Infectious Diseases of the Small Intestine

28

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Most infections that involve the small bowel are self-limiting, and endoscopic examination is rarely indicated in these cases. Suspicion of microbial enteritis can be confirmed by stool cultures and, if necessary, by detection of toxins or antigens. Persistent diarrhea and associated symptoms such as weight loss, fever, arthralgia, and neurologic deficits or findings such as anemia, inflammatory signs, eosinophilia, or signs of malabsorption warrant further investigation. Besides microscopic stool examination, the

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main tool for this purpose is flexible endoscopy with tissue sampling from the duodenum, terminal ileum, and colon. Video capsule endoscopy (VCE) can sometimes reveal changes in the jejunum or ileum, and it has a high negative predictive value to exclude such lesions. Its usefulness is limited by its inability to obtain biopsy specimens, but it can direct the type and approach of subsequent enteroscopy by providing information on localization and extent of the detected lesions.

28.1 Whipple's Disease

28.1.1 Clinical Features

Whipple's disease is very rare. It is characterized by diarrhea, malassimilation, arthritis, neurologic deficits, and psychiatric changes.

28.1.2 Etiology

The causative organism of Whipple's disease is *Tropheryma whipplei* [1]. Affected individuals may suffer from a cellular immune defect [2].

28.1.3 Diagnosis

The diagnosis is based on a small bowel biopsy with the detection of periodic acid–Schiff (PAS)-positive macrophages. It can be confirmed by a polymerase chain reaction (PCR) assay in an intestinal biopsy.

28.1.4 Endoscopy

Endoscopy may reveal a glassy, gelatinous edema; lymphangiectasia (Fig. 28.1), [3]; erosions; ulcers; and diffuse hemorrhage [4–7]. Similar findings may be noted in intestinal histoplasmosis, atypical mycobacteriosis, or multiple myeloma [8, 9].

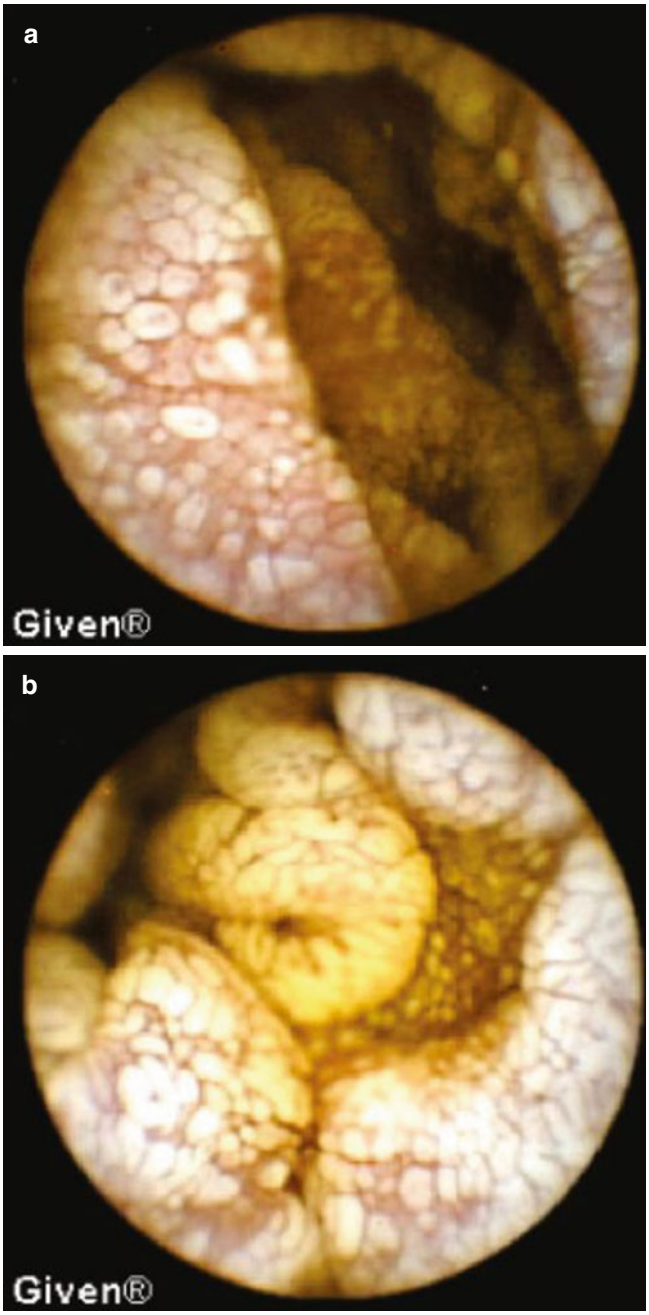

Fig. 28.1 Whipple's disease. Diffuse lymphangiectasia with swollen, whitish, club-shaped villi (**a**) and diffuse edema (**b**). (Courtesy of Ingo Franke, MD)

28.1.5 Treatment

Long-term antibiotic therapy is necessary, considering the involvement of the central nervous system. The therapeutic regimen consists of initial therapy with cephalosporin followed by a 12-month course of trimethoprim and sulfamethoxazole. Mucosal healing under this therapy has been reported, but failure of this regimen has been reported as well [10, 11].

28.2 Infection with Atypical Mycobacteria

28.2.1 Clinical Features

Intestinal involvement by atypical mycobacteria is known to occur alone or in addition to pulmonary and cutaneous involvement [12]. The infection may take an asymptomatic course or may become disseminated, especially in immunocompromised patients [13].

28.2.2 Etiology

The infection is caused by atypical mycobacteria. Over 125 species have been described, including *Mycobacterium avium-intracellulare* (MAI) and *Mycobacterium genavense*.

28.2.3 Diagnosis

Diagnosis is based on the histologic detection of acid-fast rods in biopsy material, on PCR assay, or by culturing the organism from blood, bone marrow, biopsy specimens, or sputum. Due to the ubiquitous occurrence of MAI, detection of the organism in normally sterile materials like blood is the most accurate test. Detection of environmental mycobacteria in sputum often represents colonization.

28.2.4 Endoscopy

Infection with MAI often bears an endoscopic resemblance to Whipple's disease, giving rise to the term "pseudo-Whipple's disease" (Fig. 28.2). Infections with *M. genavense* may show edema, increased friability, or lymphangiectatic areas [14].

28.2.5 Treatment

If treatment is necessary, it consists of an antimycobacterial combination regimen based on sensitivity testing and often including classic antituberculosis substances. The course of treatment may be prolonged, depending on the patient's immune status.

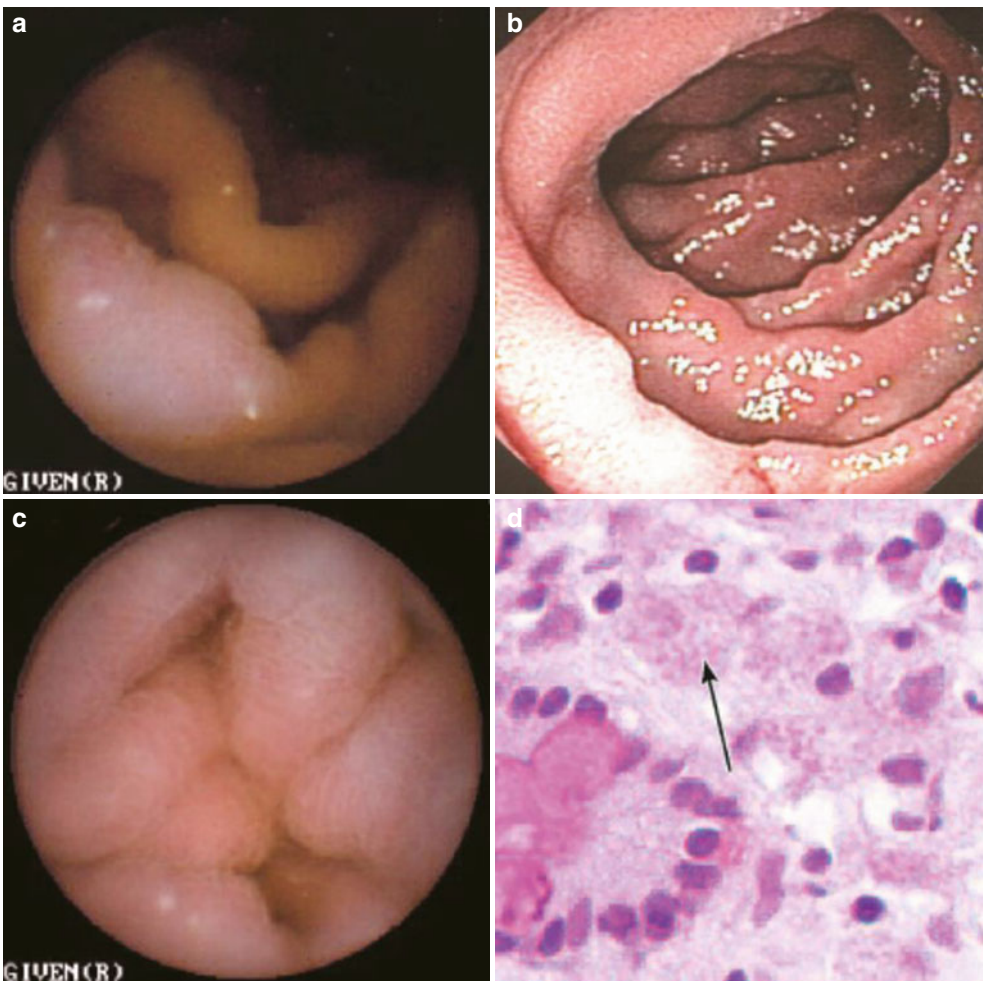

Fig. 28.2 "Pseudo-Whipple's disease" in a young man with chronic diarrhea, malassimilation, and lymphadenopathy. *Mycobacterium avium-intracellulare* was isolated from lymph nodes. The patient was HIV negative. (**a**, **c**) Examination of the jejunum with video capsule endoscopy (VCE) shows diffuse, glassy edema with indistinct villi and luminal narrowing. The capsule passed through the lumen without impediment. (**b**) Corresponding image from push enteroscopy (Courtesy of Wolfgang Cordruwisch, MD). (**d**) Histology revealed weakly PAS-positive macrophages (Courtesy of Axel von Herbay, MD)

