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Symptoms related to anorectal diseases are common among patients presenting to gastroenterologists and are sometimes challenging to diagnose and manage. During daily endoscopy practice, anorectal lesions are poorly recognized probably due to a lack of understanding and knowledge. Painless rectal bleeding and anal pain are usually caused by hemorrhoids or anal fissures. However, a high index of suspicion for anorectal malignancy should be maintained in small ulceration or fissure with slightly exophytic and indurated margins, as well as ulceroinfiltrative mass. This chapter will discuss the endoscopic diagnosis of common anorectal diseases, such as hemorrhoids, anal fissures, and hypertrophied anal papillae, as well as important conditions seen less frequently, such as anal malignancy.

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24.1 Anorectal Anatomy

Understanding anorectal anatomy is a key to evaluate anorectal diseases. Despite its short length, the anal canal produces a variety of diseases reflecting its complex anatomic and histological structure (Table 24.1, Fig. 24.1).

The most important endoscopic landmark is the dentate line, which is the mucocutaneous junction of columnar epithelium, originated from endoderm, and squamous epithelium, originated from ectoderm (Fig. 24.1c) [1, 2]. Endoscopic finding of the dentate line is a wavy demarcation formed by the anal valves (transverse folds of mucosa) at the inferior-most ends of the anal columns, sometimes accompanied with hypertrophied anal papillae (Fig. 24.1b, c).

Histologically, the mucosa of anal canal can be divided into three zones. The upper part is covered with columnar

epithelium-like colonic mucosa. The middle part is the transitional zone, where squamous epithelium gradually changes to cuboidal epithelium and then columnar epithelium [3, 4]. Endoscopically, transitional zone shows a slightly whitish or gray-colored mucosa of 6–12 mm in length just above the dentate line on the retroflexion view (Fig. 24.1b). The lower part extends from the dentate line and downwards to the anal verge and has been called anoderm (anal canal skin). It is covered by modified squamous epithelium devoid of skin appendage (hair follicles, sebaceous glands, and sweat glands), which may be partly keratinized, particularly in case of mucosal prolapse. Anal verge between the anoderm and perianal skin can be inspected on slight retraction of the perianal skin. Because the anoderm is seen as pale, smooth, shiny, and hairless when stretched compared to perianal skin, anal verge can be delineated (Fig. 24.1d).

Table 24.1 Anatomy of the anal canal and rectum

Structure	Boundary	Endoscopic finding
Rectum (Fig. 24.1a)	Rectosigmoid junction ~ anal canal	Usually have three Houston's valves (transverse folds of rectum)
(Surgical) anal canal	Anorectal ring ~ anal verge	Average 4 cm in length Surrounded by the anal sphincter mechanism
Anatomic anal canal	Dentate line ~ anal verge	Average 2 cm in length Covered with modified squamous epithelium (anoderm)
Anorectal ring (Fig. 24.1b)	Upper border of the internal anal sphincter and the puborectalis muscle	Anorectal ring cannot be seen under endoscopy, but can be palpable at approximately 1–2 cm proximal to the dentate line on digital rectal examination
Dentate line (pectinate line, Fig. 24.1b, c)	Mucocutaneous junction between columnar epithelium and squamous epithelium	Dentate line can be delineated under endoscopy, but cannot be felt in digital rectal examination Nerve innervation, blood supply, and lymphatic drainage are different above and below dentate line
Anal verge (anal margin) (Fig. 24.1d)	Junction between the anoderm and perianal skin	

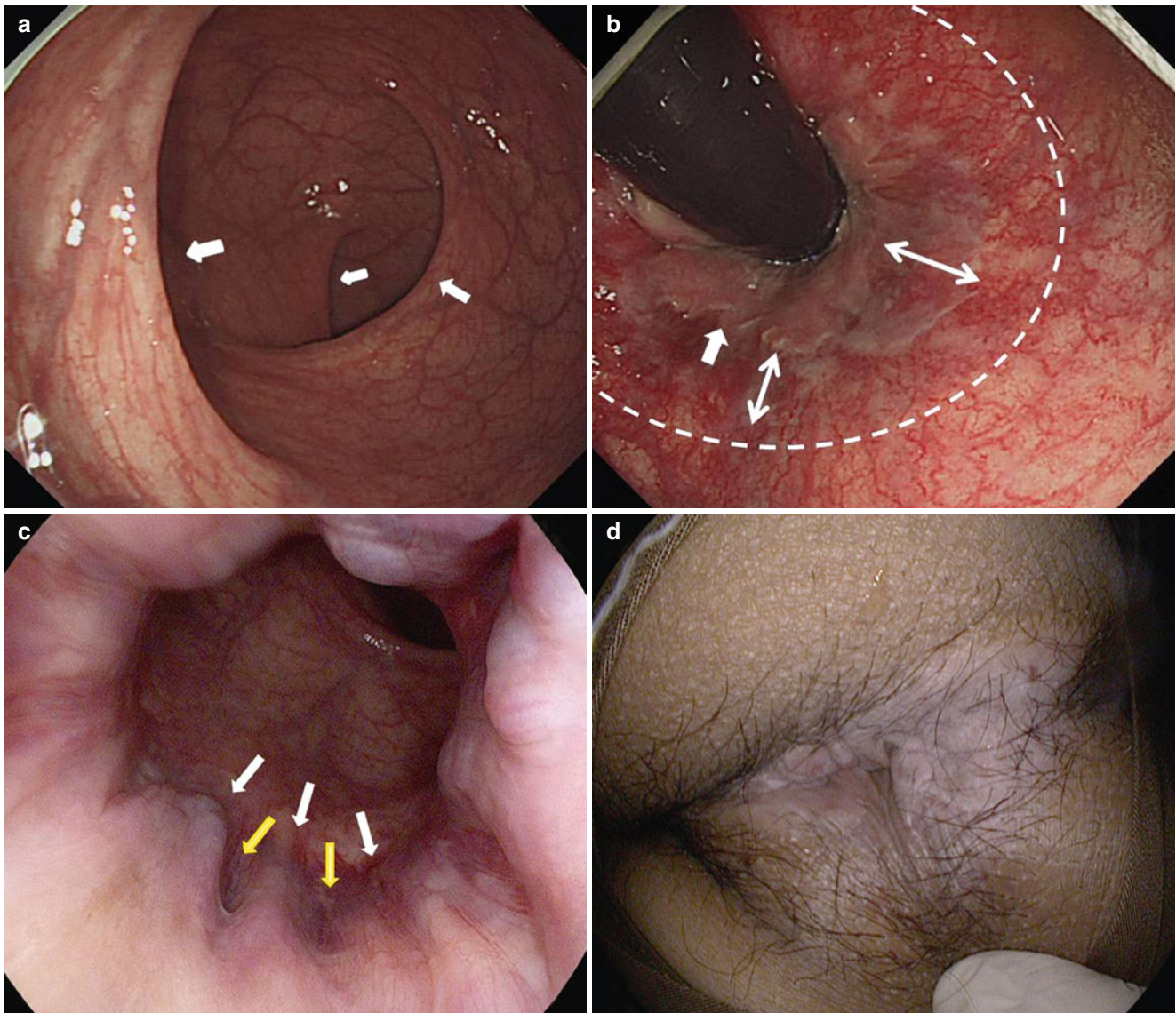


Fig. 24.1 Normal anorectal anatomy. (a) Rectum. *White arrows* (→) indicate the three Houston's valve of rectum. (b) Retroflexion view of distal rectum. *White arrow* indicates dentate line (→). *Double arrow* (↔) means the transitional zone. On the 1–2 cm proximal to transition zone, the anorectal ring is located (---), which can be palpable on digital rectal examination and cannot delineate exactly on endoscopy.

(c) Forward view of anal canal. Dentate line shows as wavy demarcated small valve-like transverse folds of mucosa at the base of anal columns of Morgagni (*white arrow*→) and anal crypt (*yellow arrow*→). (d) Forward view of anal verge. Anal verge can be delineated on slight retraction of the perianal skin

24.2 Anal Cancers

Anal cancer comprises only 1.5 % of all gastrointestinal cancers. Almost 80 % are squamous cell cancers, 16 % are adenocarcinomas, and 4 % are other types. Although incidence rates are still low, there has been a significant increase in squamous cell carcinoma over the last 50 years. The development of anal cancer has been associated with human papillomavirus (HPV) and human immunodeficiency virus (HIV) infections, history of receptive anal intercourse, history of sexually transmitted diseases, history of cervical cancer, and use of immunosuppressive medication after solid organ transplantation.

Anal bleeding is the most common symptom (45 %), followed by the anal pain or sensation of a mass (30 %). Unfortunately, these symptoms are often erroneously attributed to hemorrhoids, with subsequent delay in the diagnosis and treatment. The clinician should remember that hemorrhoids rarely cause pain (unless thrombosed); thus, patients presenting with anal pain should be carefully evaluated. Furthermore, 20 % of patients have no symptoms at the time of diagnosis. For early diagnosis of anal cancer, the careful physical examination with inspection and digital

rectal examination before endoscopy practice and routine retroflexion for complete visualization of distal rectum and anal canal during endoscopy practice should be warranted.

24.2.1 Anatomic Subsites of Malignant Anorectal Diseases

The determination of the anatomic site of carcinomas that overlap the anorectal junction may be problematic. Carcinomas located on the anorectal junction should be classified as anal canal cancers if their epicenter is 2 cm or less from the dentate line and rectal cancers if their epicenter is located more than 2 cm proximal to the dentate line or proximal to the anorectal ring on digital rectal examination (Fig. 24.2) [5]. While tumors arising in the distal anal canal usually are keratinizing squamous cell carcinomas, those arising in the transitional mucosa often are nonkeratinizing squamous cell carcinomas, which were classified as transitional cell or cloacogenic type. Recently, all nonkeratinizing squamous cell carcinomas of anal canal are considered to be variants of squamous cell carcinoma due to similar behavior with keratinizing squamous cell carcinoma.

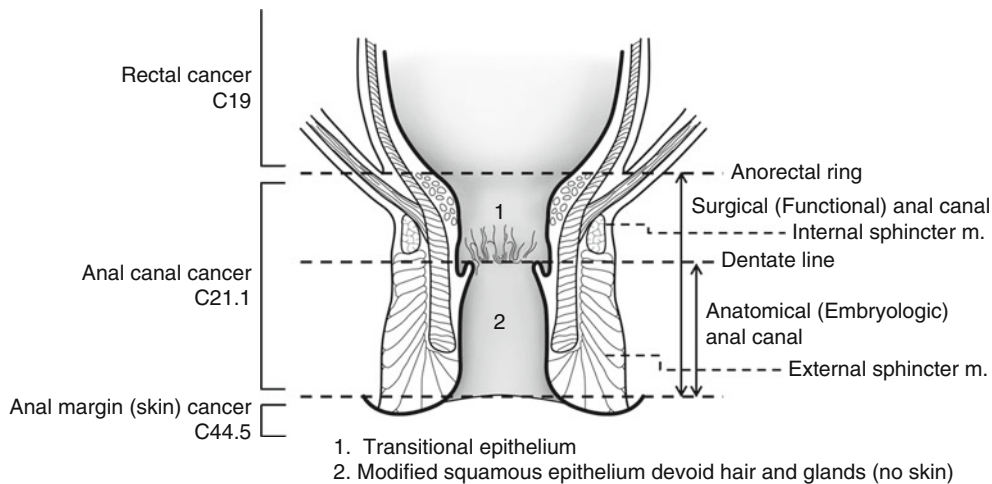


Fig. 24.2 Anatomic subsites of malignant anorectal diseases

24.2.2 Anal Canal Cancers

During a digital rectal examination before endoscopy, anal canal cancer can be identified as indurated firm nodule, lump, or mass, often with a rough or ulcerative surface, and not freely mobile. During endoscopy, the anal canal cancer may present as a small ulceration or fissure with slightly exophytic and indurated margins and irregular thickening of the anoderm and anal margin (Fig. 24.3) [5]. Anal cancer

also showed a nodular mass with an irregular surface protruding into anal canal (Fig. 24.4). The lesion may have a different color from the surrounding tissue. In advanced stages, ulceration and infiltration develop and the lesion becomes fixed to the underlying structures and may bleed (Fig. 24.5). To obtain complete view of anal canal cancer, the endoscope should be retroflexed to inspect the distal rectum and anal canal. Sometimes, small-sized tumors can be identified only in retroflexion examination of endoscope.

