Intestinal intubation showed the duodenal contents contained normal amounts of pancreatic enzymes, but there was a complete absence of bile from the small intestine as far as the jejunum. It appeared likely, therefore, that the anastomosis between the cyst and the small intestine was not sufficiently proximal to ensure adequate mixing of the pancreatic enzymes and their biliary activators during the processes of digestion. However, before further surgery could be carried out the patient requested discharge, and since that time has refused review. During her stay in hospital the patient showed no significant weight loss, haematological change, or any other clinical manifestations of a general malabsorption syndrome.

Discussion

This patient had many of the clinical features usually associated with this anomaly. She was a young woman with recurrent attacks of mild jaundice and a tumour in the right hypochondrium. The most severe attack of jaundice was associated with pregnancy and resolved following delivery. Laboratory investigations were helpful diagnostically only in so far as they confirmed the presence of an obstructive biliary lesion, and contrast radiography was of value in excluding a tumour of intestinal origin. The treatment of congenital cystic dilatation of the common bile duct is surgical, and there is no doubt that the most satisfactory procedure is choledochojejunostomy using a Roux-en-Y loop. Choledochoduodenostomy has often given satisfactory results initially, but ascending cholangitis and hepatitis have been a frequent sequel to this type of operation.

Of particular interest in the present case was the unexplained steatorrhoea observed before operation, and there is no report in the literature of this abnormality in association with choledochus cyst. A possible explanation is that distension of the upper gastro-intestinal tract with food caused intermittent but complete occlusion of the biliary tract by displacement of the cyst, so that the biliary activators of the pancreatic enzymes were absent at the most crucial phase of digestion in the upper intestine. At other times during the post-absorptive phase, biliary flow was relatively free. A further possibility is that the two conditions are entirely unrelated, the steatorrhoea being due to an idiopathic malabsorption syndrome. Although this latter possibility cannot be entirely ruled out, it is unlikely in view of the normal intestinal mucosal pattern seen on the barium-meal examination and the absence of haematological abnormality.

In spite of the relief of the obstructive element in the syndrome, the steatorrhoea continued unchanged and after operation the patient was excreting 36% of her total ingested fat. The pancreatic secretions were known to be normal in quantity and activity, and it is possible that this malabsorption was due to defective mixing of the food with the pancreatic enzymes and bile.

Summary

A case of choledochus cyst is described. Metabolic studies, with particular reference to fat absorption, were made before and after operation. The pre-operative steatorrhoea persisted after an operation which relieved the biliary obstruction.

We thank Professor W. Melville Arnott and Mr. B. N. Brooke, under whose care this patient was admitted, for their help and encouragement in the preparation of this paper. We also thank Dr. C. F. J. Cropper, of Worcester Royal Infirmary, who referred this case initially, and Mr. Garfield Thomas, who was responsible for the biochemical investigations.

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Medical Memoranda

Bilateral Rupture of Rectus Femoris Tendons in Chronic Nephritis

The following case is rare enough to warrant recording, particularly in view of the young age of the patient and the associated general disease. Rupture of both quadriceps tendons has been recorded before, but these cases have been in elderly people (Lloyd, 1949).

CASE REPORT

A man of 34 was admitted to hospital on March 17, 1956, having fallen on to his knees while going down some steps. On attempting to stand his knees gave way. He stated that for some months he had been feeling rather tired and that this came on after a bout of influenza. He had, however, been able to carry on his work.

On examination he was rather pale, with a blood pressure of 160/80 mm. Hg. He was almost completely bald, and this made him appear much older than his years. Both knees were swollen and clinically resembled a haemarthrosis. He was very tender over the upper margin of both patellae, but there was no palpable defect in the extensor apparatus. He was just able to maintain full extension, but this caused him considerable pain. He had sustained an injury to the left ankle which proved to be a first-degree external rotation fracture of the fibula. X-ray examination of the knees showed that both the upper margins of the patellae were tilted forward and there was a calcified plaque in the soft tissues immediately above. It was thought that there was a tear of the rectus femoris tendon alone without involvement of the lateral expansions, and exploration of the knees was advised.

