New approaches to the treatment of anal fissure

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According to Antropoli, pathologies of the anal canal are extremely common. About 30 to 40 percent of the population suffers from proctologic pathologies at least once in their lives. In most cases they are more annoying than dangerous. Anal fissure (AF) was recognized as a clinical entity in 1934. It is a longitudinal defect of the anal canal mucosa and anoderm extending usually from the dentate line to the external verge of the anal canal. This defect exposes the lower half or even most of the fibres of internal anal sphincter. AF is almost always accompanied by extensive tension of this muscle. Anal fissures affect all age groups but predominantly occur in the 3rd and 4th decades of life. Gatnighthouse states that fissure disease causes from 6 to 15% of office visits and 10% of operative procedures in a colorectal practice. The etiology of anal fissure has only been partially explained and remains controversial although spasm of the internal anal sphincter has been recognized to play a main role in the pathogenesis of this disease. Recent studies have cast new light on the pathogenesis of anal fissures.

Key words: anal fissure, sphincterotomy, botulin toxin

INTRODUCING

It has been hypothesized that anal fissure may be an ischaemic ulcer caused by combination of spasm of the internal anal sphincter (IAS) and poor blood supply to the posterior midline of the anal canal, the site at which the majority of fissures occur. The increased internal sphincter tone in patients with a fissure reduces anorectal blood flow at the posterior midline. Reduction of anal pressure by sphincterotomy improves anodermal blood flow at the posterior midline, resulting in fissure healing. These findings provide evidence of the ischaemic nature of anal fissures. The above mentioned considerations concerning the pathogenesis of anal fissure constitute the basis for modern therapeutic option although strict consensus has not been established. The basic requirement for a successful treatment is decrease of sphincter tone, relief of pain and restoration of normal rectoanal coordination.

The primary goal of all therapeutic measures is to break the vicious circle of inflammation, pain and anal sphincter spasm. The optimal therapy should be safe and successful about healing and continence. Lateral sphincterotomy is regarded as a gold standard in the treatment of anal fissures but one should emphasize that surgery carries potential risk of subsequent fecal/flatus incontinence that is reported up to 40 percent of cases. That is the reason why some surgeons suggest initial medical treatment of anal fissure and if it is not effective, surgical sphincterotomy may be required. Some methods of so called reversible chemical sphincterotomy paved the new way for a therapeutic option in the treatment of anal fissures. Non-permanent chemical denervation with botulinum toxin, topical application of nitroglycerin ointments or calcium channel blockers such as nifedipine have been proposed as noninvasive alternatives to surgical methods and these techniques of inducing temporary sphincter paresis have recently demonstrated encouraging results. The relief of sphincter spasm lasts long enough to allow the fissure to heal. Hallan et al. in 1988 introduced botulinum toxin (BTX) to proctology for the treatment of anismus. In 1989 injections of botulin toxin into the external anal sphincter became a new option in the treatment of AF. Nowadays BTX injections are given into the internal anal sphincter. Jost and Schimrigk in 1994 published their first results of using BTX in patients with AF. Botulinum A toxin is an endopeptidase produced by microorganism Clostridium botulinum that acts rapidly by binding to the presynaptic nerve terminal at the neuromuscular junction and at cholinergic autonomic sites. 15-25 U of Botox or 100 U of Dysport are given using an insulin syringe, no anesthesia is required. Paralysis occurs within a few minutes. In this way the toxin prevents the release of acetylcholine presynaptically, thus blocking neurotransmis-
Transmission of neuromuscular impulses resumes after the growth of new axon terminals and clinical weakening of muscle is seen for 3 to 4 months. Botulinum toxin may induce healing by simply increasing of local blood flow or by more complex effect. According to Jost, injection of BTX causes incontinence. However, this is only temporary, in the course of a few weeks, musculature is reinnervated and incontinence is completely regained. Sometimes perianal thrombosis is observed. Resting anal pressure significantly decreases after BTX injection whereas squeeze anal pressure is nearly unaffected. Resting anal pressure tends to rise by 2-3 months. Jost and other authors reported that healing rate after treatment with BTX was approximately 80 percent, sometimes even 100 percent. Jost sees 8 percent of relapses in the course of six months. In such cases, reinjections can be carried out. Other drugs which decrease internal anal spasm, improve peristalsis and promote healing of AF are nitroglycerin and diltiazem (nifedipine). The product of nitroglycerin metabolism - nitric oxide - is an inhibitory neurotransmitter in the IAS. It is applied topically twice daily for 8-12 weeks. Topical application of glycerine trinitrate (GTN) as a 0.2% ointment achieves a prompt healing of AF in 70-80 percent of cases. About 20% of patients experience a strong headache that makes them interrupt the treatment. Different results of using GTN are reported. Carapeti et al. observed high relapse rate (33%) and severe headache in 72% of cases. Gorrini applied 0.2% nitroglycerin ointment directly on AF. In 83% of patients the fissure healed after 2 weeks and in 100% after 1 month. These results refer to acute anal fissure. In the group of chronic AF, 68% of patients healed after 8 weeks. Calcium ions are an important factor in the process of contraction of smooth muscles. Nifedipine, by blocking calcium channels is commonly used in the treatment of cardiovascular disorders, and was also used in the treatment of achalasia. It inhibits the release of calcium ions into the sarcoplasm of smooth muscle cells, thus decreasing the force of contraction of muscle fibres. Recent studies have shown that this drug applied topically as a 0.2% gel in the dose of 5 mg twice daily decreases the resting tone of IAS. Treatment lasts for 6-12 weeks. With topical application its level in blood is much lower than when administered orally, so no adverse effects are observed. Additionally, nifedipine has an anti-inflammatory effect and acts favourably on microcirculation. Antropoli et al. noted healing of acute AF in 95% of patients after 21 days of the treatment. Cook et al. treated patients with chronic AF administering nifedipine orally in the dose of 20 mg twice daily. They observed healing of AF after 6-8 weeks. No cases of permanent damage to sphincters were noted. Some patients may experience a mild headache at the beginning of treatment as well as a feeling of flushing in head and legs.