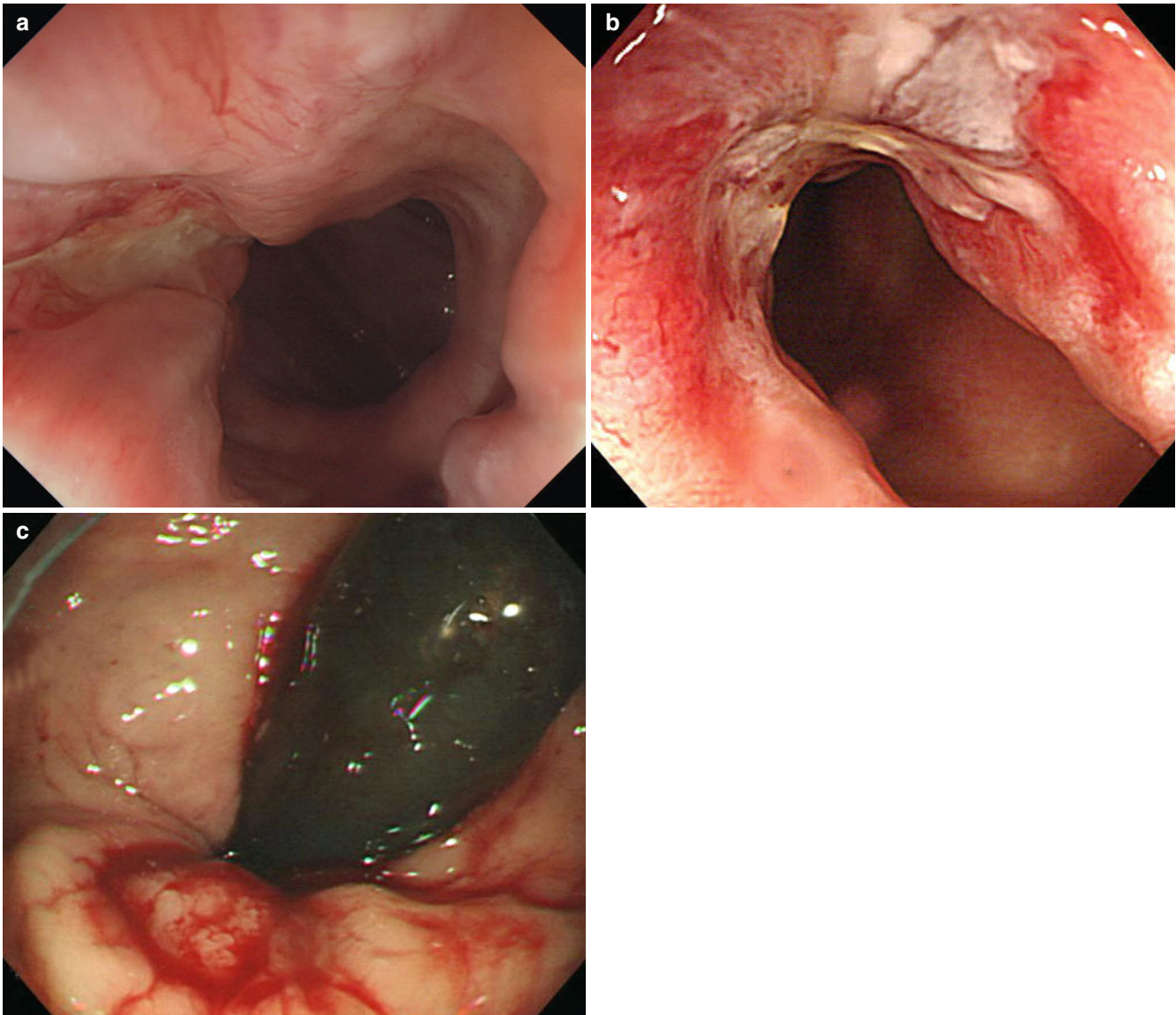


Fig. 24.3 Anal canal cancers. (a) Fissure-like squamous cell carcinoma with slightly indurated margin. (b) Small ulcerative-shaped squamous cell carcinoma with irregular thickening of the anoderm. (c)

Shallow discrete depressed-type squamous cell carcinoma covered with friable mucosa, which caused easy touch bleeding during defecation and endoscopy

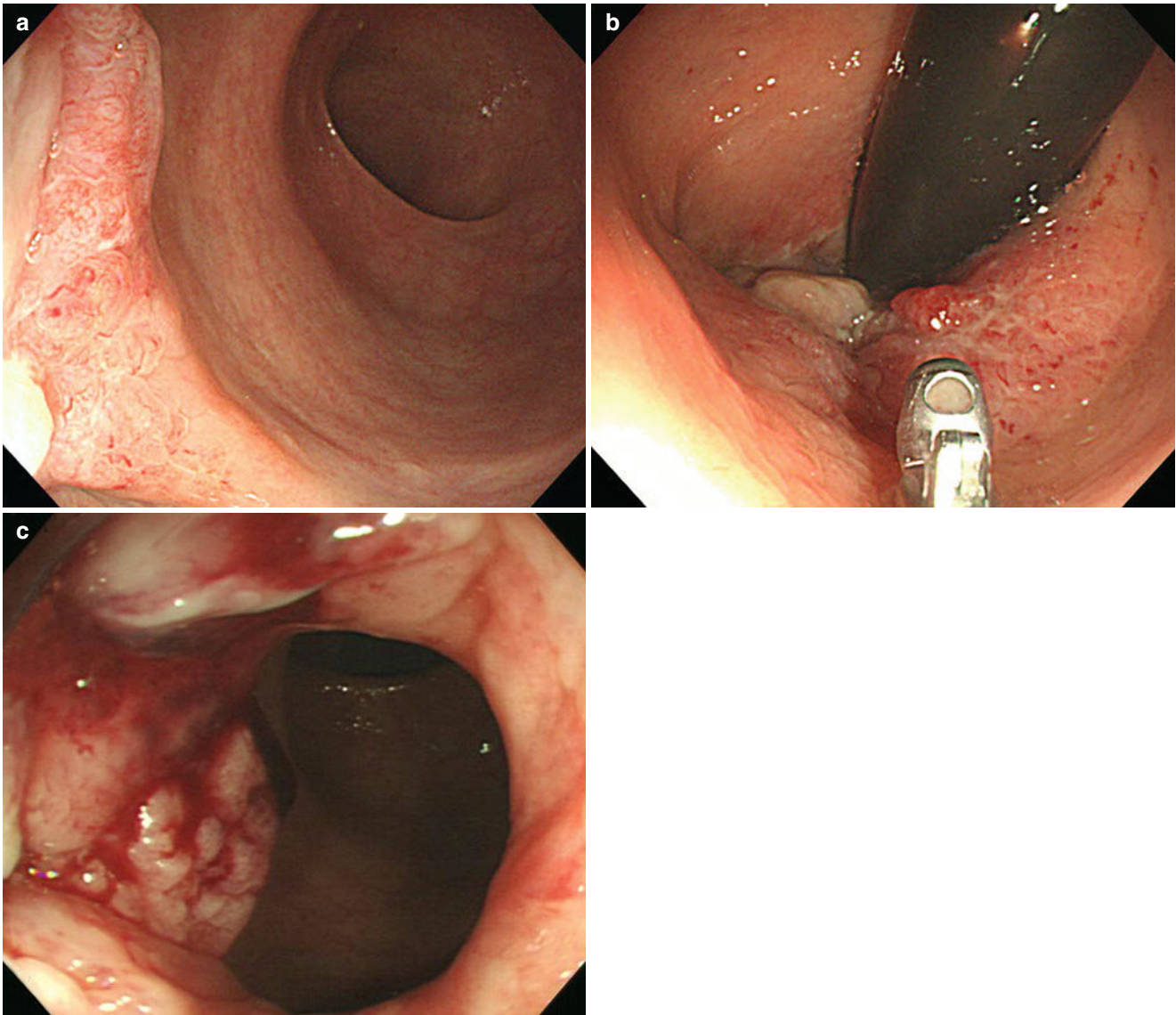


Fig. 24.4 Anal canal cancers. (a) Nodular irregular-surfaced adenocarcinoma arising from anal canal. (b) Its epicenter is 2 cm or less from the dentate line on the retroflexion. (c) Nodular irregular-surfaced pro-

truding squamous cell carcinoma is covered with friable mucosa, which caused easy touch bleeding during defecation and endoscopy

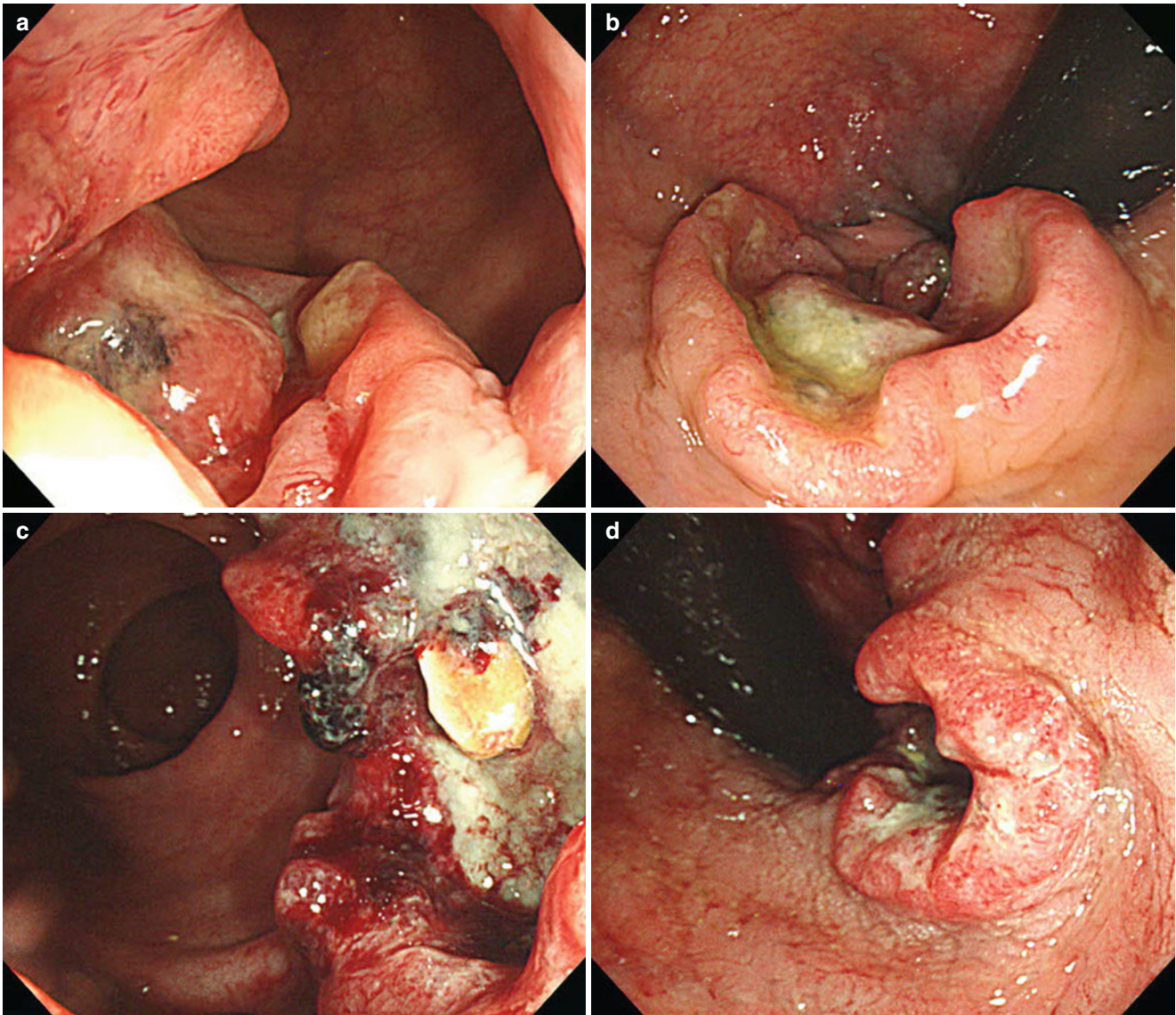


Fig. 24.5 Advanced anal canal cancers. (a) Deep ulceroinfiltrative-shaped squamous cell carcinoma. (b) 3 cm-sized ulcerative-shaped squamous cell carcinoma with exophytic margin. (c) Bleeding from

base of ulceroinfiltrative-shaped squamous cell carcinoma. (d) Deep ulceroinfiltrative-shaped squamous cell carcinoma mass with raised nodular margin

24.2.3 Anal Margin Cancers

Anal margin cancer is rare squamous cell carcinoma arising distal to the anal verge. It is considered as skin cancers and treated as such. Small lesions (<4 cm²) with

no fixation to deeper tissues are excised widely, while squamous cell cancer of the anal margin that is deeply invading is treated by chemoradiation. Careful inspection of perianal skin before insertion of endoscope is necessary (Fig. 24.6).



Fig. 24.6 Anal margin cancer. Firm hyperemic and salmon-colored patch with superficial erosions was noticed on the perianal skin distal to the anal verge

24.2.4 Anal Melanoma

Anal melanoma is rare but is an aggressive malignancy. Anal melanoma is the third most common melanoma after the cutaneous and ocular varieties. Anorectal melanoma arises from melanocytes identified within the squamous mucosa of

the anal canal. Endoscopic diagnosis can be made with visual inspection of black-colored nodular or ulcerative lesion with raised and indurate margins (Fig. 24.7). Early diagnosis and surgical excision offer the only chance for cure. However, the patients commonly present with advanced, even meta-static, disease.

