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Anal Fissures: Diagnosis and Management for the Gastroenterologist

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Anal fissures, symptomatic hemorrhoids, and other anorectal disorders are highly prevalent in the United States. Patients with these conditions, and often their referring physicians, expect that the gastroenterologist will be able to provide a comprehensive evaluation and nonsurgical care of anal fissures.[1] In the author's experience, the diagnosis of an anal fissure is too often overlooked, resulting in unresolved complaints and frustration for both patients and clinicians.

Most gastroenterology fellowship training programs do not include the evaluation and treatment of many of these issues in their core curriculum; therefore, management of anal fissures too often is deferred to the surgeons.[2,3] Some program directors have called for a more formal inclusion of these topics to the core curriculum.[2] This article is an introduction to gastroenterologists interested in providing a more complete continuum of care to their patients.[4]

Perianal complaints, most commonly the result of symptomatic hemorrhoids, are common, with prevalence rates ranging from 4.4% to 40%.[5,6] Roughly 50% of the population will experience these problems by 50 years of age, and 75% during their lifetime.[7] Anal fissures occur in an estimated 20% of patients with symptomatic hemorrhoids.[8] Unfortunately, few data exist for the overall incidence of fissures because many patients never seek care for the

condition, whereas some acute fissures heal before a patient seeks medical attention.[9] In addition, clinicians fail to identify many cases of anal fissures, as these symptoms are assumed to result from hemorrhoids and other anorectal issues. Indeed, many instances of an unsatisfactory treatment of hemorrhoids result from undetected and untreated fissures.

Anatomy and Pathophysiology

The anal canal is approximately 4 cm long, running from the anal verge to a point at the proximal aspect of the levator–sphincter complex.[10] The junction between the modified squamous epithelium of the anoderm and the columnar epithelium of the more proximal mucosa—which sits 2 cm or so from the anal verge—is called the dentate line (Figure 1).

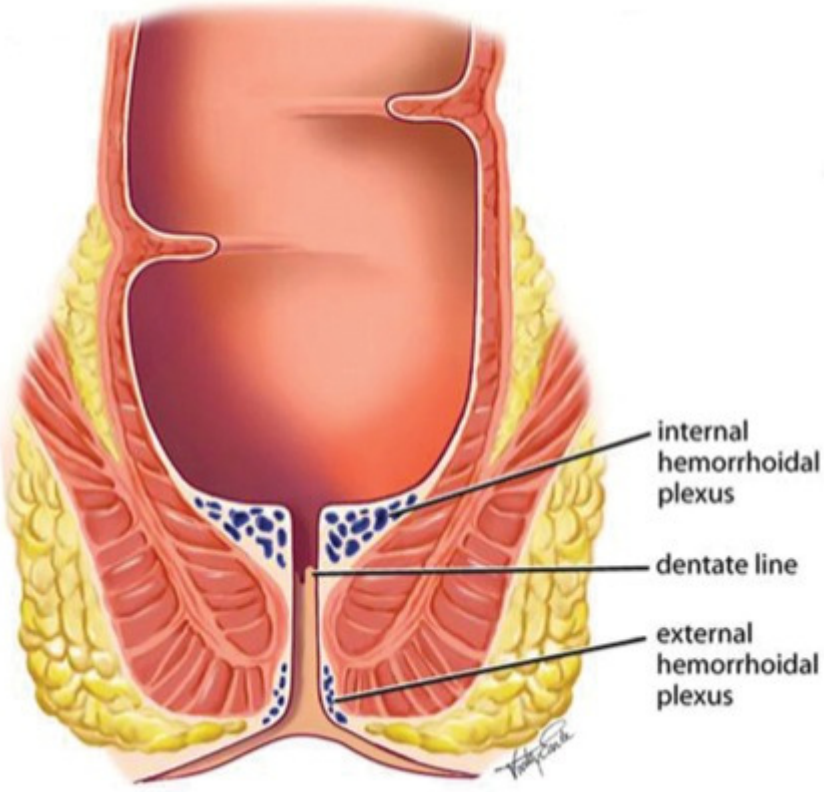

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Figure 1. Normal anorectal anatomy.
Courtesy Iain Cleator and CRH Medical.

The internal anal sphincter (IAS) is a thickened continuation of the

longitudinal smooth muscle layer of the rectum. Contracted at rest, the IAS assists with passive continence and relaxes during defecation, allowing for the passage of stool.[11] A hypertonic IAS has been associated with anal fissures and symptomatic hemorrhoid disease, although it is unclear whether the hypertonicity causes or results from these abnormalities.[11-14] This relationship may explain why so many patients have hemorrhoids and fissures concurrently.

