Acute acalculous cholecystitis immediately after gastric operation: Case report and literatures review

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Abstract

Acute acalculous cholecystitis (AAC) is a rare complication of gastric surgery. The most commonly accepted concepts regarding its pathogenesis are bile stasis, sepsis and ischemia, but it has not been well described how to identify and manage this disease in the early stage. We report three cases of AAC in elderly patients immediately after gastric surgery, which were treated with three different strategies. One patient died 42 d after emergency cholecystectomy, and the other two finally recovered through timely cholecystostomy and percutaneous transhepatic gallbladder drainage, respectively. These cases informed us of the value of early diagnosis and proper treatment for perioperative AAC after gastric surgery. We further reviewed reported cases of AAC immediately after gastric operation, which may expand our knowledge of this disease.

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Key words: Acute acalculous cholecystitis; Postoperative complication; Gastric surgery

Core tip: Acute acalculous cholecystitis (AAC) after gastric surgery is not common but it progresses rapidly once it occurs. Manifestations of AAC after gastric surgery are usually atypical and cannot be easily distinguished from other complications, which leads to delayed diagnosis and treatment and even death. Here, we report three such cases and review the literature, to give more attention to this disease. Early diagnosis and prompt surgical intervention are the keys to successful treatment.

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INTRODUCTION

Radical gastrectomy is the main procedure for advanced gastric carcinoma in spite of the rapid progress in adjuvant strategies such as chemotherapy and radiotherapy. Although the exact mechanisms are still not clear, gastric surgery may affect gallstone formation by altering cholecystokinin secretion, gallbladder reaction to cholecystokinin, and subsequently impairing gallbladder filling and emptying. It remains to be elucidated whether gallstone formation and gallstone-related symptoms in patients with previous upper gastrointestinal (GI) surgery are more frequent. It has been revealed that symptomatic postoperative gallbladder stone formation may occur in 17.5% of gastric cancer (GC) patients[1]. Until now, there has been no general consensus about whether it is necessary to remove the gallbladder during gastric surgery.

Acute acalculous cholecystitis (AAC) following radical
gastrectomy, which may result from bile stasis and gallbladder ischemia associated with vagotomy and gastrophatic ligament dissection, has been sporadically but rarely reported\(^2,3\). In the general population, AAC immediately after radical gastrectomy, a clinical situation which is not easily recognized in elderly patients, is characterized by delayed diagnosis and high mortality rate. In this paper, we report its clinical features, management strategies, and outcomes in three patients.

**CASE REPORT**

**Case 1**

A 60-year-old Chinese male patient suffered from upper abdominal pain for > 1 year, and this painful feeling had become more severe since 2 mo ago. Endoscopy revealed an ulcerated tumor at the back wall of the upper gastric body, close to the greater curvature. Biopsy showed poorly differentiated adenocarcinoma, and the clinical stage was cT2N0M0 according to preoperative computer tomography (CT), and the gallbladder seemed normal (Figure 1A). Total gastrectomy (D2 lymphadenectomy) and Roux-en-Y reconstruction were performed. He was able to tolerate liquid food on day 4 and semi-fluid on day 7 after the operation and was discharged on day 9 without any discomfort. The postoperative pathological stage was pT2N0M0. Ten days after the operation, the patient suddenly showed upper abdominal discomfort accompanied by nausea and hyperpyrexia after a greasy meal, and his body temperature was as high as 39.5 °C. Ultrasound examination was performed but no abnormality was found and the possibility of abdominal infection was considered, so antibiotic treatment was performed but was ineffective, and he was readmitted on day 13. Physical examination showed apparent tenderness in the right upper abdomen and concussive pain in the liver region, and white blood cell (WBC) count 24.35 × 10\(^9\)/L. Renal and liver function and serum amylase were within the normal range. Ultrasound and abdominal/pelvic CT showed swelling of the gallbladder and moderate effusion in the abdominal cavity and around the gallbladder. The gallbladder was about 8 cm × 4 cm × 4 cm (Figure 1B). Imipenem was used for 24 h but his symptoms were not relieved, and on day 2 after readmission, the patient suddenly presented with symptoms of septic shock and blurred consciousness, anuria, decreasing blood pressure (BP, from 100/60 to 85/55 mmHg) and increasing heart rate (HR). Emergency exploratory laparotomy was performed with fluid resuscitation and adequate intravenous antibiotics. During surgery, we found that the gallbladder was about 9 cm × 5 cm × 6 cm, and severely adhered to adjacent tissues such as duodenum and colon. There were several gangrenous lesions on the gallbladder fundus wall with no perforation. Fistulization of the gallbladder was performed, and a large amount of purulent bile was exuded. After the operation, the body temperature returned to normal, and antibiotic therapy was continued until day 5. Semifluid was started on the day 9 and he was discharged on day 14. Two months after the emergency surgery, the gallbladder fistulization tube was removed successfully, and no more associated discomfort occurred.

