

POST-GASTRECTOMY STEATORRHOEA DUE TO INTESTINAL LACTASE DEFICIENCY

S. BANK, M.B., CH.B., M.R.C.P. (LOND.), *Consultant Gastroenterologist*, G. O. BARBEZAT, M.B., CH.B., *Research Assistant*, AND I. N. MARKS, B.Sc., M.B., CH.B., F.R.C.P. (EDIN.), *Consultant Gastroenterologist; From the Gastro-Intestinal Service, Groote Schuur Hospital and Departments of Medicine and Paediatrics, University of Cape Town, and Groote Schuur Hospital, Observatory, CP*

Steatorrhea following partial gastrectomy may be associated with a disturbing loss of weight, osteoporosis, hypocalcaemia and anaemia due to the continued loss of fat, proteins, minerals and vitamins. Many factors have been incriminated in the production of the steatorrhea, but a definitive aetiological factor is not always demonstrable. Treatment ranges from a conservative medical approach with constipating agents, a trial of pancreatic replacement therapy and antibiotics, to surgical measures such as enlarging the gastric reservoir or re-establishing gastro-duodenal continuity.

The purpose of this paper is to report an unusual type of post-gastrectomy steatorrhea which is readily amenable to simple dietary measures. The steatorrhea was associated with an intolerance to dairy products and virtually disappeared on eliminating the latter from the diet.

CASE REPORT

The patient, a 49-year-old housewife, was admitted with a history of diarrhoea, loss of weight, flatulence and malaise following a vagotomy and partial gastrectomy carried out for intractable duodenal ulceration 9 months previously. The diarrhoea developed in the immediate postoperative period and was characterized by the passage of up to 10 loose, frothy, yellowish stools per day. The bowel actions tended to occur 20-30 minutes after a meal and were frequently accompanied by episodes of vomiting, headaches and extreme lethargy. Weight loss was a feature, the patient's weight having fallen from 167 to 120 lb. She was referred to the Gastro-Intestinal Service after initial treatment with anticholinergics and antacids had failed to influence her symptoms. Further questioning showed that the diarrhoea was of a particularly explosive nature after eating cheese, butter or milk with her meals. She had, in fact, omitted dairy products from her diet intermittently with marked clinical improvement. There was no history of sensitivity to dairy products before her operation and there was no childhood history of diarrhoea. Clinical examination was non-contributory apart from slight weight loss and mild hypertension of the order of 160 mm.Hg systolic and 100 mm.Hg diastolic. The haemoglobin was 13.5 G/100 ml., PCV 44%, ESR 7 mm./hr. (Westergren) and the WBC 7,200/cu.mm. Urinalysis was normal.

Laboratory investigations showed that the serum proteins, liver-function tests, blood urea, serum bilirubin and alkaline phosphates were normal. An augmented histamine test revealed a histamine-fast achlorhydria. Stool examinations were repeatedly negative for pathogens, and a culture of bile obtained at jejunal biopsy showed nasopharyngeal bacteria only. Urinary $\text{Co}^{57}\text{B}_{12}$ excretion was normal at 20.1 G/100 ml. of the administered dose after 24 hours and the serum B_{12} and folate levels were within normal range. Peroral jejunal biopsy carried out during a phase of marked diarrhoea yielded morphologically normal jejunal mucosa. Disaccharidase determinations were carried out on a 50 mg. specimen of jejunal mucosa after the method of Auricchio.¹ The disaccharidase activity per G of protein was lactase 2 units, sucrase 151 units and maltase 598 units (Table I).

TABLE I. DISACCHARIDASE ACTIVITY IN SMALL BOWEL MUCOSA IN CONTROL SUBJECTS AND A PATIENT WITH POST-GASTRECTOMY STEATORRHOEA

Author	Lactase (units/G protein) range	Sucrase (units/G protein) range	Maltase (units/G protein) range
Sonntag <i>et al.</i> ² ..	6-258	24-325	70-1,120
Haemmerli <i>et al.</i> ³ ..	7-285*	49-325	217-1,120
Sheehy & Anderson ⁴ ..	4-149	21-247	52-816
Barbezat <i>et al.</i> ⁵ ..	64-108	150-389	220-1,325
Present case	2	151	598

*Haemmerli *et al.*³ consider 7-20 units as abnormally low activity in subjects free of gastro-intestinal symptoms.

