Gross appearance of perforated duodenal ulcer and the significance of gastric metaplasia associated with duodenal ulcer

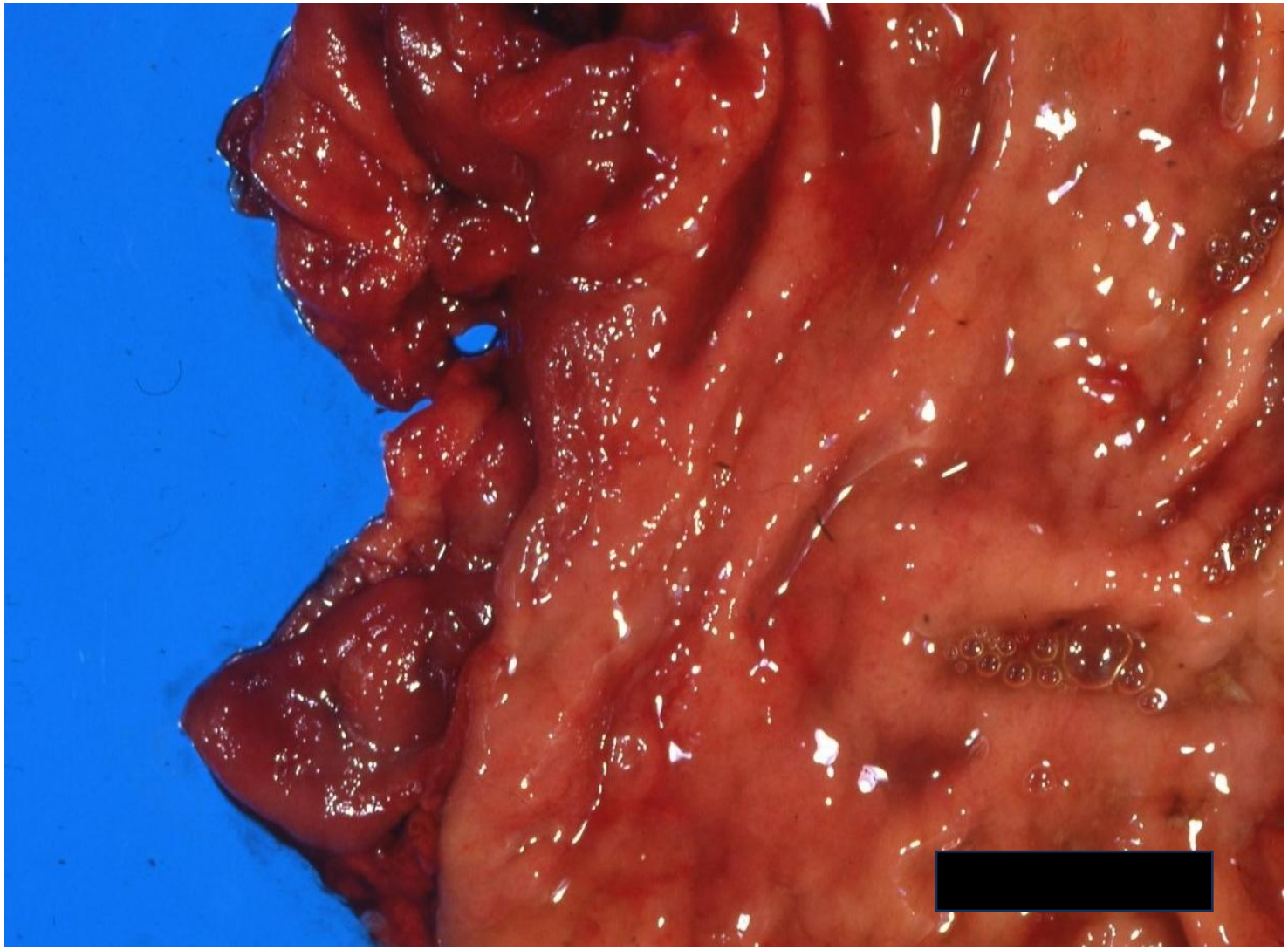
Peptic ulcer disease is provoked by *Helicobacter pylori* infection and the use of non-steroidal anti-inflammatory drugs. *H. pylori* induces gastric metaplasia, stimulates immune response and gastric acid secretion, and reduces mucosal defense, causing duodenal mucosal injury and subsequently duodenal perforation. Gastric metaplasia, frequently seen in biopsy specimens of the duodenal bulb, results from chronic active inflammation and/or duodenal ulceration. Small patches of gastric-type foveolar cells appear at the tip of the duodenal villus. The mucous cells contain PAS-reactive, galactose oxidase-Schiff-reactive and alcian blue-negative neutral mucins. Gastric metaplasia may function as a protecter against high acidity, but instead neuroendocrine cells containing somatostatin (D cells), secretin (S cells) and GIP (gastric inhibitory peptide) cells are significantly decreased. These neuroendocrine cells are known to suppress acid secretion from the stomach as a duodenal brake. The gastric metaplasia-related impairment of the duodenal brake function might lead to the recurrence of duodenal ulcer.

Duodenal perforations can either be free or contained. Free perforation arises when bowel contents leak freely into the abdominal cavity to cause diffuse peritonitis. Contained perforation occurs when the ulcer creates a full-thickness hole, but free leakage is prevented by contiguous organs such as the pancreas and omentum. Duodenal free perforation causes a sudden onset of severe pain in the upper abdomen and the leakage of the duodenal content results in diffuse peritonitis.

