response is set up around it. If the faecalith is not very big the peristalsis of the appendicular musculature may be sufficient to force it into the caecum, after which it passes along the intestine. But the inflammatory response it has evoked results in the formation of fibrous tissue—a stricture—and it is noteworthy that all the strictures in the present series were found in the same situation as the faecaliths—that is, at the proximal end of the organ. If, however, the faecalith is too large to be forced into the intestine the inflammatory response will be all the greater. The resulting oedema and compression of the surrounding tissue will compress first the lymphatics and veins, and later perhaps the arteries which drain and supply the apex of the organ. Hence it is usually at the apex that the mucosa is first seriously damaged. Once this has happened the small Gram-positive cocci and rods which Aschoff consistently found can make their way through the mucosa, become increasingly virulent, and set up an acute inflammation.

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MASSIVE SPONTANEOUS INTRAPERITONEAL HAEMORRHAGE

BY

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Massive intraperitoneal haemorrhage in the male in the absence of trauma or gross visceral disease is rare, and for that reason it was thought justifiable to put a further case on record.

Case Record

The patient, a man aged 52, was admitted to hospital with a history of a sudden onset of acute abdominal pain six hours previously. This had come on in the umbilical and hypogastric regions and doubled him up. It had since radiated to the right side of the chest and occasionally into the shoulders. On deep breathing the pain in the right hypochondrium was worse. He had had a similar attack of pain a week before. This was confined to the umbilical region and had not radiated. It had lasted three days. During the whole of the week he had had two or three attacks almost daily, the pain lasting a few seconds. There was no history of indigestion and none of trauma, but some frequency and scalding of micturition was experienced. He had been on a diet for high blood pressure and had lost 2 stone.

The previous medical history—for which I am indebted to Dr. R. O. Knowles of Birkenhead, who sent the patient into hospital—showed him to have suffered from bronchitis for some time. He had had two attacks of pulmonary oedema, as well as anginal attacks, and his systolic blood pressure was 200 mm. Hg. For this he had been dieted. He was a fairly well built man, with an emphysematous chest. On admission he was pale and somewhat cyanosed, cold, and clammy. His pulse was 96 and temperature 96.8°. The whole of the abdomen was very tender, this being especially marked in the upper abdomen and right subcostal regions. There was no real rigidity and no loss of liver dullness. In view of the shocked condition of the patient, his history and build, it was thought that he might have an acute pancreatitis. However, though there was doubt as to the actual causative lesion, there was no doubt that the case was one of acute abdominal emergency and should be explored. The pulse had remained stationary during the period before operation, being 98, and his temperature had risen to 98°.

Under spinal anaesthesia the abdomen was opened through a mid-line supra-umbilical incision and was found to be full of blood. A considerable amount was evacuated, and the hand was passed to the splenic region as it was thought that there might have been a spontaneous rupture of the spleen. Unfortunately, the spleen could not be brought into the wound, but nothing abnormal was felt on palpation. The liver, gall-bladder, pancreas, stomach, and intestines were normal; there was no retroperitoneal mass of clot, which was looked for as it was thought that the bleeding might have come from an aortic abdominal aneurysm. The kidneys felt normal, and there was no retroperitoneal collection of blood in the vicinity. The pelvis was also normal. There was staining of the mesentery and omentum, but no intramesenteric clots were present. As gross aneurysm appeared to have been excluded attention was again given to the spleen, as the quantity of blood there seemed to be greater than elsewhere. An attempt was made to bring it into the wound for inspection; this was successful, but in doing so an adhesion was torn. The spleen appeared to be normal; there was a tear in the capsule about one inch by one-quarter inch, certainly due to operative trauma. There was no pulping of the spleen or any subcapsular haematoma. As the patient's pulse was becoming very weak the abdomen was closed, a drain being left in. His pulse improved in volume once the abdomen was closed. A transfusion of one pint of citrated blood was given an hour later. After the operation his pulse rose to 150 and his temperature to 102°, and in the first twenty-four hours the pulse dropped to between 120 and 136, the temperature at the same time varying between 99.4° and 101.6°. However, thirty-six hours after the operation his pulse became weaker; he became much more dyspnoeic; and he started vomiting, dying soon after.

