Review

AETIOLOGY, CLASSIFICATION AND TREATMENT OUTCOME IN ANAL FISSURES: AN OVERVIEW

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Summary

Acute anal fissure in a high percentage of cases is associated with increased activity of the internalanal sphincter, which is demonstrated with increased pressure in the anal canal at rest and with impaired recto-anal inhibitory reflex. The increased pressure is mainly in the back part of the anal canal, which is proved by vector manometry. The key for treatment of anal fissures is decreasing of abnormal values of anal pressure at rest. Nearly 90% of acute anal fissures heal with conservative treatment. Only 20-30% of chronic fissures can be treated conservatively. The main choice of specific treatment of chronic anal fissures (ChrAF) is chemical sphincterotomy with local application of nitrates, Ca-antagonists and phosphodiesterase-inhibitors. In case of failure, we use a second-line specific therapy for treatment of ChrAF - local application of Botulinum toxin. If the latter fails, we proceed to third-line specific therapy, i.e. surgical treatment. Surgical treatment of ChrAF is divided into: operative interventions, destructing the entirety of the anal sphincter complex-anal dilation and modifications of the lateral internal sphincterotomy and operative interventions, saving the anal sphincter complex through different types of reconstruction with skin and mucosal flap. Relapse frequency is between 2 and 16%. Postoperative incontinence is between 11 and 34%. The dilemma is to choose between development of postoperative incontinence, and the excellent result – healing without relapse and continence failure of the anal sphincter complex.

Key words: anal fissure, chemical sphincterotomy, anal dilation, lateral sphincterotomy, anal incontinence

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Etiology

Anal fissure is a defect in the anodermal layer, located in the distal anal canal, underneath the dental line, 1.5-2 cm long and 1-3 mm in depth. It can be primary (acute or chronic) and secondary (associated with other disease) [1-3]. This disease occurs in all ages but it is most common during second and third decade of life.

Acute anal fissures are superficial with a base made up of connective tissue. The fibers of the anal sphincter are usually invisible and the sentinel pile is not necessarily present. In some cases the

presentation is limited to hypertrophic anal papillae in the base of the fissure. The edges of the fissure are clearly distinguished from the surrounding tissue, and there are no signs of chronic inflammation. In most cases fissures are associated with constipation and the constipation can be due to inadequate diet. It is assumed that injury to the epithelium is due to a trauma from a fecal bolus in the posterior part of the anal canal underneath the dental line. The mucosa in this area is usually exposed to trauma because of the anatomic configuration of the anorectal angle. In children, fissures are usually associated with lack of defecation regime.

In most of the cases there exist no clear predisposing factors for anal fissures in adults [4]. It is assumed that they are due to passing large and solid feces. Giving birth is an etiological factor for development of anal fissures. Martin [5] reported a connection between giving birth and anal fissure in 11% of a studied group. According to Gough and Lewis [6], a postpartum fissure is usually anterior. The pressure caused by the fetal head onto the anterior commissure during birth is one of the causes for a higher rate of anterior fissures after giving birth. An alternative theory is that the primary trauma of the perineum leads to the formation of connective tissue and a scar, which makes them more vulnerable to secondary birth trauma. Postpartal fissures are relatively rare with a rate between 3 and 9%. Abramowitz et al. [7] described only 2 fissures in 165 women during pregnancy, and 2 months after birth, 25 of them had developed a fissure. One of the causes for postpartal fissures is the presence of a post-birth spasm of the external anal sphincter [4].

Change in the regime of defecation is a common contributing factor for both genders [8]. Usually there are episodes of constipation before the occurrence of anal pain. McDonald et al. [9] have found preceding constipation in 25% of their patients. Constipation is due to an anal fissure, because of the fear of extremely intense pain during defecation. Usually, in acute anal fissure there is a high activity of the internal anal sphincter [10] and high resting pressure, which leads to prolongation of the condition [11, 12, 13]. The sphincter spasm brings about a vicious circle: anal pain, fear of defecation, and passing of firm feces, which in turn stimulates the sphincter function [14, 15].