**Case 2**

A 79-year-old male patient had repeated melena for 2 wk. Gastric endoscopy revealed an ulcerated tumor (1.0 cm in size) at the back wall of gastric fundus, close to the lesser curvature, and further biopsy confirmed poorly differentiated adenocarcinoma. The patient was ASA grade 3, had no history of gallstones or cholecystitis, but was suffering from impaired cardiac function, moderate disorder of pulmonary ventilation, and a 10-year history of cerebral infarction, without taking anti-coagulation or anti-platelet treatment. Preoperative ultrasound and CT displayed a normal gallbladder and multiple hepatic cysts (Figure 2A), and the clinical stage was cT4N + M0. Total gastrectomy (D2 lymphadenectomy) with Roux-en-Y reconstruction was performed, and the pathological stage was pT4aN2M0 pathologically. Semi-liquid diet was well tolerated on day 7 and drainage tubes were removed within 8 d postoperatively. His body temperature was normal, and he did not feel any discomfort. However, in the morning of day 9, a sudden feeling of severe pain arose in his right upper abdomen, with fever (38.5 °C), fast heart rate (120 bpm) and high WBC count (17.8 × 10\(^9\)/L). Laboratory examinations showed impaired liver and renal function: total bilirubin/conjugated bilirubin (TB/CB) 85/74 µmol/L, aspartate aminotransferase (AST)/alanine aminotransferase (ALT) 225/671 U/L, serum creatinine...
151 µmol/L. Pro-calcitonin (PCT) was elevated to > 100 ng/mL, and serum amylase was normal. Emergency abdominal CT showed an enlarged gallbladder with thickened wall, intestinal obstruction and fluid accumulation in the abdominal and pelvic cavities (Figure 2B). In consideration of the possibility of strangulating intestinal obstruction with bloody ascites, abdominocentesis was performed and 20 mL transparent and faint yellow ascites was obtained. The patient was treated with antibiotics. At 05:00 AM on the next day, typical manifestations of septic shock appeared, including hypoxemia (SpO$_2$ 85%), loss of consciousness, tachycardia (124 bpm) and a sharp decrease in BP (85/50 mmHg). Emergency exploratory laparotomy was carried out. There was severe adhesion in the abdomen among the intestines, and about 400 mL slightly turbid and yellow ascites was drained during the operation. The hypertonic gallbladder was about 10 cm × 6 cm × 5 cm, and the wall was apparently edematous with several gangrenous lesions in the body and fundus of the gallbladder. Due to the severe adhesion in the region of the hepatoduodenal ligament and difficulties in clearly detecting the structures of the bile ducts and Calot’s triangle, most of the gallbladder was removed instead of performing total cholecystectomy, and the stumps were sutured. Antibiotic therapy with meropenem was continued after the patient was transferred to intensive care after the operation, and his body temperature fluctuated around 38℃. Liver function of the first day after the operation showed: TB/CB 150.2/140.5 µmol/L, and liver protective therapy was given, but he showed an increasingly elevated direct bilirubin level, with a relatively normal level of transaminase (Table 1). For better judgment of the function of the common bile duct and hepatic ducts, magnetic resonance imaging and ultrasound were both performed several times after surgery and no dilated bile ducts and obstruction were detected (Figure 2C). However, total and direct bilirubin kept on increasing as high as TB/CB 541.1/491.4 µmol/L. The patient gradually developed severe pulmonary infection with multiresistant bacteria and fungi 2 wk after the operation. Chronic heart failure occurred and finally he died from severe multiple organ dysfunction syndrome and septic shock 42 d after the operation.

