



Not Always Hemorrhoids: Diagnosis and Management Of Anorectal Complaints

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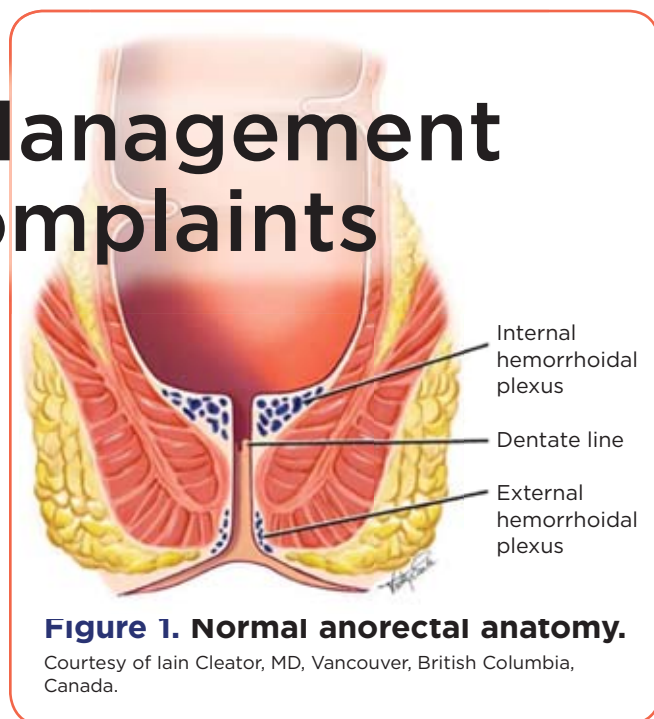


Figure 1. Normal anorectal anatomy.

Courtesy of Iain Cleator, MD, Vancouver, British Columbia, Canada.

Primary care physicians commonly refer patients with several important nonhemorrhoidal anorectal disorders to gastroenterologists. Although most of these conditions are not life-threatening, they are distressing to patients, lead to reduced quality of life, and may prevent caregivers from being able to maintain debilitated patients at home.^{1,2}

This article reviews the diagnosis and management of fecal incontinence, anal fissures, proctalgia syndromes, and pruritus ani. The information discussed herein is based on recent clinical guidelines from both the American College of Gastroenterology and the American Society of Colon and Rectal Surgeons.^{3,4}

Anatomy

The anal canal (Figure 1) consists of approximately 4 cm between the distal rectum and anal verge. The dentate line lies in the approximate midpoint of the

anal canal; this line represents the junction between the embryological endoderm and ectoderm, and is the point where the mucosa of the anal canal changes from the insensitive columnar epithelium of the rectum to the highly sensitive squamous epithelium of the anoderm. The internal anal sphincter is a thickened continuation of the circular smooth muscle of the rectum, and extends 1 to 1.5 cm below the dentate line. The external sphincter is composed of striated muscle, and at its cephalad portion is contiguous with the puborectalis muscle. It surrounds and extends below the internal anal sphincter. The internal anal sphincter is innervated by sympathetic and parasympathetic nerves that are inhibitory. The external anal sphincter is supplied by the pudendal and fourth sacral nerves.

Fecal Incontinence

Fecal incontinence is the chronic, involuntary passage of stool and/or flatus. It occurs with a prevalence, depending on the study, of as low as 2.2% to as high as 15% to 25%.^{5,6} The causes of fecal incontinence are related to abnormal anorectal structure, such as obstetric trauma, rectal prolapse, or a descending perineum; neurologic dysfunction including pudendal neuropathy, diabetes mellitus, dementia, stroke, multiple sclerosis, and spinal cord injury; and systemic states such as inflammatory bowel disease, systemic sclerosis, or diarrhea resulting from inflammatory bowel disease, post-cholecystectomy, or overflow.⁶⁻⁹ Important risk factors in women with incontinence not secondary to an underlying systemic illness are diarrhea and urgency.^{3,10}

Clinical assessment of patients with fecal incontinence centers on several important findings from history and physical examination. The Bristol Stool Form Scale and stool diaries may be used to accurately classify stool consistency and assess for concurrent, complicating diarrhea. After general questions concerning the amount, frequency, and type of incontinence are asked, special attention is directed to the presence of urgency, which may provide insight to the mechanism, and thus treatment, of incontinence. Urge incontinence is present when patients experience the urge to defecate but cannot maintain continence before voiding; in passive incontinence, defecation occurs without the patient's awareness. The significance of this distinction lies in implicating anal resting versus squeeze pressure—and thus internal versus external anal sphincter—as the dysfunctional continence mechanism.^{3,9}

The cornerstone of the physical assessment is the digital rectal exam, in which the clinician gauges anal tone, both at rest and during contraction. The digital rectal exam was demonstrated to be useful in detecting normal resting and squeeze pressure in patients with pelvic floor dyssynergia.¹¹ Based on clinical assessment, patients may undergo trials of conservative treatment if symptoms are not bothersome or further diagnostic testing if conservative measures fail or symptoms are severe.

