Short communication

# Is there a correlation between autoimmune and alkaline gastritis?

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Reactive gastropathy is a common diagnosis in gastric biopsies and reflects the stomach's response to a variety of irritants including drugs, chronic alcohol consumption as well as reflux of duodenal contents into the stomach. The latter condition, known as alkaline gastritis, has been accepted by the Sydney System as a distinct entity [1]. It results from pyloric sphincter dysfunction caused by pyloroplasty, partial gastrectomy (Billroth I or II) and age-related sphincter soothing.

Studying routine gastric biopsies, we observed rather frequent coexistence of alkaline and autoimmune gastritis. As histological features of the antral mucosa in autoimmune gastritis (AIG) may have been overlooked, we retrospectively studied gastric biopsies in order to identify a possible correlation of alkaline gastritis with autoimmune gastritis, considering that such an association has not yet been reported.

From our archives, we retrieved 1149 gastric biopsies obtained over a one-year period (2014-2015). Biopsies from gastric stumps were not included. All autoimmune gastritis cases were re-evaluated and diagnosis was based on the following appropriate criteria: lesions confined to the corpus/fundus, intense lymphoplasmacytic infiltration of lamina propria, oxyntic glands atrophy, pseudopyloric and intestinal metaplasia [2, 3] as well as enterochromaffin-like (ECL) cell proliferation evaluated on immunohistochemically stained sections for chromogranin A. Histological diagnostic criteria for alkaline gastritis included no or mild inflammation, foveolar hyperplasia, elongation and tortuosity of gastric pits, mucosa villiform transformation, mucin paucity and angulated glands, edema with dilated capillaries and upward extension of smooth muscle fibers from the muscularis mucosa into the lamina propria.

*Helicobacter pylori* gastritis was found in 228 cases (19.8%), chemical/alkaline gastritis in 88 (7.66%) cases, and non-specific gastritis in 384 (33.4%) cases, whereas no histological changes were observed in 385 (33.5%) cases. Sixty four (5.57%) biopsies carried the diagnosis of autoimmune gastritis. Upon review of these 64 biopsies, atrophy of the oxyntic mucosa was severe in 50, moderate in 10 and mild in 4 cases, while 12 cases (19%) exhibited additional findings of mild and less frequently moderate alkaline gastritis of the antral mucosa (Fig. 1). The latter subgroup had no clinical history of drug or alcohol abuse. No significant correlation was found between coexistence of the aforementioned gastritis types and demographic and histological parameters.

Although the currently established histology of AIG is characterized by a relatively normal antral mucosa, our observations suggest alkaline gastritis to be a rather common additional finding. The only indication of the presence of alkaline gastritis is the reported observation of antral foveolar hyperplasia in AIG, which however was attributed to the trophic effect of hypergastrinemia [4]. The pathogenesis of alkaline gastritis has not been fully elucidated. Prolonged exposure of the gastric mucosa to bile contents and in particular to the toxicity of bile acids is considered to be a potential pathogenetic mechanism [5, 6]. The occurrence of alkaline gastritis in the context of AIG could be ascribed to hypochlorhydria and the ensuing deficient neutralization of the refluxed

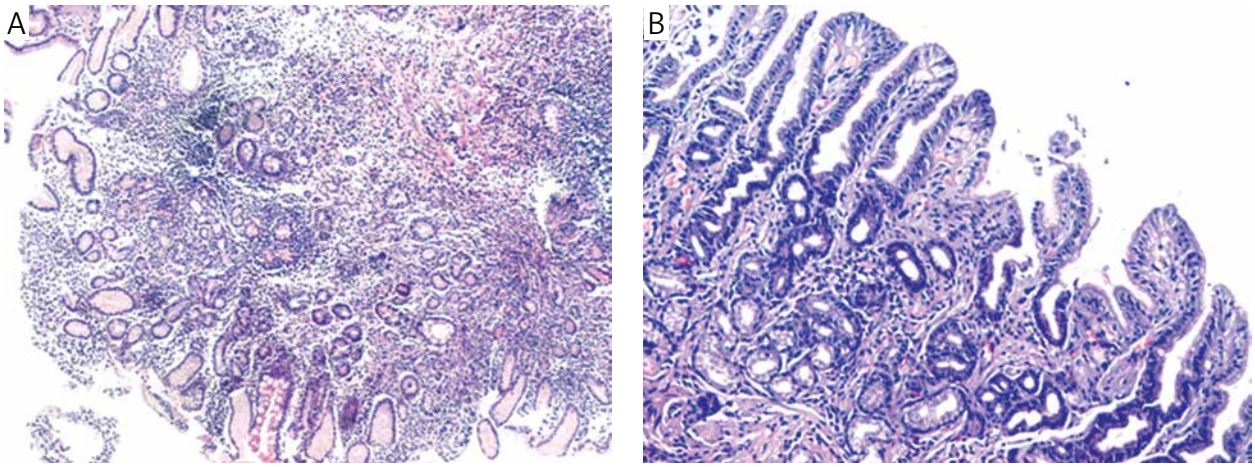


**Fig. 1.** Autoimmune gastritis: chronic gastritis with severe atrophy, pseudopyloric and focal intestinal metaplasia (A). Alkaline gastritis: elongation and tortuosity of gastric pits, villiform transformation, mucin paucity, gland angulation and upward extension of smooth muscle fibers (B)

bile acids. A possible synergistic role of age-related pyloric sphincter dysfunction cannot be excluded. A prospective study should be performed in order to clarify the significance of our observation.

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*The authors declare no conflict of interest.*

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