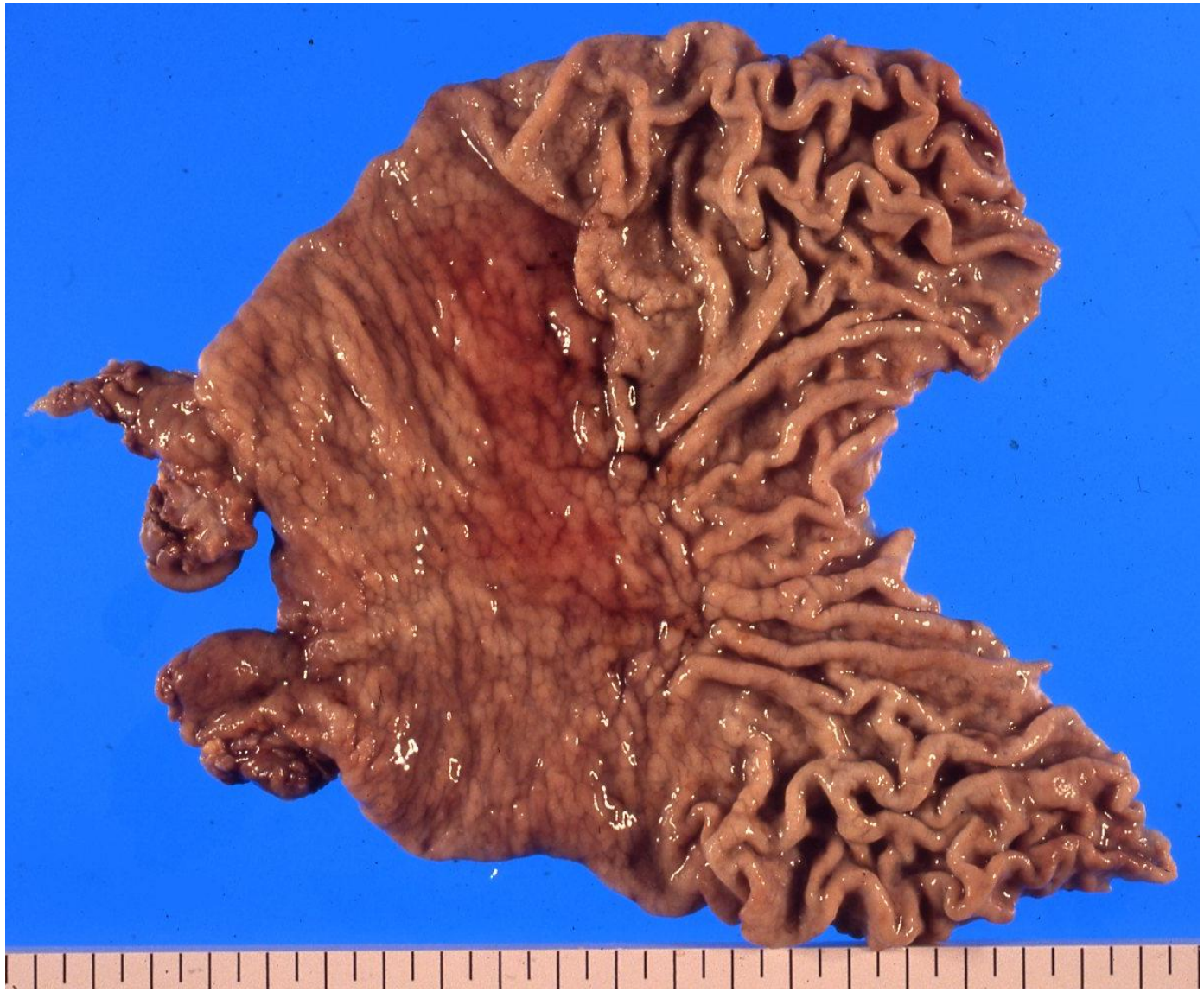
Ref.-1: Shaoul R, et al. The pathogenesis of duodenal gastric metaplasia: the role of local goblet cell transformation. Gut 2000; 46(5): 632-638. doi: 10.1136/gut.46.5.632

Ref.-2: Hara M, et al. Pathophysiology of duodenal ulcer. Immunohistochemical study on the endocrine cells in gastric metaplasia of the duodenal bulb mucosa. Igaku-no-Ayumi 1985; 134(1): 45-46 (in Japanese).

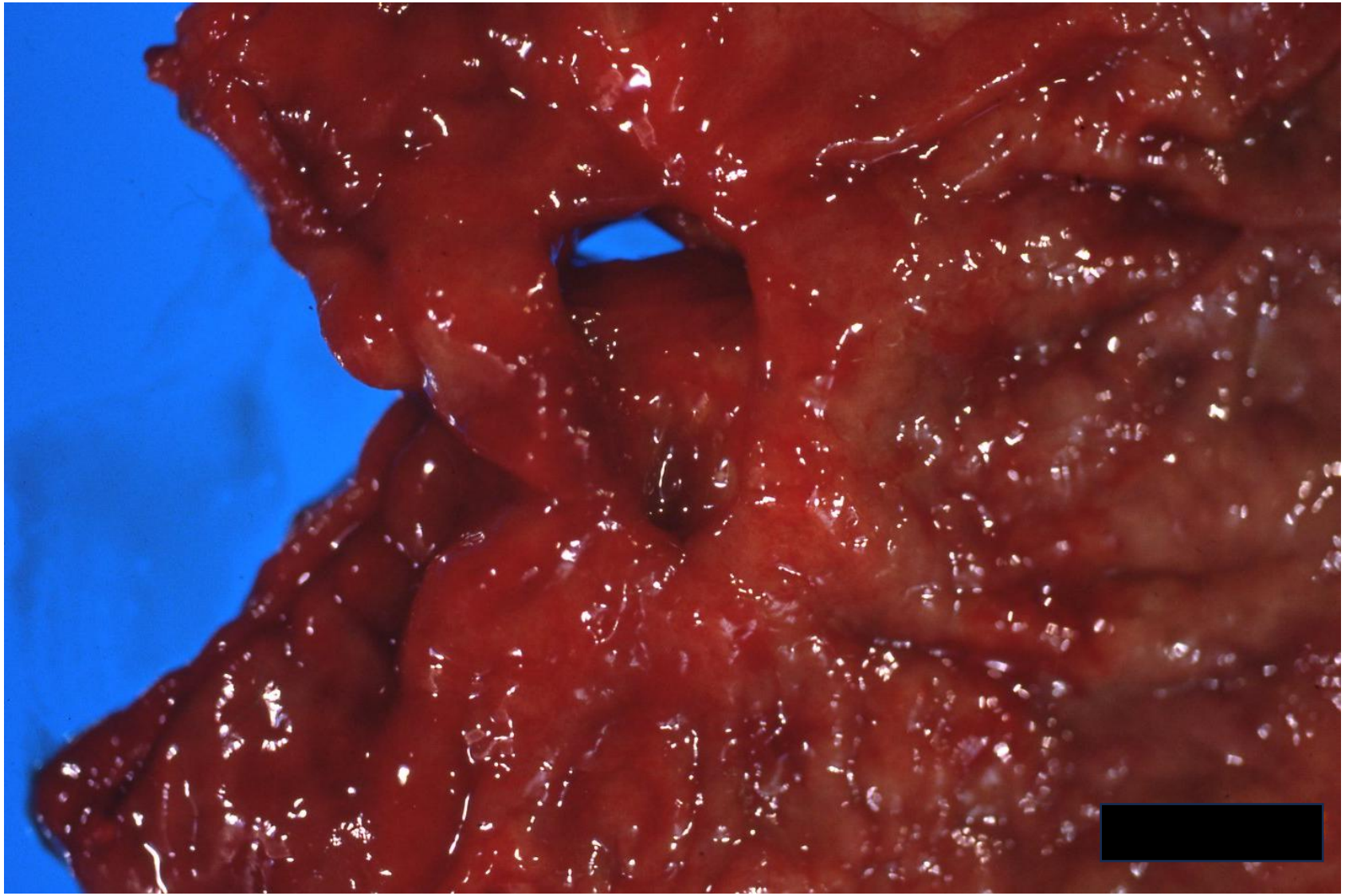
Ref.-3: Amini A, Lopez RA. Duodenal perforation. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023. Available from: https://www.ncbi.nlm.nih.gov/books/NBK553084/



