

Cholecystoduodenocolic Fistula in the Setting of Chronic Cholelithiasis

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Abstract

We present a case of a 38-year-old female who presented with several months of symptomatic cholelithiasis. Her laparoscopic cholecystectomy was delayed due to comorbidities allowing for periodic abdominal imaging. Her eventual laparoscopic cholecystectomy revealed two fistulas consistent with cholecystoduodenocolic fistula. This operation was converted to an open procedure with primary repair of both fistula tracts and successful completion of the cholecystectomy. This case highlights the difficulty of obtaining a pre-operative diagnosis of bilioenteric fistulas and the importance of converting to a laparotomy when encountered with unclear anatomy.

Keywords

laparoscopic cholecystectomy; cholecystitis; gallstone; cholecystoduodenocolic fistula

Abbreviations

RUQ: right upper quadrant; MRCP: magnetic resonance cholangiopancreatography; FNA: fine needle aspiration; G-J: gastrojejunal; HIDA: hepatobiliary iminodiacetic acid

Introduction

Bilioenteric fistulas are rare with an incidence of 0.74% in all biliary tract surgeries [1]. Cholecystoduodenal and cholecystocolic fistulas are the most commonly encountered bilioenteric fistulas with an incidence of 71.4% and 14.3%, respectively [2]. Combined bilioenteric fistulas are exceedingly rare with only a few reported cases of cholecystoduodenocolic fistulas in the medical literature [3-9]. These fistulas continue to be difficult to diagnose preoperatively despite extensive radiographic studies and pose some intraoperative technical difficulty. We report a finding of a cholecystoduodenocolic fistula in a patient with chronic cholelithiasis undergoing laparoscopic cholecystectomy.

Case Presentation

A 36 year-old African American female presented to the outpatient surgery clinic for elective cholecystectomy evaluation. She was previously evaluated eight months prior when she presented to the emergency department with nausea, vomiting and epigastric pain radiating to her back in the setting of known cholelithiasis. On admission, initial labs were WBC 10.6 k/cumm; Hgb 9.5 gm/dL, platelets 532 k/cumm, total bilirubin 0.8 mg/mL, alkaline phosphatase 82 units/L, ALT 13 units/L, AST 17 units/L and lipase 314 units/L. The patient was afebrile with diffuse abdominal tenderness and no guarding or

rebound tenderness.

RUQ ultrasound did not demonstrate definitive pericholecystic edema or wall thickening. Incidentally, a 2.2 cm x 2.6 cm hypoechoic structure adjacent to the pancreas head was discovered. The patient was admitted to the hospital and was treated symptomatically for gallstone pancreatitis. Further radiographic studies were obtained in the form of a MRCP which confirmed multiple gallstones. The gallbladder was contracted and in close proximity with the duodenum and transverse colon. Additionally, MRCP revealed a 2.7 cm x 1.8 cm x 2.5 cm enhancing mass along the superior aspect of the pancreas, periportal lymphadenopathy and peripancreatic edema around the head of the pancreas near the second portion of the duodenum (Figure 1). Two weeks later, endoscopic ultrasound with FNA of the pancreatic mass and periportal nodes revealed anaplastic large cell lymphoma, stage IIa, and gastric outlet obstruction secondary to lymphadenopathy. A G-J tube was placed for nutrition and cholecystectomy was postponed until completion of cyclophosphamide, hydroxydaunorubicin, vincristine, and prednisone therapy.

Several hospital readmissions for neutropenic candidemia, vancomycin resistant enterococcus urinary tract infection, and pulmonary embolism complicated her pre-surgical course. Her unfortunate preoperative course allowed for periodic cross-sectional imaging. The first CT with contrast revealed gallstones unchanged from her previous MRCP obtained one-month prior on her original admission (Figure 2). A second CT scan obtained a month and half later showed the same peripherally calcified stones present in the gallbladder with no biliary duct dilation (Figure 3). One month later, a HIDA scan was performed which showed no filling of the gallbladder consistent with chronic cholecystitis and on delayed imaging some gastrointestinal activity in the right mid-abdomen (Figure 4). One month after the HIDA scan, another CT without contrast showed a contracted gallbladder, calcified stones and a new focal hypodensity with vascularity surrounding the gallbladder. This was interpreted as severe focal fatty infiltration (Figure 5). Throughout these months, she remained symptomatic with intractable nausea, vomiting, persistent RUQ pain and 27 kg weight loss. After completion of chemotherapy for her lymphoma, she was deemed a surgical candidate and consented for laparoscopic cholecystectomy for symptomatic cholelithiasis.

