Gallstone Disease Following Antrectomy and Gastroduodenostomy with or without Vagotomy

O. REHNBERG, M.D., U. HAGLUND, M.D.

The incidence and prevalence of gallstones has been documented in 289 consecutive patients with peptic ulcer disease, at the time of antrectomy and gastroduodenostomy (with or without truncal or selective vagotomy) and again during a 5-year follow-up period. By comparing the preoperative prevalence of gallstone disease in one age group with the prevalence 5 years after the gastric operation in another group of patients who were 5 years younger at the operation, the incidence of gallstone production due to the gastric operation could be calculated independent of the age factor. Within 5 years of the gastric operation, 18% of the patients who were normal at the time of operation produced gallstones. The incidence of new gallstones during the 5-year postgastrectomy follow-up was the same in men and women, and was increased by 7 to 15% in each age group of men. The incidence of new gallstones was 30% after truncal and 12% after selective vagotomy (p < 0.05). Gallstone formation seems to be a sequel of Billroth I gastric resection. Truncal vagotomy in addition to the gastrectomy increases the risk of gallstone disease; patients with selective vagotomy and antrectomy had an incidence of postoperative gallstones which was the same as patients with antrectomy alone.

Gallstone disease has been presumed to be a sequel of different types of gastrectomy or vagotomy. Although high incidences have been reported, it has not been conclusively shown that postoperative patients have a higher rate of gallstone formation than nonoperated patients. Kranse has reported a significantly higher incidence of gallstones in female (but not in male) Billroth I and Billroth II gastrectomized patients than in controls. Clave and Gaspar, on the other hand, found that the incidence of new gallstones in males was almost equal to that in females after truncal vagotomy and pyloroplasty. Kranse found the incidence of gallstones to be almost equal after Billroth I or Billroth II procedures, whereas Lundman et al. reported a higher incidence in Billroth I than in Billroth II gastrectomized patients, the incidence in the latter being the same as in unoperated duodenal and gastric ulcer patients.

Most authors believe that the etiology of gallstone formation after gastric resection is denervation of the gallbladder causing dilatation either intended during truncal vagotomy, or inadvertent during selective vagotomy or gastric resection.

In the present study, the incidence and prevalence of gallstones were analyzed in duodenal and gastric ulcer patients, who were followed for 5 years after antrectomy and gastroduodenostomy with or without selective or truncal vagotomy.

Patients and Methods

Of 303 consecutive patients with gastric or duodenal ulcer operated upon electively at the Gastroenterological Unit of the Department of Surgery II, Sahlgrenska Hospital, from 1966 through 1970, 289 patients were treated by antrectomy and gastroduodenostomy with or without vagotomy and participated in a prospective 5-year follow-up. The operations were primary, save 35 patients who had previously undergone a simple suture of a perforated ulceration.

According to age and ulcer location, the material was representative of the entire surgically treated peptic ulcer population of Gothenburg with the exception of slightly fewer female patients, 21% versus 27% in the whole area. The median age at the time of antrectomy was 49 years (range 19–80). The duration of the ulcer disease was 11.5 ± 0.5 years (mean ± SEM) (n = 284).

Surgical Procedures

Antrectomy, resection of the duodenal bulb, and gastroduodenostomy were performed in every patient. The corpus antrum border was defined intraoperatively by litmus indication after betazole (Histalog®) stimulation of the acid-secreting glands. The resection was made 1 cm proximal to the indicated border. There was no further dissection of the lesser or greater omentum. Microscopic identification of parietal cells at the proximal edge of the resected specimen subsequently verified that

