LATE ENDOSCOPIC AND HISTOPATHOLOGIC CHANGES IN PATIENTS WITH BILLROTH II GASTRECTOMY FOR DUODENAL ULCER

ERDAL YILMAZ*
CANDAN TUNCER*
LEYLA MEMIS*
TEVFIK KÜÇÜKPINAR**
CAVIT ÇÖL*
SELVIN AYDIN*

SUMMARY: 25 patients who had gastric resection for duodenal ulcer 10-13 years ago were examined endoscopically. Mucosal biopsies were taken. Endoscopically 13 were normal, 12 had gastritis and 14 had bile reflux. 9 reflux and 3 nonreflux patients showed gastritis. 8 patients had neither reflux nor gastritis. In endoscopically normal patients 5 had minimal, 7 had reactive mucosal changes and 1 had chronic gastritis. In 12 patients who had gastritis endoscopically, 4 showed minimal mucosal changes and 7 showed chronic gastritis. 3 reflux patients had minimal, 5 had reactive mucosal changes and 6 had chronic gastritis histopathologically. 2 non-reflux patients had minimal, 6 reactive mucosal changes and 2 has chronic gastritis. Mean pH of reflux patients was 4.508 ± 0.418 and non-reflux patients 4.26 ± 0.629 . The difference was not significant (P > 0.05). Mean gastrin level in reflux patients was 68.89 ± 12.81 pg/ml, in nonreflux patients 56.22 ± 9.79 pg/ml. The difference was also not significant (P > 0.05). Gastritis is a serious gastrectomy complication. Bile reflux is one of the causes, but not through the pH and gastrin changes. 13 years were not sufficient to produce malignant changes. Therefore, these patients should be pursued at frequent intervals both endoscopically and histopathologically. Key Words: Gastrectomy, bile reflux, gastritis, gastric carcinoma.

INTRODUCTION

Since Thedor Billorth's first gastric resection in 1881, thousands of gastric resections most of which are for duodenal ulcer and its complications, have been done (12). Late complications of gastric resection limited the use of this procedure for benign gastric diseases (6). Histopathologic changes of the gastric mucosa are among those late complications. Balfours's (1) first report of carcinoma after gastrectomy led some investigators to search the reasons of such histopathologic changes and to find alternative surgical procedures. The purpose of this study was to

reevaluate the patients with gastric resection and to discuss the histopathologic changes and their probable causes.

MATERIALS AND METHODS

The patients who have had gastric resection for duodenal ulcer or its complications between 1975-1978 at SSK Ankara Hospital were evaluated. 25 out of 274 gastrectomy patients could be contacted and invited for controls. All of these patients had 75% distal gastric resection and Billroth II antecolic gastroenterostomy. Operative procedures to prevent bile reflux were not performed. The mean age of these patients at the time of the operation was 48, ranging between 24 and 65. This series comprised 23 men and 2 women. The mean time interval from the resection until the endoscopic examination, was 11 years (range 10-13). After the physical exami-

^{*}From Gazi University, Faculty of Medicine, Depts. of General Surgery, Gastroenterology, Pathology and Biochemistry, Ankara, Türkiye

^{**} From SSK Ankara Hospital 1. Surgical Clinic, Ankara, Türkiye.

nation, endoscopy was performed by Olympus GIF-QX-10 fibre gastroscope. Eosophagus, gastric remnant and stoma were inspected with special attention to the presence of bile in the stomach. Biopsies were taken around the stoma, specimens were fixed with 10% formaldehyde and stained with HE and PAS. Morson's (15) classification is used in cases with histopathologically gastritis.

Blood samples were taken to measure serum gastrin levels by radio immunoassay. PH of the gastric juices, obtained by the gastroscope were determined by the PH meter.

The statistical analyses were made by Student's t test.

RESULTS

Endoscopic examination revealed normal gastric and stomal mucosa in 13 patients and peristomal hyperemia and edema, accepted as signs of gastritis, in 12 patients. Endoscopically bile reflux was seen in 14 patients. Gastritis accompanied 9 out of 14 reflux patients. On the other hand, gastritis was seen in 3 non-reflux proteins. Neither reflux, nor gastritis was seen in 8 patients.

Histopathologic examination showed minimal mucosal changes in 5 cases, reactive mucosal changes in 10 cases and chronic gastritis in 9 cases. In 1 case, the specimen was inadequate for evaluation. In cases which were found normal endoscopically, 5 were found to have minimal mucosal changes, 7 reactive mucosal changes and 1 chronic gastritis. However, in 12 patients who had gastritis endoscopically, 4 showed reactive mucosal changes and 7 chronic gastritis. In this group, 1 specimen was inadequate and no mild mucosal changes were seen.

14 patients revealed bile reflux endoscopically. 3 reflux patients had minimal mucosal changes, 5 had reactive changes and 6 had chronic gastritis. Among the 11 non-reflux patients 2 had minimal mucosal changes, 6 had reactive mucosal changes and 2 had chronic gastritis.