Laparoscopic exploration of the right upper quadrant revealed the transverse colon adherent to the gallbladder. The colon was successfully mobilized from the gallbladder utilizing electrocautery and blunt dissection. A top down cholecystectomy was performed due to a severely contracted gallbladder. The gallbladder was noted to be severely adherent to the duodenum. At this point in the operation, the laparoscopic procedure was converted to open procedure because of unclear anatomy due to the inability to dissect the gallbladder off of the duodenum. A communication between the gallbladder and the duodenum was divided purposefully with electrocautery revealing a cholecystoduodenal fistula. No additional pathology was encountered when the duodenum and retroperitoneum were inspected following a Kocher maneuver. The cholecystoduodenal fistula was repaired in two layers with interrupted 3-0 vicryl sutures and reinforced with 3-0 silk Gambet sutures. Both a 4 cm and 3 cm stone were extracted, and the cholecystectomy was completed with ligation and division of the cystic artery and duct. After completion of the cholecystectomy, the colon was inspected revealing 2 mm full thickness opening within a fibrous rind on the wall of the colon consistent with a cholcystocolic fistula. A colorrhaphy was

performed with two layers of interrupted 3-0 vicryl sutures reinforced with 3-0 silk Gambet sutures. The repair was covered with an intraabdominal omental flap. One 19 french blake drain was left adjacent to each repair. Final surgical pathology of the gallbladder and fibrous rind confirmed acute and chronic inflammation with no morphological evidence of lymphoma. The patient was discharged on post-operative day nine and had no major post-operative complications; the patient was medically ready on day seven yet due to chronic pain and disposition to rehabilitation her discharge was delayed. She was seen in clinic at three and six weeks following surgery and was treated for a surgical site infection with opening of the wound and twice daily packing of the wound which resolved at six weeks.

Discussion

This case exemplified the difficulty of establishing a preoperative diagnosis of cholecystoenteric fistulas. The patient's anaplastic lymphoma diagnosis was established using multiple imaging modalities such as CT and MRI/MRCP, which were not sensitive enough to detect the presence of a fistula. In retrospective review of the patient's radiographs, the images were suspicious for a fistula given the close proximity of the duodenum and the transverse colon with the gallbladder. The T2 haste MRI image (Figure 1) showed a hypointense gallbladder indicating the presence of air and the possibility of a bilioenteric fistula. Serial CT imaging from her multiple hospital readmissions demonstrated large persistent gallstones in close proximity with the transverse colon in Figures 2, 3, and 5. Additionally, the close proximity of the stone and duodenum can be appreciated by the hyperintense G-J tube located in the duodenum in Figures 2 and 3. In Figure 2B, the black arrow indicates evidence of air in the gallbladder. In Figure 3B, the arrow shows a hypointense area connecting the gallbladder and the duodenum, which may represent a cholecystoduodenal fistula. Additionally, the adhesion between the colon and the gallbladder can be visualized by the arrow in Figure 3C. It was only until the patient's last CT scan, Figure 5, when the hypodensity surrounding the gallbladder was interpreted by the radiologist to have vessels coursing through the fatty inflammation. We suspect this may represent the cholecystocolic fistula. In Figure 5B, the adhesions between the gallbladder-duodenum can be appreciated with the black arrow and the adhesion between the gallbladder-transverse colon can be appreciated with the white arrow. By comparison of serial images, we suspect these connections between the gallbladder and colon/duodenum contained the fistula tracts.

The majority of bilioenteric fistulas present asymptotically as a result of chronic cholelithiasis. In the medical literature, 91-94% of reported fistulas are thought to be sequela of chronic gallstone disease pressure necrosis at the gallbladder infundibulum [10]. We suspect her fistulas arose from a combination of chronic cholelithiasis and 27 kg weight loss leading to a severely contracted gallbladder with persistent inflammation. Rarely, fistula formation results from peptic ulcer disease and neoplasms [11]. We do not suspect her diagnosis of anaplastic lymphoma caused fistula formation, although we considered this but the surgical pathology confirmed no malignancy. But rather her significant weight loss, malnutrition, and acute on chronic cholecystitis contributed to her contracted and necrotic gallbladder resulting in conditions favorable for fistula formation.

