



Acute acalculous cholecystitis induced by aortic dissection: report of a case

Aort diseksiyonuna bağlı olarak gelişen akut taşsız kolesistit olgusu

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Acute acalculous cholecystitis (AAC), inflammation of the gallbladder without evidence of calculi, comprises approximately 10% of all cases of acute cholecystitis. Although the mechanism of AAC has not yet been sufficiently clarified, the most commonly postulated theories regarding its pathogenesis are bile stasis, sepsis and ischemia. We present a case of AAC associated with ischemia of the gallbladder caused by aortic dissection Bakey type III.

Key Words: Acute acalculous cholecystitis; aortic dissection.

Akut taşsız kolesistit, tüm kolesistit olgularının yaklaşık %10'unu oluşturur. Kesin mekanizma tam olarak bilinmemekle birlikte, patogeneizde en önemli faktörler safra stazı, sepsis ve iskemidir. Biz bu çalışmada, Bakey tip 3 aort diseksiyonuna bağlı olarak gelişen safra kesesi iskemisine sekonder oluşmuş akut taşsız kolesistit olgusunu sunmayı amaçladık.

Anahtar Sözcükler: Akut taşsız kolesistit; aortik diseksiyon.

Acute acalculous cholecystitis (AAC) is a life-threatening entity that is increasing in frequency. It comprises approximately 10% of all cases of acute cholecystitis.^[1,2] It can emerge after burns, operations, trauma, sepsis, multiple transfusions, total parenteral nutrition, prolonged fasting, and high-dose opioid analgesia.^[1,3]

The role of ischemia in AAC has been analyzed by several authors.^[1,3] Several factors, such as abdominal, cardiovascular or trauma surgery, can induce hypoperfusion of the gallbladder, resulting in AAC. Aortic dissection may be a factor that gives rise to ischemia of the gallbladder and AAC. Only one case of AAC caused by aortic dissection was reported heretofore.^[4]

We herein present a case of AAC caused by aortic dissection Bakey type III.

CASE REPORT

A 62-year-old male with acute abdominal pain radiating to the shoulders was admitted to the hospital. Physical examination at the time of admission revealed a blood pressure of 130/90 mmHg, pulse rate 90/min and abdominal tenderness. The rest of the

physical examination was within normal limits. The leukocyte count was 14700/mm³. Other laboratory data were within normal ranges. Electrocardiogram, chest X-ray and abdominal sonography revealed no pathological findings. Computed tomography (CT) demonstrated aortic dissection Bakey's type III between the diaphragm and origin of the superior mesenteric artery (Fig. 1). Thickening of the gallbladder wall was also demonstrated. The patient was hospitalized, and conservative therapy including β -blocker was initiated. The next day, the patient developed a high temperature, and physical examination revealed abdominal guarding and rebound. He underwent emergency laparotomy, during which an inflamed necrotic gallbladder without stones was found. Further exploration revealed a low flow of the hepatic artery. Cholecystectomy was performed without ligating the cystic artery. Soon after the operation, his liver enzymes increased gradually. Liver enzymes five days postoperatively were as follows: aspartate transaminase (AST): 229 IU/L and alanine transaminase (ALT): 278 IU/L. In the following days, they subsided gradually to normal levels.

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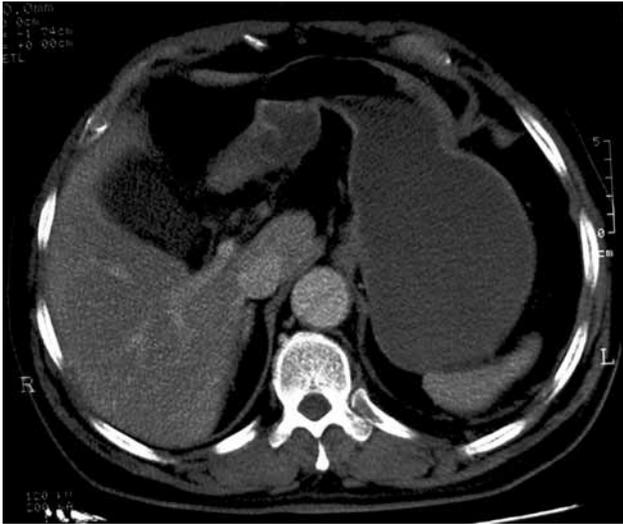


Fig. 1. Computed tomography demonstrated Bakey's type III aortic dissection.

Histological examination revealed that the gallbladder was completely necrotic. Acellular transmural severe necrosis was seen. The details of the cells could not be detected (Fig. 2).

The patient was discharged on the postoperative 8th day. He was in good health at the follow-up four months after the surgery.

