ACUTE ACALCULOUS CHOLECYSTITIS ASSOCIATED WITH STREPTOCOCCAL ANGINA IN CHILDREN

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Abstract

The authors discuss the case of a 3 year-old boy hospitalized with high fever and abdominal pain in the right flank and hypocondrium, associated to a pharynx congestion. Laboratory data showed leukocytosis with neutrophilia and moderate inflammatory syndrome. Large-spectrum antibiotherapy caused a rapid disappearance of clinical symptoms, however the echography evidences a characteristic image of an acute acalculous cholecystitis. Revaluation of the hepatic function shows an important cytolysis associated with cholestasis and negative viral markers. Pharyngian exudate shows the presence of a hemolytic group A streptococcus. The association of hepatoprotectors and parenteral nutrition was followed by a rapid decrease of the biological parameters and normalization within twelve days. The clinical, imagistic, laboratory and evolutive elements led to a diagnosis of acute acalculous cholecystitis associated with cytolysis in association with streptococcal angina.

Keywords: acalculous cholecystitis, streptococcus, echography

INTRODUCTION

Acute acalculous cholecystitis, described for the first time by Duncan in 1844, is generally associated to some severe systemic illness, even if it may occur as a postoperative complication, in immunosuppressed patients, or accompanying viral or bacterial infections (in children, most frequent being the hepatic viruses). (1, 2) Complications like gangrene and perforation are more frequent than in acute calculous cholecystitis, while the mortality ratio is high (up to 50% in adults with critical condition). (2) The paper discusses the case of a three and a half year-old boy, in whom association of a hepatocytolysis syndrome with a cholestasis one and a suggestive ultrasonographic image impeded a precise establishment of the ‘primum movens’ of the pathogenic chain.

CASE PRESENTATION

A three and a half year-old little boy from the urban area is hospitalized with high fever, inapetence, diffuse abdominal pain. The heredocolateral and the personal physiological and pathological antecedents provided no significant information. The history of the actual disease was quite recent, the onset being registered 2 days prior to the admission in our service. The general clinical exam demonstrated a normal stature-weight condition (BW=15kg, H=98cm), a slightly influenced general condition, fever (38.9°C), pallor, congestive pharynx, hypertrophic tonsils (IIInd degree), bilateral, mobile, painless submandibular adenopathy, high sensitivity to palpation in the right flank and hypocondrium. The surgical examination rejected an acute abdomen, while laboratory data demonstrated leukocytosis with neutrophilia (WBC=19080/mmc, NEU=84.1%), moderate inflammatory syndrome (ESR=24 mm/h, Fg=543 mg/dl), the hepatic and renal function tests being within normal ranges (ALT=15 U/l, urea=30 mg/dl, serum glucose=92 mg/dl), normal urinalysis, negative coproparasitological examination. The ORL exam showed eritematos angina and no tympanic inflammation. Large-spectrum antibiotherapy (ceftazidime) and symptomatic treatment with antipyretics and spasmolytics were started. The abdominal echography performed 48 hours after admission showed a homogeneous structure of the liver, enlarged gallblader, with thickened walls (6 mm), normal spleen and kidneys. (fig.1)
Fig. 1. Enlarged gallbladder with thickened walls (6mm), B 3a6l

Fig. 2. Normal gallbladder in the same patient, 10 days after the onset of the symptoms

The echographic image imposes revaluation of the hepatic function and of the lipidic profile. Four days after the onset of the disease, a significant cytolysis was noticed (ALT= 391 UI/l, AST= 227 UI/l), as well as an incomplete colestasis syndrome (ALKP=1317 U/l, GGT= 410 U/l, total bilirubine=0.4 mg/dl, conjugated bilirubine=0.15 mg/dl, unconjugated bilirubine=0.25 mg/dl), no hepatoprive syndrome (prothrombine time=15 sec., serum albumines=5 g/dl) and no alteration of the blood fats (cholesterol=176 mg/dl, triglycerids=149 mg/dl) or serum glucose (89 mg/dl). Investigations were carried out for excluding an acute hepatitis (HAV IgM, EBV IgM, CMV IgM, serology for B and C viruses), a familial hypercholesterolemia (cholesterol, LDL-cholesterol) and an intestinal parasitosis with possible hepatic consequences (blood eosinophiles, coproparasitological exam), occurring within normal limits. Pharyngian exudate identified a âhemolytic group A streptococcus. Besides the initial treatment, perfusions with hypertone glucose and hepatoprotectors were added; the child maintains a good general condition, with no subjective complaints, while the biological parameters rapidly decreased in dynamics (ALT= 234 U/l, AST= 98 U/l, ALKP= 473 U/l, GGT=211 U/l after two days), being normalized within twelve days (ALT= 32 U/l, AST= 37 U/l, GGT= 61 U/l), only alkaline phosphatasis maintaining a slight increase (ALKP=235 U/l). The ultrasonographic modifications dissapeared ten days after the onset of the disease. (fig. 2) The final diagnosis was acute acalculous cholecystitis, as complication of a streptococcal angina, with favorable evolution under antibiotherapy; the short-term prognostic is good, but dificult to evaluate along the subsequent adult life.

