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REVIEW

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Modern perspectives in the treatment of chronic anal fissures

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ABSTRACT

INTRODUCTION Anal fissures are commonly encountered in routine colorectal practice. Developments in the pharmacological understanding of the internal anal sphincter have resulted in more conservative approaches towards treatment. Simple measures are often effective for early fissures. Glyceryl trinitrate is well established as a first-line pharmacological therapy. The roles of diltiazem and botulinum, particularly as rescue therapy, are not well understood. Surgery has a defined role and should not be discounted completely.

METHODS Data were obtained from Medline publications citing 'anal fissure'. Manual cross-referencing of salient articles was conducted. We have sought to highlight various controversies in the management of anal fissures.

FINDINGS Acute fissures may heal spontaneously, although simple conservative measures are sufficient. Idiopathic chronic anal fissures need careful evaluation to decide what therapy is suitable. Pharmacological agents such as glyceryl trinitrate (GTN), diltiazem and botulinum toxin have been subjected to most scrutiny. Though practices in the UK vary, GTN or diltiazem would be suitable as first-line therapy with botulinum toxin used as rescue treatment. Sphincterotomy is indicated for unhealed fissures; fissurectomy has been revisited and advancement flaps have a role in patients in whom sphincter division is not suitable.

KEYWORDS

Anal fissures – Glyceryl trinitrate – Diltiazem – Botulinum toxin – Sphincterotomy

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An anal fissure is a split in the skin of the distal anal canal. Young adults of both sexes are affected equally. Patients present with anal pain commonly during defaecation and/or rectal bleeding. Whilst acute fissures heal spontaneously or with simple therapeutic measures, a proportion progress to form a chronic linear ulcer. Chronicity of a fissure relates to duration of greater than 6 weeks with fibres of the internal anal sphincter visible at the base of the fissure. Associated pathology may include a sentinel 'pile' distally and a fibro-epithelial polyp at the apex. Most anal fissures are idiopathic with no identifiable underlying disease process. There is no simple and unified theory to explain their genesis though constipation and lack of dietry fibre are implicated. Most fissures occur in the posterior midline; this may be anatomically related as there is a lack of tissue support posteriorly within the anal canal. Fissures associated with pregnancy are commonly located anteriorly and are often associated with low anal canal pressures. Other causes of fissures include Crohn's disease, syphilis, human immunodeficiency virus (HIV) or tuberculosis. These are secondary fissures and are most appropriately treated by addressing the underlying disease process.

Treatment of anal fissures

Conservative measures

The initial approach in the treatment of anal fissures is non-operative. An acute anal fissure may heal spontaneously or in response to medical therapy with warm baths, stool softeners, bulk laxatives, analgesics, topical anaesthetics and re-assurance.[1] Dietary bran supplements and warm sitz baths are superior to topically applied, local anaesthetic or hydrocortisone cream[2] and fibre ingestion results in fewer recurrences.[3] Regular anal dilatation to treat anal fissures is not recommended.[4] Other therapies such as cautery, suppositories and sitz baths have been disappointing with low healing rates and high long-term recurrences.[5]

Operative strategies

Most chronic anal fissures are associated with a raised internal anal sphincter (IAS) pressure and reduced vascular perfusion at the base. Current treatment has aimed at reducing resting anal pressure by diminishing sphincter tone and improving blood supply at the site of the fissure, thus promoting the healing rate.

ANAL DILATATION

Manual dilatation of the anus first described by Récamier in 1838 is a simple procedure and previously a popular treatment option. Its re-introduction by Goligher and Watts led to its popularisation.[6] However, incontinence was a concern and endo-anal ultrasonography provided an insight as to the degree of damage associated with this procedure; fragmentation is often seen.[7,8] In an attempt to minimise incontinence, some have advocated gentle digital dilatation of the anus under total neuromuscular blockade, with incontinence seen in 9 (3.8%) of 273 patients following this procedure in one study.[9] Despite these reports, incontinence is a concern and current opinion is that manual dilatation of the anus for the treatment of anal fissures is not recommended.