28.3 Tuberculosis

28.3.1 Etiology

Gastrointestinal tuberculosis most frequently involves the small intestine and ileocecum [15, 16]. Gastrointestinal tuberculosis may occur as a primary infection with *Mycobacterium bovis* or may be secondary to pulmonary infection with *Mycobacterium tuberculosis*.

28.3.2 Diagnosis

The diagnosis can be established by the biopsy detection of caseating granulomas and acid-fast rods and also by genetic testing (PCR) and culture studies. Stool PCR has proven to be as sensitive as sputum tests in patients with pulmonary tuberculosis [17], so the detection of *M. tuberculosis* in stool samples does not prove involvement of the gastrointestinal tract. On the other hand, gastrointestinal lesions can be found in asymptomatic patients with pulmonary tuberculosis [18].

28.3.3 Endoscopy

Edematous swelling and patchy redness are typical but unspecific findings in affected mucosa (Figs. 28.3 and 28.4). Ulcers are most commonly found in the ileocecal region (Fig. 28.5); strictures and obstructions also have been described [19, 20]. Differentiating intestinal tuberculosis from other ulcerative diseases of the small intestine can be challenging, especially in countries with a high prevalence of tuberculosis and Crohn's disease. In Crohn's disease, ulcers may appear longitudinal or aphthous, whereas tuberculosis may present with oblique or transverse ulcers with

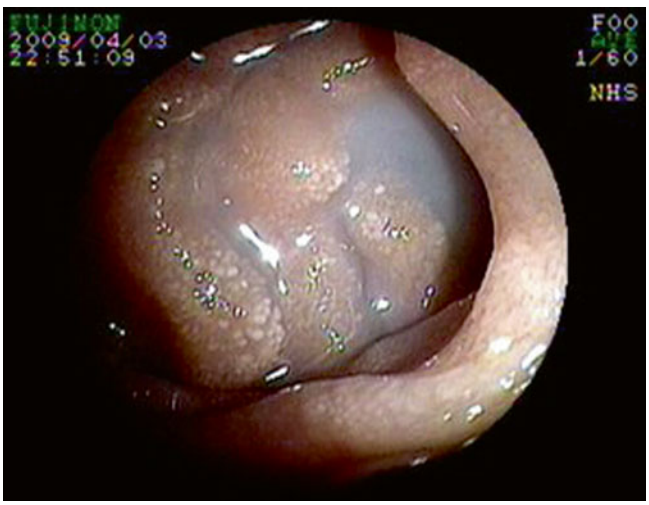

Fig. 28.3 Diffuse swelling, lymphangiectasia, and exudation in intestinal tuberculosis (double-balloon enteroscopy)

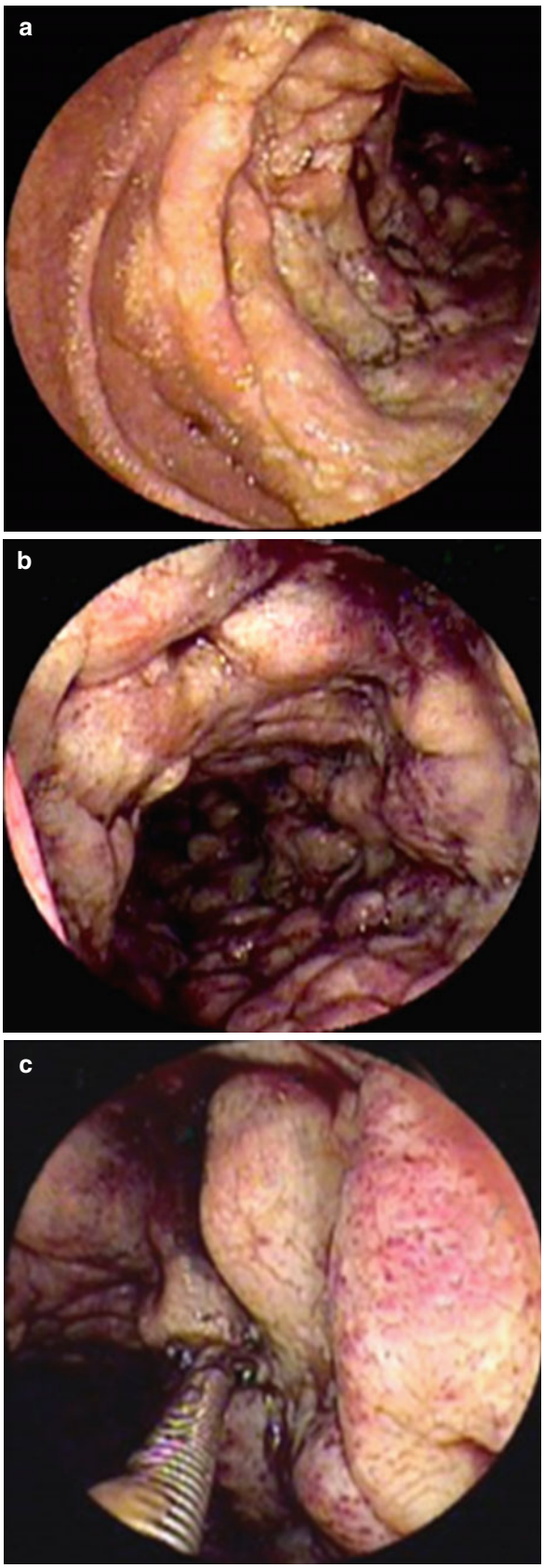

Fig. 28.4 (**a**–**c**) Diffuse infiltration of small intestinal mucosa in a patient with tuberculosis. Double-balloon enteroscopy shows diffuse swelling, erythema, and white and livid discoloration

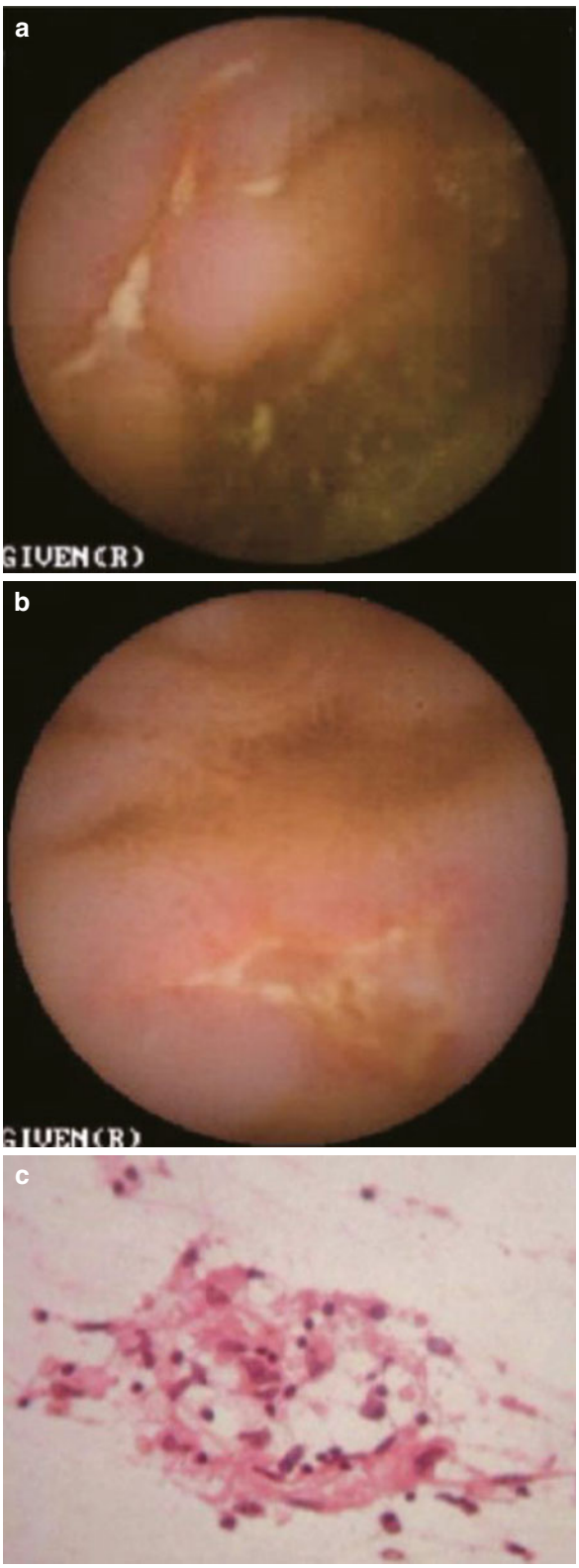

Fig. 28.5 Intestinal tuberculosis. VCE. (**a**, **b**) Edema and ulcers in the ileum (Reprinted from Reddy et al. [23], with permission from Georg Thieme Verlag). (**c**) Biopsy from the terminal ileum shows portions of a granuloma with epithelioid and giant cells, consistent with tuberculosis

a necrotic base, as well as hypertrophic or nodular lesions [21–23]. Oblique ulcers also can be found in chronic, nonspecific multiple ulcers of the small intestine (CNSU) (Chap. 32), however, and lesions related to the use of nonsteroidal anti-inflammatory drugs (NSAIDs) may present with discrete ulcerations and concentric stenosis (Chap. 30) [24, 25]. Because of the morphologic resemblance, differentiation of these lesions through biopsy is warranted.