Another theory for development of posterior and anterior fissures is based on the specifics of the blood supply of the anal mucosa in these regions [16]. The inferior hemorrhoidal artery enters the anal canal at its lateral side and thus the medial zones are potentially ischemic. The ischemia can be visualized histologically by the presence of fibrosis in this area in patients with anal fissures. Klosterhalfen et al. [16] assume that chronification of the fissures in predisposed patients can be attributed to the relative ischemia in this area. Studies of Lund and Scholfield [17] and Lund et al. [4] have demonstrated that the ischemia is a result of the anal fissure. Schouten and his group [18] have found a close correlation between anal pressure and damaged circulation, demonstrating that perfusion in the anoderm of the posterior middle line is corrupted, especially in the part of the fundus of the fissure and, based on these results, assume the anal fissure as ischemic ulcus (Figure. 1).

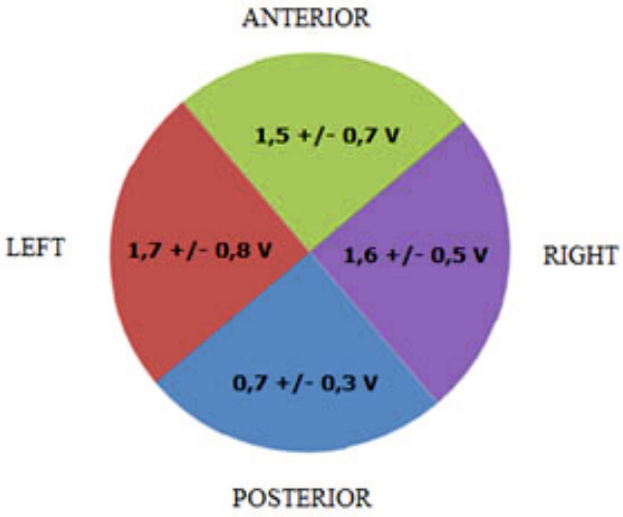

Figure 1. Perfusion in the anoderm in all quadrants of the anal canal presented as flow (v) [18]

Shouten's research correlates with postmortal angiographic studies which have shown small number of branches of the inferior rectal artery in the posterior side of the anal canal [16, 18]. Increase in the tone of the external sphincter reduces perfusion in this area which leads to the conclusion that increased tone and inadequate perfusion are responsible for the formation of anal fissure [15].

Acute anal fissure is associated with increased activity of the external anal sphincter, demonstrated by increased resting pressure of the

canal in high percentage of the cases [11, 19-22]. Increased pressure, proven by vector manometry is mainly found in the posterior part of the anal canal [23, 24]. Farouk et al. [25] have found dysfunction in the rectoanal inhibition reflex in patients with acute anal fissures.

If complaints persist for more than 6-8 weeks, the fissure is chronic. There is very strong anal pain during defecation, usually accompanied by mild bleeding.

The edges of the chronic anal fissure are dense, undermined and the fibers of the external anal sphincter are visible at the bottom. Later the fissure becomes wider and swollen due to obstruction of local lymphatic vessels, with signs of cavitation and inflammatory response. Progressing edema leads to formation of skin process and hypertrophic papilla at the upper end of the fissure [4]. Local inflammation can spread proximally and cause submucous and intrasphincter abscess in about 2% in the cases or to spread distally with formation of perianal abscess underneath the skin process with superficial fistula, beginning from the inferior edge of the fissure. This kind of fissure-fistula is present in 4% of the patients.

If an acute fissure becomes chronic in 60-65% of the patients, normal or lower resting anal pressure is seen. In the group Jones [26] studied, 55% of the patients had normal anal pressure, while in 8% of them it was low. Usually, a huge group of patients with normal or lowered resting pressure cannot be diagnosed only by digital anal examination. In 4-7% of the patients the predisposing factor was diarrhoea [3]. Previous diseases of the anal canal were registered in 26-32% of all chronic fissures. In most of the cases these were recurrences of previous anal fissures that had been successfully treated. In other cases there were previous haemorrhoidectomy or postoperative lesions of the anal mucosa which had led to stenosis and deformation, thus increasing the risk of anal fissures accompanied by constipation [4]. The same mechanism was observed in posterior fissures after successful drainage of intrasphincreric abscess (Table 1) [3, 27].

