Perforated Duodenal Ulcer Managed by Proximal Gastric Vagotomy and Suture Plication

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Twenty-one patients with acute perforated duodenal ulcer were managed by proximal gastric vagotomy without drainage and simple closure of the perforation reinforced with an omental patch. There was no operative mortality. No recurrent duodenal ulcers have developed. All patients have achieved a good to excellent clinical result from their operation. Dumping, diarrhea, and reflux gastritis have not developed. Follow-up studies extend to three and one-half years. Proximal gastric vagotomy with simple closure is safe, effective management for the patient with an acute perforated duodenal ulcer. This operation is a satisfactory compromise between simple closure alone which does not protect against recurrent ulcer and definitive ulcer operations which may subject patients who would not have further ulcer symptoms to the unnecessary risk of increased mortality, morbidity, and postgastrectomy disorders.

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SURGICAL RATHER THAN non-operative treatment is accepted for the treatment of acute perforated duodenal ulcer, but controversy exists between proponents of suture plication (simple closure) and surgeons who prefer a definitive operation to control the ulcer diathesis at the initial operation. Following simple closure of a perforated duodenal ulcer, only one in four patients[4] remains completely and permanently free of ulcer symptoms. Definitive operation as initial management, however, subjects some patients to an unnecessary operation to control their ulcer diathesis. Some of these patients may unfortunately develop "postgastrectomy sequelae" of dumping, diarrhea or reflux gastritis.

Proximal gastric vagotomy has been reported to control the ulcer diathesis and reduce postgastrectomy complications.[2] The operation has a very low mortality and morbidity rate. This study was undertaken to evaluate the effectiveness of proximal gastric vagotomy without drainage as a definitive operation in association with simple closure in patients having acute perforated duodenal ulcer.

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Clinical Material

During the past three and one-half years, the authors have performed proximal gastric vagotomy and simple closure in 21 patients who presented with an acute perforation of a duodenal ulcer. There were 17 men and four women. The age range was from 22 to 88 years with more than one-half of the patients being less than 50 years of age.

A known duodenal ulcer or typical duodenal ulcer symptoms had been present in 13 patients (62%) prior to perforation. Most patients were seen within 12 hours after the onset of symptoms, but two patients were operated upon one and three days after perforation. The patient with a duodenal ulcer perforation of three days duration was in the hospital recovering from a lobectomy for carcinoma of the lung. The diagnosis was made when pneumoperitoneum was shown by x-ray examination.

Operative Technic

After preoperative preparation consisting of nasogastric suction, correction of hypovolemia and electrolyte imbalance, and initiation of antibiotic therapy, emergency operation is performed through an upper midline incision. After identifying the site of the perforated duodenal ulcer, simple closure of the perforation reinforced with an omental patch as advocated by Graham[7] is performed using interrupted silk sutures. The peritoneal cavity is then carefully and thoroughly irrigated with a large volume of sterile saline.

Proximal gastric vagotomy is then performed using the technic described by Goligher.[5] No attempt is made to map out the parietal cell area by pH probe or dye studies; rather the antral-parietal border is determined anatomically by visualizing the insertion of the branches of the anterior nerve of Latarjet onto the lesser curvature of the stomach (the "Crow's foot"). This point is usually 7–10 cm proximal to the pylorus and coincides with the junction of gastric antrum and parietal cell mass. The dissection begins proximal to the "Crow's foot" and continues cephalad opening the anterior layer of the gastrohepatic omentum and dividing all tissue entering the anterior wall of the stomach along the lesser curvature. The neurovascular bundles are each doubly clamped with fine curved forceps, divided and ligated with silk. Only very small bites of tissues are incorporated in the clamps. Dissection continues up to the cardia. The posterior layer of the gastrohepatic omentum is then freed from the lesser curvature of the stomach beginning proximal to the insertion of the posterior nerve of Latarjet onto the antrum.

