Case Reports

ACUTE CHOLECYSTITIS ASSOCIATED WITH ECHINOCOCCAL CYST OBSTRUCTION OF THE COMMON BILE DUCT

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ECHINOCOCCAL infestation is common in Australasia, in South America and in Iceland. A Hydatid Registry is kept by the Royal Australasian College of Surgeons. It is also common in the Near East—in Greece, Southern Italy, the Balkans, Turkey, and the Levant. In the United States of America and in Britain the disease is very rare. Magath (1940) says that almost all cases occur in persons immigrant to, but not born in, North America; in these regions, hydatid disease is uncommon and the occasional appearance of a case may lead to confusion in diagnosis.

The life-cycle of the *Echinococcus granulosus* was discovered by Knabbe in 1866; this cestode usually has a cycle of development in two hosts. The adult *Tænia echinococcus* is 6 mm. in length and consists of four segments which can be found by the hundred in the upper villi of the intestine of infected dogs. About 500 ova are found in each ripe proglottis as it falls off. These are scattered by wind and water and can be ingested by the intermediate host, which may be sheep, cattle, pig, camel or man.

Barnett (1929) points out that the infested dog's hair, paws, and muzzle are found heavily contaminated, and handling them is the method of transference from dog to man. The definitive host, dog, becomes infested by feeding on the offal of infested sheep, cattle, or other intermediate hosts.

When man swallows the ova, the capsules are digested and the three-spined ova make their way through the stomach or duodenum into a venous blood-vessel, and are carried up in the portal vein to reach the liver, where they may lodge in the capillaries. About 70% of cysts are said to be found in the liver; some ova filter through and reach the pulmonary capillaries, where 10% of cysts are located. The remaining 20% of cysts occur elsewhere.

The liver is, thus, the most common location for hydatid cyst disease. The right lobe is by far the commonest site; it is affected four times more frequently than the left and in this lobe the cyst is most often in the postero-superior portion. This

is because of the greater width and more direct route of the right hepatic vein.

Hydatid cysts of the liver are nearly always primary, but occasionally may be secondary, such as occurs following the rupture of a cyst into the abdominal cavity during operation. More than one cyst is present in 25% of cases. Most authorities, e.g. Dévé (1918, 1925), Mills (1926, 1927), agree that infestation with hydatids occurs in infancy and that symptoms only begin to appear from 20 to 40 years later. Males are more often affected than females, perhaps because as boys they are more often brought in contact with dogs.

Dew (1930), believes that many cysts are almost as old as the patient. Small cysts are symptomless and even large cysts are commonly silent and complications develop. When the patient reaches 30 years of age or so, however, there is often dull pain or fullness in the right upper abdomen made worse by exercise or by the taking of a meal. There is slight jaundice in 80%. There may be urticaria or other allergic phenomena, including eosinophilia, and dyspepsia, similar to that associated with cholecystitis. Seventy per cent. of cysts present on the lower surface of the liver, and are palpable, sometimes manifesting a 'hydatid thrill', or even egg-shell crackling if a calcified cyst is sufficiently accessible. The remainder enlarge upwards, with an elevation of the diaphragm which is radiologically demonstrable, and sometimes a pleural effusion. A calcified cyst may throw a mottled or uniformly dense shadow.

The simplest diagnostic test is the Casoni reaction—intradermal injection of fresh fluid from sheep hydatid. This depends upon the allergic response of the host to the fluid of the parasite; it is negative in the 5% of cases in which there is no passage of fluid through the cyst wall. In the Indians of Northern Canada, it is negative unless fresh moose hydatid is used. There the moose is the usual host. It has sometimes been positive in tuberculous patients and in normal subjects. More elaborate is the Ghedini-Weinberg complement-fixation reaction, which has an accuracy of nearly 100%. Eosinophilia over 5% is a common finding.

The chief complication of hydatid cyst is rupture, often after injury, with dissemination of daughter

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cysts in the peritoneal cavity, the pleural cavity, the lung or bronchus with formation of a broncho-biliary fistula, or into the stomach, intestine, renal pelvis or bile passages. Such rupture may be silent, or it may be accompanied by intra-peritoneal hæmorrhage, with pain, vomiting and anaphylactic phenomena. When dissemination is into the peritoneal cavity, the rent in the liver may close, with enlargement of the peritoneal daughter cysts years later as multiple painless swellings and sometimes great enlargement of the abdomen. If the original signs of rupture have been occult, or if they are forgotten, the diagnosis is then difficult. Meanwhile the infection in the healed liver has developed again as a multilocular cyst—it is one of Dew's axioms that the presence of a multilocular cyst of the liver may be presumed in cases of peritoneal echinococcosis.

Dew (1948) states that intrabiliary rupture is one of the commoner of the major complications of hydatid disease of the liver. The rupture takes place most frequently into one of the large intra-hepatic ducts and is likely to occur even with a small centrally placed cyst. The debris frequently passes into the duodenum and pieces of laminated membrane may be discovered in the washed fæces. Obstructive jaundice and cholangitis are common. Large hydatids sometimes evacuate by this route repeatedly over a long period of years, successive descending daughter cysts giving intermittent painful jaundice rather like that of stone in the common bile duct. Dew also finds that the commonest age for rupture is between 35 and 45, that occasionally some injury precipitates the rupture, and that the ensuing biliary colic is less severe than that caused by gallstones. Diagnosis is suggested by considering the lower age group, the absence of previous attacks, the liver enlargement, and the intermittent non-progressive nature of the jaundice. It is confirmed by finding hydatid debris in the fæces, by radiology, and by positive hydatid skin tests. Frequently infection of the cyst occurs after rupture, and Dew recommends that treatment should be adequate drainage of the ensuing hepatic abscess.