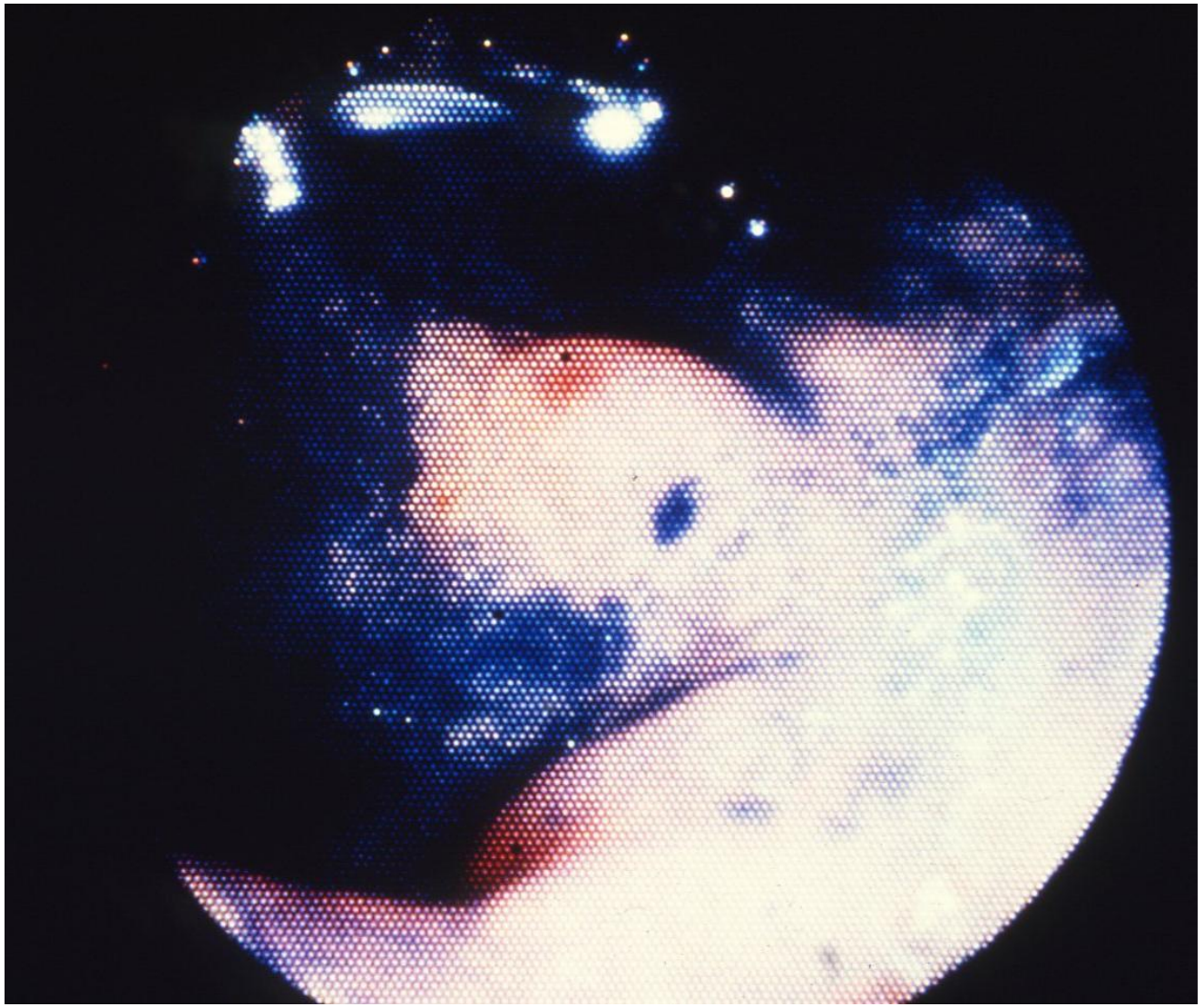
Perforated duodenal ulcer surgically removed from a 26 y-o male patient.
A small perforated hole is seen in the duodenal bulb (gross appearance).



