Bacillary dysentery, cholera and typhoid fever

Bacillary dysentery, cholera and typhoid fever are endemic or epidemic infectious diseases of intestine of Gram-negative bacilli in developing world. In the developed world, the diseases are encountered in travelers and immigrants.

Bacillary dysentery (shigellosis) is caused by infection of the Shigella genus, highly infective, virulent and invasive Gram-negative rods. It causes watery diarrhea with fever and abdominal pain, eventually progressive to dysentery (severe diarrhea with blood or mucus in stool). There are 4 groups of bacteria: *S. dysenteriae*, *S. flexneri*, *S. boydii*, *S. sonnei*. Shiga toxin released by *S. dysenetriae* provokes the most virulent disease with hemolytic uremic syndrome. The colon is the main target of infection. Clinicopathological features resemble those of shigatoxin-producing enterohemorrhagic *E. coli* O-157 infection.

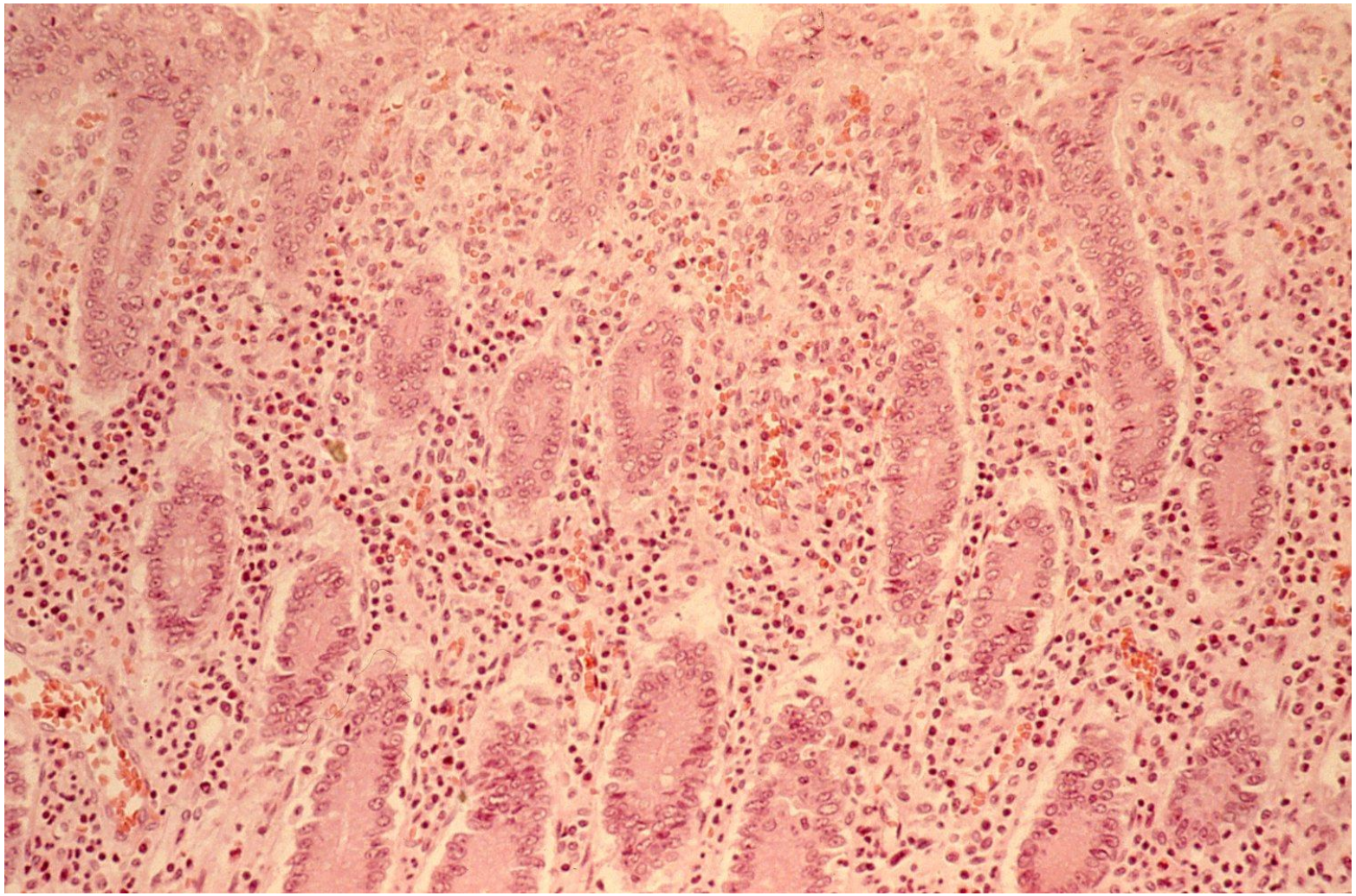
Cholera (infection of *Vibrio cholerae* O1 or O139, halophilic bacteria) accompanies acute watery diarrhea rapidly leading to dehydration, shock, electrolyte disturbances and renal failure. Latent infection is common. *V. cholerae* is a non-invasive pathogen, and the disease is provoked by exotoxins A and B. The site of infection is small bowel. No melena is encountered. Microscopically, the small bowel mucosa show only mild neutrophilic infiltration in the lamina propria without formation of erosions and ulcers.

Typhoid fever (septisemic *Salmonella typhi* infection) causes marked enlargement and ulceration of Peyer patches and lymphoid tissue in the appendix and ascending colon. Splenomegaly and lymphadenopathy are associated. The formation of typhoid nodules (aggregates of histiocytes and some neutrophils phagocytizing bacteria) is microscopically characteristic.

