Management of Postoperative Alkaline Reflux Gastritis

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A VARIETY of undesirable sequelae are known to develop following technically satisfactory operations on the stomach for peptic ulcer disease. These sequelae include diarrhea, afferent loop and stomal dysfunction, the dumping syndrome and nutritional disturbances. Equally important is bilious vomiting which may follow any operative procedure which allows free entry of duodenal contents into the stomach. Minor degrees of bilious regurgitation can often be modified by dietary measures; however, constant reflux of bile and pancreatic juice may produce severe and disabling gastritis. Such symptoms may cause incapacitation and are poorly controlled by dietary or other medical measures. The present report outlines the authors' experiences with 11 patients who underwent surgical correction for severe alkaline reflux gastritis secondary to previous gastric surgery.

Clinical Material

During a 10-year period 11 patients who had all the signs and symptoms of alkaline reflux gastritis underwent exploratory operation (Table 1). There were five men and six women whose ages ranged from 42 to 61 years. Three patients developed symptoms after vagotomy and pyloroplasty, four after gastroduodenostomy, the remaining four had undergone Billroth II gastrojejunostomy. Complaints began 1 month to 13 years after either gastric resection or pyloroplasty. All of these individuals demonstrated a similar clinical picture. Characteristic symptoms included persistent and severe midepigastric or low substernal burning pain generally aggravated by eating or the ingestion of alkalis or milk products. This pain was associated with nausea, post-prandial bilious vomiting, and weight loss. None of these patients complained of diarrhea or vasomotor symptoms traditionally associated with post-vagotomy diarrhea, intermittent afferent loop obstruction or dumping syndrome. Upper gastrointestinal roentgenograms failed to demonstrate either obstruction or rapid transit time and often showed good filling and emptying of both afferent and efferent loops when present. None of the patients were found to have guaiac positive stools, while only two individuals had the characteristic red cell morphology of a hypochromic microcytic anemia on peripheral smear. Gastric secretory studies showed an alkaline basal pH (mean 7.2) with either histamine-resistant hypo- or achlorhydria. Completeness of vagotomy was confirmed by the Hollander test.

Gastroscopy with biopsy was the single most important diagnostic study performed. Gross endoscopic appearance was characterized by a reddened, edematous, granular and friable appearing gastric mucosa often containing punctate or linear erosions. Ample reflux of bile into the stomach, often up into the esophagus, with green colored mucus lakes was a virtually constant feature. Endoscopy was also important in eliminating other entities such as marginal ulceration, stomal, afferent and efferent loop obstruction. In all cases the histologic features of a severe atrophic gastritis were found (Fig. 1). Other microscopic findings included mucosal atrophy, foci of superficial mucosal inflammation, ulceration, submucosal hemorrhage and edema. In addition, biopsies in three cases showed isolated areas of epithelial proliferation and metaplasia.

Presented at the 84th Annual Meeting of the Southern Surgical Association, Boco Raton, Florida, December 4–6, 1972.

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TABLE 1. *Summary of 11 Patients with Alkaline Reflux Gastritis*