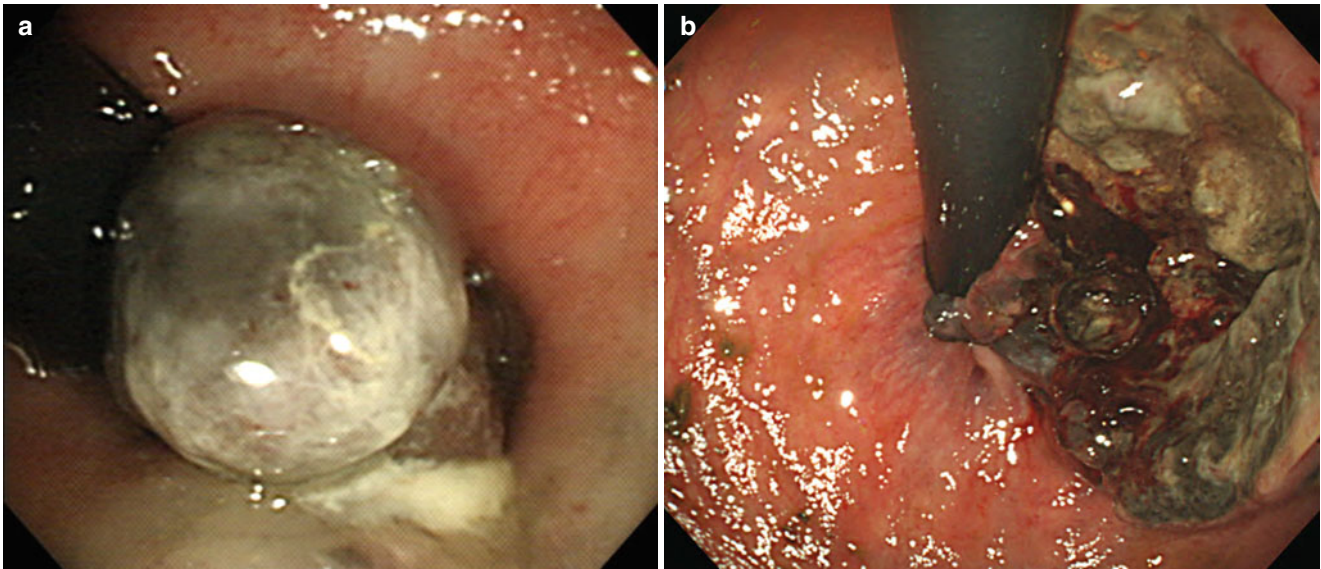


Fig. 24.7 Anal melanoma. (a) Black-colored polypoid lesion on anal canal. (b) Dirty exudates-covered black-colored ulceroinfiltrative mass arising from anal canal

24.3 Anal Intraepithelial Neoplasia

Anal intraepithelial neoplasia (AIN) is precursor of anal cancer, defined as a dysplastic condition of the epithelium of the anal canal and perineal skin. There are several other commonly used terms for this condition, such as squamous cell carcinoma in situ, anal squamous intraepithelial lesion, and

Bowen's disease. AIN is associated with HPV infection and more common in HIV-positive persons. AIN typically presents as erythematous plaque with an irregular border and surface crusting or scaling (Fig. 24.8). Several treatments have been described such as surgical excision, infrared coagulation, and anoscopy-directed lesion ablation. Prior to treatment, a thorough assessment should be performed to exclude anal cancer.

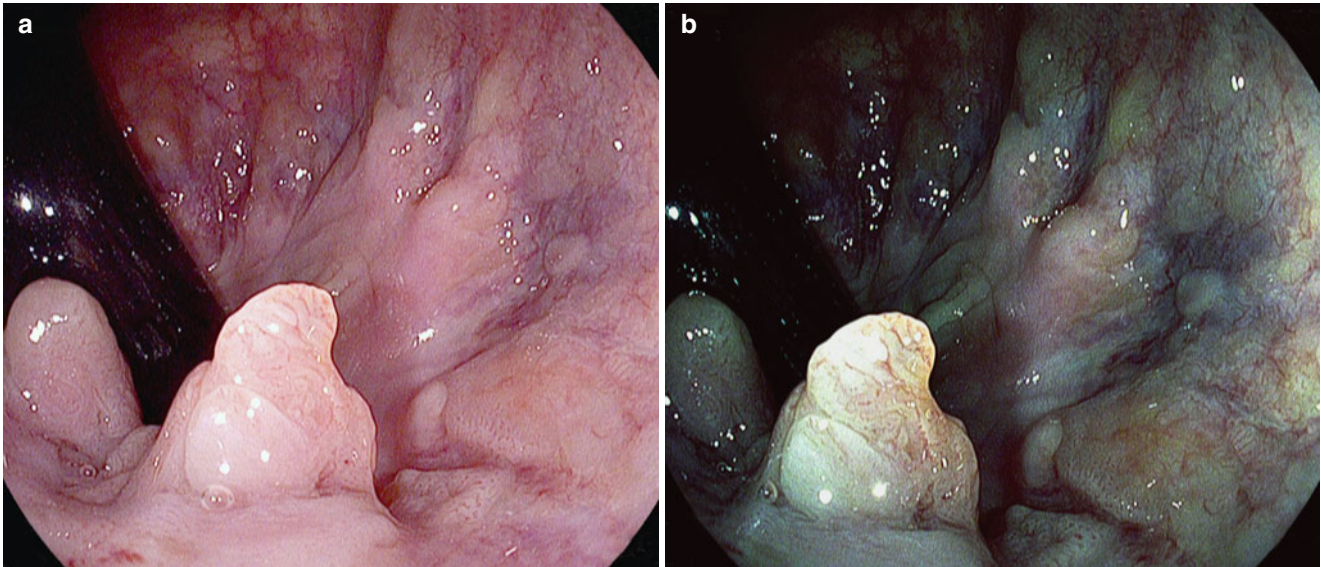


Fig. 24.8 High-grade anal intraepithelial neoplasia (squamous cell carcinoma in situ). (a) Verrucous-shaped demarcated erythematous plaque with an irregular surface is noticed on dentate line. (b) Image-enhancing image using i-Scan

24.4 Condylomata Acuminata (Anal Warts)

Condylomata acuminata, so-called anal warts, are caused by sexual transmission of HPV, although nonsexual transmission is possible. Condylomata acuminata are hypopigmented verrucous papules or plaques and vary

from very small papule to cauliflower-like bulky lesions (Fig. 24.9). Treatment is optional since these lesions do not directly progress to invasive cancer. However, patients may tend to have therapy for a variety of reasons including symptoms (burning, itching, and bleeding) or psychological distress.

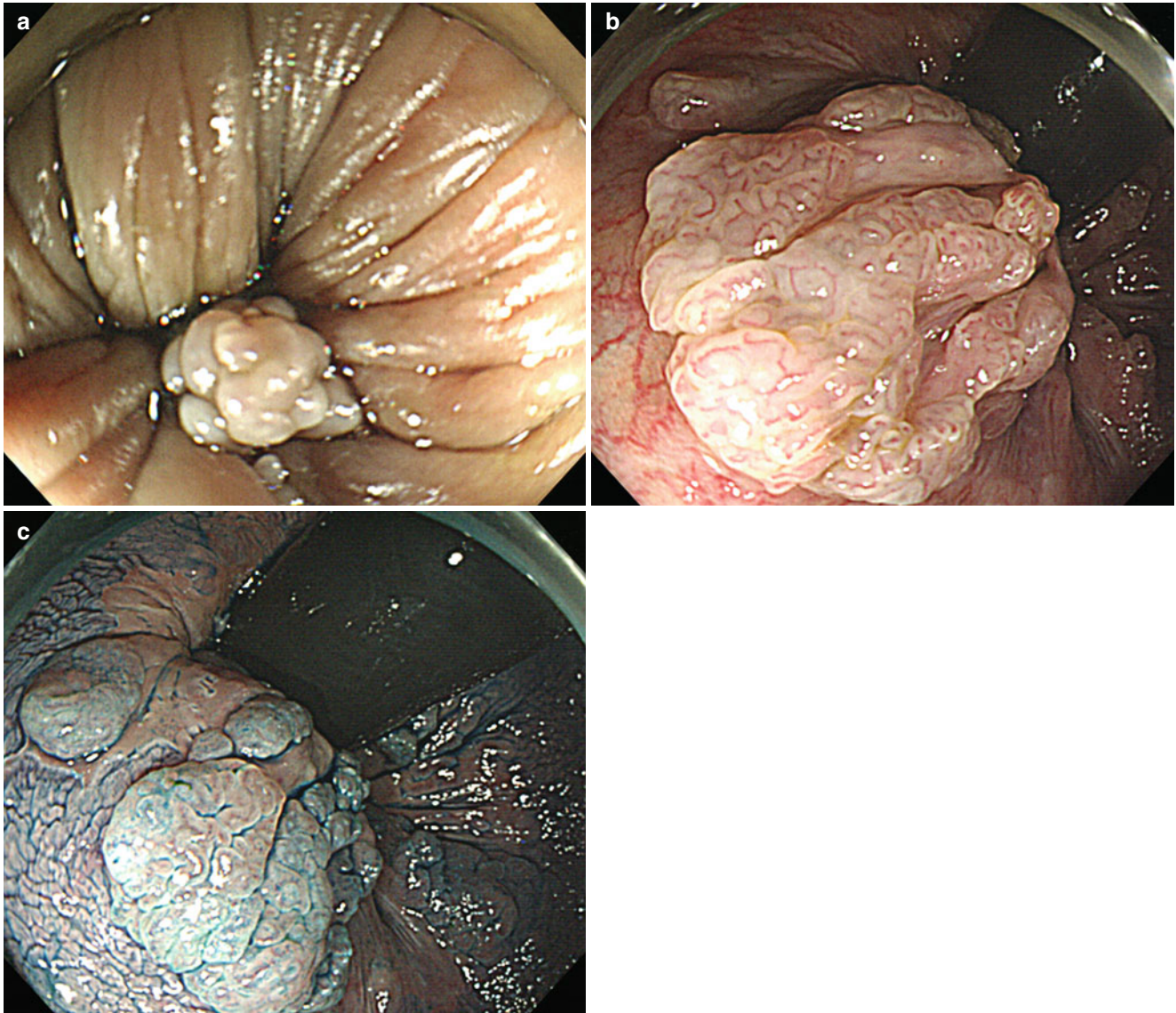


Fig. 24.9 Condylomata acuminata. (a) Small cauliflower-like surfaced polypoid lesion. (b) Geographic-shaped flat raised verrucous anal warts were noticed on retroflexion view. (c) Chromoendoscopy view using indigo carmine

24.5 Hemorrhoids

Hemorrhoids are a very common anorectal condition in general population. Hemorrhoids are classified according to their location and degree of prolapse. Internal hemorrhoids arise above the dentate line, covered by columnar or transi-

tional epithelium, and cause painless bleeding, prolapse, pruritus, hygienic disturbances, and anemia [2]. External hemorrhoids arise distal to the dentate line, are covered with squamous epithelium, and rarely bleed, although they do swell and cause pain. Mixed (interno-external) hemorrhoids arise both above and below the dentate line.

24.5.1 Internal Hemorrhoid

Internal hemorrhoids define as the symptomatic enlargement and distal displacement of the normal anal cushions. Anal cushions located on the anal canal prevent the tearing of anal canal during defecation, aid in anal continence, and provide a complete seal of the anus in closure. The abnormal dilatation and distortion of the hemorrhoidal plexus in anal cushion with destructive changes in the supporting connective tissue within the anal cushion is a paramount finding of hemorrhoids. It appears that the dysregulation of the vascular tone and vascular hyperplasia might play an important role in hemorrhoidal development and could be a potential target for medical treatment.

Bleeding is most common symptom and typically painless. Patients describe bright red blood usually seen on the toilet tissue, dripping into the toilet bowl, or streaking the outside of a hard stool. If the patient has chronic hemorrhoidal prolapse, blood or mucus against the anal skin might lead to itching.

The diagnosis of internal hemorrhoids is best made with the beveled anoscope; however, flexible endoscopy can evaluate internal hemorrhoid using forward and retroflexion view of the anorectal junction. Endoscopically, the vascular

cushions can be seen to bulge into the anal lumen, or the tissue might prolapse out through the anal canal (Figs. 24.10, 24.11, and 24.12). It is important to note that hemorrhoids are dynamic structures and maybe symptomatic only intermittently. If the patient is seen outside of a flare, the hemorrhoids can appear entirely normal. For practical purposes, internal hemorrhoids are further graded based on their appearance and degree of prolapse, known as Goligher's classification (Table 24.2).

Fiber supplements improve symptom and bleeding and should be recommended in all stages of hemorrhoidal disease. A variety of topical ointments is available and contains a combination of local anesthetics, corticosteroids, and vasoconstrictants. Flavonoid as a dietary supplement has been used as a treatment to reduce congestion and inflammation. Rubber-band ligation is the most effective for patients with second- and third-degree internal hemorrhoids. Although rubber-band ligation usually is assisted by rigid anoscopy, flexible endoscopic band ligation is possible and an effective and safe method of treatment in patients with symptomatic internal hemorrhoids. Surgical treatment is reserved for grade IV hemorrhoids or for lower-grade hemorrhoids that have not responded to conservative treatments.