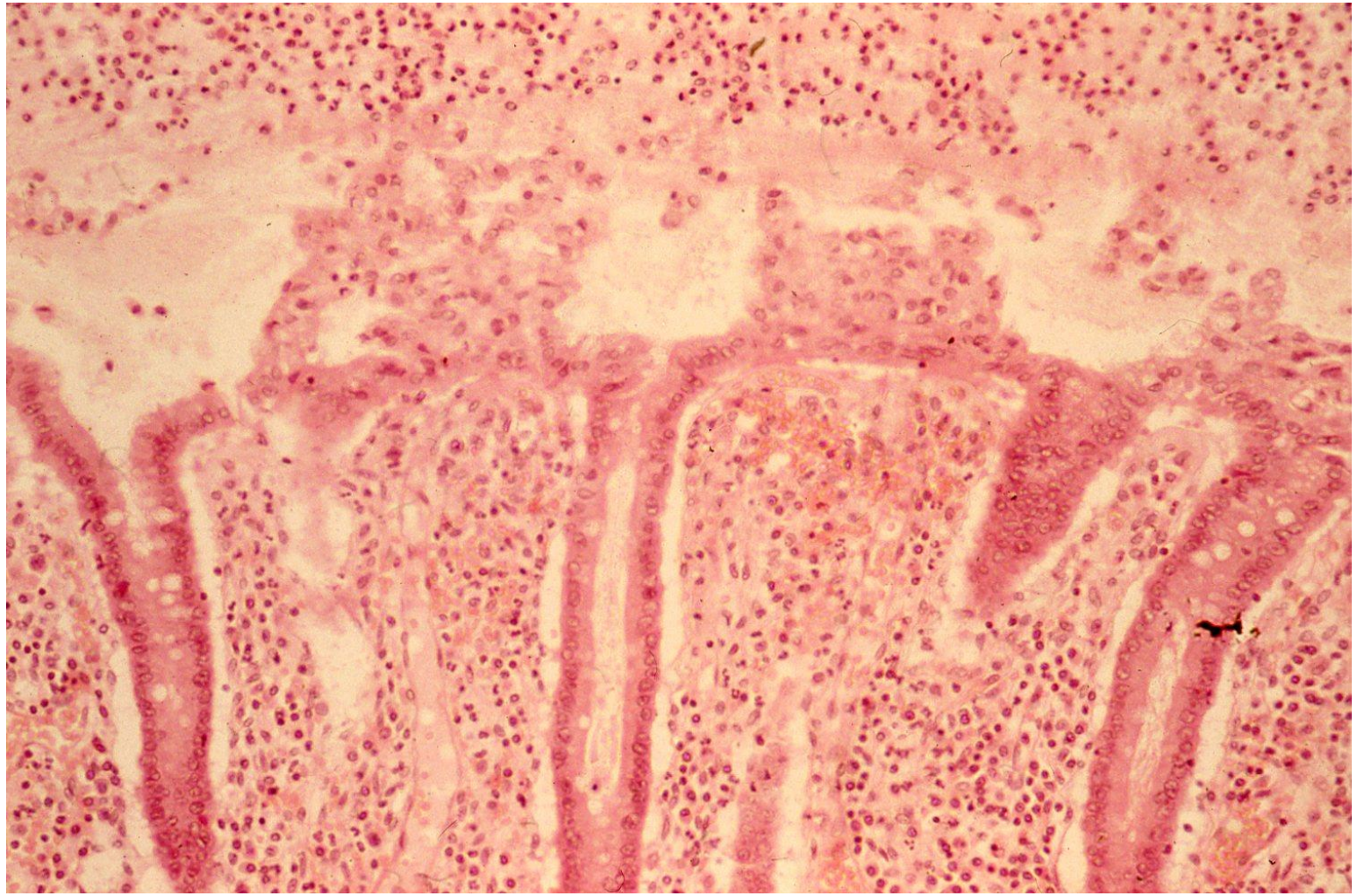
Ref.-1: Kotloff KL, et al. Shigellosis. Lancet 2018; 391(10122): 801-812. doi: 10.1016/S0140-6736(17)33296-8

Ref.-2: Kanungo S, et al. Cholera. Lancet 2022; 399(10333): 1429-1440. doi: 10.1016/S0140-6736(22)00330-0

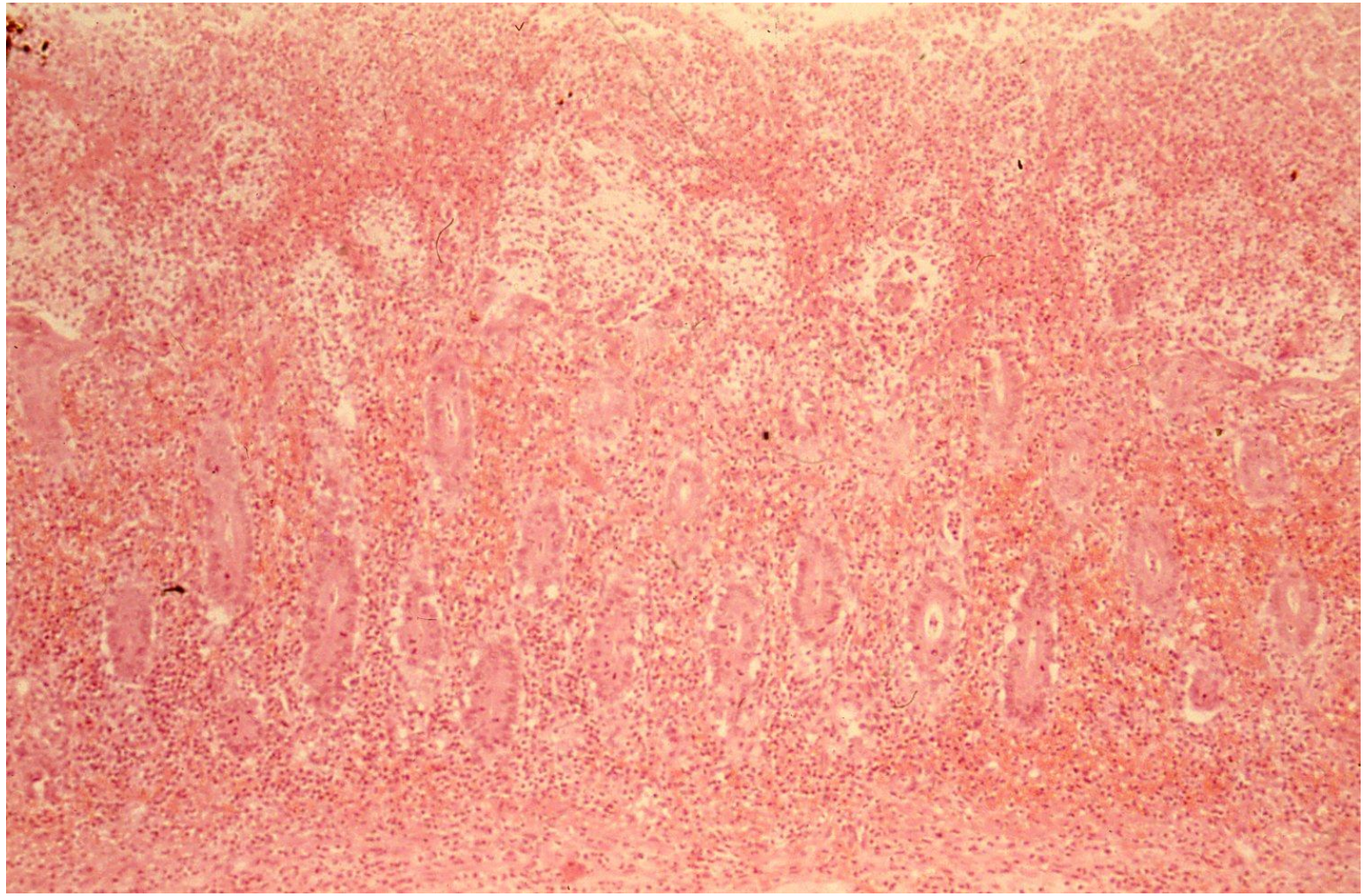
Ref.-3: House D, et al. Typhoid fever: pathogenesis and disease. Curr Opin Infect Dis 2001; 14(5): 573-578. doi: 10.1097/00001432-200110000-00011