INTERNAL ANAL SPHINCTEROTOMY

Early advocates of sphincterotomy recommended a generous division of the IAS muscle and, in some cases, total division extending to the circular muscle of the rectum. By 1959, the 'standard internal sphincterotomy' comprised division of only half of the IAS to the dentate line in its lateral or posterolateral part.[10] Posterior sphincterotomy results in a 'keyhole deformity' that can cause mucous leakage in approximately a third of patients and should no longer be performed.[11]

Notaras[12] is credited for promoting the technique of lateral subcutaneous internal sphincterotomy. In this technique, the lower part of the internal sphincter is divided by introducing the knife blade at the anal verge between the anal canal mucosa and the IAS, then directing the cutting edge laterally towards the IAS. Hoffman and Goligher modified this technique by passing the blade between the internal and external sphincters and cutting medially. Both the subcutaneous and open techniques seem equally efficacious with regards to extent of division and effect on anal pressures.[13]

There are certain principles that should be noted:

1. The sphincterotomy should be away from the fissure site so that intact mucosal bridges fill the gap between divided muscle fibres to allow rapid healing.
2. The entire thickness of the lower internal sphincter must be divided, as any remaining intact fibres go into intense spasm to compensate for the divided fibres.
3. The mucosa over the sphincterotomy site should not be breached as this would predispose to infection.
4. The upper one-third of the sphincter must remain intact for continence.
5. The length of the sphincterotomy should be 'tailored' to the length of the anal fissure.

Sphincterotomy induces a sustained reduction of maximum resting anal pressure.[14] The largest review of the sequelae of internal sphincterotomy for chronic fissure *in ano* showed rates of flatus incontinence in 715 patients, occurring 'sometimes' to 'infrequently' in 255 (35.7%), faecal urgency in 35 (4.9%) and soiling in 152 (21.2%).[15] Incontinence may be minimised by a 'tailored sphincterotomy' where the sphincter is divided to the length of the fissure; this does not appear to compromise the healing rate.[16]

FISSURECTOMY/FISSURECTOMY AND SPHINCTEROTOMY

Fissurectomy has a role in midline fissures complicated by underlying fistula. Though further work by Bode *et al.*, Gingold and Di Castro *et al.* demonstrated fissurectomy as a viable treatment option, its use has remained sporadic. The recent use with pharmacological agents such as topical isosorbide dinitrate to treat fissures has led to its re-introduction.[17]

ANAL ADVANCEMENT FLAP

This is indicated for patients with primary or recurrent fissures and for women with a complicated obstetric history with low resting anal canal pressure. In order to aid selection of suitable cases, prior manometry and endosonography is employed. This operation avoids further disruption to the internal sphincter and avoids factors that might otherwise jeopardise continence. Skin flaps can either be triangular (Y-V), a square-shaped sliding graft or a C-anoplasty.[18]

Endo-anal ultrasound imaging of the anal sphincters

Anal dilatation can be regarded as an uncontrolled tearing procedure that damages the sphincter muscles with considerable detriment to anal function (Fig. 1). Lateral

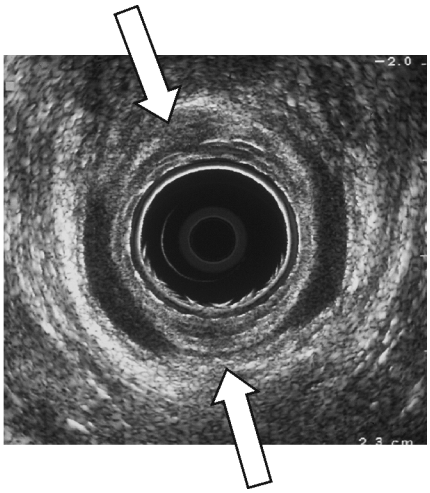

Figure 1 Endo-anal ultrasound demonstrating the appearance of the internal anal sphincter (IAS) after manual dilatation for chronic anal fissure. The white arrows indicate the defects in the IAS.

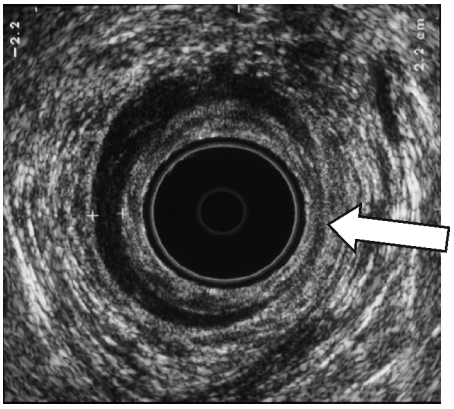

Figure 2 Endo-anal ultrasound demonstrating the appearance of the internal anal sphincter (IAS) after lateral sphincterotomy for chronic anal fissure. The white arrow indicates the defect in the IAS.