Correspondence to: O. Rehnberg, M.D., Department of Surgery II, Sahlgrenska Hospital, S-413 45 Gothenburg, Sweden.
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the antrum was completely removed. Care was taken to resect all of the duodenal bulb by excising it close to the pancreaticoduodenal junction at the back and another few centimeters aborally at the front. The lesser curvature of the gastric remnant and the gastroduodenal anastomosis were sutured with double layers of chromic catgut 000 and 00, forming a wide stoma because of the generous excision of the anterior duodenal wall. Vagotomy was added solely on the basis of a preoperative gastric acid secretion capacity exceeding 40 mmol/h. Vagotomy was thus performed in 49% of the duodenal ulcer patients, in 22% of the patients with combined duodenal and gastric ulcer, and in four per cent of the gastric ulcer patients (that is, in 112 patients altogether). As the gastric acid secretion capacity was lower in the female patients and decreased with age, vagotomy was performed particularly in the young (including 77% of the patients below 30 years of age) and in the male duodenal ulcer patients (55% of the men vs. 18% of the women). Most of the vagotomies were selective, but truncal vagotomy was performed in 30 cases by a technique described by Broomé. All intraabdominal organs were palpated. Special attention was paid to the presence or absence of stones in the gallbladder.

Additional surgical procedures included cholecystectomy in 11 patients, exploration of the common bile duct in four, plastic repair of the crura in 15 (mainly in vagotomy cases), incidental appendectomy in 11, Meckel's diverticulectomy in two, splenectomy in one, repair of incisional hernia in one, and temporary gastrostomy in 43 patients.

Follow-Up

One month, 3 years, and 5 years after the operation the patient was called for check-ups, including a thorough clinical examination, determination of body weight, and gastric acid secretory tests. Three and 5 years after the operation, oral cholecystography and various blood tests were performed. In seven patients with weak or no contrast filling of the gallbladder and no visible biliary stones, an intravenous cholangiogram was performed. A few cases that remained doubtful were recorded as free of gallstone disease. Missing data seemed to be randomly distributed according to checks against age, sex, ulcer location, operation method, and Visick score.

Method of Calculation

To answer the question of whether the operation caused an increased incidence of gallstones, the prevalence of gallstones was recorded at operation in all patients according to their age groups and was compared to the prevalence 5 years after the antrectomy in patients who had reached the same age group by that time. The postgastrectomy prevalence of gallstone disease included the prevalence at the antrectomy and the incidence in the 5-year period after the operation. Unoperated peptic ulcer patients could thus be compared with patients of the same age operated by antrectomy 5 years previously (Table 3).

Statistics

Significance levels were determined according to Chi square and Fischer's exact tests.

Results

Prior to gastric surgery, the prevalence of gallstones in female patients with peptic ulcer disease was 5 times that of males (Table 1). Within the 5-year period after Billroth I gastrectomy, with or without vagotomy, 18% of the patients with preoperatively normal gallbladders by palpation produced gallstones. This percentage was not significantly related to the sex or age of the patient (Table 2).

The incidence of gallstone formation after selective vagotomy was 12% (7/60), and after truncal vagotomy was 30% (6/20) (p < 0.05). In males following gastric resection the prevalence increased significantly (p < 0.02) (7–15%) in each age group (Table 3). The gallstones seemed to be produced early after operation, as 83% of the cases were diagnosed within 3 years of the 5-year follow-up (expected rate 60%).

In male patients, the mean preoperative body weight (72.5 kg) was identical in patients with or without gallstone production after the antrectomy. In male patients who produced gallstones after surgery, the mean change within 5 years after the gastrectomy was +1.5 kg versus +1.8 kg in those without gallstone production.
Only five out of 37 patients who produced gallstones during the 5-year follow-up underwent cholecystectomy during this time.

Discussion

In this study, it has been demonstrated that antrectomy and gastroduodenostomy increase the prevalence of gallstone formation, and that this increase is more pronounced if a truncal vagotomy—but not a selective vagotomy—is added.

In a population study from Malmö and Prag,\textsuperscript{10} the prevalence of gallstones in females was 1.5–2 times that of males; in the Framingham study,\textsuperscript{11} this increased prevalence in females was 3–6 times. In the latter investigation, the 10-year incidence rate of gallstone disease in females was 2–4 times that of males. No association has been reported between peptic ulcer disease and gallstone formation.\textsuperscript{10,12} Therefore, the equal incidence of gallstone formation in males and females 5 years after antrectomy and gastroduodenostomy (Table 2) implies that there is a major increase in gallstone formation after this procedure, as previously reported by Clave and Gaspar.\textsuperscript{1} This increase in gallstone incidence was not associated with an increase in body weight.

