ACQUIRED DIVERTICULOSIS OF THE SMALL BOWEL

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Historical Review

Although the pathological anatomy of acquired diverticulosis of the small bowel was described in 1807 by Astley Cooper,[1] the first report of this condition in a living patient did not appear before 1906.[2] In 1920 the condition was demonstrated radiologically for the first time[3] and by 1943, 122 descriptions of this condition had been traced in the literature.[2] In 1950 Kozol *et al.*[4] performed a laparotomy on a patient who had massive gastro-intestinal haemorrhage, when diverticula of the jejunum were noticed, but not implicated, and the abdomen was closed. When bleeding persisted, a second laparotomy was done, the portion of the small bowel harbouring the diverticula was resected and the haemorrhage stopped. The resected segment showed multiple diverticula, many of which contained fresh blood. Ulceration of some diverticula seemed to be the source. This episode stimulated their perusal of the available literature, which included 300 cases of small bowel diverticulosis. Of these, only 6 cases were complicated by haemorrhage. Since that time various complications have been described.

Classification

According to Edwards,[5] diverticula of the small bowel can be classified as follows:

1. *Congenital diverticula*
   - A. Meckel's diverticulum
   - B. Non-Meckellian
     - (*i*) Giant diverticulum
     - (*ii*) Diverticula associated with ectopic pancreatic nodules
     - (*iii*) So-called 'cyst-like' diverticula
2. *Acquired diverticula*
   - A. Primary or hernia type
   - B. Secondary type
     - (*i*) Diverticula owing to pathological conditions of small bowel wall
     - (*ii*) Traction or pulsion diverticula
     - (*iii*) Pseudo-diverticula, e.g. choledocho-duodenal fistula

The subject under discussion here is the acquired traction or pulsion diverticulum, which should be more accurately termed a saccule.

Pathological Anatomy and Diagnosis

Acquired diverticula occur on the mesenteric border of the bowel, at the sites of penetration by nerves or blood vessels. Most frequently they occur in the proximal jejunum, less frequently in the whole of the jejunum, and even more rarely in the jejunum and ileum. The duodenum is involved in about 25% of all cases,[6] and may rarely be the only part of the small bowel affected. The condition is apparently more common in men than women, occurring mostly in people over the age of 60.

Although it is estimated that only about 10% of the general population have diverticulosis of the colon, this condition co-exists with small bowel diverticulosis in about 30% of all cases.[6, 7] Other pathological conditions that have been described in patients with diverticulosis of the small bowel are: gallstones, peptic ulcer, hiatus hernia, ulcerative colitis and carcinoma of stomach, pancreas or colon.[7]

The wall of an acquired diverticulum consists of only mucosa, submucosa and serosa, and since the ostium is relatively large and the contents mostly of a fluid nature, obstruction is comparatively rare.[8] They vary in size, some being as large as golf balls or even larger, while others may be hardly noticeable. The largest occur most often in the proximal jejunum.[7] They may be very difficult to demonstrate radiologically, at operation or at necropsy. When empty, they can escape the eye completely, but the injection of air into the bowel lumen, however, will nearly always demonstrate them.[3] No doubt, then, in many cases diverticula will be missed unless suspected and diligently sought.

The incidence of diverticulosis of the small bowel in the general population has been estimated by various authors to be from 0·26% to 1·3%.[7, 8]

When solitary, a diverticulum will most commonly occur in the jejunum.[2, 9] It is well known, however, that new diverticula may develop at a later stage, and a case has been described where completely normal bowel, left after resection of a pathological segment, developed multiple new diverticula, which were seen at a subsequent laparotomy after an interval of years.[2]

An affected segment of small bowel seems to lose its power of propulsion to a marked degree and although violent churning movements, sometimes in a retrograde direction, can be observed under the X-ray screen, stasis of the contents very often occurs.[7] Peristalsis is apparently not smoothly propagated over the entire length of the small bowel, and these violent peristaltic waves seem to stop abruptly at certain points without having achieved much. The segment of small bowel containing the diverticula, as well as the normal segment proximal to it, may be enormously dilated, while the normal portion distal is usually empty and of normal diameter.[7] The term 'jejunal dyskinesia' has been coined in an effort to describe these violent ineffective contractions and it has been suggested that a neuromuscular imbalance akin to that of Hirschsprung's disease might be implicated, but histological study of affected small bowel has so far failed to show any organic changes in the ganglia.[7]

