Eosinophilic cholecystitis: An infrequent cause of acute cholecystitis

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ABSTRACT

Eosinophilic cholecystitis (EC) is a rare disease that is characterised by eosinophilic infiltration of the gallbladder. Its pathogenesis is unknown, although many hypotheses have been made. Clinical and laboratory manifestations do not differ from those of other causes of cholecystitis. Diagnosis is histological and usually performed after analysis of the surgical specimen. We report the case of a woman aged 24 years, with symptoms of fever, vomiting and pain in the right upper quadrant. When imaging tests revealed acalculous cholecystitis, an urgent cholecystectomy was performed. Histological examination of the surgical specimen revealed eosinophilic cholecystitis. No cause of the symptoms was found.

Key words: Eosinophilic cholecystitis. Acalculous cholecystitis.

INTRODUCTION

Eosinophilic cholecystitis is an uncommon condition of the gallbladder. It is characterised by an inflammatory infiltrate constituted mainly of eosinophils. Its aetiology is often unknown, although cases have been associated with hyper-eosinophilic syndrome, parasitosis, infections, drugs and medicinal herbs. Clinically, it is indistinguishable from common cholecystitis, although peripheral eosinophilia is sometimes observed, as is the case in hyper-eosinophilic syndrome and parasitic disease. When the effect is limited to the bladder, the treatment of choice is cholecystectomy, and the prognosis is usually favourable.

CASE REPORT

A 24-year-old woman presented to the emergency department complaining of abdominal pain, located in the epigastrium and radiating to the right upper quadrant, together with nausea, vomiting and fever of 39 °C for the past two days. The presence of choluria was also reported. The patient had no personal or family history of interest. She smoked about five cigarettes per day and was a habitual consumer of oral contraceptives. Physical examination revealed good general condition, with cutaneous-mucous jaundice and tenderness in the right upper quadrant, and a positive Murphy sign. Other results of the examination were normal. Laboratory analysis revealed the following alterations: Total bilirubin 3.76 mg/dL (range 0-1.2 mg/dL); direct bilirubin 3.5 mg/dL (range 0-0.5 mg/dL); indirect bilirubin 0.26 mg/dL (range 0-0.75 mg/dL); alanine aminotransferase 174 U/L (range 28-100 U/L); aspartate aminotransferase 49 U/L (range 28-100 U/L); C-reactive protein 149.12 mg/L (range 0-5 mg/L); leukocytes 15,500 µl (range 4.8-10.8 µl); neutrophils 86.3 % (range 40-74 %); lymphocytes 4.2 % (range 19-48 %); monocytes 6.9 % (range 1-9 %); eosinophils 2.2 % (range 0-7 %); prothrombin activity 60.8 % (range 70-120 %), INR 1.34 (range 0.85-1.2); APTT 28.2 sec (range 23.5-33.2 sec). Abdominal ultrasound findings: Thin-walled acalculous gallbladder; non-dilated bile duct; no evidence of pancreatic abnormalities. In view of the clinical and laboratory findings, the patient was admitted to monitor the evolution of the condition and for further study.

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During admission, abdominal and cholangio MRI were performed to assess the bile duct, obtaining the following results: no dilatation; no evidence of bile duct filling defects; space-occupying lesion of 9 mm in segment 8 with uptake and density suggesting hemangioma; no evidence of alterations in pancreas or liver. Further analyses were performed, which revealed increased total bilirubin, decreased direct bilirubin, increased leukocytosis, increased C-reactive protein, and normal levels of amylase, transaminases and cholestatic enzymes. The patient had malaise, with increased pain despite analgesia, and painful abdominal tenderness, with a tightening in the epigastric right upper quadrant. In view of this situation, an urgent abdominal CT scan with IV contrast was performed (Fig. 1), which showed perivesicular oedema without calculi or biliary dilatation, with a small amount of free fluid in the Douglas pouch. There were no images suggestive of perforation or pancreatitis.

The patient’s clinical condition was worsening and presence of cholecystitis was suspected, and so an urgent cholecystectomy was performed, which revealed a thickened gallbladder wall with oedema on the rear surface. The pathology examination revealed the presence of a transmural infiltration, and of a more intense infiltration in the muscular layer, by eosinophilic polymuclear leukocytes (Fig. 2).

After surgery, the patient was asymptomatic and was discharged a few days later.

DISCUSSION

Eosinophilic cholecystitis (EC) is a rare and poorly understood disease of the gallbladder, which was first described in 1949. It can be considered an inflammatory condition of the gallbladder, in which the inflammatory infiltrate consists primarily of eosinophils (1).

The aetiology of EC is unknown. In patients with eosinophilic infiltrate affecting other organs and tissues, it has been suggested that these lesions could be due to a local allergic reaction to substances released at sites of inflammation within the target organ or tissue. It has also been hypothesised that EC may be caused by hypersensitivity to bile acids (2,3).

Cases have also been reported secondary to infections, parasitosis, allergies, hyper-eosinophilic syndrome, eosinophilia-myalgia syndrome, eosinophilic gastroenteritis, drugs and herbal medicines (4,5). In the absence of evident causes, we consider the present case to be an idiopathic EC (6).

EC is three times more common in patients with acalculous cholecystitis than in patients with cholelithiasis (6).

EC does not present any clinical or laboratory manifestation to distinguish it from common cholecystitis, and so it is difficult to detect prior to cholecystectomy and histological examination of the surgical specimen. Peripheral eosinophilia may or may not be present; when it is, it has been associated with hyper-eosinophilic syndrome, eosinophilic gastroenteritis and parasitosis. In addition, symptoms secondary to the eosinophilic infiltration of other organs have been described (8).

Diagnosis is histological; EC is said to be present when the cellular infiltrate in the gallbladder wall is composed of more than 90 % eosinophils, and the cholecystitis is chronic with an eosinophilic (lympho-eosinophilic) component if the infiltrating inflammatory leukocyte population contains 50-75 % eosinophils (9). In imaging tests,
ultrasound results may be normal or show signs suggestive of cholecystitis (gallbladder distension, wall thickening, perivesicular liquid or sonographic Murphy sign). A CT scan may reveal similar features, with perivesicular oedema or decreased attenuation in the adjacent liver, indicative of perihepatitis (10).

EC prognosis is favourable. When the disease is confined to the bladder, the treatment of choice is cholecystectomy, preferably performed laparoscopically. Treatment with corticosteroids can be effective when the bile ducts are affected, or when the condition is associated with eosinophilic gastroenteritis.

It is generally accepted that EC should not be considered a separate entity, because the clinical and laboratory manifestations are indistinguishable from those of common cholecystitis, and therefore it is considered more a histological finding than a pathology in itself. The importance of EC lies in the fact that it can be associated with other diseases, and therefore, when it is observed, possible associated syndromes should be investigated.

REFERENCES