Fissures have been described as a crack, tear, or ulceration of the squamous lining of the anal canal distal to the dentate line. They most commonly occur in the posterior midline (up to 90%) or in the anterior midline (up to 8% of men with fissures and up to 25% of women). Fissures not occurring in the midline should raise concern for other diseases, including Crohn's disease, tuberculosis, AIDS, syphilis, and carcinoma.[15]

Although the etiology of anal fissures is unclear, it is generally agreed that trauma to the anoderm during defecation plays a role in their development. Most acute fissures heal spontaneously, but angiographic and laser Doppler flowmetric studies may explain why many fissures have so much difficulty healing. These studies demonstrate that the posterior midline is poorly vascularized, anodermal blood flow is compromised in this area, and that the hypertonicity of the IAS that occurs in many of these patients further diminishes anodermal perfusion. It therefore seems reasonable to consider chronic, nonhealing fissures to be ischemic ulcers, and explains the rationale behind many of the therapeutic options discussed below.[16,17]

Acute fissures are superficial and often heal with conservative treatment. Those that do not spontaneously heal may deepen to expose the underlying IAS muscle, and chronic changes, including thickening and fibrosis of the area, a "sentinel tag," hypertrophic papilla, spasm, or even relative stenosis of the anal canal, may develop (Figure 2).[15]

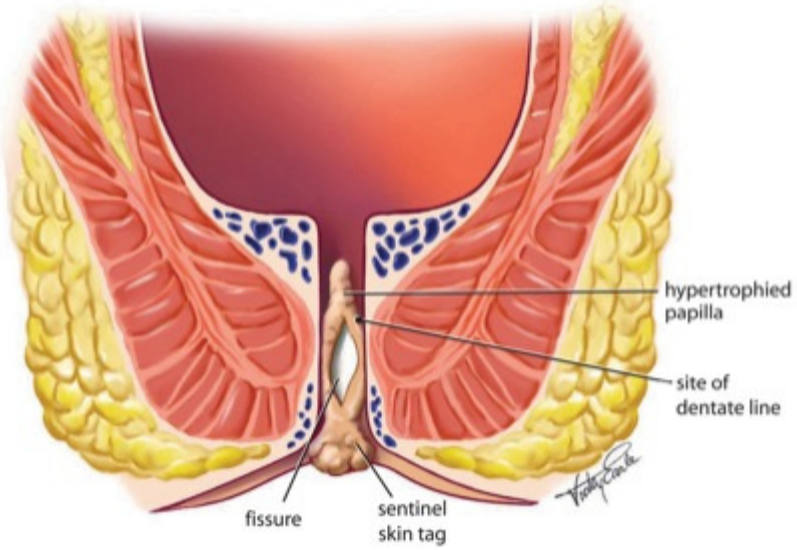

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Figure 2. Chronic anal fissure with associated findings.
Courtesy Iain Cleator and CRH Medical.

Diagnosis

The classic symptoms of pain during and especially after a bowel movement should immediately raise one's concern for an anal fissure.[18] Unfortunately, patients often relate these symptoms to their hemorrhoids, which can delay correct diagnosis. Of note, chronic fissures may have partially healed. In these patients, pain may not be present; indeed, many patients report that their hemorrhoids are flaring up when in fact a large or hard bowel movement worsens a partially healed fissure.

Internal hemorrhoids are covered by relatively insensitive mucosa, and so typically do not cause pain.[19] Visual inspection may demonstrate the open wound of the fissure, but as the wound typically resides within the anal canal (between the dentate line and the anal verge), digital examination of this area seems to be most reliable in making this diagnosis. Tenderness (whether minimal or severe) in the midline, particularly the posterior midline, along with palpable scarring, induration, or other mild changes may represent a partially healed fissure, even in the absence of a visible fissure. The findings on digital examination may be very subtle, but their identification and subsequent treatment may play a key role in trying to alleviate all of a patient's perianal symptoms.[1,2,20]

Anoscopy can be quite helpful, as many intraanal fissures may be seen, but flexible endoscopic evaluation tends to miss many anorectal issues, particularly those within the anal canal. Kelly et al prospectively demonstrated that anoscopy would identify 99% of anal lesions, where colonoscopy identified 78% of issues on straight withdrawal, and 54% of issues when used in retroflexion.[21] For these reasons, a combination of visual, digital anorectal, and (if the patient is not too tender) anoscopic examination seems to be the best way to evaluate these patients for the presence of a fissure. If any of these methods raises our suspicion of a fissure, then conservative treatment is warranted.

Treatment

Nonsurgical

Diet and behavioral modification: As many as 50% of patients with acute fissures will respond to the addition of dietary fiber along with sitz baths or the application of moist heat. Adults should consume 25 to 30 g of fiber daily, along with sufficient fluid intake to create a large bulky stool resulting in a physiologic dilatation.[22] The increased intake of fiber should continue indefinitely in order to minimize recurrence. The applied heat functions by relieving sphincter spasm, which is responsible for most of a patient's pain.[23,24]

Topical anesthetics: Topical anesthetics such as lidocaine may improve symptoms, but they do not promote healing[25] and should be used only in addition to other therapies.

Topical nitrates: Nitrates are metabolized by smooth muscle cells, releasing nitric oxide. Nitric oxide is the principal nonadrenergic, noncholinergic neurotransmitter in the IAS, and its release results in the relaxation of the sphincter muscle.[26] Relaxation of the IAS allows for increased blood flow to the anoderm, resulting in the healing of a significant percentage of chronic anal fissures.

An updated Cochrane review, which included 16 studies, found topical nitrates to be marginally better than placebo in the treatment of anal fissures.[15] The main drawback of topical

nitroglycerin is the troubling side effect of headaches, many of them severe, which occur in as many as 70% of patients who receive the medication. In a comparative study treating patients with 0.1%, 0.2%, and 0.4% compounds, headaches associated with the use of nitroglycerin appeared to be dose dependent, but the efficacy was not.[9] A small amount of a low concentration of nitroglycerin (0.125%) inserted into the anus 3 times daily for up to 12 weeks may maximize the clinical benefits of the medication without minimizing patient complaints.

Topical nitrates are contraindicated with the use of the erectile dysfunction medications sildenafil, vardenafil, and tadalafil because of the risk for developing symptomatic hypotension.[27-29] Studies suggest that this interaction no longer is a concern 24 hours or longer after a dose of sildenafil or 48 hours after a dose of tadalafil.[30-32]

Topical calcium channel blockers: In an effort to avoid the headaches associated with topical nitroglycerin, topical compounds containing calcium channel blockers can be used. These agents block the flow of calcium into the smooth muscle cells, also lowering pressure in the IAS.[33] Nifedipine and diltiazem are the 2 medications most frequently studied. Although these drugs appear to have a lower incidence of adverse effects, there are insufficient data to conclude that they are superior to placebo.[15] For this reason, these medications should be reserved for patients taking medication for erectile dysfunction, as the safety profile seems better than that for the use of organic nitrates.[34]

Botulinum toxin type A; chemical sphincterotomy: Botulinum toxin type A is neurotoxin that blocks the release of acetylcholine at the neuromuscular junction, with muscular paralysis resulting within a few hours. The effect typically lasts 3 to 4 months, until axonal regeneration occurs with the formation of new nerve terminals.[35] Injection of the medication into the IAS promotes relaxation more uniformly than topical agents, and can lead to the healing of a chronic fissure. The use of botulinum toxin for this indication is not FDA approved, and so some insurers will not reimburse for the treatment, forcing patients to incur greater cost for the treatment.[36]

Botulinum toxin therapy produces rates of healing superior to placebo. There is no consensus, however, regarding the dosage, sites of administration, number of injections, and other aspects of treatment.[15] Doses ranging from 5 to 100 units injected into the IAS, external sphincter, and intersphincteric groove as well as varying numbers of injections all have been reported to promote healing of an anal fissure.[37] The most common approach, involving 2 injections (on either side of the fissure) with a total of 20 to 30 units of botulinum toxin, has been shown to produce significant clinical response and low rates of postinjection incontinence.[38] Patients who fail to respond to topical treatment also appear to benefit from the therapy.[39]

Surgical Measures

When conservative approaches fail, surgery may be necessary for the definitive management of a chronic anal fissure. Dilatation of the anal canal was first described in the 1860s and popularized in the 1960s, but this procedure was later demonstrated to cause potentially significant damage to both internal and external sphincters that could result in permanent incontinence. Another surgical technique included excision of the fissure with a posterior midline sphincterotomy. This combination of procedures could cause a “keyhole deformity” of the introitus along with incontinence, so this proce- dure lost favor as well.[37]

Lateral internal sphincterotomy: Lateral internal sphincterotomy has become the surgical treatment of choice for patients resistant to medical management. A Cochrane review found lateral internal sphincterotomy to be superior to anal dilatation, posterior fissurectomy/sphincterotomy, botulinum toxin, and other therapies. The procedure can be performed open or closed with similar results.[15]

Lateral internal sphincterotomy is associated with healing rates ranging from 90% to 100% and recurrence rates of 1% to 3%, but is accompanied by postsurgical incontinence that reached 50% in some studies.[40] Fortunately, the majority of studies seem to indicate that the rate of incontinence is less than 6%.[22]

Johnson and Steele stated: “One must be alert for the patient who presents with a chronic anal fissure in the face of normal or diminished sphincter tone. This often does not require complex manometric evaluation, and can simply be determined in an office physical examination. … Certainly, a sphincterotomy is often not appropriate in this situation, and may lead to worsening continence.”[36] In such circumstances, an anal advancement flap may be considered. Perry’s review cited a few small series of these procedures,[41,42] noting that they “showed promise” for this type of patient but indicated that larger trials were still necessary.[15]

Conclusion

Anal fissures are a common health problem in the United States, and the symptoms they cause often are assumed to be secondary to hemorrhoids. Although hemorrhoidal disease occurs frequently, at least 20% of patients with hemorrhoids also have fissures, and clinicians must address both conditions to achieve a satisfactory clinical response. The physical examination, including a good anorectal evaluation, is key to making the diagnosis of a fissure. A series of nonsurgical therapies are available and will be effective for a majority of patients. Should these conservative measures fail, surgical referral is appropriate. Although many physicians refer fissures directly to surgery, the risk for postsurgical incontinence warrants an initial attempt to manage these problems medically.

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