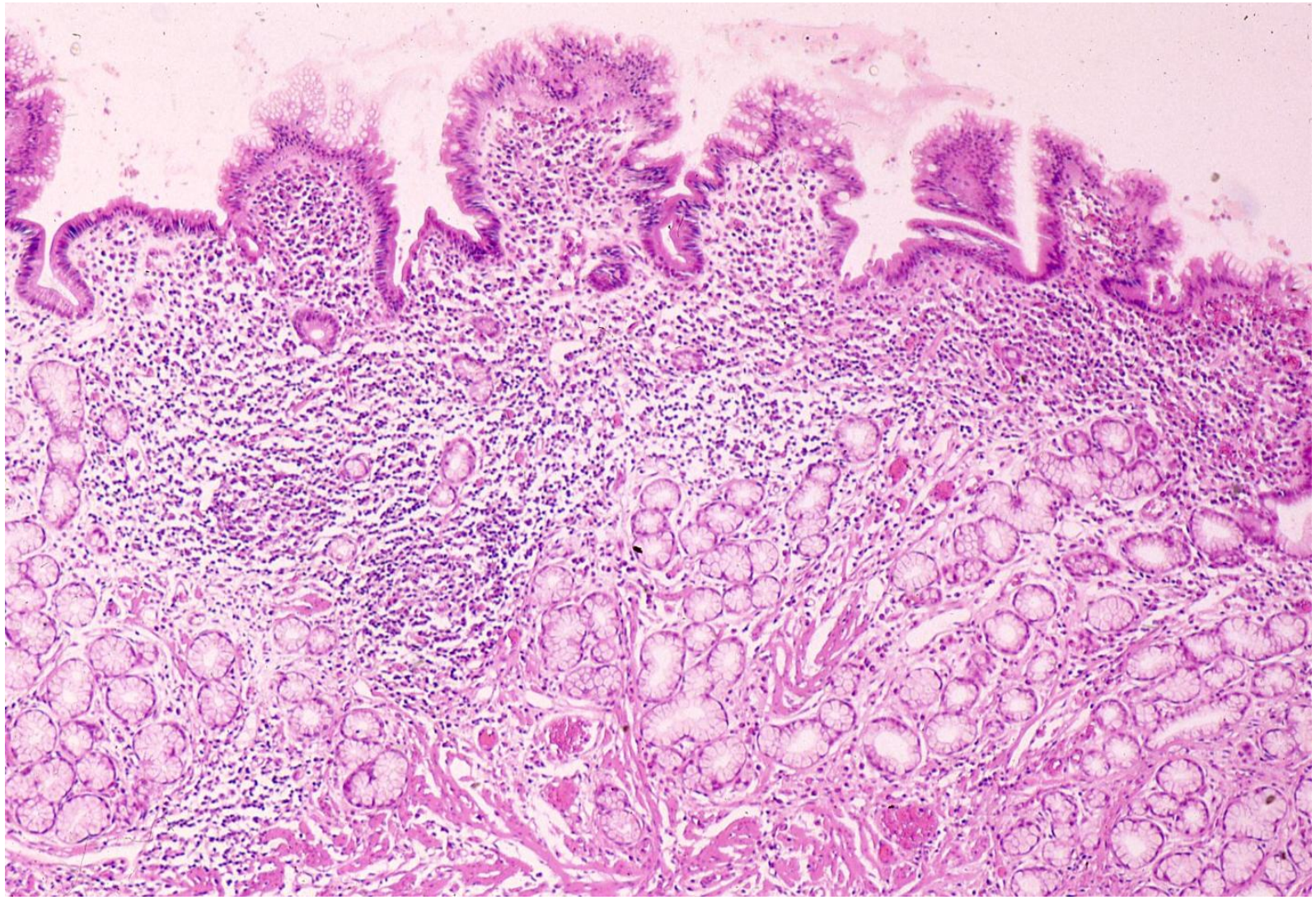
Perforated duodenal ulcer surgically removed from a 43 y-o male patient.
A large perforated hole is seen in the duodenal bulb (gross appearance).



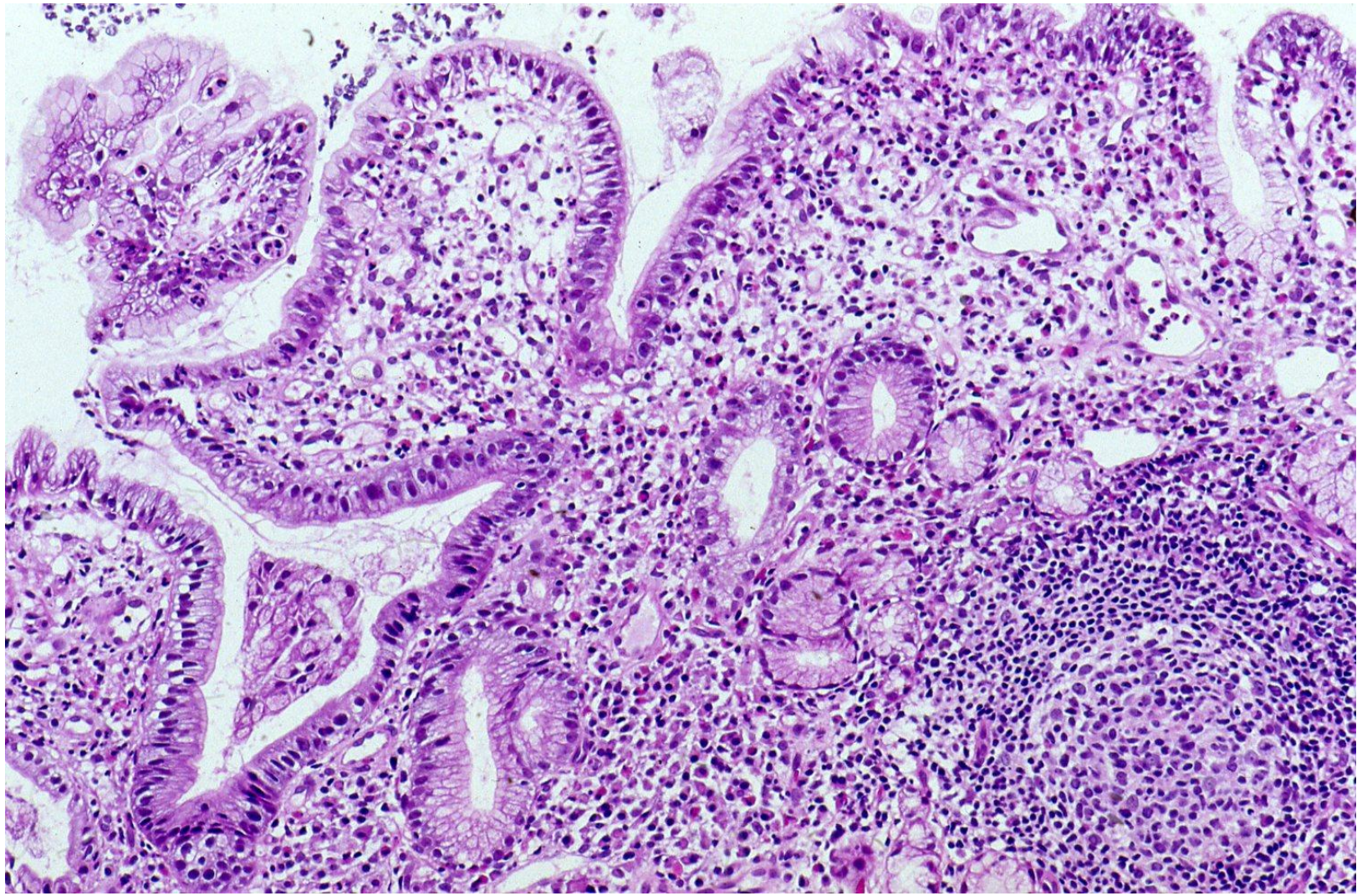
Perforated duodenal ulcer surgically removed from a 63 y-o male patient. A large perforated hole is seen in the deformed duodenal bulb (gross appearance).