Table 1. Symptoms and aetiological factors in anal fissures

	Lock a. Thomson, 1977 Number - 188	**Birmingham series Number - 355**
Symptoms %		
Pain	87	82
Bleeding	82	74
Itching	44	14
Swelling	29	32
Leakage	7	4
Regime of defecation %		
Constipation	14	24
Diarrhoea	4	7
Pathology %		
Postpartal	3	9
Previous anal diseases	26	32
Duration (weeks)	11	21

Classification of anal fissures

Anal fissures can be classified as primary and secondary. Primary anal fissures can be acute or chronic. According to their position in the anal canal they are anterior, posterior, lateral or multiple [Table 2]. Localization is determent by examination and confirmed by anoscopy. Over 2/3 of the fissures are single and posterior. In the Birmingham series 89% were posterior, 7% were anterior, 2% were on both sides of the midline, and 2% were lateral [27]. Anterior fissures were more common in women (21%), while in men these were 9%.

Table 2. Location of anal fissures in percent [2, 3, 6, 9, 27]

Site	Lock a. Thomson (1977) n=188	Shub et al. (1978) n=393	Gough a. Lewis (1983) n=97	McDonald et al. (1983) n=81	Birmingham series n=355
Anterior	14	27	12	7	7
Posterior	75	66	82	88	89
Both	8	7	2	1	2
Lateral	3	0	3	4	2

Some diseases can imitate anal fissures. Perianal sepsis, which is a complication in patients with neutropenic leukemia, can present with the signs of anal fissure. Squamous carcinoma, basocellular carcinoma and adenocarcinoma in the lower third of the rectum, complicated with anal sepsis can be misdiagnosed as fissures. Similar symptoms can be found in the anal form of Paget's disease, intersphincteric abscesses, proctalgia fugax, thrombotic hemorrhoids and pruritus ani. All these conditions require careful differential diagnosis.

Changes of the anal pressure in patients with anal fissure

Patients with acute anal fissure have significantly increased resting anal pressure as compared with control groups [22], the pressure being a result of increased activity of internal anal sphincter caused by continuous ultraslow waves [11]. Many studies have confirmed that resting anal pressure in the anal canal increases when acute anal fissure is present [15, 19, 28, 29, 31, 32]. Abcarian et al. [33] and Orthmann and Shuster [34] have reported abnormal rectoanal inhibition reflex in high percentage of patients with anal fissure. Ultraslow waves have been registered by Hancock [11] in 10 out of 12 patients with acute anal fissure, in contrast to the control group, for which the result was 2 out of 40. Cerdan has found not only increased resting anal pressure but also higher values of pressures in patients with acute anal fissure [12]. Resting anal pressure is normal in patients with chronic anal fissure but it varies in wide ranges [22]. Two populations of patients with chronic anal fissure can be distinguished: those with high resting anal pressure and those with low or normal resting anal pressure [29]. This data was confirmed by the Oxford group [26]. Out of 40 patients with chronic anal fissure, 15 (37%) had high anal pressure, 22 had normal anal pressure (55%), and 3 had low anal pressure (8%).

Evolution of the disease

Over 80% of acute anal fissures heal without treatment, while the rest progress to chronic fissure. When chronification occurs, chances for spontaneous healing drop to 20-30%. Thus, a fissure can heal, remain static, progress to fissure-fistula, or be complicated by abscess. Exception from this are the secondary fissures in Crohn's disease, in which self-cure with systemic treatment without local therapy can be expected in 80% of cases [35]. Sweeney et al. reported that 69% of the fissures in Crohn's disease are cured after systemic treatment of the underlying disease [36].

Treatment of anal fissures

Normalization and regulation of the act of defecation as well as use of laxatives are essential for the success of treatment. Symptomatic therapy includes the use of a bidet, showers, a bathtub, diet with high amount of fiber. Hot baths reduce the symptoms but do not have an affect to anal pressure [37]. Gabriel [38] has reported good results from local therapy with opiates and silver nitrate. Local anesthetics such as xylocaine and lidocaine 2% gel can reduce the pain in patients with acute anal fissure but it can lead to sensibilization in 2% of the patients [39, 40]. Steroid drugs can reduce the swelling and can lead to spontaneous cure. High rate of remissions has been reported by Jensen [41] who used steroids and anesthetics locally and controlled the constipation.

