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Case Report

Cholecystogastric Fistula Penetrating Duodenum and Caused a Duodenal Obstruction: A Case Report

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Abstract

Background

Cholecystoenteric fistula, a rare complication of gallstone disease, can cause obstruction of the digestive tract by the form of gallstone ileus or Bourveret syndrome. Cholecystogastric fistula is a minor variant of cholecystoenteric fistula with a frequency of approximately 5% of cholecystoenteric fistula. We describe an extremely rare case report of the cholecystogastric fistula, caused by dense calculous cholecystitis, penetrated the duodenum, caused duodenal obstruction and needed differentiation from gallbladder cancer infiltrate to neighboring organ.

Case Presentation

A 58-year-old man suffering from nausea and abdominal fullness those had persisted for one month referred to our hospital. The patient's abdomen was distended but not associated with tenderness. Upper gastrointestinal tract endoscopy revealed much amount of diet residue, deformity of the pylorus, small depressed lesion with central small nodule at the posterior wall of the antrum and stenosis of the duodenal bulb without tumorous findings. Contrast enhanced computed tomography revealed a significantly expanded stomach, a significant thickness of the gallbladder wall, and the ampulla of the duodenum pulled into and pressed between the gallbladder and the antrum. Magnetic resonance imaging showed no signal in the round gallbladder lumen indicating the lumen was occupied for a calculus without bile juice. After preoperative preparation, we conducted the open surgery, however, the cholecystitis was very intense and the confirmation and detachment of the structure of biliary tract have finally reached the biliary injury. We performed cholecystectomy, extrahepatic bile duct resection, antrectomy, hepaticojejunostomy and reconstruction with Roux-en Y manner. Pathologically, a narrow fistula was found between the root of the small nodule at the posterior wall of the antrum and the gallbladder. In the cut surface of the fistula, between the antrum and the gallbladder, the bulb of the duodenum with the proper muscle layers of both anterior and posterior wall was intervened and the penetrated by the cholecystogastric fistula.

Conclusion

This case demonstrated that dense cholecystitis can have a potential to develop cholecystogastric fistula and cause unique duodenal obstruction by penetration of the duodenum unlike past reports.

Keywords: Cholecystogastric fistula; Cholecystoenteric fistula; Cholecystoduodenal fistula; Calculous cholecystitis; Duodenal obstruction; Gallbladder cancer

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Background

Cholecystoenteric fistula (CEF) is a rare complication of gallstone disease, occasionally found during operation, reported to as high incidence as 0.5 to 0.9 % [1-2]. CEFs usually present as cholecystoduodenal fistulas (CDFs) and contribute to 68% of the cases as a result of the proximity of the duodenum to the gallbladder, and less common variants, cholecystocolic or cholecystogastric fistulas, account for 17% and 5% of the cases respectively. Cholecystogastric fistula (CGF) is believed to be the least frequent among CEFs due to the thickness of the gastric wall. Most of CEF is associated with complication of the cholecystolithiasis and could cause obstruction of the digestive tract by the form of gallstone ileus or Bouveret syndrome; the latter is a minor variant of CEF associated gallstone ileus characterized with gastric outlet obstruction caused by a large gall stone passed through a CDF or CGF [3-6]. However, CGF penetrating duodenum and directly cause duodenal obstruction and showed similar clinical presentation of gastric outlet obstruction to Bouveret syndrome is seemed to be extremely rare and to our best knowledge, this is the first report of such complicated disease. We describe a case report of the CGF that was caused by dense calculous cholecystitis penetrated the duodenum and caused duodenal obstruction needing differentiation from gallbladder cancer infiltrate to neighboring organ.

