Gastritis Cystica Polyposa - Report of a Case

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SUMMARY: A case of gastritis cystica polyposa is presented. A 45-year-old woman developed gastritis cystica polyposa at the gastroduodenostomy anastomotic site four years after Billroth II gastrectomy for adenomatous polyp. There was no association of gastric cancer in the lesion.

INTRODUCTION

Gastritis cystica polyposa (GCP) is a rare benign condition which develops at the gastrointestinal anastomotic site. Many nomenclatures were given to this condition including gastritis cystica, multiple polypoid cystic gastritis, and stomal polypoid hypertrophic gastritis. Since Qizilbash (1975) and Iwashita, et al (1982) reported the case of an association of gastric cancer, this condition has been focused on as a premalignant condition. There hitherto has been 12 cases of such a rare coexistence of the two lesions. In this paper, a case of GCP is presented. This case had no associated cancer in the stomach. Terms appearing in this paper are used according to the general rules for the gastric cancer study in surgery and pathology in Japan.

CASE REPORT

A 45-year-old woman was admitted to our hospital on April 17 in 1972 because of anorexia and vomiting of eight months' duration. She lost 4kg in the last 10 months. She had undergone Billroth II gastrectomy for gastric polyps (adenomatous polyps) in 1968 at her age of 41.

A stomach polyp was detected at the anastomotic site on the upper GI series and endoscopic examination in June 1970. Since that time she had been followed-up periodically. Because she developed the above symptoms and the polyp increased in size, she was readmitted to the hospital for operation. Her past history included appendectomy at 24, and her family history showed her father died of gastric cancer at her 62 years of age.

Physical examination on admission was not remarkable. The previous operation scars were seen at the upper abdominal midline and the right lower quadrant. Her blood pressure was 104/66 mmHg, pulse 90/minute regular, body temperature 36.6°C. Laboratory data showed RBC 408×10^9/mm^3, Hb 11.8g%, Hct 37%, white blood cell count 2,700/mm^3 with 1% of basophile, 3% of eosinophils, 2% of bands, 44% of polymorphs, and 50% of lymphs. The platelet counts were
6.1 × 10⁴/mm³. The liver and renal function tests and serum electrolytes were all within normal limits, including a total protein of 6.3g%, albumin of 3.8g%.

A barium swallow showed a polypoid tumor, measuring 2cm, at the greater curvature side of the anastomotic site and a filling defect being 1cm in width along the anastomotic site. An endoscopic examination showed three polypoid lesions at the anastomotic site.

At operation (May 2, 1972), the initial operation was found to be Billroth II type gastrectomy in a retrocolic fashion with no Braun's anastomosis. The anastomotic site was about two times as thick as usual, and a polypoid mass was palpable at the greater curvature side. After the stomach and jejunum around the anastomotic area were resected, the gastrointestinal continuity was restored by making a Billroth I anastomosis. The resected specimen showed a polypoid lesion at the greater curvature and a protruded fold of the stomach along the gastrojejunostomy anastomotic site (Fig. 1). The surface of the lesion was fine-nodular, similar to the brain convolution seen in the giant rugae

Fig. 1. The resected specimen showing a polypoid lesion and a giant fold along the gastrojejunostomy anastomatic site with a fine-nodular surface

Fig. 2. A polypoid lesion at the gastrojejunostomy site. Arrow indicates the anastomotic junction (H & E, ×3.3).

Fig. 3. Microscopic findings of the polypoid lesion (H & E, ×10)
of Menetrier's disease. A histologic examination showed elongation of the gastric pits, hyperplasia and cystic dilatation of the pseudopyloric glands and their submucosal invasion (Figs. 2 and 3). The diagnosis of GCP was established. There was no evidence of malignancy.

The patient is well and has no symptoms or signs of recurrence of GCP at the time 19 years after the second gastric resection.

