duct injury (BDI) is a rare complication with a varied (but clinically urgent) presentation.

METHODS A prospectively maintained database of all patients with BDI at laparoscopic cholecystectomy (LC) referred to a tertiary specialist hepatobiliary centre between 1992 and 2011 was searched systematically to identify patients with a symptomatic HAP. Care and outcome of these patients was studied.

RESULTS Eight (6 men) of 236 patients with BDI (3.4%) with a median age of 65 (range: 54–76) years presented with symptomatic HAP. Median time of presentation of the HAP from the index LC was 31 (range: 13–816) days. Bleeding was the dominant presentation in 7 patients. One patient presented late (>2 years) with abdominal pain alone. Computed tomography angiography was the most useful investigation. Angioembolisation was successful in 7 patients. One patient died, and another patient developed liver infarction. Three patients (38%) developed biliary strictures after embolisation. Seven patients are alive and well at a median follow-up of 66 months.

CONCLUSIONS Presentation of HAP is often delayed. A high index of suspicion is necessary for the diagnosis. Computed tomography angiography is the first-line investigation and selective angioembolisation can yield successful outcomes.

KEYWORDS

Cholecystectomy – Bile-duct injury – Pseudoaneurysm

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On a global scale, laparoscopic cholecystectomy (LC) is one of the most commonly undertaken general-surgical procedures. An estimated 49,000 procedures in the United Kingdom and ≈750,000 procedures in the USA are carried out each year.^{1,2} Prevalence of bile-duct injuries (BDIs) remains high (0.2–0.6%) compared with that reported for an open cholecystectomy (0.1%).^{3,4} The risk of BDIs is well recognised, but iatrogenic vascular injuries and their outcomes are appreciated less well. Clinically significant isolated vascular injuries at a LC are thought to be rare, but can occur in ≈25% of patients who suffer a BDI.⁵

Vascular injuries at a LC may be evident as overt bleeding from the hepatic arterial tree or hepatic portal vein. Delayed presentations include hepatic infarction and consequent liver abscess from inadvertent ligation of the hepatic artery (or even acute hepatic failure).⁵ Another form of delayed or late presentation includes a symptomatic pseudoaneurysm involving the right hepatic artery (RHA). This late presentation is due to unrecognised injury at the primary surgical procedure (PSP) or a consequence of local inflammatory processes (eg bile leak or infected collections causing erosion of the arterial wall).^{6–8} Hepatic-artery pseudoaneurysms

(HAPs) have a clinically unpredictable course and can present weeks to months after the index LC.

Here, we report our experience with HAPs after a BDI sustained at a LC. Being a tertiary care referral centre for such injuries in the region, our experience in managing BDIs has developed from a specialist outreach service to include dedicated long-term follow-up.

Methods

A prospectively maintained database of all patients referred with BDIs to the Queen Elizabeth Hospital (QEH; Birmingham, UK) between 1992 and 2011 was searched systematically to identify patients with a HAP and their outcomes were analysed.

A consultant-led specialist outreach service is in operation at QEH. The consultant, when notified, attends regional hospitals to manage the BDIs identified intraoperatively.⁹ Our policy is to undertake, whenever possible, immediate or early definitive repair.¹⁰ The other cohort of patients referred are those whose injuries were not recognised at the index LC and, consequently, presented late with

complications such as biliary leak, collections, peritonitis or symptomatic pseudoaneurysms. Patients were followed up at 6–12-month intervals to assess progress and identify potential long-term complications.

Results

A total of 236 cases of a BDI were recorded during the study period. Most cases were referred from other hospitals in the region. Eleven cases were from different referring hospitals and 4 BDI cases were from the authors own hospital (QEH). HAPs were diagnosed in 8 (3.4%) patients in this cohort and all were referrals from other hospitals in the region. Six (75%) patients were men and the median age was 65 (range, 54–76) years. HAP presentation was at a median of 31 (range, 13–816) days after the index LC.

BDI: chronology and initial management

Median duration of hospital stay for the index admission for entire cohort was 13 (range, 3–41) days. All patients underwent an elective LC for symptomatic gallstone disease. The suspicion, diagnosis and type (Strasberg classification) of a BDI was varied. In brief, 6 of the 8 injuries were suspected during the index LC and converted to an open procedure with the type of BDI being classified as “major”. The type, initial management and final outcome of BDIs is summarised in Table 1.

HAP: chronology, presentation and management

The time to diagnosis of a HAP from the initial LC was 13–812 (median, 31) days. In 2 patients, the HAP was diagnosed during the same hospital admission for LC, these patients underwent a primary repair for the BDI with a hepaticojejunostomy. This was further complicated by bile leak and resulted in a prolonged hospital stay. The remaining 6 patients were discharged home after BDI management but developed a range of symptoms that necessitated re-admission to hospital. In 5 of these patients, the symptoms leading to hospital re-admission were attributable directly to the HAP, whereas the other patient was re-admitted with biliary peritonitis and developed a HAP during the second admission to hospital.