In reflux patients, the mean pH was 4.508 ± 0.418 , while in the non-reflux patients it was 4.26 ± 0.629 . The mean serum gastrin levels were 68.89 ± 12.81 pg/ml and 56.22 ± 9.79 pg/ml in the reflux and non-reflux patients, respectively. The mean pH was 3.5 in 5 patients whose gastrin levels were above normal. 2 of those patients showed minimal mucosal changes, 1 had reactive changes and 2 had chronic gastritis.

Table 1: Correlation between endoscopic and histopathologic examination.

| HISTOPAH. EXAM. | NORMAL | GASTRITIS |
|--------------------------|--------|-----------|
| Minimal Mucosal Changes | 5 | - |
| Reactive Mucosal Changes | 7 | 4 |
| Chronic Gastritis | 1 | 7 |
| TOTAL | 13 | 11* |

^{* 1} specimen was inadequate for evaluation.

Table 2: Correlation between bile reflux and histopathologic examination

| HISTOPAH. EXAM. | REFLUX PATIENTS | NON-REFLUX PATIENTS |
|--------------------------|--------------------|------------------------|
| Minimal Mucosal Changes | 3 | 2 |
| Reactive Mucosal Changes | 5 | 6 |
| Chronic Gastritis | 6 | 2 |
| TOTAL | 14 | 10* |

^{* 1} specimen was inadequate for evaluation.

DISCUSSION

The severity of late effects of extensive gastric resections done for duodenal ulcer or its complications, led the surgeons to perform lesser procedures (21). Malnutrion, anemia, metabolic bone diseases, dumping syndrome and gastritis are amoung such undesired effects.

Gastritis is probably the most unpreventable side effect of this operation (7). In our series, 12(48%) patients were diagnosed as gastritis endoscopically. Although histopathologically varying degrees of mucosal changes were found in all 24 patients (96%), only 8(32%) of them showed frank chronic gastritis. Table 1 shows the correlation between endoscopic and histopathologic changes. In literature, after gastric resection and Billroth II operations, gastritis were reported about 90% of the operated patients (4,7,9,11,16). We may postulate that if we increase the number of biopsies taken during endoscopy and the time interval between the operation and the examination, we may obtain a higher percentage of gastritis. Therefore, it is important to stress that gastritis should be diagnosed not only endoscopically, but also histopathologic examination is mandatory.

Various reports stated that bile reflux may play an important role in the etiology of gastritis (6, 11). Reflux was observed in 14 out of 25 patients (56%). Endoscopic gastritis accompanied with 9 of 14 reflux patients (62.3%). Patients who had bile reflux endoscopically, 3 showed minimal mucosal changes, 5 reactive mucosal changes and 6 chronic gastritis. In non-reflux patients, 2 had minimal mucosal changes and 6 had reactive mucosal changes and 2 had chronic gastritis (Table 2). The increase of mucosal changes with bile reflux, gave us the impression that reflux is an important etiologic factor in pastgastrectomy gastritis, which is also supported by an other author (7).

There was no carcinoma in our series. However, there are numerous reports stating the possibility of carcinoma in the gastric remnan. Damellof (5), Schrumpf (20), Savage and Johnkson (19) and Geboes (8) reported gastric cancer possibility in the gastric remnant as 3.3% 6.4%, 1.6% and 8.9%, respectively. In 1962, Morson (14) stated that all of these histopathologic changes should be

regarded as premalignant. In our series, the interval between the operation and the examination was 10-13 years and 96% of the patients had histopathologic changes. Therefore, it has vital importance to follow the patients who had gastric resection for benign diseases endoscopically and histopathologically very closely and detect any early carcinomatous changes.

The mean PH of the reflux patients was 4.508 ± 0.418 and non-reflux patients was 4.26 ± 0.629 . The difference is not statistically significant (p > 0.05). PH values of these patients are more alkaline than normal and duodenal ulcer patients, but it is obvious that bile reflux does not have a significant effect on the gastric juice pH. This indicates that, bile reflux doesn't directly affect the gastric mucosa by changing the PH values to a more alkaline medium. Werner et al. (24) declared a correlation between bile reflux and mucosal changes. On the other hand. Chelie (3), Haare (10) and Pääkönen (17) could not find any correlation between postgastrectomic reflux and gastric mucosal changes. It has been reported that reflux causes histopathologic changes by affecting gastric microbial flora (2). In another study parallel to this one, a possible correlation between the altered microbial flora and the histopathologic changes was proposed by the same research team (18).

Gastrin was proposed to be one of the causes of post-gastrectomy gastritis (13). However, in this study it was shown that gastrin levels were 68.89 ± 12.81 pg/ml and 56.22 ± 9.74 pg/ml in reflux and non-reflux patients, respectively. The difference is not statistically significant (p>0.05). A similar result is obtained by Heerden *et al.* (7).