Operation.—The lesion was found to be almost identical on both sides, that on the right being slightly more extensive. A 2-in. (5-cm.) crescent of rectus femoris tendon had been torn off the top of the patella along with a sliver of bone. There was no evidence of calcification in the tendon itself. The lateral expansions were intact, the tear being much less extensive than in the usual quadriceps rupture. The patellae were displaced forward at the site of injury and the rectus femoris tendons tucked in behind. A repair was easily accomplished with silk mattress sutures.

The patient was rather lethargic after operation, but his condition did not cause any concern until the third day, when it became obvious that there had been a sudden serious deterioration in his condition. Investigation showed: blood urea, 312 mg. per 100 ml.; CO₂-combining power, 11.2 mEq/l.; Hb, 50%; serum phosphate, 12.2 mg. per 100 ml. He was passing normal quantities of urine, the specific gravity of which was 1010, with an albumin estimation of 70 mg. per 100 ml. On going into his previous history it was discovered that he had been rejected by the Army during the war because of albuminuria. He was regarded as a case of chronic nephritis, probably in the terminal phase. He shortly developed a pericarditis and died in uraemic convulsions on March 26.

Post-mortem Examination.—Both kidneys were found to be small and sclerotic. Sections of the rectus femoris tendons at the site of rupture showed no evidence of calcification or of degenerative changes.

COMMENT

It is felt that simultaneous rupture of both rectus femoris tendons in so young a patient must be an almost unique occurrence. The severe debility from his renal failure may have been a contributory cause, although there was no evidence of degenerative changes in the tendons. It is interesting to note the very limited extent of the lesion, distinguishing it from the more common form of quadriceps rupture. In the latter injury it is most unusual for the

patient to be able to hold the knee fully extended against gravity, a sign which was absent in this case. The unusual tilt of the patellae therefore proved a useful guide in making the diagnosis.

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Fatal Reaction Following the Intravenous Injection of Congo Red

While the Congo-red test for secondary amyloidosis is not now extensively used, it is advised as a helpful diagnostic procedure in primary systemic amyloidosis (Rukavina *et al.*, 1956) and for the detection of amyloidosis in association with rheumatoid arthritis (Fearnley and Lackner, 1955). In view of this, it is considered of importance to record a death following the intravenous injection of Congo red.

CASE REPORT

A 47-year-old man underwent a right pneumonectomy for carcinoma in 1943. This was followed by an empyema which did not finally heal till 1950, during which time several plastic operations were required. Since 1950 he had experienced increasing shortness of breath and progressive reduction in his exercise tolerance. Three weeks before admission to hospital he developed marked swelling of his feet.

On admission he was cyanosed and dyspnoeic at rest. There was no increase of jugular venous pressure. Signs of bronchospasm were present over the left chest. Obvious oedema of the feet was noted. A heavy albuminuria was found without casts or red blood cells. The blood urea was 23 mg. per 100 ml.; plasma protein, 5.3 g. per 100 ml. (albumin 3.4 g.; globulin 1.9 g.); blood cholesterol, 148 mg. per 100 ml. Secondary amyloidosis following the empyema was suspected, and it was decided to perform a Congo red test.

Ten millilitres of a 1% solution of Congo red in normal saline was prepared at the bedside from a sealed ampoule of the powder. This solution was injected intravenously immediately afterwards. About two minutes later the patient complained of severe difficulty in breathing. Respiration ceased within seconds, and unconsciousness with deep cyanosis rapidly ensued. The radial pulse became impalpable soon after the loss of consciousness. Approximately five minutes after the loss of consciousness ventilation was started through an endotracheal tube and the chest was opened for cardiac massage. After one hour spontaneous respiration and heart beat were restored, but after a further three-quarters of an hour he collapsed and died without fully regaining consciousness.

