Subvesical Duct Detected by Magnetic Resonance Cholangiopancreatography (MRCP) in a Patient with Bile Leak after Laparoscopic Cholecystectomy

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ABSTRACT
We report a case of bile leaks post-laparoscopic cholecystectomy (LC) with initial treatment failure by common bile duct stent insertion. The injury of a subvesical duct running from gallbladder fossa toward an area of fluid accumulation that was not revealed by computed tomography and endoscopic retrograde cholangiopancreatography previously, was eventually found by magnetic resonance cholangiopancreatography (MRCP) and proved to be the cause of bile leak. Also, several tiny branches in the right liver instead of a main trunk and another subvesical duct draining into the common bile hepatic duct was noted. These anatomic variations were scarcely reported, especially by MRCP.

The aim of this case report is to discuss the link between biliary tree anomaly and bile leak due to bile duct injury during LC in our experience treating one patient. Also, we review related literature to understand more on prevention or management of subvesical duct injury.

Key Words: Subvesical duct injury, Laparoscopic cholecystectomy, Bile leak, Biliary tree anomaly.

INTRODUCTION
The “subvesical ducts” (once called the “ducts of Luschka”), bile ducts connected with the gallbladder fossa,1 are the second most location where bile leaks often occur.2,3 Thus, if these ducts were injured during laparoscopic cholecystectomy (LC), bile leaks may occur and may not be easily diagnosed and treated by common bile duct (CBD) stent insertion due to the leak site near gall bladder fossa may far away from the drainage by stent in CBD. Moreover, inefficient stent function even with partial obstruction may hamper healing or induce recurrence of leakage.

We report a case of bile leak that could not be treated successfully by CBD stent insertion initially, and though unusual, this was attributed to stent obstruction. Finally, subvesical duct injury with biliary tree anomaly (right hepatic duct was replaced by several tiny branches) was proved by magnetic resonance cholangiopancreatography (MRCP). All biliary tree anatomic variations in this patient were scarcely reported, especially by image of the MRCP.

METHODS
A 50-year-old male presented in the emergency department with epigastric pain with bloating sensation for one day. Physical examination showed no fever or icteric sclera. Abdominal examination revealed soft but tenderness over right upper quadrant, and positive Murphy’s sign. Laboratory data showed white cell count 10520/ul (normal
range: 3590–9640/mm³), CRP 12.04 mg/dl (0–0.3 mg/dl), aspartate aminotransferase (AST) 64 IU/L (15–37 IU/L), ALT 87 IU/L (16–63 IU/L), total bilirubin 10.4 mg/dl (00.2–10.0 mg/dl), direct bilirubin 00.5 mg/dl (0–0.2 mg/dl). Abdominal computed tomography (CT) showed thickened gallbladder wall about 14 mm and gallstones without stones in CBD (Figure 1A-B). Acute cholecystitis was impressed and therefore emergent laparoscopic cholecystectomy was performed.

Dissection before cholecystectomy was difficult in this case due to severe edematous and fibrotic change inside and around the Callot triangle. Meticulously combined sharp and blunt dissection with combined antegrade and retrograde approach could safety sever the cystic duct from CBD. We found the cystic duct entering into the gall bladder. Ligation and division was done using a vascular staple technique with 45 mm GIA stapler.

Also, dissection was very close or superficial liver injury of gall bladder fossa but not enter deeply into liver parenchyma. The operative time was relatively longer (200 minutes) than usual and external blood loss was 80–100 ml. No bile spillage was noted with the white gauze test on operated field.

Due to severe inflammatory change and severe fatty liver change, there was some diffuse oozing in the operative field. We left a closed vacuum suction drain at the end of the operation.

RESULTS

Two days after the operation, bile fluid was noted in the sub-hepatic drain, and bile leak was suspected. Conservative treatments including nothing by nothing by mouth (NPO) and intravenous fluid were applied unsuccessfully. Twelve days after LC, the patient underwent endoscopic retrograde cholangiopancreatography (ERCP), and biliary and pancreatic plastic stents were inserted (Figure 2). The following day after ERCP, the amount of fluid in the drain dramatically decreased and the patient improved and was discharged smoothly after one week. There was no bile fluid noted at follow-up one week later.

However, bile fluid about 250–300 cc/day was noted again in the following week (2 weeks post ERCP). Due to suspected bile duct obstruction, the patient was admitted again though there was no obvious fluid accumulation noted on abdominal CT. He went through repeated ERCP on admission the following day 5 owing to persistent bile leak. CBD stent partially occluded by yellowish substances (Figure 3) was noted, but no leak was seen on common hepatic duct and CBD. As a result, the old

![Figure 1. (A) Severe inflammation of gall bladder with thickened and layered gallbladder wall. (B) Several gall stones.](image-url)
dysfunctional stent was replaced with a new one. In addition, during the ERCP, we had difficulty in trying to insert the guidewire into the right hepatic duct (Figure 4A). When the guidewire was finally inserted to one of the branches of bile duct on the right side, the unusual track of the guidewire was noted. Moreover, the main right hepatic duct was not found when contrast filling the right intrahepatic biliary tree in correspondence to the left hepatic duct. Instead, several tiny branches were noted (Figure 4B). Abdominal MRCP was arranged one week later due to persistent bile leak and biliary anomaly. The result of the MRCP showed intact CBD and intact cystic duct stump. Also, a subvesical duct running from gallbladder fossa toward an area of fluid accumulation (Figure 5A, extravasation of contrast), where its distal end might have been incidentally cut during LC, proved to be the cause of bile leak was noted. In addition, several tiny branches in charge of the bile drainage of the right liver, including another subvesical duct draining into common bile duct (Figure 5B), strongly expressed the existence of such accessory duct in this patient.