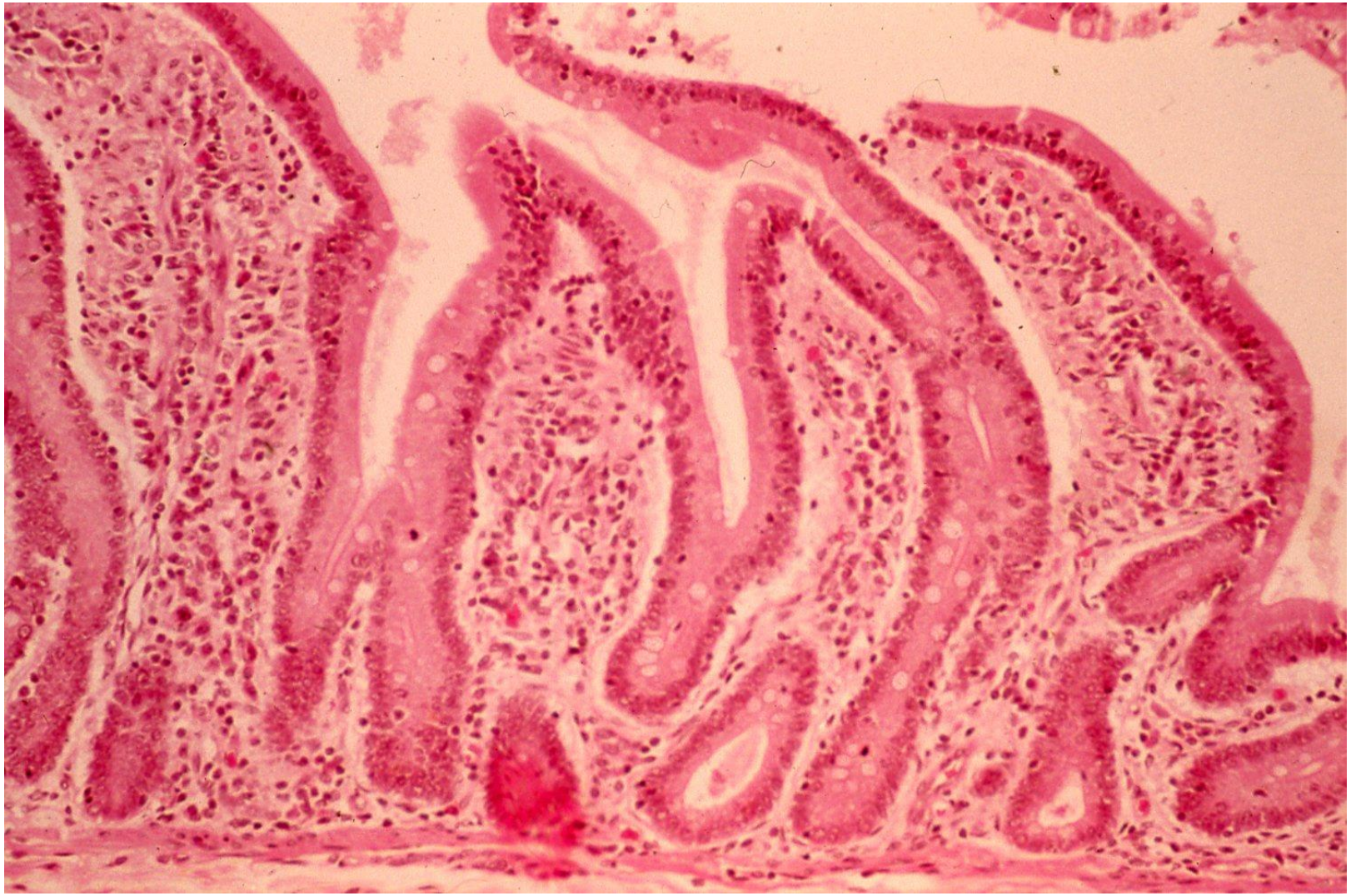
Early phase of bacillary dysentery (shigellosis). Increase of acute inflammatory infiltrate is seen in the lamina propria mucosae. Crypt destruction is minimal. H&E-1