sphincterotomy results in a discrete defect of the IAS; in women, the extent of the division can be greater due the relatively shorter anal canal length (Fig. 2). Endo-anal ultrasonography has also identified inadvertent division of the EAS or inadequate division as reasons for failure of a fissure to heal.[19]

Chemical sphincterotomy

This term refers to pharmacological manipulation of anal sphincter tone as an alternative modality to surgery for the treatment of anal fissures. The optimal treatment for anal fissures is to induce a temporary reduction of anal canal pressure to promote healing of the fissure without permanently disrupting normal sphincter function. A reduction in anal sphincter tone is achievable by enhancing IAS relaxation through direct action on internal anal sphincter smooth muscle cells. These mechanisms serve to reduce intracellular Ca^{2+}, which reduces the tonic state of the muscle. These can occur through nitric oxide donation, direct intracellular Ca^{2+} depletion, muscarinic receptor stimulation, α-adrenergic inhibition or β-adrenergic stimulation.[20]

Nitric oxide donors

GLYCERYL TRINITRATE

Glyceryl trinitrate (GTN) and isosorbide dinitrate act as nitric oxide donors and probably aid healing through an increase in local blood flow secondary to a reduction in intra-anal pressure and perhaps also by vasodilatation of the vessels supplying the anal musculature. Early studies with GTN focused on optimal dose schedules, healing rates and side effects. These were validated by numerous trials. Whilst GTN was advocated as first-line treatment for chronic anal fissures with encouraging results, concerns about its effectiveness in clinical practice outside clinical trials emerged.[21–25] There was also evidence that the duration of topical GTN was limited[24] and that GTN was possibly ineffective altogether.[25] Furthermore, data from randomised, controlled trials have shown that GTN is not superior to lateral sphincterotomy.[26,27] It would seem that those fissures present for greater than 6 months and those with an associated sentinel pile are more likely to fail treatment.[28] Alternative modes such as nitroglycerin patches have shown promise but have not been established as common practice.[29]

ISOSORBIDE DINITRATE

Isosorbide dinitrate (ISDN) is an alternative nitric oxide donor that has been used successfully in the treatment of anal fissures. The problems encountered are similar to those with GTN but long-term effectiveness has been questioned.[30,31]

Calcium antagonists

Nifedipine is a dihydropyridine calcium-channel blocker (less correctly referred to as a 'calcium-antagonist'), which inhibits calcium ion entry through voltage-sensitive areas of vascular smooth muscle and myocardium. Topical and oral formulations of nifedipine have been evaluated but not used in routine clinical practice.[32,33]

Diltiazem, a non-dihydropyridine calcium-channel blocker, also effects vascular smooth muscle relaxation and dilatation. Topical 2% diltiazem reduces maximum resting pressure (MRP) by approximately 28% and this effect lasts 3–5 h after application.[34] Early studies by Carapeti *et al.*[35] and Knight *et al.*[36] reported healing rates of chronic anal fissures of 67% and 73%, respectively. Side effects are minimal with diltiazem and include peri-anal dermatitis. Oral diltiazem has been assessed as part of a randomised trial and shown to heal anal fissures; however, significant side effects were noted.[37] The topical formulation of diltiazem has been subjected to rigorous scrutiny and is a valid alternative to GTN with similar reductions in MRP, improved healing rates and lower rates of recurrence.[38–41] There are also data to suggest that topical diltiazem heals GTN-resistant fissures.[42]

Muscarinic agonists and sympathetic neuromodulators

Carapeti *et al.*,[35] using 1% bethanecol gel in 10 volunteers, showed a dose-dependent reduction in the maximum resting anal canal pressure. Its use in a small trial showed that after 8 weeks, in 9 of 15 (60%), the fissure had healed with no side effects. After an initial encouraging study with oral indoramin as an α_1-adrenoceptor antagonist which reduced maximum resting anal canal pressure,[43] Pitt *et al.*[44] proceeded to conduct a double-blind, randomised, placebo-controlled trial of oral indoramin to treat 23 patients with

Table 1 **Effect of injection of botulinum toxin type A injected into the external anal sphincter in patients with anal fissures**

Authors	Dose (Units)	Patients (*n*)	Review (weeks)	Healing rate (%)	Maximum reduction in resting pressure (%)	Maximum reduction in voluntary pressure (%)
Jost & Schimrigk (1993)	2.5	1	12	100	No data	No data
Jost & Schimrigk (1994)	2.5	12	12	83	No data	No data
Jost & Schimrigk (1995)	5	26	12	81	No data	No data
Jost (1997)	2.5–5	100	12	82	No data	No data
Jost & Schrank (1999)*	20 40	50	12	76 (20 U) 80 (40 U)	No data	No data
Jost & Schrank (1999)	5 10	20 30	12	70 (5 U) 63 (10 U)	No data	No data
Jost (2001)	200	10	4	70	No data	No data
Thompson *et al.* (2002)#	50	26	12 (not complete)	81	No data	No data

*Dysport used for injection.
#Botulinum toxin type B used for injection.

