

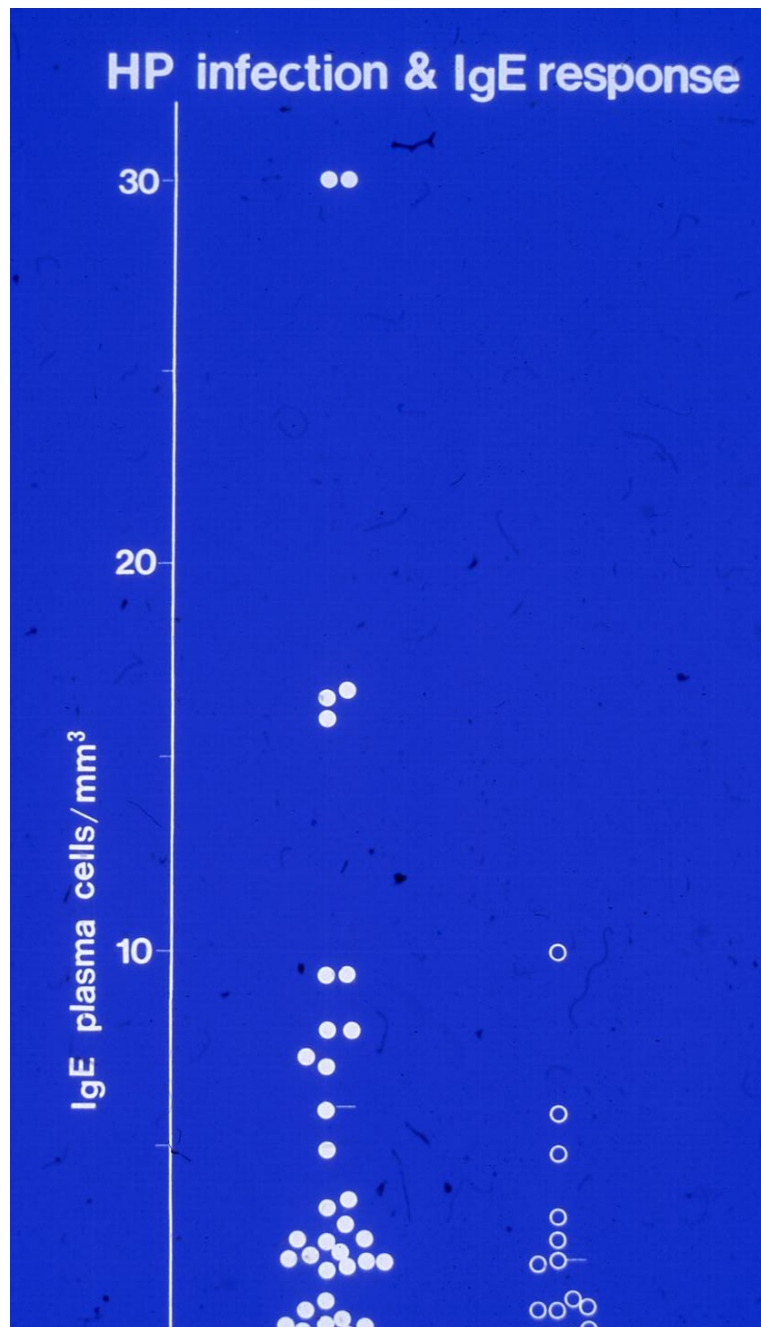
Helicobacter pylori infection and IgE response in the gastric mucosa

In the gastric mucosa, IgA plasma cells predominate, and IgM plasma cells follow. IgG plasma cells are increased in response to *Helicobacter pylori* infection, and lymphoid follicle formation is activated. In foci of intestinal metaplasia, IgG plasma cells and lymphoid follicles significantly decrease.

IgE plasma cells are increased in the lamina propria mucosae, in response to *H. pylori* infection. IgE deposition in the germinal center (on the follicular dendritic cells) is also observed. The number of eosinophils and mast cells is significantly increased in *H. pylori* gastritis. Allergic responses against *H. pylori* may be functioning in the *H. pylori* gastritis.