It can be concluded that gastritis an important side effect of gastric resection. Bile reflux is probably one of the causes of gastritis, but reflux does not affect the gastric mucosa by changing the gastric juice PH. Gastrin is not responsible for such mucosal changes. 13 years are not sufficient for gastric carcinoma to develop at the gastric remnant. However, to detect such malignant changes, gastrectomy patients should be followed both endoscopically and histopathologically at frequent intervals.

REFERENCES

- 1. Balfour OC: Factors influencing the life expectancy of patients operated on for gastric ulcer, Ann Surg 76:405-403, 1922.
- 2. Carboni M, S Guadagni, MA Pistoia, C Amicucci, D Tuscano, P Negro, PLR Simith, CH Waters: The microflora of the gastric juice after Billroth I and Billroth II partial gastectomy, Scand J Gastreonterol 21: 461-470, 1986.
- 3. Cheli RA Glacosa, F Molinari: Chronic atrophic gastritis and duodenogastric reflux, Scand J Gastroenterol (Suppl) 16:125-127, 1981
- 4. Domellöf L, S Eriksson, KG Janunger: Late occurrence of the precancerous changes and carcinoma of the gastric stump after Billroth II resection, Acta Chir Scand 141:292-297, 1975.
 - 5. Domellöf L, S Eriksson, KG Janunger: Carcinoma and possible

- precancerous changes of the gastric stump after Billroth II resection, Gactroenterol 73:462-468, 1977.
- Fischer AB: Twenty-five years after Billroth II gastrectomy for duodenal ulcer, World J Surg 8:293-302, 1984.
- 7. Fisher AB, N Graem, LA Christiansen: Causes and clinical significance of gastritis following Billroth II resection for duodenal ulcer, Br J Surg 70:322-325, 1983.
- 8. Geboes K, P Rutgerts, L Broeckhaert et al: Histologic appeanances of endoscopic gastric mucosal biopsies, Ann Surg 192:179-182, 1980.
- 9. Gjeruldsen ST, J Myren, B Fretheim: Alterations of gastric mucosa following a graded partial gastrectomy for duodenal ulcer, Scand J Gastroenterol 3:465-70, 1986.
- 10. Hoare AM, EL Jones, J Alexander-Williams et al: Symptomatic significance gastric mucosal changes after surgery for peptic ulcer, Gut 18:195-300, 1977.
- 11. Janunger KG, L Domellöf, S Eriksson: The development of mucosal changes after gastric surgery for ulcer disease, Scand J Gastroenterol 13:217-23, 1978.
- 12. Kenedy T: Billroth II symposium: The failures of gastric Surgery and their management, Br J Surg. 68:677, 1981.
- 13. Martin F, IB Macleod, W Sircus: Effect of antrectomy on the fundic mucosa of the rat Gastroenterology 59:437-444, 1970.
- 14. Morson BC: Percancerous lesions of the upper gastrointestinal tract, JAMA 179:311-315, 1962.
- 15. Morson BC, IMP Dawson, FA Jones: Gastrointestinal Pathology Blackwell Scientific Publications, P. 80-94, 1972.
- 16. Nielsen JA, EH Thaysen, H Olesen et al: Fundal gastritis after Billroth II type resection in patients with duodenal ulcer, Scand J Gastroenterol 7:337-43, 1972.
- 17. Paakkönen M, S Aukee, K Syrjanen et al: Gastritis, duodenogastric reflux and bacteriology of the gastric remnant in patients operated for peptic ulcer by Billroth I Operation, Ann Clin Res 17:32-36,
- 18. Rota S, E Yilmaz, C Tuncer ve ark Parsiyel gastrektomili hastalarin mide suyu florasinin incelenmesi: Infeksiyon Dergisi 2(3):303-310, 1988
- 19. Savage A, S Jones: Histological appearances of the gastric mucosa 15-27 years after partial gastrectomy. J Clin Path 32:179-186, 1979.
- 20. Schrumpf E, J Stadas, J Myren et al: Mucosal changes in the gastric stump 20-25 years after partial gastrectomy, Lancet 2:467-469, 1977
- 21. Schwartz SI et al: Stomach. In Principles of Surgery Singapore, McGraw-Hill Book Company, P1123, 1985.
- 22. Stalsberg H, S Taksdal: Stomach cancer following gastric Surgery for benign conditions, Lancet 2:1175-1177, 1971.
- 23. Van Heerden JA, SG Phillips, MA Adson et al: Postoperative reflux gastritis. Am J surg 129:82-88, 1975.
- 24. Werner B, A Leppin, I Seiler et al: Dodenaler reflux und gastritis im Billroth I magen, Deutsch Med Wochen schr 100:2385-2388, 1975.

Correspondence: Doç. Dr. Erdal Yilmaz Gazi Üniversitesi Tip Fakültesi, Ankara, TURKIYE.