Patient	Age & Sex	Underlying Disease	Original Operation	Time Interval Prior to Symptoms	Weight Loss (pounds)	Anti-reflux Operation	Weight Gain (pounds)	Comments	Follow-up
P. T.	50 F	Perforated ulcer	Vagotomy, pyloroplasty, closure of perforation	9 yrs.	26	Hemigastrectomy Roux-Y	19		7 yr. 6 mo.
M. L.	49 M	Bleeding duodenal ulcer	Vagotomy Pyloroplasty Suture ligation	3 yrs. 5 mo.	28	Hemigastrectomy Roux-Y	36	Antacids without relief	5 yr. 6 mo.
J. B.	47 F	Hiatal hernia High acids	Fundoplication Vagotomy Pyloroplasty	1 yr. 4 mo.	32	Hemigastrectomy Roux-Y	21	Temporary relief on cholestyramine	2 yrs.
M. G.	61 M	Gastric ulcer	Vagotomy Antrectomy Billroth I	8 yrs.	59	Roux-Y limb	34	Pulmonary embolus, postoperatively	6 yrs.
A. J.	55 M	Gastric & Pyloric ulcer	Antrectomy Billroth I	4 mos.	21	Roux-Y limb	24	Previous transthoracic vagotomy without relief	3 yr. 9 mo.
C. N.	60 F	Gastric ulcer	Vagotomy Antrectomy Billroth I	2 yrs.	55	Roux-Y limb	43		8 yrs.
A. S.	59 F	Gastric ulcer	Vagotomy Antrectomy Billroth I	18 mo.	36	Roux-Y limb	29		7 yrs.
J. U.	42 M	Perforated duodenal ulcer	Vagotomy, 70% gastric resection Billroth II	13 mo.	54	Roux-Y limb	19	Anemic, antacids without relief, Previous conversion to Billroth I without relief	8 yr. 6 mo.
C. D.	52 F	Bleeding duodenal ulcer	70% gastric resection Billroth II	1 mo.	20	Roux-Y limb	30	Vagotomy for marginal ulcer Cholestyramine without relief	2 yr. 9 mo.
C. B.	53 F	Duodenal ulcer	Vagotomy 70% gastric resection Billroth II	13 yrs.	?	Roux-Y limb	?	Cholestyramine without relief	1 yr.
S. D.	47 M	Duodenal ulcer	Vagotomy 70% resection Billroth II	10 yrs.	42	Modified Tanner Roux-Y	39		3 yr. 8 mo.

Two patients underwent antacid therapy with no relief of symptoms. Three others were given Cholestyramine, a basic anion exchange resin which binds bile salts in the intestine thereby removing them from the enterohepatic circulation. Of the latter, two had no symptomatic response although the course of treatment was short; a third patient (J. B.) had some symptomatic relief with weight gain and gastric mucosal improvement after taking Cholestyramine (4 Gm. t.i.d.) for 6 months.

In three individuals who had undergone a previous vagotomy and pyloroplasty, reoperation consisted of hemigastrectomy with a precolic Roux-en-Y gastrojejunostomy (Fig. 2). The afferent limb was implanted 40 cm. distal to the gastrojejunostomy. In four patients with previous gastroduodenostomy, the Billroth I anastomosis was disassembled, the duodenum was closed and a Roux-en-Y anastomosis was established. The remaining four patients had undergone previous Billroth II subtotal gastrectomy. One patient (J. U.) underwent conversion to a Billroth I gastrectomy. He returned 7½ months later with intractable bilious vomiting and a Roux-en-Y anastomosis was constructed. A second individual (C. D.) underwent vagotomy for a marginal ulcer which subsequently healed. However, 2 years later he was seen again and the diagnosis of alkaline reflux gastritis was made by endoscopy. After a brief trial of Cholestyramine a Roux-en-Y limb was constructed. The remaining two patients had Roux-en-Y gastrojejunostomies 10 and 13 years after the previous gastric operation. One (S. D.) had conversion to a Y anastomosis with a modified jejunostomy as suggested by Tanner[15] (Fig. 3).

There were no deaths. Following operation, one patient developed a pulmonary embolus which successfully responded to medical management. A second patient required reoperation 3 months later because of partial obstruction of the Roux-en-Y limb from adhesions. Follow-up has ranged from one to 8½ years. All of the patients have had dramatic relief of symptoms with a