Ref.-1: Tsutsumi Y, et al. Immune aspects of intestinal metaplasia of the stomach: an immunohistochemical study. Virchows Arch A Pathol Anat Histopathol 1984; 403(4): 345-359. doi: 10.1007/BF00737285

Ref.-2: Kamoshida S, et al. Detection of *H. pylori* in the gastric mucosa and its histopathological significance. Byori-to-Rinsho 1995; 13(12): 1689-1698 (in Japanese).



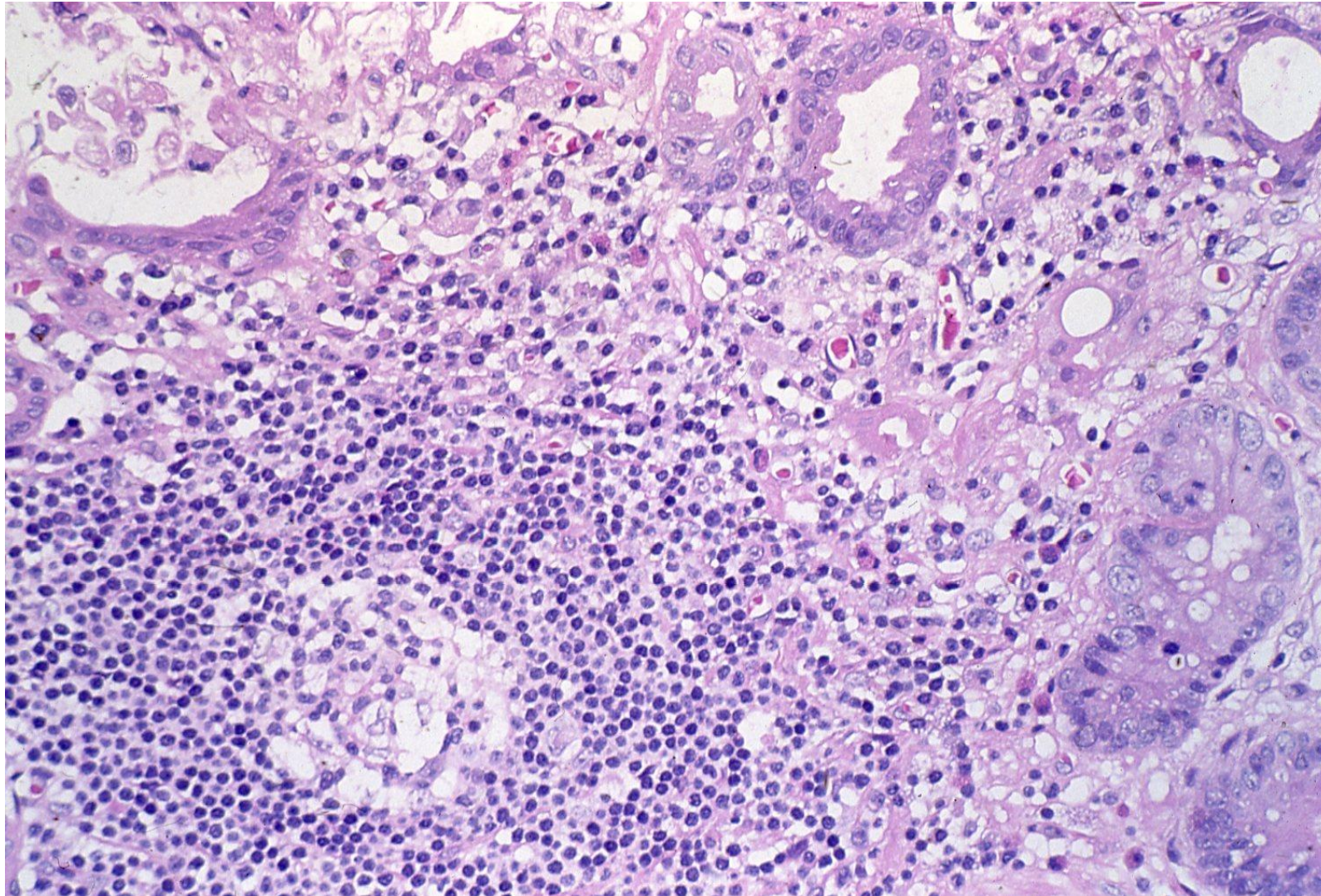
IgE plasma cells are significantly increased in *H. pylori*-infected gastric mucosa

Table 1. IgE plasma cells in patients with or without *Helicobacter pylori* infection