28.3.4 Treatment

Treatment consists of a combination regimen like that used for pulmonary tuberculosis. Recent studies showed similar effectiveness for 6-month and 9-month regimens [26].

28.4 Intestinal Spirochetosis

28.4.1 Etiology

The organisms causing intestinal spirochetosis are spirochetes such as *Borrelia eurygyrata*, *Brachyspira aalborgi*, *Brachyspira hyodysenteriae*, and *Serpulina pilosicoli*. There is no clinical relation to borreliosis or syphilis. The detection of intestinal spirochetosis in asymptomatic individuals may represent colonization, but it should be considered in the differential diagnosis of symptomatic patients, especially those suffering from immunodeficiency [27].

28.4.2 Clinical Features

Patients may present with abdominal pain, which sometimes resembles appendicitis. Chronic diarrhea, constipation, and weight loss also have been reported.

28.4.3 Diagnosis

Diagnosis can be established by biopsies of the colorectal or small bowel mucosa. Spirochetes can be detected in the specimens by microscopy and can be differentiated by PCR [28].

28.4.4 Endoscopy

Colonoscopic examination often shows normal mucosa. In some patients, lymphoid follicular hyperplasia or hemorrhagic lesions can be found [29]. Possible findings at small bowel capsule endoscopy are lymphoid follicular hyperplasia and aphthous ulcers (Fig. 28.6).

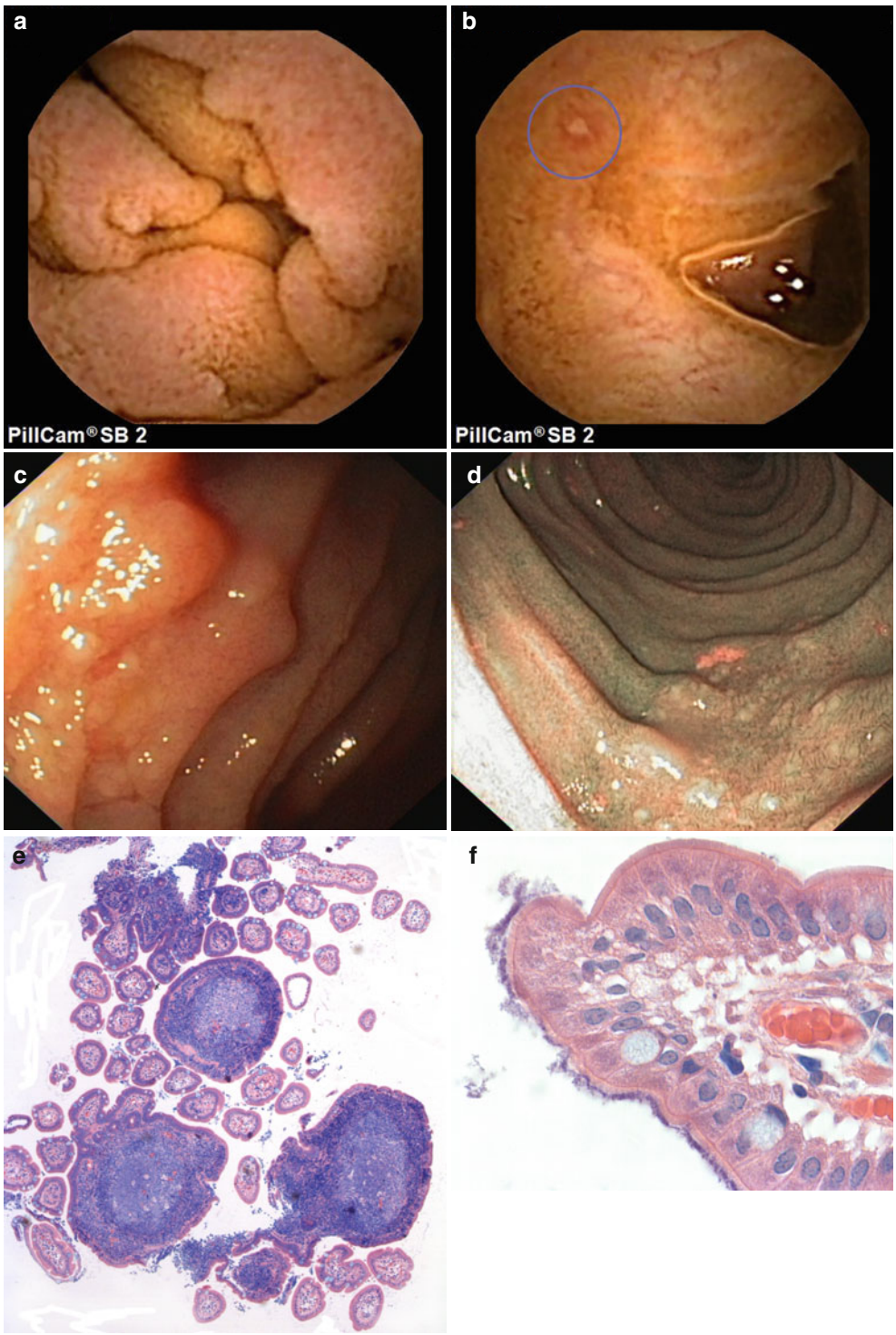

Fig. 28.6 Intestinal spirochetosis. Lymphoid hyperplasia in the proximal duodenum (**a**) and aphthae (*circle*) in the proximal and mid-small bowel (**b**) (VCE, courtesy of Brigitta Reinke, MD.) Single-balloon enteroscopy shows nodules (**c**) and an irregular mucosal defect without ulceration (**d**). Biopsy specimen (Giemsa stain) showed lymphoid hyperplasia (**e**) and a pathognomonic bacterial layer on the mid-small bowel mucosa (**f**), confirming intestinal spirochetosis. (Courtesy of Ilske Oschlies, MD)

28.4.5 Treatment

Symptomatic intestinal spirochetosis can be treated with nitroimidazoles; metronidazole is the first-line drug of this type.

