CASE REPORT

Acute acalculous cholecystitis after abdominal wall repair (Rives-Stoppa)

Jurrian C Reurings, Ruben P D Diaz, Luit Penninga, David R Nellensteijn

Department of Surgery,
St. Elisabeth Hospital,
Willemstad, Curaçao

Correspondence to
Dr Luit Penninga, LP@ctu.dk

Accepted 19 March 2014

SUMMARY

Acute acalculous cholecystitis (AAC) is a rare condition normally occurring in critically ill patients. Compared to acute calculous cholecystitis, AAC is associated with complications and has a worse outcome. Hence, knowledge of this condition is very important. We describe a case of a 31-year-old man who developed AAC after abdominal wall repair with mesh (Rives-Stoppa procedure) 1 day after discharge from the hospital. To the best of our knowledge, this is the first paper to report AAC after abdominal incisional hernia repair. Although it is known to be more common in critically ill patients, AAC can also occur postoperatively in outpatients. Early recognition and treatment of AAC may improve outcome.

BACKGROUND

Acute acalculous cholecystitis (AAC) comprises about 5% of all cases of acute cholecystitis.^{1 2} Compared to acute calculous cholecystitis (ACC), AAC often has a more fulminant course, and is more frequently associated with gallbladder gangrene, perforation and empyema.³ Acute cholecystitis is acalculous in about 33% of cases in postoperative patients.³ Although AAC is described in outpatients it remains a condition mostly seen in critically ill patients in association with severe trauma, non-biliary tract surgery, extensive burns, sepsis and prolonged fasting with or without total parenteral nutrition.⁴ For this reason the diagnosis is often missed in outpatients. Delay in diagnosis of AAC in outpatients may lead to an increase in morbidity and mortality. We present a case of a patient who developed AAC after abdominal wall repair after discharge from the hospital.

CASE PRESENTATION

A 31-year-old male patient underwent abdominal wall repair with mesh (Rives-Stoppa procedure) for an incisional hernia. The postoperative course was uncomplicated and the patient was discharged after 5 days. One day after discharge the patient was readmitted with nausea, vomiting and abdominal pain. On physical examination the right upper quadrant of the abdomen was painful. Vital signs were normal with a temperature of 36.7°C.

INVESTIGATIONS

Haemoglobin was 12.8 g/L, white cell count $13.3 \times 10^9/L$, and C reactive protein 12 mg/dL. Liver enzymes were slightly elevated; aspartate aminotransferase 378 IU/L, alanine aminotransferase 133 IU/L, alkaline phosphatase (AF) 253 IU/L, γ -glutamyl transpeptidase 218 IU/L, total bilirubin 2.1 mg/dL and serum amylase 113 IU/L. Abdominal ultrasound was

performed but complicated by the presence of intestinal air, the presence of a postoperative subcutaneous haematoma and the mesh. Although, a gallbladder without stones and with normal sized intrahepatic and extrahepatic bile ducts was found. A subsequent CT of the abdomen was performed to establish firm diagnosis, and showed acalculous cholecystitis with thickening of the gallbladder wall, pericholecystic fluid and inflammation of the surrounding fat tissue (figure 1).

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of AAC includes cholecystitis due to sludge or microlithiasis, not seen on ultrasound/CT scan, malignancies of the gallbladder and/or biliary system and cholecystitis associated with a viral infection such as herpes or a parasite infection like malaria.

TREATMENT

The patient was treated conservatively with intravenous fluids, analgesics, anti-inflammatory drugs and cessation of oral intake. According to the Dutch guidelines on gallbladder disease, we did not routinely start antibiotic treatment.

OUTCOME AND FOLLOW-UP

The patient recovered uneventfully and was again discharged after 8 days. After 6 weeks the patient was re-examined with ultrasonography and no gallstones were found. At that time, he was fully recovered and had returned to work. At 6-month follow-up, the patient was well without recurrence of the hernia. Furthermore, the patient had been free from any biliary colic, or gallbladder-related symptoms or complications.

Figure 1 CT of the abdomen showing acalculous cholecystitis with thickening of the gallbladder wall, pericholecystic fluid and inflammation of the surrounding fat tissue.

CrossMark

To cite: Reurings JC, Diaz RPD, Penninga L, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2014-203781

DISCUSSION

Our patient developed AAC after abdominal hernia repair with mesh (Rives-Stoppa procedure). AAC has been reported after different types of non-hepatobiliary surgery, but not after abdominal wall repair. In our patient AAC was recognised early and conservative treatment was started, and the patient recovered without further complications.

AAC is an inflammation of the gallbladder. AAC occurs more often in older men; male–female ratio 2–3:1 and average age >60 years.^{1 5}

The exact pathophysiology of AAC remains unclear; bile stasis and ischaemia of the gallbladder may lead to inflammation and infection.⁶ Histopathological findings show frequently ischaemia, and gangrene in acalculous cholecystitis.⁵ Positive bile cultures are found in about 61–73% of the cases, and these are predominantly Gram-negative microorganisms including *Escherichia coli* and *Klebsiella*.^{1 3} Relative ischaemia and stasis of bile result in inflammation and secondary invasion of bacteria causing gangrene, empyema and perforation.