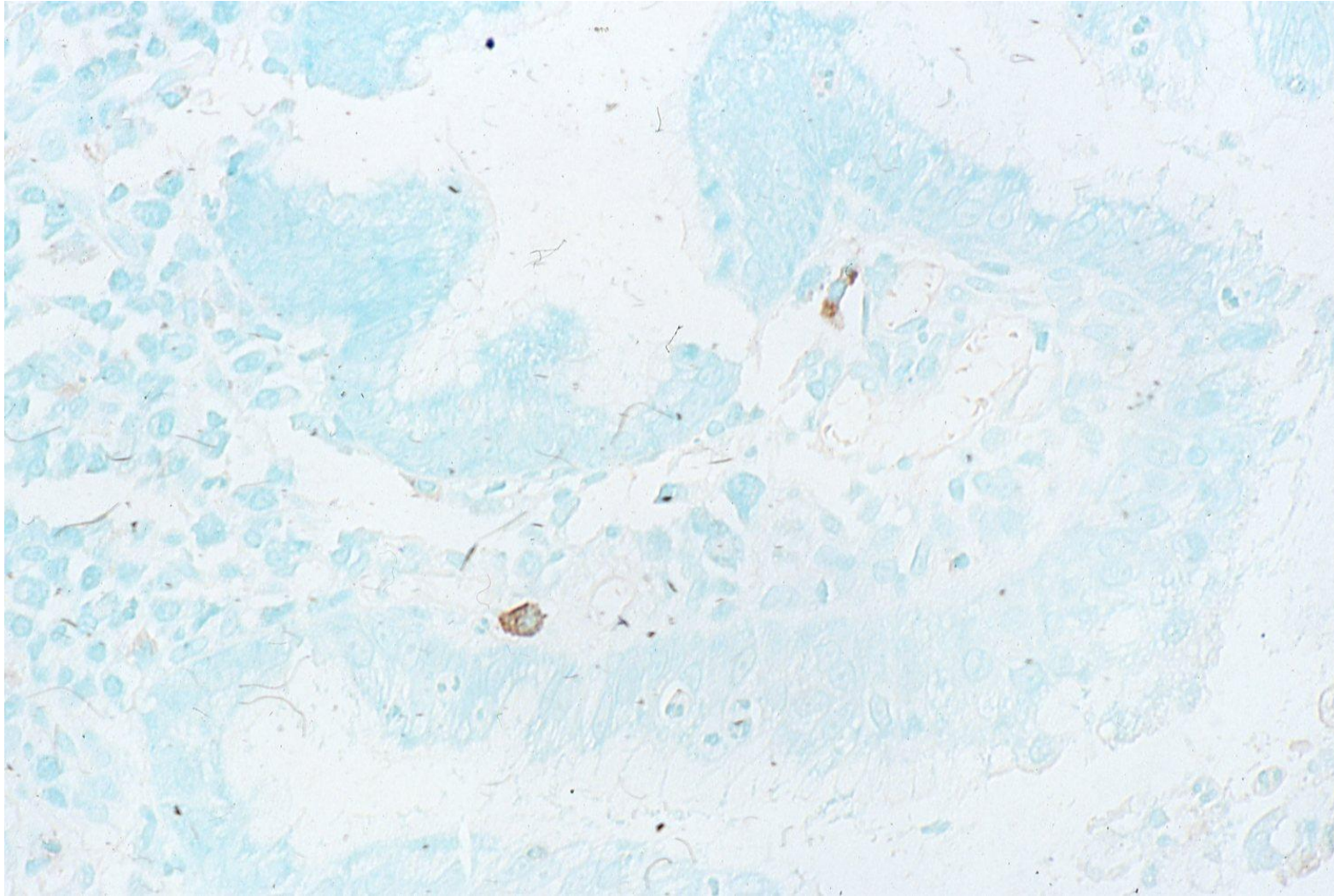
<i>Helicobacter pylori</i>	No. of IgE plasma cells (/mm ²)		
	< 0.5	0.5~1.5	> 1.5
Positive (n=65)	30(46.2)	21(32.3)	14(21.5)
Negative (n=35)	23(65.7)	12(34.3)	0(0)
Total (n=100)	53(53.0%)	33(33.0%)	14(14.0%)