It has been shown previously\textsuperscript{13} that there is good agreement between oral cholecystography and the palpatory findings at laparotomy. In the present investigation, gallstone disease after gastrectomy was defined as visible stones on cholecystography or an I.V. cholangiogram. If anything, the gallstone incidence by this definition is underestimated. It seems unlikely to us that careful peroperative palpation gives falsely negative findings of gallstone disease to an extent that might affect the conclusions of this investigation. As contrast absorption from the gut is frequently impaired after gastric operations, a comparison of pre- and postoperative cholecystograms would probably have been less valid. It is furthermore emphasized that the comparison between selective and truncal vagotomy is not affected by the differing diagnostic criteria before and after operation. The method of statistical calculation might be biased by different disposition to produce gallstones after gastric surgery in different age groups. The similar postgastrectomy gallstone incidence in different age groups, however, contradicts this source of error.

Conclusion

The results of this study demonstrate that gallstone disease is a sequel of Billroth I gastric resection, inducing an excess incidence of about ten per cent within 5 years. Truncal vagotomy further enhances this incidence by an additional ten per cent, perhaps by causing a dilatation of the gallbladder.\textsuperscript{1,14,15} Selective vagotomy in addition to the gastric resection does not further enhance the incidence of gallstone disease. These observations are supported by the findings of Parkin et al.\textsuperscript{16} and Rudick and Hutchison\textsuperscript{15} that there is dilatation of the gallbladder after truncal, but not after selective, vagotomy. As to whether vagotomy increased the lithogenicity of the bile, the opinions are conflicting.\textsuperscript{5,17–20} The mechanism of enhancement of gallstone production after gastric resection has no obvious explanation. The theoretical possibility of an accidental transection of not only the celiac division of the posterior branch of the vagal nerve, which seems possible, but also of the hepatic branches of the anterior vagus at a well-defined antrectomy, seems very unlikely.

We found that the stones following antrectomy occur soon (within 3 years) after the operation. The reason might be either that only certain patients are predisposed to gallstone formation, or that the predisposition is transitory. The latter explanation is perhaps a more reasonable one. Clave and Gaspar\textsuperscript{1} found that most of the new gallstones were formed within 4 years of truncal vagotomy and pyloroplasty. In dogs, Tompkins et al.\textsuperscript{5} recorded a lithogenic bile after vagotomy. However, the lithogenicity disappeared during an 8-week postoperative period. Clave and Gaspar\textsuperscript{1} stated that the fasting volume of the gallbladder was enhanced only during the first 2 years after truncal vagotomy. In male patients 1 to 5 years after truncal vagotomy and pyloroplasty, Stempel and Duane\textsuperscript{17} recorded a less lithogenic bile than in controls.

To summarize, it can be concluded that the risk of acquiring gallstone disease will probably increase about three times after Billroth I gastrectomy (Table 3). Selective vagotomy in addition does not further increase the incidence of gallstone formation and should, therefore, be preferred to truncal vagotomy.

Table 3. Comparison of Gallstone Prevalence in Male Patients before, and 5 Years after, Antrectomy with or without Vagotomy

<table>
<thead>
<tr>
<th>Age</th>
<th>Prevalence before surgery</th>
<th>Prevalence 5 years after surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;45</td>
<td>1/100 = 1%*</td>
<td>13.9/77 = 8%†</td>
</tr>
<tr>
<td>46-55</td>
<td>6/70 = 9%</td>
<td>14.6/61 = 24%</td>
</tr>
<tr>
<td>≥56</td>
<td>6/62 = 10%</td>
<td>23.4/94 = 25%</td>
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</tbody>
</table>

* These patients are 45 years or younger at the operation.† These patients are 45 years or younger 5 years after the operation.

References