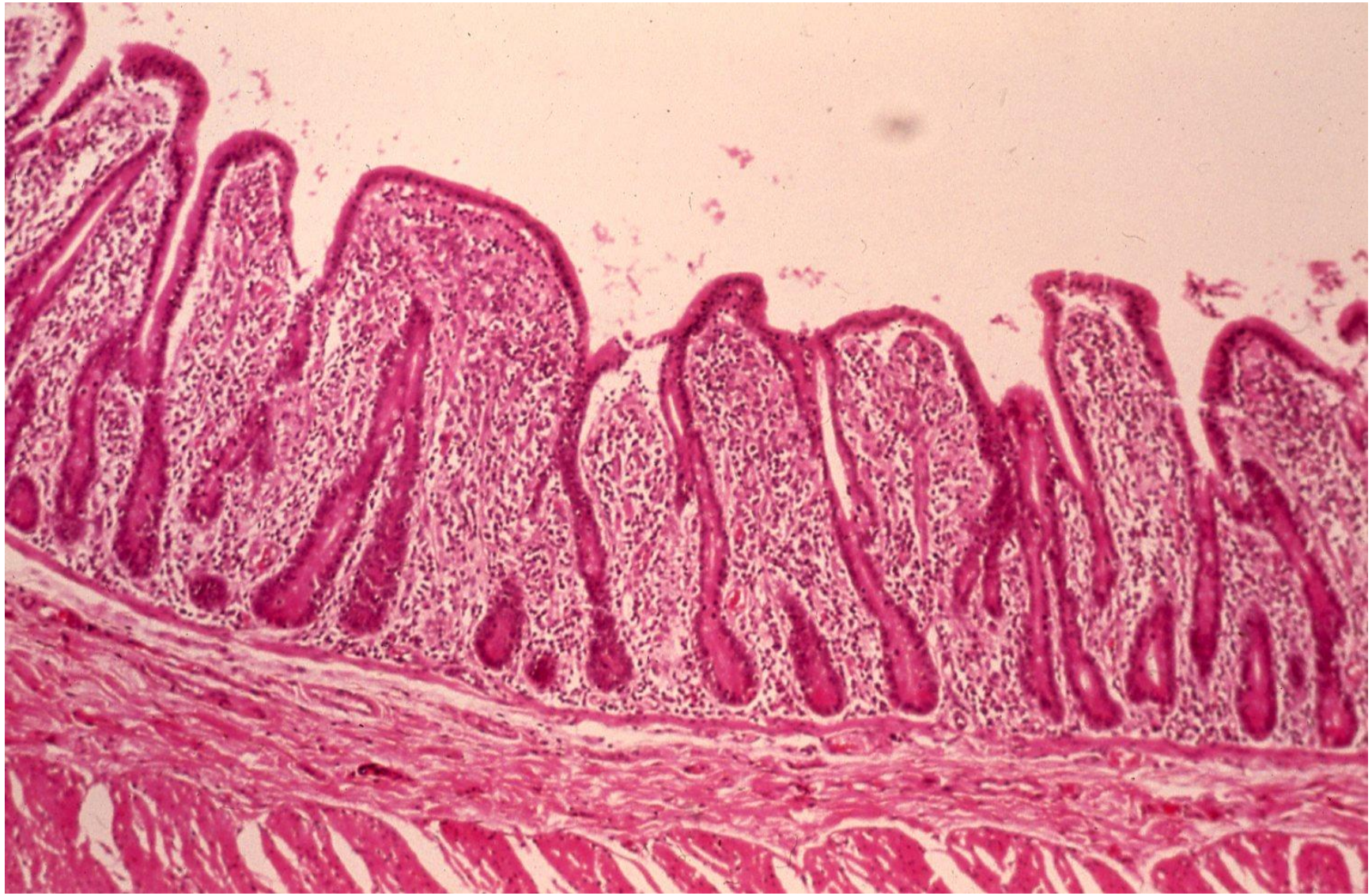
Early phase of bacillary dysentery (shigellosis). Increase of acute inflammatory infiltrate is seen in the lamina propria mucosae. Crypt destruction is minimal. Inflammatory exudation is evident on the eroded mucosal surface. H&E-2



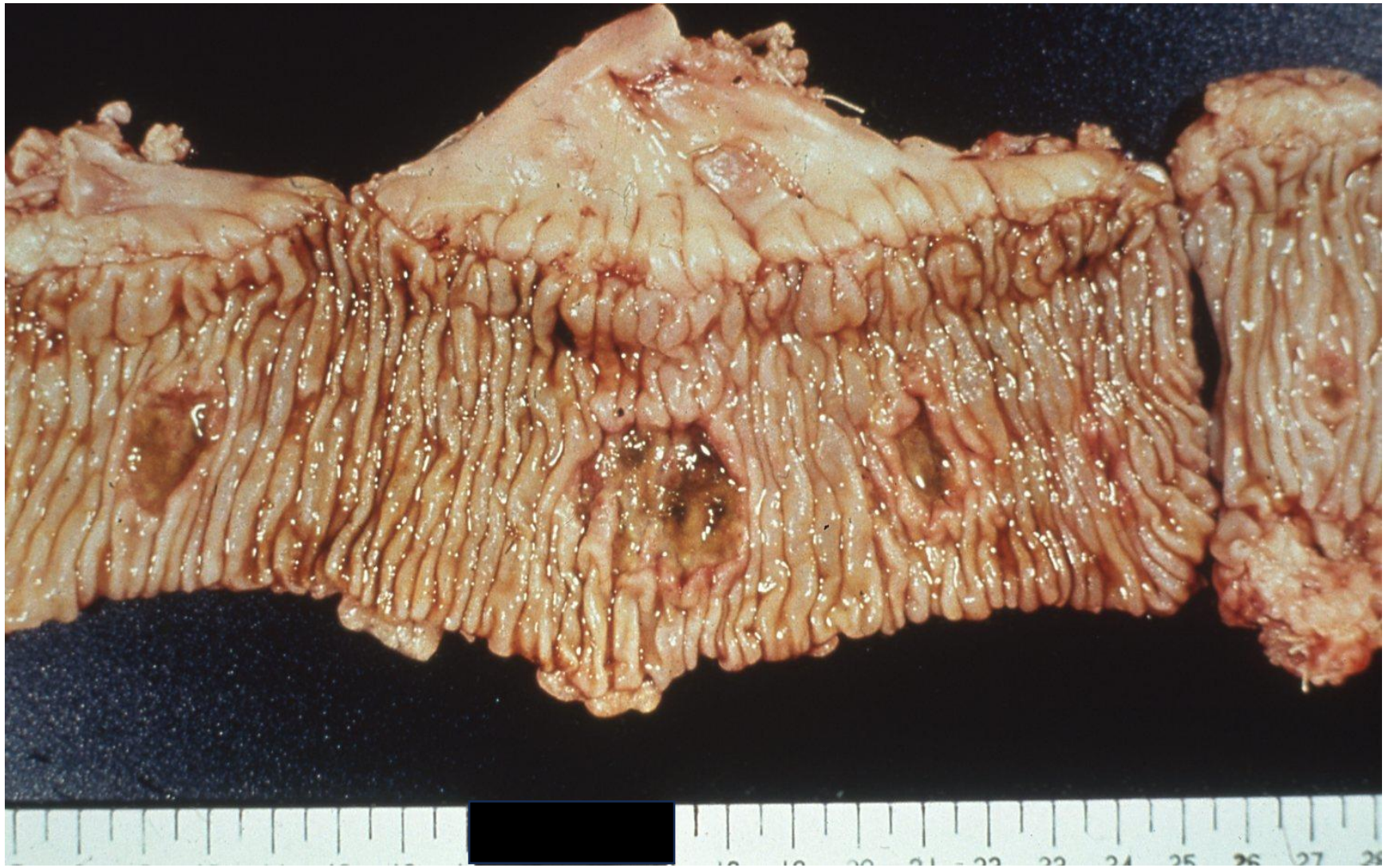
Advanced phase of bacillary dysentery (shigellosis). Marked hemorrhage is seen in the mucosa and submucosa. Crypt destruction is associated. Inflammatory and hemorrhagic exudation is evident on the eroded mucosal surface. H&E-3