Overt bleeding was the dominant clinical feature in 7 patients, 4 of whom had hemodynamic compromise that necessitated blood transfusion. The only patient without bleeding presented with abdominal pain as the dominant symptom >2 years after surgery.

Upper gastrointestinal endoscopy confirmed haemobilia in 1 patient. Otherwise, computed tomography angiography was the first-line investigation that led to the diagnosis, along with selective hepatic arterial angiography and coil embolisation within 24 hours (Fig 1 and Fig 2). HAP diameter ranged from 3cm to 4.3cm. Six patients had successful embolisation of the HAP in a single attempt and repeat embolisation was required in 1 patient.

Table 1 Type of bile-duct injury, initial management and outcome of those who developed a pseudoaneurysm in the hepatic artery

Age (years) and sex	Type of injury	Initial management	Outcome of bile-duct injury
74, Male	Strasberg A	Not recognised on table. Readmitted with biliary peritonitis. ERCP failed twice. Laparotomy and repair of leak in cystic stump.	Needed further percutaneous drainage of a collection. Died of multiple-organ failure 29 days after embolisation of pseudoaneurysm.
70, Female	Strasberg A	Not recognised on table. ERCP + stenting and percutaneous drainage.	Leak settled in a few days. Well at 16-month follow-up.
76, Male	Strasberg E2	On-table repair: hepaticojejunostomy by specialist team.	No leak; no stricture.
56, Male	Strasberg E2	Recognised on table; T-tube drainage.	Developed mid-CBD stricture 20 months later needing hepaticojejunostomy.
66, Male	Strasberg E2	On table. Hepaticojejunostomy by specialist team.	Early bile leak, settled with PTC and drainage. Developed anastomotic stricture that was dilated by a percutaneous approach 56 months later.
64, Female	Strasberg E2	On-table repair: hepaticojejunostomy with reconstruction of the injury to the right hepatic artery.	Bile leak on day-12, resolved with percutaneous drainage.
59, Male	Strasberg E3	Suspected right-posterior sectoral duct injury oversewn by primary surgeon.	Developed obstructive jaundice from biliary stricture (E3). Hepaticojejunostomy done 3 months after a laparoscopic cholecystectomy. Well at 43-month follow-up.
54, Male	Strasberg E3	On-table hepaticojejunostomy by primary surgeon.	Developed anastomotic stricture 1 year after initial surgery necessitating revision

ERCP = endoscopic retrograde cholangiopancreatography; CBD = common bile duct; PTC = percutaneous transhepatic cholangiography

Figure 1 Angiogram showing a pseudoaneurysm (diameter, 4.5cm) in the right hepatic artery

Figure 2 Post-embolisation angiogram showing no flow in the pseudoaneurysm

One patient died in hospital from sepsis and multiple-organ failure 4 months after the index BDI and nearly 1 month after embolisation of the HAP. The remaining 7 patients have been followed up for a median of 66 (range, 2–99) months. The chronology, presentation and outcome of HAPs is summarised in Table 2.

Discussion

Visceral artery aneurysms are rare but, the hepatic arterial territory is the second most commonly involved site.¹¹ ‘True aneurysms’ are more common than pseudoaneurysms however, in the hepatic artery, these occur with equal frequency.¹¹ HAPs have been reported in relation to acute cholecystitis,¹² xanthogranulomatous cholecystitis,¹³ acute pancreatitis, trauma, liver transplantation and percutaneous interventions. Interestingly, the most common cause of symptomatic pseudoaneurysms involving the cystic artery or RHA is after a cholecystectomy. Since the first report by Pistorius in 1994, there have been reports of ≥70 cases of pseudoaneurysms involving the cystic artery or HAPs after a LC. However, the literature on HAPs after a BDI at a LC comprises just five reports.^{14–18}

The true prevalence of arterial injuries at a cholecystectomy is likely to be higher than that reported in the literature because reports pertain to symptomatic presentations and do not capture the population of asymptomatic patients. Halasz and workers found a prevalence of RHA injury of 7% in 71 cadavers who had undergone a presumed uncomplicated cholecystectomy.¹⁹ A BDI increases the risk of associated RHA injury. A review by Pulitano *et al.* concluded that the reported prevalence of RHA injury in association with a laparoscopic BDI was 12–47%,²⁰ these included transections, ligation or thrombosis of the RHA diagnosed by angiography or at a subsequent exploration. In some series that have reported a specific complication related to a BDI (eg anastomotic stricture) the prevalence of arterial injury was significantly higher (32–61%).^{18,21,22} Those reports have an inherent, inevitable selection bias, and Strasberg and colleagues estimate that the overall prevalence of RHA injury in patients with a BDI is ≈25%.⁵

Similarly, the true prevalence of a HAP after a LC is difficult to estimate. Sansonna and workers reported a risk of acute cholecystitis for all patients having a LC of 3 in 1,000,000, and that for patients undergoing an emergency LC of 1 in 100,000.²³ Prevalence of a pseudoaneurysm in the context of a BDI is 2.6–4.5%^{14,18} (it was 3.4% in our institution).