**Case 3**

A 72-year-old male patient had been suffering from upper abdominal distension accompanied with repeated nausea and vomiting for nearly 1 mo. Gastric endoscopy showed a large mass in the antrum with involvement of the pylorus, and biopsy showed mucinous adenocarcinoma. Preoperative CT and positron emission CT (PET-CT) showed lymph node metastasis in the peri-gastric, hepatic portal and peri-pancreatic region at cT4N3M1. According to the strong wishes of the patient and his family, reductive distal gastrectomy with Billroth I reconstruction was performed. On day 6 after the operation, the patient complained of tachypnea and fever (38.9℃). CT and ultrasound showed fluid accumulation and intra-abdominal infection, so a drainage tube was placed guided by ultrasound, and tentative antibiotic treatment was given. On day 12, he presented with an incision infection. On day 18, his temperature returned to normal, and repeated CT showed that fluid accumulation in the abdomen decreased. Semi-liquid diet was well tolerated on day 21 and drainage tubes were all removed by day 28 after surgery, so he was discharged on the next day. On day 37, the patient suddenly felt upper abdominal distension accompanied by nausea and vomiting, and the body temperature was still normal. He was treated by adequate fluid perfusion and fasting in the emergency room but the symptoms were not released. Right upper abdominal pain and fever arose on the next day, and he was readmitted again. Physical examination showed temperature 38.9℃, heart rate 105 bpm, BP 160/80 mmHg, and apparent tenderness and concussion in the right upper abdomen, with positive Murphy’s sign. Laboratory examination showed WBC count was elevated (18.05 × 10$^9$/L). Liver function, renal function and serum amylase were all within the normal range. Emergency ultrasound showed an enlarged gallbladder (9 cm × 4.5 cm in size) with exudation around it (Figure 3). Percutaneous transhepatic gallbladder drainage was practiced immediately, and the
AAC is defined as acute cholecystitis (AC) without detection of any gallstones, which accounts for 2%-12% of AC cases [4,5]. AAC immediately after gastric operation is also rare. To gain a better understanding of the clinical fluid drained out was slightly turbid, and antibiotic therapy was continued. His temperature returned to normal and abdominal pain was apparently resolved on the next day. On day 4, he started a semi-fluid diet, and he was discharged on day 5 after readmission, with the drainage tube in place. On day 19, the tube was removed.

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features of AAC and guide our diagnosis and treatment, we reviewed previous studies. We systematically searched the English-language articles in the PubMed (1950-2013), using the following terms: “acute cholecystitis”, “acute acalculous cholecystitis”, “postoperative acute cholecystitis” and “postoperative acute acalculous cholecystitis”, and 7928 articles were obtained, among which, 1952 were case reports and 101 were meta-analyses or systematic reviews. The studies after gastric surgery were clearly defined, and the criteria for no stones on imaging, no stones detected at surgery, and histology-confirmed acute cholecystitis were used. Cases excluded were those with postoperative AAC after trauma, non-gastric operations, and where the antecedent surgery was not clearly identified. Patients with common bile duct and intrahepatic stones were also excluded. All obtained articles were carefully examined and their reference lists were systematically reviewed to identify other studies for potential inclusion in this review. None of the 101 reviews concerned postoperative AAC except for one Japanese case report [6]. Finally, 13 articles/case reports and 34 cases (as well as the three cases reported in the present report) were included in the following review (Table 2, partially adapted from Crichlow et al [7]). We concluded that this disease occurred mostly in men (25 men and 2 women) at an average age of 64.13 ± 11.32 years (range: 37-79 years), similar to that in previous reports of male and elderly predominance [4,8-10]. The mean time to manifestation of postoperative AAC symptoms was 17.6 d (range: 7-45 d). According to the available data, gangrene formation was