Once the decision to proceed with further diagnostic studies is made, patients with chronic diarrhea or other risk factors for mucosal or systemic disease should be considered for colonoscopy. After systemic disease is eliminated as a cause of incontinence, the initial diagnostic test is anorectal manometry (ARM; Figure 2) with

balloon expulsion testing if a defecatory disorder is suspected.³ During ARM, the resting and squeeze pressures are assessed to identify those with abnormally weak pressures. Patients with weak pressures who fail conservative measures and are candidates for surgery should undergo further imaging of the pelvic floor or anal canal, either with magnetic resonance defecography or rectal endoscopic ultrasound.³

The treatment of fecal incontinence begins with counseling on dietary habits and their potential to exacerbate symptoms. Poorly absorbed carbohydrates and caffeine may contribute to urgency, and should be avoided. A food diary may prove useful to uncover trigger foods.³ Patients with irritation of the anoderm should be counseled to avoid aggressive wiping and astringents and to use a zinc oxide barrier cream. Evidence suggests that, especially in patients with liquid stools, antidiarrheal agents (loperamide, diphenoxylate-atropine, and codeine) can reduce episodes of incontinence and improve stool consistency.¹² Of the 3 medications, loperamide has the preferable side-effect profile.

In patients who fail supportive and medical care, further diagnostic studies, as outlined above, and additional therapy targeted to any abnormalities identified are warranted. In patients with weakness of the external anal sphincter, biofeedback therapy may be useful in improving continence and is a recommended approach, but robustly designed studies are lacking.^{3,13} In those with low pressures of the internal anal sphincter, submucosal injection of a bulking agent—non-animal-stabilized hyaluronic acid-dextranomer—can reduce the frequency of incontinence episodes.¹⁴

Anal sphincteroplasty is reserved for patients who have both failed conservative measures and have anatomic sphincter defects on endoscopic ultrasound or magnetic resonance defecography, although by 60 months the surgery has a 50% failure rate.³ Most patients with obstetric injuries typically experience short-term improvement with anal sphincteroplasty, but 10% to 14% have sustained results.⁴ Colostomy is an option for those with severe, refractory incontinence who fail the above measures.

Anal Fissures

Anal fissures are longitudinal tears of the anoderm distal to the dentate line. In 90% of cases they involve the posterior midline, but tears can occur in the anterior midline, usually in women.¹⁵ Of note, some anal fissures can occur secondary to other disease processes, particularly Crohn's disease, sarcoidosis, syphilis, tuberculosis, HIV, or anal carcinoma. Anal fissure can be acute or chronic, with the latter lasting more than 8 to 12 weeks.¹⁵

Anal fissures are characterized clinically by pain during or shortly after defecation that often is associated with bright red rectal bleeding. Patients may report a tearing sensation after passage of hard stool, although diarrhea, parturition, or receptive anal intercourse also may lead to anal fissures.



Figure 2. The mcompass anorectal manometer.

Photo courtesy of Medspira.

Acute fissures appear as simple, ovoid tears of the anoderm that may resemble an ulcer.¹⁵ The tear may expose muscular fibers of the anal sphincter that are evident in the base of the fissure. Chronic fissures often develop a sentinel skin pile that is palpable in the distal aspect, as well as proximal hypertrophied anal papillae, often visible during retroflexion during colonoscopy.³

Most anal fissures—greater than 44% in one study—will resolve without surgical intervention.¹⁶ Initial therapy consists of sitz baths, fiber, and topical analgesics.^{3,15,17} In patients who do not respond to such therapies, the next step in management is the addition of a topical vasodilator, either 0.2% nitroglycerin ointment or 2% nifedipine or diltiazem ointments.

Data for the efficacy of topical nitroglycerin are mixed, and a recent Cochrane review revealed that it is only marginally better than placebo.¹⁸ Regardless of its efficacy, headache remains a limiting factor in up to 20% to 30% of patients, and may lead to discontinuation of therapy.¹⁸⁻²⁰

A recent systematic review of topical diltiazem versus topical nitroglycerin found that diltiazem was associated with a lower incidence of side effects, headache, and recurrence, and was equally effective as nitroglycerin.²¹ A small, randomized trial found that oral and topical nifedipine are comparable in terms of pain relief and healing, with insignificantly more adverse events in those who administer orally.²²