Lymphoid follicles with IgE-deposited germinal centers in chronic gastritis

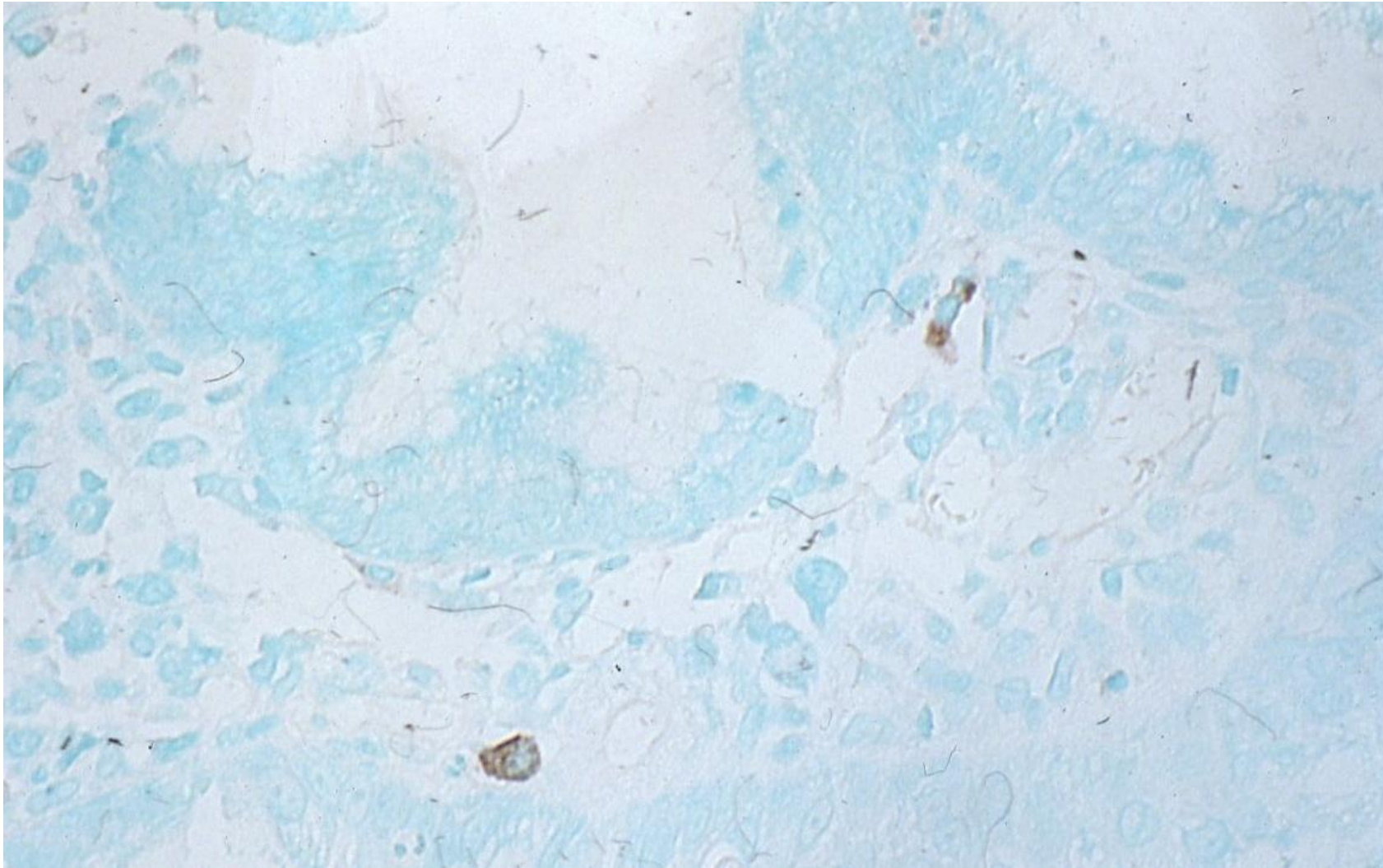
- 1) Incidence of IgE-positive germinal centers:
 surgical cases 7/47 (15%)
 biopsy cases 3/31 (10%)
- 2) *Helicobacter pylori*-positive
- 3) The germinal centers are small-sized, occasionally with hyaline change
- 4) IgE-bound mast cells are increased
- 5) Eosinophilic infiltration is observed in more than half of cases
- 6) Intestinal metaplasia is frequently seen adjacent to IgE-deposited germinal centers.