Mortality rates in ACC are low, with a reported mortality of about 3%.¹ In contrast reported mortality rates in AAC are about 21%.⁷ Overall high morbidity rates in AAC are reported; gangrene of the gallbladder is seen in about 55–63% of the cases.^{5 6} The frequent occurrence of gangrene in association with AAC supports the possible role of ischaemia in the pathogenesis. Also a gallbladder perforation rate of approximately 15–20% in AAC has been reported.^{3 5 6} These complications may occur more frequently when diagnosis and treatment are delayed.⁴

Traditionally, AAC is diagnosed in critically injured patients, for example, after trauma or major burn injury, gastrointestinal surgery and sepsis.^{1 6} Previous reports have shown that AAC also occurs in outpatients or patients without predisposing conditions.^{4 8}

The clinical presentation of AAC is similar to ACC. About 70–80% of the patients have pain in the right upper quadrant of the abdomen, only 5–8% have diffuse abdominal pain or epigastric pain. Most patients (70–95%) have leucocytosis, and fever is present in about 55%. Slightly elevated liver enzymes occur frequently, due to inflammation of the gallbladder and the surrounding liver parenchyma, including the common bile duct. Less common symptoms are nausea, vomiting and jaundice.^{2 4}

The primary diagnostic modality is abdominal ultrasonography with a sensitivity of 30–92%, and a specificity of 93–98%.^{9 10} When ultrasonography is inconclusive a CT scan should be made to establish firm diagnosis, as CT scan in this context has a sensitivity and specificity reaching 100%.⁹

Conservative treatment of AAC consists of intravenous fluid administration, cessation of oral intake and treatment with analgesics. The Dutch guidelines do not recommend routine start of antibiotics for mild to moderate cholecystitis. The Tokyo guidelines recommend treatment with wide-spectrum antibiotics for moderate to severe acute cholecystitis.¹¹ However, no strong scientific evidence exists to support or refute the use of antibiotics for acute cholecystitis.

Our patient did not show signs of sepsis, and we primarily decided to treat him conservatively without antibiotics. However, we would have initiated antibiotic treatment if our patient showed any signs of clinical deterioration.

While, there lacks confirming evidence regarding the role of cholecystectomy for patients with acalculous cholecystitis, patients with ACC normally undergo cholecystectomy.¹² Meta-analyses comparing early laparoscopic cholecystectomy (within 7 days) with delayed laparoscopic cholecystectomy in patients with ACC have not found significant differences

regarding mortality, bile duct injury and serious adverse events.¹³ However, recent randomised trials have found less morbidity in patients undergoing early laparoscopic cholecystectomy.¹⁴ Critically ill patients or patients with high comorbidity can be treated with percutaneous drainage, followed by a late cholecystectomy (>6 weeks).¹²

To the best of our knowledge, this is the first reported case of AAC after abdominal wall surgery. The present case highlights that AAC can occur in outpatients after abdominal wall surgery and not exclusively in critically ill patients. Patients with AAC may develop gangrene, empyema and perforation of the gallbladder, which are associated with high morbidity and mortality. This may be prevented by early recognition and treatment of AAC, and a high index of suspicion, also in outpatients.

Learning points

- Acute acalculous cholecystitis (AAC) is a rare complication after abdominal (wall) surgery.
- AAC is associated with high morbidity and even mortality, and diagnostic and therapeutic delay may increase morbidity and mortality.
- AAC can also occur in outpatients after surgery, and not only in critically injured patients.

Contributors All authors have contributed to the preparation of the manuscript in a sufficient way. Furthermore all authors have read and approved the final version of the manuscript.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Glenn F, Becker CG. Acute acalculous cholecystitis. An increasing entity. *Ann Surg* 1982;195:131–6.
- 2 Howard RJ. Acute acalculous cholecystitis. *Am J Surg* 1981;141:194–8.
- 3 Devine RM, Farnell MB, Mucha P Jr. Acute cholecystitis as a complication in surgical patients. *Arch Surg* 1984;119:1389–93.
- 4 Savoca PE, Longo WE, Zucker KA, et al. The increasing prevalence of acalculous cholecystitis in outpatients. Results of a 7-year study. *Ann Surg* 1990;211:433–7.
- 5 Ryu JK, Ryu KH, Kim KH. Clinical features of acute acalculous cholecystitis. *J Clin Gastroenterol* 2003;36:166–9.
- 6 Kalliafas S, Ziegler DW, Flancbaum L, et al. Acute acalculous cholecystitis: incidence, risk factors, diagnosis, and outcome. *Am Surg* 1998;64:471–5.
- 7 Crichlow L, Walcott-Sapp S, Major J, et al. Acute acalculous cholecystitis after gastrointestinal surgery. *Am Surg* 2012;78:220–4.
- 8 Parithivel VS, Gerst PH, Banerjee S, et al. Acute acalculous cholecystitis in young patients without predisposing factors. *Am Surg* 1999;65:366–8.
- 9 Mirvis SE, Vainright JR, Nelson AW, et al. The diagnosis of acute acalculous cholecystitis: a comparison of sonography, scintigraphy, and CT. *AJR Am J Roentgenol* 1986;147:1171–5.
- 10 Puc MM, Tran HS, Wry PW, et al. Ultrasound is not a useful screening tool for acute acalculous cholecystitis in critically ill trauma patients. *Am Surg* 2002;68:65–9.
- 11 Yoshida M, Takada T. Antimicrobial therapy for acute cholecystitis: Tokyo Guidelines. *J Hepatobiliary Pancreat Surg* 2007;14:83–90.
- 12 Huffman JL, Schenker S. Acute acalculous cholecystitis: a review. *Clin Gastroenterol Hepatol* 2010;8:15–22.
- 13 Gurusamy KS, Davidson C, Gluud C, et al. Early versus delayed laparoscopic cholecystectomy for people with acute cholecystitis. *Cochrane Database Syst Rev* 2013(6):CD005440.
- 14 Simorov A, Ranade A, Parcells J, et al. Emergent cholecystostomy is superior to open cholecystectomy in extremely ill patients with acalculous cholecystitis: a large multicenter outcome study. *Am J Surg* 2013;206:935–40.

Copyright 2014 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow