Gastritis

The understanding of gastritis has increased markedly following elucidation of the role of H. pylori in chronic gastritis.

Type A gastritis

This is an autoimmune condition in which there are circulating antibodies to the parietal cell. This results in the atrophy of the parietal cell mass, hence hypochlorhydria and ultimately achlorhydria. As intrinsic factor is also produced by the parietal cell there is malabsorption of vitamin B12 which, if untreated, may result in pernicious anaemia. In type A gastritis the antrum is not affected and the hypochlorhydria leads to the production of high levels of gastrin from the antral G cells. This results in chronic hypergastrinaemia. This in turn results in hypertrophy of the ECL cells in the body of the stomach which are not affected by the autoimmune damage. Over time it is apparent that microadenomas develop in the ECL cells of the stomach, sometimes becoming identifiable tumour nodules. Very rarely these tumours can become malignant. Patients with type A gastritis are predisposed to the development of gastric cancer and screening such patients endoscopically may be appropriate.

Type B gastritis

There are abundant epidemiological data to support the association of this type of gastritis with H. pylori. Most commonly type B gastritis affects the antrum, and it is these patients who are prone to peptic ulcer disease. Helicobacter-associated pangastritis is also a very common manifestation of infection, but gastritis affecting the corpus alone does not seem to be associated. However, there are some data to suggest that Helicobacter may be involved in the initiation of the process. Patients with pangastritis seem to be most prone to the development of gastric cancer.

Intestinal metaplasia is associated with chronic pangastritis with atrophy. Although intestinal metaplasia per se is common, intestinal metaplasia associated with dysplasia has significant malignant potential and if this condition is identified the patient should be regularly screened endoscopically.

Reflux gastritis

This is caused by enterogastric reflux and is particularly common after gastric surgery. Its histological features are distinct from other types of gastritis. Although commonly seen after gastric surgery, it is occasionally found in patients with no previous surgical intervention or who have had a cholecystectomy. Bile chelating or prokinetic agents may be useful in treatment and as a temporising measure to avoid consideration of revisional

surgery. Operation for the condition should be reserved for the most severe cases.

Erosive gastritis

This is caused by agents which disturb the gastric mucosal barrier; NSAIDs and alcohol are common causes. The nonsteroidal-induced gastric lesion is associated with inhibition of the cyclo-oxygenase type 1 (Cox 1) receptor enzyme, hence reducing the production of cytoprotective prostaglandins in the stomach. Fortunately, many of the beneficial anti-inflammatory activities of NSAIDs are mediated by Cox 2, and there is at present much activity to produce Cox 2 inhibitors which will spare some of the side effects of these agents.

Stress gastritis

This is a common sequel of serious illness or injury and is characterised by a reduction in the blood supply to superficial mucosa of the stomach. Although common, this is not usually recognised unless stress ulceration and bleeding supervene, in which case treatment can be extremely difficult. The con-dition also sometimes follows cardiopulmonary bypass. Prevention of the stress bleeding from the stomach is much easier than treating it, and hence the routine use of H2 antagonists with or without barrier agents, such as sucralfate, in patients who are on intensive care. These measures have been shown to reduce the incidence of bleeding from stress ulceration.

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