**DISCUSSION**

Acute cholecistitis is a rare condition in pediatric age, its exact incidence being not known. Various studies have shown that affections of the gallbladder in children have a frequency of 1.3 cases/1000 adult patients, and that 4% of all cholecystectomies are performed in children. (2) The etiology of acute cholecystitis is dominated by vesicular lithiasis or pseudolithiasis, the causes of which are listed in table 1.

<table>
<thead>
<tr>
<th>Causes of vesicular lithiasis in children</th>
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<tr>
<td>• chronic hemolysis</td>
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<td>• abdominal surgery</td>
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<td>• trauma</td>
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<td>• sepsis</td>
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<td>• acute renal failure</td>
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<td>• prolonged parenteral nutrition</td>
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<td>• Chron disease, cystic fibrosis</td>
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<td>• prolonged fasting, hypocaloric diets</td>
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<td>• rapid weight loss</td>
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<td>• obesity</td>
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<td>• pregnancy in adolescents</td>
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<td>• drugs (ceftriaxon)</td>
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Acute acalculous cholecistitis (CAA), with a 5-10% frequency in adults and with incidental citations in pediatric literature, is also described.(2) The most frequent CAA causes are listed in table 2.(4-10)

The major risk for the occurrence of the disease is represented by the association of 4 of the following factors: shock, sepsis, hyperalimentation, prolonged fasting, intravenous
administration of narcotics and repeated transfusions; nevertheless, in some situations, the infectious factor was the only one responsible for the pathogenicity of the condition. Increase in mucous viscosity, caused by dehydration, increased cholesterol saturation of the bile and biliary stasis, the inflammation and the oedema which compromise the normal blood flow being some of the mechanisms involved. The most frequently occurring clinical signs are fever, abdominal pain (diffuse in small children or localized in the upper right quadrant in older ones), vomiting, sometimes jaundice. (10, 11) Leukocytosis with neutrophilia, a possible inflammatory syndrome and modifications of the hepatic function (increased transaminases, GGT, bilirubine, alkaline phosphatases) may suggest the diagnosis. The investigation of choice is ultrasonography, which may visualize distension of the gallbladder, wall thickening (>3.5 mm), the presence of sludge or pericolecystic liquid. (11) Generally speaking, the treatment is a conservative one, insertion of a naso-gastric tube, parenteral administration of liquids and antibiotherapy assuring a favorable result in most of the cases. Daily clinical and echographic monitoring is indispensable, in order to evidence possible complications and to recommend surgical indications. (11) The case here under analysis discusses mainly the problem of the differential diagnosis between an acute acalculous cholecystitis associated with cytolyis or of an acute viral hepatitis complicated with CAA. The onset with fever and abdominal pain may appear in both entities; the absence of jaundice is not unusual in type A viral hepatitis, if considering the high frequency of the anicteric forms. At the same time, leukocytosis with a left-deviated formula, the inflammatory syndrome occurring in the beginning of the condition, in the presence of an initially normal hepatic function and the suggestive echographic image represented essential arguments in the diagnosis of the biliary infectious process. Association with streptococcal infection was the only risk factor identified. Rapid clinical remission under antibiotherapy, in contrast with the sudden alteration of the hepatic function which, nevertheless, shows a favorable evolution within an extremely short time interval, associated to a negative serology for the hepatic viruses, constituted the main elements differentiating it from an acute hepatitis.

**CONCLUSIONS**

Even if it is a rarely occurring diagnosis in pediatrics, acute acalculous cholecystitis should be evoked in children with fever and abdominal pain localized in the upper right quadrant, in the presence or even in the absence of major risk factors, if the echographic image is a suggestive one. Daily monitorization of such patients through ultrasonography is indispensable for a correct choice between the conservative treatment and the surgical one.

**References**

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