Case Report

A 58-year-old man visited his local doctor with the complaint of nausea and abdominal fullness those had been persisted for one month and referred to our hospital. The patient had never experienced abdominal pain. He had comorbidities of hypertension and diabetes

mellitus and both were well controlled by medication. Physical examination showed height of 171.7cm, body weight of 67.6 kg, body mass index (BMI) of 21.2 kg/m2, blood pressure of 110/86 mmHg, pulse rate of 86 /minute and body temperature of 37.2 ℃. The patient's abdomen was distended but not associated with tenderness. Routine laboratory tests showed no inflammatory reaction (white blood cell count: 4630/μL; C-reactive protein: 0.16 mg/dL), no liver dysfunction (total bilirubin: 1.04 mg/dL; AST: 20 IU/L; ALT: 18 IU/L, ALP: 47 U/L; LDH: 171 U/L), no renal dysfunction (BUN: 11.9; creatinine: 0.64 mg/dL), and no malnutrition (total protein: 7.8 g/dL; albumin: 4.5 g/dL). Serum values of such tumor makers as CEA or CA19-9 showed within normal level (1.6 ng/mL and 6.1 U/mL; respectively). The patient underwent upper gastrointestinal tract endoscopy and revealed much amount of diet residue, deformity of the pylorus, duodenal ulcer scar (Figure 1A), stenosis at the duodenal bulb (Figure 1B), and we were able to just barely navigate the endoscope through to the descending portion of the duodenum (Figure 1C). In the stenotic portion of the duodenal bulb, the findings of the exposure of epithelial tumor were not observed. At the posterior wall of the antrum, there was a small depressed lesion with the small mucosal nodule in the center (Figure 1D). Contrast enhanced computed tomography (CECT) revealed a significantly expanded stomach, a significant thickness of the gallbladder wall with clear round outline of the lumen side with 13 mm inside diameter (Figure 2A). The liver at the site where the gallbladder was attached showed an intensive contrast effect than other parts of the liver in early phase of enhancement protocol, and showed same density with other parts of liver parenchyme in latter phase, reflecting a severe inflammation. The gallbladder contacted to gastric antrum and showed a little ill-defined border. In the sagittal view, the ampulla of the duodenum was depicted to be pulled into and pressed between the gallbladder and the antrum (Figure 2B). In the coronal view, the similar imaging findings were depicted (Figure 2C). Magnetic resonance imaging (MRI) showed no signal in the gallbladder indicating the lumen of the gallbladder was occupied for a calculus without bile juice in the T2 weighted imaging (T2WI) (Figure 3A). Magnetic resonance cholangiopancreatography (MRCP) showed that the common hepatic duct was slightly pressed to left-dorsal direction by the thickened gallbladder without irregular narrowing or disruption (Figure 3B). In the sagittal view of fast imaging employing steady state acquisition (FIESTA), the duodenal blub was pulled into and pressed between the gallbladder and the antrum and each border was remained to be unclear (Figure 3C). After intubation of nasogastric tube, drainage of gastric content, fluid infusion and administration of antibiotics were initiated. Preoperative imaging diagnosis was unable to distinguish between cholecystitis involving adjacent organs and that due to gallbladder cancer. Because a passage obstruction due to duodenal stenosis was present, at least surgical relief of the stenosis was required. Therefore, we made the surgical policy that a resection similar to that for gastric cancer was performed with antrectomy, and the gallbladder was removed by cholecystectomy similar to that for cholecystitis. Rapid pathological diagnosis was planned during the surgery. If the diagnosis had revealed with gallbladder cancer, in addition to cholecystectomy, liver bed resection combined with extrahepatic bile duct resection, lymph node dissection, and hepaticojejunostomy would have been performed. An open surgery was performed on five days after admission.

The gallbladder was covered with the adhered and thickened greater omentum, the duodenum and the gastric antrum. We exfoliated them and incised thickened hepatoduodenal ligament, and isolated the proper hepatic artery and the common hepatic artery then taped them each other, and the portal vain was also isolated. However, extrahepatic bile duct including the cystic duct was not isolated in the thickened connective tissue. The stomach was cut at the antrum using a stapler and the upper portion of the descending part of the duodenum which was taken from the pancreas head was also cut using a stapler. At this time, the outline of the gallbladder and bile duct remained to be unclear. We dissected the gallbladder from the liver bed from the fundus and in this process the round cholesterol calculus was extracted from the thickened gallbladder, and then the common hepatic duct was separated, therefore we conducted extrahepatic bile duct resection, hepaticojejunostomy and reconstructed by Roux-en Y manner. Postoperatively, the leakage of the hepaticojejunostomy was complicated, the patient was relieved by conservative treatment and discharged on the postoperative day 27.