PATHOLOGICAL REPORT

At the necropsy the abdomen still contained a considerable amount of blood, with what appeared to be a greater proportion in the splenic region and an accumulation in an inguinal hernia sac. The stomach was dilated, and the small intestine also slightly distended but otherwise normal, as also were the liver, pancreas, kidneys, suprarenals, and bladder. The spleen was normal in size and texture. There were two tears in the capsule, each about one inch by one-quarter inch, which were considered by me to have been secondary to the operative trauma. The abdominal aorta showed only very slight evidence of atheroma. The heart was enlarged. The orifices of the coronary arteries were normal. There was slight blood-staining on the upper surface of the diaphragm. Though the individual splanchnic vessels were not carefully dissected the absence of any retroperitoneal clot was thought to exclude any aneurysmal rupture.

Comments

Cases of massive intraperitoneal haemorrhage in the male in the absence of trauma or gross visceral disease appear to fall into two main groups:

1. An older group of patients, aged 44 to 60, with marked arteriosclerosis and high blood pressure, the haemorrhage coming from a ruptured splanchnic vessel. Six cases have been described which can be included in this category; of these, two were not operated on, being discovered post mortem (Moorehead and McLester, 1936), and four were operated on. In three of the latter (Starcke, 1923; Green and Powers, 1931; Buchbinder and Greene, 1935) the bleeding point was found, the vessel was ligatured, and the patients recovered. In the fourth case (Hilliard, 1918), where no bleeding point was discovered, the patient died.

2. A younger group, in which the vascular system is healthy and no bleeding point is found. Three cases come under this heading: Churchman, 1911—a man aged 48; Hartley and McKechnie, 1934—a man aged 31; and Bruce, 1937—a man aged 34. Of these only the last-named survived laparotomy.

It is suggested (Bruce, 1937) that the haemorrhage in the second group may be due to rupture of a miliary aneurysm or at a junctional area, and some support is given to this by the record of two patients who had a massive intraperitoneal haemorrhage from an aneurysm of a splanchnic vessel in the absence of gross arterial disease in the abdomen (Illingworth, quoted by Bruce (1937), a man of 75 with aneurysmal rupture of the middle colic artery; Budde, 1925, a man of 27 with aneurysmal rupture of the left gastro-epiploic artery).

Though from the clinical and operative findings the case described would be considered to belong to Group 1, it should, I think, be placed in the second group in the absence of any gross disease of the main abdominal blood vessels.

I am indebted to Dr. Hugh Smith for the post-mortem findings.

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Clinical Memoranda

Pseudo-hypoglycaemic Attacks

In view of the anxiety and distress caused to the parents and the physician by the occurrence of hypoglycaemic attacks during the treatment of diabetes by insulin, the following case record has considerable interest and importance. In the first place, it is interesting to find a young patient—and one, be it noted, who never to our knowledge had had a genuine hypoglycaemic attack—simulate this therapeutic accident. He had, however, through association with another patient in whom hypoglycaemic attacks were the great bugbear of her parents, opportunities of learning of such catastrophes, and it is not unlikely that this was the source of his knowledge. The importance of the clinical story is that it reveals how treatment may be unwarrantably interfered with through the administration of the very article of food to which the patient is intolerant and the withholding of the very means by which this intolerance is circumvented. Indeed, in the patient above referred to as being very liable to hypoglycaemic attacks, the fear of such has led to interruption of the treatment and the rapid development of coma on more than one occasion.

