STEATORRHOEA DUE TO INTESTINAL RESECTION AND STRICTURES

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The resection of large amounts of small bowel is likely to produce evidence of malabsorption. The clinical picture then encountered differs somewhat from that seen in the sprue syndrome[9] and in those conditions where stagnation of intestinal contents (e.g. blind loops, multiple strictures) is responsible for the steatorrhoea.[8, 10] In the case now presented several factors were combined: he suffered from massive resection, a blind loop (thought not to be significant) and intestinal strictures, with the added complication of mucosal ulceration. Partial relief was obtained from removal of the strictured-ulcerated area, and at present the patient's metabolic status presumably reflects the fact that his residual small bowel consists of only 75 cm. of upper jejunum.

CASE REPORT

W.F., a Coloured male then aged 27 years, was first admitted to this hospital on 20 November 1954, complaining of attacks of abdominal pain and vomiting during the previous 5 years, and recent weight loss. Physical examination revealed dextrocardia (subsequent investigations showed that he had total situs inversus). There was moderate gynaecomastia but the testes felt normal. The blood pressure was 130/80 mm. Hg. There were no abnormal findings in the abdomen. The urine was normal and the blood indices were Hb. 15 g.%, ESR 10 mm. in the first hour (Westergren), and WBC 11,000 per c.mm. The Wassermann reaction was negative. Several stools contained occult blood, but a sigmoidoscopy was negative. The urinary 17-ketosteroid excretion was 8·3 mg. in 24 hours.

During an attack of pain the abdomen became distended, with increased bowel sounds; a plain X-ray film at that time showed fluid levels in the small bowel. Laparotomy was performed: a 5-cm.-long constriction was found 15 cm. from the ileo-caecal valve, and 25 cm. of terminal ileum were resected; histology (Dr. J. A. H. Campbell) was that of regional enteritis (Crohn's disease).

The patient did not remain well for long, and he was readmitted on 26 January 1955 with severe abdominal pain. At operation there was localized peritonitis in the ileo-caecal region, loops of ileum being matted together. An unstated length of small bowel was resected, together with the caecum, and an anastomosis was made between the end of the remaining small intestine and the side of the ascending colon. Pathological examination (Dr. M. Sacks) again showed regional enteritis; there were fistulae between the loops of intestine.

Since this operation he has suffered from diarrhoea. At first his bowels acted up to 15 times a day, but after 3 months they became less active. The stools are pale, bulky and offensive. He entered hospital again on 21 May 1957, because the diarrhoea had become worse during the preceding 9 months and because he had developed abdominal cramps and distension and dyspnoea on effort. He weighed 96 lb. and was very pale. His fingers were slightly clubbed. There was a grade-3 blowing systolic murmur, maximal at the apex of the heart. The blood pressure was 130/80 mm. Hg. The gynaecomastia was unchanged and the testes were normal. The abdomen was distended; the liver edge could be felt 5 cm. below the left costal margin. The urine was normal. The blood indices were Hb. 5 g.%, VPC 20%, ESR 1 mm. and WBC 5,000 per c.mm.; the peripheral blood smear was hypochromic. Some of the relevant investigations are listed in Table I. The blood urea and electrolytes were normal. On electrophoresis, the serum-protein pattern was normal. Apart from the evidence of dextrocardia, the electrocardiogram was within normal limits. Uropepsin excretion was 4 units per hour and he secreted 10·6 mEq. of free HCl in 1 hour in an augmented histamine test (both normal). Bacteriological examination of the stools was negative, but they contained occult blood. The bone marrow was considered to be basically normoblastic, with evidence of iron deficiency, but several cells resembling megaloblasts were seen. A liver biopsy was reported (Dr. W. B. Becker) as showing marked fatty change. Barium studies confirmed the situs inversus; both enema (Fig. 1) and meal (Fig. 2) showed a narrowed segment of small bowel near the anastomosis, and the meal showed flocculation.