With longer NPO and parenteral nutrition, the leak healed gradually. After JP drain amount gradually decreased, we removed the Jackson Prat drain without complications. Delay in remission of bile leak at this time was due to intra-abdominal surgical site infection with abscess formation and the fistula healed gradually with antibiotic treatment and longer NPO with parenteral support. There was no recurrent biliary fistula nor abnormal liver enzymes with regular follow up for 2.5 years.

**DISCUSSION**

If injury of the subvesical duct occurred and was not noticed during LC, postoperative bile leak may occur. Subvesical ducts are rarely found during initial laparoscopic cholecystectomy. In one study, 230 patients receiving LC operated by a single surgeon, subvesical bile ducts were seen intraoperatively and ligated only in 3.47% of patients (8 cases).4

Case reports of subvesical ducts are not rare, but those with proven by MRCP is uncommon. Most cases are diagnosed with ERCP when bile leak was suspected or direct visualization during LC. In our case, due to a tiny right biliary tree and a great distance between the injury point of the subvesical duct and central biliary tree, ERCP could not detect the injured subvesical duct in charge of bile leak. However, such anomaly gave us some hints that this patient might have other biliary anomaly like subvesical duct. Another unique aspect of this case is that the biliary tree is abnormal on the right side with several tiny branches. Although these tiny branches are not the etiology of post-LC bile leak, they may affect the recovery of bile leak due to poor bile drainage function of the right liver even when the CBD stent was
Figure 4. (A) When trying to insert the guidewire into right hepatic duct, the guidewire easily went toward left hepatic duct. (B) No leak was seen on endoscopic retrograde cholangiopancreatography and biliary tree on the right side liver consisted of several tiny branches draining into main bile duct instead of merging into right hepatic duct.

Figure 5. (A) Subvesical duct running from gallbladder fossa toward an area of fluid accumulation (arrow) in magnetic resonance cholangiopancreatography. (B) Several tiny branches in charge of the bile drainage of right liver, including another subvesical duct (arrow) draining into common hepatic duct.
applied. In addition, the discovery of another subvesical duct draining to CBD in MRCP (Figure 5A-B) convinced us of the possibility of intraoperative injury of similar anomalous subvesical duct.

There are many anatomic variations and no standardized definition accepted universally for subvesical duct. Also, many published articles used the term of “ducts of Luschka”, but with different descriptions. Therefore, a systemic review done by Schnelldorfer et al. concluded that the subvesical bile duct is a topographic description of a bile duct (or bile ducts) which run in contact with the gallbladder fossa. In addition, Schnelldorfer et al. classified the subvesical ducts into 4 types, including (I) superficial variations of segmental and sectorial bile ducts, (II) superficial or intercommunicating accessory bile ducts (supernumerary from the formal biliary tree), (III) hepaticocholecystic ducts (directly draining into the gallbladder), and (IV) aberrant bile ducts (network of small bile ducts within the connective). Despite having this classification, we were still unable to clearly determine which type the two subvesical ducts in our case belong to. The injured subvesical duct looks like type III and the intact one may be classified as type II. The prevalence of each subvesical duct type is unavailable, but overall prevalence is approximately 4%.

The various ways to detect subvesical duct have been reported in the literature. Pre-operative imaging of the subvesical ducts, including drip-infusion cholangiography with computed tomography or cholangiography via other methods are not routinely arranged. Intraoperative detection and direct visualization of injured subvesical ducts are not easy and common. When bile leak was suspected after LC, abdominal sonography or CT scan can help detect fluid collection and drainage. In addition, ERCP, MRCP, fistulography, and hepatobiliary iminodiacetic acid scintigraphy (suboptimal anatomic detail) can also be used to detect bile leak and subvesical duct injury. Moreover, the subvesical duct injury can be detected during reoperation if needed.

Several studies mentioned that more than 90% of bile leaks will resolve after ERCP treatment. Most cases of bile leak due to subvesical duct injury can be successfully managed by ERCP with or without sphincterotomy plus stent insertion. The management mentioned above is usually to treat the bile leak that the amount is more than 200 ml per day. For the cases with smaller amount of bile leak, with a drain placed during LC, spontaneous closure takes about 6–8 weeks. However, complications may develop after stent placement, such as stent occlusion and proximal or distal stent migration. Prevalence of these two complications were both approximately 30.1%. Like our patient, stent occlusion can be caused by inspissated bile, sludge, or debris within the stent. Once the occluded stent obstructs the bile flow, it will raise the pressure of the biliary tree and aggravate the bile leak. Stent removal and replacement can resolve stent occlusion. In addition, the biliary stent can maintain patent for about 90 days. If stent migrates proximally (upward), it can be managed by endoscopic retrieval with basket or balloon. Distal stent migration is rarely complicated with perforation, but surgery is needed if it occurs. Therefore, if there are symptoms and signs like cholangitis after CBD stent insertion, ERCP is the only way to identify the causes of the complication after stenting.

In conclusion, this case provides a unique situation of bile leak after LC due to operative injury of the subvesical duct identified in MRCP, and is associated with anomaly of biliary tree in the right side liver proved by ERCP and MRCP. We also suggest re-evaluating the stent function if there is persistent or rapid recurrence of
biliary fistula to rule out the possibility of occlusion by sandy stone or sludge.

When bile leak occurs after LC, subvesical duct injury should be taken into consideration. It is easily unnoticed even with routine intraoperative cholangiogram if the surgeon does not have knowledge of the existence of such anomaly. Therefore, proper management and, if possible, prevention by careful dissection during the operation is the fundamental to solve this kind of complication.

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