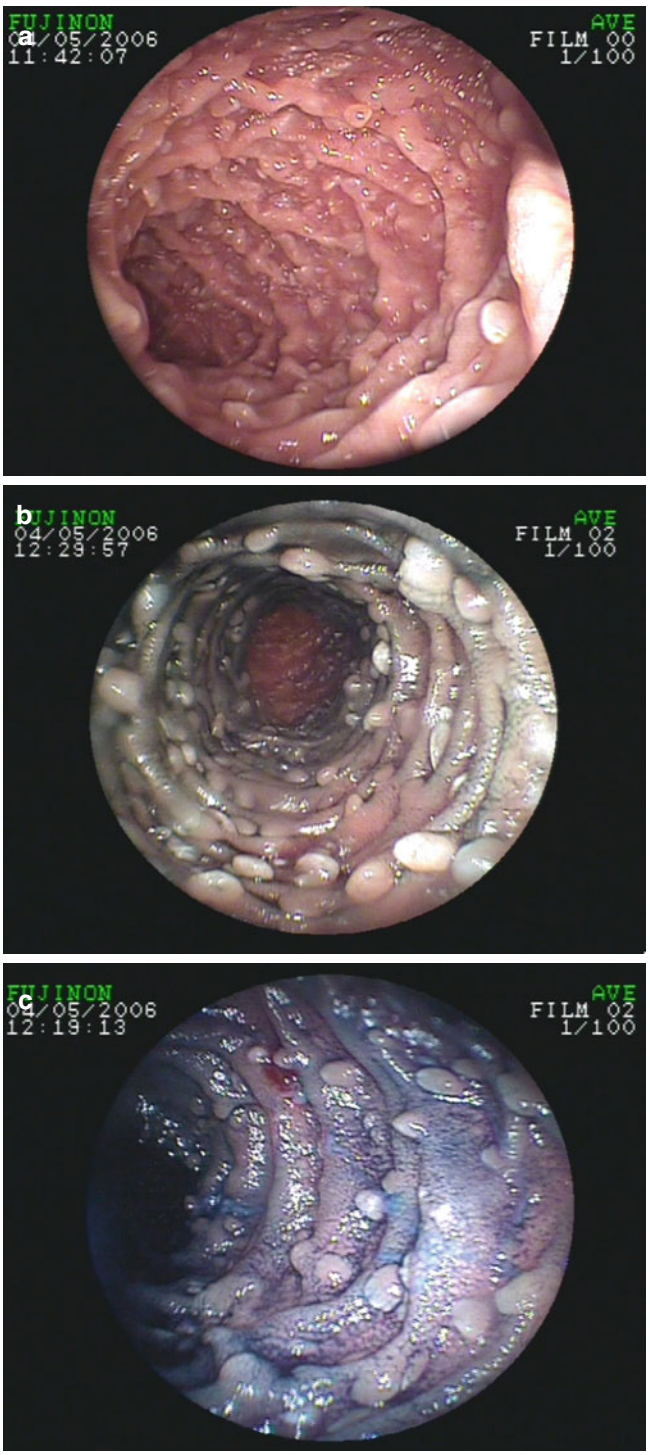

Fig. 28.7 (**a**–**c**) Immunoproliferative small intestinal disease (IPSID) with multiple, diffusely distributed nodules over a long segment of the small intestine. Double-balloon enteroscopy (DBE). A native image (**b**), DBE with Fujinon intelligent color enhancement (FICE), DBE chromoendoscopy with indigo carmine

28.5 Immunoproliferative Small Intestinal Disease

28.5.1 Etiology

Immunoproliferative small intestinal disease (IPSID) is a form of extranodal marginal zone lymphoma (mucosa-associated lymphoid tissue (MALT lymphoma)) involving the small intestine. The production of truncated alpha heavy chains also led to the term *alpha heavy chain disease*, and the endemic presentation in the Middle East and Africa led to the term *Mediterranean lymphoma*. Bacterial infection of the small intestine as with *Campylobacter jejuni* has been attributed to the development of IPSID [30].

28.5.2 Clinical Features

Clinical presentation includes abdominal pain, secretory diarrhea, and malabsorption [31].

28.5.3 Endoscopy

Multiple nodules of the small intestine can be observed [32–34] (Fig. 28.7).

28.6 Cytomegalovirus Enteritis

28.6.1 Etiology

Cytomegalovirus (CMV) is a DNA virus from the group of herpesviruses. After primary infection, the virus persists and may later be activated or reactivated. Severe courses of disease can be found in patients with immunosuppression due to malignant diseases, pharmacologic treatment, or AIDS.

28.6.2 Clinical Features

The infection often remains asymptomatic, but it may affect the lungs, central nervous system, retina, liver, biliary tract, and all portions of the gastrointestinal tract. Mid-gastrointestinal bleeding, ulcers, perforation, and necrosis have all been described in the small bowel [35–38].

28.6.3 Diagnosis

The diagnosis is based on the biopsy detection of typical cytomegalic cells (“owl’s eye cells”) (Fig. 28.8), antigen detection in biopsy samples, and PCR assay in peripheral

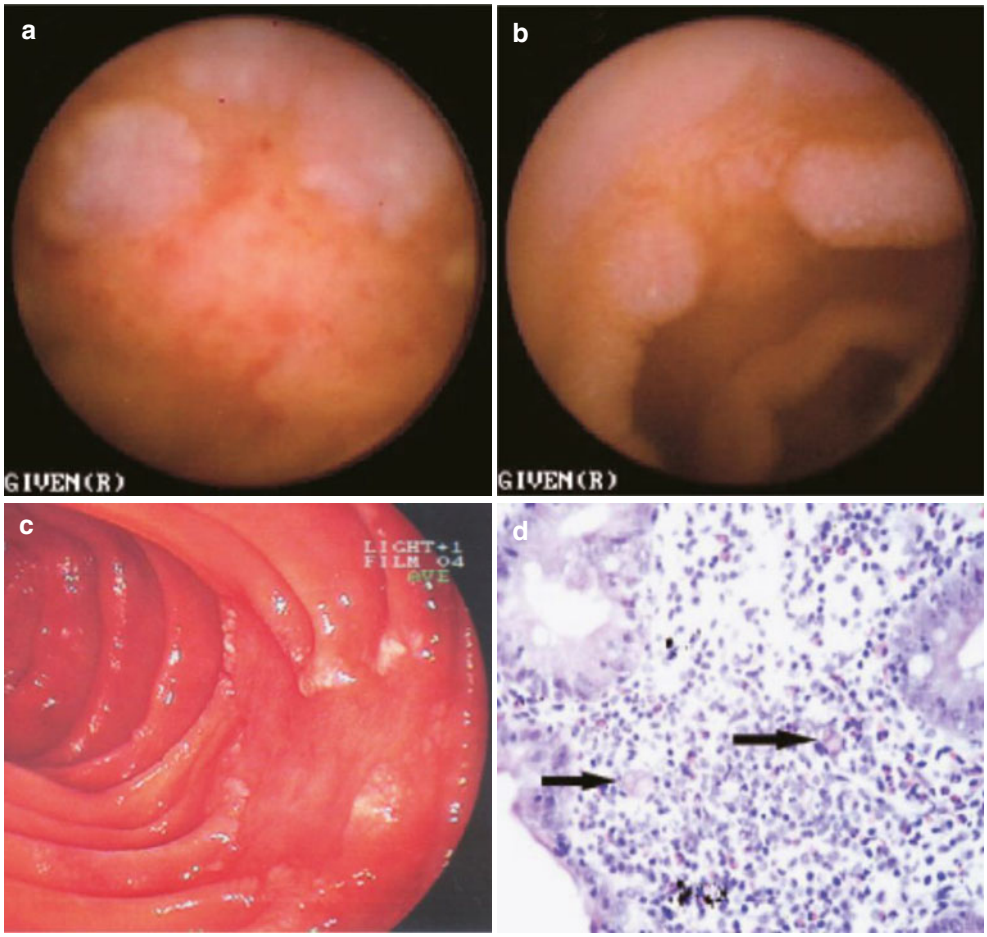

Fig. 28.8 Cytomegalovirus (CMV) enteritis in a 59-year-old man with cachexia and tetraparesis. The HIV-negative patient was taking methotrexate for rheumatoid arthritis. Capsule endoscopy was performed for chronic diarrhea and malassimilation. (**a**, **b**) VCE reveals small erosions, petechiae, and punched-out mucosal defects. (**c**) Punched-out, noninflamed ulcers on enteroscopy (Courtesy of Christoph Manegold, MD). (**d**) Histology shows typical owl's eye cells (*arrows*) (Courtesy of Andreas Gocht, MD)

blood lymphocytes. IgM antibodies may be detectable in immunosuppressed patients.

28.6.4 Endoscopy

CMV ulcers often show no inflammatory reaction and have a punched-out appearance. Typically the ulcer base is not covered by fibrinous exudate [39] (Fig. 28.8).

28.6.5 Treatment

Most immunocompetent patients do not need treatment. Immunosuppressed patients may be treated with intravenous ganciclovir or oral valganciclovir; cidofovir or foscarnet can be used as second-line drugs.

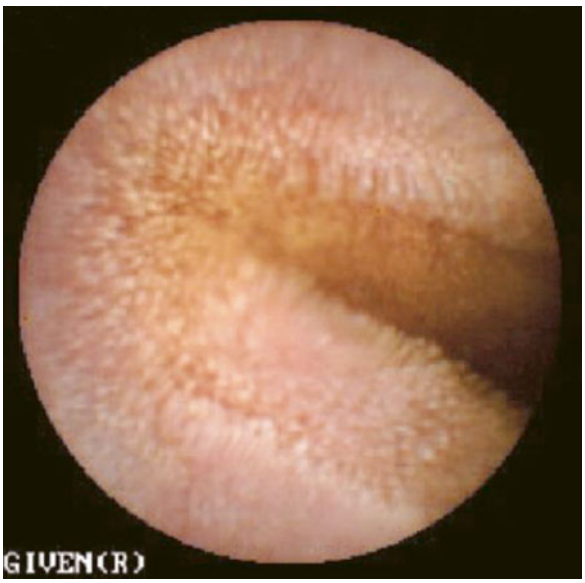

Fig. 28.9 Diffuse, nonspecific lymphangiectasia in an AIDS patient with malassimilation

28.7 AIDS

Patients with AIDS can develop enteritis due to a variety of causes [40]. These include mycobacteriosis, histoplasmosis, CMV infection, strongyloidiasis, and cryptosporidiosis [13, 41, 42]. Histologic and/or microbiologic identification of the causative organism is essential for planning a specific therapy. Malassimilation in patients suffering from AIDS may result from enteritis caused by HIV itself. HIV enteropathy is diagnosed by exclusion (Fig. 28.9). Endoscopic sign of HIV enteropathy may be diffuse, unspecific lymphangiectasia [43]. These enteropathies have become less common with the widespread use of highly active antiviral therapy, but they may be difficult to diagnose and treat [44].

Thinking the other way around, in patients with unusual findings at VCE, a detailed history is important and HIV testing might be considered.

