Primary gastric lymphoma is a rare tumour. It is thought that low grade gastric mucosa associated lymphoid tissue lymphoma has not been previously reported to occur in a patient with gastrojejunostomy. This report describes such a case. The patient presented with bleeding from non-healing stomal ulcers. The ulcers healed and there was regression of the tumour after eradication of Helicobacter pylori.

CASE REPORT

A 50 year old man was admitted with history of melena for the past three days. He had had a similar episode of melena three months previously that lasted five days and resolved on conservative treatment. Three units of blood were transfused. Endoscopy was not performed. He had type II diabetes mellitus that was well controlled with oral hypoglycaemic drugs. He was also receiving oral pantoprazole 40 mg per day. Elective gastrojejunostomy and truncal vagotomy were performed for peptic ulcer in 1974. Between 1974 and to date he was asymptomatic and was not taking any medication. The patient denied consuming anti-inflammatory drugs and was very particular about his medication. He was a non-smoker and was very particular about his medication. He was also receiving oral pantoprazole 40 mg per day. Three units of blood were transfused. Intravenous pantoprazole was given.

Histological examination of the biopsy specimens showed focal areas of damage to the epithelial lining of the gastric mucosa with diffuse infiltration of the lamina propria by centrocyte-like cells. There was formation of lymphoid follicles with expansion of the marginal zone. In some of the follicles, centrocyte-like cells were also seen in the centre. The centrocyte-like cells were seen infiltrating through the lining epithelium of the gastric pits (lymphoepithelial lesions). At places hyalinisation and goblet cell transformation of the lining epithelium was also evident (fig 2). Antral biopsy specimens showed presence of H pylori. Ultrasound and contrast enhanced computed tomography of the abdomen did not disclose any abnormality. Two separate fasting serum gastrin measurements, performed a week apart, were within normal range. Immunohistochemically, the centrocyte-like cells were CD 20 positive. The patient refused endoscopic ultrasound examination. The patient was diagnosed to have low grade B cell gastric mucosa associated lymphoma.

Anti-H pylori treatment was given consisting of clarithromycin 500 mg twice daily, amoxyclillin 1 g twice daily, and lansoprazole 30 mg twice daily for two weeks. UGI endoscopy...
Figure 2  Histological appearance of the gastric mucosa. There are focal areas of damage to the gastric epithelial lining with diffuse infiltration of the lamina propria by centrocyte-like cells. There is formation of lymphoid follicles with expansion of the marginal zone. Centrocyte-like cells are also seen in the centre of the follicle. Lymphoepithelial lesions are also visible (haematoxylin and eosin original magnification ×80).

performed at the end of treatment showed considerable improvement in the ulceration at the gastrojejunostomy site. Histological examination of the biopsy specimens obtained from around the gastrojejunostomy site also showed noticeable improvement with decrease in the lymphoepithelial lesions and centrocyte-like cells in the lamina propria. The urease test was negative and antral biopsy specimens did not detect H pylori. A repeat endoscopy, performed two months after stopping anti-H pylori treatment, showed noticeable improvement in the appearance of the gastrojejunostomy stoma (fig 3). The urease test continued to be negative and antral biopsy specimens did not detect the presence of H pylori. Biopsy samples obtained from around the gastrojejunos- tomy stoma showed normal appearance. However, the gastric glands were sparse and there was evidence of fibrosis attributable to healing. There was no evidence of lymphoepithelial lesions or centrocyte-like cells (fig 4).

DISCUSSION
The digestive tract is the most common site of involvement for extra nodal non-Hodgkin’s lymphoma. The involvement of the gastrointestinal tract by lymphoma may be primary5–7 or secondary due to disseminated nodal disease.8–11 Because of the unique properties of the gastrointestinal system, Issacson and Wright,12 in 1983, introduced the term MALT; thereby classifying primary gastrointestinal lymphoma as a distinct entity with unique histological and biological features.13–15

Figure 3  Endoscopic appearance of the gastrojejunostomy stoma. There is noticeable improvement in the appearance compared with figure 1.