The key in treatment of anal fissures is in reducing the abnormal values of resting anal pressure. Failure to reduce resting pressure is related to persistence of symptoms [42]. Almost 90% of the acute anal fissures heal after conservative treatment [43]. Only 20-30% of chronic fissures can be cured conservatively.

First line in specific treatment of chronic anal fissures (CAF)

First line in specific treatment of chronic anal fissures (CAF) is chemical sphincterotomy.

Local admission of nitrates

Important progress in conservative treatment is admission of drugs, which reduce resting sphincter tone [44]. Short-term healing has been reported after using nitric oxide donors to reduce the sphincter tone and resting anal pressure [45, 46]. Reduction of the anal pressure with glyceryl trinitrate has a short-term effect – between 15 and 90 min. [47]. Most of those studies report good results in 40-50% of cases but there is a high relapse rate. Headache is a common side effect of this therapy.

One of the main drawbacks of local therapy is defining an optimal dose [48]. In an ointment, the dose depends on the content of the active substance and the amount of the ointment applied, which can be hardly controlled. Lund and Scholefield [17] used 0.5 g 0.2% cream applied 2 times per day. In a multi-centric study in USA Bailey et al. [49] applied an intraanal applicator to control the dose. The device dosed 374mg of nitroglycerin. Healing was achieved in 50% of cases, and it was not dose-dependent.

Another problem is tolerance to the drug after long-time use but this is not taken into consideration in clinical practice.

Of the large number of studies made to compare local administration of drugs containing nitrates with placebo, none has statistically shown the advantages of mono-therapy with nitrate-containing drugs [50, 51, 52]. The drawback of these large-scale studies is due to the high percentage of recovery in the placebo group. These results render the results from treatment with nitrates insignificant.

The high percentage of recovery in the placebo group is due to spontaneous cure of some acute fissures. Despite this result, meta-analysis have demonstrated that nitric oxide donors are considerably more effective than placebo [53].

Long-term results from administering nitrates vary. It is suspected that relapse shortly after nitrate therapy is due to discontinuation of the therapy because of lack of symptoms. Early relapses can be treated with an additional course of nitrates with success in up to 90% of the cases [54]. The patients who do not tolerate the therapy well are usually found with well-presented sentinel piles. These patients do not respond to this treatment and early relapses occur [55].

Calcium channel blocker (CCB)

Local and oral CCBs have been proved effective in reducing resting anal pressure. CCBs work by reducing the pace of depolarization of muscle cells [56]. Oral administration of Nifedipine or diltiazem can lead to hypotension [57]. Most commonly CCB used are diltiazem and nifedipine [57, 58, 59, 60].

Several small studies proved that local treatment with 2% gel diltiazem leads to recovery in 70% of patients with chronic anal fissure [54, 59]. Although these drugs do not cause headache, they can cause local skin reaction and are considerably more expensive than glyceryl trinitrate (GTN).

Garffin et al. [57] applied local diltiazem in patients with intolerance to GTN and in patients with complaints after GTN treatment and are not eligible for sphincterotomy.

The results from random researches comparing topical use of nifedipine with placebo [60] are shown on Table 3.

Table 3. Local administration of Nifedipine vs placebo as therapy for chronic anal fissure

	Nifedipine (0.3%) n=55	Controls n=55
Heal of the fissure	54/55 (98%)	8/55 (14%)
Side effects	0/55	0/55
Relapses	3/55 (5%)	n.a.

Additional comparison between nifedipine and GTN showed that GTN is more effective in the cure of fissures but with more side effects [61]. Another comparison between topical application of diltiazem and GTN shows similar results from the therapy but in the group treated with GTN there was headache as a side effect in 33% of the patients [62].

Phosphodiesterase inhibitors

One of the side effects of sildenafil (Viagra Pfizer) is the relaxation of the external anal sphincter with a subsequent drop in resting anal pressure. This has been proved by in-vitro

research where the effects of sildenafil were studied on animal anal sphincters [54]. Although these drugs can be favoured in the treatment of chronic anal fissures there no clinical evidence to back up their use.

Nowadays, nitric oxide donors are first-line agents in treatment of chronic anal fissures, with a choice between 0.2% and 0.4% GTN. In cases of intolerance to GTN or the symptoms persist after an 8-week course, therapy continues with local administration of diltiazem. If this proves inadequate, recommendations are to proceed to second-line drug therapy.