Of the few reported cholecystoenteric fistulas, cholecystectomy and fistula repair were completed using only laparoscopic technique [8]. In this case, the unclear anatomy influenced the decision to convert to open laparotomy. This allowed for sufficient exploration of the fibrous rind on the colon leading to the

discovery of the second fistula. Without this crucial finding, this patient had the potential to develop serious complications. This patient also had two separate fistula tracts which is distinct from most previous reports which report one continuous fistula tract between gallbladder, colon and duodenum [1, 3-9].

This patient exemplified the difficulty of diagnosing bilioenteric fistulas without classic sequela such as gallstone ileus. Additionally, this reinforced the importance having certain threshold to convert to open laparotomy when encountered with unclear anatomy. Upon conversion to a laparotomy this allowed for sufficient exploration and definitive repair of the fistula.

Figures

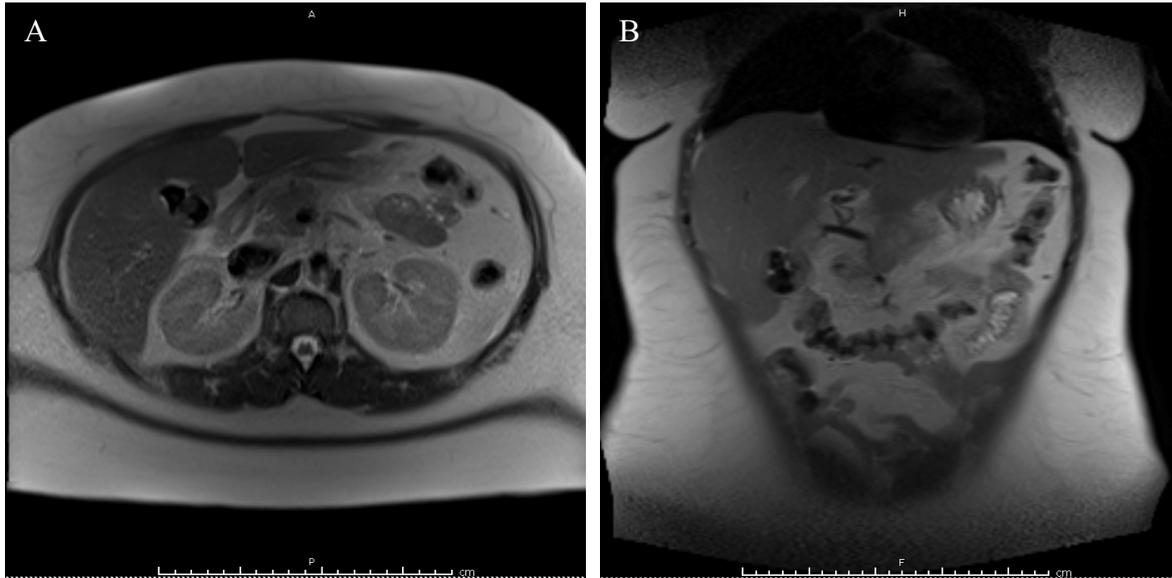


Figure 1: MRCP obtained on original hospital admission demonstrating pneumobilia.

