ACALCULOUS CHOLECYSTITIS IN INFANCY AND CHILDHOOD*

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SUMMARY

A case of acalculous cholecystitis in a 17-month-old baby is discussed.

Special emphasis is laid on the importance of the diagnosis, and the danger of overlooking this condition in infancy and childhood. It is pointed out that in all cases with a right-sided pain and peritonitis the possibility of cholecystitis should be borne in mind.

A definite line of treatment is established after reviewing the cases in the literature.

Acute cholecystitis is a rare phenomenon in children under the age of 2 years. The first paediatric case of cholecystitis and cholelithiasis was reported by Gibson[1] in 1922. In 1767 Lieutaud, at autopsy made the same finding in a 25-day-old infant. Howse was probably the first to treat a patient successfully when he performed a cholecystectomy on a 5-year-old child with acute noncalculous cholecystitis in 1922.[2]

Lucas and Walt[1] reported a case of acute noncalculous cholecystitis in a 2-year-old boy. This case shows a similarity to ours in symptoms, signs and presentations. In 32 cases, they discovered from the literature, only 8 cases were under the age of 3 years. Brenner,[3] in his collective review reported 570 patients between the ages of 10 and 15 years.

We would like to add to the literature another case of acalculous cholecystitis in a 17-month-old child.

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Case Report

A male Bantu baby aged 17 months was referred to our outpatient department with the diagnosis of acute appendicitis. The baby presented with one episode of vomiting 2 days previously, crying continuously and refused to take feeds. There was no history of gastroenteritis, parasitic infestation or any other systemic disease. The child had been normal since birth and had never had anaemia or jaundice. On examination, the baby was found to be well nourished but restless and had a pulse of 140/min and a temperature of 102°C. No jaundice, anaemia or oedema was present. Abdominal examination revealed marked muscle rigidity and rebound tenderness, spreading from the right iliac fossa to the right hypochondrium. Rectally he was tender on the right side, but no mass was present. The stool was normal.

Laboratory investigations were as follows: haemoglobin 12·5 g/100 ml, white blood-cell count 18 000/mm^3 and scanty pus cells on urine analysis.

A tentative diagnosis of acute appendicitis was made but because of the few pus cells in the urine it was decided to keep the baby under observation. Intravenous infusion, antibiotics and sedation were started and the baby had nothing by mouth.

A half-hourly pulse chart showed a gradual rise in pulse rate and on examination we found a marked increase in abdominal symptoms after 6 hours. At this stage the point of maximum tenderness was the right hypochondrium, and the possibility of a retrocaecal peri-appendicular abscess was considered.

It was decided to do a laparotomy immediately and to make a right paramedian incision because of the uncertainty as to the cause of the peritonitis.

The baby had a normal appendix and on examining the abdomen a large, tense, acutely inflamed gall bladder projected from the liver. The hepatic, cystic and common ducts were normal. There were no tumours or parasites, and the duodenum seemed normal.

A cholecystectomy was performed, a corrugated drain inserted and the abdomen closed in layers. The gall bladder and its contents were sent directly to the laboratory for histology, microscopy, culture and sensitivity tests.

The postoperative period was uneventful and the baby was discharged after 20 days.

Postoperative urinary culture, stools and special investigations gave no further clues as to the aetiology of the cholecystitis. No calculi were seen in the gall bladder or cystic duct.

Microscopy, culture and sensitivity tests of the contents of the gall bladder showed a moderate number of Gram-positive bacilli, no pathogenic organisms were grown and the Ziehl-Nielsen stain was negative. The histology report was as follows: 'Gall bladder markedly dilated (1 × 3·5 × 1 cm) with a thin wall. The appearance is that of mucocele of the gall bladder. Sections of this gall bladder showed marked oedema and chronic inflammation of the wall. The mucosa showed areas of proliferation of columnar cells'.

The child was seen again in January 1971 with no postoperative complications and was doing well.

DISCUSSION

The first comprehensive presentation on the subject was made by Potter in 1938.[4] Hopkins *et al.*[5] have made the point that childhood cholecystitis is rarely diagnosed preoperatively and this was true in our case.

It has repeatedly been stated that in contradistinction to adults, in whom there is a female preponderance of 4:1 of biliary tract disease, in children it occurs more often in males, with a ratio of 3:2.

Morales *et al.*[6] found 38 cases with cholecystitis and cholelithiasis, with maximum incidence in the 9 - 13 age group. They noted that females predominated over males with a ratio of 3:1. Brenner[7] reported a female to male ratio of 5:2 in 244 cases.

Aetiology

Gastro-intestinal infection with secondary spread to the gall bladder is given as one of the aetiological factors. The most frequently cultured organisms are *E. coli*, Salmonella and cholera. Many cases have negative cultures and viral infection has been suggested as a possible factor.

Parasitic infestations such as *Giardia lamblia* and *Ascaris lumbricoides*, causing obstruction of the common duct, were found in a number of cases.

It has been suggested that cystic duct obstruction secondary to fibrous bands or enlarged glands is a cause, but this factor has been difficult to prove.

Congenital anomalies of the cystic duct and common duct have been demonstrated. Forshall and Rickham[8] had 3 cases in which they could prove cystic duct abnormality with careful dissection. It was not stated whether they found atresia or stenosis in these cases. The possibility of a stenotic cystic duct was the only aetiological cause for the mucocele and colecystitis we found in our case. According to the pathology report, this was not so.

Haemolytic streptococcal septicaemia, glomerulonephritis, erysipelas, metabolic disorders, haemolitic anaemias and hereditary factors have been indicated as predisposing to gall-bladder disease.

The possibility that an acute systemic infection and concomitant dehydration might lead to bile stasis and concentration of organisms in the gall bladder with resultant inflammation and cholecystitis has been mentioned by various authors.

Diagnosis

The tendency to overlook the diagnosis of acute cholecystitis in infancy and childhood because of its rarity, must be guarded against. As in our case, the most frequent diagnosis made is that of acute appendicitis. Herein lies the danger that when at appendicectomy a normal appendix is found, the real condition is completely overlooked, with grave consequences to the patient.

The clinical picture of cholecystitis is characterized by nausea, vomiting and anorexia of 2 - 6 days duration. There is fatty-food intolerance in 14·3% of cases.[7] Jaundice was found in 24·2% of children but no definite classification as to the presence of calculi, in comparison with acalculous colecystitis, could be found in the literature.

The variation and location of the pain often make the diagnosis difficult, but in all cases a right-sided abdominal tenderness should make one consider a cholecystitis.

In retrospect, from experience with our case, it might be of importance to note that tenderness was maximal in the right hypochondrium although the pain started in the right iliac fossa. A definite abdominal mass could be felt in 10·7% of cases mentioned by Richard and Brenner.[7]

Special investigations include a while cell count of haemoglobin, erythrocyte sedimentation rate and a cholecystogram.

Treatment

Preoperative intravenous infusion of fluids and antibiotics followed by a cholecystectomy is the treatment of choice. In children the mortality rate is low and the postoperative morbidity far less than in cases where a cholecystotomy was done.

Only in toxic moribund cases should a cholicystostomy be done under local anaesthesia. This should be followed after 6 weeks with an elective cholecystectomy because of danger of recurrent disease.

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