CASE REPORT

The patient is a boy, aged 12 years and 9 months, who has been under observation since 1930. A sister is also a diabetic. Both children have been dieted and been given insulin, and have done fairly well. They have been in hospital on many occasions to have the diet regulated, or because of an upset of balance in consequence of a septic focus or some other intercurrent infection. The boy has been a particularly difficult patient. He resents all restrictions, and unfortunately parental discipline is very unsatisfactory.

On March 6, 1937, he was readmitted to hospital in order to start a high carbohydrate diet and for the regulation of the insulin dosage. At first an attempt was made to stabilize him on a diet containing 66.5 grammes protein, 57.5 grammes fat, and 110 grammes carbohydrate per day. This was found very difficult; there was considerable glycosuria and slight acetonuria. Insulin was increased from 14, 14, and 12 to 18, 18, and 16 units before breakfast, dinner, and supper respectively. The boy thought he was being kept in hospital longer than had been promised; he became more and more impatient, and was always asking when he was going home.

March 20.—At 10 p.m.—that is, four hours after his evening dose (14 units) of insulin—he was found by the nurse unconscious, cold, and clammy, with a poor pulse and unable to swallow. He was given immediately 50 c.cm. of normal saline intravenously and 15 grammes of glucose in 2 oz. of orange juice, and by 10.15 p.m. he was quite conscious and seemed better.

March 23.—The diet was changed to 70 grammes protein, 62 grammes fat, and 140 grammes carbohydrate, with 53 units of insulin daily. On this diet there was no acetonuria, but moderate glycosuria before dinner.

March 25.—At 12.6 p.m. he received 18 units of insulin preparatory for the midday meal. Eight minutes later he became drowsy; he refused to eat his dinner. When shaken he drank some orange juice, but he kicked much, and there were peculiar jerking movements of the arms and legs, which suggested to the resident staff a convulsion. Blood was taken for sugar estimation and he was then given 20 c.cm. of a 10 per cent. solution of glucose intravenously and 30 grammes of glucose by mouth. He was quite normal at 5 p.m. The report of the blood examination revealed 0.156 gramme of glucose per c.cm., a finding which at once raised the question of the attack having been psychic in nature.

March 29.—At 12.15 p.m., about three minutes after the injection of insulin, he had another attack similar to the one described above. Blood was again taken for examination and revealed 0.148 gramme of glucose and 10.4 mg. of calcium per 100 c.cm. Nothing at all by way of treatment was given on this occasion, in view of the previous blood finding, and he came round almost at once.

April 1.—About 11 a.m. (last dose of insulin at 8 a.m.) he became drowsy and ultimately would pay no attention to anything. By 12 noon he was apparently unconscious, and neither the midday dose of insulin nor the midday meal was given. I saw him at 2.30 p.m. He was very drowsy. He refused to answer questions or do anything asked of him, but he could easily be roused by pressure behind the angle of the jaw. The pulse was of good volume and numbered 60 per minute. The opinion was expressed at the bedside that he was "putting it on" and that nothing was to be done. He was then left alone. At 3 p.m. he sat up and said that he would like to have his dinner. This was given with the usual dose of insulin. Urine passed just before the meal was yellowish-green to the Benedict test but contained no acetone. He had another attack of drowsiness at 10 p.m. which lasted for ten to fifteen minutes, but no attention was paid to him and he was soon all right.

On the following day the boy was told that he would be allowed home on a certain date and that we only wished to see how he could stand the extra sugar. From then he had no further attacks; he co-operated well and was quite contented The diet was changed to 70 grammes protein, 35 grammes fat, and 200 grammes carbohydrate per day, with 24, 22, and 22 units of insulin before breakfast, dinner, and supper respectively. During the last week of residence there was no glycosuria and no acetonuria, and, as promised, he was discharged on April 26, when he weighed 62¾ lb.

Since dismissal in April he has been on the same diet, and since October on protamine insulin, 30 units a day. He has been well of himself, but there is still irregular glycosuria. There have been no recurrences of the above peculiar pseudo-hypoglycaemic attacks, and on December 2 he weighed 67½ lb.

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