pitched tinkling variety; transmitted cardiac sounds were audible over the whole abdomen. It was possible to elicit splashing in distended loops of bowel. On rectal examination the pelvic peritoneum was found to be tender. The remainder of the examination was negative.

At operation it was found that several feet of small bowel had herniated from above through a defect in the terminal mesentery, and the weight of the prolapsed closed loops had in turn obstructed that part of the terminal ileum which formed the lower boundary of the hiatus. The viability of the loops was not in doubt, and reduction was affected without difficulty. It was then found that the caecum had undergone rotation and had accompanied the small bowel through the defect. After returning the caecum to its normal position it was seen to fill through the ileo-caecal valve. The appendix, which was normal in appearance, was not removed. It was noted that the mesenteric defect was ovoid in shape and about 3 in. (7.5 cm.) in length. The margins were rigid and smooth; the medial margin was swollen by the presence of a chain of enlarged glands suggestive of tuberculous adenitis. The congestion and oedema surrounding the defect obscured the main vessels. From the lateral side it was possible to bring across a longitudinal fold of peritoneum and attach it to the rigid medial margin of the defect with interrupted thread sutures, and thus the mesenteric hiatus was completely occluded. Several months after operation progress had been entirely satisfactory.

COMMENT

There is little doubt that in this case tuberculous mesenteric adenitis played an all-important part. There were vague pains in the right lower abdomen up to the age of 18, and, although at this time appendicular disease was suspected, the probability is that the real cause of the discomfort was mesenteric adenitis. It may be suggested that adhesions formed between the glands and the adjacent terminal mesentery and produced a tenting. The recent strain on a tented, fibrosed, and atrophied mesentery would be sufficient to tear an opening which was soon enlarged by the herniation of small bowel. Within five days of the strain the patient had his first symptom of obstruction, and operation was carried out five days later; in 10 days the margins of the defect would no longer be expected to present features of a recent tear.

The strongest supporter of the developmental theory would find it hard to attribute the case described to congenital causes. One must accept a congenital cause for mesenteric defects encountered in young people, but it is difficult to believe that patients with large defects can reach adult life without internal herniation and obstruction supervening. The defect encountered in the previously reported case was attributed to congenital causes, but, on looking back and in the light of the recent case, one cannot be so certain. In a hurried operation on a critically ill patient with distended coils of small bowel it would be quite easy to miss evidence of previous inflammation, if any such evidence remained. It is always interesting to note what few local signs persist several months after an appendix abscess has resolved, and therefore it would not be unreasonable to assume that an inflammatory condition in the right iliac fossa could eventually produce a mesenteric defect without showing, at the time of operation for the acute obstruction, any evidence of such inflammation. The fact that the ileo-caecal region is the common site for both acute and chronic inflammatory conditions would account for the predominant incidence of mesenteric defects in the terminal mesentery.

JOHN A. BATY, F.R.C.S.Ed.

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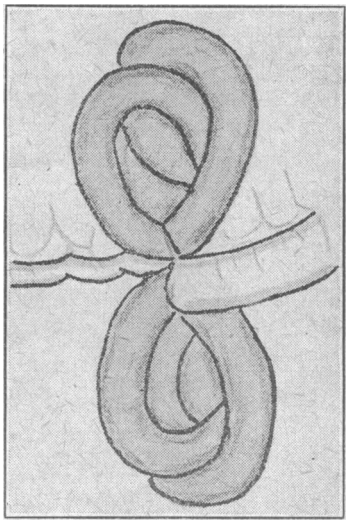
Transmesenteric Herniation Due to Tuberculous Mesenteric Adenitis

My colleague, John Baty, recently read a paper on the subject of transmesenteric hernia at a meeting of the Shrewsbury Clinical Club, and described the case published above. I have since encountered a further case in which the defect was certainly due to tuberculous mesenteric adenitis.