Second line in specific treatment of CAF

A relatively new approach in the treatment of CAF is the application of botulinum toxin [63]. Botulinum neurotoxins accumulate in the neuromuscular transmission site and block only cholinergic nerve endings and non-adrenergic response of nerve endings mediated by nitrooxyde. Therefore, in combination with GTN they may produce a greater effect. Local administration of botulinum toxin is not harmful [64]. It leads to healing in 3 of 5 cases by reducing resting anal pressure by 23% [65]. Jost [66] has reported that in 78 of 100 patients there was no pain after an injection of Botox. Early healing was reported in 83% of the patients, and slower healing – in 79%. Transient faecal incontinence was detected in 7 patients.

In the study conducted by Lindsey et al. healing was reported in 43 (96%) [67]. Temporary low degree of incontinence was registered in 18%.

There are different views about the site of injection and treatment plan. Most authors inject 2.5 to 20 units of Botox in the intrasphincteric groove on both sides of the fissure. The maximum single dose is 50 units. Common complications include hematoma at the application site and postoperative pain [68] .

Botox therapy is recommended for recurrent fissures in cases of failure of first-line specific therapy and when there are contraindications to sphincterotomy. Combining Botox with GTN in the treatment of recurrent anal fissure achieve healing in 76% of patients [69].

Sclerotherapy

Sclerotherapy using sodium tetradecyl sulfate after local infiltrative anesthesia leads to healing in 80% of the cases [70]. However, it may lead to abscess formation at the site of application. Despite the good results, sclerotherapy is not recommendable [2].

Hyperbaric oxygenation

Hyperbaric oxygenation as a method of treatment of CAF is described by Cundall et al. [71].

Cryodestruction

Savin, (1975), and O'Conor, (1976) have described the application of cryodestruction in hemorrhoids, when combined with anal fissure, with good results. In case of a failure of conservative treatment, third-line specific therapy in the treatment of chronic anal fissure is administered [72,73].

Third line therapy of CAF

Third line therapy of CAF is surgical treatment.

The aim of surgical treatment is to reduce the activity of the internal anal sphincter and provide healing of the anal fissure [8, 15, 31, 74]. There are two fundamentally different surgical approaches in the treatment of chronic anal fissures:

A. The first approach is to reduce anal pressure by anal dilation or internal sphincterotomy [43, 75].

B. The second approach is applicable in patients at risk of developing incontinence. This approach includes curettage or cutting the fissure, and covering the defect with a mucosal flap [76-79] . An alternative to that is a combination of fissurectomy with application of nitrooxyde and botox [80, 81].

Anal dilatation (AD)

Most colorectal surgeons believe that anal dilatation is not applicable in the treatment of anal fissures. With this approach, there is a risk of anal incontinence, and patients should be informed about it [82, 83]. Side effects of anal dilation include bleeding, hematoma, discomfort, first and second degree anal incontinence, and urine incontinence or retention. In case of hemorrhoids, anal dilation can lead to prolapse [84].

Transient incontinence after anal dilatation was reported in more than 30% by Wats et al. [84]. Permanent incontinence was found in more than 10% of patients [75]. The incidence of incontinence vary widely and depend on the degree of dilatation and associated risk factors: age over 60 years, vaginal birth, previous operations on the perineum and anus, neurological diseases.

Open lateral sphincterotomy (OLST)

OLST requires selection of the patients. It is not recommended in patients at risk of incontinence: previous birth trauma, age over 60 years, previous anorectal operations, neurological diseases and registered low levels of resting anal pressure [32]. The length of the sphincterotomy incision corresponds directly with the grade and development of postoperative anal incontinence. However, this length should also comply with clinical situation [85-87].

Complete healing of anal fissure after lateral sphincterotomy occurs in 92-100% of cases [3, 88-90]. Temporary first and second grade incontinence in up to 9% of cases was reported by Leong et al.[91], which subsides within 2-3 months. Permanent incontinence developed in 5-6% of cases [89, 92]. Garcia - Aguilar et al. [93] reported incontinence of gas in 30% , incontinence of fluid in 27% and incontinence of feces in 12% of cases. Percentage of incontinence is higher in open than with closed sphincterotomy. These results indicate that sphincterotomy should not be made before treatment with pharmacological agents and botox, and without warning the patient of possible failures.