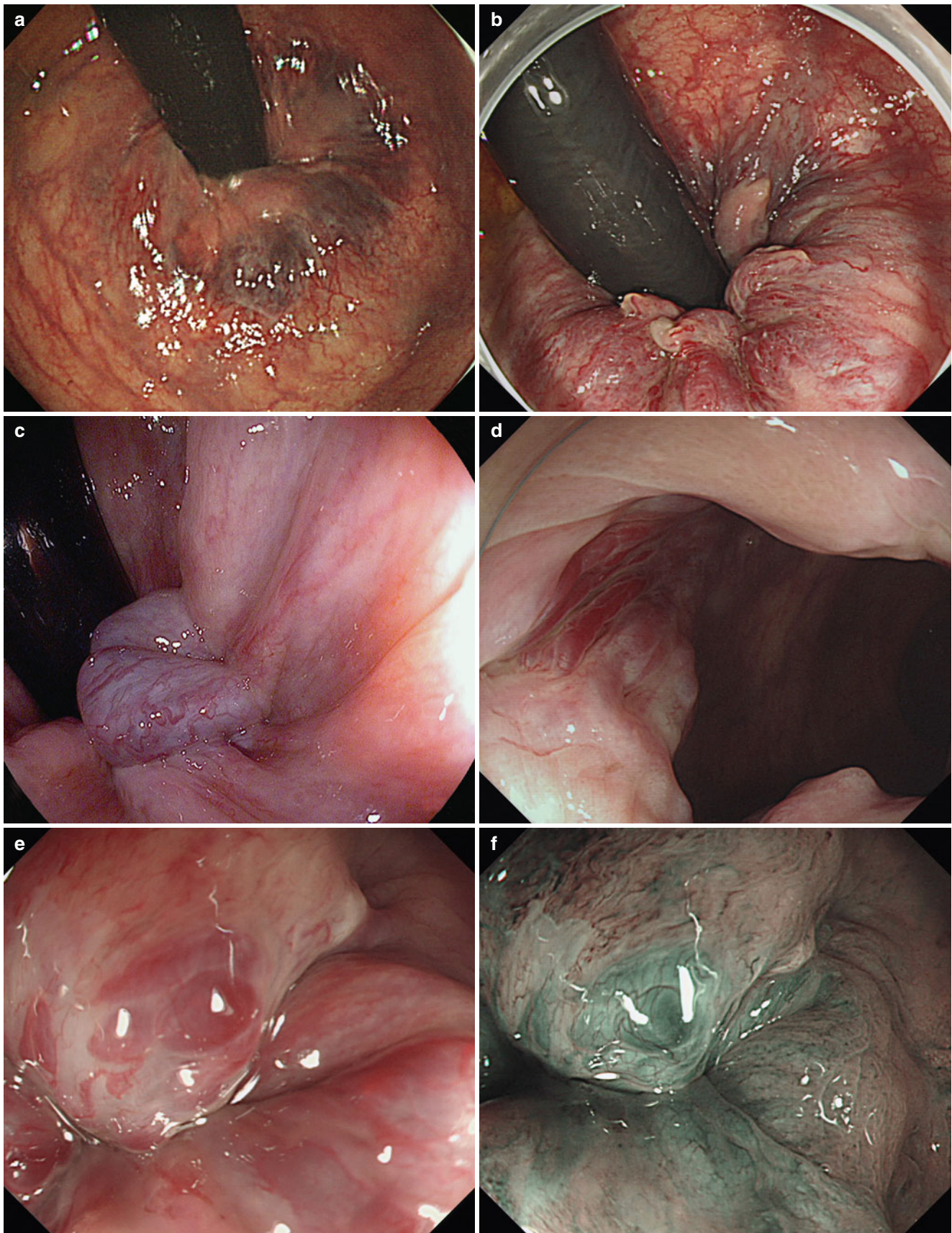


Fig. 24.10 Internal hemorrhoids, grade I. (a) Small non-bleeding internal hemorrhoids are seen in retroflexion. (b) Small non-bleeding internal hemorrhoids with hypertrophied anal papillae are shown. (c) Prominent internal hemorrhoid without prolapse. (d) Forward view of

small non-bleeding internal hemorrhoids with dilated vascular channel. (e) Dilated vascular channel on internal hemorrhoid, which is the equivalent of the red color sign of esophageal varices. (f) Narrowband imaging of internal hemorrhoids

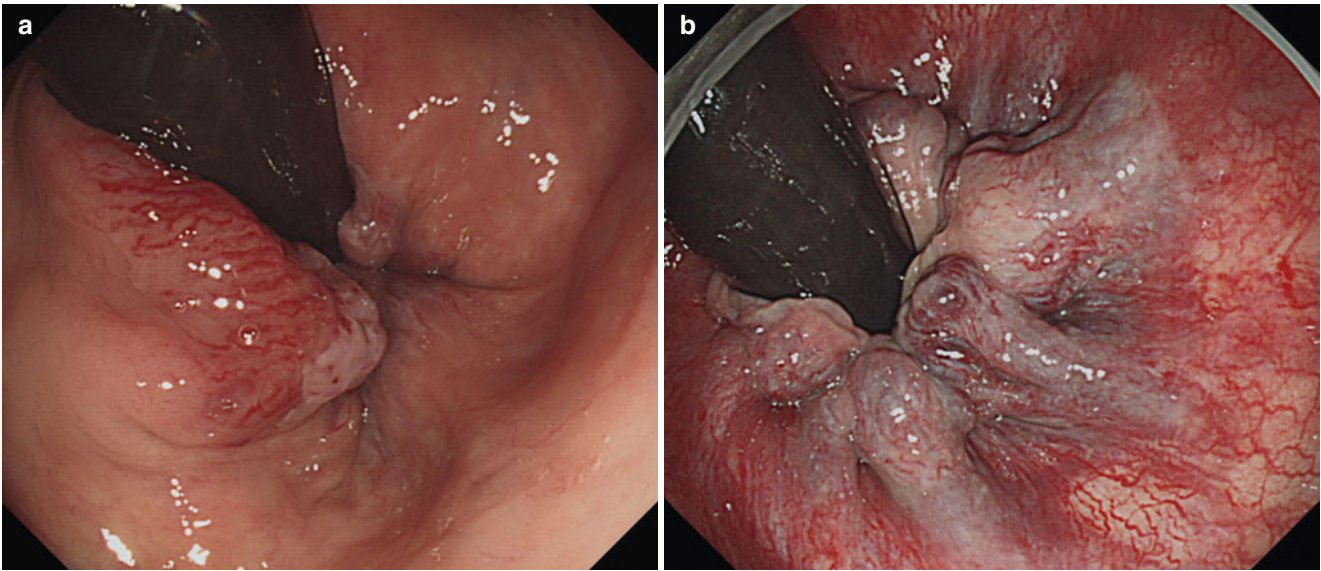


Fig. 24.11 Internal hemorrhoids, grade II. (a) Prominent non-bleeding grade II internal hemorrhoids. (b) Prominent internal hemorrhoids with dilated vascular channel. (c) Prominent non-bleeding internal hemorrhoids. (d) Congested prominent internal hemorrhoids with reddish

color at the 5-, 9-, or 12-o'clock position seen in retroflexion. (e, f) Protrusion of the anal cushions beyond the anal verge with straining and returning spontaneously

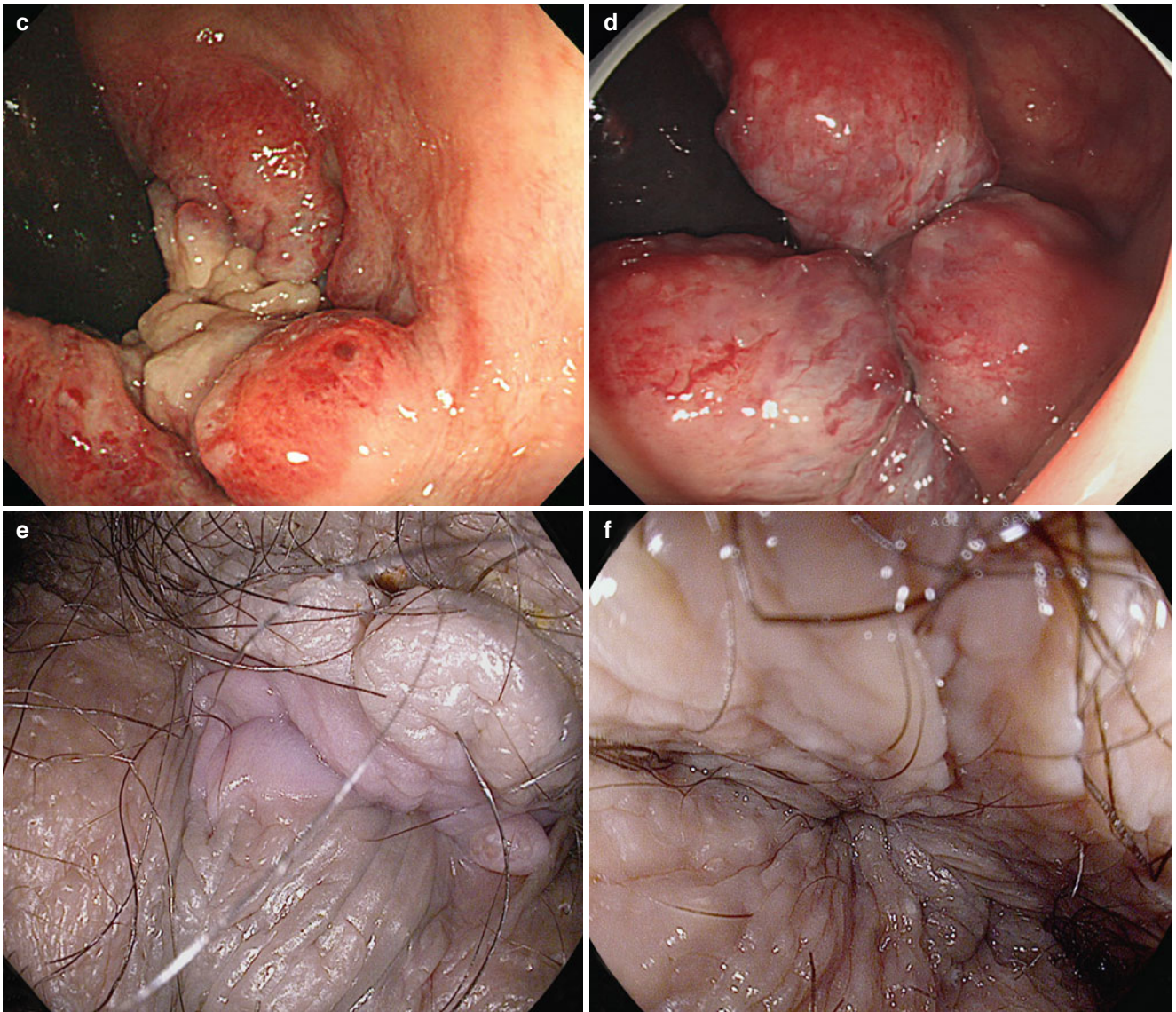


Fig. 24.11 (continued)

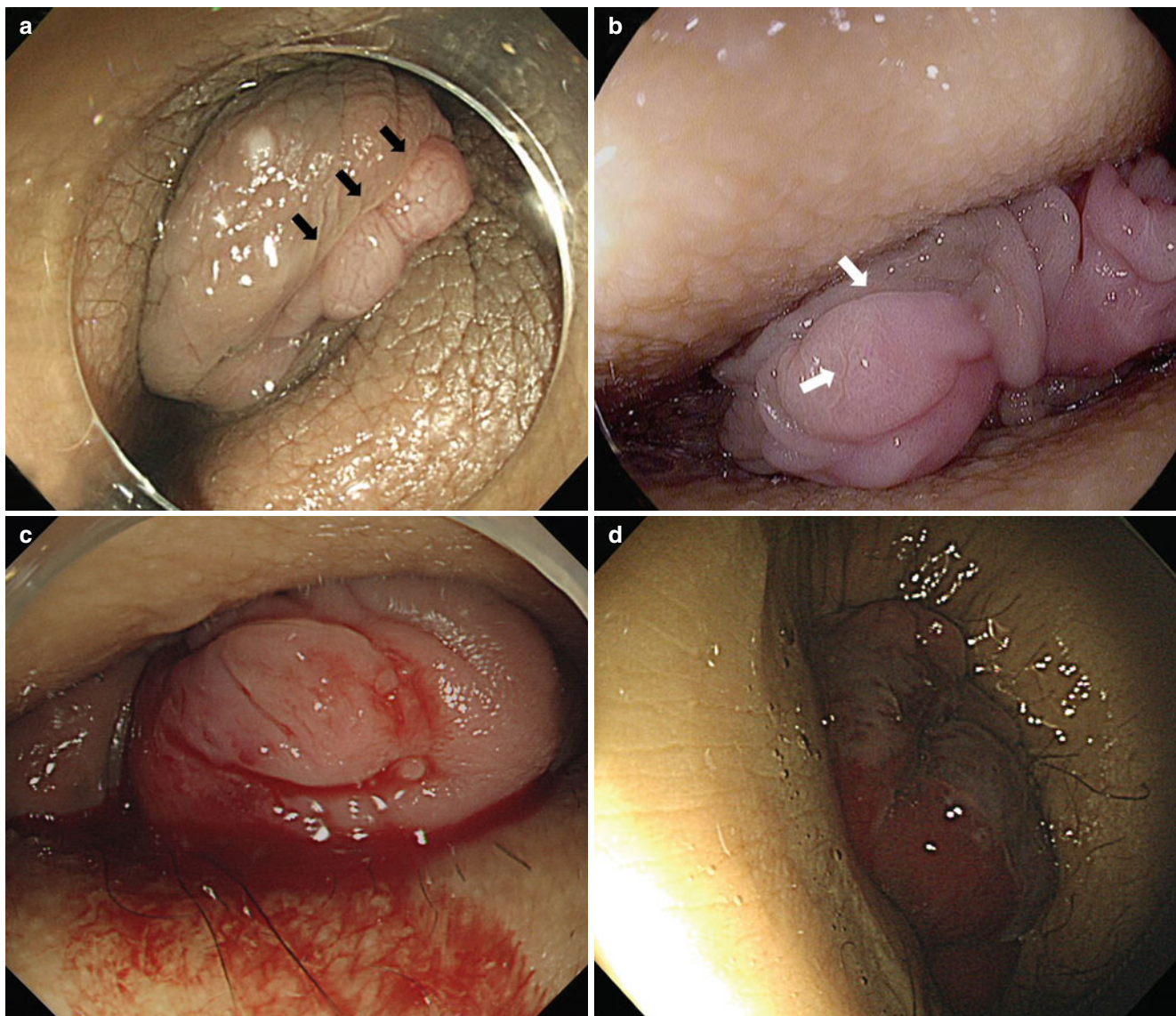


Fig. 24.12 Internal hemorrhoids, grade III or IV. (a) Reducible prolapsed hemorrhoid, but fall out again. Observed dentate line (→) indicates mixed interno-external hemorrhoids. (b) Prolapsed hemorrhoid

stays out at all times and is irreducible. (c) Prolapsed grade III internal hemorrhoids with bleeding. (d) Thrombosed internal hemorrhoids involving circumferential rectal mucosal prolapse

Table 24.2 Grade of internal hemorrhoid

Grade I	The anal cushions project into the anal canal with minimal bleeding or may be asymptomatic, but they do not prolapse
Grade II	The anal cushions protrude beyond the anal verge with straining or defecating and reduce spontaneously when straining ceases
Grade III	The anal cushions protrude either spontaneously or with straining and will require manual reduction into anal canal
Grade IV	The prolapse stays out at all times and is irreducible. If they are reducible, they fall out again Acutely thrombosed incarcerated internal hemorrhoids and incarcerated, thrombosed hemorrhoids involving circumferential rectal mucosal prolapse were included Irreducible, strangulated hemorrhoids are a surgical emergency

24.5.2 External Hemorrhoid

External hemorrhoids are visible at the anal verge and actually represent residual redundant skin from previous episodes of external hemorrhoidal inflammation and edema. They usually cause no symptoms and do not bleed, because they are covered with squamous epithelium. However, some people are uncomfortable during anal wiping due to irritation or the uncomfortable sensation of redundant tissue. Before endoscopy practice, inspection of anal verge using slight retraction of the perianal skin diagnosed the protrusion of external hemorrhoid (Fig. 24.13).