CASE REPORT

A man aged 31 was admitted at 3 a.m. on September 11, 1951, from a sanatorium where he had been a patient on account of extensive bilateral fibroid pulmonary tuberculosis with some cavitation. Owing to his severe deafness it was difficult to elicit the history, but he admitted only slight occasional indigestion before the gradual onset at 10 p.m. on September 10 of a dull epigastric ache associated with nausea. The pain gradually became severe, and he had vomited thrice. The bowels had acted the previous morning. On admission there was some tenderness and guarding in his upper abdomen, but no rigidity and no distension. The pain did not appear to be colicky, and it was thought that the patient was unlikely to be suffering from either intestinal obstruction or a perforated ulcer. In view of the advanced pulmonary disease it was considered unwise to open the abdomen except for most definite indications. After 100 mg. of pethidine he at first appeared to be improving and did not vomit again; but the pulse rate tended to rise, and by the afternoon of September 11 there was a resonant suprapubic swelling typical of strangulated bowel.

A right lower paramedian incision, made under local analgesia, revealed blood-stained peritoneal exudate and gangrenous small intestine, but this could not be satisfactorily examined until general anaesthesia was induced by Dr. M. J. Harker with thiopentone, gallamine triethiodide, gas, oxygen, and trichlorethylene. The appearances at first suggested strangulation by a band or volvulus associated with a band, but when the whole affected area was delivered it was seen that there were two large loops of gangrenous bowel, one on each side of the mesentery, evidently communicating through a small defect near the attachment of the intestine (see illustration). We were immediately reminded of Baty's attribution of the mesenteric defect in his case to tuberculous adenitis, but at the time of operation we could not find any enlarged lymph nodes in the oedematous mesentery—which supports his contention that it is easy to miss evidence of previous inflammation. We did, however, observe numerous tubercles on a portion of the dilated ileum.



The strangulated bowel was clearly beyond hope of recovery, and the two loops were therefore excised in one piece, together with the defect in the mesentery, and continuity of the bowel was restored by axial anastomosis. The post-operative course was surprisingly uneventful; gastric suction was not employed, as it was felt that a tube in the pharynx might aggravate the chest condition, and in fact he did not vomit after the operation. After some hesitation intravenous fluid was begun at midnight on September 11/12, but the needle escaped from the

vein before 500 ml. had been administered, and, as bowel sounds were then audible, no further intravenous fluid was given. The bowels acted after a glycerin suppository on September 14 and naturally on the 16th. In view of the risk to other patients in the ward he was transferred back to the sanatorium the next day. His cough then appeared no worse than before his operation. On January 3, 1952, his chest condition remained unchanged and he has had no further abdominal symptoms.

Dr. M. Symons reports as follows on the specimen: "Approximately 100 cm. of small intestine, purple in colour, and containing a foul-smelling mixture of blood and faeces. At the centre there is a hole in the mesentery, through which loops of gut from above and below have passed in opposite directions. On the gut side of the hole are a number of large hard lymph nodes. Microscopically the mesentery shows extensive haemorrhage into loose connective tissue: it contains lymph nodes which are almost entirely replaced by hard tubercles with no caseation."

Comment.—Gangrene of the bowel appears to be uncommon in transmesenteric hernia, and I am not aware of a previously reported case in which the hernial orifice was available for histological examination.

I am indebted to Mr. Baty for making his paper and the record of his case available to me, to Dr. Harker for anaesthetizing this poor-risk subject, and to Dr. Symons for his careful examination of a somewhat unattractive specimen.

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Spontaneous Rupture of the Stomach

Cases of this condition are extremely rare. According to Leger and Maes (1947) only 30 cases have been reported. A further case was recorded by Ladkin and Davies (1948). I have been able to find only four cases in the British literature, and all ended fatally. The present case is, I believe, the first recorded survival in the British literature and the second in the whole literature.

CASE REPORT

A married woman aged 67, the mother of ten children, was admitted on December 3, 1949, for an "acute abdomen." She had been healthy until 1939, when she began to suffer from indigestion and pain in the epigastrium, and *x*-ray examination revealed a gastric ulcer. Operation was refused, and for ten years she was treated medically. In spite of this, she suffered from intermittent epigastric discomfort, sometimes accompanied by nausea and vomiting. Her appetite remained good. Her bowel habit and micturition were normal.