The development of hematoma and fistula as a complication of open sphincterotomy are rare. Usually found are bleeding, urinary retention, delayed healing, pain, pruritus ani, anal leakage and perianal abscess [92].

Concealed subcutaneous lateral sphincterotomy (CSLST)

With this method, selection of cases is obligatory. There is a considerable risk of developing postoperative incontinence, especially in patients with previous anal surgery, elderly patients and those with birth trauma. The advantages of CSLST, as compared with OLST, is that there is no cut. Thus, post-operative pain is reduced and the recovery period is shorter. The incidence of incontinence is lower as compared with OLST [93]. Moreover, transanal ultrasound control during the procedure could be applied. Marby et al. [28] have reported recurrence in 50% of patients with concealed sphincterotomy under local anesthesia, while recurrence was seen in 17% of the cases undergone CSLST under general anesthesia. Postoperative management in CSLST is the same as in anal dilatation, including laxative medications and diet. General anesthesia without muscle relaxant is preferable because of the possibility to distinguish the intrasphincteric groove. Hoffmann et Goligher [94] ; Millar [99] ; Oh [95] ; Lewis et al. [89]; Leong et al. [91] have reported development of hematoma in 4-6% of cases, abscess in 1-2% and fistula in 0.5-1%. Incontinence was found in 9 -14% as transitory and in 6-7% as permanent. According to Garcia-Aguilar et al. [93] permanent incontinence rate was higher.

Results from the of surgical treatment

Clinical results of anal dilatation, OLST and CSLST are demonstrated in Table 4.

Table 4. Comparing treatment outcomes of CAF in OLST, CSLST and AD

	OLST	**OLST**	**OLST**	**AD**	**AD**	**CSLST (OA)**	**CSLST (LA)**
	Lock a. Thomson (1977) n=82	Bailey et al. (1978) n=418	Lewis et al. (1988) n=103	Watts et al. (1964) n=90	Oh (1978) n=200	Hoffman a.Goligher (1970) n=99	Millar (1971) n=99
Relapse %	0	8	8	16	2	3	0
Incontinence%	0	2	6	28	3	12	3
Bleeding, hematoma%	1	1	1	0	4	22	6
Fistula %	0	1	0	0	0,5	1	1
Abscess %	0	0	2	0	2	1	1
Prolapse of hemorrhoids %	0	0	0	1	0	2	1
Urinary retention %	1	0,2	0	0	0	0	0

GA –general anesthesia; LA-local anesthesia

Anatomic efficiency of the performed sphincterotomy can be determined by intra-anal ultrasonography. Small defects are associated with a higher risk of relapse [96].

Relapse rate

Incidence of relapses after anal dilatation reaches 16% [84]. The incidence of relapses after anal dilatation is higher than after sphincterotomy [97, 98]. OLST is associated with a recurrences rate of 0-8% [3, 88, 89] and CSLST under local or general anesthesia with 2-3% recurrence rate [95, 99, 100]. Lewis [89] compares OLST with CSLST, and found that persistence of the fissure was more common in OLST (8%) as compared with CSLST (5%).

Incontinence

Incontinence due to sphincterotomy performed in Leeds was evaluated and compared with results of 100 healthy subjects (control group). Incontinence first degree was reported in 11% of healthy subjects, as compared with a figure almost identical in patients with CSLST – 11%. Incontinence rate after an open back sphincterotomy was significantly higher – 34% [19], and after anal dilatation it was 31% [84]. Lewis et al. [89] have demonstrated that transient incontinence is more common in OLST (14%), as compared to CSLST (9%). Long term incontinence was 6% and 7%, respectively. nielsen et al. [75] performed transanal sonography in patients after anal dilatation and found that 11 of 18 continent patients had sphincter defects - 10 internal and 2 external. The width of the intrasphincteric defect was greater in women probably due to the shorter length of the anal canal. Therefore sphincterotomy, especially in women with previous birth trauma, should be undertaken with great care. incontinence after internal sphincterotomy is mostly transitory [90, 91, 101]. Permanent incontinence is more common after anal dilatation as compared with internal sphincterotomy 12% vs 3% [75,90], and dilatation has negative effects on the quality of life [102, 103].