TABLE I

	Period		
	I	*II*	*III*
Serum			
Proteins (g.%)			
Albumin	3·9	4·3	3·9
Globulin	1·6	2·5	2·8
Cholesterol (mg.%)	109	250	101
Calcium (mg.%)	7·4	11·5	9·6
Phosphorus (mg.%)	4·2	4·2	
Urine			
Calcium (mg./24 hours)	63	105	52
17-ketosteroids (mg./24 hours)	3·6		6·1
Tests of Absorption			
Xylose (g. excreted/5 hours)	3·6		2·7
Fat (%) (3-day balance, 70 g./day)	65	33	56
Glucose tolerance curve	Flat	Flat	Flat

Periods
I: Pre-operative, May 1957.
II: Soon after operation, August.
III: 4 months after operation, November.

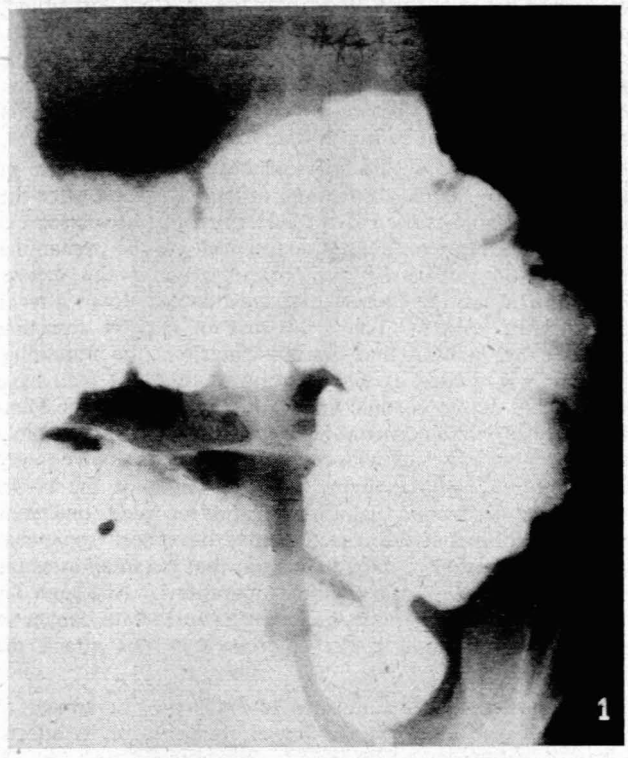

Fig. 1. Barium enema.

The anaemia did not respond to the parenteral administration of vitamin B12 and folic acid. He received a high-calorie diet, vitamin supplements, oral chlortetracycline and blood transfusions, and his weight rose to 109 lb. At this stage the cardiac murmur was no longer audible.

On 15 July 1957 Dr. D. J. du Plessis performed a laparotomy. The small bowel was found to be only 90 cm. long, and to be dilated and hypertrophied. Proximal to the entero-colostomy site was a blind loop of ascending colon, 15 cm. long. In the jejunum were 2 ring-like narrowings 5 cm. apart, the distal one being 4 cm. from the anastomosis. The distal 15 cm. of small bowel were resected, with the blind pouch of ascending colon, and an end-to-end jejuno-colostomy was made; approximately 75 cm. of small intestine were left behind.

The mucosa at the site of the strictures was ulcerated. Histologically, again, the picture was that of regional enteritis (Dr. C. J. Uys). He made a good recovery from the operation. The bowel actions have decreased in number from 6-7 a day soon after the operation to 3-4, 4 months later. When re-assessed in November 1957 his weight was maintained at 110 lb., and he was neither dyspnoeic nor anaemic (Hb. 15 g.%, smear normochromic). The blood pressure was 120/80 mm. Hg. He had not suffered from a recurrence of the abdominal pain. The electrolytes and blood urea were normal. A barium meal with the usual type of suspension again showed clumping; with a non-flocculating medium the mucosal pattern was normal and the jejunum was slightly dilated. Other data are recorded in Table I.

DISCUSSION

The history at the time of the patient's admission in May 1957 suggested that he had steatorrhoea, and this was confirmed by the fat balance. The biochemical tests (flat glucose tolerance curve, low xylose absorption) and the flocculation-pattern on barium meal indicated an enterogenous cause.[9] The barium studies also showed a narrowing proximal to the entero-colonic anastomosis; as the previous operations had been for regional enteritis, a recurrence was thought to have produced strictures. In view of the positive tests for occult blood in the faeces, indicating mucosal ulceration, active enteritis was diagnosed. The abdominal pain was thought to be due to partial intestinal obstruction. Laparotomy confirmed these impressions.

