Anal fissure: a review

Patients often present to general and colorectal clinics with symptoms of anal fissures and can even present acutely to hospital. Conservative remedies have healing rates of up to 85% and have largely replaced surgery as first-line management. This review highlights the cardinal points in the aetiology, pathogenesis and treatment of anal fissures.

Anal fissures are longitudinal or elliptical tears in the distal anal canal, extending below the dentate line to the anal verge. The condition is the commonest cause of severe anal pain. The symptoms can be highly distressing, often out of proportion to the findings on physical examination (Kim and Wong, 1997).

Aetiology and pathogenesis

The majority of fissures encountered in clinical practice are idiopathic in origin. The lesion is usually encountered in younger and middle-aged adults but may occur at the extremes of age. Anal fissure is the most common cause of rectal bleeding in infants. Men and women are equally affected, but women are more likely to develop anterior fissures. Anterior fissures account for 10% of all fissures in women *vs* only 1% of fissures in men. The majority of fissures in both men and women are located in the posterior midline. Multiple or lateral fissures should arouse suspicion of another underlying pathology including Crohn's disease, ulcerative colitis, tuberculosis, human immunodeficiency virus infection or syphilis (Lund and Scholefield, 1996). The majority of fissures seen in patients with inflammatory bowel disease occur in the posterior midline and are painful in at least half of cases.

Anal fissures are less likely to develop in those individuals who eat plenty of raw fruit and vegetables, and wholegrain breads, and more likely to affect those who eat highly processed foodstuffs (Jensen, 1988).

The aetiology and pathogenesis of this condition is not completely understood. Generally, anal fissures have been associated with the passage of hard, constipated stool that, theoretically, traumatizes the anal canal leading to the onset of symptoms. The anal canal can also be traumatized by the passage of very loose stool (Metcalf, 2002). It is unclear why fissures are most commonly located in the posterior midline, and why some heal spontaneously while others become chronic. Studies have suggested that ischaemia may play a role in the aetiology of anal fissures (Carapeti and Philips, 2000).

There is a well-established association between anal fissure and increased anal resting pressure (Arabi et al, 1977; Hancock, 1977). Hypoperfusion caused by increased anal resting pressure is thought to be the critical element that prevents healing. Reduced transcutaneous oxygen tension has been found within the anus of patients with fissures (Hiltunen and Matikainen, 1986; Schouten et al, 1996). Oxygen tension correlates inversely with anal pressure and is increased after sphincterotomy. Also, fissures occur most commonly at sites of 'watershed' vascular supply: the posterior midline in men and both the anterior and posterior midline in women (Schouten et al, 1994).

Engel et al (2002) studied completely excised fissures. A defect in the anal mucosa was present in 14 of the 17 specimens analysed. The anal mucosa was undermined adjacent to the defect in 11 patients and a fibroepithelial polyp was present in six patients. The histological data were thought to be consistent with specific anodermal scar tissue, with or without an epidermal defect, in 16 patients.

The perpetuation of the fissure may be the result of an abnormality of internal sphincter function leading to chronicity. Several investigators have demonstrated high resting pressures in fissure patients. Gibbons and Read (1986) showed that anal sphincter patients demonstrated an initial relaxation followed by an abnormal 'overshoot' contraction. This overshoot phenomenon may explain the sphincter spasm and pain that result after anorectal stimulation during defecation. One hypothesis suggests that ischaemia is the underlying pathophysiology for fissure development. Gibbons and Read (1986) suggest that high resting pressures recorded in chronic anal fissure patients are unlikely to be the result of spasm, but probably represent an increase in basal sphincter tone. This implies that the elevated resting pressure is a primary event rather than a consequence of the fissure.

Klosterhalfen et al (1989) studied the topography of the inferior rectal artery and suggested a causal relationship with chronic primary anal fissure. Post-mortem angiography showed that in 85% of unselected autopsy cases, the posterior commissure was considered the end of the capillary system for the inferior rectal artery. Therefore the posterior commissure is less perfused than other sections of the anal canal, providing an anatomical basis for hypoperfusion in the posterior midline.

