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Case Report

Two Cases of Primary Gastric Lymphoma, Mucosa-Associated Lymphoid Tissue (MALT)-type

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Two cases of early primary gastric lymphomas, mucosa-associated lymphoid tissue (MALT)-type, in 65 years old and 50 years old females, are reported. The both lymphomas showed small ulcerated lesions. In these ulcerated lesions, centrocyte-like (CCL) cells and monocytoid cells proliferated around the enlarged germinal centers. Thus, MALT type malignant lymphoma was identified. Monoclonality of these lymphoma cells could be recognized by means of in-situ-hybridization of immunoglobulin light chain gene to see monoclonal signals in lymphoma cells differentiated in to plasma cells in the superficial layer of the mucosa. Furthermore, *Helicobacter pylori* could be found in the mucous material of the gastric glands in the both cases. Clinically these lymphoma were well responsive to local excision.

Key words: Stomach, MALT-type lymphoma, Helicobacter pylori

Introduction

The normal stomach does not contain mucosa associated lymphoid tissue (MALT). It is considered that many extranodal lymphomas such as those in the gastrointestinal tract, salivary gland, lung, and thyroid arise from MALT[1-3]. The study of Genta, et al[2] supported the concept that MALT, which may be induced by the infection of *Helicobacter pylori*, is a precursor in the development of primary gastric lymphoma. We encountered two cases of MALT type gastric lymphomas revealing small ulcerated lesions. Here, we reported these lymphomas as early phase MALT type lymphomas with discussion about the detection of monoclonality of the lymphoma cells and examination for *H. pylori*.

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Clinical summary

Case 1

A 65-year-old woman complaining of anal prolapse had taken a routine medical checkup in every two years. One year ago, gastroendoscopical examination revealed a shallow depressed lesion under the gastric angle. She had complained of continuous epigastralgia from five months ago. That gastric lesion had been enlarged gradually. Surgical resection of the stomach was performed.

Case 2

A 50-year-old woman visited a hospital because of continuous epigastralgia for two years. She was diagnosed as chronic gastritis clinically and had received some medicines. One month before operation, epigastralgia worsened and she was admitted to the hospital. Gastroendoscopical examination disclosed an irregular ulcerative lesion with protruded margin at the cardia. Surgical resection of the stomach was performed.

Pathological findings

The principal histopathologic features of each case were similar.

The resected stomach of the case 1 showed a small and shallow ulcerated lesion (1.2 cm in diameter) at the lesser curvature of the pyloric antrum. Microscopically, in the lesion, enlarged germinal centers surrounded by thin mantle zone associated a pale marginal area comprising centrocyte-like (CCL) cells and plasma cells (Figure 1a). Clusters of CCL cells destroyed the glandular epithelium, forming so-called lymphoepithelial lesion (LEL) (Figure 1b). Immunohistochemically these CCL cells were positive for L26 (CD20). In in-situ-hybridization (ISH) of immunoglobulin (Ig) light chain genes[4] (DAKO In Situ Hybridization Education Kit, No. K 003) plasma cells having signals of Ig lambda type light chain gene dominated in the superficial layer