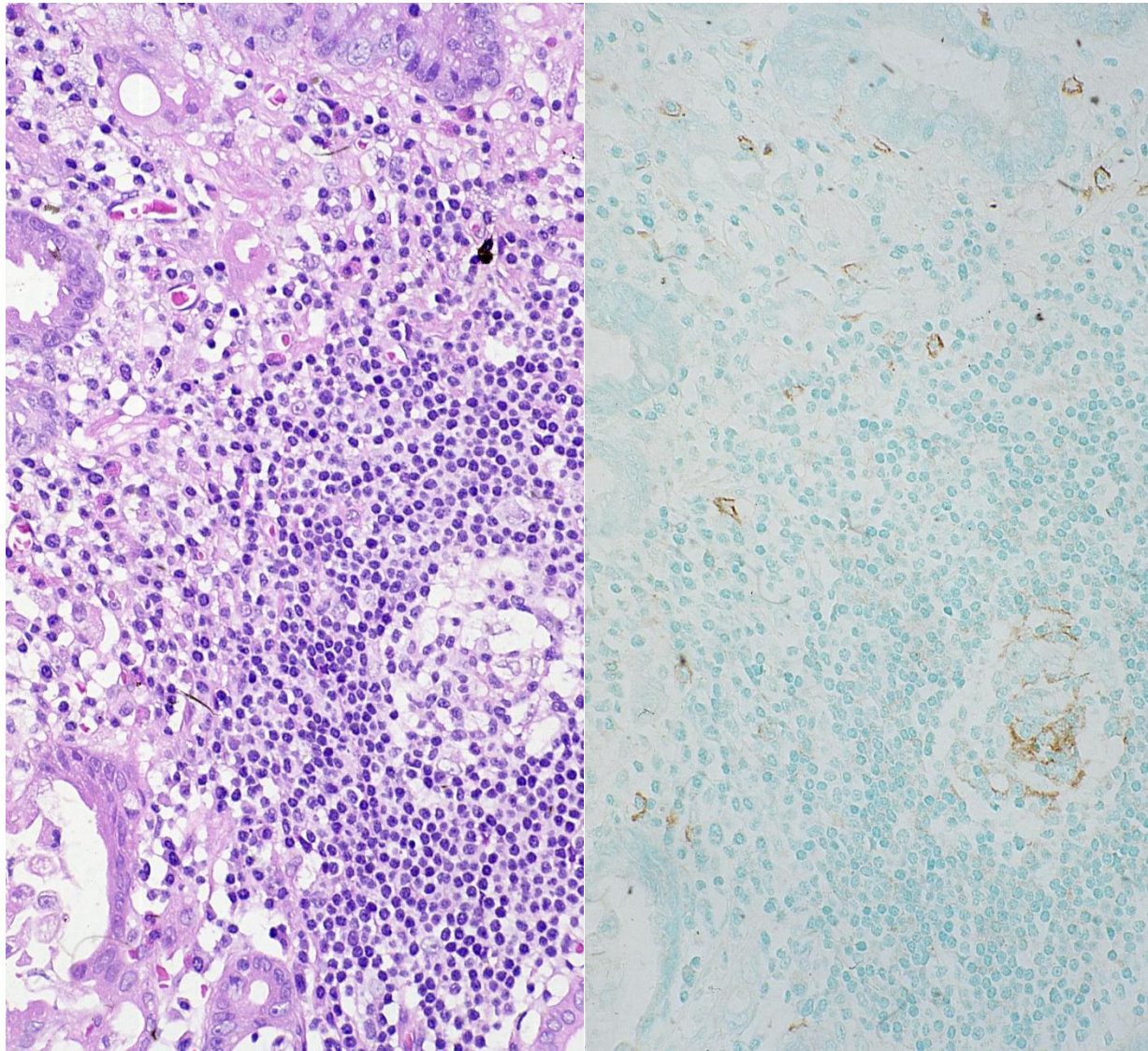
H. pylori-infected gastritis frequently accompanies formation of lymphoid follicles with germinal centers. H&E