| Ref. | Year | Gender | Age (yr) | Prior surgery | Days after surgery | Treatment | Gangrene formation | Survival |
|------|------|--------|----------|---------------|-------------------|-----------|--------------------|----------|
| Schwegman et al [27] | 1953 | Male | 43 | TG | 11 | CO/PTGD | No | Yes |
| Glenn et al [26] | 1979 | Male | 65 | PG | 45 | CE | NA | No |
| Howard et al [25] | 1981 | Male | 42 | DG | NA | CE | No | NA |
| Lens et al [24] | 1981 | Male | 75 | TG | NA | CO/PTGD | NA | Yes |
| Glenn et al [29] | 1982 | Male | 55 | DG | 42 | CO/PTGD | NA | Yes |
| Becker et al [23] | 1986 | Male | 37 | TG | 7 | CE | No | NA |
| Fabian et al [22] | 1986 | Male | 68 | PG | NA | CE | NA | No |
| Jensen et al [21] | 1987 | Male | NA | TG | NA | NA | NA | NA |
| Jensen et al [21] | 1987 | Male | NA | TG | NA | NA | NA | NA |
| Berge [5] | 1987 | Male | NA | TG | NA | CO/PTGD | NA | Yes |
| Wu et al [20] | 1995 | Male | 56 | TG | 18 | CE | Yes | No |
| 1995 | Male | 62 | TG | 21 | CO/PTGD | NA | Yes |
| 1995 | Male | 66 | TG | 19 | CE | Yes | No |
| 1995 | Male | 64 | TG | 8 | CE | Yes | Yes |
| 1995 | Male | 65 | TG | 11 | CE | Yes | Yes |
| 1995 | Male | 57 | TG | 14 | CE | No | Yes |
| 1995 | Male | 75 | TG | 18 | CE | Yes | Yes |
| 1995 | Male | 72 | TG | 16 | CE | Yes | Yes |
| 1995 | Male | 74 | TG | 18 | CE | No | Yes |
| 2001 | Female | 71 | PG | 11 | CE | Yes | Yes |
| 2001 | Female | 73 | PG | NA | NA | CE | NA | Yes |
| 2001 | Female | 75 | PG | NA | NA | CE | NA | Yes |
| 2001 | Female | 78 | PG | NA | NA | CE | NA | Yes |
| 2001 | Female | 80 | PG | NA | NA | CE | NA | Yes |
| 2001 | Female | 81 | PG | NA | NA | CE | NA | Yes |
| 2001 | Female | 82 | PG | NA | NA | CE | NA | Yes |
| 2001 | Female | 83 | PG | NA | NA | CE | NA | Yes |
| Liu et al [19] | 2010 | Female | 64 | DG | 9 | AA | NA | Yes |
| 2010 | Male | 76 | DG | 14 | CO/PTGD | NA | Yes |
| 2010 | Male | 64 | TG | 14 | CO/PTGD | NA | Yes |
| Liu (our report) | 2013 | Male | 60 | TG | 10 | CO/PTGD | Yes | Yes |
| 2013 | Male | 79 | TG | 9 | CE | Yes | No |
| 2013 | Male | 72 | DG | 37 | CO/PTGD | NA | Yes |

TG: Total gastrectomy; DG: Distal gastrectomy; PG: Proximal gastrectomy; NA: Not available; PTGD: Percutaneous transhepatic gallbladder drainage; CE: Cholecystectomy; CO: Cholecystostomy; AA: Antibiotics alone.
Table 3 Statistical analysis for all 34 cases of acute acalculous cholecystitis after gastric operation  n (%)  

| Gender       | Age (yr) | Surgery | Treatment | Gangrene formation |
|--------------|----------|---------|-----------|--------------------|
| Male         | < 65     | ≥ 65    | TG        | CO/PTGD | AA | Yes | No |
| Female       | 1 (50.0) | 1 (50.0) | 8 (72.7)  | 8 (61.5) | 1 (20.0) | 3 (23.1) |
|              | 8 (61.5) | 1 (100.0) | 3 (42.9)  | 6 (57.1) | 1 (100.0) | 0 (0.0)  |
|              | 5 (38.5) | 1 (66.7)  | 1 (9.1)   | 0 (0.0)  | 1 (20.0)  | 0 (0.0)  |
|              | 1 (100.0)| 8 (66.7)  | 3 (33.3)  | 1 (14.3) | 4 (66.7)  | 1 (20.0) | 0 (0.0)  |
|              | 5 (38.5) | 1 (100.0) | 4 (33.3)  | 1 (14.3) | 4 (66.7)  | 1 (20.0) | 0 (0.0)  |
|              | 1 (100.0)| 1 (100.0) | 1 (100.0) | 0 (0.0)  | 1 (20.0)  | 0 (0.0)  | 0 (0.0)  |
|              | 5 (38.5) | 1 (100.0) | 1 (14.3)  | 0 (0.0)  | 1 (20.0)  | 0 (0.0)  | 0 (0.0)  |
|              | 1 (100.0)| 8 (66.7)  | 1 (100.0) | 0 (0.0)  | 1 (20.0)  | 0 (0.0)  | 0 (0.0)  |
|              | 5 (38.5) | 1 (100.0) | 1 (14.3)  | 0 (0.0)  | 1 (20.0)  | 0 (0.0)  | 0 (0.0)  |
|              | 1 (100.0)| 8 (66.7)  | 1 (100.0) | 0 (0.0)  | 1 (20.0)  | 0 (0.0)  | 0 (0.0)  |
|              | 5 (38.5) | 1 (100.0) | 1 (14.3)  | 0 (0.0)  | 1 (20.0)  | 0 (0.0)  | 0 (0.0)  |
|              | 1 (100.0)| 8 (66.7)  | 1 (100.0) | 0 (0.0)  | 1 (20.0)  | 0 (0.0)  | 0 (0.0)  |
|              | 5 (38.5) | 1 (100.0) | 1 (14.3)  | 0 (0.0)  | 1 (20.0)  | 0 (0.0)  | 0 (0.0)  |
|              | 1 (100.0)| 8 (66.7)  | 1 (100.0) | 0 (0.0)  | 1 (20.0)  | 0 (0.0)  | 0 (0.0)  |
|              | 5 (38.5) | 1 (100.0) | 1 (14.3)  | 0 (0.0)  | 1 (20.0)  | 0 (0.0)  | 0 (0.0)  |
|              | 1 (100.0)| 8 (66.7)  | 1 (100.0) | 0 (0.0)  | 1 (20.0)  | 0 (0.0)  | 0 (0.0)  |
|              | 5 (38.5) | 1 (100.0) | 1 (14.3)  | 0 (0.0)  | 1 (20.0)  | 0 (0.0)  | 0 (0.0)  |
|              | 1 (100.0)| 8 (66.7)  | 1 (100.0) | 0 (0.0)  | 1 (20.0)  | 0 (0.0)  | 0 (0.0)  |
|              | 5 (38.5) | 1 (100.0) | 1 (14.3)  | 0 (0.0)  | 1 (20.0)  | 0 (0.0)  | 0 (0.0)  |
|              | 1 (100.0)| 8 (66.7)  | 1 (100.0) | 0 (0.0)  | 1 (20.0)  | 0 (0.0)  | 0 (0.0)  |
|              | 5 (38.5) | 1 (100.0) | 1 (14.3)  | 0 (0.0)  | 1 (20.0)  | 0 (0.0)  | 0 (0.0)  |