Three weeks before admission the symptoms became progressively worse in spite of greater care in diet. The vomiting became frequent; it was pronounced at the end of the day, and latterly every meal had been vomited. The vomiting gave some relief from the discomfort and fullness. Her last normal meal was three days before admission. The next day she had only a small meal, and the day before admission she spent in bed without taking any solid food. Repeatedly during the 48 hours before admission she unsuccessfully attempted to vomit; the pain in the epigastrium was more pronounced, and she was seized with sharp stabbing pain in the left hypochondrium, which lasted a few seconds and relieved the desire to vomit. This acute pain was then replaced by a constant dull pain generalized in the upper abdomen, maximal at first in the left hypochondrium and later in the left iliac fossa.

On examination her general condition was poor; she was shocked, dehydrated, and very apathetic. Her tongue was furred and dry. Her pulse was 112, respirations 32, and temperature 97° F. (36.1° C.). The cardiovascular and respiratory systems were normal. There was obvious wasting. The epigastrium was distended, and tenderness and rigidity were present throughout the abdomen, with the maximum point of tenderness in the left hypochondrium. A tentative diagnosis of perforated gastric ulcer was made and immediate operation carried out.

Under nitrous oxide, oxygen, and ether anaesthesia (Dr. C. R. Blaiklock) the abdomen was opened through a midline supra-umbilical incision. A small amount of turbid fluid was at once apparent. The stomach was grossly distended, the greater curvature being well below the umbilicus. The wall was thin, smooth, and greyish white in colour. The pyloric antrum was thickened, causing complete obstruction, but the duodenum appeared normal. A large amount of greenish grey, turbid, slightly offensive fluid, with fragments of undigested food (peas, onions, and raisins) was found in the left hypochondrium. On handling the stomach, some of the contents escaped through a linear slit in the anterior wall fairly close to the lesser curvature and near the cardia. The slit was 1 in. (2.5 cm.) long, running parallel to the lesser curvature. The edges were clean-cut and paper-thin, and there appeared to be no local pathological lesion. Examination of the surrounding stomach wall revealed no evidence of acute or chronic inflammation, necrosis, or previous scarring. There were no adhesions. A short distance from the slit, near the cardia, the loss of a circular-shaped portion of serosa was noticed. No evidence of any other pathological lesion was found in the region of the rupture.

Closure of the slit was not easy owing to the thinness of the edges of the stomach wall. A continuous suture was inserted, then a second Lembert layer suture, and a stomach tube was passed for lavage. Some difficulty was encountered owing to spasm of the cardia. Because of the pyloric stenosis a posterior gastro-enterostomy was performed. The soiled area was swabbed out. During this operation the capsule of the spleen was damaged, and profuse haemorrhage followed, necessitating splenectomy. The abdomen was closed and drainage was established through a separate incision in the left hypochondrium.

The patient made a satisfactory recovery and left the hospital three weeks after the operation. One year later she had gained weight, was taking ordinary diet, and was enjoying a normal life.

A radiological report on April 17, 1950, by Dr. B. Klukvin stated: "Oesophagus normal. No deformity of the stomach could be seen at the site of rupture. Meal entered the stomach freely. Posterior gastro-enterostomy functioning normally. No barium could be persuaded to pass the pylorus."

COMMENT

A study of the literature reveals that various factors must be considered as the causes of spontaneous rupture of the stomach. The distension of the stomach, with the increase in gastric pressure, is a striking feature. It may be associated with overloading or overdrinking, sometimes combined with the administration of bicarbonate of soda. It may occur with or without any pathological condition. It may follow after operation, and in one instance occurred during labour.

In the case reported pyloric obstruction was complete and associated with spasm of the cardia. No doubt this condition was accompanied by reflex dilatation of the stomach—the intragastric pressure exerted by paroxysmal aerophagia, retching, and violent, almost unproductive vomiting, steadily increasing until rupture of the thin wall relieved the pressure.

I should like to thank Mr. R. J. Rutherford for permission to record this case.

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