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 gallbladder 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

was some pitting oedema. There was no associated acute arthritis. The cervical, right pectoral and abdominal muscles were also very tender, and there was suprapubic tenderness. The prostate was not enlarged, and the passage of a Gibbon's catheter failed to drain any urine.

Investigations. X-ray abdomen: No opaque calculi visible. Chest X-rays: Some left ventricular enlargement, lung fields clear. X-rays of his hands, feet and knees did not show any changes to suggest gout. There were osteo-arthritic changes in both knees. Blood urea: 312 mg./100 ml.; K. 5.4, Na. 126, Cl. 95 mEq./l. Uric acid 9 mg./100 ml., E.S.R. 90 mm./hr. (Westergren), Hb. 98%, WBC 20,100, polys. 91%, lymphs. 4%, monos. 5%. ECG: Moderate LV enlargement, LBBB.

Progress. Throughout his admission the urinary output never exceeded 200 ml./24 hr. and urine examination showed an excess of protein, a large number of pus cells and a few red cells. Urine culture revealed a heavy growth of *Staphylococcus pyogenes* sensitive to most antibiotics.

In spite of intravenous fluids, penicillin and hydrocortisone i.v. the patient deteriorated rapidly, and on October 4, 1962, his blood urea had increased to 368 mg./100 ml. He became stuporous and died on October 6, 1962.

Necropsy findings. The muscles of the chest wall were found to contain collections of exudate between the muscle fibres. This was also present on the right side of the neck. The arms were very oedematous and the underlying muscles also contained this gelatinous exudate. The right elbow was opened but there was no pus in this joint. The trachea and main bronchi contained watery fluid and the lungs showed oedema of the lower lobes. The heart showed left ventricular hypertrophy and the coronary arteries were markedly atheromatous. There were several old scars posteriorly in the wall of the left ventricle. The heart valves were normal and there was no evidence of endocarditis. Examination of the abdomen revealed some enlargement of the spleen and liver. The latter showed early nutmeg changes. The kidneys (left 135 g., right 185 g.) showed patchy atrophy of their cortices. Crystalline deposit was visible in the pyramids of the collecting tubules. There was no evidence of acute inflammation of the bladder, ureters or renal pelves. The psoas muscles also contained interfascicular collections of exudate. Examination of the brain and meninges was normal.

Histological examination confirmed the presence of linear collections of pus between the muscle fibres. There was marked oedema, but little evidence of muscle degeneration, except for toxic necrosis in the immediately adjacent areas. Clearly the process was essentially an acute pyogenic one.

The kidneys showed a diffuse interstitial oedema and cellular infiltration composed of mixed polymorphs and lymphocytes. Though variable in concentration the polymorphs nowhere accumulated to the extent of producing abscesses. The glomeruli was mainly normal

with occasional old hyalinization. The convoluted tubules showed marked toxic degeneration. The medullary and collecting tubules contained occasional amorphous casts but no significant cellular aggregations. The picture was essentially that of an acute interstitial nephritis.

Culture of the exudate from the muscles produced a heavy growth of *Staphylococcus pyogenes* of phage type 52A/79/+ which was identical to the organisms found in his urine. This supported the diagnosis of staphylococcal septicæmia which had caused acute interstitial nephritis and renal failure.

Discussion

Muscle involvement in staphylococcal septicæmia is noted by a number of authors including Ryle (1949). There is an interesting paper on the subject of pyæmia by Gamgee, which was read before the Medical Society of University College on November 25th, 1852. He stressed the importance of muscle involvement and emphasized that the pus was not collected in circumscribed cavities but was 'disposed in longitudinal strata, evidently in the interfascicular cellular tissue'. His description made over a century ago corresponds well with the macroscopic findings in our present case.

In a large series of 122 cases Skinner and Keefer (1941) record only three cases with muscle invasion, but these patients also had infection elsewhere. The overall mortality in this series was high, 82%. However, as Ryle noted, some patients with staphylococcal septicæmia survived even in the prepenicillin era. These patients were usually young and the infection eventually became focalized into an abscess, e.g. perinephric, which was amenable to surgical drainage.

Skinner and Keefer (1941) also state that the most constant clinical feature was the presence of high remittent fever. In our case the temperature remained mainly in the region of 97°F. Absence of a febrile response may have been due to his advanced age. In addition, infectious fever may be masked by uræmic hypothermia (Shreiner and Maher 1961).

To conclude, widespread staphylococcal muscle invasion and anuria, without fever, metastatic abscesses elsewhere or any obvious source of infection would certainly seem most unusual. Culture of muscle biopsy material would probably have established an early diagnosis. However, the patient's condition was unfortunately too advanced to benefit from treatment.

I am particularly grateful to Dr. T. Manners for his full post-mortem report.

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