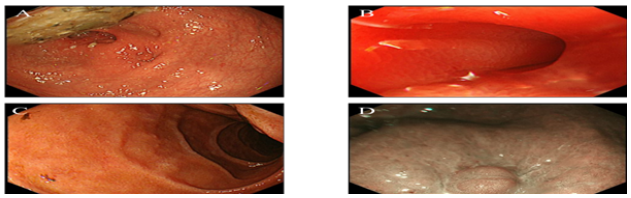

Figure 1: Upper gastrointestinal tract endoscopy findings. A: endoscopy revealed much amount of diet residue, deformity of the pylorus and duodenal ulcer scar. **B:** The endoscopy also revealed a stenosis at the duodenal bulb without the findings of the exposure of epithelial tumor. **C:** The endoscope was barely inserted to the descending portion of the duodenum passing through the stenotic portion. **D:** At the posterior wall of the antrum, there was a small depressed lesion with the small mucosal nodule in the center.

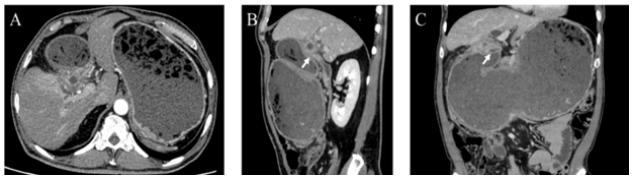

Figure 2: CECT findings. A: Axial view. CTCE revealed a significantly expanded stomach, a significant thickness of the gallbladder wall with clear round outline of the lumen side. The hepatic bed received more intense contrast effect than the surrounding liver tissue in the early phase of imaging. **B:** Sagittal view. The ampulla of the duodenum (arrow) was depicted to be pulled into and pressed between the gallbladder and the antrum. **C:** Coronal view. The similar imaging findings were depicted (arrow: the pressed ampulla of the duodenum between the thickened gallbladder and the antrum).

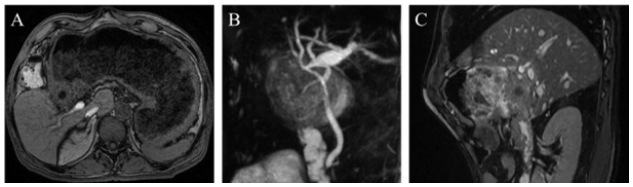

Figure 3: MRI findings. A: Axial view of the T2WI. MRI showed no signal in the gallbladder indicating the lumen of the gallbladder was occupied for a calculus without bile juice. **B:** MRCP findings. The common hepatic duct was slightly pressed to left-dorsal direction by the thickened gallbladder without irregular narrowing or disruption. **C:** Sagittal view of FIESTA. The duodenal bulb was pulled and pressed between the gallbladder and the antrum and each border was remained to be unclear.

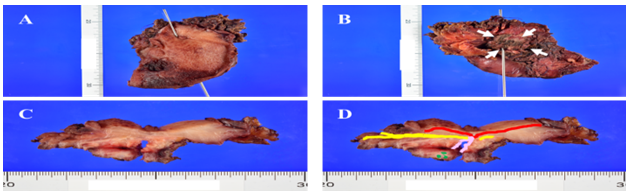

Figure 4: Macroscopic findings of the resected specimen consisted with the antrum, the duodenum, the gallbladder and uncertain bile duct. **A:** View from gastric mucosa side. On the posterior wall of the antrum, a small nodule was found in the center of the small depressed lesion, and between the root of the nodule and the gallbladder, a narrow fistula was formed. Arrows indicate the pylorus. **B:** View from gallbladder side. The fistula reached on the gallbladder mucous membrane side. Arrows indicate the gallbladder. **C:** The cut surface of the fistula between the antrum and the gallbladder. The bulb of the duodenum with the proper muscle layers of both anterior and posterior wall was intervened and the diagnosis with CGF penetrated the bulb of the duodenum was made. **D:** Markings-added picture to Figure 4C. Red line indicates gastric mucosa, yellow lines indicate duodenal mucosa, pink line indicates granuloma tissue and green areas indicate gallbladder mucosa.

