Acute-on-chronic acalculous cholecystitis in a Black child
A case report and review of the literature
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Summary
A case of acute-on-chronic acalculous cholecystitis in a 7-year-old Black child is reported. The incidence, causation, differential diagnosis and management of acalculous cholecystitis in childhood and the confusion caused by the terminology are briefly discussed.

Case report
A 7-year-old Black boy was admitted to hospital after the sudden onset of right hypochondrial pain and bilious vomiting. Two months before admission he had spent 2 days in hospital because of a similar episode, which had resolved spontaneously.

Examination revealed a toxic, pyrexial child with a pulse rate of 110/min. No jaundice was noted, the abdomen was not distended, and bowel sounds were normal. A tense and very tender well-defined mass was palpable in the right hypochondrium, descending with the liver on inspiration. There was muscular guarding in the right hypochondrium. The rest of the abdomen was soft but mildly tender to palpation. Rectal examination was negative.

Plain radiographs of the abdomen showed few air/fluid levels, and there was a suggestion of a mass in the right hypochondrium. The haemoglobin value was 13.2 g/dl and the white cell count 9.6 x 10^9/l. Amylase and urobilinogen levels and liver function test results were normal.

A diagnosis of acute torsion of the gallbladder was made. At laparotomy the gallbladder was found to be enlarged, inflamed, and surrounded by fibrous adhesions. No stones were palpable in the gallbladder or the common bile duct, which appeared of normal calibre. The liver looked normal. Ascaris worms were palpable in the terminal ileum. An uneventful cholecystectomy was performed. The pathologist reported a thickened and inflamed gallbladder wall; histological examination revealed ulceration of the mucosa with an acute-on-chronic inflammatory infiltrate.

Discussion
Confusion in the discussion of childhood cholecystitis stems from the term 'acute hydrops of the gallbladder'. Hydrops is defined as an acute obstructive distension of the gallbladder without the presence of stones in the gallbladder or bile ducts, without microscopic evidence of inflammation of the gallbladder wall and without infection in the bile. Ternberg postulated that acute hydrops may represent a first stage in the pathogenesis of acute acalculous cholecystitis — inflammation and infection fol-
lowing the obstruction. Chamberlain and Hight\(^1\) found only 25 patients described in the literature to fulfil the definition of acute hydrops, and added 4 of their own. The patients presented with right hypochondrial pain, a mass, and vomiting. A correct diagnosis was made pre-operatively in only 3 cases, the rest being diagnosed as having an appendix abscess or an intussusception.

Acalculous cholecystitis in childhood may be associated with congenital abnormalities of the biliary tract, severe systemic disease or injury.\(^2\) In one series 60% of the patients with acalculous cholecystitis or acute hydrops had a history of preceding illness or injury. Three per cent had congenital biliary tract disease. Bile culture in patients with no associated disease is sterile, but in those with preceding illness the bile commonly contains organisms suggestive of haematogenous spread to the gallbladder from a site of primary infection. The associated conditions were upper respiratory infections, scarlet fever, prematurity with sepsis, gastro-enteritis and burns.\(^3\)

Biliary stasis in the gallbladder may result from external compression of the cystic duct by hyperplastic lymph nodes, or stasis following fever, dehydration, prolonged fasting and ileus. Forshal and Rickham\(^4\) suggested that congenital abnormalities of the cystic duct may be an important factor in some cases; the abnormalities become apparent only if the duct is carefully dissected and sections are examined. In 3 of 6 children with gallbladder disease in their series the operative specimens were examined in detail and anatomical abnormalities were found. Glenn\(^5\) postulated that spasm of the sphincter of Oddi and regurgitation of pancreatic juice into the gallbladder caused inflammatory reaction.

Acute acalculous cholecystitis may follow closed abdominal trauma, as is demonstrated by a five-fold increase in the incidence of the disease in civilians during the Nigerian civil war.\(^6\)

Numerous infectious agents have been described as causes of cholecystitis, usually part of systemic infection. Enteric bacteria, streptococci, staphylococci, diptheroid, \textit{Pseudomonas}, leptospires, \textit{salmonella}\(^7\) and schistosomes\(^8\) have all been implicated. Biliary symptoms in the Black child are often attributed to \textit{Ascaris lumbricoides}, which in some regions infests 95% of the population, particularly preschool children.\(^9\) The worms may migrate into the common bile duct and cause transient obstructive jaundice.\(^10\) We wish to emphasize that only massive infestation in the small bowel can lead to colonization of the duodenum resulting in migration into the common bile duct, and that cholecystitis without jaundice and palpable parasites in the common bile duct cannot be attributed to \textit{Ascaris} infestation.

Despite the frequently incorrect pre-operative diagnosis, the indications for laparotomy in a child with acute cholecystitis are always present.\(^11\) More difficult diagnostic dilemmas concern children with chronic acalculous cholecystitis. Forshal and Rickham\(^12\) suggest that mild attacks escape recognition (as in our patient on his first admission) and that cases exist among the enormous number of children with undiagnosed abdominal pain seen at paediatric and surgical clinics. Adams and Foxley\(^13\) described an accurate technique for the diagnosis of acalculous cholecystitis. Twenty-six patients in whom oral cholecystography was negative but symptoms of biliary tract disease persisted were studied with 24-hour delayed films of the gallbladder. All of them had persistent opacification and in all a diseased acalculous gallbladder was found at operation. In 22 control subjects visualization was normal at 12 hours, but there was no opacification in the delayed films. The delay in expulsion of the dye suggests an abnormality in contractility, which mirrors disease in the gallbladder wall.

There is some doubt as to the correct surgical procedure for acute acalculous cholecystitis in childhood. Ternberg and Keating\(^1\) state that tube cholecystostomy is probably adequate treatment. Forshal and Rickham\(^4\) consider this a mistake, since even a macroscopically normal gallbladder can be associated with an abnormal cystic duct.

Barnes\(^14\) described a 4-year-old child who underwent tube cholecystostomy for acute acalculous cholecystitis. The postoperative tube cholangiogram was normal, but 16 months later the child returned with an acute abdomen. Laparotomy revealed acute cholecystitis which was treated with cholecystectomy. Glenn\(^5\) believes that after cholecystostomy calcareous or amorphous material can form in the gallbladder, predisposing to gallstone formation.

Castle \textit{et al.}\(^15\) have claimed that there is an increase in the risk of carcinoma of the gallbladder after cholecystectomy. The interval between cholecystostomy and the diagnosis of carcinoma ranged from 3 months to 40 years.

We believe that cholecystectomy is the operation of choice for acute cholecystitis in childhood. In spite of its rarity, acute and chronic cholecystitis must be considered as possible diagnoses in the child with acute, recurrent or chronic abdominal pain.

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\textbf{REFERENCES}