Development of a HAP after a LC is probably multifactorial. The primary injury is likely to be mechanical and may occur at the time of the cholecystectomy, and may include injury at dissection or diathermy injury (direct or conduction injury). In our series, 1 patient had an arterial injury diagnosed at the PSP. Secondary injury possibly occurs from bile leak and infection. Five patients out of 8 cases in our series had a bile leak before they developed a pseudoaneurysm, and we feel that this is an important risk factor for pseudoaneurysm development. Metal clips in direct contact with the arterial wall can also contribute to wall damage (as found in 1 patient). Most patients present within 6 weeks of the PSP but, in some cases, the presentation may be delayed for >1 year. Three patients in our series had a delayed presentation, one being as late as 2.5 years after the PSP. Pseudoaneurysms can a diameter of 7cm,²⁵ and may even present with pressure symptoms or pain (as noted in 1 patient in the present study).

Urgent selective hepatic arterial angiography and embolisation is first-line treatment for a symptomatic HAP because it has the advantages of speed, targeted therapy, minimal invasiveness and potential for repetition if necessary. Disadvantages are collateral damage by spillover embolisation of other arteries and the risk of hepatic ischaemia. Surgical exploration is not the initial recommended therapeutic option because it involves re-operative laparotomy in a hostile field with its attendant difficulties (adhesions and altered anatomy). If repeat angioembolisation fails or if it is not possible logistically, then other options are percutaneous image-guided thrombin injection^{24,25} and surgical exploration. Surgical options include exclusion (ligation of feeding vessels) and excision of the HAP. Endoscopic ultrasound-guided injection and obliteration of selected visceral pseudoaneurysms has been described,²⁶ and it may be possible to apply this method in HAPs if the anatomy is favourable and expertise available.

Table 2 Bile-duct injury and a pseudoaneurysm: chronology, presentation and outcome of a hepatic-artery pseudoaneurysm

Age (years) and sex	Time to discharge (days)	Pseudoaneurysm presentation	Time from cholecystectomy (days)	Management	Outcome
76, Male	12	Bleeding from drainage site; Hypotension	13	Needed two embolisations 3 days apart	Developed segmental infarction of the right lobe of the liver. Well at 10-month follow-up.
59, Male	16	Blood in the drain	48	Successful embolisation	Needed delayed repair of bile-duct injury (E3). Well at 41-month follow-up.
54, Male	13	Rectal bleeding; Hypotension	17	Successful embolisation	Anastomotic stricture needing revision. Well at 81-month follow-up.
56, Male	5	Haematemesis and melena	25	Successful embolisation	CBD stricture needed hepaticojejunostomy. Well at 99-month follow-up.
66, Male	41	Blood in percutaneous drain placed for anastomotic leak.	31	Successful embolisation	Needed percutaneous dilatation of anastomotic stricture. Well at 78-month follow-up.
74, Male	3	Hypotension; anaemia	86	Successful embolisation	Died 29 days after embolisation of multiple-organ failure.
70, Female	7	Upper abdominal pain	812	Successful embolisation	Well at 16-month follow-up.
64, Female	21	Blood in drain	13	Unable to embolise selectively. However, no flow in the pseudoaneurysm at the end of angiography	Well at 2-month follow-up.

The extent of the impact of concomitant hepatic arterial injury on the outcome of biliary injury is controversial. Several studies have reported a poor outcome with increased mortality and morbidity (bleeding, liver abscess, liver necrosis, leaks and anastomotic strictures).^{22, 27–31} However, two of the largest series involving 110 patients with a combined BDI and arterial injury did not find an adverse impact of the arterial injury on the long-term outcome of the BDI (though there was an increased incidence of morbidity in the short- and medium-term).^{18,21} In our series, 1 patient developed hepatic necrosis after embolisation of the hepatic artery, whereas 3 patients developed anastomotic strictures diagnosed 11, 19 and 55 months after angioembolisation. Due to the small number of patients who developed strictures, drawing definitive conclusions on their natural history and relationship (if any) with embolisation is difficult. However, strictures present typically at least many months later and not at immediate follow-up, which supports a policy of periodic surveillance.

Conclusions

A HAP is not an uncommon sequel in the context of a BDI. Any patient with a history of a BDI who presents with gastro-intestinal bleeding, hypotension or otherwise unexplained abdominal pain should be investigated for a possible pseudoaneurysm. The risk is especially high if the patient has had a bile leak after initial surgical repair of a BDI. Early and urgent computed tomography angiography should form part of the diagnostic workup in this subset of patients. Such

patients should be cared for in a setting that has the support of specialist hepatobiliary surgeons and offers ready access to interventional radiology, which is often lifesaving.

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