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REVIEW

Update on the management of anal fissure

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Summary Anal fissure is an ulceration of the anoderm in the anal canal. Its pathogenesis is due to multiple factors: mechanical trauma, sphincter spasm, and ischemia. Treatment must address these causative factors. While American and British scientific societies have published recommendations, there is no formal treatment consensus in France. Medical treatment is non-specific, aimed at softening the stool and facilitating regular bowel movements; this results in healing of almost 50% of acute anal fissures. The risk of recurrent fissure remains high if the causative factors persist. If non-specific medical treatment fails, specific medical treatment can be offered to reversibly decrease hypertonic sphincter spasm. Surgery remains the most effective long-term treatment and should be offered for cases of chronic or complicated anal fissure but also for acute anal fissure with severe pain or for recurrent fissure despite optimal medical treatment. Surgical treatment is based on two principles that may be combined: decreasing sphincter tone and excision of the anal fissure. Lateral internal sphincterotomy (LIS) is the best-evaluated technique and remains the gold standard in English-speaking countries. Since LIS is associated with some risk of irreversible anal incontinence, its use is controversial in France where fissurectomy combined with anoplasty is preferred. Other techniques have been described to reduce the risk of incontinence (calibrated sphincterotomy, sphincteroplasty). The technique of forcible uncalibrated anal dilatation is no longer recommended.

Introduction

Anal fissure (AF) is an ulcer situated in the mucosa and the lower part of the anal canal from the anoderm up the dentate line. Clinical history and physical examination help to differentiate AF from hemorrhoidal disease, anoperineal Crohn's disease, various sexually-transmitted infections, and fissure due to underlying cancer. AF is the second most common reason for proctologic consultation after hemorrhoidal disease. It occurs more commonly in young adults with similar incidence in either sex. Its occurrence is less common beyond age 65 when other associated diseases must be considered [1,2]. It is particularly common after childbirth affecting 15% of women [3].

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Anal fissure is considered acute when it is of recent onset (less than six weeks), and chronic if it has been present for a longer period, taking on a characteristic aspect that includes perianal skin tag, fibrotic edges, and a proximal papilla.

A fissure may cause severe pain or be essentially asymptomatic depending on the degree of anal sphincter spasm. There is often associated low volume rectal bleeding. Secondary reflex constipation is very common due to the fear of pain associated with bowel movements. The fissure is located posteriorly in 85% of cases and anteriorly in 15% of cases.

When rectal bleeding is a presenting symptom, the presence of a fissure should not preclude full colonoscopy to rule out other colorectal lesions.

Pathophysiology

Anal fissure has multiple pathogenic factors, which form the basis for therapeutic approaches.

The initial event is triggered by the passage of hard and bulky stools (or the sudden evacuation of liquid stool) resulting in a tear the anoderm.

Two other factors account for the persistence of the fissure. Hypertonia or spasm of the internal sphincter can be primary or a secondary reflex to the pain caused by the raw ulceration, resulting in a vicious circle of repeated anal trauma due to reflex sphincter spasm caused by fear of defecation. This primary role of spasm has been demonstrated by anal manometry of patients with chronic anal fissure: the resting tone of the internal anal sphincter is high with little relaxation [4]. The second predisposing factor is local ischemia of the anoderm, which impairs the healing of the fissure. The anoderm is vascularized by branches of the inferior rectal artery. These arterioles reach the mucosa after piercing through the internal anal sphincter. Studies have shown that the area of the posterior commissure is less well vascularized and therefore at risk of ischemia [5,6]. Sphincter spasm promotes mucosal ischemia by reducing the blood flow through the arteries as they pass through the internal anal sphincter [7,8].

Post-partum anal fissure, which often involves the anterior anal commissure, is usually not associated with sphincter spasm, having a different mechanism related to constipation, hormonal levels and perineal dynamic changes that impair healing.

Non-specific medical treatment

The lack of prospective studies with extended follow-up of acute and chronic anal fissure has made it difficult to understand the natural history of anal fissure. The healing rate of chronic anal fissure with medical management ranges from 8 to 31% (estimated by analyzing the placebo arm in controlled trials) [9—13].

Medical management is the standard first-line of therapy due to its safety and simplicity of implementation. It aims to regularize bowel movements and improve patient comfort. Bowel regulation is enhanced by a high fiber diet and mild laxatives chosen according to the quality and intensity of constipation (stool softeners, osmotic laxatives, mineral oil). A study from the 1980s showed the effectiveness of high fiber diet in the treatment of acute anal fissure. It resulted in healing in 87% of cases within 3 weeks [14]. Continuation of a high fiber diet for one year (at least 5 g/day) prevented the recurrence of the fissure, with only 16% recurrence compared to 68% for patients taking placebo [15]. Two large retrospective series (876 and 393 patients) reported a healing rate of about 45% at 5 years [16].

Topical medications, consisting of local anesthetics, vitamins or anti-inflammatory agents, are often used but no study has proven their superiority over simple topical lubricants. The use of anesthetic-containing lubricants is considered no better than placebo [17]. Suppositories are often used to lubricate the anal canal and facilitate the passage of stool.

Analgesics are often required for patient comfort. NSAIDs, tylenol, and opioid analgesics are used.

Conservative medical treatment for 3 weeks results in healing in almost 50% of acute anal fissures. The risk of recurrence remains high if the underlying cause (such as anal sphincter spasm) persists or if stool softeners are prematurely discontinued; such treatment should be continued for long periods.

Specific medical treatment

Specific medical treatment is aimed at reversible reduction of anal sphincter tone (since sphincter spasm is responsible for persistence and/or appearance of anal fissure) thereby also promoting improved vascularization of the anoderm, which is essential to the healing process. A recent Cochrane Collaboration reviewed the medical management of acute and chronic anal fissure in adults and children; this included 75 randomized controlled trials in the analysis and 5031 patients [17].

Topical nitrates

Dilute nitroglycerin (NTG) ointment (0.05—0.4%) has proved its effectiveness in the control of sphincter spasm-related pain and the healing of chronic anal fissure compared to placebo (48.9% versus 35.5%) [17]. Two applications per day for 6 to 8 weeks should be prescribed [18]. The most common side effect of NTG is headache, which occurs in 20—30% of cases and may require discontinuation of treatment in one out of five cases. Headache is dose-dependent and can be prevented by oral analgesics and by gradually increasing the NTG dosage over four to five days [19,20].

The healing rate is similar to that obtained with injection of botulinum toxin or topical application of calcium channel blockers, but it is significantly less than after LIS or use of anal dilators at home. There is no significant difference between intra-anal application at the dentate line or at some distance therefrom [17]. The rate of long-term recurrence ranges from 51 to 67% [18,21—23]. The use of topical NTG has decreased the need for surgery in some practices [24], while others find that it only delayed eventual surgery [25].

While the Cochrane study did not note an influence of the NTG concentration (0.2 and 0.4%), one group reported higher healing rates (40.4% versus 54.1%) at higher concentrations (0.2% versus 0.4%) [19]. In France, only one form (Rectogesic®, a 0.4% preparation of NTG) is approved by the French marketing authority (Autorité de mise sur le marché [AMM]) and it is not reimbursable.