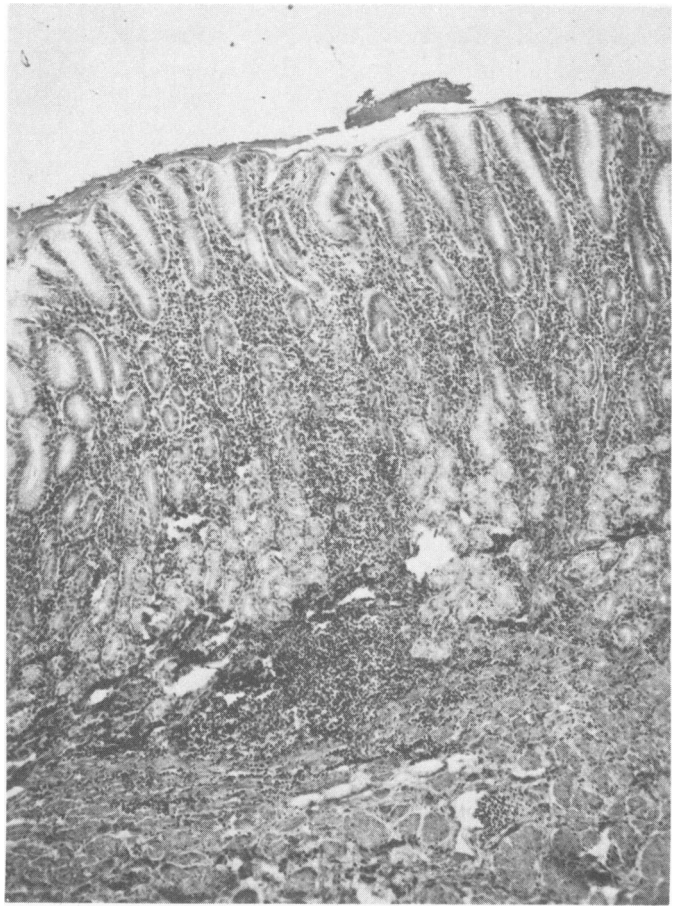

FIG. 1. Photomicrograph of gastric biopsy in a patient with alkaline reflux and atrophic gastritis. Microscopic features include focal mucosal atrophy, lengthened gastric pits and submucosal edema with intense lymphocyte infiltration. Hematoxylin and eosin ×50.

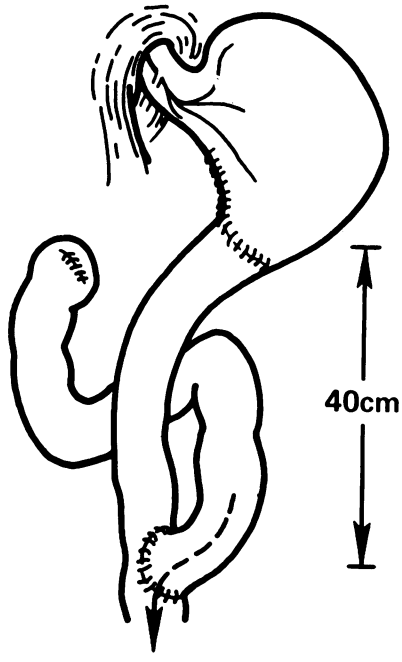

FIG. 2. Diagrammatic representation of the author's technique of precolic Roux-en-Y limb gastrojejunostomy.

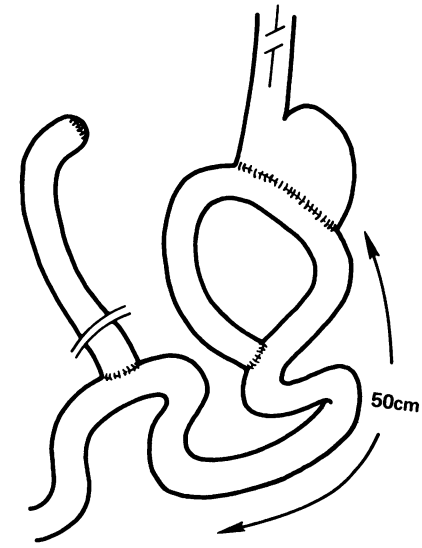

FIG. 3. Tanner's method of modified Roux-en-Y jejunojejunostomy constructed in patients with a previous gastrojejunostomy for relief of alkaline reflux.

19 to 43-pound weight gain. The principal complaint remaining in some patients was early satiety related to the remaining small gastric remnant. Subsequent endoscopy reveals a return to normal of the gastric mucosa. None of the patients had evidence of ulcer formation secondary to diversion of the alkaline stream from the gastric outlet.