As the dissection approaches the gastroesophageal junction, the peritoneum overlying the distal esophagus is incised in a transverse direction, and the abdominal esophagus is encircled with a tape or narrow Penrose drain. It is important to continue the dissection cephalad onto the abdominal esophagus for a distance of approximately 5 cm above the angle of His. Coursing along the left lateral wall of the esophagus is a constant vagal nerve fiber that enters the fundus of the stomach. This nerve arises high from the posterior nerve of Latarjet and may be missed unless the dissection is extended upward onto the abdominal esophagus. Professor Guessipi Grassi has referred to this vagal fiber as "the criminal nerve".[8]

After skeletonizing the distal 5–7 cm of the abdominal esophagus, a very superficial circumferential incision is made around the esophagus with a #15 scalpel blade to divide any fine longitudinal nerve fibrils that run distally to the proximal cardia.

As a final step the anterior and posterior walls of the lesser gastric curve are plicated with interrupted fine silk sutures from the esophagogastric junction distally to the insertion of the nerves of Latarjet onto the antrum.

No pyloroplasty or other gastric drainage procedure is performed. The gastric antrum remains vagally innervated via the nerves of Latarjet, but the proximal parietal cell mass is vagally denervated. The abdominal midline incision is closed in anatomical layers without using peritoneal or wound drains. Patients are maintained on nasogastric suction and intravenous fluids until intestinal peristalsis resumes and a liquid diet is tolerated (usually three days).

Results

There has been no operative mortality in the patients who had their acute perforated duodenal ulcer managed by simple closure and proximal gastric vagotomy without drainage. One late death occurred in the patient who had carcinoma of the lung; but he had no complications from the operation for treatment of his perforated duodenal ulcer.

One patient had a non-fatal pulmonary embolus after operation. His perforated duodenal ulcer occurred soon after a traumatic amputation of his lower extremity. Two patients developed superficial wound seromas after operation, but their hospital stay was not prolonged.

Follow-up studies from 6 months to three and one-half years are available on these patients. Eleven patients have been followed from one to two years, five from two to three years and one for three and one-half years. No proven or suspected recurrent duodenal ulcers have developed.

One patient who had been hospitalized for cirrhosis of the liver, ascites and pancreatitis was found to have a lesser curvature 0.5 cm gastric ulcer at the angularis as well as a perforated duodenal ulcer. His perforated ulcer was managed by Graham closure and proximal gastric vagotomy. The gastric ulcer was not treated. Gastroscopy performed one month after operation showed that the duodenal ulcer was healed but the gastric ulcer was still present. Repeat gastroscopy three weeks later showed no gastric or duodenal ulcer. This patient has been followed more than two years with annual gastroscopy. The duodenum appears normal and the gastric ulcer on the lesser curve has remained healed, but superficial erosions suggesting gastritis have been noted on the greater curvature of the stomach. The patient continues to be an alcoholic abuser.

A 43-year-old man is of interest because he had been taking prednisone for five years for treatment of rheumatoid arthritis prior to perforation of his duodenal ulcer. After closure of his perforation and proximal gastric vagotomy, he has been maintained on prednisone 5 mg daily for more than two years. He has had no symptoms of peptic ulcer disease. His basal acid hourly output is 1.8 mEq, and his Hollander test is negative.

"Postgastrectomy sequelae" have been minimal. Two patients had epigastric fullness for three weeks after operation. One patient had mild diarrhea for the first month after operation, but is asymptomatic now.

No patient has symptoms of dumping syndrome, and none has reflux gastritis. The overall clinical results from the operation are considered excellent in 18 patients and good in three.

No preoperative gastric analysis studies could be obtained because the patients presented with acute perforated duodenal ulcer. Five patients have had postoperative gastric analysis studies. The basal acid output has ranged from 0.1 to 2.0 mEq/hr. All five patients have negative Hollander tests.