Apart from lesions obviously of infectious origin, patients with HIV infection or AIDS may present with other rare and unusual gastrointestinal manifestations caused by inflammation, medication, or yet undiagnosed infections [45]. Examples of mesenteric panniculitis or severe gastric ulceration are shown in Figs. 28.10 and 28.11.

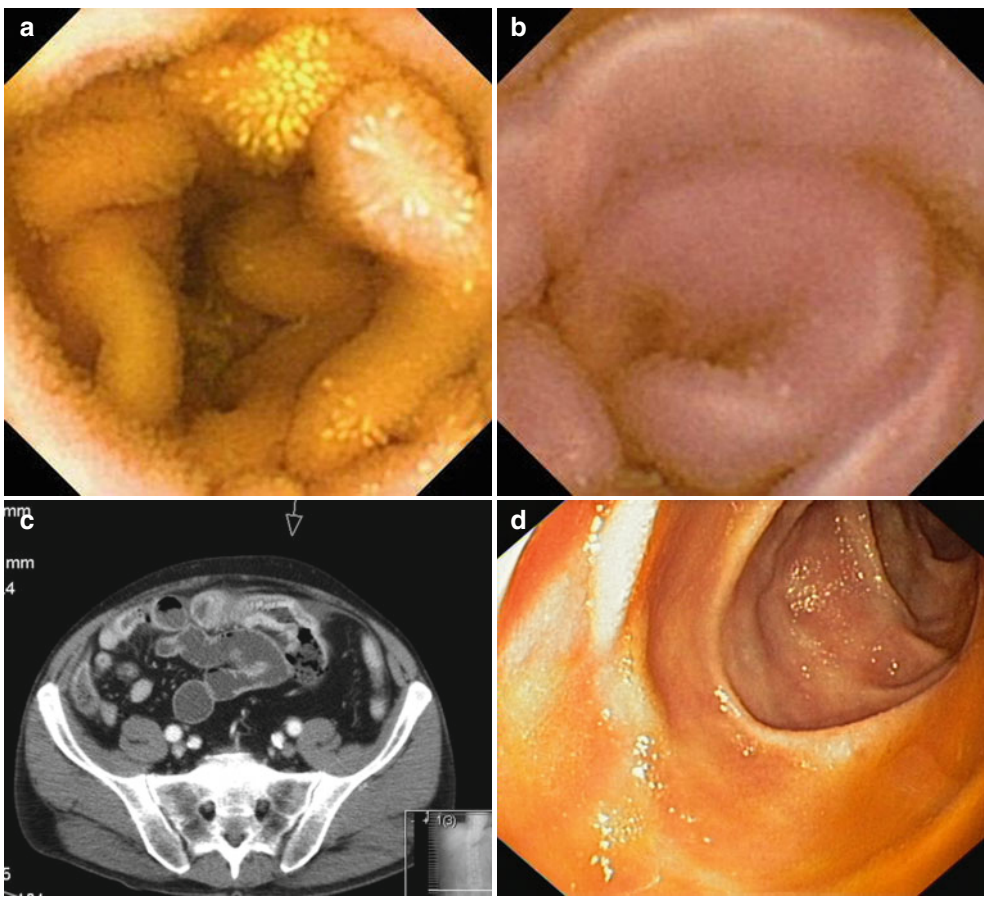

Fig. 28.10 Panniculitis in an HIV-positive patient. VCE shows lymphangiectasia (**a**) and thickened, fibrotic small bowel wall (**b**). A CT scan (**c**) shows inflammation of the visceral tissue. (Courtesy of Roman Fischbach, MD.) Single-balloon enteroscopy (**d**) demonstrates fibrosis and reduced motility of the jejunum

Panniculitis may occur as a rare complication of AIDS. This group of diseases is characterized by inflammation of adipose subcutaneous or visceral tissue. A diagnosis of panniculitis can be suggested by CT scans and verified by biopsy sampling. Further classification is made by histologic characteristics. If fat necrosis and inflammation predominate, the condition is called *mesenteric panniculitis*, whereas if fibrosis and retraction predominate, the condition is known as *retractile mesenteritis*. As the inflammatory reaction affects the mesentery and visceral fat tissue, only indirect signs, such as lymphangiectasia or mucosal fibrosis leading to hypomotility, can be observed (Fig. 28.12).

Although capsule endoscopy does not provide the possibility of biopsies, it may present an interesting possibility to investigate the entire gastrointestinal tract in HIV patients with a single-use endoscope (Fig. 28.13).

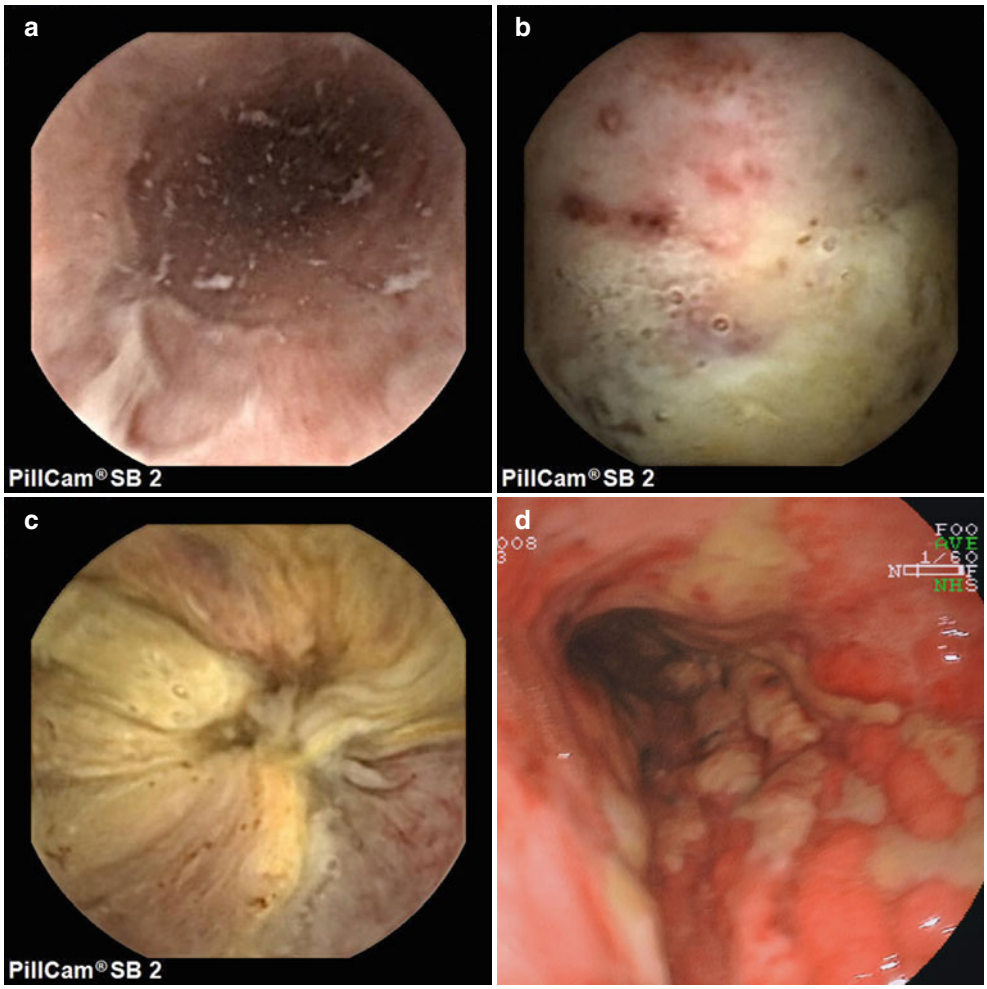

Fig. 28.11 Severe ulceration of the entire stomach in an AIDS patient with dysphagia. (**a**) Reflux esophagitis. Ulcerated stomach in VCE (**b**, **c**) and in gastroscopy (**d**). Histology was nonspecific

