Coexistent pathology (Non-core)

Based on the updated Sydney system, chronic gastritis is classified into *Helicobacter* gastritis, ex-*Helicobacter* gastritis, chemically induced/reactive gastritis, autoimmune gastritis and other special forms of gastritis.[1] *Helicobacter* gastritis and autoimmune gastritis are recognised risk factors for gastric carcinoma. Both cause atrophic gastritis with intestinal metaplasia, which may develop into dysplasia/adenoma and further progress to intestinal-type adenocarcinoma. In addition, pyloric gland adenoma may arise in a background of autoimmune atrophic gastritis,[2] which can also progress to gastric carcinoma.

Gastric polyps include fundic gland polyp, hyperplastic polyp and different types of adenoma. Hyperplastic polyps can be seen in the setting of long-term gastritis, and intestinal metaplasia may be seen in large hyperplastic polyps, which may progress to dysplasia and eventually to invasive carcinoma. Rarely, dysplasia is seen in fundic gland polyps, but it almost never progresses to adenocarcinoma. Gastric adenomas include intestinal type, foveolar type, pyloric gland adenoma and oxyntic gland adenoma, all of which can progress to invasive carcinoma.[3]

Other risk factors associated with gastric carcinoma include previous gastric surgery and Epstein-Barr virus (EBV) infection. In addition, approximately 10% of gastric cancers develop in a familial/ hereditary setting, including hereditary diffuse gastric cancer in patients with *CDH1* mutations and patients with Lynch syndrome with microsatellite instability (MSI)-high gastric cancer. Some patients with familial adenomatous polyposis can have multiple foveolar-type adenomas, which have a potential to become invasive carcinoma but at a consistently low rate.[3] In addition, synchronous gastric carcinoma is rare; however, in one report from Asia, synchronous gastric cancer is seen in approximately 10% of gastric cancer patients.[4]

References

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