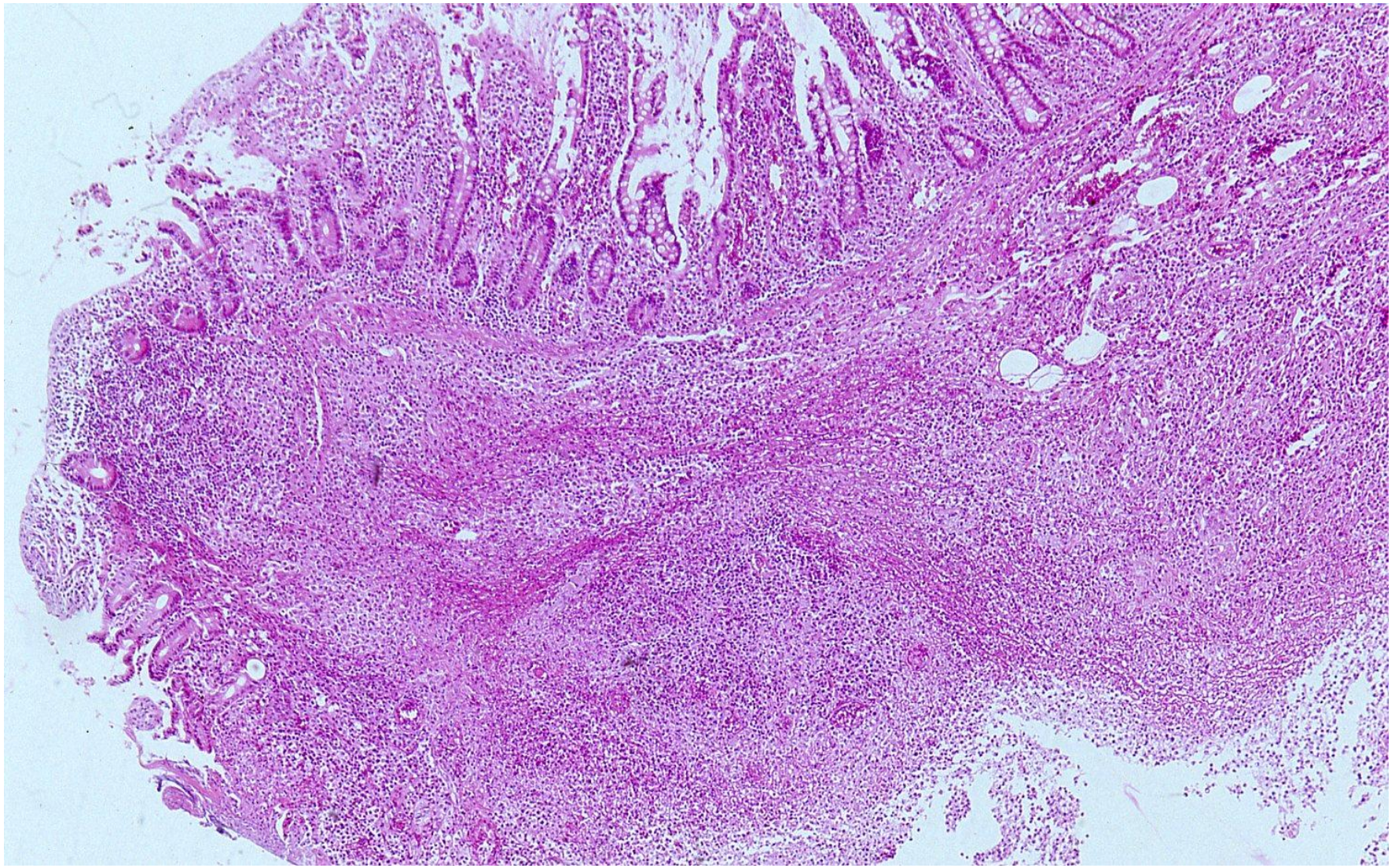
Cholera (*Vibrio cholerae* infection) 7 hours after onset. The jejunal mucosa of a 3-year-old boy at autopsy. Inflammatory infiltrate is increased in the lamina propria mucosae. Mucosal destruction is not associated in cholera. The villous structure is retained at this stage. H&E-4