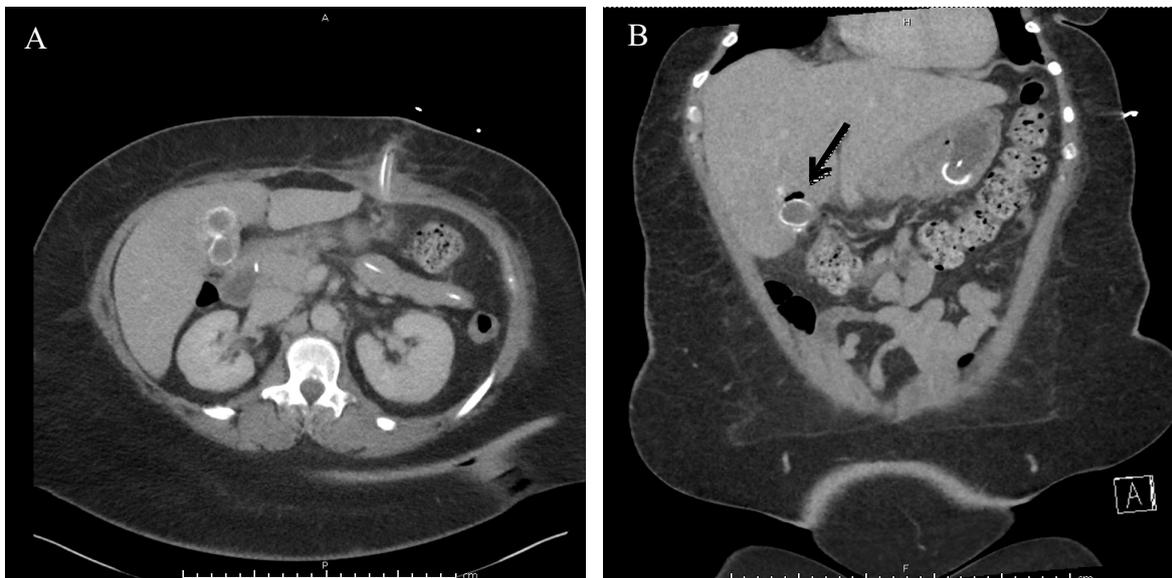


Figure 2: Abdominal CT with contrast demonstrating the presence of cholelithiasis. (A) The proximity of the duodenum and gallbladder can be appreciated by the hyperintense structure of the G-J tube. (B) The black arrow indicates the presence of pneumobilia superior to the gallstone.