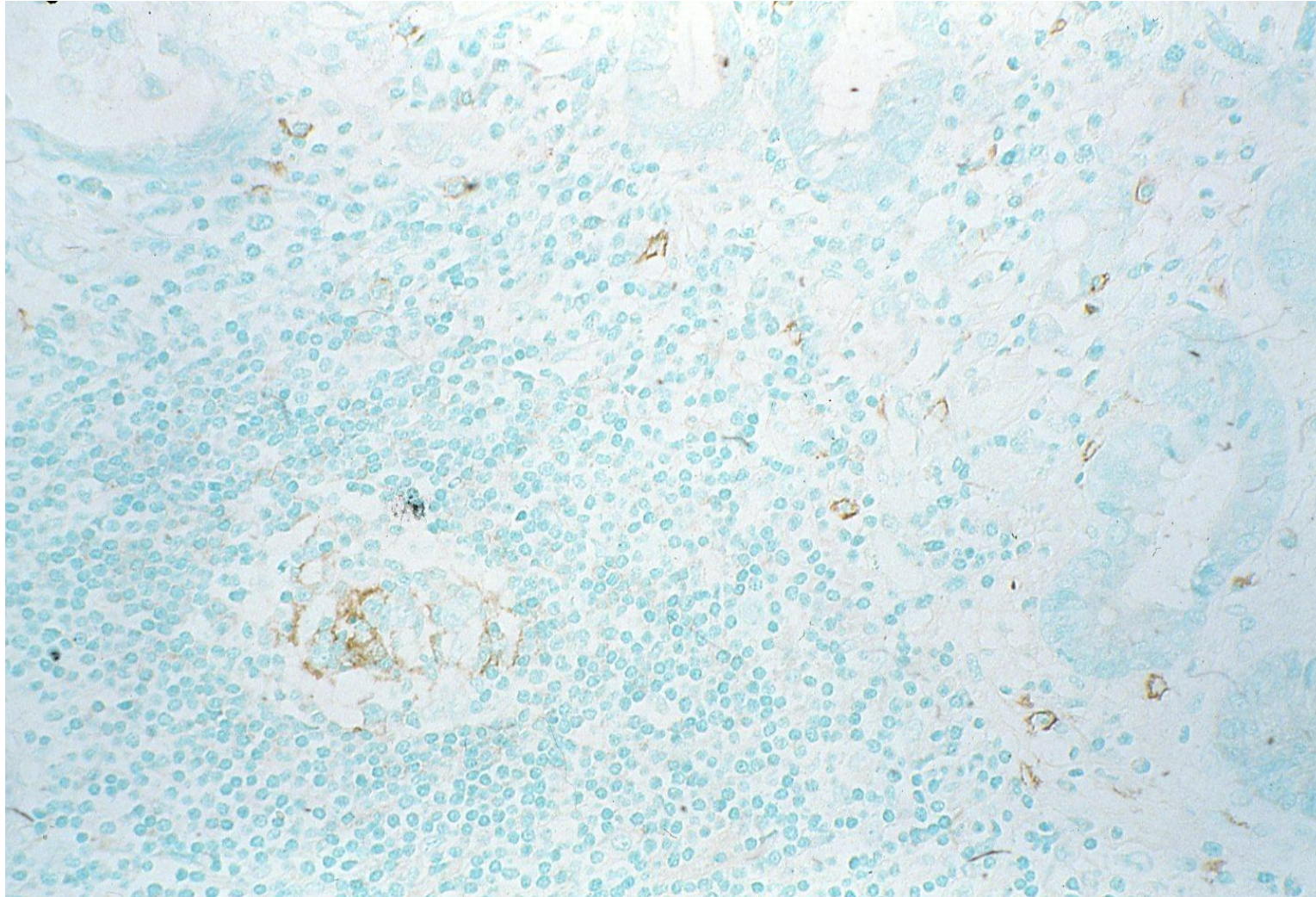
IgE plasma cells are scattered in the lamina propria mucosae of *H. pylori*-infected gastritis. Immunostaining for IgE (1)