Figure 4  Gastric histology. The mucosa appears normal. However, the gastric glands are sparse. Note the presence of fibrosis attributable to healing. The centrocyte-like cells and lymphoepithelial lesions have disappeared (haematoxylin and eosin original magnification ×80).

Overall, primary gastric lymphomas are rare and represent only 1%–5% of all malignant disorders of the stomach. Most primary gastric lymphomas are of the B cell variety non-Hodgkin’s lymphoma. These can be either low grade MALT lymphoma, or high grade, diffuse, large cell lymphoma.2 Primary gastric lymphomas are believed to arise from MALT that is found in Peyer’s patches. In response to an antigenic stimulation, a benign reactive process takes place, leading to formation of lymphoid follicles composed of B cells at different stages of development. Later, on persistence of the antigenic stimulation, small centrocyte-like cells from the marginal zone of the lymphoid follicle invade the epithelial lining, giving rise to classic lymphoepithelial lesions. The lesion is referred to as MALT lymphoma or low grade MALToma.16–18 The antigen responsible for the changes in these patients is H pylori.15

As mentioned earlier, the formation of MALToma is attributable to colonisation of the gastric mucosa by H pylori. The stomach is devoid of lymphoid tissue but chronic gastritis, attributable to colonisation of the gastric mucosa by H pylori, leads to accumulation of lymphoid tissue in the stomach.16 This accumulation gradually leads to changes resulting in MALToma formation.17 H pylori is found in the gastric mucosa of more than 90% of patients with gastric MALToma.17–19 More importantly, MALT lymphomas are known to regress after eradication of H pylori.19–21 Occasionally, even high grade diffuse B cell lymphoma of the stomach20 as well as that of the duodenum have been observed to regress after eradication of H pylori.21

The diagnostic feature of MALToma in this patient, as in all patients with MALToma, was the typical histological results. Lymphoepithelial lesions are diagnostic of MALToma.15–18–22 Nest formation can also occur in various inflammatory and reactive conditions and may be a cause for diagnostic confusion with lymphoepithelial lesions. However, in these situations, immunohistochemistry is helpful and diagnostic. Lymphoepithelial lesions are monoclonal while nest formation is polyclonal.

Sometimes, follicular colonisation may be confused with reactive follicles. However, follicular colonisation has no dendritic cells or macrophages with chromophilic bodies. Moreover, centrocyte-like cells are found in the marginal zone rather than in the germinal centre.14–16–22–24

Occurrence of stomal ulcers is not uncommon after vagotomy and gastrojejunostomy. Gastric malignancies are also known to occur in patients who have undergone gastrojejunostomy in the past.25–26 However, to the best of our knowledge, MALToma occurring at the site of gastrojejunostomy has not been reported. Initially we were of the opinion that the bleeding in this patient was from stomal ulcers. What prompted us to obtain biopsy specimens was the fact that the patient was receiving continuous treatment with...
a potent proton pump inhibitor and bled despite that. Another finding that prompted us to obtain biopsy samples was the presence of nodular lesions on and around the gastroenterostomy site. The endoscopic appearance also prompted us to consider the possibilities of malignancy and tuberculosis as differential diagnosis. As far as cancer is concerned, the chances of development of cancer seem to be more common after Billroth II anastomosis. Similarly, stomal ulcers too are commonly seen after Billroth II anastomosis because of retained antrum. The cancer develops at the site of the Billroth II anastomosis, or in the gastric body or fundus. None the less, cancer after gastrojejunostomy has also been reported.25 26

In the case in discussion, there was a noticeable improvement in the gastric histology, two months after anti-H pylori treatment. This denotes a good prognosis, with less chance of recurrence.27

Authors’ affiliations
S P Misra, V Misra, M Dwivedi, Departments of Gastroenterology and Pathology, Motilal Nehru Medical College, Allahabad, India

Correspondence to: Professor S P Misra, Department of Gastroenterology, Motilal Nehru Medical College, Allahabad 211 001, India; spmisra@sancharnet.in; misrasp@rediffmail.com

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S P Misra, V Misra and M Dwivedi

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