Symptoms and Complications

About 60% of those known to have this condition never develop symptoms and the condition is then discovered incidentally at laparotomy, on barium X-ray studies done for other reasons, or at necropsy.[6]

Symptoms include vague postprandial discomfort in the epigastrium and left hypochondrium, which may be described as 'nervous dyspepsia'. Eructation of gases and passage of abnormal amounts of flatus may occur and give relief. These dyspeptic symptoms may at times be more severe and concomitant vomiting may occur. Symptoms may then be very suggestive of incomplete or even total obstruction of the small bowel.[7]

These patients are often in poor physical condition, owing to considerable weight loss, and are sometimes

anaemic. When those with so-called jejunal dyskinesia are operated on, no organic obstruction will be found, although the segment proximal to the affected portion of small bowel may be grossly dilated and the distal segment collapsed and devoid of contents.

On the whole, patients exhibiting the above symptoms rarely require surgery. Conservative therapy, consisting of small amounts of soft foods, taken at frequent intervals, combined with antispasmodic drugs, and rest after meals, is the method of choice.

Occasionally steatorrhoea may be the main symptom and it may be found that the absorption of glucose, iron, folic acid, amino acids and vitamin B_{12} are defective in varying degrees, depending on which portion of small bowel harbours the diverticula. Glucose, iron and folic acid are absorbed in the proximal jejunum, fats and amino acids through the entire length of the small bowel and vitamin B_{12} mainly in the distal ileum. This malabsorption syndrome may be complicated by a macrocytic anaemia of varying degree[7, 10] and cases simulating pernicious anaemia, even with signs of subacute combined sclerosis of the spinal cord, have been described.[7] The malabsorptive state is apparently caused by stasis of small bowel contents in the diverticula with excessive growth of intestinal organisms (*E. coli*) which are not normally found in these parts of the bowel. The villi of the affected portions are flattened and defective absorption ensues. The state is analogous to that sometimes found where blind loops of small bowel exist.[10]

Indications for Surgery

Indications for surgery, although well described, may vary from one surgeon to another. It is not strange therefore that one authority found it necessary to operate on 38% of patients under his care,[7] while others claim that only 10% of patients require surgery.[6, 8] Definite indications for surgery are the following complications:

(a) *Acute inflammation of a diverticulum*, whether perforation occurs or not.

(b) *Perforation of a diverticulum*. This may occur spontaneously or be the result of acute inflammation. A blow on the abdomen may cause a diverticulum to rupture and a foreign body may perforate it. The consequences of perforation are obvious—escape of bowel contents with ensuing peritonitis, either localized or generalized. In an isolated case, small perforations have been known to give rise only to leakage of gas into the peritoneal cavity and to sub-serous accumulation of gas. These perforations have been so small that the semi-liquid bowel contents could be filtered out,[7] allowing only gas to escape.

(c) *Haemorrhage from a diverticulum* may occur and may be a dangerous complication[3, 4, 6, 7, 9] as seen in case 2 of this report. This condition should always be considered in the differential diagnosis of a middle-aged patient presenting with haematemesis and/or melaena, especially after a previous episode of jejunal dyskinesia. Myburgh[11] has pointed out that haemorrhage owing to diverticulosis of the large bowel in old people, although often alarming, seldom has fatal results.

(d) *Intestinal obstruction*. Jejunal dyskinesia may be so severe that a laparotomy becomes imperative. X-rays of the abdomen have been known to demonstrate fluid levels which were assumed to denote obstructed loops of small bowel, but which proved at operation to be accumulations of fluid and gas in large diverticula.[8]

Incomplete organic obstruction of the third and fourth parts of the duodenum may be caused by accumulation of semi-fluid contents in a large diverticulum situated behind these parts (case 1 of this report).

(e) *Acute inflammation of a diverticulum* may cause adhesion to another viscus or to the abdominal wall. Volvulus of the small bowel with complete organic obstruction may ensue.[12] As most diverticula are situated in the proximal jejunum, this is a very dangerous variety of small bowel obstruction.