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found in 64.3% of patients (9/14) at the time of diagnosis and treatment, and overall mortality rate was 25% (5/20). All the statistical results are listed in Table 3.

Although various factors are associated with AAC, such as critical illness, surgery, trauma, burns, and total parenteral nutrition, bile stasis and ischemia of the gallbladder wall are the two principle proposed etiologies. After operation, persistent fasting, hypovolemia, parental nutrition and even ileus all can result in bile stasis. From pathological examination, we usually obtain evidence for gallbladder ischemia, which documents multiple arterial occlusions with minimal venous filling. Associated risk factors for ischemia include hemorrhage, dehydration, administration of vasoactive drugs, and microcirculation disorder such as long-term hypertension, diabetes and congestive heart failure, which is similar to that of Case 2 (hypertension, history of cerebral infarction, and persistent infusion of vasoactive drugs during surgery). In these patients, any insult can exacerbate gallbladder ischemia.

The symptoms and treatments differed among the present three cases, as did the outcomes. However, there was still something in common that could be preliminary pro-
posed. Clinical manifestations are usually not typical, but the disease course can progress rapidly, which can even threaten patients’ lives. In the perioperative period, local inflammatory exudation and adhesion are severe, which may further increase the pressure within the biliary tract and promote bacteremia or even septic shock. In contrast, patients usually suffer from weakness, dehydration and suppressed immunity, which damage the compensational ability to some extent when infection occurs. In the present three cases, the time of onset ranged from day 9 to 38 after prior surgery. All three patients demonstrated high fever and right upper abdominal discomfort, with sharply elevated WBC counts. In Cases 1 and 2, C-reactive protein and PCT were apparently elevated (not available in Case 3), and finally bacteremia and septic shock developed. These symptoms are easily confused with other complications immediately after gastric surgery, especially with duodenal stump leakage. Although ultrasound and CT are helpful in diagnosis, sometimes diagnosis can only be confirmed during emergency exploratory laparotomy. If diagnosed and managed in time, more efficiency treatment and better outcome can be expected, as in Case 3.

Percutaneous transhepatic gallbladder drainage (PTGD) can seldom be considered first, due to the difficulty of early precise diagnosis of immediate AAC after gastric surgery. Unfortunately, the exact reason for obstructive jaundice in Case 2 was not clear, because there was no evidence of bile duct occlusion. Furthermore, the management of AAC is still controversial. Actually, three different treatment strategies were carried out in these three patients, including cholecystostomy, cholecystectomy, and PTGD, and hospitalization days, cost and outcomes also completely differed among them. The major clinical information for all the three cases is listed in Table 3.