Discussion

Attempts have been made preoperatively to distinguish between patients who may need definitive operation from those who need only simple closure. Greco and Cahow[9] recommend definitive operation be limited to male patients between the ages of 40 and 64 who have a history of chronic duodenal ulcer disease prior to perforation. In a recent review of our experience with patients having perforated duodenal ulcer, we advocated definitive operation only for good-risk patients with a history of previous duodenal ulcer disease.[16] At that time we reported four patients managed by simple closure of their perforated ulcer and proximal gastric vagotomy. Since these patients did well with no incapacitating side effects from their definitive operation, we continued using this procedure for patients with perforated duodenal ulcer.

Jordan[15] has recently reported his experience with treatment of 157 patients with perforated duodenal ulcer. Forty patients were managed by simple closure alone because of the patient's condition. Other patients were randomized between vagotomy-hemigastrectomy and vagotomy drainage. Reoperation was required in three patients after vagotomy-drainage and in one patient after vagotomy-hemigastrectomy. The frequency of postoperative gastric complaints was essentially the same in each group. To reduce postoperative gastric sequelae and still protect against ulcer disease, Jordan started using proximal gastric vagotomy (parietal cell vagotomy) without drainage. He has reported this in 13 patients with perforated duodenal ulcer. All patients had good to excellent results with follow-up to two years.

Johnston[13] and Goligher[6] in England have reported excellent results using proximal gastric vagotomy in 15 patients with perforated duodenal ulcer. Barroso[3] in Brazil has managed 13 patients with perforated duodenal ulcer by simple closure and proximal gastric vagotomy without drainage. He had one death from respiratory difficulty. The other patients have had excellent results.

The incidence of undesirable side effects from a definitive operation to control the ulcer diathesis is less after proximal gastric vagotomy without drainage than after other operations. The incidence of dumping, diarrhea, and reflux gastritis is significantly reduced.[14] Gastric emptying remains essentially normal. When performed correctly, proximal gastric vagotomy protects against the early recurrence of ulcer. Hallenbeck[11] has emphasized the need for correct surgical technic in performing proximal gastric vagotomy, especially in the periesophageal dissection.

Proximal gastric vagotomy without drainage was performed experimentally by Griffith and Harkins in 1957,[10] but not used in man until 1969 when Johnston and Amdrup independently performed the operation in patients intractable to medical therapy for duodenal ulcer. In 1964 Holle[12] introduced proximal gastric vagotomy in patients with duodenal ulcer disease, but have always added a concomitant pyloroplasty. Johnston is apparently the first surgeon to use proximal gastric vagotomy without drainage in a patient with an acute perforated duodenal ulcer.

Proximal gastric vagotomy without drainage is widely used in Europe for the surgical treatment of duodenal ulcer. Johnston[13] has collected over 5,000 patients from a survey of the literature. The operative mortality is only 0.3%. The safety of the operation, minimal morbidity after operation, and reduced incidence of sequelae from "postgastrectomy syndrome" are well accepted. However, the effectiveness of the operation in long-term control of the ulcer diathesis is not known. Preliminary and interim reports extending up to 7 years are encouraging with ulcer recurrent rates of 0–6.4%.[1] Goligher[6] reports only one patient with a recurrent duodenal ulcer in 100 patients who have been followed more than five years.

Proximal gastric vagotomy without drainage added to simple closure of perforated duodenal ulcer appears to be a satisfactory compromise between simple closure alone which does not protect against continuing or recurrent ulcer symptoms and definitive ulcer operations which may impose the risk of postoperative gastric side effects on patients who may not have further ulcer symptoms. Experience with 21 patients indicates that proximal gastric vagotomy is a safe, effective additional procedure to add to simple closure for the patient with an acute perforated duodenal ulcer.

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Discussion

Dr. Paul H. Jordan, Jr. (Houston, Texas): Highly selective vagotomy, or parietal cell vagotomy, was first used in combination with a pyloroplasty for the treatment of duodenal ulcer by Fritz Holle about 12 years ago. This type of vagotomy, used without drainage, was introduced independently by Johnston and Amdrup approximately 7 years ago. The results of the latter procedure with respect to reducing the adverse effects of dumping, diarrhea, bilious vomiting, sometimes associated with conventional types of gastric surgery, have been excellent, as the present study by Sawyers and Herrington indicates.