Multiple intestinal strictures cause steatorrhoea by producing a 'stagnation syndrome'.[6, 9] Megaloblastic anaemia is often found; in the present case the bone marrow was probably normoblastic and the anaemia did not improve on treatment with vitamin B12 and folic acid. It was obviously due largely to blood loss, but malabsorption may have contributed to the picture.

The post-operative course has shown conclusively that the strictures were not the principal cause of the steatorrhoea; in fact, 4 months after the operation the fat absorption was lower than it had been prior to surgery. Obviously, the extensive intestinal resection must be incriminated. Had his intestinal length been adequate, excision of the strictured area would have caused the fat absorption to return to normal, as occurs after restoration of proper bowel continuity in the blind-loop syndrome.[10]

What did surgery achieve in this patient? (1) The source of blood loss has been removed. (2) He no longer suffers from intestinal obstruction. (3) The strictures can no longer interfere with absorption (assuming that they might have done so).

The radiological sign of clumping of barium has been said to occur only in those cases of steatorrhoea which are due to

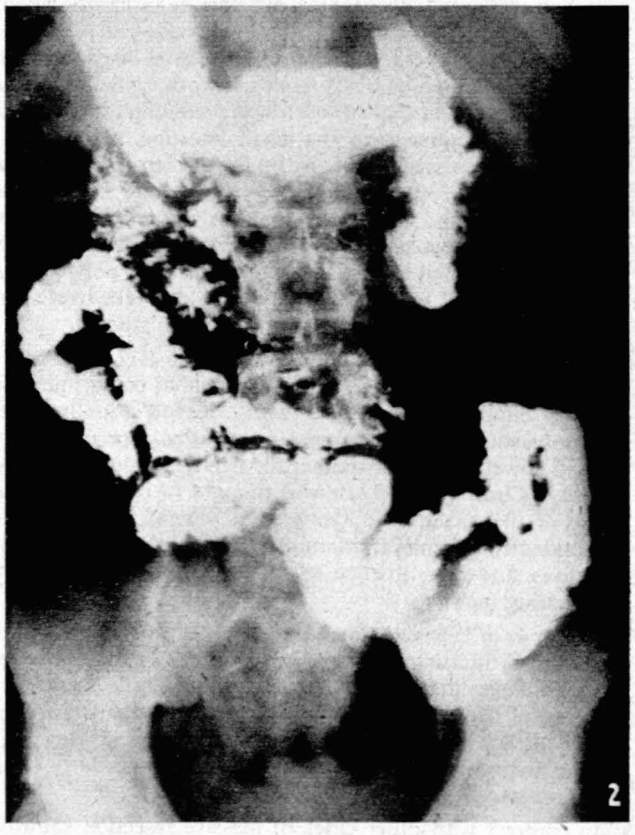

Fig. 2. Barium meal.

sprue, Whipple's disease and intestinal lymphoma;[12] it is thought to be due to flocculation of the barium by mucus secreted in response to the presence of unabsorbed short-chain fatty acids. Gardner[6] did not find this sign in blind-loop steatorrhoea, and one would therefore not expect it in the analogous condition of multiple strictures. Changes in the mucosal pattern with non-flocculating barium are probably of greater significance in the radiographic investigation of malabsorption.

Regional enteritis has been thought to cause steatorrhoea on the basis of extensive bowel disease, but it is likely that localized strictures are more often to blame. Support for this suggestion is obtained from a study by Ensrud and Sauer,[5] who divided their 38 medically-treated cases of regional enteritis into 3 groups, according to the amount of bowel involved. Of the 11 patients in whom less than 12 inches of the bowel was affected, 4 had a malabsorption syndrome, whereas of the 19 in whom more than 36 inches was diseased, or in whom there were skip lesions or internal fistulae, steatorrhoea was noted in 5. Only 1 of the 8 cases in the intermediate group suffered from malabsorption.