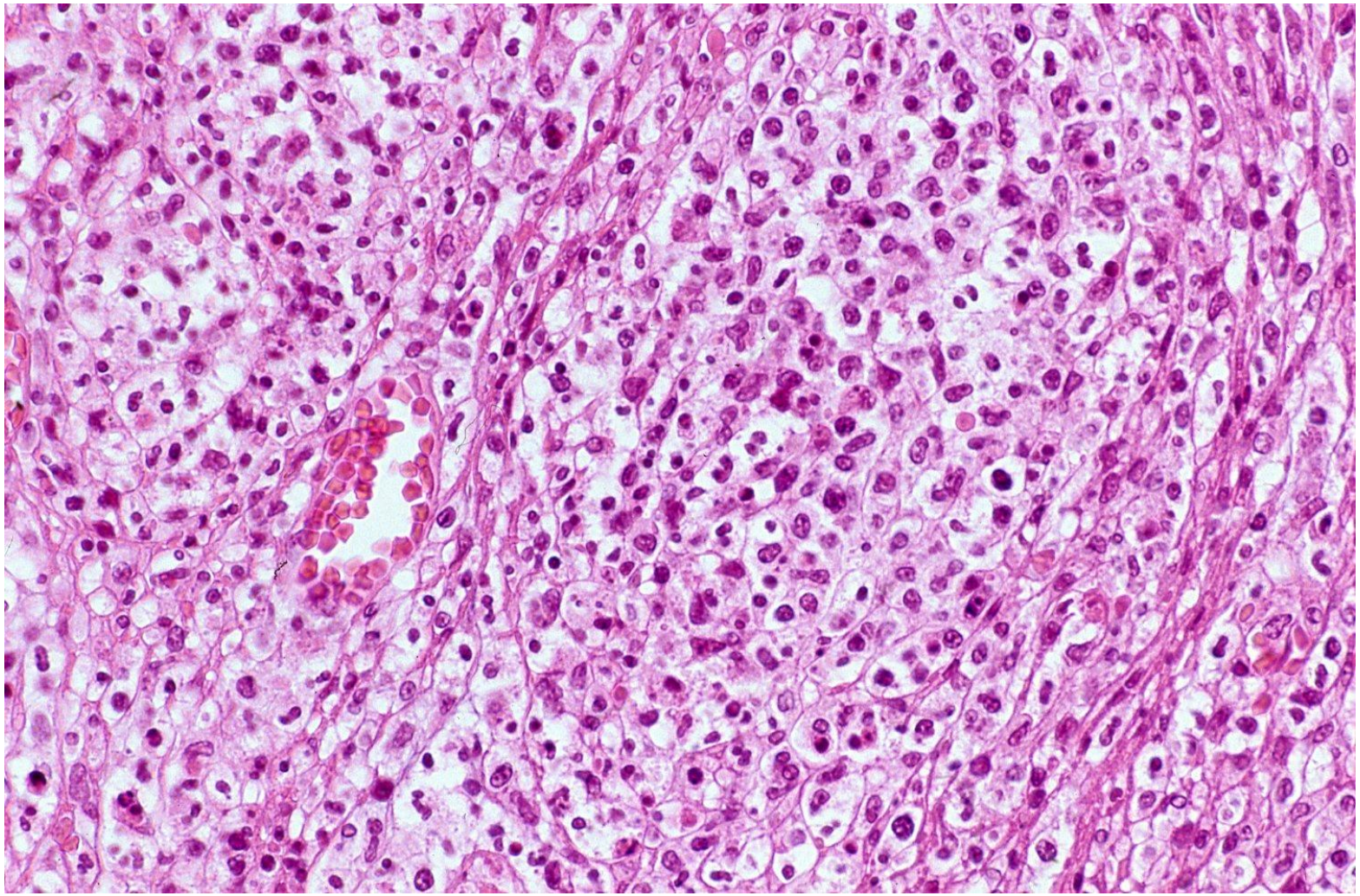
Cholera (*Vibrio cholerae* infection) 24 hours after onset. The jejunal mucosa of a 2-year-old boy at autopsy. Inflammatory infiltrate is increased in the lamina propria mucosae. Mucosal destruction is not associated in cholera. The villous structure is shortened. H&E-5



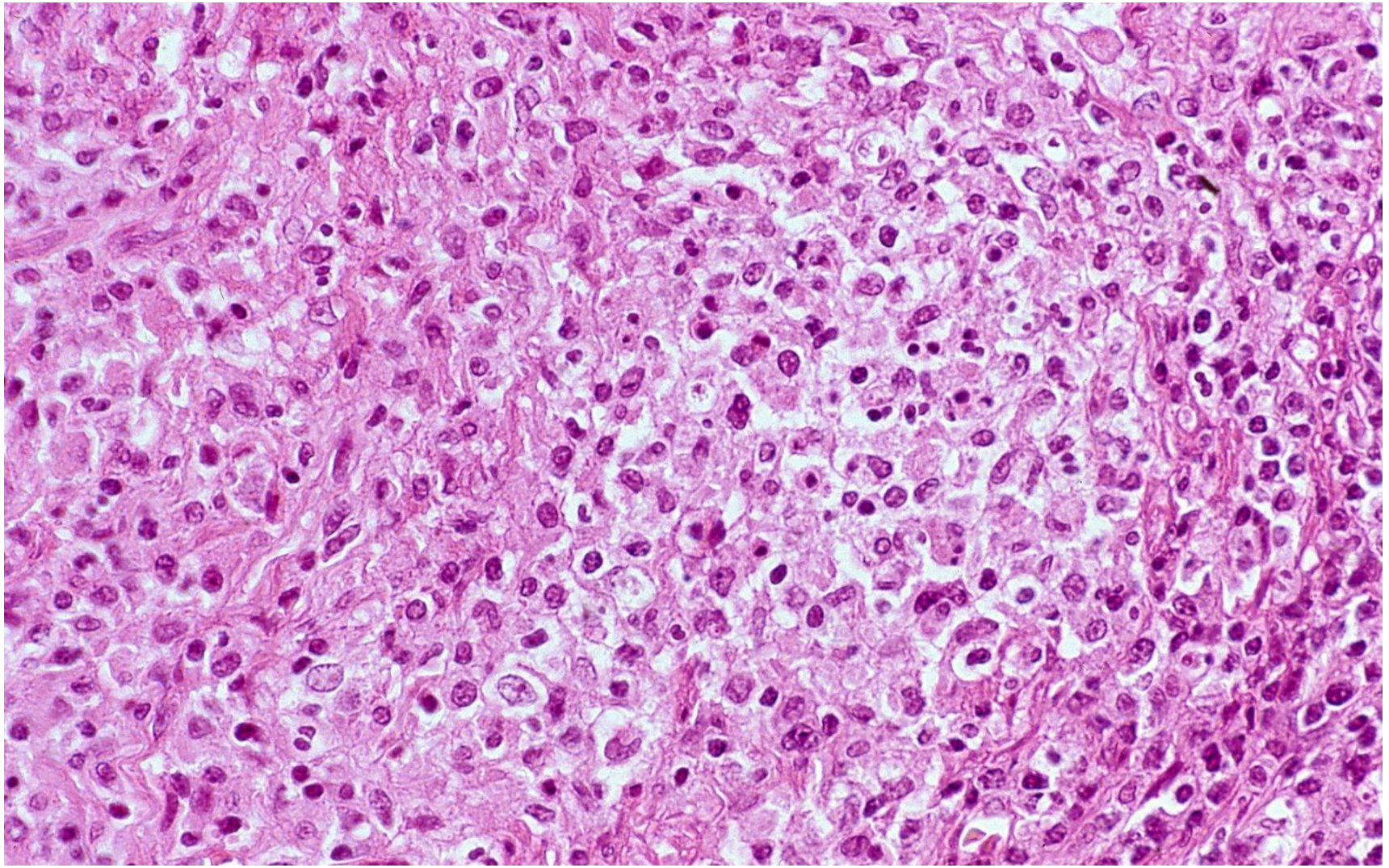
Typhoid fever (*Salmonella typhi* infection). Intestinal perforation happened 3 weeks after onset in a male patient aged 30's. The surgically resected ileum shows ulcerations at Peyer's patches.