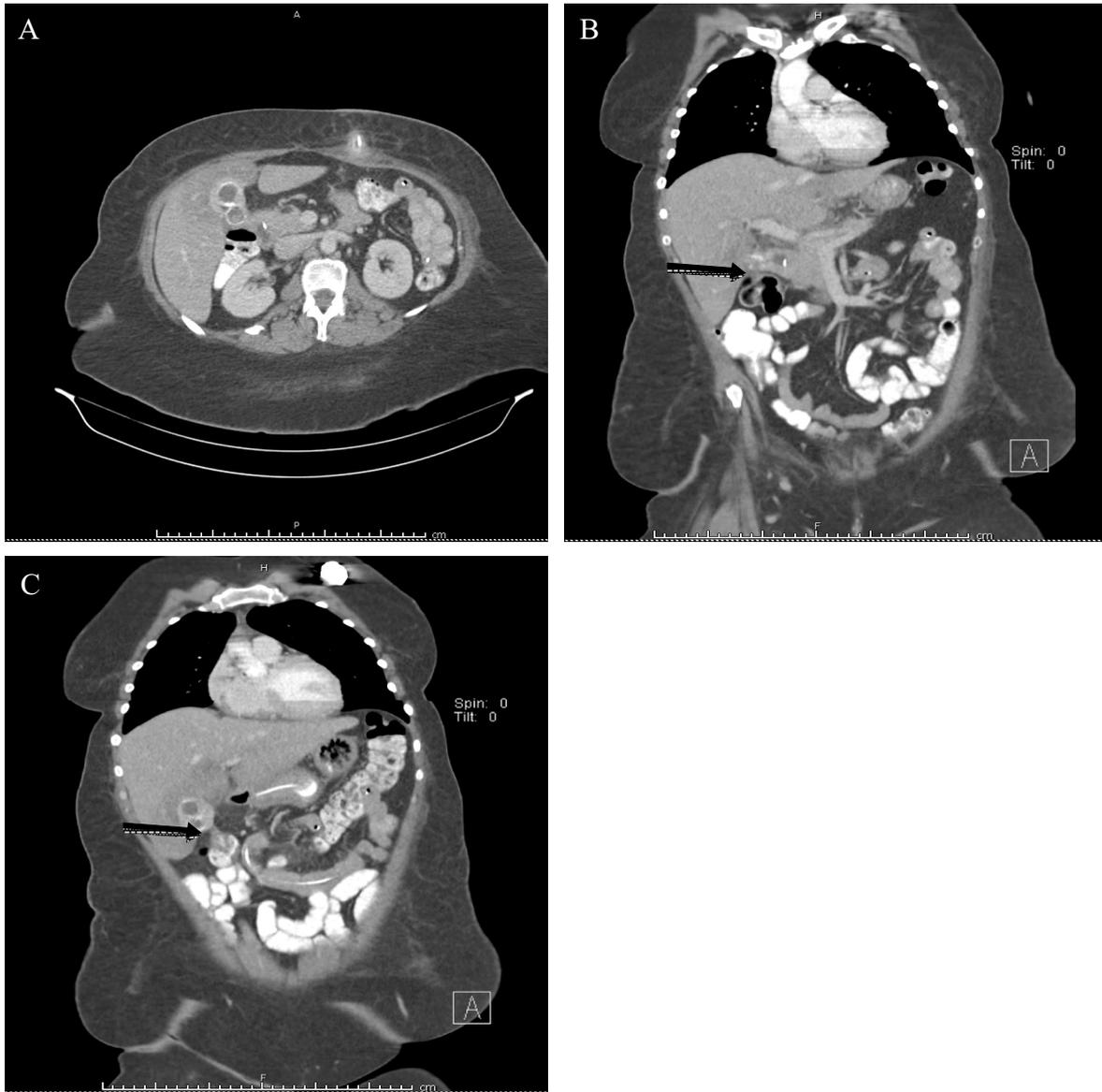


Figure 3: Series of abdominal CT with contrast images obtained approximately 2.5 months after original admission. (A). Axial image demonstrating the presence of gallstones and close proximity to the duodenum. (B). The black arrow indicates the structure suspected to be early signs of fistula formation. (C). This is demonstrated again in an alternant coronal plane.

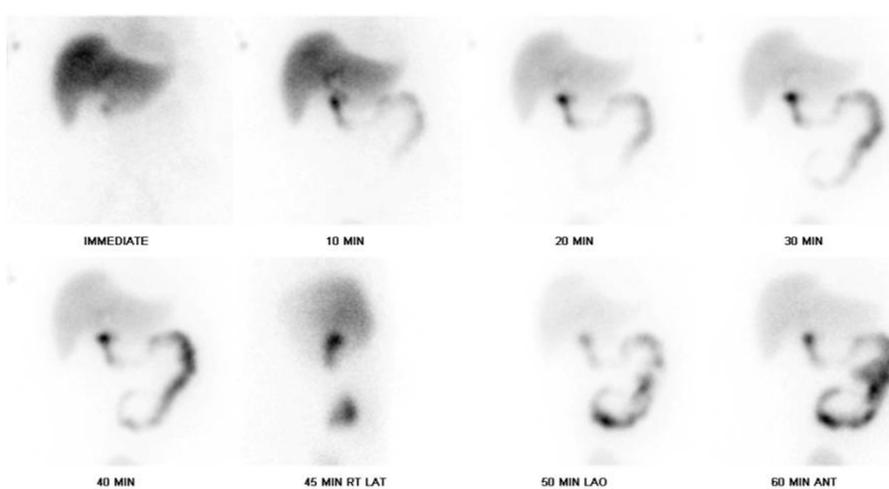


Figure 4: HIDA scan demonstrating incomplete gall bladder filling suggestive of cholelithiasis and delayed activity in the right upper abdomen.