Table 2 **Effect of injection of botulinum toxin type A injected into the internal anal sphincter in patients with anal fissures**

Authors	Dose (Units)	Patients (*n*)	Review (weeks)	Healing rate (%)	Maximum reduction in resting pressure (%)	Maximum reduction in voluntary pressure (%)
Giu *et al.* (1994)	15	10	8	70	25	21
Mason *et al.* (1996)	0.125–1 ng	5	12	60	16	No data
Espi *et al.* (1998)	10–15	36	24	65 (10 U) 81 (15 U)	No data	No data
Maria *et al.* (1998)	20	15	4 & 8	53 (4 weeks) 73 (8 weeks)	28	44
Maria *et al.* (1998)	15–20	57	8	44 (15 U) 67 (20 U)	29 (15 U) 28 (20 U)	17 (15 U) 13 (20 U)
Brisinda *et al.* (1999)	20	25	8	96	29	6
Minguez *et al.* (1999)	10–21	69	24	83 (10 U) 78 (15 U) 90 (21 U)	5 (10 U) 13 (15 U) 16 (21 U)	17 (10 U) 17 (15 U) 35 (15 U)
Fernandez *et al.* (1999)	80	76	12	67	No data	No data
Maria *et al.* (2000)	20	50	8	74	32	5
Lysy *et al.* (2001)	20 ± ISDN	30	12	73 (ISDN) 66	24 (ISDN) 21	4.8 (ISDN) 4.4
Madalinski *et al.* (2001)	50	13	No data	54	No data	No data
Gecim (2001)	5	27	6	80	No data	No data
Brisinda *et al.* (2002)	20–50	150	8	89 (20 ± 30 U) 96 (30 ± 50 U)	30 (20 ± 30 U) 34 (30 ± 50 U)	0 (20 ± 30 U) 8 (30 ± 50 U)
Katory *et al.* (2002)	50	20	6	80	No data	No data

