Medical Memoranda

Aetiology of Pancreatic Apoplexy

Acute haemorrhagic pancreatitis is usually due to a fulminating infection which either gives rise directly to a haemorrhagic exudate or does so indirectly by the liberation of pancreatic ferments which destroy the walls of the small blood vessels. A few cases, however, of so-called pancreatic apoplexy appear to originate with massive haemorrhage, and the following report throws light on the pathogenesis. The necropsy was one of three similar necropsies seen by me in the last 20 years.

Pathological Report on a Case

A housewife aged 52 (Case No. 179480) was admitted to the City General Hospital, Sheffield, suffering from acute pancreatitis of 10 hours' duration, and she died 26 hours later.

Post-mortem Examination, Feb. 26, 1945 (relevant details only).—The body was that of a very obese female. The peritoneal cavity was full of blood-stained fluid, and the omentum and mesentery were diffusely spotted with fat necrosis. The body and tail of the pancreas were diffusely infiltrated with blood and there was considerable retroperitoneal clot in the left perirenal region. The head and neck of the pancreas were diffusely suffused with blood in their posterior portion, but the anterior parts were only slightly affected, though blood clot surrounded the entire organ. The spleen appeared normal, though surrounded by blood clot, and on dissection it was evident that the haemorrhage had originated in a rupture of the inferior of two large splenic veins before they united to form the main splenic vein about 4 in. from the hilum of the spleen. There was cholesterosis of the gall-bladder, but no calculi or other abnormalities were found in the biliary or pancreatic ducts. No fat necrosis was present in the thorax.

Comment

These three cases all suggest that the primary lesion in the final catastrophe was haemorrhage from rupture of a splenic vein, and that the effused blood injured the pancreas, leading to secondary autolytic digestion, etc., in the region. That the process is not in the reverse direction is suggested from observations on other necropsies of the same condition, which are of primary autolytic or infective origin and which never, so far as I know, lead to the opening of the wall of a large blood vessel. It may be that the initial process is a mild inflammation which produces adhesions between the usually mobile splenic veins and the pancreas, and that some movement may precipitate the final rupture, but I am unable to adduce any proof of this.

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A Case of Achalasia of the Cardia

In a typical case of achalasia of the cardia the symptoms are accompanied by radiological evidence of enlargement of the oesophagus. It is uncertain, however, whether the enlargement follows the onset of symptoms or whether the sequence of events is reversed. The case described below throws light on this point because enlargement of the oesophagus followed the onset of symptoms.

Case History

Mrs. A., aged 59, a housewife, was admitted to hospital on April 5, 1944, with a history of dysphagia and bouts of vomiting since Oct., 1940. She dated the symptoms from the time when her house was bombed. She had lost 34 lb. in weight and her appetite was poor; her diet consisted mainly of soft foods, including milk. There were no abnormal signs on physical examination. The vomitus contained no free hydrochloric acid.

Her oesophagus was *x*-rayed in Oct., 1941, a year after the onset of dysphagia, and was found to be normal. About the same time Mr. F. C. Ormerod examined it endoscopically with the same result. In April, 1942, radiography of the oesophagus showed that no barium passed into the stomach for five minutes and that there was a fusiform dilatation of the oesophagus. These radiological findings were confirmed in April, 1944, the dilatation being somewhat greater than two years previously.

Commentary

The following conclusions can be drawn from this case. (i) Achalasia was probably present for a year before the patient was first investigated in 1941, and yet the oesophagus appeared normal to clinical test. (ii) Definite oesophageal dilatation occurred quite rapidly over a period of six months (Oct., 1941, to April, 1942).

Hurst and Rake (1929–30) have set out in detail their view of the manner in which the oesophageal enlargement comes about. It may be summarized as follows. (*a*) As a result of inflammation of Auerbach's plexus in the region of the cardia, the neuromuscular mechanism is upset and a functional obstruction to the transfer of material from the oesophagus to the stomach is produced. (*b*) The obstruction becomes progressively worse, but for a time hypertrophy of the circular muscle fibres of the oesophagus "compensates" for the obstruction. (*c*) Finally, further compensation can no longer occur and the oesophagus enlarges. In very advanced cases it may also lengthen.