Permanent incontinence first degree was reported in 30% of patients after OLST and in 24% after the CSLST. Second degree of incontinence was reported in 27% in OLST, as compared with 16% for the CSLST. Third degree incontinence was 12% in OLST against 3% in CSLST [93]. These frightening results prove that sphincterotomy implies a risk of incontinence that is higher in OLST [104]. The dilemma is between incontinence and healing.

Sphincterotomy or anal dilatation

Olsen et al. [105] compare anal dilatation with CSLST. They registered recurrence in 3 of 10 patients after anal dilatation, and in only 1 in 10 patients after CSLST. In two patients in each group transitory incontinence occurred.

Open or covered sphincterotomy:

Boulos and Araujo [106] compare CSLST with OLST. No recurrences were recorded, and postoperative pain was similar in both groups. Bruising was more common in CSLST. Transitory incontinence of first degree was similar in both groups. All operations were performed under general anesthesia.

A randomized study of sphincterotomy to the level of the top side of the fissure – 4-5 mm below the level of linea dentata, or sphincterotomy reaching the level of linea dentata, has shown significantly better fissure healing with longer sphincterotomy and greater reduction in anal pressure. This, however, was associated with high-degree of anal incontinence [107]. Based on these observations, the authors recommend cutting the internal sphincter high and above the upper edge of the fissure.

Advanced flap (flap plastic):

The concept of cutting the fissure and covering the defect with a skin flap or mucosa is attractive because it does not entail the risk of incontinence, there are good chances of healing, especially in ischemic lesions owing to the fact that skin or mucousal flaps contribute to better blood supply of the area. These techniques are applicable to complicated fissures with anal stenosis since the mucosa is used to cover the upper part of the fissure defect.

Nyam et al. [75] have report the results of islet advanced flap plastic surgery in 21 patients with recurrent fissures with low resting anal pressure at risk of incontinence in standard therapy. Anal ultrasound examination proved sphincter defects in 15 patients from the group described. All plastics healed, sensation was preserved and violation of continence was established.

Island plastic surgery is considered for in the treatment of anal fissure in patients with low resting pressure in the anal canal, where the risk of incontinence after conventional sphincterotomy is high. The role of the anoplastic is well known in the treatment of patients with anal stenosis or after fissurectomy or

hemorrhoidectomy [108]. Advanced flap plastic can be complicated with sepsis, flap peeling, scarring and poor results.

Our experience

We studied 313 patients with chronic anal fissure. In 8 patients, chemical sphincterotomy with 0.2% nitroglycerin unguent was applied. Gel nifedipine 0.2% was used in 11 patients. Other treatments included: cryodestruction - 61 patients, anal dilatation - 37 patients, open lateral sphincterotomy - 82 patients, plastic with proximal mucosal flap - 18 patients, and a combination of surgical techniques - 96 patients.

In the patients with medication sphincterotomy we found 50% relapse in the group with nitroglycerin versus 100% epithelization in the group treated with nifedipine gel, without violation of continence.

At the end of the first postoperative month, in the group with cryodestruction mild degree incontinence was present in 80% of the patients, and 18% had moderate degree incontinence (severity assessed by FISI). The results found on the sixth postoperative month in the patients treated with cryodestruction were excellent: there were no cases of anal incontinence, and no pain or epithelialization were present.

In our series, the relapse rate after AD was 2.5%, and the cases of permanent mild degree anal incontinence accounted for 11.2%.

Six months after In our study, after open lateral sphincterotomy, no relapses occurred, and the anal incontinence registered in this group was 11.0%.

In the patient series, in which plastics with proximal mucosal flap was applied, no relapse and continence disorder were found.

The analysis of our results and data from the literature allows us to adopt the principle of consistency in choosing a treatment strategy in patients with chronic anal fissure. Our first level of choice was medication sphincterotomy. In case of failure, it was proceed to patient-tailored surgical techniques, taking into consideration.

The choice of treatment should be consistent with the specifics of the perineal anatomy and the physiology of the act of defecation in each individual patient. An inappropriate surgical intervention on the anal sphincter complex may lead to complications after treatment that could be far more serious than complaints before the intervention.

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