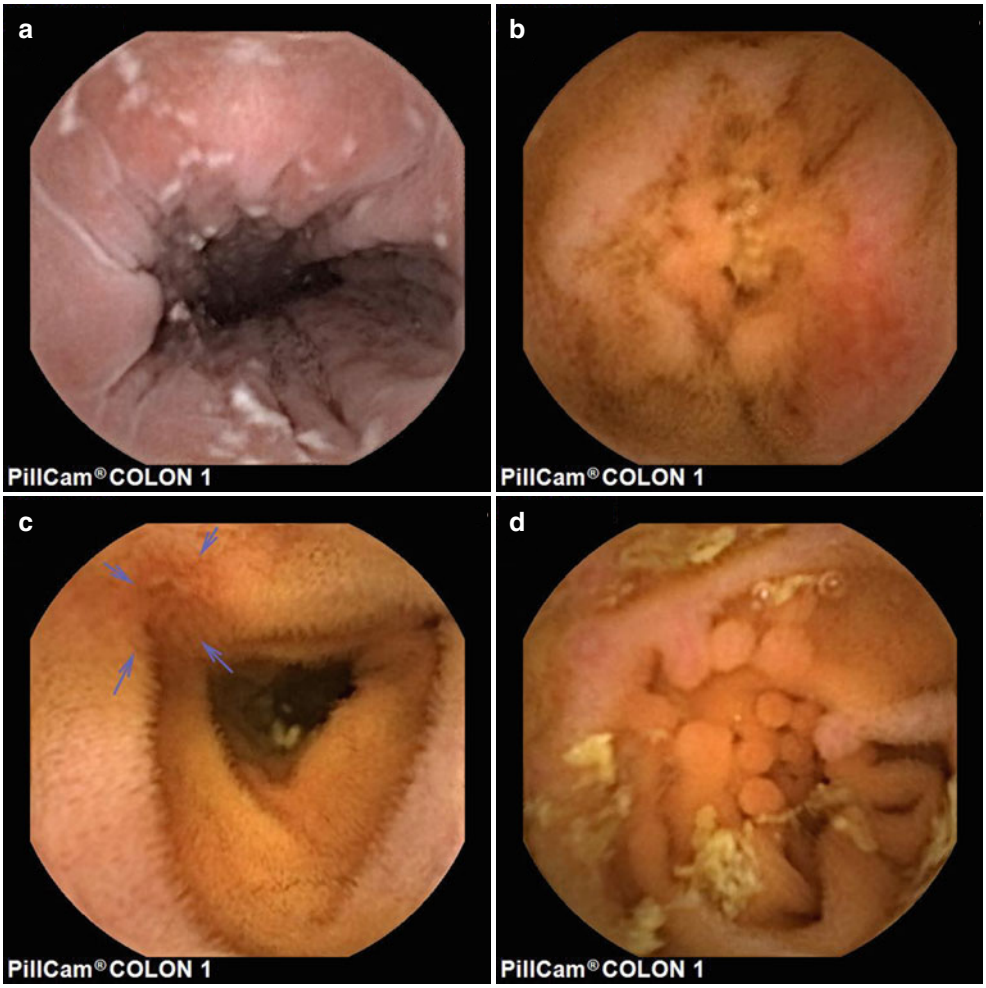

Fig. 28.12 PillCam COLON capsule for anemia in a patient positive with HIV and CMV serology shows monilial esophagitis (**a**), as well as erosions (*arrows*) (**b**, **c**), and lymphoid hyperplasia (**d**) of the small bowel

28.8 Blastomycosis

28.8.1 Etiology

South American blastomycosis is a fungal infection caused by *Paracoccidioides brasiliensis*.

28.8.2 Clinical Features

Besides sometimes mutilating cutaneous forms, visceral manifestations can affect multiple organs, including the lung, brain, and others, including the small intestine in rare cases [46]. Immunosuppression or diabetes frequently accompanies blastomycosis [47].

28.8.3 Diagnosis

Fungal cultures and antibody tests can be used for diagnosis. A characteristic feature at histology is the steering wheel-like appearance of the pathogen (Fig. 28.13).

Fig. 28.13 Histopathology of *Paracoccidioidomycosis brasiliensis*. Methenamine silver stain (Courtesy of Dr. Lucille K. Georg, Centers for Disease Control and Prevention, Atlanta, GA, USA)

28.8.4 Endoscopy

Lesions may be nodular, ulcerous, or stenotic [48]. Besides nodules and ulcers, lymphangiectasia also can be seen at endoscopy [34] (Fig. 28.14).

28.8.5 Treatment

Systemic amphotericin B has been widely used for treatment.

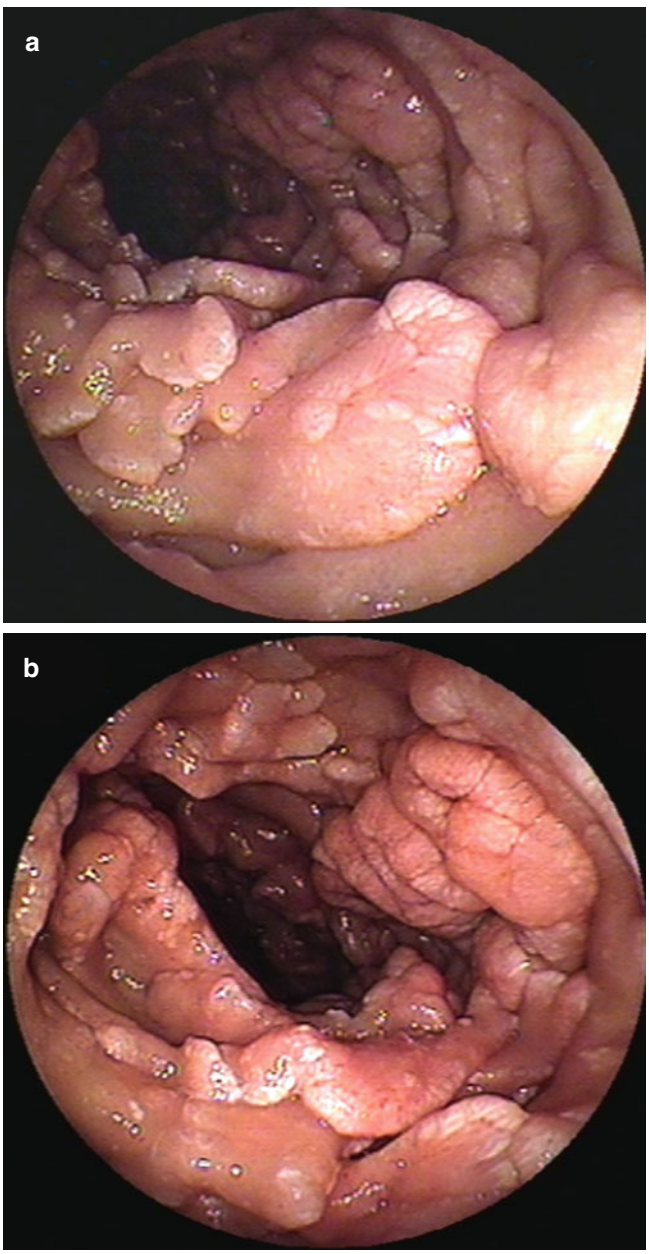

Fig. 28.14 (**a**, **b**) Small intestinal blastomycosis with diffuse, nodular, and patchy mucosal swelling and white villi seen at double-balloon enteroscopy

28.9 Giardiasis

Giardiasis is caused by the protozoon *Giardia lamblia*, which is present worldwide in varying frequency, with a higher prevalence in the tropics. *G. lamblia* preferably affects the upper gastrointestinal tract.

28.9.1 Clinical Features

The typical manifestation is diarrhea, in some cases causing severe illness. Most infected persons are asymptomatic or have only minor, nonspecific symptoms, but the course is sometimes prolonged [49]. The infection may be self-limiting. Imidazole derivates are effective in treatment [50].

28.9.2 Diagnosis

The first-line diagnostic test is stool examination. The diagnostic yield of an enzyme-linked immunoassay is superior to the previously used microscopic evaluation.

28.9.3 Endoscopy

Endoscopy is usually normal in giardiasis. Rarely, lymphoid hyperplasia of the entire small intestine (Fig. 28.15) or villous atrophy may be visible, for example, in patients with immunoglobulin A deficiency [51]. At endoscopy, it is possible to obtain duodenal aspirate for immediate microscopic examination, but histology from duodenal biopsy is preferable to duodenal aspirate. Histology usually demonstrates normal intestinal architecture with trophozoites adhering to the mucosa (Fig. 28.16).

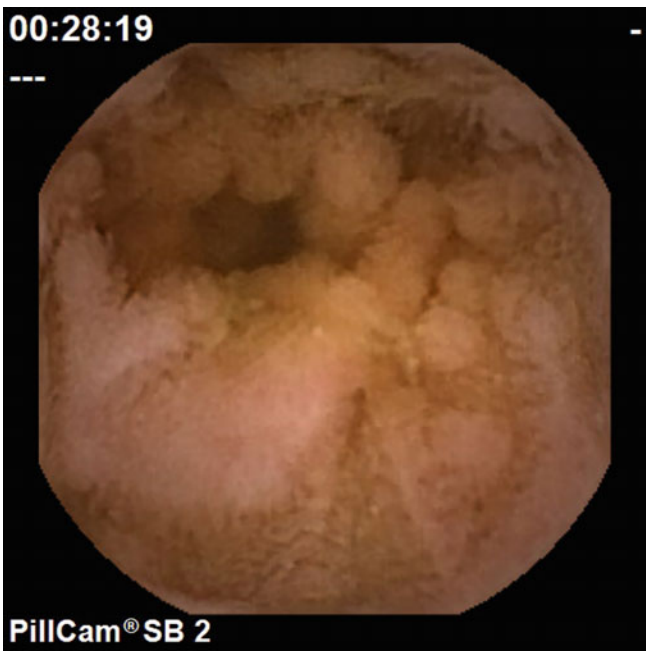

Fig. 28.15 Giardiasis. Lymphofollicular hyperplasia throughout the entire small intestine in a young patient with Giardiasis and underlying common variable immunodeficiency disease (CVID, Courtesy of Jörg Albert, MD)