At necropsy the empyema was soundly healed. There were approximately 30 oz. (850 ml.) of blood in the left chest, clotted blood in the pericardium, and bruising of the myocardium. No evidence of amyloidosis was found.

COMMENT

Reactions to intravenous Congo red in the form of "chilly sensations" have been described by Keith *et al.* (1915). Strasser (1927) mentioned the occurrence of chills and fevers, and Rossak (1933) noted abdominal pain, flushes, palpitation, and malaise. More recently Discombe (1949) reported severe reactions following its injection—for example, nausea, sweating, headache, and vomiting—and mentioned knowledge of at least one death.

Hallgrims (1939) described a death due to the injection of Congo red. The patient developed symptoms and signs suggestive of damage to the nervous system during the course of the injection and died the same day without recovering consciousness. Selikoff and Bernstein (1946) published an account of six cases with severe systemic reactions following injection. Two of these patients died. These reactions occurred within a few minutes of completing the injection and the manifestations were neurological—that is, intense headache, unconsciousness, and hemiplegia. Congo-red tests had been performed previously in all six patients and none gave a history of allergy.

Macht and Grumbein (1938) showed experimentally that Congo red had a depressant effect on the circulatory and respiratory systems, and this was confirmed by Richardson and Dillon (1939). The latter felt, however, that the dose used in clinical work left a wide margin of safety. There would appear to be four factors, all or one of which may have contributed to the severity of the reaction in the present case.

1. *The Solution was prepared with Normal Saline.*—Richardson and Dillon (1939) found that solutions of Congo red in saline were much more toxic for experimental animals than those prepared in water. Such saline solutions were stable for 24 hours only. This increased toxicity appeared to be due to flocculation which occurred in the saline solution. Somers and Whittet (1956) were able to confirm the greater danger with saline solutions. It is customary to use sterile water in clinical practice, and we were unaware of this increased toxicity at the time.

2. *Incomplete Solution of the Powder.*—The Congo-red powder may not dissolve readily, and incomplete solution is not easily detected. Discombe (1949) claims that reactions following injection of Congo red are due to the presence of particulate material, and Somers and Whittet (1956) found that solutions injected into mice before the powder is completely dissolved were highly toxic. The experience of the latter also suggests that batches of the powder vary as regards solubility. This danger would appear to be a strong argument against the use of freshly prepared solutions, which are usually advised on account of the instability of dissolved Congo red. Incompletely dissolved material could be removed by filtration, and, indeed, Somers and Whittet (1956) have shown how solutions of Congo red may then remain stable for several months—a much longer period than previously accepted. From this it seems that previously prepared filtered and autoclaved solutions may be safer.

3. *Variation in Batches of the Drug.*—Macht *et al.* (1939) found striking differences in toxicity between various batches of the dye, and this was greatly increased by the presence of impurities. A similar experience was reported by Somers and Whittet (1956). It is impossible to say what part this factor played in the reactions in the present case, but it later transpired that the ampoule of powder used had been in the ward for a considerable period of time, and possibly several years.

4. *Anaphylaxis.*—Selikoff and Bernstein (1946) believed that anaphylaxis was the basis of the reactions which occurred in their cases. They suggest that the Congo red, being an azo-dye, combined with the patients' own serum to form an azo-protein to which they became sensitized.

Clinical Pathology Notes (University College Hospital Publication) contains a warning regarding the danger of the Congo-red test in the presence of allergy. In the experience of the authors of this handbook, reactions have occurred where this test has been performed in patients with allergic disease (F. V. Flynn, 1956, personal communication).

The presence of bronchospasm in this patient suggested that he was an allergic subject, and it may well be that the train of events was a manifestation of acute sensitivity to the Congo-red dye.

I am indebted to Mr. G. A. Mason for permission to publish this case.

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