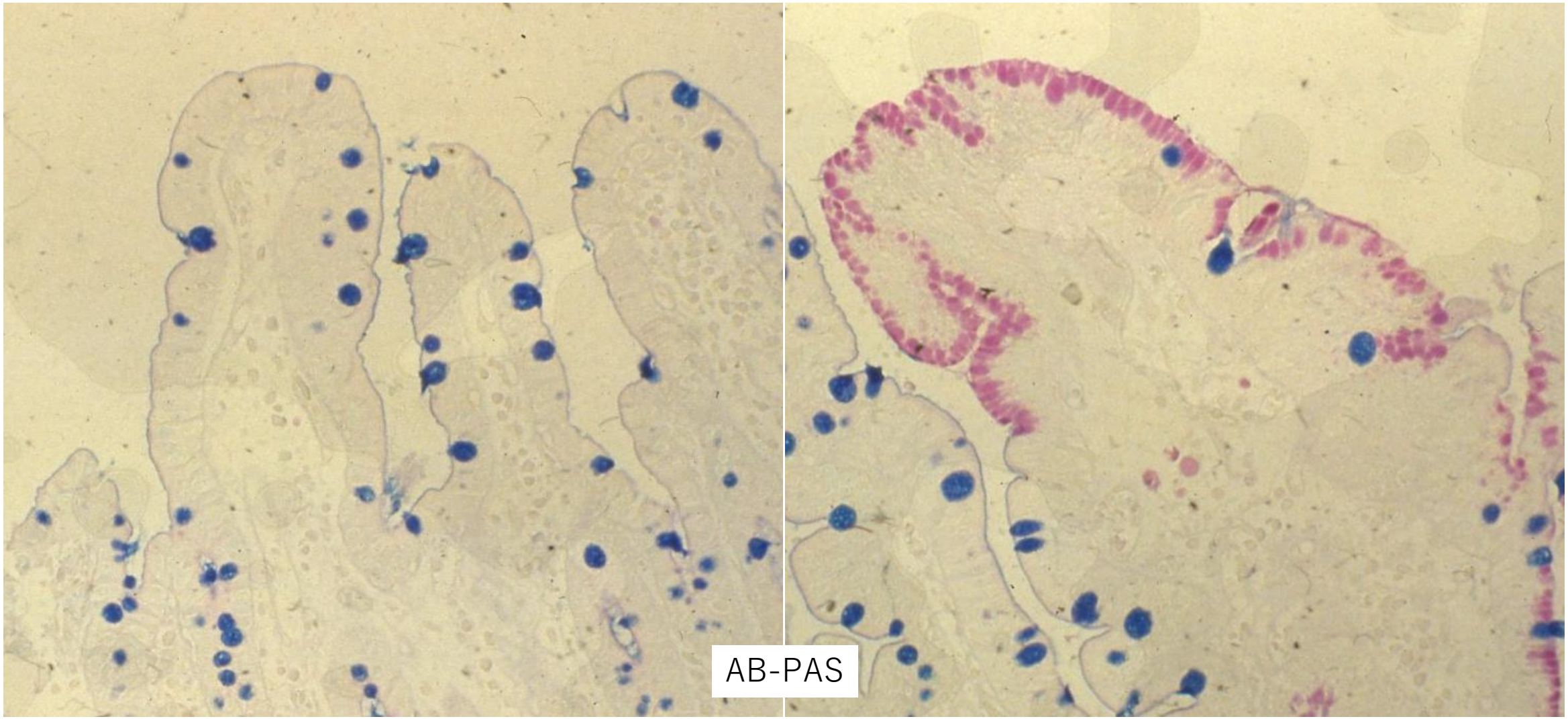
Chromoendoscopy of healed duodenal ulcer with gastric metaplasia seen in a male patient aged 30’s. The normal duodenal mucosa is diffusely stained blue because of the absorption of the dye. In the mucosal area with gastric metaplasia seen at the site of the healed duodenal ulcer, blue staining is lacking.



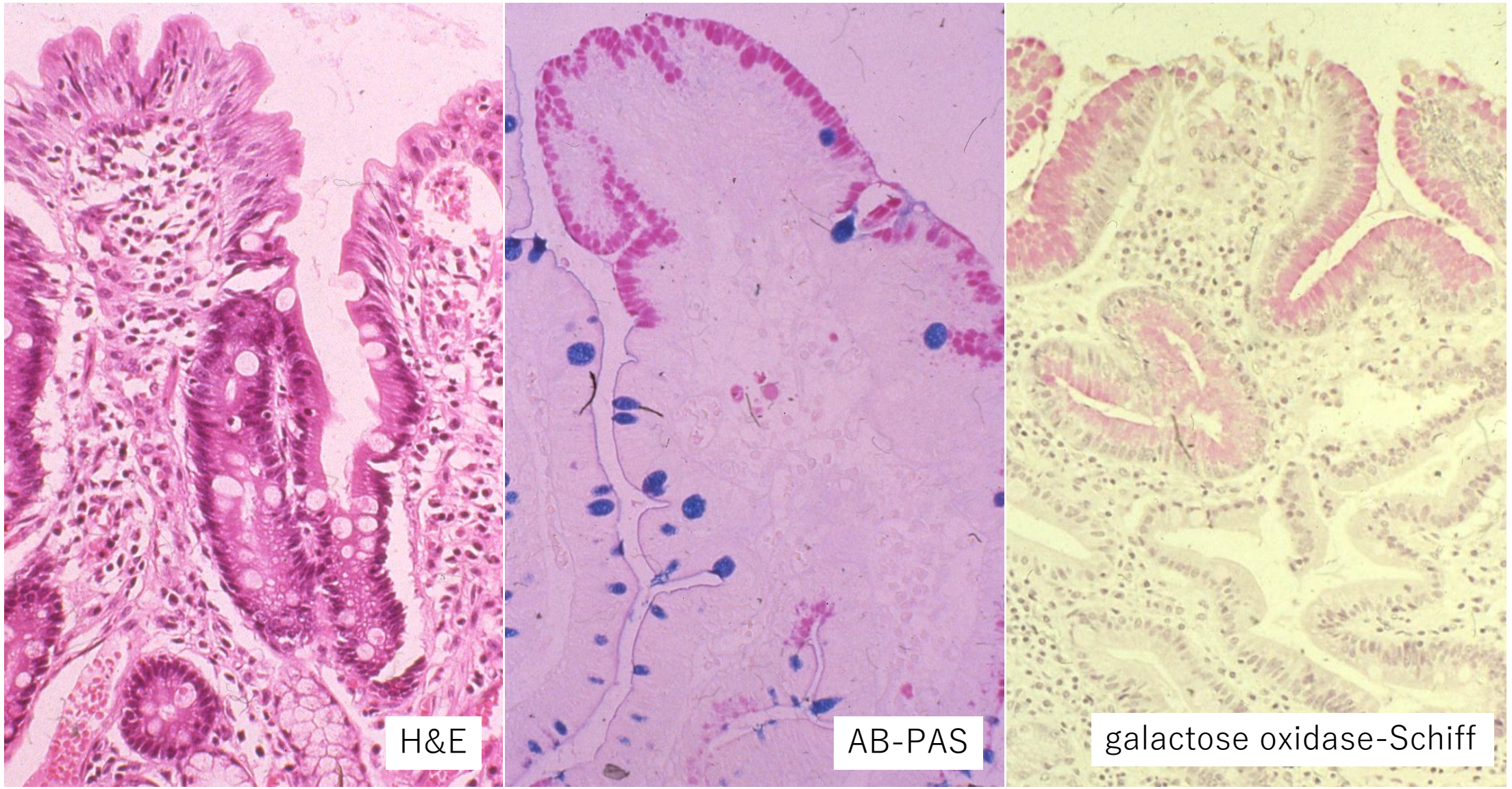
Healed duodenal ulcer with gastric metaplasia seen in a male patient aged 30’s (H&E). The shortened villi are lined by gastric foveolar-type mucous cells. Moderately active chronic inflammation is associated (chronic duodenitis).