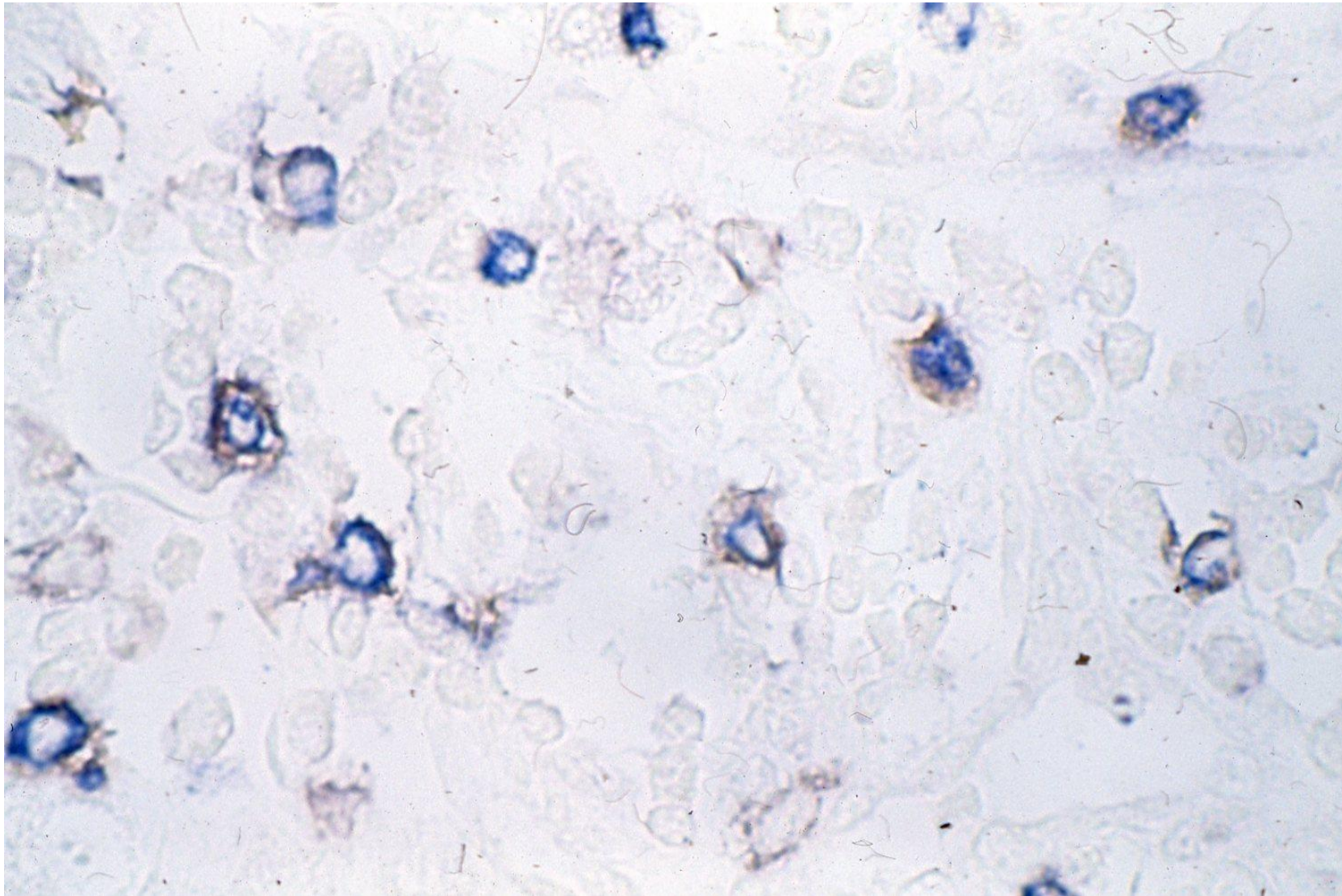
IgE plasma cells are scattered in the lamina propria mucosae of *H. pylori*-infected gastritis. Immunostaining for IgE (2)



H. pylori-infected gastritis frequently accompanies formation of lymphoid follicles with germinal centers. Deposition of IgE is seen in the germinal center. IgE-bound mast cells are scattered. H&E (left) and immunostaining for IgE (right)



H. pylori-infected gastritis frequently accompanies formation of lymphoid follicles with germinal centers. Deposition of IgE is seen in the germinal center. IgE-bound mast cells are scattered. Immunostaining for IgE



Mast cells in *H. pylori*-infected gastritis. IgE is bound to the plasma membrane of mast cell tryptase-positive mast. Double immunostaining for mast cell tryptase (blue in color) and IgE (brown in color).