(f) *Neoplastic changes* may occur in a diverticulum.[6]

SURGICAL TREATMENT

Complications as severe as those mentioned require resection of the affected small bowel, with end-to-end anastomosis. An affected segment of small bowel having undergone volvulus, is best resected at operation although the blood supply may prove adequate after reduction.

Where no organic obstruction can be demonstrated at operation and the obstructive symptoms and signs are blamed on jejunal dyskinesia, most authors recommend that a resection of the affected segment should be performed, and that entero-anastomosis, to bypass the affected segment, should not be resorted to, except in poor-risk subjects, because symptoms may persist or recur. Patients, however, have been treated with success by such bypass entero-enterostomy,[8] of which case 1 of this report is an example.

If in a case where jejunal dyskinesia has been severe enough to warrant a laparotomy, the small bowel is found to be affected to such an extent that resection of the whole portion would be too drastic, it is advised that only the proximal jejunum be subjected to a limited resection as the largest diverticula usually occur there.[7]

Diverticula which are incidentally demonstrated by barium X-ray series, or at laparotomy done for another condition, do not require surgery, unless the diverticula are found to be very large and the unaffected small bowel severely dilated. This should be regarded as a progressive condition which will cause trouble in future, and is best resected at an early stage.[7]

CASE REPORTS

The two cases described here, illustrate some interesting facets of the condition.

Case 1

A White female, Mrs. A.D.W. aged 76 years, was admitted to hospital on 23 March 1963, complaining of pain in the left hypochondrium, which came on soon after meals, accompanied by severe heartburn. Present for a year, the symptoms had become progressively worse and in this time she had lost 30 lb. in weight. At times she experienced nausea and tended to vomit after meals. The vomitus consisted of food just eaten and bile, and never contained blood. Appetite was poor but bowel actions were normal.

On examination it was obvious that this old lady must have lost considerable weight, although her general condition was good for her age. She had cataracts of both eyes.

Her abdomen was slightly distended but no peristaltic waves could be seen. There was slight tenderness in the epigastrium and left hypochondrium. The spleen and liver were not palpable.

A full blood count showed no abnormalities. The liver functions were normal and the blood-urea was not elevated.

A barium meal examination showed a grossly dilated stomach, and such thickened gastric mucosa on the greater curvature that a carcinoma could not be excluded. The first, second and third parts of the duodenum were enormously dilated approaching the diameter of the stomach. A few diverticula were noticed in the proximal jejunum.

At laparotomy on 25 March 1963 extensive diverticulosis of the duodenum, jejunum, ileum and colon was found. At the duodeno-jejunal flexure a diverticulum somewhat larger than a golf ball was seen, stretching up behind the fourth part of the duodenum. It contained thickened material, which could only be expressed with difficulty, and on account of its bulk, had compressed and nearly completely occluded the duodenum from behind. There was also a large active ulcer on the anterior wall of the first part of the duodenum but no sign of gastric neoplasm (Fig. 1). Considering her age, and the