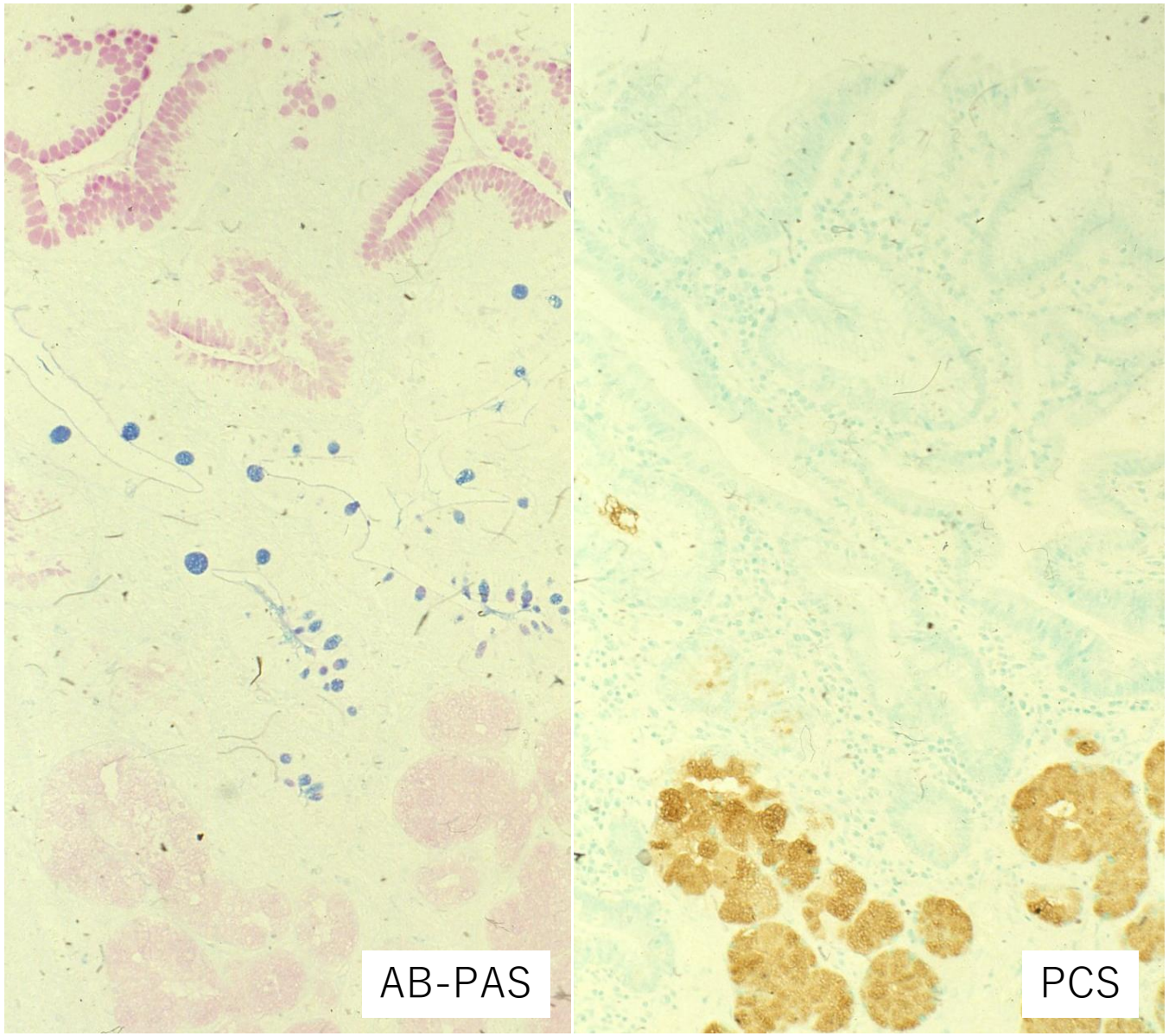
Healed duodenal ulcer with gastric metaplasia seen in a male patient aged 40’s (H&E). The shortened villi are lined by gastric foveolar-type mucous cells. Moderately active chronic inflammation with lymphoid follicle formation is associated (chronic duodenitis).