DISCUSSION

The pathogenesis of AAC is multifactorial. Several mechanisms have been associated with the development of AAC, such as systemic sepsis with release of mediators, generalized or local ischemia and biliary stasis.^[1]

Previous reports showed that AAC exhibited arterial occlusion with poor venous filling whereas arterial

dilatation and regular filling of the capillary network were consistent with features of acute gallstone-associated cholecystitis.^[5,6] These findings suggest that ischemia of the gallbladder seems to have an important role in the pathogenesis of AAC. Ischemia induces injury and promotes bacterial invasion of the injured wall of the gallbladder.^[3] This process can be initiated by factor XII (Hageman factor) activation. Tissue breakdown from surgery or trauma or multiple transfusions of blood and blood products can induce factor XII activation that affects the gallbladder wall.^[1,3]

The result of several reports have documented an increased incidence of AAC because of hypotension and hypoperfusion of the gallbladder in patients with shock and in those who underwent abdominal, cardiovascular or trauma surgery.^[1,3,7,8] The incidence of AAC is 0.2%-0.05% after cardiac operations such as cardiopulmonary bypass or valve operations.^[8] After the elective or emergency repair of abdominal aorta aneurysm, this pathology is seen in 0.3% and 13.6%, respectively.^[3]

Our case is the second documented case of AAC caused by aortic dissection. We found only one previous case, reported by Roth et al.^[4] They suggested that AAC was induced by aortic dissection and explained this entity asserting two hypotheses: 1- Aortic dissection induced thrombosis of the cystic artery on the basis of low splanchnic flow; 2- Angiography catheter gives rise to thrombosis avulsion from the aortic wall, resulting in embolism of the cystic artery.

We did not perform angiography in our case; therefore, the first hypothesis is plausible. Low hepatic artery flow recognized in our exploration also supports this hypothesis. Low splanchnic flow due to aortic dissection as a causative factor for AAC supports the

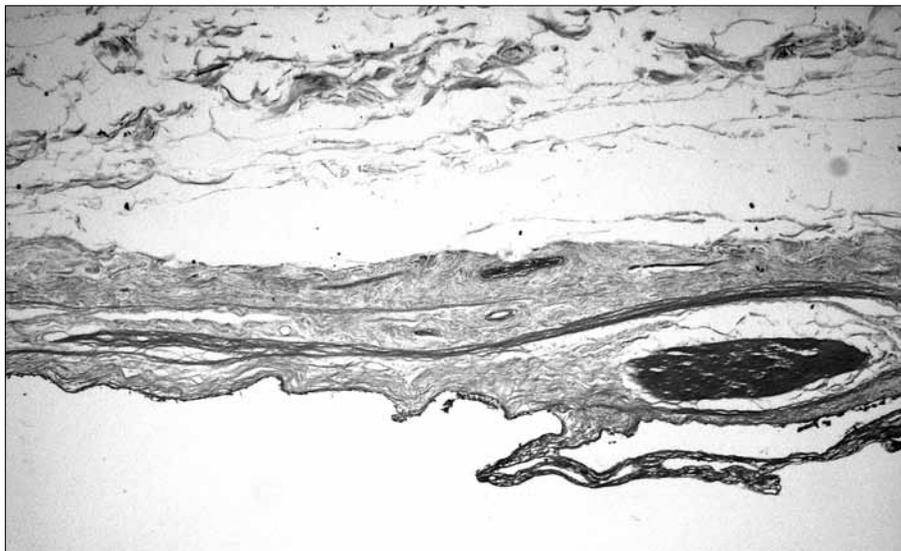


Fig. 2. Acellular transmural necrosis of the gallbladder (H-E x 100).

theory that ischemia, generalized or localized, plays an important role in the pathogenesis of AAC.

Transient liver ischemia caused by the dissection was accompanied with gallbladder gangrene in our case, as described in the previous case. Liver enzymes increased gradually but did not exceed 280 IU/L, and subsided to normal levels gradually.

The clinical and laboratory diagnosis of AAC is often difficult. The best diagnostic imaging techniques are ultrasonography (US) and CT.^[1,8] However, several studies have shown that US is associated with high false-negative rates.^[9] In conformity with these studies, US was not diagnostic in the present case. The thickened gallbladder wall without cholelithiasis indicating AAC was revealed by CT. Radionuclide cholescintigraphy (RC) and morphine cholescintigraphy (MC) have been utilized to confirm the diagnosis of AAC, with high sensitivity rates. In Kalliafas's study,^[1] RC was proposed as the first diagnostic modality for outpatients if the suspicion of AAC is high. The authors proposed that CT should be performed initially if the suspicion for biliary sepsis is low.

Once a diagnosis has been made, the AAC can be treated conservatively. When patients fail to improve, cholecystectomy is the gold standard. Cholecystostomy, a less invasive procedure, can be an alternative to cholecystectomy in critically ill or elderly patients.^[10] In the present case, there was pain and tenderness but no guarding or rebound initially. Therefore, these findings were attributed to aortic dissection, and conservative therapy was initiated. We performed emergency laparotomy and cholecystectomy when peritoneal signs (guarding and rebound) occurred.

Surgeons need to be aware of this condition be-

cause open cholecystectomy is the correct treatment for AAC caused by aortic dissection. In this situation, conspicuous risk of ischemic gallbladder necrosis is present; therefore, conservative therapy should not be considered for treatment. In addition, laparoscopic cholecystectomy should be avoided because increased abdominal pressure gives rise to an increase in the mean arterial pressure, resulting in complications related to aortic dissection.

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