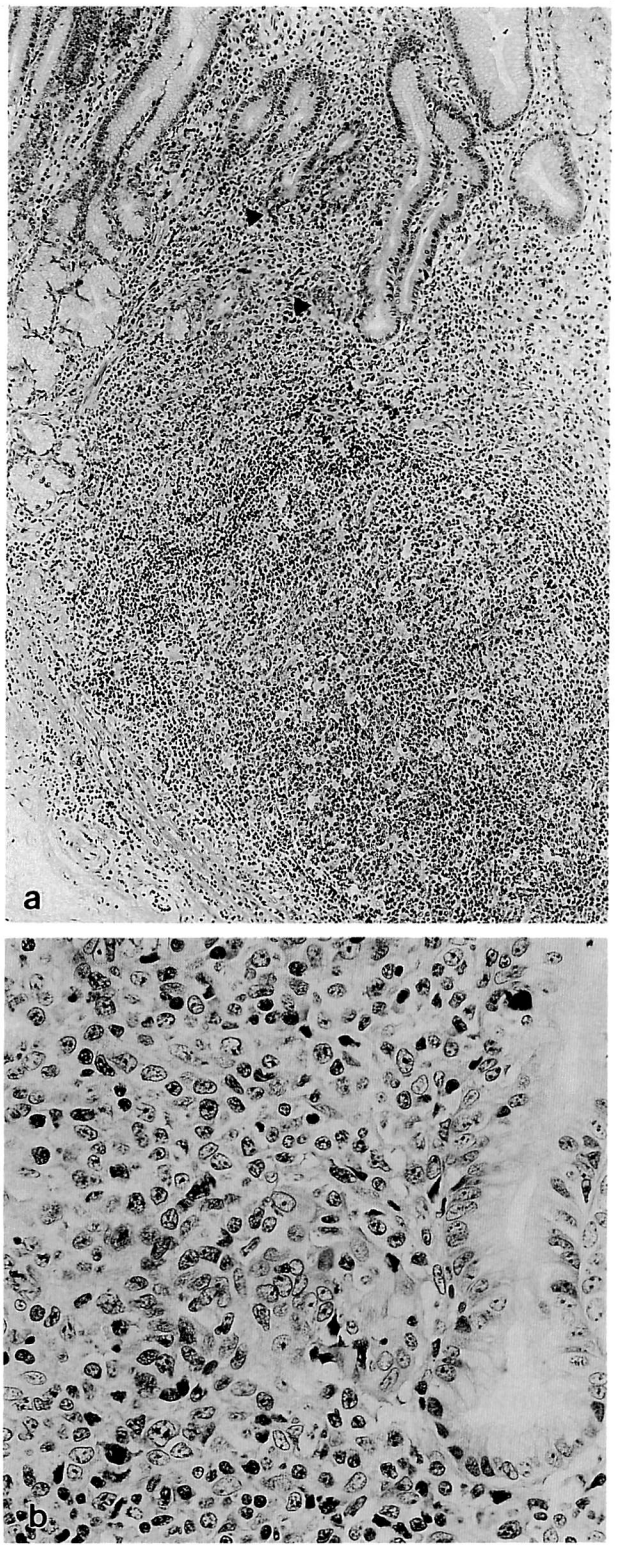

Fig. 1. a) There are reactive lymphoid follicles and lymphomatous infiltrate, which forms lymphoepithelial lesions (arrow heads) in the case 1. (HE, ×85)
b) Note destruction of the gastric proper glands by centrocyte-like cells (Lymphoepithelial lesion). (HE, ×310)

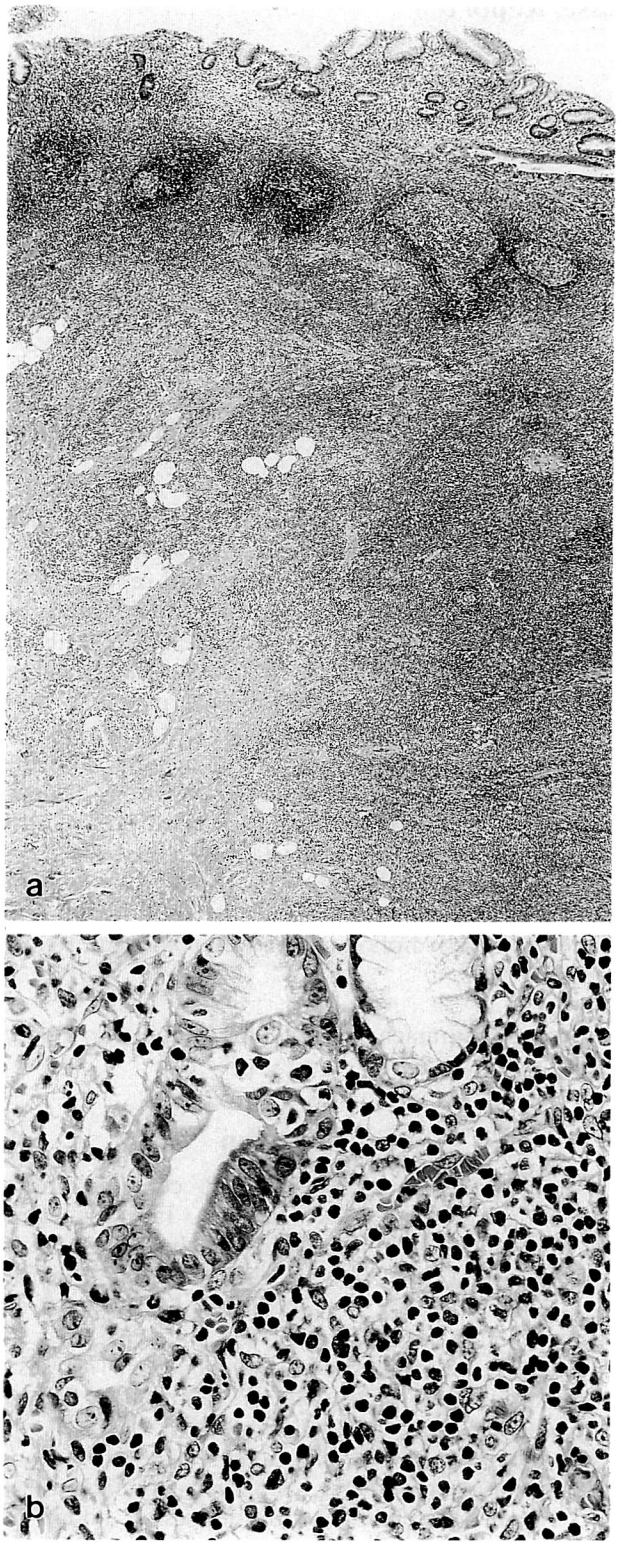

Fig. 2. a) There are small reactive lymphoid follicles in the mucosal layer and lymphoma cells infiltrating diffusely into the submucosa in the case 2. (HE, ×31)
b) Infiltation of monocytoid cells into a gastric proper gland. (HE, ×310)