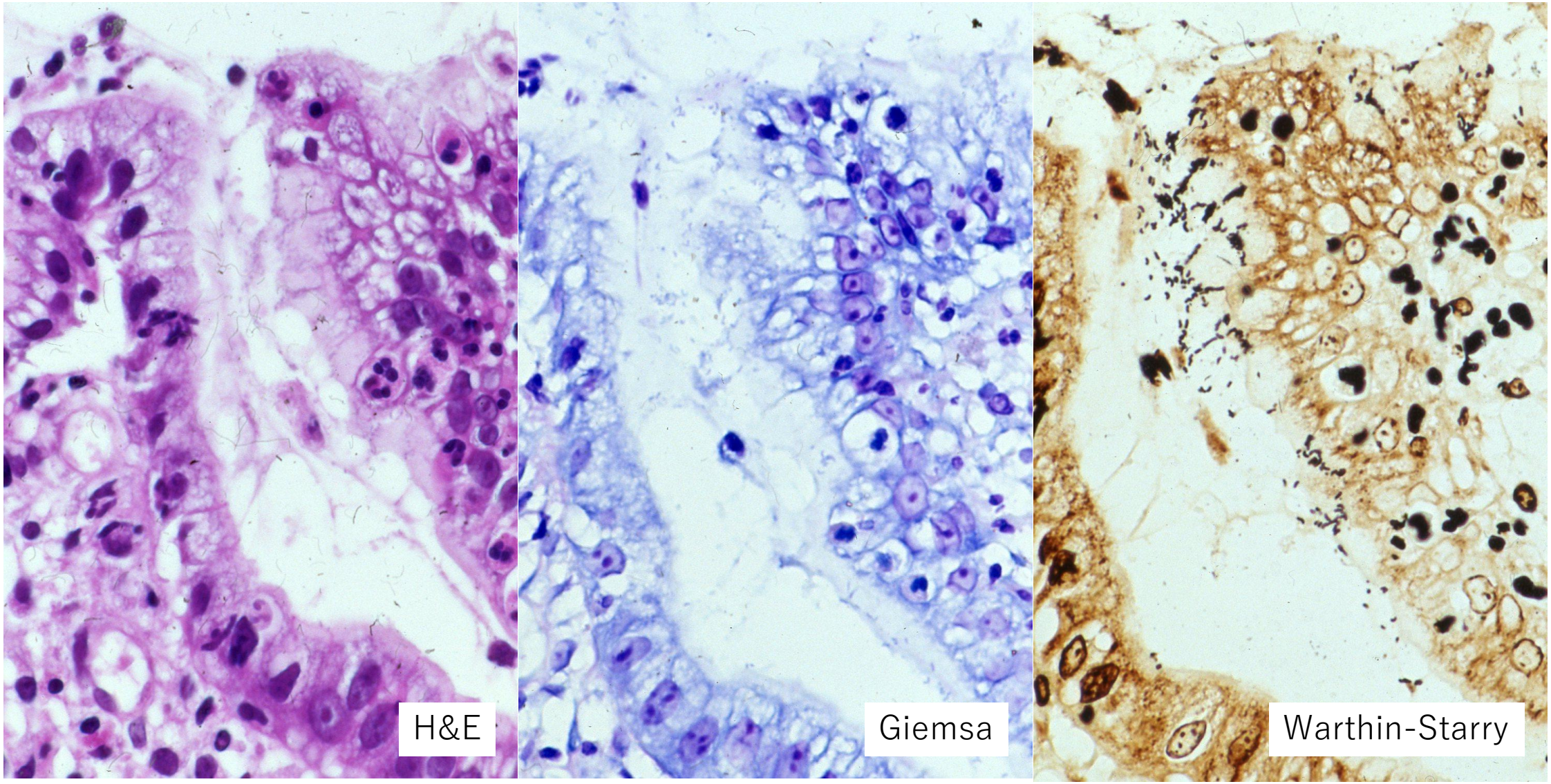
Normal (left) vs. gastric metaplastic (right) duodenal mucosa (AB-PAS). In the normal duodenal mucosa, goblet cells and the surface coat are stained blue with the Alcian blue dye. In chronic duodenitis with gastric metaplasia, the tip of the shortened villi is stained magenta with the PAS reaction. At the bottom of the villi, alcianophilic goblet cells are distributed.



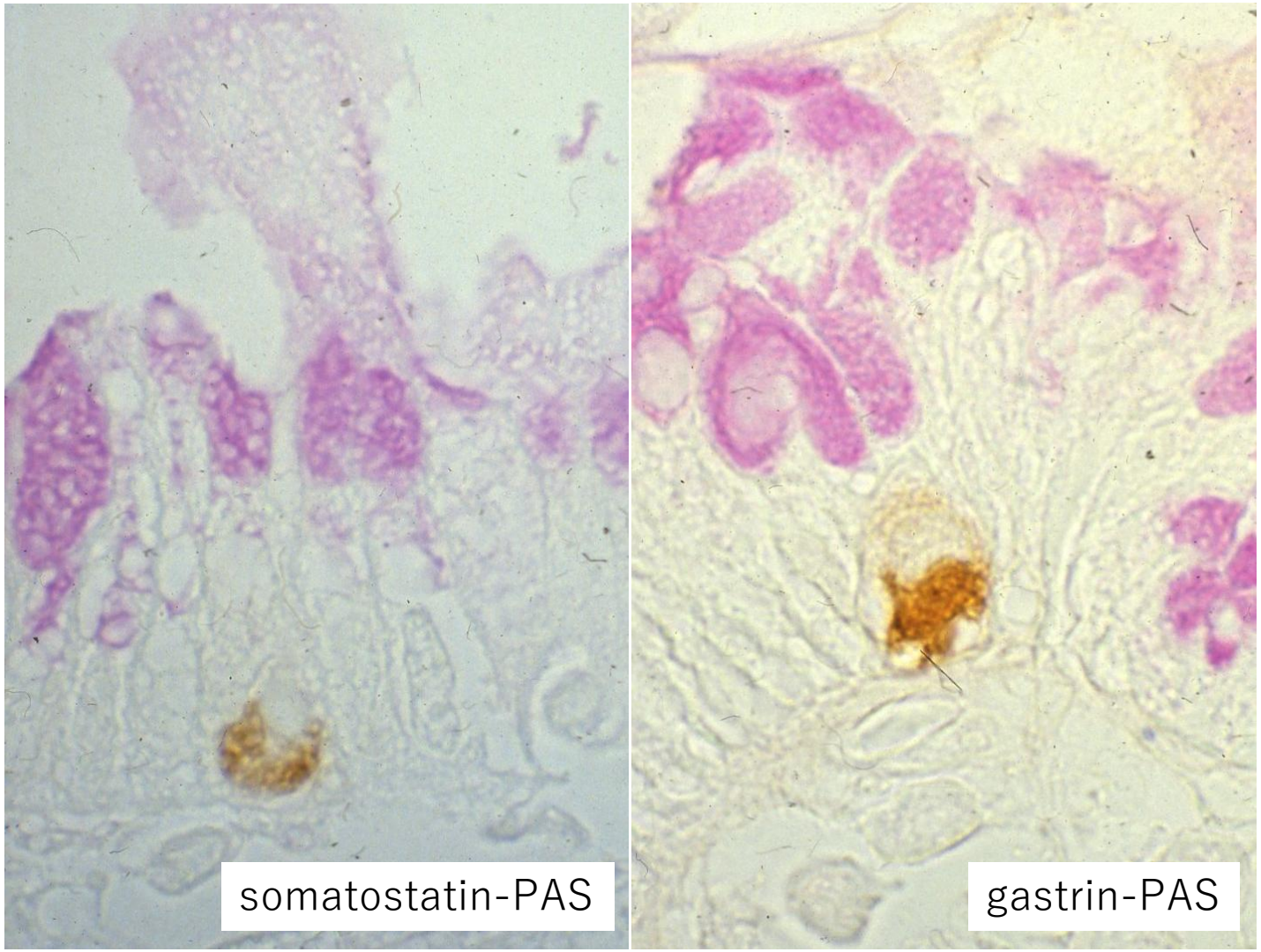
Gastric metaplastic of the duodenal mucosa associated with healed duodenal ulcer. The tip of the shortened villi is replaced by gastric foveolar-type mucous columnar cells (left: H&E). The gastric metaplasia contains neutral mucin (stained magenta with AB-PAS, center), and is stained magenta with galactose oxidase-Schiff reaction (right), as is in normal gastric foveolar cells.