Patients who fail vasodilator therapy may be considered candidates for botulinum toxin injection or surgery. Botulinum toxin is minimally invasive, but lacks consensus on dosage, location, and number of injections. It leads to the healing of 60% to 80% of fissures.²³ Side effects are minimal but may include incontinence of flatus and stool in a minority of patients. Recurrence is common, affecting up to 42% of patients who, in turn, may be retreated with success rates up to 63%.^{24,25}

Patients who fail repeated botulinum toxin injections should be referred to a surgeon for consideration of lateral internal sphincterotomy, which is superior to all other medical and minimally invasive strategies, albeit with a small risk for incontinence.^{18,26}

Proctalga Syndromes

Proctalga Fugax

Proctalga fugax is the sensation of rectal or anal canal pain that is typically severe and of short duration, lasting just seconds to minutes. The disorder likely is common, but most patients do not seek medical attention for the condition.²⁷ The exact etiology is unknown; however, abnormalities of pudendal nerve conduction and spasm of the smooth muscle of the anus have been implicated as causes.^{28,29} In evaluation, clinicians should exclude other diseases that lead to pain, such as anal fissures, thrombosed hemorrhoids, proctitis, solitary rectal ulcer, coccydynia, ulcers secondary to inflammatory bowel disease, and malignancy. Pelvic

inflammatory disease and prostatitis or prostate cancer also may cause rectal pain. It is thus important to exclude these pathologies with appropriate diagnostic studies, if clinical suspicion exists.³

Treatment of proctalga fugax is seldom needed or practically feasible given the short duration of symptoms. Reassurance should be provided that it is a benign condition. Treatment of patients with severe and debilitating symptoms is a matter of debate.³ A recent review details a stepwise approach to management, which first consists of warm baths and topical nitroglycerin followed by inhaled salbutamol, warm enemas, and oral clonidine. More invasive therapy involves injection of a local anesthetic and botulinum toxin.³⁰

Chronic Proctalga

Chronic proctalga, also known as levator ani syndrome, is defined by chronic, recurrent episodes of rectal pain or aching, each episode lasting more than 20 minutes; tenderness with retraction of the puborectalis muscle; and the exclusion of other etiologies of symptoms.^{3,31} The presumed pathophysiology is continuous contraction of the pelvic floor muscles. On exam, pain may be noted on digital rectal exam with levator tenderness.

Treatment of chronic proctalga is targeted to achieve relaxation of the striated pelvic floor muscles. Several strategies exist, including sitz baths, botulinum toxin, and levator ani massage. Biofeedback was associated with adequate pain relief in 87% of patients who had pain on palpation of the levator ani muscles. This response was durable for 12 months and greater than that seen with electrical stimulation and digital massage.³² Botulinum toxin does not appear to be of significant benefit in chronic proctalga.³³

Pruritus Ani

Pruritus ani is a dermatologic condition characterized by itching and/or burning in the perianal region. It occurs more commonly in men than women, and is estimated to affect 1% to 5% of the US population.³⁴ Pruritus ani is categorized as primary or secondary, with up to 75% of cases being secondary.³⁵ Etiologies of secondary pruritus ani are numerous and considered inflammatory, such as psoriasis, atopic dermatitis, seborrheic dermatitis, lichen planus, and lichen sclerosus; nonsexually transmitted infections, including *Candida*, *Enterobius*, *Streptococcus*, *Staphylococcus*, and herpes zoster; and premalignant or malignant, such as anal intraepithelial neoplasia and Paget's disease.³⁴ Primary pruritus ani is diagnosed once secondary causes have been excluded. It has been linked to fecal contamination.³⁶ The physical exam may show erythematous skin, lichenified skin, ulcerations, and excoriations.

Treatment of pruritus ani depends on the cause. *Candida* may be treated with topical or systemic antifungal agents, as well as treatment of hemorrhoids, fissures, spasms, and mucosal prolapse, if present, as these may contribute to fungal infection.³⁴ Bacterial infections

may be treated with topical or systemic antibiotics. Enterobiasis is diagnosed by cellophane tape preparation and responds to albendazole.³⁷ Primary pruritus ani is treated conservatively with attention to proper anal hygiene. Patients should be instructed to use only unscented toilet paper and to avoid excessive wiping, alcohol wipes, witch hazel, perfumes, and dyes. Controlling fecal seepage with bulk agents, antidiarrheal agents, and treatment of hemorrhoids and prolapse also should be employed. A zinc oxide barrier also can be applied.

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Conclusion

Anorectal complaints are common and often self-diagnosed incorrectly by patients as hemorrhoids. Fecal incontinence, anal fissures, proctalgia syndromes, and pruritus ani may be diagnosed by history and careful inspection of the perineum and a digital rectal exam. Additional testing with endoscopy, ARM, and imaging may be needed to confirm diagnoses and exclude inflammatory and malignant conditions. Many anorectal diseases can be concurrent, and addressing each is needed for maximum patient benefit.