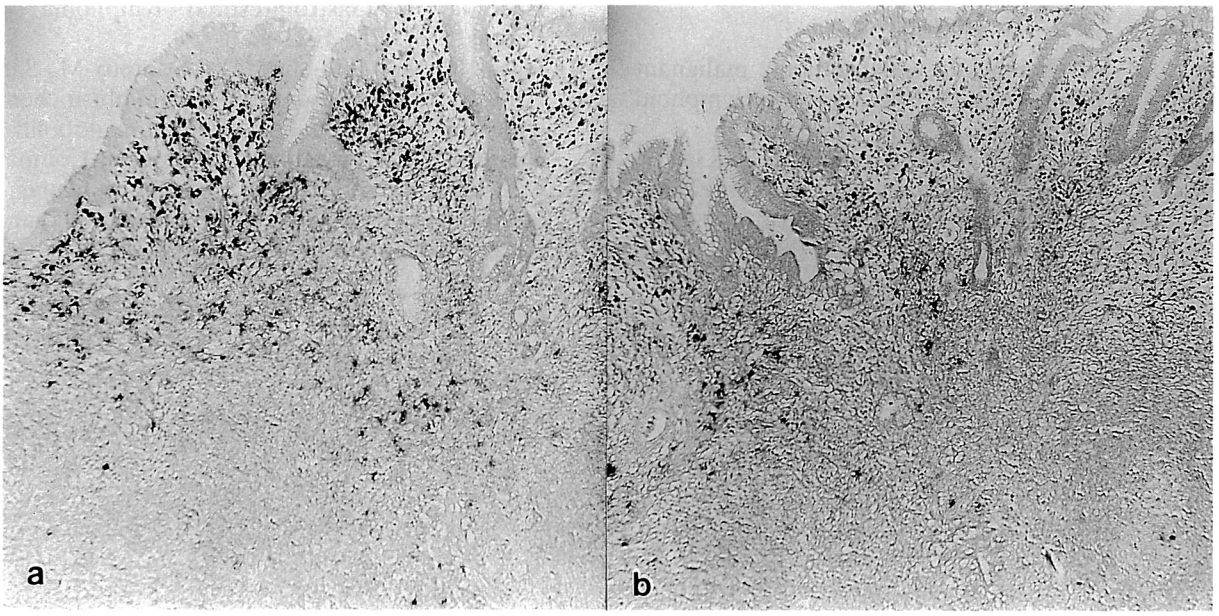

Fig. 3. In situ hybridization for detection of immunoglobulin light chain genes in the case 2. (×85)
a) λ light chain gene b) κ light chain gene

of the propria mucosae.

The resected stomach of the case 2 showed an ill-defined protruded tumor. The tumor was composed of a collection of irregularly thickened folds with multiple small erosions. Microscopically diffuse and mixed proliferation of CCL cells, monocytoid cells and a small number of immunoblast-like cells was found in the lesion (Figure 2a). These cells infiltrated into the deep submucosa. Small reactive lymphoid follicles were associated in the lesion. Some CCL cells had abundant pale cytoplasm and irregular-shaped "cleaved" nuclei. These CCL cells formed LEL in parts (Figure 2b). Immunohistochemically, these cells were positive for L26. The immunoblast-like cells showed IgM in their cytoplasm. In ISH of Ig light chain gene, the plasma cells showed signals of lambda type light chain gene in the superficial layer of the propria mucosae (Figure 3).

No lymph node involvement was noted in the both cases.

H. pylori infection was recognized in the mucosa of the both cases histochemically (by Giemsa stain) and immunohistochemically (by anti-*Helicobacter pylori* antibody (DAKO)).

According to the above findings, we designated these cases as an early phase of B-cell malignant lymphoma of MALT type.

Discussion

In the both cases in this issue, infection of *H. pylori* and hyperplastic lymph follicles of MALT were recognized. Because the preceding *H. pylori* infection seems to be a very important step for the development of MALT and maybe for an occurrence of MALT lymphoma[2], an effort for detecting *H. pylori* should be made at the diagnosis of early MALT type lymphoma.

Both cases show LELs in the mucosal layer. Isaacson[3] described that LELs give rise in the stomach, lung, and thyroid gland to the pathognomonic histopathologic feature of MALT lymphoma. Because non-neoplastic LELs include many T-cells, it may be a diagnostic clue for MALT type lymphoma to see immunohistochemically whether B-cells form LELs or not in the lesion.

MALT type lymphoma cells reveal frequently plasma cell differentiation. But it is difficult without antigen-retrieval method by immunohistochemistry and ISH of Ig light chain genes to see monoclonal plasma cells in the mucosa. And the ISH method was more useful for the detection of monoclonality of Ig in plasma cells in the mucosa on the paraffin-embedded specimen, as reported here.

The lymphoma cells were limited within the mucosal layer in the case 1. In case 2, the lymphoma cells, which infiltrated into the deep submucosa, were composed of many CLL cells and immunoblasts. Thus, the histological grade of lymphoma was more progressive in the case 2. It may be related to the fact that the clinical course of case 2 was longer than that of case 1.

Reference

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