**DISCUSSION**

The first case of GCP was documented in 1965 by Nickolai and Muller\(^2\) as gastritis cystica. Since then the detection of these lesions has increased steadily. In Japan, the first case of GCP was described by Kameyama, \textit{et al.}\(^8\) in 1972. GCP develops mostly in men following Billroth II gastrectomy and rarely following gastroenteric anastomosis\(^{4,9,10}\). The interval from the first operation ranges from six months to 42 years\(^9\).

Recently GCP has been focused on as a possible precancerous lesion. To our knowledge, only three cases of GCP associated with gastric cancer have been reported in the western literature\(^{5,9,10,11}\). In Japan, Iwashita documented the first case of a coexistence of GCP and focal early carcinoma, and since then ten cases have been reported as listed in Table 1. Franzin, \textit{et al.}\(^11\) considered that GCP is a possible precancerous lesion because of the similarities of the site and the histologic features of GCP to those of experimental stomal polyps in rats after partial gastrectomy. They stated that the most common histologic changes in case of GCP were atrophy of gastric glands, cystic dilatation of gastric glands, intestinal metaplasia and dysplastic changes which occur early in the postoperative period; and that local chronic ischemia and inflammatory reaction as a consequence of gastric surgery and suture at gastroenterostomy together with bile reflux and increase in gastric pH were responsible for the development of GCP and carcinoma. Our patient had no association of cancer. However, a special attention should be paid to the subsequent development of carcinoma from GCP.

**REFERENCES**


**Table 1.** Ten reported cases of gastritis cystica polyposa associated with cancer of the stomach in the Japanese literature

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>First operation</th>
<th>Time interval(\star)</th>
<th>Type</th>
<th>Early gastric cancer</th>
<th>Size**</th>
<th>Depth</th>
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</thead>
<tbody>
<tr>
<td>1982</td>
<td>Iwashita</td>
<td>B-II</td>
<td>18y</td>
<td>I</td>
<td>tub(_b)</td>
<td>2×2</td>
<td>m</td>
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<tr>
<td>1982</td>
<td>Fukuchi</td>
<td>B-II</td>
<td>19y</td>
<td>IIa</td>
<td>tub(_b)</td>
<td>5×3</td>
<td>pm</td>
</tr>
<tr>
<td>1982</td>
<td>Kondo</td>
<td>B-II</td>
<td>19y</td>
<td>elevated</td>
<td>tub(_b)</td>
<td>1.5×1</td>
<td>sm</td>
</tr>
<tr>
<td>1982</td>
<td>Kondo</td>
<td>B-II</td>
<td>21y</td>
<td>elevated</td>
<td>tub(_b)</td>
<td>3.5×3</td>
<td>pm</td>
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<tr>
<td>1986</td>
<td>Okamoto</td>
<td>B-II</td>
<td>26y</td>
<td>IIc</td>
<td>tub(_b)</td>
<td>7×1.5</td>
<td>sm</td>
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<tr>
<td>1987</td>
<td>Ishikawa</td>
<td>B-II</td>
<td>16y</td>
<td>I</td>
<td>tub(_b)</td>
<td>3.5×3</td>
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<td>1988</td>
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<td>B-II</td>
<td>28y</td>
<td>I</td>
<td>tub(_b)</td>
<td>0.7×0.7</td>
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<td>25y</td>
<td>I</td>
<td>tub(_b)</td>
<td>7×4</td>
<td>sm</td>
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<td>1988</td>
<td>Hosokawa</td>
<td>B-II</td>
<td>23y</td>
<td>IIa+IIc</td>
<td>sig</td>
<td>4×3</td>
<td>sm</td>
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<td>1988</td>
<td>Lien</td>
<td>B-II</td>
<td>36y</td>
<td>IIa+IIc</td>
<td>tub(_b)</td>
<td>1.6×1.5</td>
<td>m</td>
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</table>

\(\star\): years from the first gastrectomy, **: centimeters, B-I: Billroth-I gastrectomy, B-II: Billroth-II gastrectomy, tub\(_b\): well differentiated adenocarcinoma, tub\(_c\): moderately differentiated adenocarcinoma, tub\(_p\): poorly differentiated adenocarcinoma, sig: signet ring cell carcinoma, m: mucosal cancer, sm: submucosal cancer