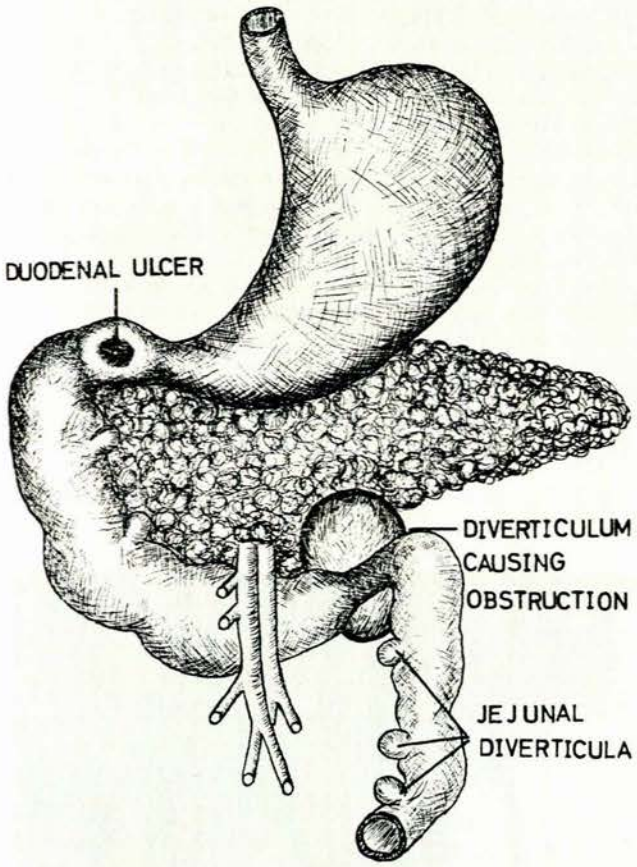

Fig. 1. Anatomy of the pathology in case 1.

extensive nature of her disease, it was decided not to do any resection. A vagotomy and pyloroplasty was performed for the peptic ulcer and after mobilizing the duodenum from behind the superior mesenteric vessels, a side-to-side anastomosis between the third part of the duodenum and first portion of the jejunum completed the operation. The patient made an uneventful recovery and when discharged on 4 April 1963, the heartburn and epigastric pain had disappeared and her appetite was much improved. Within a month she had increased her weight by 10 lb.

A year later she was readmitted for review and in the interval had had no further heartburn or epigastric pain. A vague postprandial epigastric discomfort sometimes manifested itself, but was not at all severe and she had never vomited since her operation. Her general condition was excellent and no abdominal tenderness could be elicited. A barium meal examination was negative, other than for some dilatation persisting in the second part of the duodenum, although this was considerably less than the year before. Numerous diverticula were again demonstrated in her small bowel. The entero-anastomosis was patent, and functioned well. At this stage she underwent an operation for cataract and was discharged on 7 May 1964.

Case 2

A White male, A.N.G.W. aged 65 years, was transferred to our care on 17 May 1963, from a neighbouring hospital, complaining of diarrhoea for the previous 6 weeks. For a few days before his transfer, he had noticed that his stools had become pitch-black, and he had vomited large amounts of fresh blood on a few occasions. He complained of colicky pains in the left hypochondrium. His appetite had been poor for some time and he had lost considerable weight during the previous 2 months.

On examination he was anaemic (Hb was 7·9 G/100 ml.) and severely shocked. The abdomen was slightly distended and there was marked epigastric tenderness. A preliminary diagnosis of bleeding peptic ulcer was made and a blood infusion was commenced. The patient was informed that an emergency laparotomy might have to be considered, but he refused operation. His condition fortunately improved, and after transfusion of 5 pints of blood, haemorrhage seemed to have stopped and his blood pressure remained within normal limits.

Three days after admission, a barium meal examination demonstrated an ulcer in the pyloric antrum of the stomach. A second ulcer in the first part of the duodenum was suspected. Numerous small diverticula were demonstrated in the second and third parts of the duodenum. It was again suggested to the patient that a laparotomy should be done, but he again refused, and left the hospital on 22 May 1963.

On the same day he was readmitted to the neighbouring hospital, from which he had originally come, and was treated conservatively until his death on 18 June 1963. The cause of death was noted as 'multiple diverticula of the duodenum and jejunum with perforation of a diverticulum'.

COMMENT

Some interesting points are illustrated by the above two cases:

1. Diverticulosis of the small bowel, although seldom causing symptoms, may be a serious and even fatal condition.
2. The occurrence of peptic ulceration and diverticulosis of the colon, concomitant with diverticulosis of the small bowel, should be kept in mind.
3. A large diverticulum situated behind the third part of the duodenum can cause an obstruction of this part.
4. A bypass entero-enterostomy, although not generally accepted as the best possible operation for this condition, proved in our case to be quite adequate in relieving the obstruction.

SUMMARY

Two cases of acquired diverticulosis of the small bowel are presented. One case exhibited a rather unusual form of organic obstruction, which was relieved by entero-enterostomy. Both cases had peptic ulcers as well. One patient had severe haemorrhage either from his diverticula or his ulcer, which stopped after a few days. He eventually died of the effects of perforation of a diverticulum.

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