A glucose-tolerance test was carried out following the oral administration of 50 G of glucose and the test was repeated, on alternate days, using 50 G of sucrose, maltose and lactose respectively. The tolerance test following maltose showed the typical post-gastrectomy alimentary hyperglycaemia. The latter was also apparent, but to a lesser degree, after glucose and sucrose. These curves contrasted sharply with the flat tolerance test following lactose (Fig. 1).

Fat absorption was measured by means of labelled triolein and biochemical methods. The stool radioactivity and faecal fat content were determined on each of the 2 successive 5-day

collections. The daily fat intake during both test periods ranged from 70 to 120 G. Dairy products were eliminated from the diet during the first period but given freely in the second.

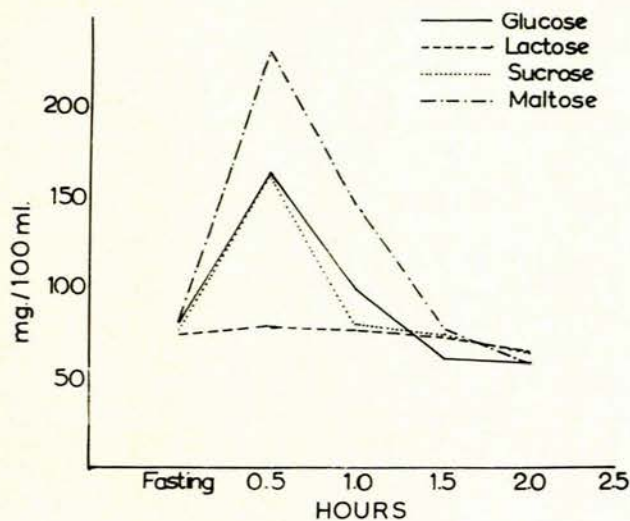


Fig. 1. Glucose-tolerance curves after the administration of 50 G of disaccharide in patient with post-gastrectomy steatorrhoea.

The stool radioactivity and faecal fat was 8.0% of the administered dose and 3.4 G/day respectively during the first collection period, and 19.2% of the administered dose and 14.4 G/day respectively during the second period (Fig. 2).

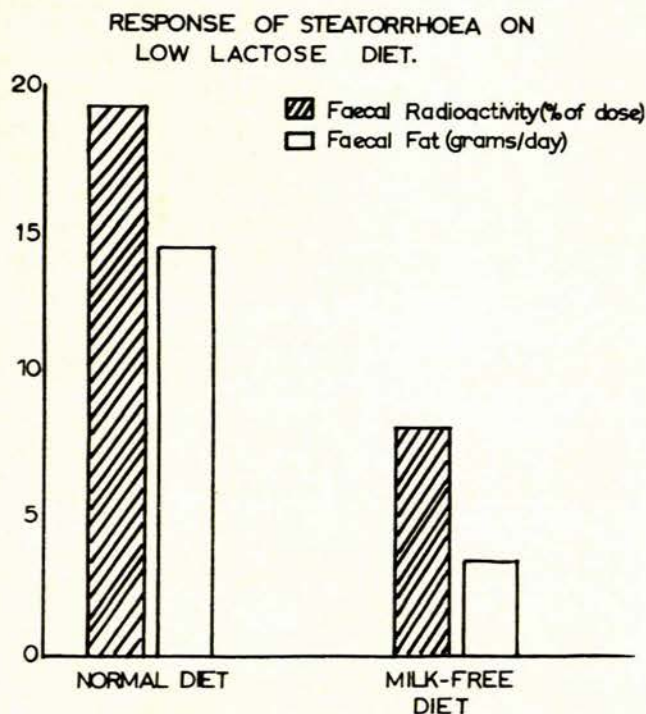


Fig. 2. Response of steatorrhoea on a low-lactose diet in patient with post-gastrectomy lactose intolerance.

The patient became asymptomatic on a high-protein and moderate fat and carbohydrate diet with a low lactose content. There was a progressive increase in weight, the patient gaining 10 lb. in 3 weeks.