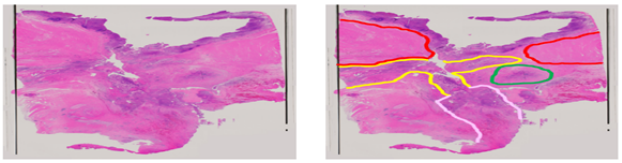

Figure 5: Microscopic findings. **A:** The structure of the duodenum, accompanied by muscle layers and Brunner's glands, can be observed on both sides of the fistula, and it comes to that the fistula penetrated the duodenum. **B:** Marking-added picture to Figure 5A. Tissue of the cholecystic duct can be observed in the area circumscribed by green line, closed to the stomach whose proper muscle was indicated by red lines, the duodenum indicated by yellow lines and the fistula indicated by pink lines.

Pathological Findings

Macroscopic findings

The specimen consisted with the antrum, duodenum, gallbladder and uncertain bile duct. On the posterior wall of the antrum, a small nodule of the 5 mm size was found in the center of small depressed lesion, and between the root of the nodule and the gallbladder, a narrow fistula was formed (Figures 4A,B). In the cut surface of the fistula between the antrum and the gallbladder, the bulb of the duodenum with the proper muscle layers of both anterior and posterior wall was intervened and the diagnosis with CGF penetrated the bulb of the duodenum was made (Figure 4C,D).

Microscopic findings

The structure of the duodenum, accompanied by muscle layers and Brunner's glands, can be observed on both sides of the fistula, and it comes to that the fistula penetrates the duodenum (Figures 5A,B). Tissue of the cholecystic duct can be observed (circumscribed by green line in Figure 5B) closed to the stomach, the duodenum and the fistula. Around the fistula, thickened fibrotic tissue including thickened nerve bundle was formed and was considered to be resulted by chronic inflammatory reaction. No findings of the malignancy were evident.

Discussion

Cholecystolithiasis can rarely cause gastrointestinal obstruction, which is known as gallstone ileus, where a large gallstone falls into the gastrointestinal tract through a CEF, among them, especially causing impaction in the proximal duodenum or pylorus due to gallstones passing through a CDF or CGF is known as Bouveret syndrome. The presented case involved the formation of a CGF from cholecystitis with extensive inflammation. Interestingly, during this process, the duodenum was pulled and trapped between the gallbladder and stomach, leading to the unique occurrence of duodenal obstruction through penetration unlike the past reported CDF or CGF cases. The gallstone itself remained within the gallbladder and was not directly involved in the obstruction of the gastrointestinal tract. This case demonstrated the potential for calculous cholecystitis to cause duodenal obstruction by forming CGF penetrating the duodenum, as seen in this presentation, however, it is considered extremely rare for duodenal obstruction to occur in this fashion, and as far as we could find in our search, no similar reports were found.

The mechanism of formation for a CEF is understood to occur as follows: gallstones are formed, and due to cholecystitis, dense adhesions are formed between the gallbladder and surrounding viscera. Large gall stones, especially those filling the gallbladder, continually compress the gallbladder wall, leading to local ischemia, ulcer formation, necrosis, and eventually the formation of a fistula between the adjacent viscus. In the presented case, the gallbladder was densely adhered to the duodenal bulb, and the opposing walls of the duodenum and stomach were continually compressed in a fashion in whom the duodenal bulb was trapped between the gallbladder and stomach, leading to the formation of dense adhesions also between the duodenum and stomach. Ultimately, it was thought that this led to the formation of a CGF that penetrated both layers of the duodenum. Therefore, in this case, it was considered that a unique progression occurred, where a special variant of CDF was formed during the formation process, followed by penetration of the thick gastric wall, resulting in the formation of the CGF penetrating the duodenum. The chronic gastric dilatation resulting from duodenal obstruction might have contributed to the formation of the CGF by fixation of antrum and received continuous mechanical pressure to the local antral area. This might have been one of the important factors to facilitate the formation of such fistula.