Schouten et al (1994) demonstrated that anodermal blood flow at the posterior midline is less than in the other anal canal segments, and that perfusion of the anoderm at the posterior commissure is strongly correlated to anal pressure. Gibbons and Read (1986) proposed that the higher incidence of anal fissures seen in young men might be explained by their higher maximum anal resting

Dr D Wray *is Senior House Officer in General Surgery,* **Miss S Ijaz** *is Specialist Registrar in General Surgery and* **Mr S Lidder** *is Senior House Officer in General Surgery in the Department of General and Colorectal Surgery, Lister Hospital, Stevenage SG1 4AB*

Correspondence to: Miss S Ijaz

pressure, which would induce a relative ischaemia of the anoderm secondary to decreased blood flow.

Clinical features

Symptoms are often out of proportion to the underlying pathology. In most patients, the symptoms are so characteristic as to be nearly diagnostic. The prominent symptom is severe anal pain, usually on defecation, which may last between a few minutes to a few hours afterwards with a tight, throbbing quality. It can be described as knifelike, cutting or tearing in character. This discomfort can often be ameliorated, at least temporarily, by sitz baths (Jensen, 1986). Rectal bleeding is seen in 75–100% of cases and is characteristically small in quantity, bright red and only occurs during straining or defecation. A minority of patients may also complain of mucous anal discharge and pruritus ani (Klosterhalfen et al, 1989). An obvious sentinel pile may be present. Constipation is a frequent complaint and is often described as the initiating event. Patients may also recall diarrhoea triggering their symptoms. Occasionally patients with a painful fissure may develop micturition problems, including dysuria, retention, urgency or frequency. Anal fissures may also lead to dyspareunia (Lund and Scholefield, 1996).

Diagnosis

The patient's history often strongly suggests the correct diagnosis, as does the presence of a sentinel pile. Retraction of the perianal skin usually allows one to visualize the fissure directly, even in patients with significant spasm. Visual examination may disclose a posterior oedematous tag and, on parting the buttocks, an associated fissure may be seen. Discomfort is often severe enough to prevent digital rectal examination. If conservative measures fail to heal the fissure then rigid sigmoidoscopy should be undertaken, under anaesthesia if necessary, to exclude specific causes of fissure, including inflammatory bowel disease (especially Crohn's disease), anal syphilis, anal herpes, anal carcinoma, lymphoma, anoreceptive intercourse (with or without human immunodeficiency virus infection) and, in children, sexual abuse. Contrast enema or colonoscopy is indicated on an individual basis, dictated by clinical features. An intersphincteric perianal abscess may mimic an acute anal fissure, presenting as severe anal pain with minimal physical findings and merits an examination under anaesthesia for diagnostic purposes. As discussed previously, the vast majority of fissures present posteriorly and a minority present anteriorly. A fissure located in the lateral position should alert the clinician to another cause. If infection, neoplasia or inflammatory bowel disease is suspected then appropriate biopsies, cultures, endoscopy or radiological studies should be carried out (Metcalf, 2002).

On examination, the triad of a chronic anal fissure includes a sentinel pile, an indurated ulcer and a hypertrophied anal papilla. The fissure edges become indurated and undermined, and the circular fibres of the internal sphincter are visible in the depths of the ulcer. A sentinel pile develops distally as does a hypertrophied anal papilla at the ulcer's apex. Once this stage is reached it is very unlikely that spontaneous healing will occur, and intervention is usually necessary. A chronic fissure may be associated with anal stenosis, especially as a result of previous anal surgery, for example following a haemorrhoidectomy (Kim and Wong, 1997).

Management

Conservative measures

Most acute fissures and up to 40% of chronic fissures will heal spontaneously aided by conservative measures to relieve pain (Carapeti and Philips, 2000). Constipation should be treated with bulk laxatives and dietary fibre supplements in order to avoid straining, which can cause persistent trauma and bleeding. Sitz baths relieve the spasm associated with a fissure. Dodi et al (1986) provided the first objective evidence for this recommendation with patients showing diminished resting anal canal pressures from baseline after immersion at 40°C but remaining unchanged after immersion at 5°C and 23°C. Jensen (1986) advocated warm sitz baths and bran as the treatment of choice for an acute fissure and showed no particular advantage to using proprietary medications.

Local application of anaesthetic cream such as 2% or 5% lignocaine may provide some symptomatic relief, but must be applied into the anal canal directly onto the fissure. However, data suggest this may not be an effective means of treatment (Bhardwaj and Parker, 2007). Topical steroids such as 1% hydrocortisone cream have been used to reduce inflammation, and it has been suggested that a significant proportion of acute anal fissures do heal after 3 weeks application of Proctosedyl (cinchocaine and hydrocortisone) ointment (Anonymous, 1970), although most acute fissures heal spontaneously regardless. The use of local anaesthetic agents can occasionally lead to skin sensitization, and topical steroids should be used sparingly and only for a limited period.