When dentate line can be identified, hemorrhoid considered as mixed interno-external hemorrhoids or grade IV prolapsed internal hemorrhoids. Treatment usually is reassurance and proper anal hygiene, including delicate washing of the anal area and avoidance of aggressive wiping with harsh toilet tissue. However, thrombosed external hemorrhoid can cause severe perianal pain. A painful, edematous, bluish and firm mass or nodule can be seen during perianal inspection (Fig. 24.13d). Sitz baths, ice packs, stool softeners, and oral analgesia are usually sufficient, but prompt surgical excision of the thrombus can shorten the recovery period.



Fig. 24.13 External hemorrhoids. (a) Small external hemorrhoid covered with squamous epithelium. (b) Large external hemorrhoid covered with squamous epithelium. (c) Large external hemorrhoid covered with

squamous epithelium. (d) External hemorrhoid with thrombus. A painful, edematous, bluish, and firm nodule can be seen during perianal inspection

24.6 Anal Fissure

Anal fissure is defined as a tear, crack, or ulceration of the anal canal (Fig. 24.14). It may be commonly brought on by the passage of a large, hard stool. They are most commonly seen in the posterior midline portion of the anal canal, following as anterior midline (Fig. 24.15). This area may have decreased blood flow due to the configuration of the vasculature of anus and loosen in support of the anoderm due to the arrangement of the anal muscles. When anal fissures are found laterally, Crohn's disease, occult abscesses, syphilis, tuberculosis, leukemic infiltrates, carcinoma, herpes, or HIV infection should be considered as underlying causes of anal fissure (Fig. 24.16) [2]. Pain and bleeding are developed associated with defecation. Spasm of the internal anal sphincter due to pain may exacerbate symptoms and induce more damage.

External inspection is usually sufficient to make a diagnosis. Gentle separation of the buttocks to expose the

perianal area may facilitate examination. Digital rectal examination produces extreme pain and sphincter tone is markedly increased. Endoscopy may result in significant pain in acute anal fissure and may require anesthesia. During the endoscopy, acute fissures show simply split or crack in the anoderm without exposed internal sphincter fibers, whereas chronic fissures show rolled edges, fibrosis of the edges, deep ulceration with exposure of the underlying internal sphincter muscle, enlargement of the tissue at the dentate line (hypertrophied anal papilla), and edematous skin tags at the distal anal verge (sentinel pile). The classic triad includes a linear mucosal tear exposing the internal sphincter fibers, hypertrophied anal papilla, and a sentinel skin tag (Fig. 24.14). Stool softener and local anesthetic agent are first line treatment in acute and chronic fissure. Endoscopic botulinum toxin injection into the internal anal sphincter or surgical internal anal sphincterectomy with or without fissurectomy may be effective in refractory cases.

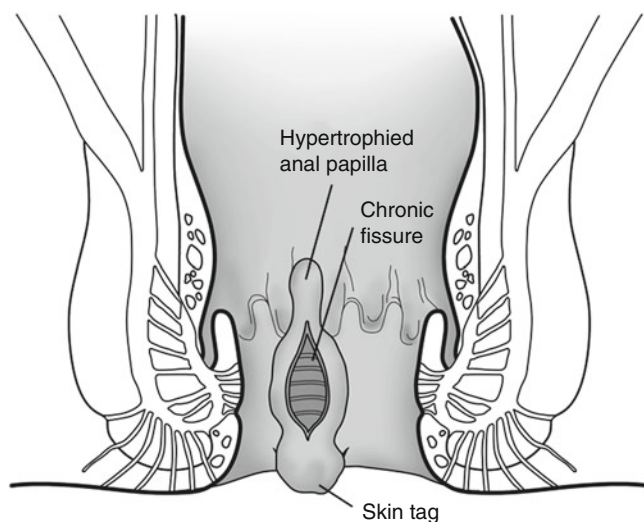


Fig. 24.14 The classic triad of chronic anal fissure. Although an acute fissure is simple tear in the anoderm, chronic fissure has rolled edges, a hypertrophied anal papilla proximally, a distal skin tag, and ulceration of anoderm exposed internal anal muscle

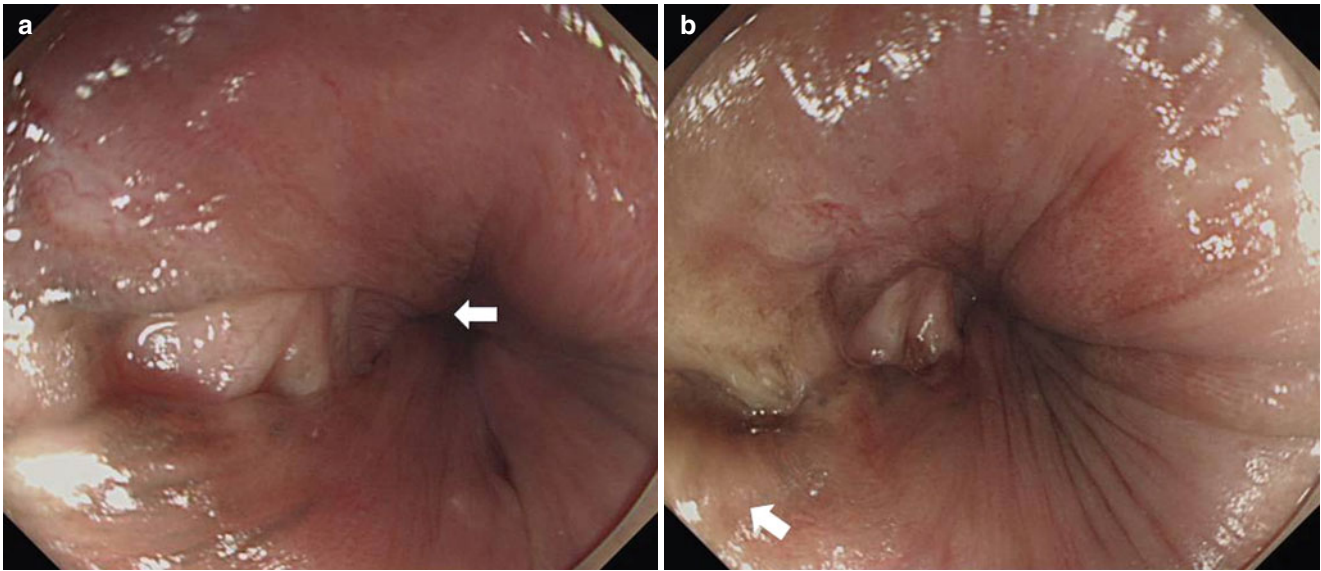


Fig. 24.15 Chronic anal fissure. (a) Tear of anoderm with exposure of the underlying internal sphincter muscle at the posterior midline portion of the anal canal (→). (b) A small skin tag can often be seen at the distal or caudal extent of the chronic fissure (→)

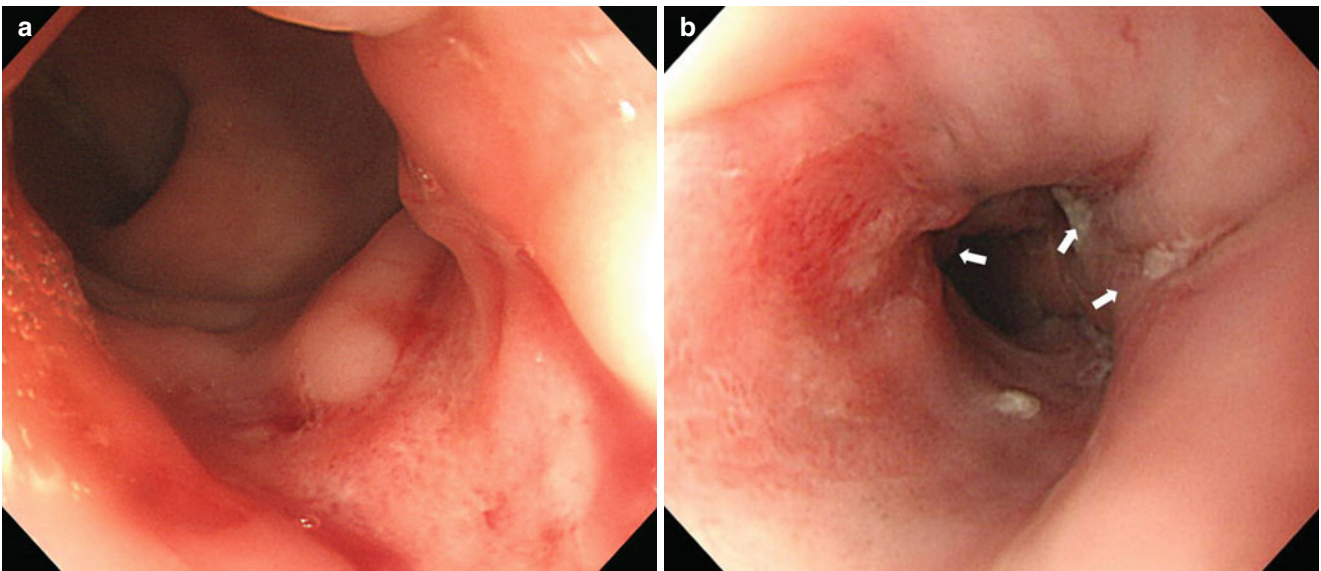


Fig. 24.16 Anal fissure associated with Crohn's disease. (a) Ulceration of anoderm at the lateral portion of the anal canal. (b) Multiple anal fissures associated with Crohn's disease

24.7 Hypertrophied Anal Papillae and Fibroepithelial Polyp

Hypertrophied anal papillae, called as fibrous polyp or anal tag, are skin tags that project up from the dentate line and arise from the base of the rectal columns of Morgagni. Although they are one of the most frequently found anal lesions during endoscopy, hypertrophied anal papillae are traditionally considered as a component of the classic triad of chronic anal fissure. However, they may be developed by chronic local inflammation and made at the end stage of thrombosed hemorrhoids. In clinical practice, hypertrophied anal papillae must be differentiated from polyps, hemorrhoids, or other tumors. They can be endoscopically differentiated from an adenomatous polyp by their white- or gray-colored smooth surface and their origin from the lower aspect of the dentate line in the anal canal (Fig. 24.17). They are usually asymptomatic and usually treatment is not required.

With passage of time, papillae continue to grow in size. The hypertrophied anal papilla is spherical or elongated with a greater diameter ranging from a few mm up to 4 cm. A papilla is liable to acquire considerable fibrous thickening over a period of time when it gets a rounded expanded tip, which is known as a fibroepithelial polyp. This is due to piling up and consolidation of chronic inflammatory tissues at the proximal part of the fissure at the dentate line. On the endoscopy, the fibroepithelial polyp is relative large and crumbly and coarse surface is often with pigmentation due to chronic inflammation (Fig. 24.18). The surface may show superficial ulceration. Therefore, biopsy is usually performed to assess pathologic evaluation. When biopsy forceps grasp hypertrophied anal papilla or fibroepithelial polyp, patients feel the pain sensation like pinching the skin, because of somatic sensory nerve innervations. Histologically, it consists of a fibrous stroma covered by squamous epithelium, which usually is slightly hyperplastic and maybe keratinized.