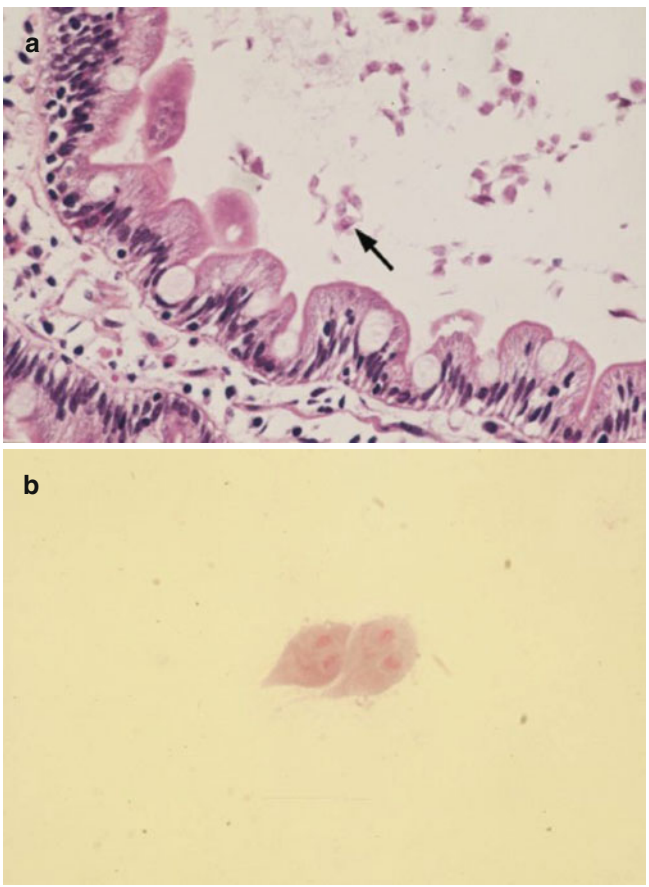

Fig. 28.16 Giardiasis. (**a**) Duodenal biopsy shows trophozoites in the overlying mucus. (**b**) Trophozoites at higher magnification

28.10 Helminthiases

28.10.1 Epidemiology

Helminth infections are more prevalent in the tropics and subtropics, so it is more common to find parasitic worms during capsule endoscopy in those regions [52]. Human pathogenic helminthes can be divided into nematodes (roundworms), cestodes (tapeworms), and trematodes (flukes). Nematodes are found most frequently in the small bowel lumen. *Ascaris lumbricoides*, the largest of these, may be up to 30 cm long. Smaller nematodes are the whipworm *Trichuris trichiura*, the pinworm *Enterobius vermicularis*, and the hookworms *Ancylostoma duodenale* and *Necator americanus* (up to 12 mm) and *Strongyloides stercoralis* (2 mm). For *Necator americanus*, occurrence of eosinophilic enteropathy has been observed by capsule endoscopy [53]. Tapeworms (*Taenia solium*, *Taenia saginata*, or *Diphyllobothrium latum/nihonkaiense*) may reach a length of up to several meters in the small intestine, causing malnutrition, vitamin deficiency, obstruction, and even intestinal bleeding [54, 55].

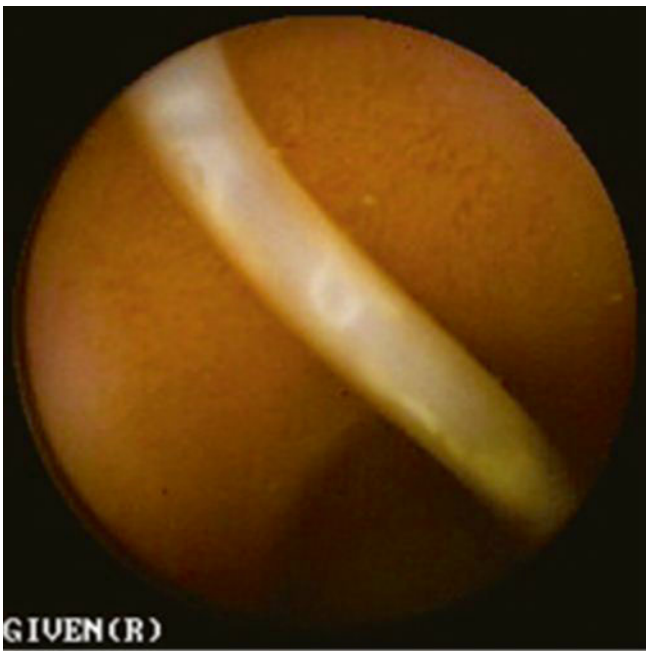

Fig. 28.17 *Ascaris* as seen on VCE

28.10.2 Development

Adult nematodes live in the human intestine, and therefore humans are the definitive host. The infection may be acquired through the oral ingestion of eggs or by larvae penetrating the skin. With strongyloidiasis or *E. vermicularis* infection, autoinfection can occur.

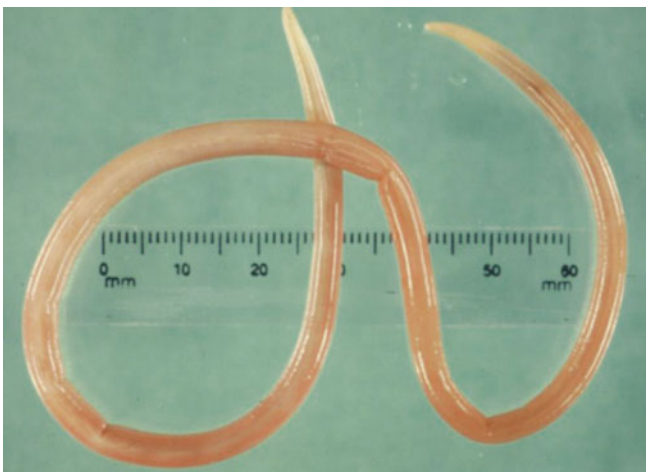

Fig. 28.18 Adult *Ascaris*

28.10.3 Endoscopy

Motile worms of varying size may be observed in the bowel lumen [56]. Ascarids can easily be identified by their large size (Figs. 28.17 and 28.18). On the other hand, the tiny *Strongyloides* is hardly visible at all (Fig. 28.19). Flexible endoscopy with the possibility of biopsy sampling may be desirable [57–59]. *Enterobius* usually occurs in the cecum, where multiple small worms may be seen (Fig. 28.20). *Trichuris* is a long worm (smaller than *Ascaris*) with a thin proximal end (Fig. 28.21), which is not seen in hookworms (Figs. 28.22 and 28.23). The largest helminths are tapeworms, consisting of multiple proglottides and reaching a length of several meters (Figs. 28.24 and 28.25) [60]. Visualization of adult worms at endoscopy or in stool, sometimes combined with microscopic detection of worm eggs, enables a correct classification of the parasites. Serology is rather insensitive because of frequent cross-reactions.

The outer cuticle of the parasites remains intact even after the worms have been killed. Wormlike structures with irregular outlines in the small bowel are usually food residues.

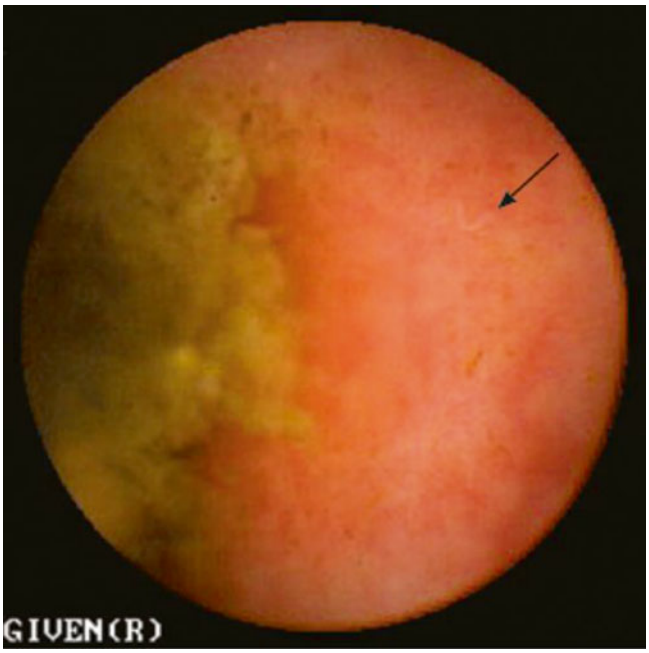

Fig. 28.19 *Strongyloides* (*arrow*). This small worm is hardly visible on the still image. (Courtesy of Annette Stelzer, MD)