Case Report

History. N.A., aged 13 years, admitted to the Baptist Hospital, Gaza, on December 15, 1961. She complained of sudden, severe abdominal pain for 48 hours prior to admission. The pain, to start with, was colicky in nature and generalized; later, it localized to the right upper quadrant of the abdomen and became dull-aching in character. The patient vomited three times on the day of admission and had diarrhoea. No previous history of illness.

On examination. Temperature 37.4° C., pulse 75/min., B.P. 100/75 mm. Hg. The patient was acutely distressed and was slightly icteric. Heart and chest, normal; abdomen, tenderness in the right upper quadrant. A large, very tender mass was felt in the region of the gallbladder, extending to about 4 cm. below the right costal margin. A tentative diagnosis of acute cholecystitis with hydrops of the gallbladder was made.

Laboratory findings. Hb 85%. WBC, total 11,050/cu. mm—polys. 72%, lymphs. 27%, eosinos. 1%. Stools: Negative for ova and amœba.

FIG. 1.—The two daughter cysts removed from the common bile duct with the gallbladder removed in the second operation.

Procedure. The patient was given conservative treatment; Fowler's position, sedation, antibiotics, iv. fluids and vitamins (B complex and K). After 24 hours she seemed to have improved with decrease in the size of the mass. However, and despite the above treatment, subsequently, the swelling enlarged and the tenderness and rigidity increased. Exploratory laparotomy was performed on December 19, 1961.

Operation. A small amount of bile was found free in the peritoneal cavity. The gallbladder was markedly inflamed, œdematous, with areas of gangrene in its wall. Three hydatid cysts were also found in the slightly enlarged liver; two large ones on the inferior surface of the right lobe and a third smaller one on the diaphragmatic surface of the same lobe. The cysts were treated in the usual way: i.e. aspiration, formalin injection, evacuation and dusting with penicillin. The cavities were closed around three drainage tubes. No attempt at removal of the gallbladder or exploration of the bile ducts was made, due to the marked inflammatory œdema in the field. Cholecystostomy was done using a Foley's catheter. A Penrose drain was inserted in the subdiaphragmatic region. The three drainage tubes were removed on the 19th post-operative day and the cholecystostomy tube on the 21st day. It was noticed that the tubes started to drain bile in the immediate post-operative period and continued to do so after removal of the tubes. Even the site of the cholecystostomy tube started to drain bile after temporary and apparent sealing off. A cholangiogram was taken—by injection of lipiodol through one of the fistulae—on February 8, 1962, i.e. after 50 days from surgery. It showed dilatation of the intrahepatic bile

ducts with dilatation and obstruction of the common bile duct.

The second operation was undertaken on February 24, 1962, i.e. about two months after the first, under general intubation anaesthesia. The old scar was excised. Adhesions of the colon and great omentum to the gall-bladder were dissected. The common bile duct, which was found to be dilated (about 1.5 cm. in diameter), was explored through an incision below the junction of the cystic duct; two dark-greenish (bile-stained) daughter cysts were removed from the common bile duct at the entrance of the cystic duct. The common bile duct was probed and dilated using Bakes dilators and a T-tube choledochostomy instituted. Cholecystectomy was also done; the gallbladder was removed with some difficulty due to dense adhesions from previous inflammation and surgery (Fig. 1).

A post-operative 'T' cholangiogram done on March 9, 1962, i.e. 12 days after the second operation, showed a free passage of the dye into the duodenum with no apparent obstruction of the common bile duct. The T-tube was removed on the same day; this was followed by no drainage of bile at all.

All wounds healed well and the patient left the hospital on March 12, 1962, in a very satisfactory condition.

Summary

A case of multiple hydatid cysts of the liver with rupture into the bile passages and obstruction of the common bile duct by daughter cysts has been reported. Whether acute cholecystitis was a coincident disease in this case or a clinical presentation of hydatid disease due to obstruction of the cystic duct by hydatid cyst is not definitely known.

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STAPHYLOCOCCAL MUSCLE INVASION AND ANURIA

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MOST of the clinical features of staphylococcal septicæmia are well established and the more recent literature is chiefly concerned with the problem of treating antibiotic-resistant strains. I therefore felt it would be refreshing to return to the bedside and report the following unusual syndrome.

Case Report

The patient, a man of 77, was admitted to Ashington Hospital on October 2, 1962, with a one-week history of increasingly severe muscle pains. The muscles of the arms, neck, thighs, anterior chest and abdomen were chiefly affected, and the slightest movement precipitated intense pain, so that he was forced to lie motionless, and was quite unable to reach out to his locker for a drink.

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He had felt unwell for three months and had lost half-a-stone in weight. However, there was no history of fever, nor had he suffered from any recent skin infections. For the past four months he had experienced difficulty passing urine for a few days at a time and over the past year he had suffered from occasional dysuria due to the passage of gravel in his urine. Furthermore, we discovered that he had been oliguric for one week.

Over the previous ten years he had suffered from osteo-arthritis of both knees, which had been treated intermittently with butazolidine. There was no family history of gout.

On examination he was clearly extremely ill. His tongue was very dry and his breath had the characteristic uræmic factor. His temperature was subnormal, 96.8° F., pulse 88/min., and blood pressure 120/70. Rales at left base; no neurological signs.

Both forearms and thighs were remarkably swollen and the underlying muscles were extremely tender. The skin in these areas was reddened, warm, and there