Discussion

Bilious regurgitation often associated with persistent epigastric pain is not an uncommon complaint following gastric resection. The relative frequency of bile vomiting and regurgitation varies from 8–35% of patients.[4,19] Most authors have traditionally attributed these symptoms to the "afferent loop syndrome" with stasis of bile and pancreatic secretions in the afferent loop of a gastrojejunostomy followed by the sudden retrograde inflow of duodenal contents into the stomach.[6,7,20] However, this same pattern of symptoms can follow any type of gastric operation in which no afferent loop has been constructed such as a Billroth I gastroduodenostomy or vagotomy and pyloroplasty.[17,18,19]

In the past 20 years the British literature has documented the fact that emesis of duodenal contents may be caused by the accumulation of bile in the stomach rather than in the afferent intestinal loop.[4,15,19] This symptom complex has been called "postoperative alkaline reflux gastritis" and is related to any post-surgical condition in which the pyloric mechanism is either destroyed or bypassed allowing free reflux of duodenal contents into the stomach. Lawson[9] first demonstrated the inflammatory gastric mucosal changes that could occur in animals secondary to duodenal and biliary reflux; later DuPlessis[5] suggested similar changes in man. Schinler[13] by means of repeated gastroscopic examina-

tions subsequently showed that constant reflux of duodenal and pancreatic juice produced gastritis. Siurala and Tawast[14] reported that the incidence of chronic gastritis was significantly increased among patients with bile constantly present in their stomachs. Toye and Williams[17] were able to reproduce these symptoms in two patients who had Billroth II gastrectomies by instilling a quantity of bile and pancreatic secretions directly into the stomach. The symptoms could not be elicited in the same patients by infusing the afferent loop with duodenal aspirate. More recent publications have documented the fact that postoperative reflux alkaline gastritis is probably more common than the older literature would suggest.[1,10,18] Equally current is the demonstration of increased bile reflux into the stomachs of patients with gastric ulcer.[2,12]

Symptoms of bilious vomiting may develop immediately after operation or, for uncertain reasons, after a symptom-free interval of months or years. The epigastric and substernal burning pain is most likely related to the associated gastritis and esophagitis from the irritating alkaline secretions and would explain why antacids are ineffective. Barium studies are of little help except to eliminate marginal ulceration, stomal or efferent loop obstruction. The clinical history, gastric secretory data, and endoscopy with biopsy are the most important diagnostic aids. The uniform finding of histamine-resistant hypochlorhydria or achlorhydria eliminates an acid-pepsin mechanism as the cause for gastritis. Endoscopy documents the free reflux of bile into the stomach. The most severe gastric mucosal changes are usually found at or close to the gastroenteric junction. Chronic atrophic gastritis with diffuse areas of mucosal atrophy and foci of edema and inflammation is the constant histologic feature. In some patients with long-standing symptoms, gastric mucosal biopsies will also show metaplasia resembling the small intestine. This widespread mucosal atrophy can account for the uniformly depressed gastric acid secretion as shown by Kuster.[8] It should also be noted that gastric atrophy is not recognized as a sequelae to vagotomy, further supporting the belief that epithelial damage is directly related to chronic bilious wash of the stomach lining.[11]

In addition to documenting the presence of alkaline reflux gastritis, it is important to meticulously eliminate other more common postgastrectomy sequelae. The dumping syndrome, postvagotomy diarrhea and other nutritional problems can be excluded since increased intestinal transit time and other vasomotor symptoms would be the prominent complaints rather than postprandial biliary emesis. Marginal ulceration is diagnosed either by upper gastrointestinal roentgenograms, endoscopy or at operation. It is also associated with gastric hypersecretion rather than hypochlorhydria and mucosal destruction occurs on the small bowel side of the anastomosis. Gastric stomal dysfunction and afferent loop syndrome usually occur early in the postoperative period and may be diagnosed by gastroscopy or at the time of laparotomy.