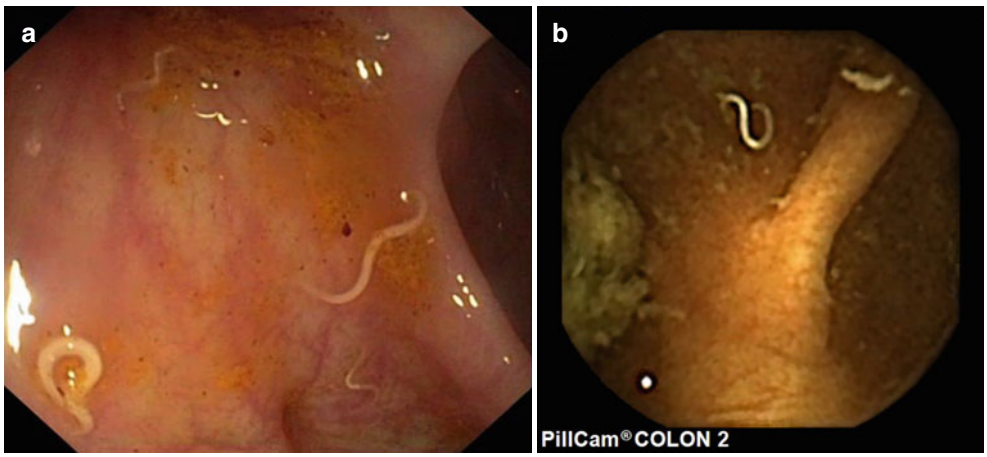

Fig. 28.20 (**a**) Colonoscopy shows pinworms (*Enterobius vermicularis*) in the cecum of a man with chronic anal fissures. (**b**) Incidental finding of pinworms in the cecum by PillCam COLON 2, which was used to complement an incomplete colonoscopy. Additionally, a polyp in the right colon was detected. (Courtesy of Michael Philipper, MD)

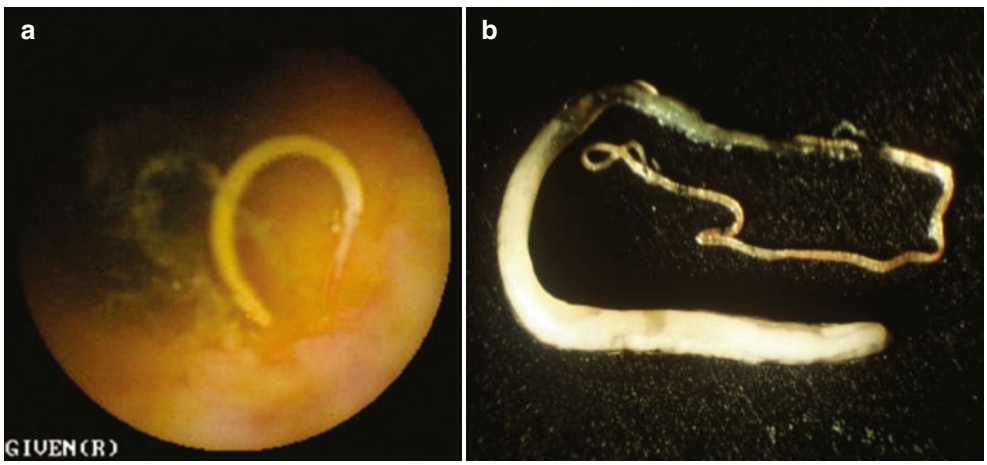

Fig. 28.21 Whipworm. (**a**) Image from VCE in the small intestine (Courtesy of Bruno Neu, MD.) (**b**) Adult whipworm

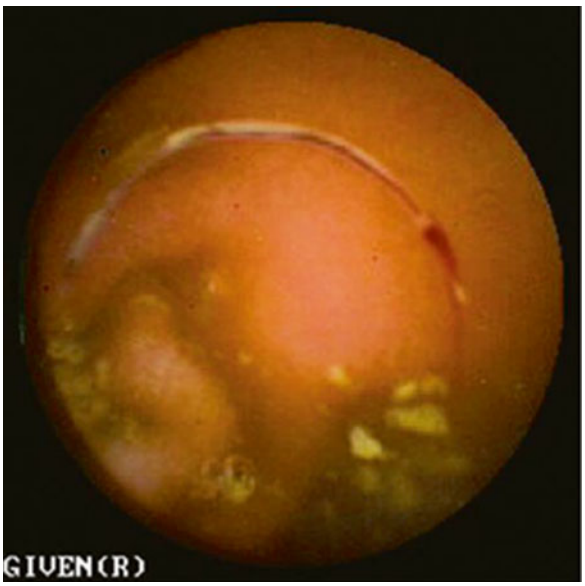

Fig. 28.22 Worm in the small intestine, most likely hookworm

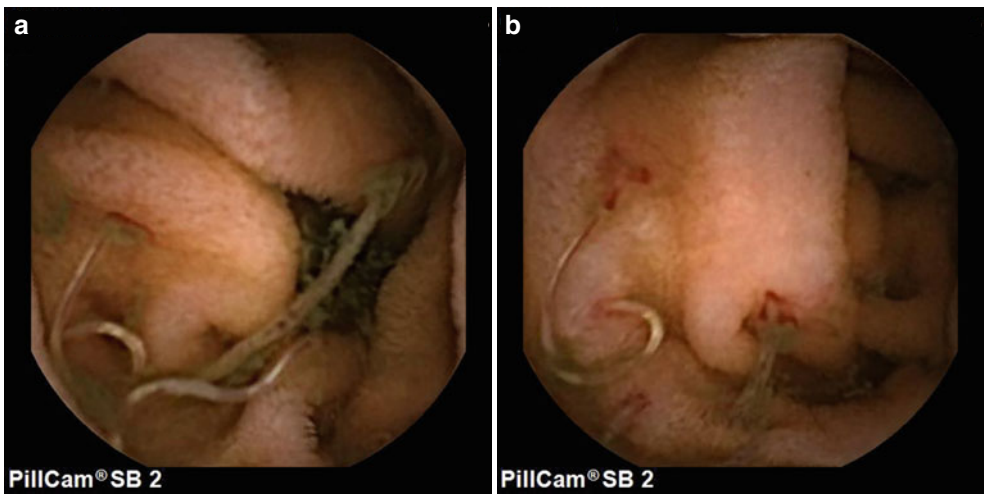

Fig. 28.23 (**a**, **b**) Hookworms penetrating the small bowel mucosa with visible blood loss in a patient with anemia (Courtesy of Selva Mony, BSC, RN, and Tariq Iqbal, MD)

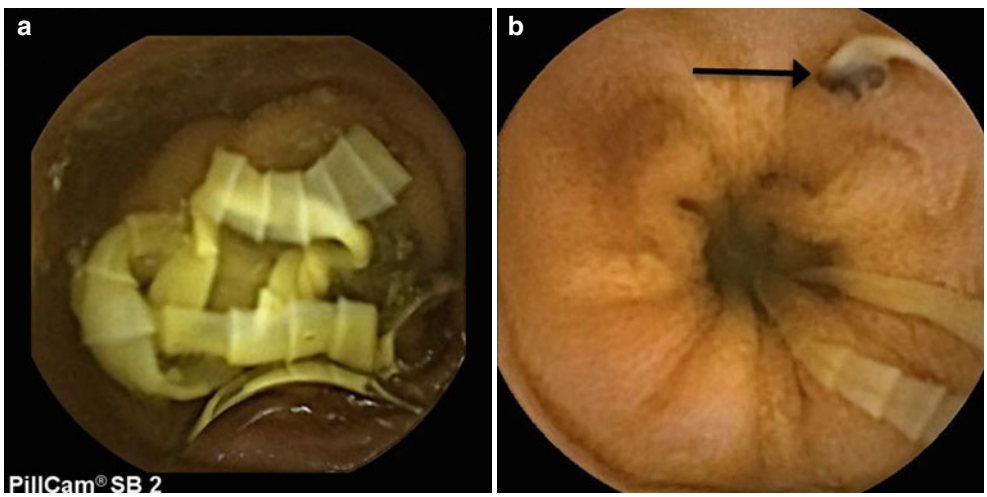

Fig. 28.24 *Taenia*. (**a**) The proglottides are clearly visible. (**b**) The head is visible (*arrow*) (Courtesy of Ingo Steinbrück, MD)

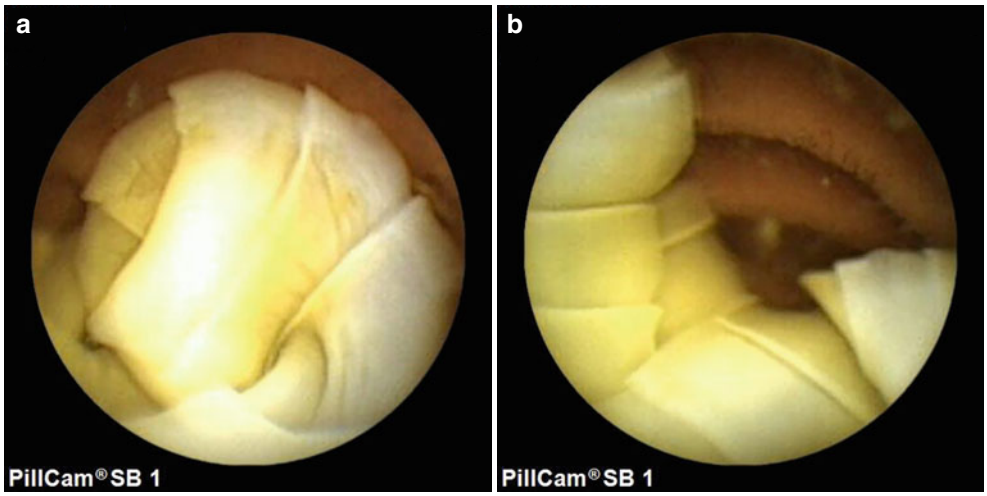

Fig. 28.25 (**a**, **b**) *Taenia*. Lumen-filling proglottides (Courtesy of Wolfgang Fortelny, MD)

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