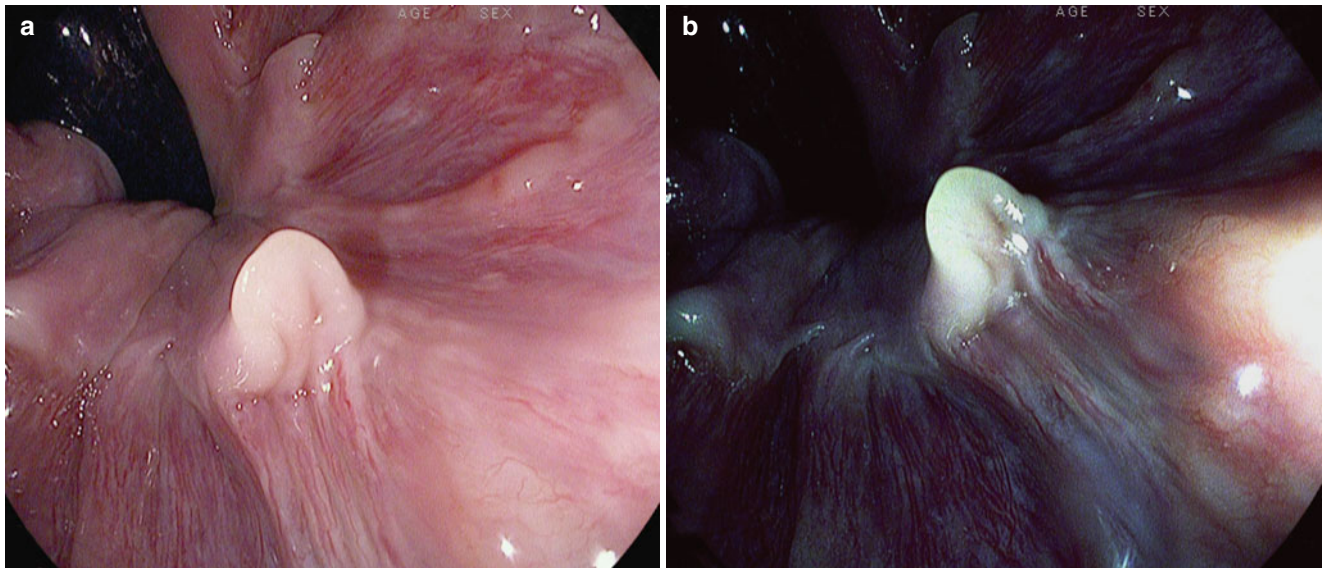


Fig. 24.17 Hypertrophied anal papillae. (a) Small-sized white-colored smooth surfaced hypertrophied anal papillae projected at the dentate line. (b) Image-enhancing image i-Scan. (c) Forward view of small hypertrophied anal papillae, which can be differentiated with polyp by

white-colored smooth surface. (d) 5 mm-sized hypertrophied anal papillae originating from dentate line. (e) Forward view of hypertrophied anal papillae on the anal canal. (f) Elongated hypertrophied anal papillae with internal hemorrhoid

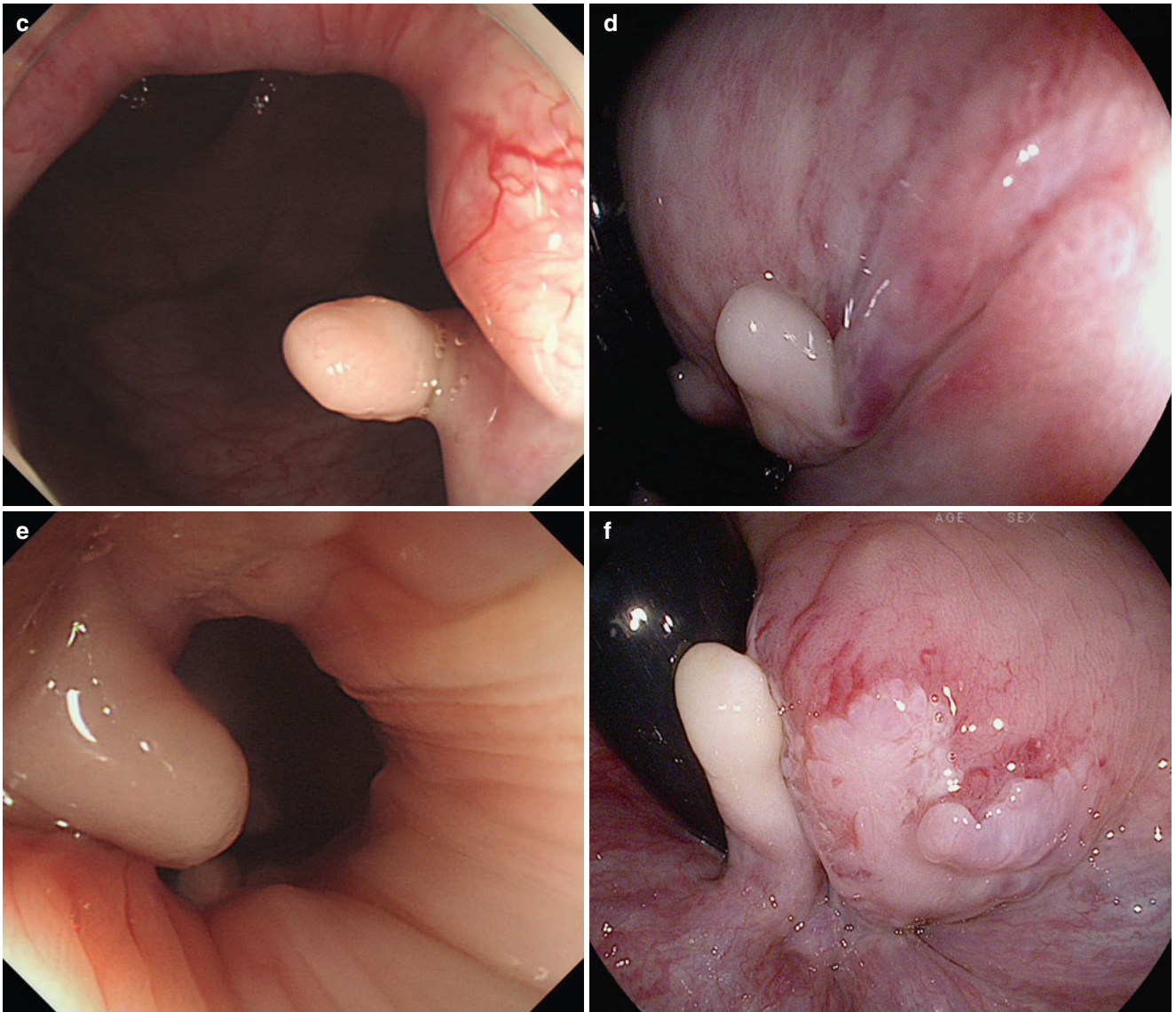


Fig. 24.17 (continued)

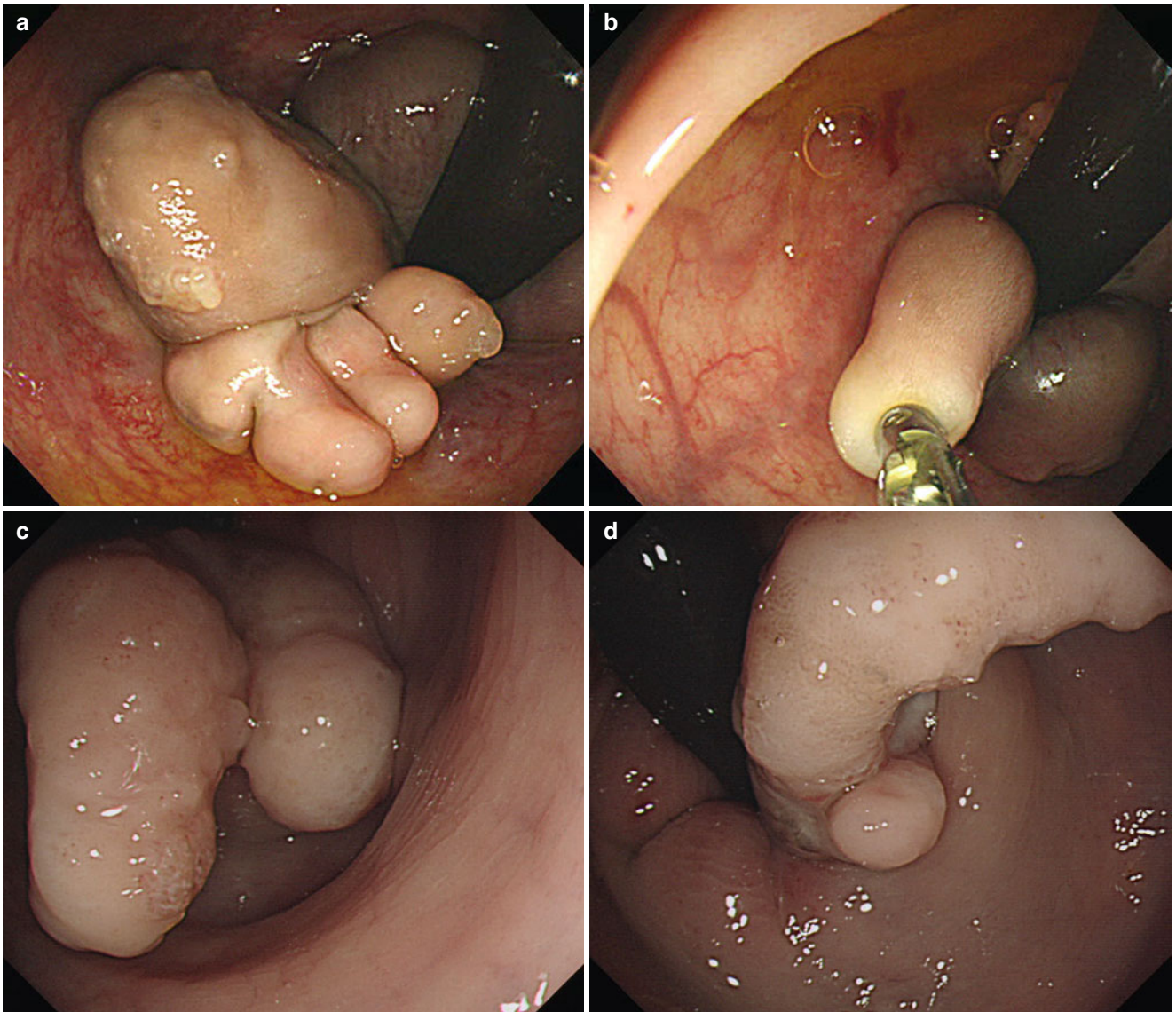


Fig. 24.18 Fibroepithelial polyp. (a) 4 cm- to 1 cm-sized multiple fibroepithelial polyps. (b) Patients feel pain, when biopsy forceps pinch fibroepithelial polyp. (c) Crumbly and coarse-surfaced fibroepithelial polyp. (d) Fibroepithelial polyp originating from dentate line

24.8 Perianal Abscess and Anal Fistula (Fistula-in-ano)

Most of anorectal suppurative diseases resulting from infection of the anal glands extend from the anal crypts. Acute infection can cause an abscess and lead to anal fistula. In addition, anal fistula is associated with Crohn's disease, hematologic malignancies, tuberculosis, actinomycosis, trauma, and anal surgery [4]. The anorectal abscess can be diagnosed based on typical symptoms, such as swelling, throbbing, and continuous pain, and signs, such as erythema,

swelling, and marked tenderness on the anorectum and perianal skin (Fig. 24.19a). Because antibiotics alone are not adequate, incision and drainage should be performed (Fig. 24.19b).

Anal fistula is a tunnel that connects an internal opening, usually at an anal crypt, with an external opening, usually on the perianal skin. A fistula is diagnosed by seeing blood, pus, and sometimes stool drain from its external opening. If the tract seals over, pus can accumulate within the tract and pain can develop. Pus sometimes may be drained from the opening when the fistula tract is palpated (Fig. 24.20).

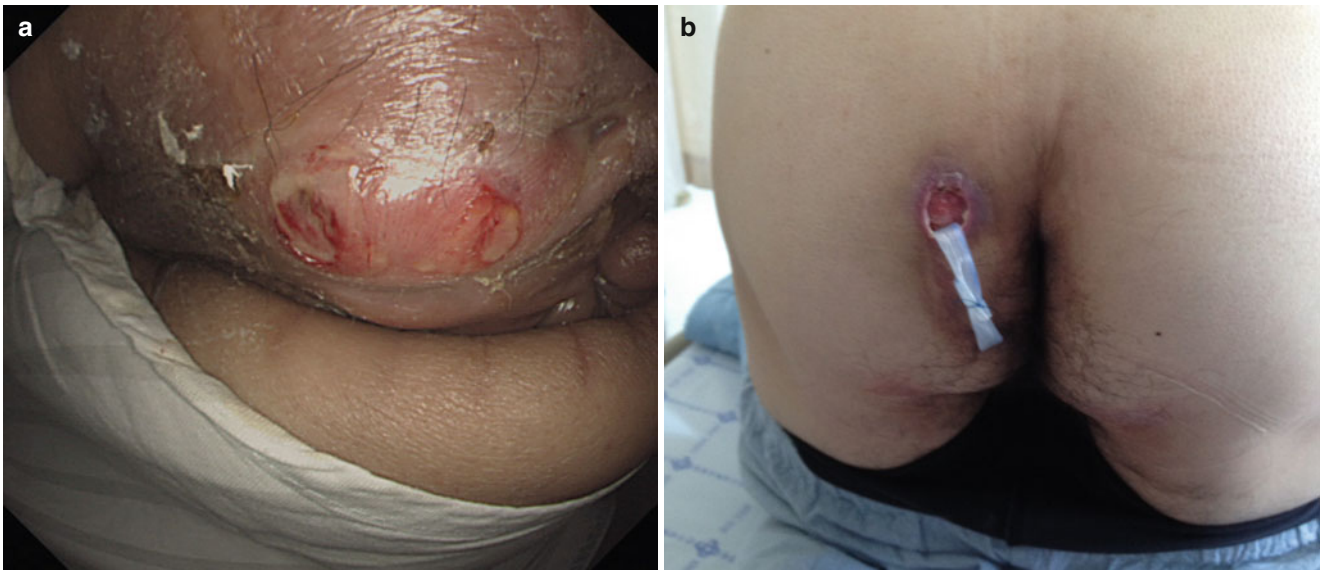


Fig. 24.19 Perianal abscess. (a) Erythema, swelling, and marked tenderness on buttock. (b) Abscess drainage

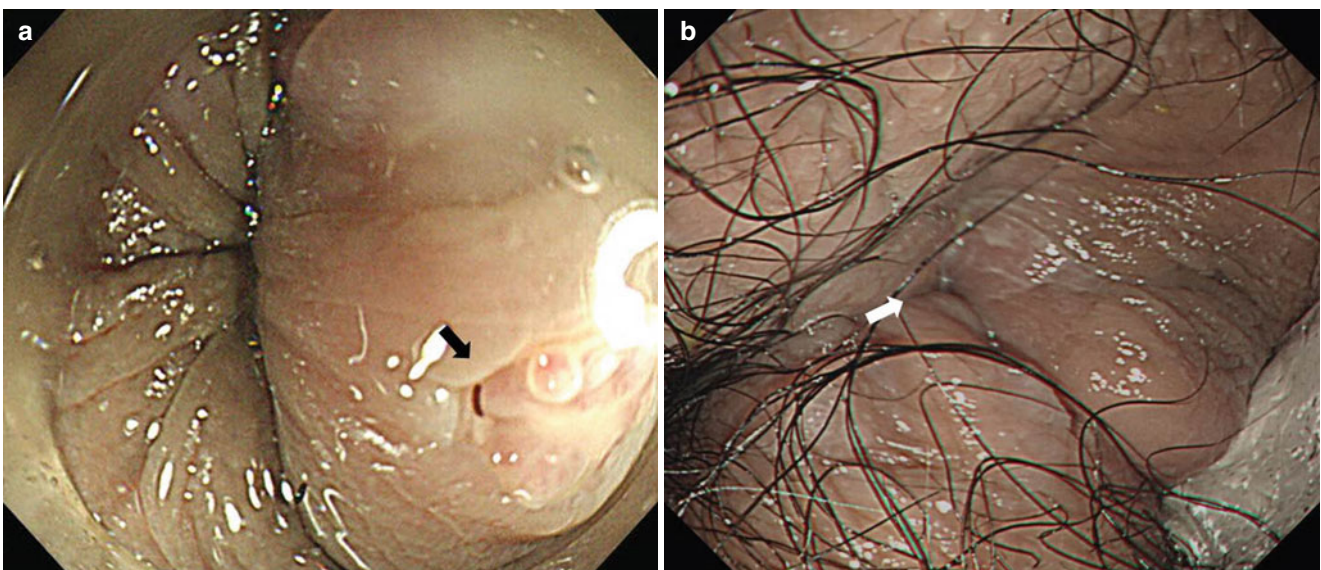


Fig. 24.20 Anal fistula. (a) External opening (→) of fistula at perianal skin. (b) Pus drainage from external opening (→) of anal fistula

24.9 Proctitis

Proctitis, the inflammation of the rectum, can be caused by several infectious and noninfectious etiologies (Table 24.3). The symptoms and endoscopic appearance of many of these diseases can mimic other conditions and may pose diagnostic difficulties.

