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Gastric biopsies from a 52-year-old male with mild unspecific abdominal complaints.

What is your diagnosis?





		Hallankas	to a deal of	er die staar die se dat is	
		Helicobec	ter pytori e	radication if positive	
+				¥	
Patients with atrophic gastritis or intestinal metaplasia (IM)				Patients with dysplasia	
+	+	+		4	
Mild to moderate	IM only in the	Atrophy OR IM in		Endoscopic reassessment at a reference center with HD-0	
atrophy only in	ophy only in antrum OR IM		am and	4	
the antront, no wi	only in the corpus	corpe	15.	Visible lesion?	
Family history of gastric cancer ¹ , incomplete IM ¹ , autoimmune gastritis, or persistent H. pylori infection		+		4	+
		First-degree family history of gastric cancer ²		No*	Yes
				J.	+
				HD-CE in 6 months (high grade dysplasia)	Staging and
				to 12 months (low grade dysplasia)	resection
No	Yes	No	Yes	4	
				If no visible lesion (re)stage gastritis and	
$\downarrow \downarrow$	4	4	4	tonow up accordingly	1
No surveillance	S.	arveillance pr	referential	y with HD-CE with guided biopsies of irregular a	reas
	1	4	Ţ		1
		Every 3 years		Every 1-2 years	

Diagnosis:

Chronic-atrophic gastritis with complete and incomplete intestinal metaplasia.

Comment:

Upon endoscopy, the mucosa appeared largely normal, only slightly irregular (not shown). Routine biopsies were taken from antrum and corpus mucosa and showed mild expansion of the lamina propria by mononuclear cells, i.e. lymphocytes and plasma cells, no active inflammation (Panel A). There was moderate gland atrophy. Helicobacter stains were negative.

Metaplastic intestinal differentiation of the mucosa was frequently observed and can be described as follows.

In some parts, we detected well-defined goblet cells alternating with eosinophilic enterocytes displaying a luminal brush border also known as glycocalyx (Panels A-B). At the bottom of some of these glands, Paneth cells were present within the crypt epithelium (Panel C). These parts qualified for classification as "complete-type" intestinal metaplasia.

In other parts, goblet cells were less well defined and were found amongst gastric foveolar cells containing apical mucin droplets of varying sizes; enterocytes with brush border (and also Paneth cells) were lacking (Panel D). These parts qualified for classification as "incomplete-type" intestinal metaplasia.

Does this distinction matter?

Yes, it does. Gastric cancer risk is higher when incomplete intestinal metaplasia (defined by intestinal differentiation in the absence of enterocytes with brush border and Paneth cells) is identified compared with complete-type intestinal metaplasia (defined by the presence of enterocytes with brush border and/or Paneth

cells). Accordingly, individuals with incomplete intestinal metaplasia have shorter surveillance intervals (Panel E; MAPS II Guidelines 2019).

Two final remarks:

Slight nuclear irregularities are common within the proliferation compartment of metaplastic glands and can also be observed here. They may be marked when active inflammation is present and have been referred to as metaplastic atypia. Their presence does not bear any clinical consequences. Surface maturation discriminates these changes from dysplasia.

To correctly identify incomplete intestinal metaplasia on an H&E stain, there needs to be enough haematoxylin included, which stains goblets cells somehow bluish in contrast to neighbouring foveolar cells that appear more pinkish (compare Panel D for illustration). This is relevant also for biopsies from the gastroesophageal junction, where Barrett's metaplasia has to be differentiated from "pseudo-goblet cells" within cardiac mucosa.

For further reading:

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