Autoimmune metaplastic atrophic gastritis

Autoimmune metaplastic atrophic gastritis (AMAG) is an autosomal dominant trait inherited autoimmune disease that the patients have antibodies to parietal cells and their components (which include intrinsic factor and the proton pump H^+,K^+□ATPase). The antibodies attack parietal cells, resulting in hypochlorhydria and decreased production of intrinsic factor. Hypochlorhydria leads to G-cell hyperplasia and elevated serum gastrin levels. Elevated gastrin levels lead to enterochromaffin-like cell hyperplasia, which occasionally undergoes transformation to a carcinoid tumor.

In some patients, AMAG may be associated with chronic *Helicobacter pylori* infection, although the relationship is not clear. Gastrectomy and chronic acid suppression with proton pump inhibitors cause similar deficiencies of intrinsic factor secretion.

The lack of intrinsic factor leads to vitamin B_{12} deficiency that can result in a megaloblastic anemia (pernicious anemia) or neurologic symptoms (subacute combined degeneration). Serum B_{12} levels should be obtained. No treatment is needed other than parenteral replacement of vitamin B_{12}.

The diagnosis of atrophic gastritis can only be ascertained histologically. The endoscopic findings are not helpful for diagnosis, but endoscopy is essential to perform multiple gastric biopsy sampling. The clinicians usually obtain at least 2 biopsy samples from the gastric antrum, 2 from the corpus and 1 from the incisure, and submit to pathology in separate vials.

Histologically, during the early phase, multifocal diffuse infiltration of the lamina propria by mononuclear cells and eosinophils occurs, as does focal T-cell infiltration of oxyntic glands with glandular destruction. Focal mucous neck cell hyperplasia and hypertrophic changes of parietal cells also are observed. During the florid phase of the disease, increased lymphocytic inflammation, oxyntic gland atrophy, and focal intestinal metaplasia occur. Diffuse involvement of the gastric corpus and fundus by chronic atrophic gastritis associated with intestinal metaplasia characterizes the end stage. Some patients present with gastric polyps, mostly nonneoplastic hyperplastic polyps and polypoid areas of preserved islands of relatively normal oxyntic mucosa that may appear polypoid endoscopically. The antrum is spared.

There is 3 fold increase for gastric carcinoma with AMAG patients. The issue of surveillance endoscopy for cancer screening is unsettled; follow-up examinations are unnecessary unless histologic abnormalities (eg, dysplasia) are present on initial biopsy or symptoms develop.

Helpful laboratory tests of autoimmune gastritis include: antiparietal and anti-IF antibodies in the serum; achlorhydria, both basal and stimulated, and hypergastrinemia; low serum cobalamin (B-12) levels (<100 pg/mL); and Shilling test.