Pharmacological treatments

Nitric oxide has emerged as one of the most important neurotransmitters mediating internal anal sphincter relaxation. Neural stimulation of these nitric oxide-containing nerves releases nitric oxide, causing the internal anal sphincter to relax. Topical application of a nitric oxide donor, such as glyceryl trinitrate, can lower the sphincter pressure and heal anal fissures. Over two thirds of chronic anal fissures can be treated with an 8-week course of 0.2% glyceryl trinitrate ointment applied topically three times a day. Variable results have been reported in the literature. Some of this variability may be attributed to differing definitions of a chronic anal fissure. Healing rates of between 50% and 85% have been reported in the treatment of chronic fissures (Metcalf, 2002). Side effects of therapy tend to be mild and limited to headache, which has been reported in 20–40% of patients.

Similarly, 1% isosorbide dinitrate ointment applied five to six times a day results in healing of 80% of chronic anal fissures. However, early recurrence of symptomatic fissures may occur in over one third of patients after initial healing and a regimen requiring such frequent application may lead to poor compliance.

Many recurrent fissures can be successfully treated with a further course of topical nitrates and these agents are now considered a good first-line treatment for chronic anal fissures, avoiding the need for a surgical sphincterotomy in a significant proportion of patients.

Purified botulinum toxin, a potent biological agent that inhibits presynaptic release of acetylcholine from cholinergic nerve endings and causes temporary paresis of striated muscle, lowers resting anal sphincter pressure when injected into the smooth muscle of the internal sphincter. Jost and Schimrigk (1993) treated 26 patients with botulinum toxin with 81% of the fissures completely healed at 3 months follow-up. Limitations of this treatment are its cost, occasional perianal thrombosis and unknown long-term efficacy. It is gaining popularity and is now becoming an effective therapeutic option, again sparing patients from the hazards of sphincterotomy.

The calcium-channel blockers nifedipine and diltiazem also reduce resting anal pressure. Jonas et al (2002) reported on a group of patients with chronic anal fissure who had failed glyceryl trinitrate therapy. Topical 2% diltiazem gel was applied to the distal anal canal twice daily. Maximum anal resting pressure was significantly lowered during therapy, and 49% were healed after 8 weeks of treatment. Carapeti et al (1999) reported similar results.

Manual anal dilatation

Recamier in 1838 is widely cited as giving the first description of anal stretch. Its popularity has waxed and waned over the years and currently it is out of favour. Surgeons have been attracted to the procedure because of its simplicity and because it can easily be performed by relatively junior medical staff. However, the procedure is difficult to standardize and may impair anal continence permanently. Recurrence rates vary from 2–57%, but there may be flatus incontinence or soiling in as many as 40% of patients, and frank faecal incontinence has been reported in up to 16% of patients (Carapeti and Philips, 2000).

Internal anal sphincterotomy

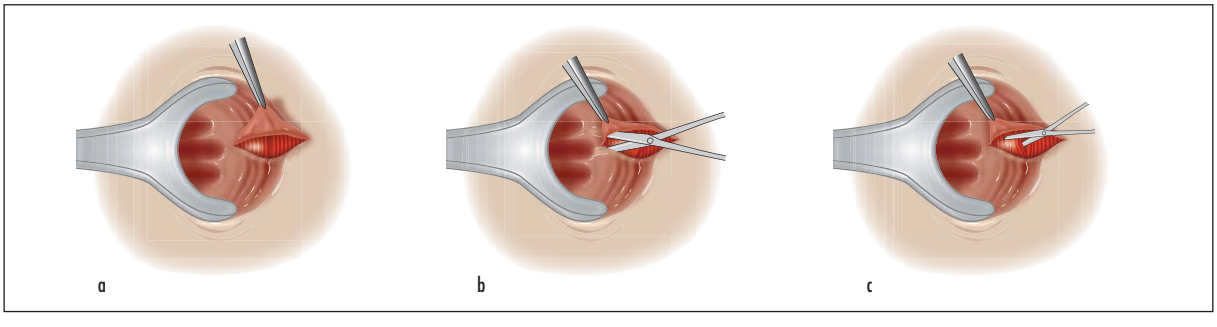
Failing conservative measures, lateral anal sphincterotomy is the procedure of choice. Incontinence rates postoperatively have been as high as 30% in the larger studies (Khubchandani and Read, 1989), and this has raised concerns over the safety of the operation. Department of Health statistics (Hospital Episode Statistics, 2007) indicate a 47% reduction in the numbers of procedures performed for anal fissures since 1999, largely attributable to a combination of these factors.