The present series emphasizes several points which have not been previously stressed in the surgical literature. Three patients developed alkaline reflux after pyloroplasty. Most instances have developed after gastric resection or gastroenterostomy.[10,15,18] Bartlett[1] reported the case of a single patient with alkaline reflux after vagotomy and pyloroplasty while four other patients developed symptoms after partial gastric resection. Our findings serve to support the concept that reflux of duodenal contents can occur whenever the pyloric sphincter mechanism is damaged or bypassed. Guaiac-positive stools and iron-deficiency anemia with occult blood loss were not uniformly present. Only two of our patients had low hemoglobin and serum iron levels with hypochromic microcytic anemia. Both of these individuals had undergone Billroth II gastrectomy. Recent reports by Van Heerden[18] and Mackman[10] have stressed the presence of anemia and guaiac-positive stools as important diagnostic findings. The present report would suggest that Cholestyramine therapy is relatively ineffective in controlling the symptoms of alkaline reflux. However, one of our patients did receive temporary symptomatic relief and some mucosal improvement. As more extensive studies are undertaken in the use of Cholestyramine, they may prove that this drug therapy needs to be instituted earlier or may be more effective in milder cases of alkaline reflux.

A variety of surgical procedures have been proposed in the treatment of this condition.[4,16] The most satisfactory method has been construction of a Roux-en-Y limb to prevent reflux. Tanner[15,16] has described a modified Roux-Y loop (Fig. 3). In patients with Billroth II gastrectomy, he divides the afferent loop leading to the gastrojejunostomy and implants both ends into the jejunum beyond the stomach. The afferent loop is anastomosed 50 cm. below the gastrojejunostomy making further reflux virtually impossible. The second outlet from the gastric pouch provides an additional outlet from the stomach in case the original efferent limb fails to function properly. The importance of at least a 40–50 cm. Roux-en-Y limb has been emphasized by Bartlett[1] who found that a shorter distance failed to prevent antiperistaltic reflux into the gastric remnant. This method appears applicable primarily in patients with Billroth II gastrojejunostomy. We have utilized this operation in a single patient primarily to shorten the afferent limb at the time of reconstruction. Both Van Heerden[18] and Bartlett[1] have reported great success with this technic. We have favored hemigastrectomy and antecolic

Roux-en-Y limb gastrojejunostomy in those individuals who develop reflux after pyloroplasty (Fig. 2). In symptomatic patients with Billroth I gastroduodenostomy or Billroth II gastrojejunostomy a single limb Roux-en-Y efferent limb of 40 cm. is constructed. If a vagotomy has not been performed previously, this is also recommended. Mackman[10] has also reported good results in four patients with this type of efferent limb.

An increased risk of stomal or jejunal ulceration must be considered following this procedure due to diversion of the alkaline stream. However, this risk appears to be overestimated since all patients generally undergo vagotomy and high gastric resection. We have not noted stomal ulceration in any of our patients, some of whom have been followed for more than 8 years. This has been the experience of Tanner[16] and others.[4,10,18] It appears to be important to perform bilateral truncal vagotomy even in the presence of histamine-resistant achlorhydria. Capper and associates[3] have noted a return of acid secretion after alkaline diversion by a Roux-en-Y loop. Repeat endoscopy with biopsy in some of our long-term patients has suggested that a more normal appearing gastric mucosa develops once reflux ceases.

Summary

The clinical, endoscopic and gastric secretory findings in 11 patients are reported in whom the diagnosis of alkaline reflux gastritis was made. Bilious vomiting with persistent epigastric and substernal pain and weight loss developed after various gastric operations. Endoscopic visualization of alkaline reflux and biopsy-proven atrophic gastritis in addition to histamine resistant hypochlorhydria or achlorhydria confirms the diagnosis. The importance of eliminating other postoperative digestive sequelae has been stressed. Dramatic relief of symptoms was produced by construction of a modified Roux-en-Y jejunal limb allowing the afferent loop to enter the jejunum at least 40 cm. distal to the gastrojejunostomy.

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