Jackson[7] states emphatically that metabolic disturbances are rare in patients in whom less than 2/3rds of the small bowel has been resected, provided that the remaining segment is healthy. In the present patient the possibility of recurrent enteritis must always be a hazard, still further to deplete his limited absorptive capacity. His residual bowel length approaches the danger mark; with less than 18 inches, the prospect is grim.[7] Pietz[13] notes that patients retaining a segment of ileum appear to do better than those having an equivalent length of jejunum, and suggests that the ileo-caecal valve may be the important factor. An alternative explanation may be deduced from the work of Benson *et al.* in rats;[1] they found that olive oil was preferentially absorbed from the third quarter of the small intestine. However, a recent study of normal humans by Borgström *et al.*[3] contradicts this thesis; the intestinal contents were sampled at various levels, and it appears that fat, protein and carbohydrate are almost completely absorbed within 100 cm. of the duodeno-jejunal flexure. In spite of this, it is possible that lower segments of small bowel may be able to take over some of these functions—this aspect remains to be studied.

According to Booth and Mollin[2] vitamin B12 is absorbed from the ileum, and not from the proximal small intestine. This is difficult to reconcile with Jackson's finding that megaloblastic anaemia does not occur after experimental or clinical bowel resection, whether the ileum has been removed or not.[7] Chronic blood loss was thought to be the principal cause of the anaemia in the present patient, who had not responded to parenteral vitamin B12 and folic acid, but the strictures and the absence of the ileum may have been aggravating factors.

Linder *et al.*[11] have defined the effects of massive resection of the small intestine as *undernutrition with special adaptation*. At this stage the present patient shows little secondary evidence of depletion and is in a better nutritional state than before his latest operation. When he presented in May 1957 he had hypocalcaemia, hypoproteinaemia and a fatty liver, but there were no signs of vitamin-B deficiency.

In contrast with other cases of massive resection showing the undernutrition-adaptation syndrome, the patient under discussion has a normal blood pressure, has not lost libido, does not crave salt, has a normal serum-potassium level, shows no evidence of psychological deterioration, and is able to maintain his weight. His urinary 17-ketosteroid excretion is higher than it was before the strictures were resected, but slightly lower than it had been when he first attended in 1954.

In spite of the severe steatorrhoea he shows signs of improvement—his fat absorption, at its nadir soon after the latest operation, has risen to 56%. His post-operative progress may be due to continuing adaptation of the remaining intestine; this must also have occurred after the second operation, when the intense diarrhoea settled down after a few months. As in 'Toni', the case of massive resection reported by Jackson and his associates,[8, 11] the remaining jejunum was dilated and hypertrophied, but this may have been partly due to chronic intestinal obstruction. The blind loop probably had no metabolic significance as it presumably was able to empty itself with peristalsis[4] and consisted entirely of colon. It is unfortunate that the length of the bowel removed at the second operation was not recorded; one gains the impression that the resection may have been somewhat generous. There is nothing to suggest that his small intestine was abnormally short before the operations. Although his ultimate prognosis is poor, he is not in immediate danger of death from inanition if the enteritis does not attack the remaining bowel.

The gynaecomastia and situs inversus do not appear to have any relationship to the bowel condition or its effects.

SUMMARY

A patient who had previously undergone extensive bowel resections for regional enteritis is discussed. He was studied while suffering from inflammatory strictures in his remaining 90 cm. of jejunum, and again after the diseased area had been excised. He showed the features of enterogenous steatorrhoea, anaemia due to blood loss, and partial intestinal obstruction. Resection of the affected bowel, in which the pathological process was recurrent regional enteritis, cured the latter two complaints. It was not possible to evaluate the role the strictures played in the production of malabsorption.

I wish to thank Dr. Louis Mirvish for permission to report this case. Dr. W. P. U. Jackson kindly allowed me to read his contribution to Modern Trends in Gastro-enterology[7] in proof. The cooperation of Dr. Velva Schrire and Dr. D. J. du Plessis is greatly appreciated. Mr. B. Todt made the reproductions of the X-ray plates.

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