Table 24.3 Causes of proctitis

Infectious proctitis	
Distal proctitis	Proctocolitis
Chlamydia trachomatis	Entameba histolytica
Neisseria gonorrhoeae	Campylobacter spp.
Syphilis (Treponema pallidum)	Salmonella spp.
Herpes simplex virus	Shigella spp.
	Clostridium difficile
	Cytomegalovirus
Chronic radiation proctopathy	
Ischemic proctitis	
Prolapse proctitis	
Solitary rectal ulcer syndrome	
Idiopathic hemorrhagic proctitis (ulcerative proctitis)	

24.9.1 Infectious Proctitis

Proctalgia (rectal pain), a change in bowel habits, and a mucoid or bloody anal discharges between bowel movement suggest infectious proctitis. Infectious proctitis can have a number of etiologies and the most infectious proctitis is accompanied with infectious colitis by organisms causing dysentery (Fig. 24.21). The localized distal proctitis in men who have intercourse with men or women with a history of anal sex is usually caused by sexually transmitted infections,

such as *Chlamydia trachomatis*, *Neisseria gonorrhoea*, syphilis, or Herpes simplex virus. Chlamydia is the most prevalent sexually transmitted organism and can cause proctitis, mild symptom of pruritus ani, and anal mucopurulent discharge compared to other sexually transmitted proctitis. Endoscopic appearance of Chlamydia proctitis is usually nonspecific erythema, diffuse friability, small ulceration, and numerous nodular lesions caused by lymphoid follicle hyperplasia. The diagnosis is made by detecting specific DNA sequences using PCR in the rectal biopsy specimen (Fig. 24.21e, f).

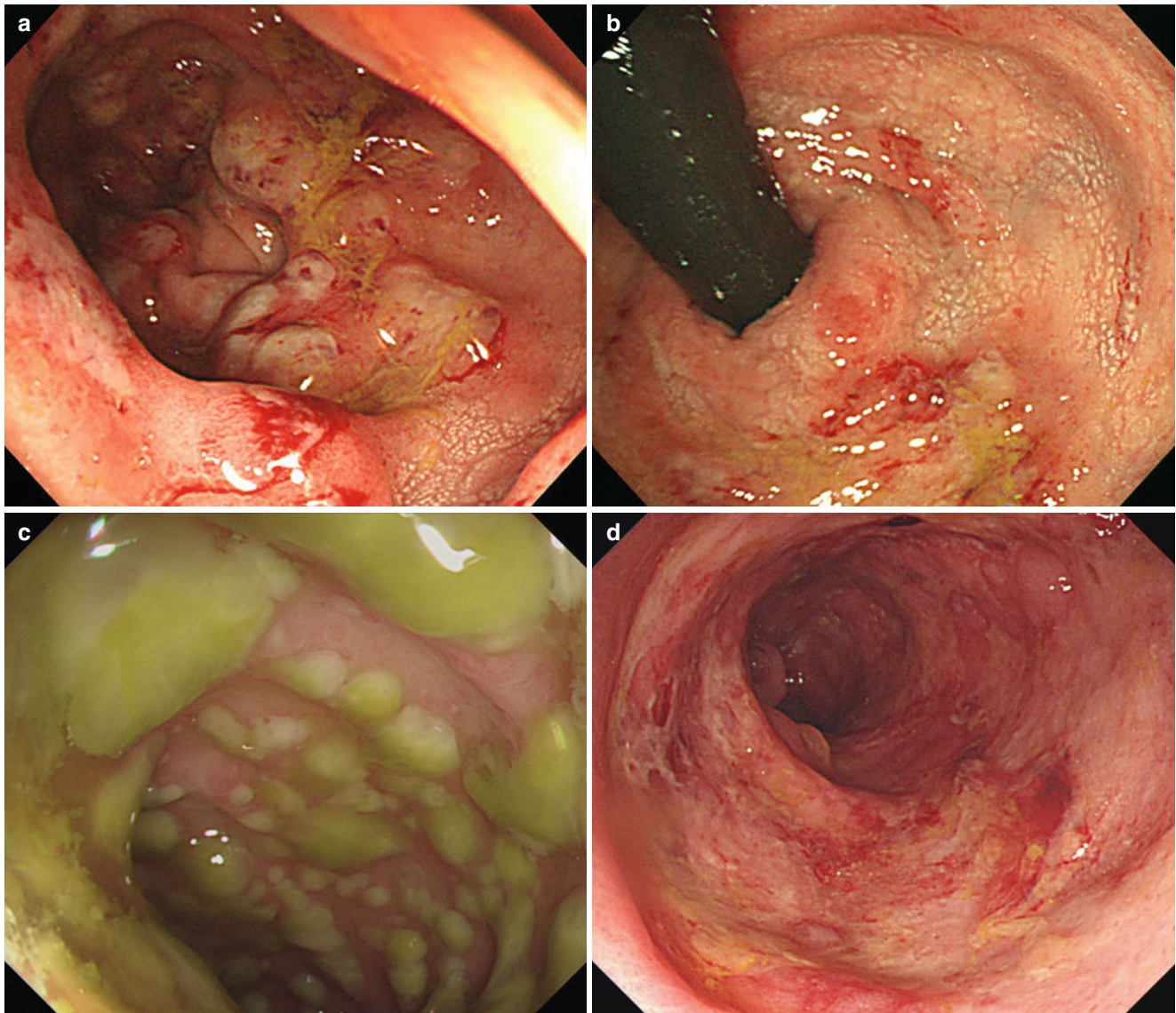


Fig. 24.21 Infectious proctitis. (a) Amebic proctitis. (b) Amebic proctitis. (c) Clostridium difficile-associated proctitis. (d) Ulcerative proctitis with CMV infection. (e) Chlamydia proctitis. (f) Chlamydia proctitis

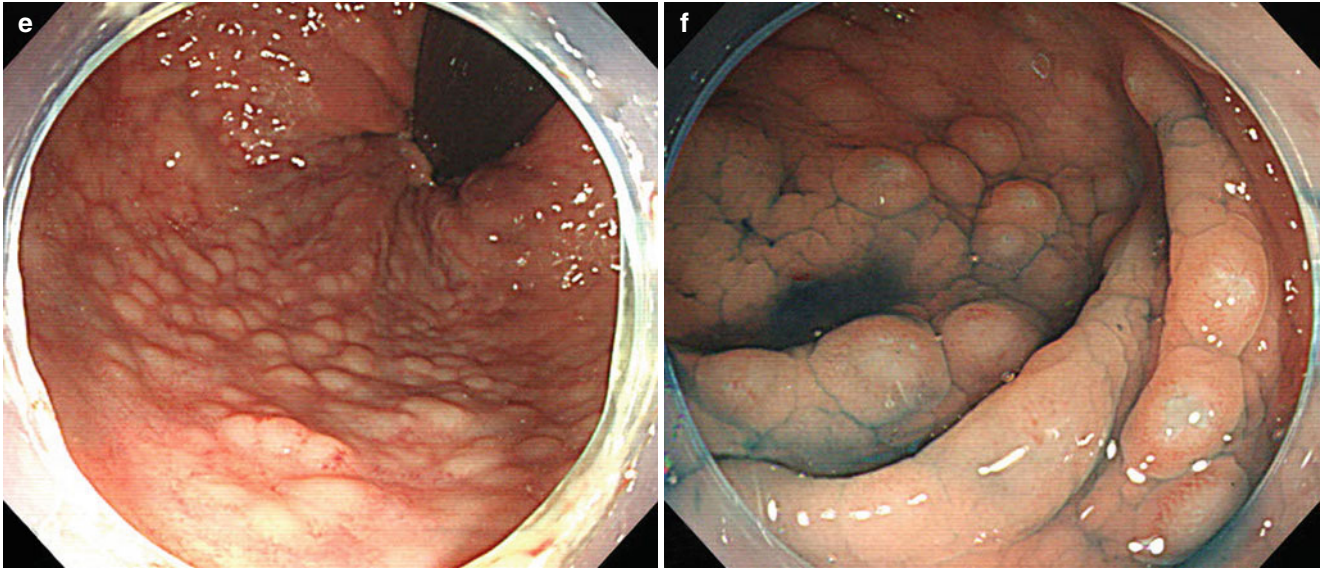


Fig. 24.21 (continued)

24.9.2 Chronic Radiation Proctopathy

The radiation proctitis is a misnomer because there is no inflammatory component found on the biopsy sample, but there is the presence of ischemic endarteritis of the submucosal arterioles and submucosal fibrosis. Chronic radiation proctopathy can occur from 9 months to 30 years after pelvic radiation injury, although patients typically present within 2 years after radiation. Endoscopic finding varies from diffuse, friable angioectatic lesions to frank ulceration

(Fig. 24.22). Rectal stenosis may be found. Bleeding is most problematic symptom and tenesmus, diarrhea, and defecatory urgency can be developed. Treatment of radiation proctopathy that is found incidentally on endoscopy is not usually indicated unless symptoms, such as hematochezia, tenesmus, diarrhea, and defecatory urgency, affect the patient's health status or quality of life. Complications of radiation proctopathy include anemia, rectal strictures, rectovesical fistula formation (Fig. 24.22), and increased risk of colorectal cancer.

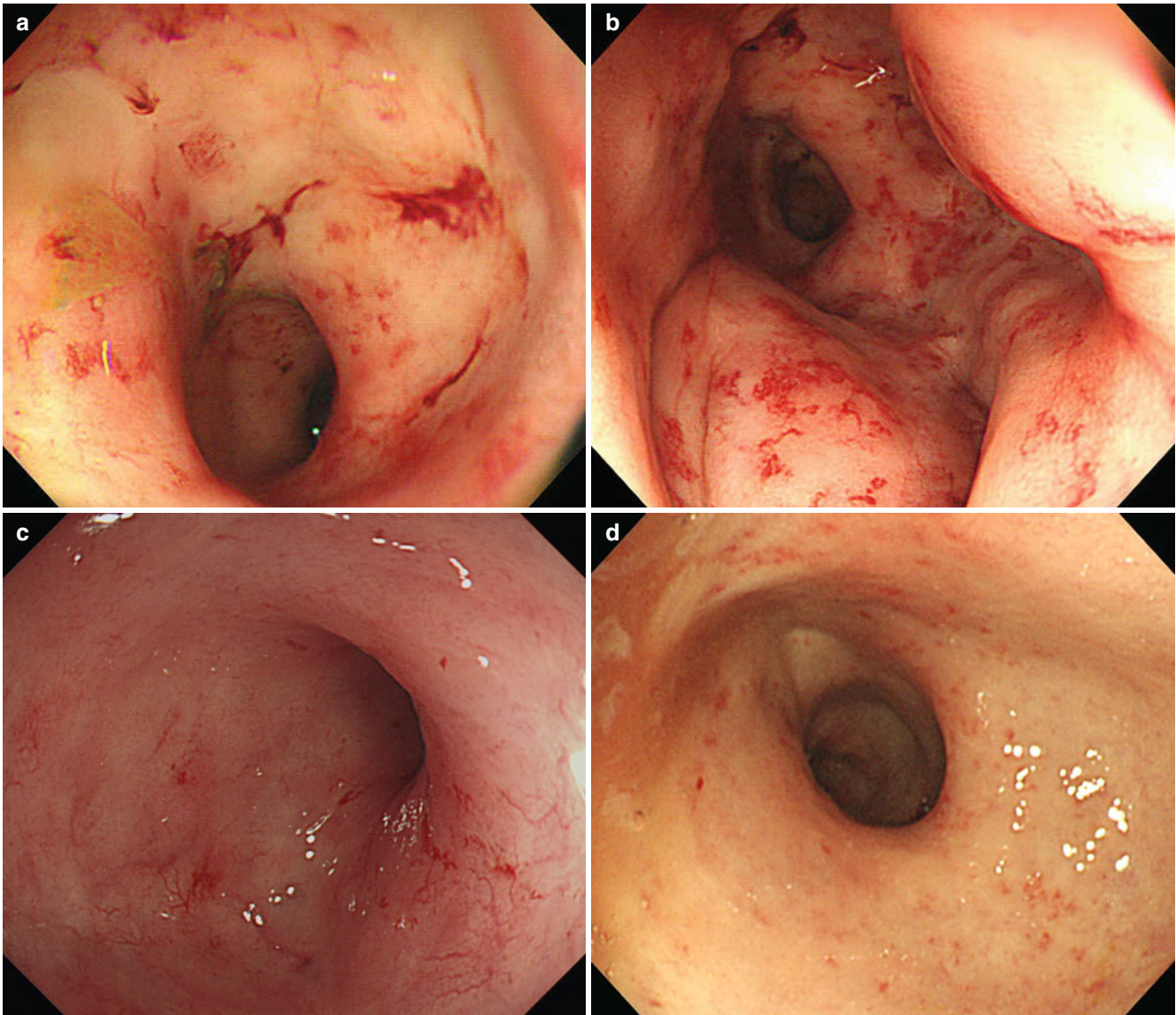


Fig. 24.22 Chronic radiation proctopathy. (a, b) Diffuse, friable angioectatic lesions were noticed on the rectum with luminal narrowing. (c) Cicatricial change with angioectasia on the rectum. (d) Edematous rectal mucosal change with angioectasia

