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Leading article

Postvagotomy diarrhoea: is there a place for surgical management?

Postvagotomy diarrhoea is now uncommon because surgical treatment of peptic ulcer disease is rarely needed and highly selective vagotomy has superceded truncal vagotomy and drainage. The two important risk factors are vagotomy in younger patients and additional cholecystectomy.[1 2] Additional cholecystectomy is associated with the highest faecal bile acid output and faecal bile acid concentration (as distinct from output) is elevated above the normal control values.[2]

Definition and incidence

Estimates of the incidence of postvagotomy diarrhoea vary because there is no standard definition. Significant diarrhoea should only be diagnosed when it results in a poor outcome (Visick III-IV) or a curtailment of the patient's activity and social life with watery diarrhoea (> three motions/day) with urgency and occasional incontinence.* The diarrhoea is frequently episodic when it is usually associated with dumping (39/56). Steatorrhoea is rare. The reported incidence of severe diarrhoea varies from 0·5–8%, mean 4%.[3-6]

Aetiology

Postvagotomy diarrhoea is often associated with vasomotor dumping symptoms and because of the coexistent rapid emptying of liquid as distinct from solid meals,[7] a common underlying pathophysiological mechanism may be responsible for both.[8] The primary abnormality may be loss of duodenal regulation of gastric emptying with gastric incontinence, the stomach emptying under the influence of posture and exposing the upper small bowel to a hyperosmolar load especially after a liquid meal. Although important, rapid gastric emptying is not the only factor as severe diarrhoea may be encountered without vasomotor symptoms. Remedial surgery to slow gastric emptying by pyloric reconstruction or take down of a gastroenterostomy often improves the vasomotor dumping without materially influencing the severity of the diarrhoea.[9-12] Rapid small bowel transit is a universal finding,[13] perhaps following the hyperosmolar load (secretory diarrhoea).[13] Small bowel motility however is disturbed by vagotomy with a reduction of the fed pattern of activity such that the migratory complexes return early and sweep the intestinal contents rapidly through the small intestine.[14] This study explains why diarrhoea is not relieved after remedial surgery to retard gastric emptying. Rapid transit induces malabsorption with the delivery of a high volume/solute load containing bile salts and hydroxy-fatty acids to the colon and a combined osmotic and secretory diarrhoea. While the faecal bile salt output is elevated,[15] the concentration is within normal limits except in patients after vagotomy and cholecystectomy. Thus a specific bile acid malabsorption has not been demonstrated.

An alternative hypothesis for post vagotomy diarrhoea proposes gall bladder distension with postprandial contraction and delivery of a large bile load into the intestine.[16]

Conservative management

The management of the patients with post vagotomy diarrhoea is difficult. Dry meals and a low fat diet are helpful and may help in a few well motivated and cooperative patients. Codeine phosphate and domperidone are ineffective except in large doses and with strict timing in relation to meals.[17] The benefit of cholestyramine[18] has been confirmed in a number of clinical trials[19 20] in the short term but I have not seen sustained improvement beyond six months in any patients. The poor longterm outcome with cholestyramine may reflect the episodic nature of the condition or poor compliance. Oral preparations of long acting somatostatin analogue have not yet been assessed.

Surgical management

Remedial surgery for the patient with intractable diarrhoea is necessary but should be delayed for at least 18 months because some patients improve by adjusting their diet and eating habits. The whole gastrointestinal tract must be assessed before remedial surgery. Among our 56 patients, four examples of organic disease have been encountered (coeliac disease (two), blind loop, gastroileostomy, and carcinoma of the descending colon). While revision of the drainage procedure often improves vasomotor dumping symptoms, relief of severe diarrhoea, is uncommon. For patients with persistent dumping symptoms and diarrhoea, pyloric reconstruction or take down of the gastroenterostomy should be the first option but patients must be warned that the diarrhoea may persist.

Surgical treatment is also designed to slow small bowel transit.[4] Good results have been reported by the use of an antiperistaltic segment located at 100 cm from the ligament of

*In the author's series of 56 patients.

Treitz[21] but I have not had any favourable results with this procedure. Indeed 10 of 13 patients treated with antiperistaltic jejunal segments have required reversal because of episodes of severe postprandial colic, intestinal obstruction and gross bacterial overgrowth. A better tolerated and more physiological brake is obtained using the distal onlay reversed ileal graft first described by Sadowski in dogs subjected to massive resection of the small intestine.[22] This procedure creates a passive non-propulsive segment, 10–12 cm long some 30 cm from the ileocaecal junction and has been used successfully in patients with severe postvagotomy diarrhoea.[23] It restores the orocaecal transit time after a lactulose meal as estimated by breath hydrogen from 61·0 (12·4) min to near normal 74·4 (5·8).

The most important surgical message is directed at the prevention of this complication. Truncal vagotomy should be avoided and the highly selected operation used whenever possible. Cholecystectomy should be avoided in patients treated previously by vagotomy or gastrectomy. Symptomatic gall stone disease developing in these patients is best managed conservatively. If stones are encountered during surgery, cholecsytolithotomy is a better option than cholecystectomy.

A CUSCHIERI

Ninewells Hospital,
Dundee

Correspondence to: A Cuschieri,
Ninewells Hospital, Dundee DD1 9SY.

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