FATAL PERFORATION OF THE CAECUM
IN A CASE OF SPRUE

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Cases of perforation of the intestine in sprue are extremely rare, and those so far reported have been associated with ulcers in the small bowel. Faber (1904) reported a case of death from peritonitis following rupture of an ulcer in the small intestine. At necropsy sixteen small ulcers were found at various parts of the lesser bowel, but the large intestine was free. Fischer and von Hecker (1922) reported several small ulcers in the lower jejunum and ileum, and Mackie and Fairley (1929) described a superficial healing ulcer in one of their cases about half an inch in diameter, located two inches above the ileo-caecal valve. In two cases reported by Manson - Bahr (1924) perforation had occurred. One of these—a case of ten months' duration—showed a large ulcer in the ileum six feet from the ileo-caecal valve opposite the mesenteric attachment; it measured one inch in diameter, and had perforated in two places, producing a fatal peritonitis. In the other case twelve ulcers varying in size from 0.5 to 1 c.cm. were found in the ileum, the mucosa of which was acutely inflamed. One small ulcer 3 mm. in diameter was found in the upper part of the jejunum; multiple perforations had resulted in peritonitis and death. The present case is of special interest because no evidence of ulcers in the small or large bowel was found at necropsy, and the perforation involved the caecum. An epitomized history is recorded below.

Case Record

Mrs. S., aged 58 years, was admitted to the Hospital for Tropical Diseases on February 28th, 1934, seriously ill with tropical sprue.

Past History.—Born in England, she had gone to India in 1900, and had remained there until 1926, since when she had not been out of this country. There was a history of malaria in 1901, and of dysentery, without recurrence, in 1902.

Recent History.—The onset of the immediate trouble occurred in September, 1925, when the patient began to have large, pale, frothy stools and flatulence, having lost much weight. These symptoms persisted, and she returned to England in March, 1926. The tongue was then sore with ulcers and fissures, and intestinal symptoms were marked. In 1927 she received three months' treatment with milk diet and temporarily improved. Then in November, 1930, she became definitely worse, receiving treatment including injections of liver extract over a period of six months without any great

benefit; subsequently she returned home, improving up to a certain point. By June, 1933, she had begun to lose weight; the stools were bad, flatulence marked, and the tongue sore; in July an anal fissure developed. Since Christmas vomiting occurred infrequently and she had lost 3 stone 9 lb. in weight. Attacks of tetany had also been present for a considerable period, the last being about a fortnight before admission. The stools were pale, sometimes formed and sometimes fluid; flatulence was severe, and she could not retain solid food, vomiting it almost immediately.

Physical Examination (February 28th).—Inspection revealed a thin, elderly, much emaciated woman with pale mucous membranes and conjunctivae. She was edentulous; the tongue was smooth and pale with hyperaemic edges. She was very weak on admission, and a grave prognosis was given. There was an apical systolic murmur, but no cardiac enlargement, and the breath sounds, though harsh, were normal. Abdominal distension was present, the muscles were atonic and wasted, and peristaltic movements were visible through the thinned abdominal parietes. Some tenderness was noted on deep pressure over the appendix area. An anal fissure was found on the posterior aspect of the sphincter, and above this the rectum felt ballooned.

Laboratory Records

(March 1st).—Red blood counts, 1,700,000 per c.mm.; haemoglobin, 50 per cent. (Haldane); colour index, 1.3; average diameter of corpuscle, 8.6 μ (halometer). The blood picture was typical of megalocytic anaemia; megalocytes were numerous; anisocytosis and poikilocytosis were marked; normoblasts were scanty. Biochemical examinations were postponed until March 5th, when the serum calcium was 8.7 mg. per 100 c.cm., the

serum phosphorus 2.5 mg. per 100 c.cm.; and the glucose tolerance curve showed a maximal rise of 54 mg. per 100 c.cm. one and a half hours after the administration of 50 grams of glucose.

Progress Notes.—The case ran an apyrexial course with normal pulse rate and respirations until the terminal crisis with sudden perforation and peritonitis. A high protein, low fat, low carbohydrate dietary in the form of "sprualac" introduced by Fairley in 1932 was used, and initially three injections of campolon (each 6 c.cm.) were given; a sub-maximal reticulocytosis of 10.4 per cent. (see Graph) occurred on the sixth day, and subsequently fluctuations in the counts occurred throughout the course of the disease, the percentage, however, always remaining above the normal. Though there was improvement in the alimentary features of the case, looseness of the bowels persisted, and there was but little gain in weight; though the red corpuscles showed some increase in numbers the haemoglobin percentage remained stationary (see Graph). Analysis of the stools on March 16th showed: total fat, 19.2 per cent.; neutral fat, 2.6 per cent.; fatty acids, 16.6 per cent.

On March 19th the patient was put on two tubes of "exhepa" daily (equivalent to $\frac{1}{2}$ li. whole liver), and this later was increased; daily injections of campolon (2 c.cm.) were also given, as indicated in the Graph. Again an appreciable increase in the red cells occurred, but the haemoglobin showed no improvement. A convalescent mixed diet was allowed on April 9th, and though the patient was troubled with flatulence she put on 2 lb. in weight, and progressed satisfactorily during the next week. The red cells also increased, and on April 16th the count showed red blood cells, 3,585,000 per c.mm.; haemoglobin, 50 per cent.; colour index, 0.7; average diameter of corpuscle, 8 μ ; megalocytes, microcytes, anisocytosis, and poikilocytosis still present.

Haematological response in a case of tropical sprue dying from perforation of the caecum with peritonitis.

