Envoi SPECIALIST PATHOLOGISTS inside insights

Update No.2 - 2019 Dr Christophe Rosty

# Autoimmune gastritis

Autoimmune gastritis (AIG) is a chronic inflammatory disease targeting the parietal cells of the oxyntic mucosa which produce hydrochloric acid and intrinsic factor. It is a major cause of iron and vitamin B12 deficiencies. Affected patients are at risk of pernicious anaemia and gastric neoplasia. Often underdiagnosed, AIG prevalence is estimated at 2% in patients > 60 years and up to 25% in patients with iron deficiency. The female to male ratio is 3:1.

***Pathogenesis:***

Auto-antibodies against the proton pump (H+/K+ ATPase) result in destruction of parietal cells and progressive atrophy of the oxyntic mucosa in the fundus and body. This leads to:

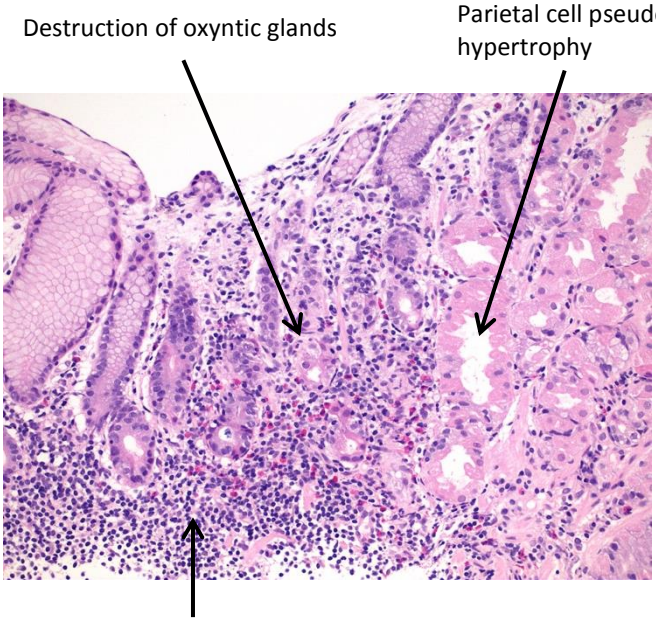
- Decreased gastric acid
  - decreased absorption of iron, bacterial overgrowth
  - increased gastrin from G-cells in the antrum results in enterochromaffin-like (ECL) cell hyperplasia and type 1 neuroendocrine tumours
- Decreased intrinsic factor
  - decreased uptake of vitamin B12 in the ileum
- Intestinal metaplasia
  - Risk factor for dysplasia and adenocarcinoma

***Clinical presentation:***

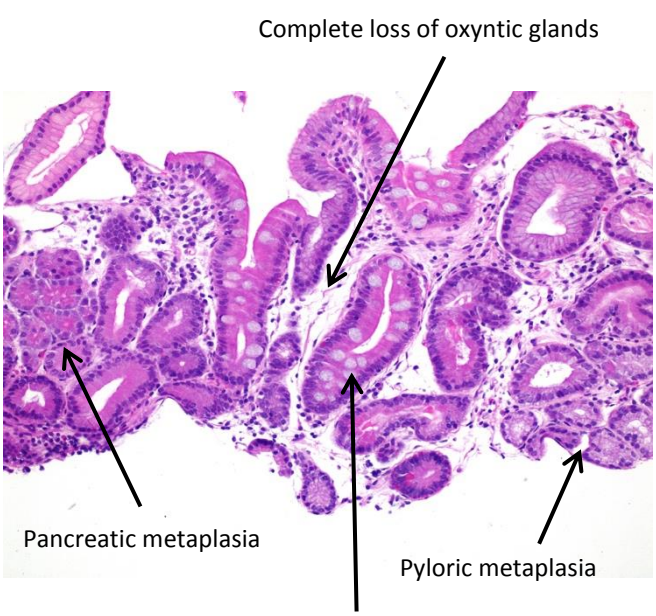
Iron deficiency with or without microcytic anaemia is a common presentation in younger women and is typically refractory to oral iron. Vitamin B12 deficiency (pernicious anaemia) occurs later with risk of neurological complications, glossitis and diarrhoea.

***Diagnosis - Histology:***

Early AIG




Late AIG



Endoscopic biopsies should be taken from the gastric body/fundus and the antrum and preferably placed in separate containers. The biopsies should target the flat mucosa of the gastric body rather than polypoid mucosa which often shows hyperplastic polyps which are insufficient for the diagnosis of AIG. ECL cell hyperplasia develop as the oxyntic mucosa becomes progressively atrophic. The antral mucosa is normal or shows mild reactive gastropathy.

The differential diagnosis includes *H. pylori* gastritis (particularly in patients on PPI) and IBD. *H. pylori* infection can be associated with AIG and should be excluded, but its role in the pathogenesis is unclear.

***Diagnosis - Blood tests:***

Serologic tests are necessary to confirm the diagnosis, particularly in early forms

- Anti-parietal cell Abs: high sensitivity (80%), low specificity
- Anti-intrinsic factor Abs: low sensitivity (50%), high specificity (100%)
- Serum gastrin: elevated (confounding factor is PPI use)

***Complications:***

- Neurological complications secondary to cobalamin deficiency -> subacute combined degeneration, peripheral neuropathy, neuro-psychiatric manifestations
- Gastric dysplasia, adenoma and adenocarcinoma
- Type 1 neuroendocrine tumours (carcinoid)

***Management:***

- Supplementation of iron, folates and vitamin B12
- Eradication of *H. pylori* if present
- Investigation for other autoimmune diseases including thyroiditis, type 1 diabetes

***Endoscopic surveillance:***

No surveillance guidelines specific to AIG currently exist. European guidelines for the management of precancerous lesions of the stomach make the following recommendations:

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| • Extensive atrophy/Intestinal metaplasia | 3-year surveillance |
| • Low grade dysplasia without endoscopic lesion | 1-year follow up |
| • High grade dysplasia without endoscopic lesion | Immediate endoscopic assessment with multiple biopsies and surveillance at 6mo-1yr interval |
| • Type 1 gastric NET | Endoscopic resection, somatostatin analogues, Netazepide (gastrin/CCK-B antagonist) |

***References:***

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