A closed technique was first described in 1969 by Notaras whereby the internal anal sphincter was divided under the skin in a manner similar to performing a tenotomy. The patient is examined under general anaesthetic in the lithotomy position. A Parks' anal retractor is then inserted and gently opened, stretching the internal sphincter (*Figure 1a*). The lower border of the internal sphincter is demonstrated by pushing blunt forceps into the intersphincteric groove in an upward direction. A narrow-bladed scalpel is then introduced through the perianal skin on the lateral side and pushed subcutaneously upwards. The internal sphincter is then divided to the required length by cutting outwards and the blade extracted, the sphincter defect can then be palpated (*Figure 1b* and *1c*). Haemostasis is achieved by the application of pressure and the wound is left open. Unless the surgeon is very skilled at this closed technique, it is often safer to divide the sphincter under direct vision by making a small lateral incision in the perianal skin. Surgeons traditionally incised the sphincter laterally rather than at the site of the fissure because it was thought that sphincter healing would be compromised in the relatively hypoperfused midline.

The results of lateral anal sphincterotomy have been reported from many centres (*Table 1*). Incontinence rates vary between surgeons depending on case selection and also on the perceived optimum length of sphincterotomy. The latter has always been a subject under dispute.

A prospective study of the extent of internal anal sphincter division at operation using endoanal ultrasound suggested that more of the internal anal sphincter was divided than intended. This is more of an issue in women who have a shorter anal canal. Common practice is to limit the sphincterotomy to a level equivalent to the proximal margin of the fissure. In 1999, Nelson's meta-

Figure 1. Technique of closed lateral anal sphincterotomy. a. Radial incision across the intersphincteric groove. b. Internal sphincter separated from the anoderm. c. Internal sphincter divided, wound closed or left open.



analysis of operative techniques for anal fissures found no difference between open and closed lateral sphincterotomy in terms of fissure persistence or incontinence rates.

Anal advancement flaps

Advancement flaps are used to cover the area left by the excised fissure with perianal skin to promote primary healing. Broad-based V-Y or rhomboid advancement flaps have been used successfully. This procedure is suitable for patients who have had surgery or trauma to the anal sphincter and those where further division of the internal sphincter would be likely to impair continence. In such cases, it is prudent to check preoperatively that the internal sphincter has been divided, with the help of anal ultrasound. If chronic fissures persist with normal or low resting anal sphincter pressure then advancement flaps are indicated. The operation is technically more demanding than sphincterotomy and may fail, sometimes leaving a large wound that can take a long time to heal. It should therefore be used with caution (Carapeti and Philips, 2000).

Conclusions

Anal fissures are a common and very distressing cause of anal pain. The mainstay of treatment is first-line therapy with ointments or botulinum toxin and if this fails then a careful and conservative approach to a lateral anal sphincterotomy can offer patients an excellent cure rate with minimal complications. BJHM

Table 1. Results of lateral anal sphincterotomy

Reference	No. of patients	Technique	Incontinence rate (%)	Recurrence rate (%)	Follow-up period
Notaras (1971)	82	Closed	Flatus 2.4 Soiling 7 Faeces 1	0	6 months
Milito et al (1983)	117	Open	Flatus 32.5 Soiling 3.4	0	1 year
Khubchandani and Read (1989)	657	Both	Flatus 36 Soiling 21 Faeces 4.9	2.3	–
Nyam and Pemberton (1999)	585	Open	Flatus 31 Soiling 39 Faeces 23	8	6 years

KEY POINTS

- Anal fissures may present with anal pain out of proportion to physical findings.
- Anal fissures are usually located in the posterior midline and multiple or lateral anal fissures should arouse suspicion of concomitant disease processes that may require further investigation.
- Hypoperfusion locally in the anal canal may lead to chronicity of anal fissures.
- A treatment plan would involve stool softeners, bulk laxatives, topical and oral analgesia and sitz baths; a full 6–8-week course of 0.2% glyceryl trinitrate or 2% diltiazem ointment applied 2–3 times a day or injection of botulinum toxin. If this fails, then lateral anal sphincterotomy (either closed or open) is indicated.

Conflict of interest: none.

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