On April 18th, at 5.15 p.m., the patient complained of a sudden severe abdominal pain, and vomited copiously. She was in great pain, lying with the knees drawn up, and exhibited a greyish pallor and drawn facies. The abdomen was very distended, definitely rigid, and pressure increased the pain. Perforation with peritonitis was diagnosed, and Mr. A. H. McIndoe did an immediate laparotomy.

Operation.—There was a gush of gas and faecal fluid when the peritoneum was incised, a diffuse generalized peritonitis with ileus being found. A small circular hole about 1.5 cm. in diameter was found posteriorly, situated at the junction of a mobile caecum and ascending colon some two and a half inches above the base of the appendix. The bowel wall was very thin in the vicinity, but no ulcer or growth was palpated.

Necropsy.—The caecum and adjacent ascending colon were distended and thinned, and a circular hole was found in the bowel as indicated above. There was no induration or thickening around the perforation, and no signs of surrounding inflammation suggesting that perforation had occurred at the site of a former ulcer. A vein draining the area was thrombosed. Further details of the necropsy will be published separately later.

Comment

Though the patient had definitely improved as a result of treatment and the red corpuscles had increased by 1,885,000 per c.mm., the haemoglobin failed to increase in the way the vast majority of sprue cases do. The patient, as already stated, was very weak on admission, and it appears probable that venous thrombosis in a thinned and over-distended bowel led to sudden bursting of a devitalized area of the caecal wall. In the light of subsequent happenings it would probably have been better if the convalescent diet had been of higher protein and lower carbohydrate composition. Iron therapy was withheld for fear of irritating the bowel and increasing the diarrhoea.

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The Christchurch Hospital, New Zealand, publishes a review of the work of the radium and deep therapy departments between 1924 and 1934, which has been compiled by Dr. P. Clennell Fenwick, F.R.C.S.Ed., who is in charge of the departments in question. In his introductory note he states that "during the last nine years every request for additional equipment has been granted at once, and we are now so well equipped that we are able to carry out the same methods of treatment that are in use in Europe and in America." In May, 1930, a special consultation clinic was organized, which meets weekly and is composed of all members of the honorary and departmental staff. Each patient is examined by the members, and re-examinations are made at regular intervals. Carefully worked out recording and "follow-up" systems are in routine use. The consultation clinic has been found to be especially useful in the case of persons who ask for examination under the impression that they have cancer; and the united assurance of an expert committee that such is not the case has done much not only to render the persons happier but to encourage people to come for advice. Since 1930 the clinic has examined 785 new patients, while the total attendances for examination and re-examination have been 2,004. Brief descriptions are given of the methods of treatment employed, together with summaries of the results obtained. There are also useful notes of the after-care of cancer patients and upon the mental aspect in carcinoma. A useful series of precautions to be observed in treatment appears at the close of the report.

Clinical Memoranda

TORSION OF HYDATID OF MORGAGNI SIMULATING ACUTE APPENDICITIS

The following case seems to merit report as illustrating one of the more uncommon conditions which may present the clinical picture of acute appendicitis.

A well-developed girl of 16 years was seen about five hours after the onset of abdominal pain, at first generalized, but later localized in the right iliac fossa, accompanied by vomiting. Temperature, 99°; pulse, 96. There was diminution of abdominal movement, some rigidity in the lower right quadrant, and tenderness just below McBurney's point. A fairly definite diagnosis of acute appendicitis was made. An hour later the abdomen was opened by Davis's incision: a considerable amount of sero-sanguineous fluid escaped from the abdomen. The appendix, which showed injection of its peritoneal coat, was removed. As the condition of the appendix seemed incompatible with such an amount of bloody fluid the pelvis was explored. The right ovary was polycystic. The uterus and left appendages were in all respects normal. The two right hydatids of Morgagni were larger than normal, and presented pedicles about 1.5 cm. long: the uppermost one resembled a ripe cherry in size and appearance. It was tense, and its pedicle was twisted on itself through 1½ turns in a counter-clockwise direction. The pedicle was ligated and the strangulated hydatid removed. The other hydatid was also removed, as the occurrence of a similar mishap seemed not unlikely in view of the abnormal length of its pedicle. A wedge-resection of the right ovary was done. The abdomen was closed without drainage. Convalescence was uneventful.

The points of interest in this case are: (1) the accurate simulation of acute appendicitis, and (2) the large amount of fluid poured out in six hours from a small strangulation.

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MYOIDEMA

Myoidema is a physical sign which seems to have been almost forgotten, as it is not mentioned in some of the recent textbooks of medicine, and is not known to the younger generation of doctors. It is a localized contraction of wasting muscle, produced by a sharp tap, and is most often seen in cases of pulmonary tuberculosis. It is readily obtained by striking the pectoralis over the second and third ribs with the end of the middle finger, as if percussing rather forcibly. In all cases where the tuberculosis is active a small lump of muscle will at once arise at the stricken point and quickly disappear. It may be seen in other diseases where there is loss of flesh with febrile temperature. One condition is, so far as I know, peculiar to pulmonary tuberculosis, and that is, the presence of myoidema on one side of the chest only; this happens when there is active disease at one apex, and that slight. In the more advanced cases there is often a difference on the two sides as to the readiness of myoidema to appear, the more ready contraction being produced on the more diseased side. In acute cases it can be got in every muscle of the body. A very fair estimate of the rate of progress of the disease at the time is given by this phenomenon—the more readily it is produced the more acute the case, and, if it cannot be obtained, the tuberculosis is quiescent.

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