Typhoid fever (*Salmonella typhi* infection). Intestinal perforation happened 3 weeks after onset in a male patient aged 30's. At the site of ulcerated Peyer's patch, accumulation of macrophages is pronounced. H&E-a



Typhoid fever (*Salmonella typhi* infection). Intestinal perforation happened 3 weeks after onset in a male patient aged 30's. At the site of ulcerated Peyer's patch, macrophages and some neutrophils are accumulated to form typhoid nodules. H&E-b



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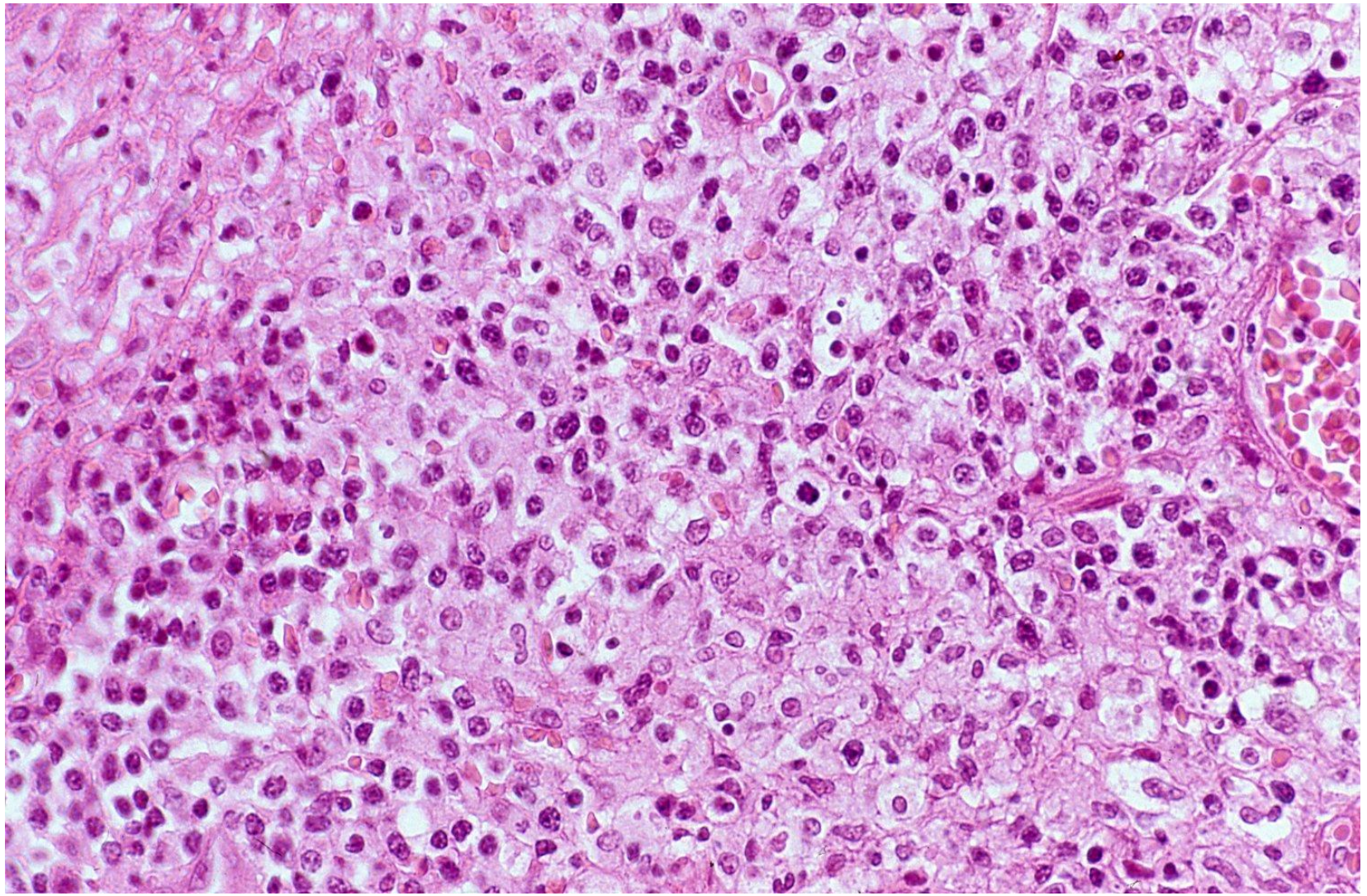