According to the CECT findings, a 13mm gallstone, which was also confirmed on MRI, was stuck inside the gallbladder, and the relatively homogenous thickening of gallbladder was showed around the central gall stone, and an outline of the gallbladder could barely distinguish it. The gastroduodenal mucous membrane close against gallbladder could be also confirmed to maintain a continuity without irregularity or duodenal mural thickening, and was different from the conventional image of the gastroduodenal permeation of the advanced gallbladder cancer, for instance, defined as loss of the fat plane between the gallbladder mass and the duodenum with or without evidence of duodenal mural thickening and mucosal irregularity or extrinsic duodenal compression [7-9]. Although inflammation was revealed around the hepatic artery close to the gallbladder, an apparent encasement of the hepatic artery was not shown. Lymphadenopathy in the hepatoduodenal ligament was also not depicted. The hepatic bed received more intense contrast effect than the surrounding liver tissue in the early phase of imaging, and it was visualized with equal contrast to the surrounding liver tissue in the late phase, it was

suggested that it was not the liver bed infiltration of the advanced gallbladder cancer but influence of inflammation. Although the common bile duct was pushed, the wall of bile duct was smooth and not seen in irregular stenosis or disruption by swollen gallbladder. The thickened gallbladder wall was more intensely contrasted in a latter phase than an early phase and seemed to suggest fibrosis of the gallbladder wall. These findings suggested influence due to intense inflammation of the gallbladder unlike image evidence of conventional advanced gallbladder cancer. Detailed interpretation of the image findings is crucial. However, we could not completely deny the possibility of invasion due to advanced gallbladder cancer still, because we had not experienced cholecystitis severe enough to cause obstruction in the duodenal bulb or poor extension of the descending part of the duodenum before, and while there are few reported cases, there have been reports of the development of CEF consequent to a gallbladder malignancy [11-13]. Because a passage obstruction due to duodenal stenosis was exist, and at least surgical relief of the stenosis and detailed confirmation of the cause was required, we planned the open laparotomy and cholecystectomy with quick pathological diagnosis during surgery. After having expected relief of inflammation by treatment with administration of antibiotics and gastric drainage, we conducted the operation, however, the cholecystitis was very intense, and the confirmation and the detachment of the structure of biliary tract have finally reached the biliary injury with difficulty than preoperative assumption. It was thought that the preparations that could support any situation were necessary on facing the operation of such a case. If it could relieve by noninvasive treatment, it must be more appropriate, and after relief of the dense inflammation, we should inspect it in detail and make a further treatment policy.

Conclusion

We reported the extremely rare case of CGF penetrated the duodenum and caused duodenal obstruction difficult to differentiate with gallbladder cancer. This case demonstrated that dense calculous cholecystitis can have a potential to develop CGF and cause unique duodenal obstruction by penetration of the duodenum unlike past reports of gallstone related digestive tract obstruction. When encountering a case of gastric outlet obstruction associated with severe calculous cholecystitis, surgeons should also consider the possibility of a CGF with duodenal penetration. Detailed interpretation of imaging findings and establishment of treatment strategies based on the patient's condition are crucial. When performing surgery during the acute phase, it is essential to be prepared for any possible scenarios.

List of Abbreviations

CEF: Cholecystoenteric fistula; **CDF**: Cholecystoduodenal fistula; **CGF**: Cholecystogastric fistula; **BMI**: Body mass index; **AST**: Aspartate aminotransferase; **ALT**: Alanine aminotransferase; **ALP**: Alkaline phosphatase; **LDH**: Lactate dehydrogenase; **BUN**: Blood urea nitrogen; **CEA**: Carcinoembryonic antigen; **CA19-9**: Carbohydrate antigen 19-9; **CECT**: Contrast enhanced computed tomography; **MRI**: Magnetic resonance imaging; **T2WI**: T2 weighted imaging; **MRCP**: Magnetic resonance cholangiopancreatography; **FIESTA**: Fast imaging employing steady state acquisition.

Declarations

Ethics approval and consent to participate.

All procedures used in this research were approved by the Ethics Committee of Marunouchi Hospital (#).

Consent for Publication

Written informed consent was obtained from the patient for the publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Availability of Data and Materials

All data generated or analyzed during this study are included in this published article.

Competing Interests

The authors declare that they have no competing interests.

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None.

Author's Contribution

NH participated in the operation and drafted the manuscript. SO performed the preoperative investigation, conducted the operation and interpreted the results. AS, JI and TY participated in the operation and the revision of the manuscript. MK1 conducted the radiological examination and interpreted the images. MK2 conducted the pathological examination and interpreted the results. All authors read and approved the final manuscript.

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