The long-term results with respect to the incidence of recurrent ulcer is still awaiting a final answer. At the present time it appears that the recurrence rate is in the vicinity of 5% when the procedure is properly performed.

With respect to the utilization of this operation for duodenal ulcers, with the complications of bleeding, perforation, and obstruction, or for gastric ulcers, our information is more rudimentary and fragmentary. The results of parietal cell vagotomy in patients with perforated duodenal ulcer that Dr. Sawyers has reported is a cautious extension of the use of parietal cell vagotomy into an area less well explored than has the use of the operation for the elective treatment of duodenal ulcer.

This report, as well as several others, some of which he mentioned, that have appeared on this subject, suggests that the results with parietal cell vagotomy, when combined with a patch closure of the perforation, provides an excellent way to handle many patients with perforated ulcer.

Although many patients with perforation not treated with a definitive procedure will continue to have symptoms and require further surgery, there is a substantial number that will have no further trouble. The difficulty is our inability to precisely identify those patients who will and those who will not continue to have ulcer symptoms if untreated.

Being unable to do this, it seems unreasonable, then, to perform definitive surgery of the conventional variety on all patients who have a perforation, and thus incur a significant incidence of undesirable side effects in patients who might not have required definitive surgery.

Dr. Sawyers' results, therefore, suggest that parietal cell vagotomy without drainage may be a satisfactory compromise between the use of simple closure that does not protect patients against recurrent ulcer, and the use of vagotomy and hemigastrectomy, or vagotomy and drainage, which impose increased morbidity on those patients who might not have had further ulcer disease.

We therefore concur with Drs. Sawyers and Herrington in their thesis, and our results in 23 patients similarly treated completely support their conclusions.

I would like to ask Dr. Sawyers and Dr. Herrington what precautions do they take at the time of the operation to avoid performing parietal cell vagotomy in a patient who may have a pyloric lumen of inadequate size, leading to obstruction?

The other question is: How do they feel about the application of this procedure in patients with a prepyloric ulcer which has the same clinical characteristics as a duodenal ulcer, or with the garden variety of gastric ulcer at the angularis incisura that is unaccompanied by a duodenal ulcer?

Dr. Edward R. Woodward (Gainesville, Florida): I want to ask the authors in how many cases they were not able to perform this for their patients in whom the inflammatory reaction was advanced to the point where this wasn't feasible. I would suspect that probably the lesser curvature is quite well protected by the liver, and that this particular area may be much more accessible than we would commonly think.

We haven't used definitive surgery extensively in patients with perforation, largely because in our locale we tend to get patients rather late; but I certainly think that the concept is sound.

Are there patients, however, where a nonrecurring stress relating to the perforation, which was present in two of their cases, would indicate that recurrence of perforation or development of persistent duodenal ulcer symptoms would be most unlikely? Wouldn't it be prudent to omit parietal cell vagotomy in such patients?

Also, in their manuscript I noticed that they plicate the serosa-covered posterior and anterior walls of the stomach over the area where the branches of the vagi have been transected, and I wonder if they feel this is essential. We don't do it, and have found no cause to regret it. What do they think the advantages are of this maneuver?

Dr. Patricia C. Moynihan (Jackson, Mississippi): I deal primarily in pediatric surgery, but I thought it might be appropriate to share with you some of the cases that we have seen in the neonate in recent years. As a matter of fact, in the last two years we have had 8 cases of newborn infants who had duodenal ulcer problems, and these were divided into 6 infants who had anterior perforation and two infants who had posterior bleeding also. The male-to-female ratio was 1:1.

Simply, we could categorize these patients into two categories. One is the stress infant, who is the premature infant with