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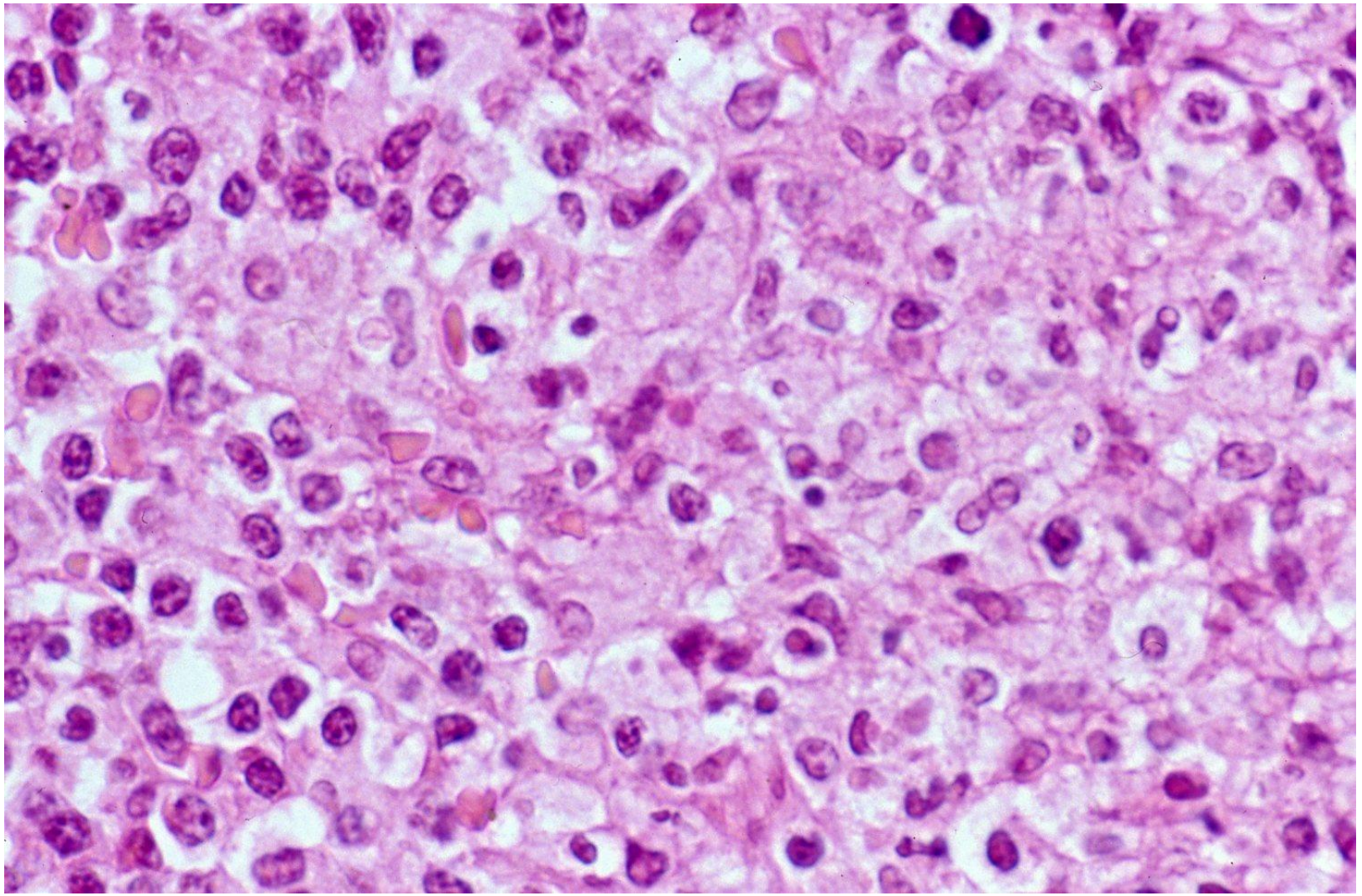
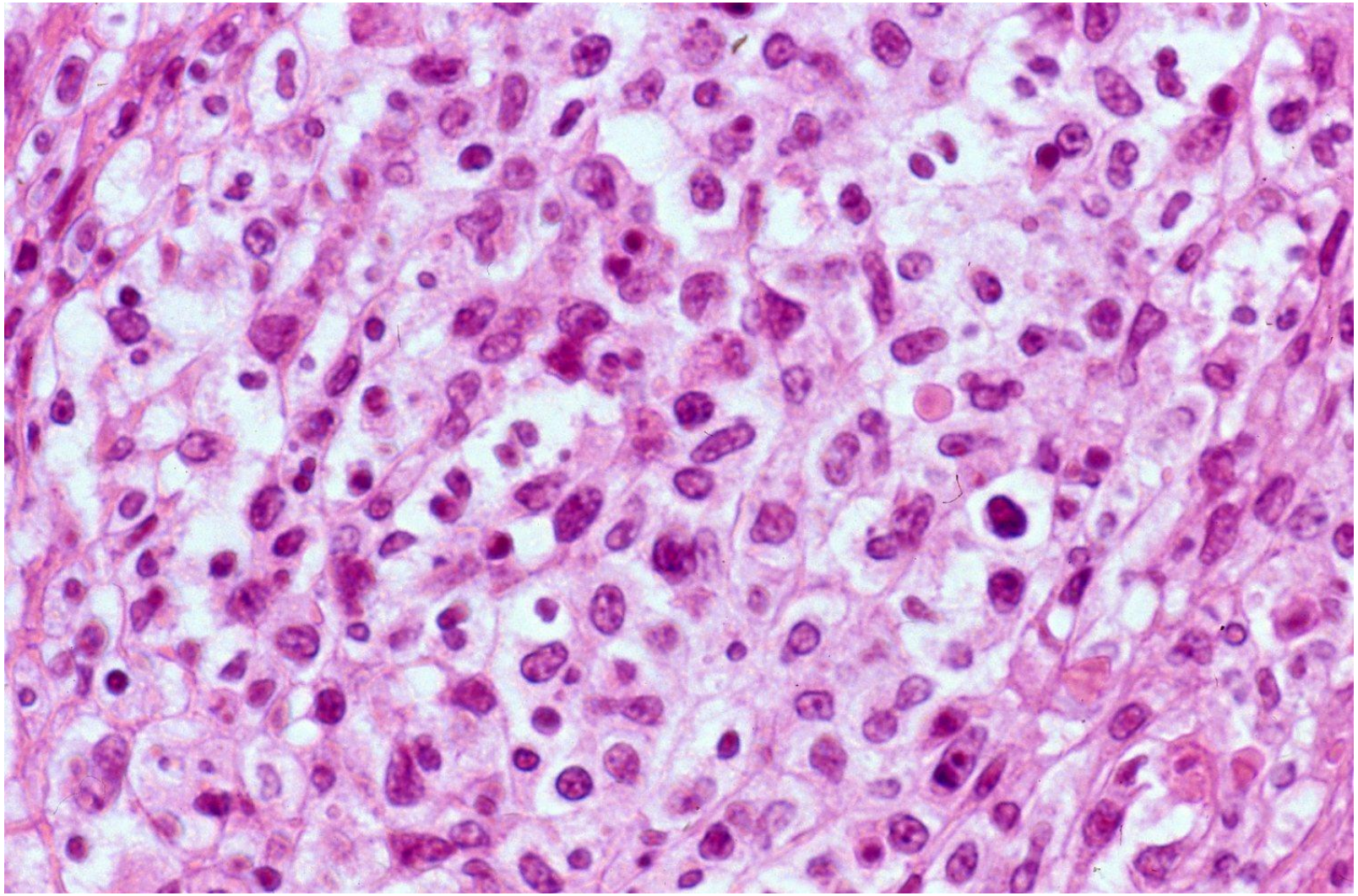


Typhoid fever (*Salmonella typhi* infection). Intestinal perforation happened 3 weeks after onset in a male patient aged 30's. At the site of ulcerated Peyer's patch, macrophages and some neutrophils are accumulated to form typhoid nodules. H&E-e



Typhoid fever (*Salmonella typhi* infection). Intestinal perforation happened 3 weeks after onset in a male patient aged 30's. At the site of ulcerated Peyer's patch, macrophages and some neutrophils are accumulated to form typhoid nodules. H&E-f