There is a good deal of evidence to support this hypothesis. Many cases with clinical and radiological evidence of achalasia of the cardia show, post mortem, few or no ganglion cells in Auerbach's plexus in the region of the cardia (cf. Rake, 1927). Hurst and Rake (1929–30, p. 499) refer to three cases in which the wall of the oesophagus was found at necropsy to be hypertrophied though the lumen was of normal size, and in which the changes referred to above were present in Auerbach's plexus. This lesion of the oesophagus was symptomless and was thought to represent the stage of "compensation." Hurst (1943) records a case (Case 4, p. 70) in which the dilated oesophagus in the course of a few years became S-shaped, so that the patient could no longer use a mercury bougie.

The missing link in this chain of evidence was the demonstration that an oesophagus with a lumen of normal size could enlarge. Bockus (1943) has described this change, though oesophagitis with ulceration of the mucosa in the region of the cardia complicated his case. The present case shows that the enlargement can occur in an organ which appears normal radiographically and endoscopically.

We wish to thank Dr. C. M. Hinds Howell for permission to publish this case, and Mr. F. C. Ormerod for letting us use his endoscopic observations.

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Neurological Complications in Tropical Macrocytic Anaemia

In view of the statements made by several authorities (Wills, Clutterbuck, and Evans, 1937; Trowell, 1943), that nervous symptoms and subacute combined degeneration have never been found in nutritional macrocytic anaemia, a report of the following case may prove interesting.

Case Record

A coloured woman aged 22 was admitted to the Belize Hospital on Feb. 2, 1943. At the time of admission she was in the seventh month of pregnancy. Her illness had begun three months previously with an attack of fever, which was followed by the onset of numbness in her hands and feet and a sensation of tightness around the waist. Trembling and weakness ensued whenever she tried to stand up, and gradually she lost the ability to walk. Temperature 99° F. on admission. The fundus of the uterus was felt 2 in. above the umbilicus. On examination there was marked loss of power in the upper and lower limbs. Sensation for pain and touch was diminished on the inner aspects of both legs, and there was considerable impairment of the sense of position in the upper limbs and in the left leg. The abdominal reflexes and knee- and ankle-jerks were all absent, and no plantar response could be elicited. Physical examination was otherwise negative. Urine: trace of albumin. Stool: no ova. Blood count: red cells, 2,810,000 per c.mm.; Hb, 68% (Dare), or 11 g. per 100 c.cm.; C.I. (corrected to Haldane), 1.4; M.C.V., 128.5 c.μ; M.C.H.C., 35.5%; white cells, 5,400 (no differential count done); icterus index, 5. Alcohol-histamine test meal: free HCl present. Kahn reaction, negative. Treatment consisted of 6 c.cm. of anahaemin (B.D.H.) during the first week, followed by 18 c.cm. of liver extract (Lilly) during the next three weeks. On March 18 a blood count showed: red cells, 2,730,000 (reticulocytes 3%); Hb, 70% (11.2 g.); C.I., 1.5; M.C.V., 118.5 c.μ; M.C.H.C., 35%; white cells, 7,240. Subsequent treatment consisted of 7 c.cm. of liver extract weekly, acid. hydrochlor. dil. m. 10 t.d.s., and thiamine 9 mg. daily, up to the time of delivery on April 11. The patient's mental attitude during this period became more cheerful, and she said she felt stronger, but no demonstrable improvement in her neurological condition could be detected until she gave birth to a somewhat premature baby weighing 5 lb. After that time her symptoms improved rapidly, and by the end of a month she was able to walk. When last seen, in Nov., 1944, she was in good health, and there had been no recurrence of her nervous symptoms.

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