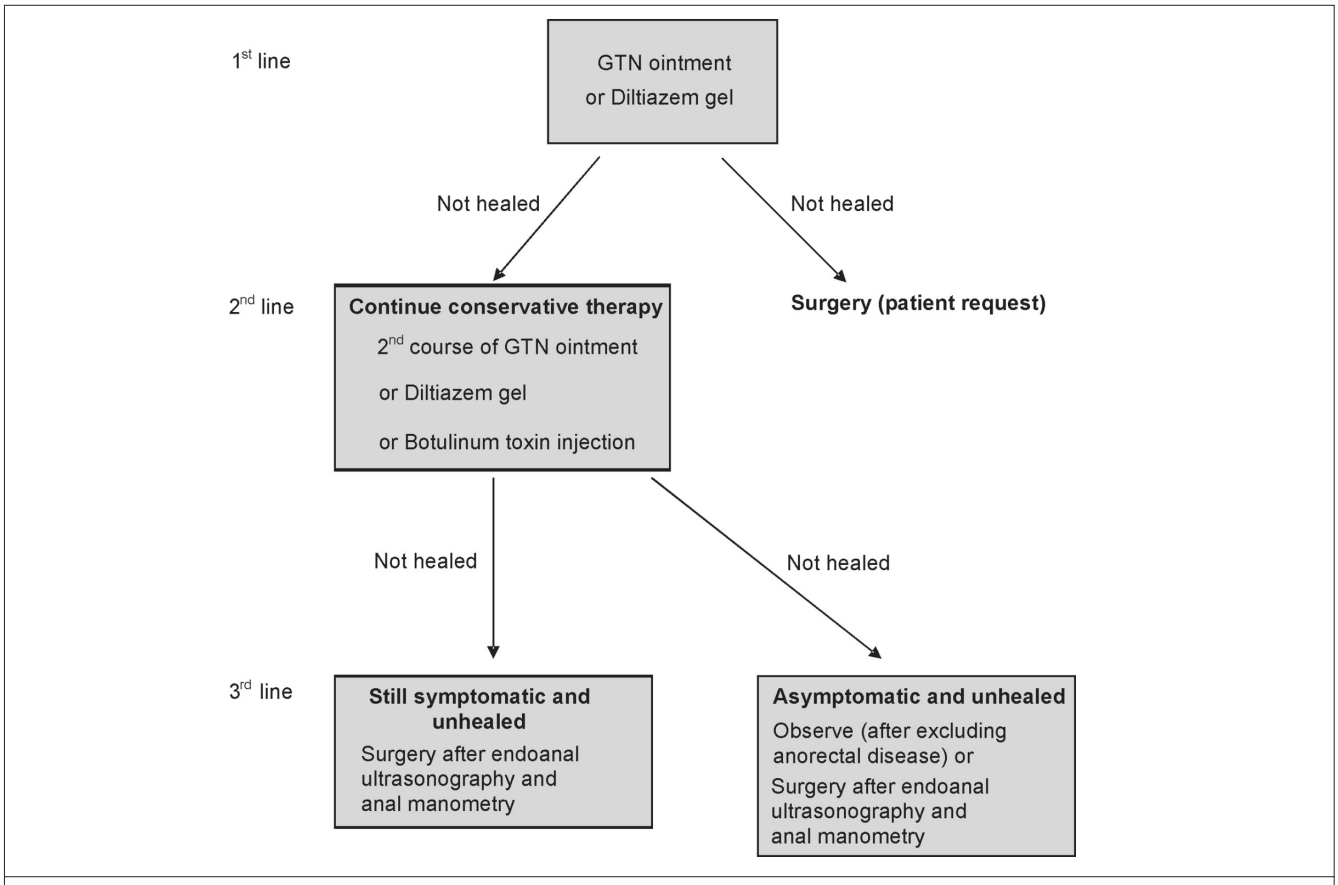

Figure 3 Treatment algorithm for chronic anal fissures. If patients heal, a follow-up out-patient appointment should be considered.

chronic anal fissure. Nine subjects withdrew within the first 2 weeks of treatment due to side effects which included fatigue, dizziness, headache, dry mouth, nasal congestion and retrograde ejaculation. At 6 weeks, the fissure healed in only one (7%) compared with two (22%) in the placebo group.[44] The trial was terminated.

Botulinum toxin

The precise anatomical position in which to inject botulinum toxin has been a matter of some debate as comparable healing rates are seen when injected into the internal or external sphincter (Tables 1 and 2). Jones *et al.*[45] have shown that botulinum toxin reduces the internal sphincter tone through its effect on the sympathetic nervous system. Despite concerns with the injection, patients seemed to accept its use in the out-patient setting, though in the UK most users would advocate injection under general anaesthesia. To address this, investigators have treated chronic anal fissures with botulinum delivered in an out-patient setting through a high-pressure device.[46] This mode of delivery needs further exploration.

As with diltiazem, botulinum toxin is effective in treating fissures that have failed to heal with topical agents. Botulinum toxin can also be combined with surgical modalities. Lindsey *et al.*[47] showed that, following injection of 25 U of Botox into the internal sphincter combined with fissurectomy in 30 patients (19 of whom had failed both GTN and botulinum toxin injection), 28 (93%) had healed after a median of 16.4 weeks' follow-up. Dysport is an alternative tolerable commercial formulation of botulinum toxin to Botox; however, the change in dose needs attention as, in one study by Brisinda *et al.*,[48] patients with fissures were randomised to receive 50 U of Botox formulation or 150 U of Dysport.

Conclusions

The first-line treatment of chronic anal fissures with topical agents has led to management algorithms that can be employed effectively.[49] Lindsey *et al.*[50] gave an excellent overview of the current treatment of chronic anal fissures and introduced the idea of poly-pharmacy with and without surgery. This synergy between topical, injectable and operative modalites requires continued appraisal. A practical algorithm that we think is evidence based and encompasses changing practice for the treatment of anal fissures is shown in Figure 3.

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Inter- and intra-observer variation of the Schatzker and AO/OTA classifications of tibial plateau fractures and a proposal of a new classification system

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We would like to apologise for the mispelling of two names and for errors in Table 3 in this article which was published in the May issue. The underlined names are now correct and a corrected Table 3 is given in this erratum.

Table 3 Proposed new classification for tibial plateau fractures. Fractures are described according to the number of condyles involved as well as the split/articular surface depression combination

Number of condyles involved
Unicondylar (medial or lateral)
Bicondylar
Type of fracture
Pure split
Articular surface depression without split
Articular surface depression with split