When medical treatment fails then surgical management is necessary. Sphincterotomy has a long history. In 1833 Dupuytren was the first who suggested division of anal sphincter fibres in order to treat AF. In 1835 Brodie and in 1863 Hilton proposed the incision of internal sphincter in the posterior midline in the area of fissure itself. In 1951 Eisenhammer also proposed the division of IAS. Bennett and Goligher in 1962 noticed that sphincterotomy performed in the posterior midline led to key-hole deformity of anus. In their opinion it was the cause of post-operative soiling. Bennett and Duthie in 1964 observed such a deformity in 12 out of 16 cases. Notaras in 1969 described lateral subcutaneous sphincterotomy. This modification prevents creation of a large wound opening thus protecting against infection. Subcutaneous layer of EAS is preserved with this method. Wound healing is better and no deformation is observed. Routinely performed sphincterotomy is made just above the dentate line. Littlejohn et al. pointed out that during tailored lateral sphincterotomy (sphincterotomy reaching the proximal margin of AF) the incidence of sphincter dysfuction is much lower what is associated with a much shorter transection of muscle fibres. Posterior sphincterotomy was studied in 1992 with the finding that 1/3 of patients had significant incontinence problems. In 1991 The American Society of Colon and Rectal Surgeons made the following statement: The treatment for fissure-in-ano refractory to nonoperative therapy is internal sphincterotomy. The same Society recommends caution before performing lateral sphincterotomy, particularly in elderly patients or those with diarrhoea, irritable bowel syndrome, diabetes or recurrent fissure after previous surgery. It seems that final outcome of the treatment depends also on the kind of anaesthesia used. Keighley et al. compared the results of sphincterotomy performed under local and general anaesthesia. Their results confirm the superiority of general anaesthesia. Local anaesthesia does not provide an adequate relaxation of IAS leading often to an incomplete sphincterotomy.

In conclusion one can say that so many therapeutic options of anal fissure show that none is optimal. Taking into account various complications associated with surgical treatment we should first implement conservative treatment both in acute and chronic anal fissures. When it fails there is time for lateral sphincterotomy.

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