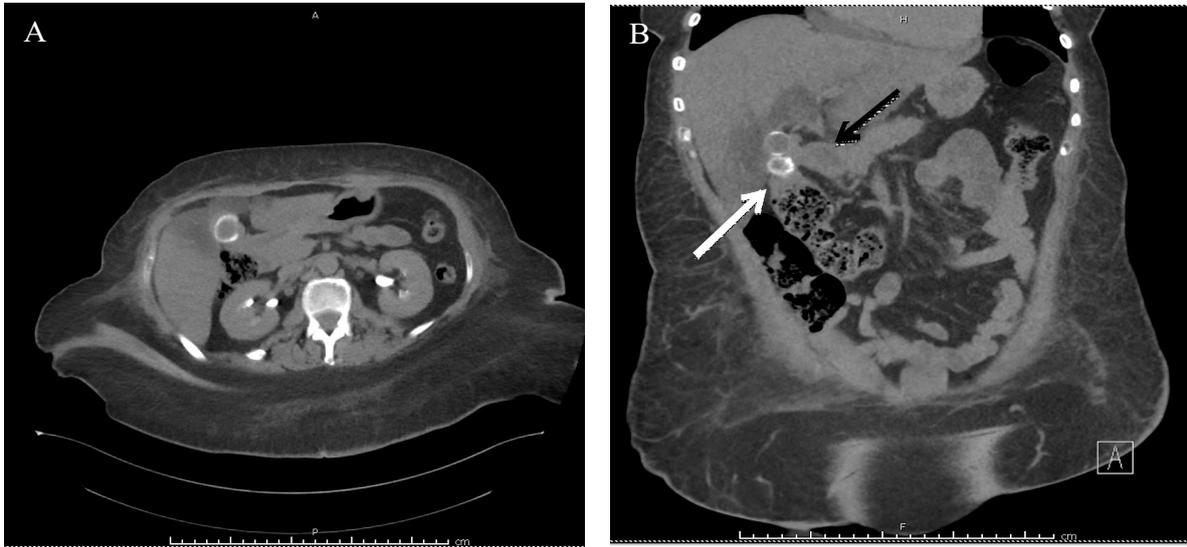
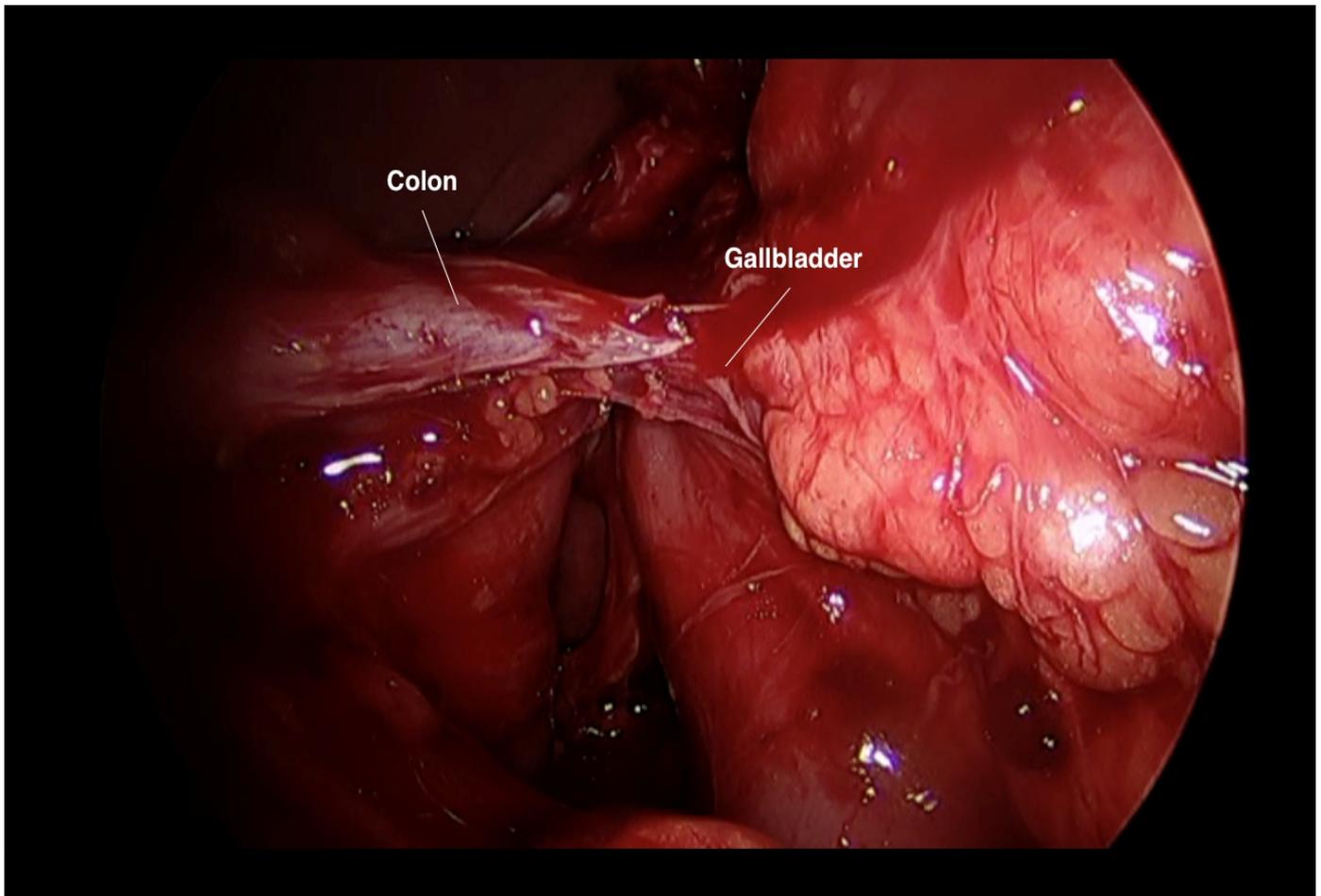
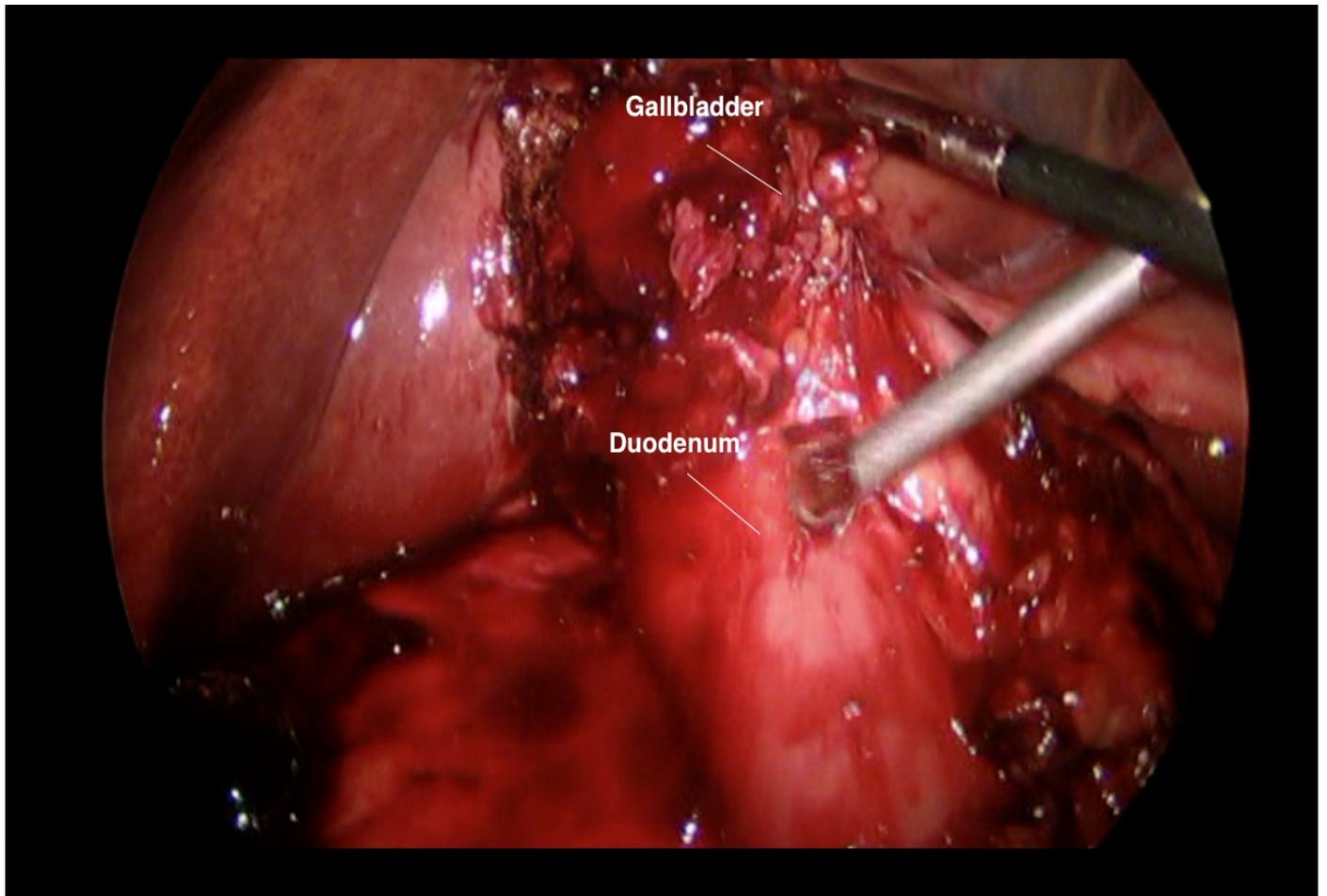
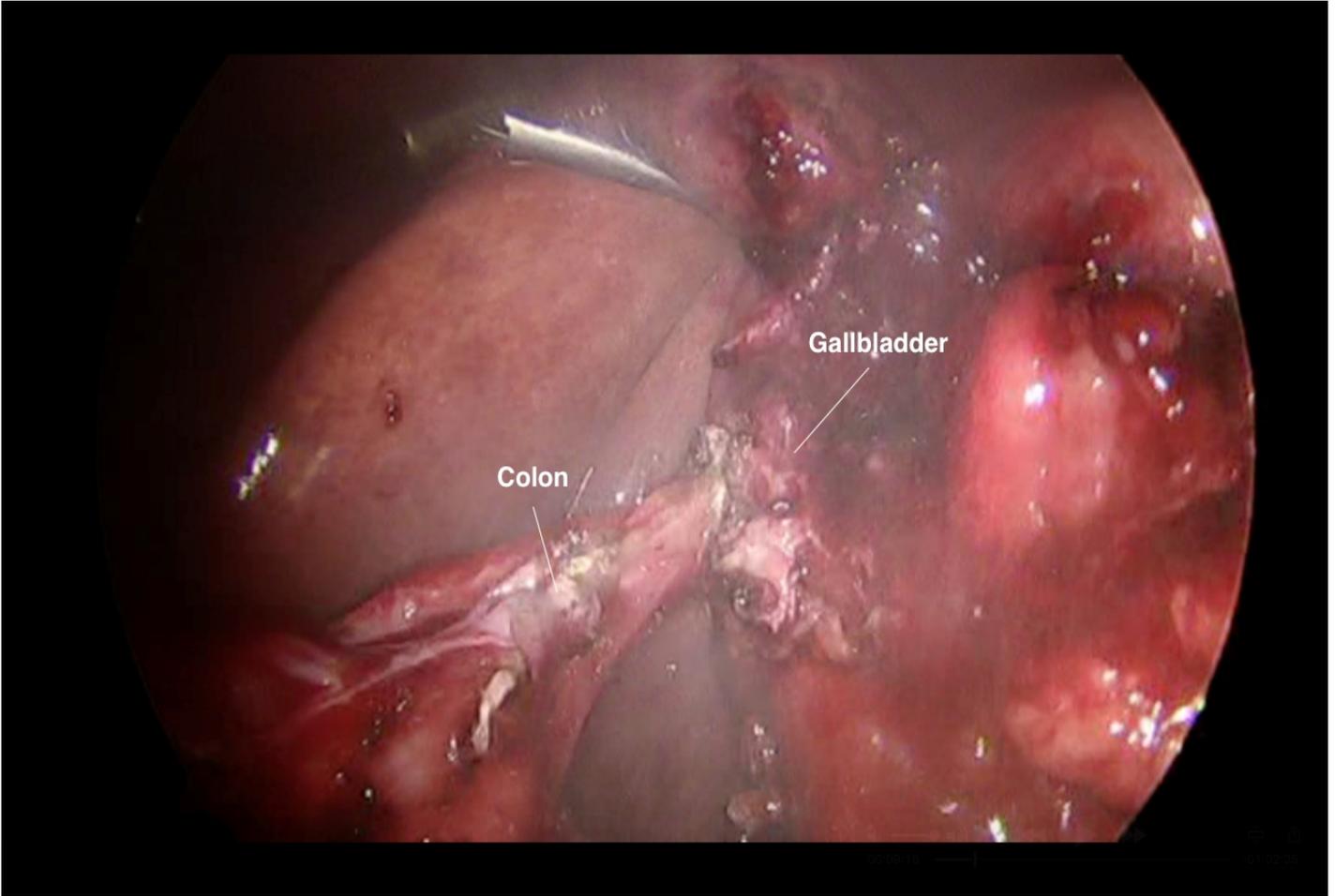


Figure 5: Abdominal CT scan without contrast demonstrating interval increase in gallstone size and close adherence to surrounding intestinal structures. (A). Severe focal fatty infiltration with vascularity surrounding the gall bladder. (B). The white arrow indicates adhesions between the transverse colon and gallbladder suspicious for a cholecystocolic fistula. The black arrow indicates a similar adhesion connecting the duodenum and gallbladder suspicious for a cholecystoduodenal fistula.

Intraoperative Images





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