DISCUSSION

Clinically, lactose intolerance is characterized by the development of diarrhoea, abdominal cramps and flatulence after the ingestion of milk. The stools are usually frothy or watery in appearance, but steatorrhoea has been reported in a number of children and infants with lactose and other disaccharide intolerance.⁸⁻⁹ The development of steatorrhoea in adults with milk intolerance would appear to be uncommon and only 1 case of the association could be found in the literature.

Kern *et al.*¹⁰ described a patient with mild milk intolerance which became very much more severe following resection of the ileum and ascending colon for an internal hernia. The steatorrhoea which occurred after operation in their patient could be increased by the administration of milk or lactose and fat absorption was improved by the removal of these products from the diet. The clinical diagnosis of lactose intolerance was supported by the finding of a flat glucose-tolerance curve after the administration of lactose. The lactase activity in a jejunal mucosa biopsy appeared to be low but technical difficulties precluded an exact quantitative estimation.

Our patient shared many of the features of the case reported by Kern *et al.* Clinical steatorrhoea, occurring for the first time after gastrectomy for a duodenal ulcer, was subsequently shown to be due to milk intolerance. The flat glucose-tolerance test after 50 G of oral lactose, the low jejunal mucosal lactase activity and the clinical and biochemical remission of steatorrhoea when milk products were eliminated from the diet suggested that the steatorrhoea was directly related to the impaired intestinal handling of lactose, in a bowel compromised to some extent by the effect of resection or short-circuit.

Steatorrhoea may occur after partial gastrectomy for a variety of reasons. While an afferent loop component could not be excluded with certainty in the present case, the fact that clinical and biochemical steatorrhoea recurred whenever milk products were reintroduced pointed strongly to lactose intolerance being the dominant aetiological factor in the steatorrhoea. Malabsorption of fat has not been noted previously in patients with lactose intolerance after gastrectomy.^{11,12}

Most authors agree that the diarrhoea associated with disaccharide intolerance results from irritant organic acids produced by bacterial degradation of non-hydrolysed disaccharide in the large bowel.³ It is conceivable that the presence of bacteria in the small bowel of such patients may cause similar changes in the jejunum or ileum; the organic acids may interfere with fat hydrolysis and absorption and thus lead to steatorrhoea. In this connection it is of interest that the operative procedures in the present case and in that reported by Kern *et al.* both predispose to bacterial invasion of the small bowel. Bacterial decomposition of excess free lactose may therefore have taken place at a small bowel level in both these patients. The non-contributory small bowel culture in the present case does not negate this possibility.

A history of milk intolerance before gastrectomy was not elicited in the present case and the question whether the low mucosal lactase activity existed before gastrectomy or occurred as a result of the operation cannot be answered. Low lactase activity has been reported in

16-55% of subjects free of gastro-intestinal symptoms.^{4,13} On the other hand, symptomatic lactose intolerance occurs in a wide variety of disorders including gastro-intestinal resection, infection, granulomatous conditions, protein malnutrition and congenital abnormalities.¹⁴ Whether these conditions inhibit or interfere with intestinal disaccharidase activity temporarily or merely serve to highlight a pre-existing low mucosal disaccharidase state has not yet been clarified. It should be stressed that the intestinal mucosa in most of these conditions is morphologically normal on light microscopy, and that a return of low mucosal disaccharidase levels to normal values after an apparent cure of the symptoms has not been recorded to date. These findings suggest that patients with an inherently low level of lactase activity respond to a variety of gastro-intestinal insults by developing symptoms of lactose intolerance. Prolonged milk restriction may be necessary in some patients, and in others the symptoms of lactose intolerance may remit spontaneously with the successful treatment of the precipitating disease.

The steady weight gain that occurred in our patient after the steatorrhoea was contained by a high-protein, moderate-fat and low-lactose diet, implied that the lactose-induced steatorrhoea following gastrectomy carries with it all the risks of the more usual causes of post-gastrectomy steatorrhoea.

SUMMARY

A case of post-gastrectomy steatorrhoea due to lactose intolerance is reported. Low mucosal activity was found on

disaccharidase determinations of a peroral jejunal biopsy specimen. A clinical and biochemical remission in the steatorrhoea occurred when dairy products were eliminated from the diet.

The pathogenesis of the steatorrhoea is considered and it is suggested that a variety of factors may convert a previously occult lactose intolerance to the overt manifestations of the disease in patients with inherently low mucosal lactase activity.

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