In most cases, it is difficult to remove the inflammatory gallbladder, especially to dissect the triangle of Calot, due to prior surgery, local exudation and adhesions, therefore, cholecystostomy was performed in the first case, because of the difficulty and impropriety of cholecystectomy. Regardless of the prior operation, the optimal treatment of acute cholecystitis with sepsis in critically ill patients is not well defined, and there is only limited evidence about whether percutaneous drainage or cholecystectomy is preferred. The reported mortality rates do not show any difference. However, we considered on the basis of the present three cases and former reports that timely surgical intervention is usually necessary, such as exploratory laparotomy, especially when the disease progresses without definite diagnosis, or direct ultrasound-guided PTGD if a diagnosis of AAC has been confirmed. Of note, mortality is predominantly related to the severity of the underlying disease rather than the ongoing gallbladder sepsis because prompt treatment is performed in time, as in Case 2.

From the experience of our three cases and literature review, we preliminarily propose that for patients with AAC immediately after GC, timely management is necessary. Patients after surgery for GI anastomosis may profit from supportive examinations such as CT to exclude complications like leakage of the anastomosis. However, in these circumstances, minor cholecystitis can be neglected, and fluid accumulation around the gallbladder can result from the former operation, especially when symptoms are atypical. Occasionally, an emergency situation requires immediate exploratory laparotomy to save the patient’s life, or even for the purpose of confirming the diagnosis, although such an operation is not always the best choice. However, if we are sure that cholecystic sepsis has arisen, ultrasound-guided PTGD is the proper course to treat the sepsis. It has been reported that 35% of AAC patients treated with PTGD did not need any further treatment for cholecystitis and eventually remained symptom free. Besides, the rate of complication is much lower in PTGD treated patients compared with that of cholecystitis (8.7% vs 47%) (25). PTGD has even been recommended in some studies as a definitive treatment for AAC (24,25). PTGD has been shown to be a life-saving, minimally invasive, alternative management in high-risk AAC patients (25,26), especially for those who have undergone a recent gastric operation. Last but not least, there were some other important problems in the whole process of treatment in addition to delayed diagnosis and management. For example, gastrojejunal bypass in a symptomatic patient might be less invasive in an elderly GC patient with a stage T4N + M1 tumor, as in Case 3, in spite of the insistence of the patient and his family. In Case 2, we considered exploratory laparoscopy to confirm the diagnosis, but finally we abandoned it, because we were not sure whether intestinal obstruction had occurred, in which situation, inhalation pneumonia could have been caused by laparoscopy. Exploratory laparoscopy might be another effective treatment choice in these situations.

AAC is a rare but severe complication immediately after gastric operation. It can advance rapidly and be hard to diagnose at an early stage. Emergency surgical intervention is usually necessary when it happens, and if there are no contraindications, cholecystectomy combined with antibiotic therapy should be performed. If inoperability and diagnosis of AAC are confirmed, a cholecystostomy and PTGD are possible and usually effective procedures.

**COMMENTS**

**Case characteristics**
Three male gastric cancer patients presented with fever and abdominal pain after gastric surgery.

**Clinical diagnosis**
Murphy’s sign and percussion pain in the liver region were more or less positive in these cases.

**Differential diagnosis**
Duodenal stump leakage, postanastomotic leakage, abdominal cavity infection, intestinal obstruction, and incisional discomfort.

**Laboratory diagnosis**
White blood cell count and pro-calcitonin were significantly elevated.

**Imaging diagnosis**
A swelling gallbladder with fluid accumulation can be found by computed tomography or ultrasound.
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Pathological diagnosis
Bronchoscopy and biopsy have revealed wild-type K-ras adenocarcinoma of the colon, CDX2/CK20 positive and TFF1/CX7/CD56 negative.

Treatment
Pericutaeous transhepatic gallbladder drainage or other timely surgical interventions such as cholecystectomy and cholecystostomy are necessary.

Related reports
Acute acalculous cholecystitis (AAC) after gastric operations has seldom been reported, but the disease is usually serious once it happens. Besides, the argument about synthetic cholecystectomy during gastric operation has never stopped.

Experiences and lessons
This case report not only presents the symptoms and treatments of AAC after gastric operation, but also indicates the severity of the disease and necessity of timely management; otherwise, septic shock may happen, which will threaten patient’s life.

Peer review
This article reviews and analyzes reported cases to confirm the prevalence and common treatments of AAC after gastric operation, and is helpful in clinical settings.

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