24.9.3 Solitary Rectal Ulcer Syndrome

Solitary rectal ulcer syndrome is rare and presents with bleeding, passage of mucus, straining during defecation, and a sense of incomplete evacuation. The name of the syndrome is a misnomer, since patients can often present with lesions that are neither solitary nor ulcerated. The lesions are located in the anterior rectal wall within 10 cm of the anal verge in the majority of patients. Endoscopic findings vary and can include mucosal ulcerations, polypoid and mass lesions, or

simply erythema (Fig. 24.23). The histology of solitary rectal ulcer has a characteristic appearance that includes a thickened mucosal layer with distortion of the crypt architecture. The lamina propria is replaced with smooth muscle and collagen leading to hypertrophy. The pathogenesis of the solitary rectal ulcer is incompletely understood and often related to straining or abnormal defecation and rectal mucosal prolapse. Treatments for solitary rectal ulcer syndrome range from changing diet and fluid intake to surgery depends upon symptoms and whether rectal prolapse is present.

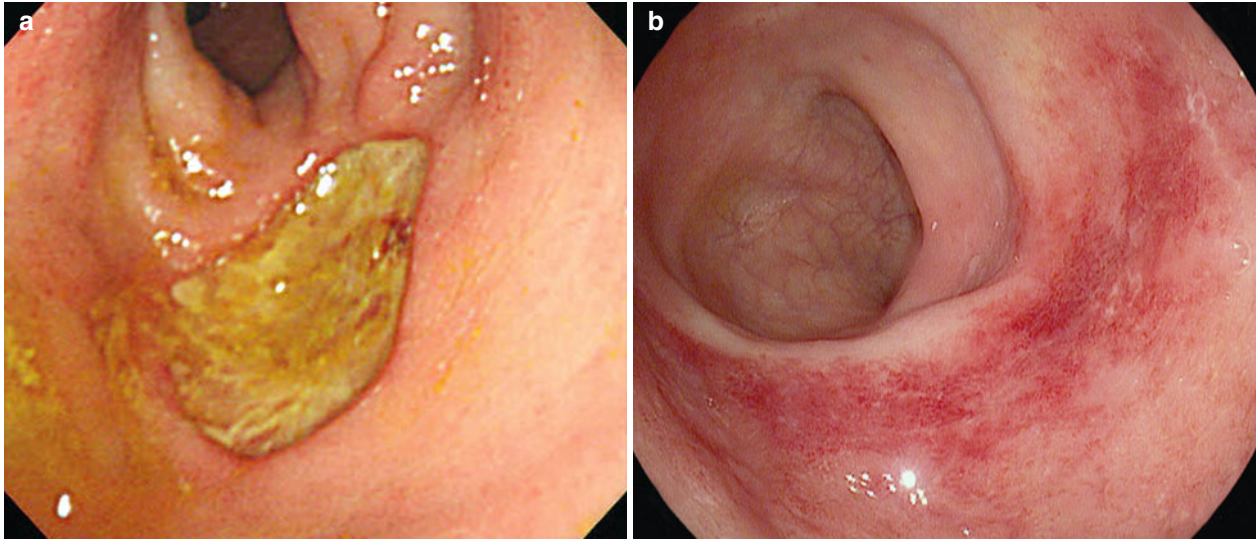


Fig. 24.23 Solitary rectal ulcer syndrome. (a) Well-demarcated solitary ulcer with flat base at the mid-rectum. (b) Erythematous mucosal changes only in the mid-rectum

Interesting Case

A 62-year-old female presented with a sensation of mass on anus. Inspection revealed a 2 cm-sized indurated nodule at the outside of anal verge (Fig. 24.24). After excisional biopsy, the patient was diagnosed with invasive squamous cell carcinoma of the anal canal. She was treated with concurrent chemoradiation and achieved complete remission.

Concurrent chemoradiation is standard therapy for squamous cell carcinoma of anal canal, whereas adenocarcinoma of the anal canal behaves like adenocarcinoma of distal rectum and is treated as such.

Lymphatic drainage of anal canal cancers depends on the location of the tumor in relation to the dentate line. Because cancers below the dentate line drain to the inguinal and femoral nodes, patients presenting with anal cancer should undergo physical and radiographic evaluation of inguinal nodes. Radiographic evaluation most commonly includes abdominal and pelvic CT scans, but there has been recent interest in the use of positron emission tomography (PET) scan, because recent studies have reported better sensitivity in identification of nodal disease.

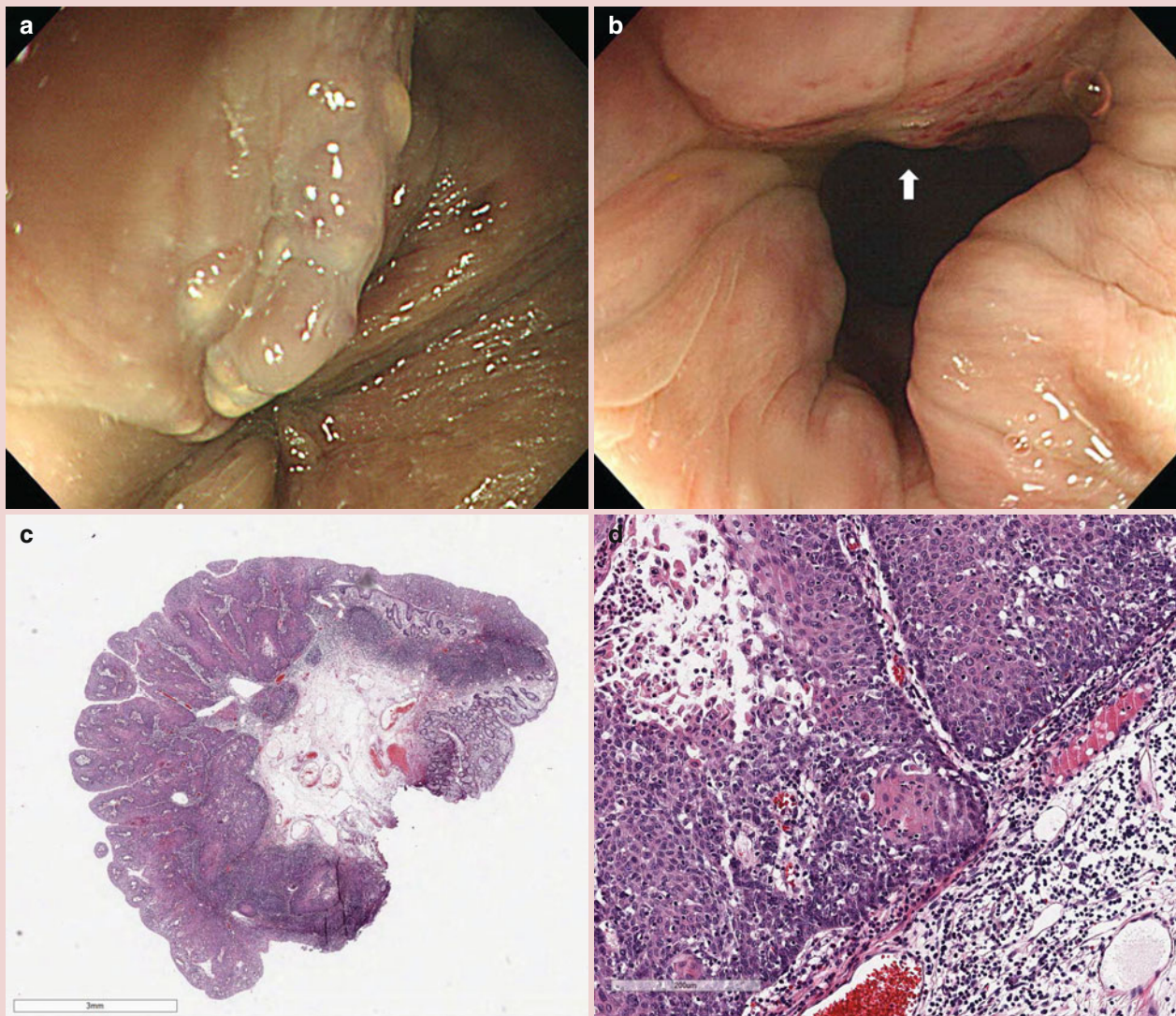


Fig. 24.24 Endoscopy and radiologic studies. (a, b) 2 cm-sized indurated firm nodule was noticed, which originated from anal canal (→). (c, d) Excisional biopsy revealed invasive squamous cell carcinoma. (e, f) Left inguinal lymph node metastasis was noticed on the CT scan and PET scan. (g) After concurrent chemoradiation,

previous indurated firm nodular lesion disappeared completely on endoscopy. (h) There was no significant hypermetabolism at the left inguinal lymph node and anus on follow-up PET scan. The patient was considered in complete remission state. Patient achieved complete remission with concurrent chemoradiation

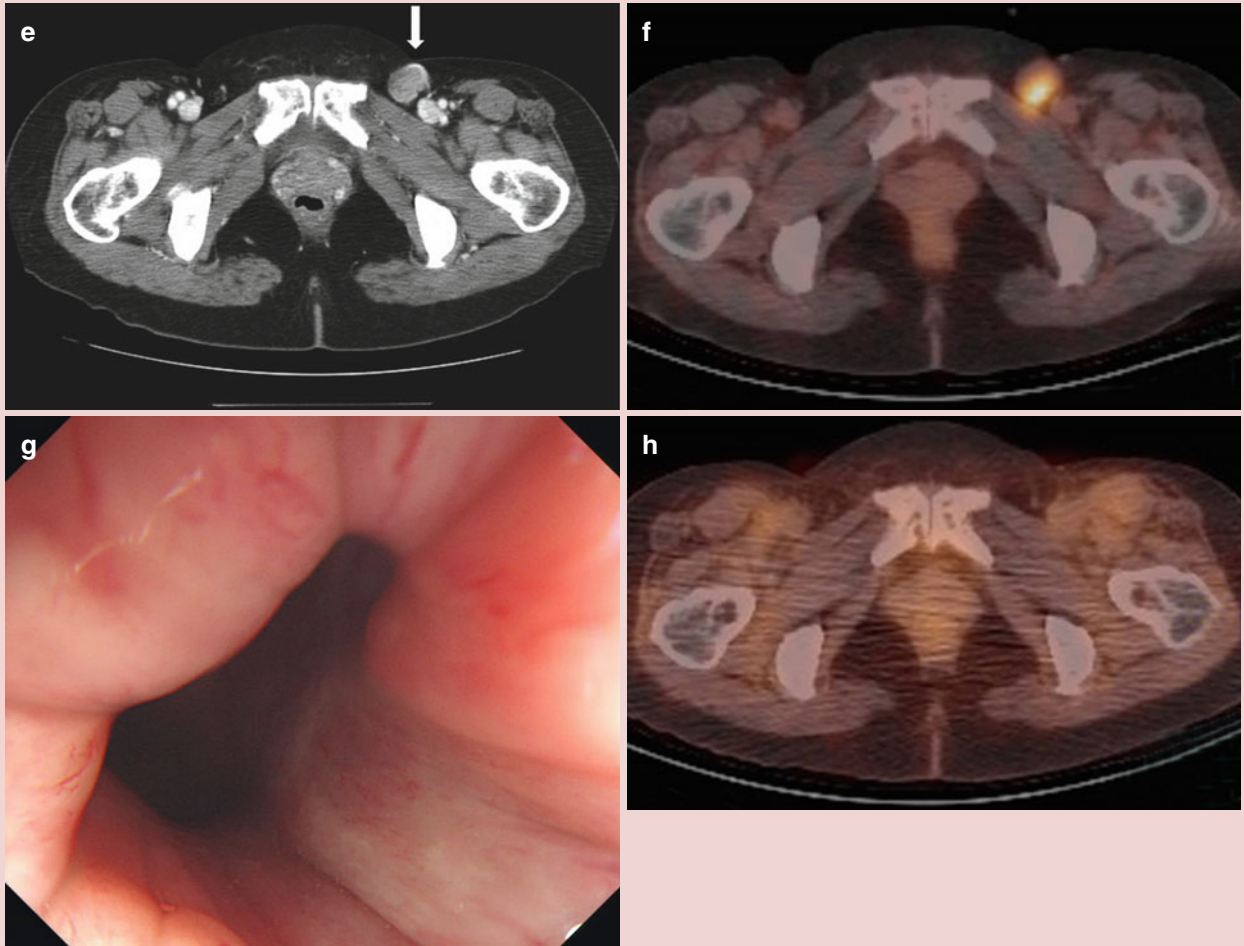


Fig. 24.24 (continued)

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