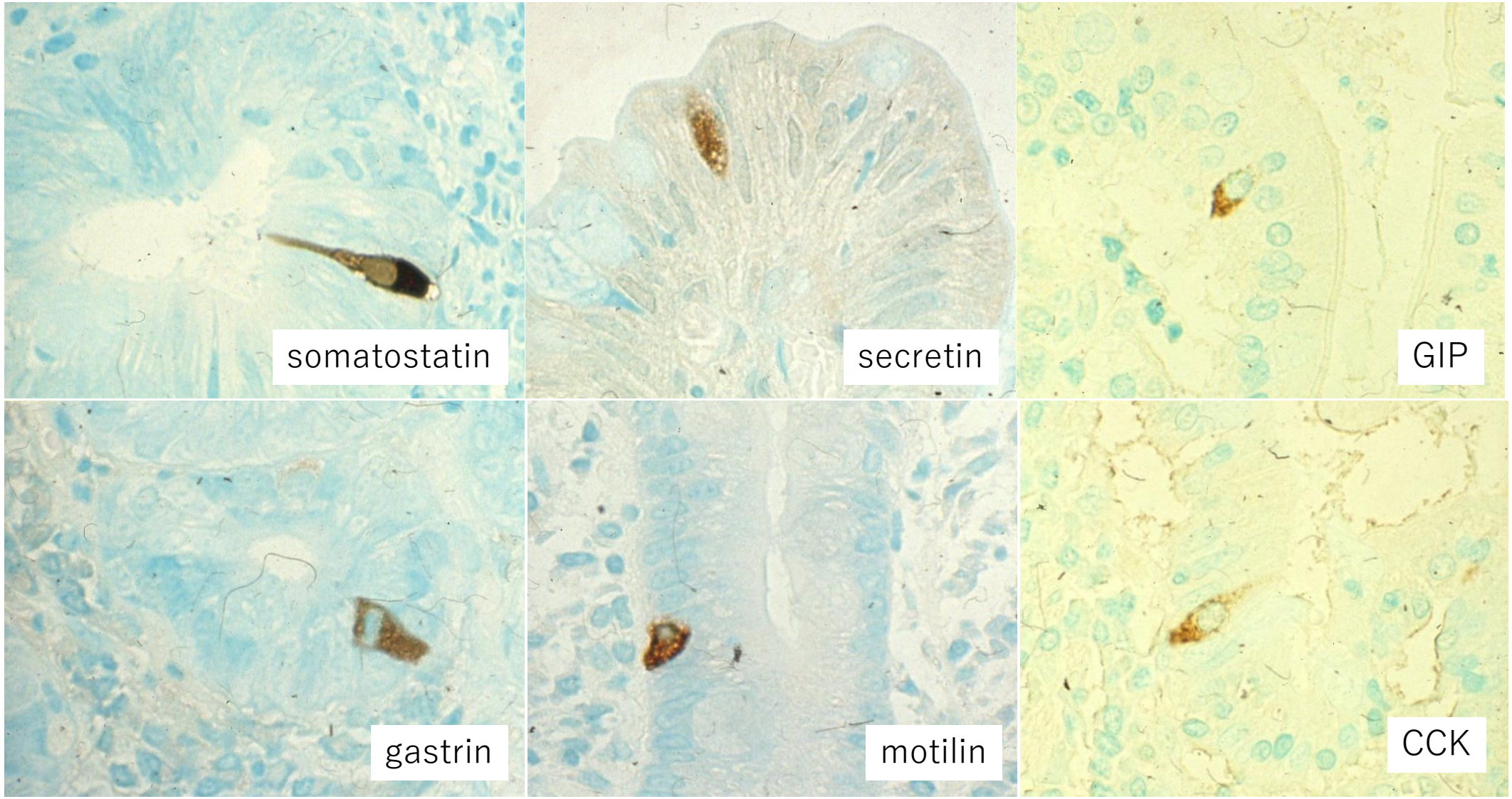
Duodenal mucosa with gastric metaplasia in a case of duodenal ulcer scar (left: AB-PAS, right: PCS: paradoxical Concanavalin A staining). In chronic duodenitis with gastric metaplasia, the tip of the shortened villi is stained magenta with the PAS reaction. PCS stains Brunner's gland cells in brown.



Duodenal mucosa with gastric metaplasia in a case of duodenal ulcer scar (left: H&E, center: Giemsa, right: Warthin-Starry's silver). On the surface of gastric foveolar-type cells, colonization of *Helicobacter pylori* can be observed.



Neuroendocrine cells in gastric metaplasia of the duodenal bulb mucosa. A few neuroendocrine cells immunoreactive for somatostatin (left) and gastrin (right) are observed, while the cells containing intestinal-type peptides (see next slide) are no longer seen, suggesting a loss of duodenal brake (See GI-235-1-small bowel). The loss of normal villous structure may cause the loss of the intestinal-type neuroendocrine cells producing secretin, motilin and GIP. The immunostaining was combined with PAS reaction.



Neuroendocrine cells in normal duodenal bulb. Immunostaining for 6 peptide hormones demonstrates somatostatin, gastrin, secretin, motilin, GIP (gastric inhibitory peptide) and CCK (cholecystokinin) in NE cells. Somatostatin, gastrin and CCK cells are mainly seen in the crypts, while secretin, motilin and GIP cells are predominantly distributed in the villi.