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Straining And Anal Sphincters: Cause And Effect Relation In Benign Anal Disorders

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Abstract

Anal disorders are one of the most commonly encountered diseases in a regular surgical out-patient clinic. However, the understanding of this entity remains obscure amongst the patients and evaluation by the clinician is mainly restricted to the relief of symptoms only. This group of disorders affects most of the population with a wide range from younger to elderly and across the gender. The process of defecation is complex, reflex dependent and the synchronous action of various sphincter mechanisms. Straining during defecation is against physiological principles and causes structural damage to soft tissue structures situated in and around the anal canal. This is an attempt to focus on developing a basic understanding of the complex process of defecation, explain the effect of individual response to symptoms and stress the importance of education of the clinician and patient in complete recovery of the patient.With the advances in technology and improved clientele awareness, the diagnosis of the disease has become easy, and management is directed towards the relief of the symptoms. Surgical intervention is planned to depend upon the severity of symptoms. This is a review article and compilation of literature from various texts to stress upon an understanding of the etiopathogenesis of a variety of anal disorders and their evaluation. Excerpts from various articles and textbooks are used to come to conclusions. There is also a role of individual experience in the surgical field to understand the symptomatology and response of patients to treatment.

Key Words

Constipation; Straining; Defecation; Internal Anal Sphincter; Anal manometry

Conflicts of Interest

Nil to declare

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I. Introduction

Benign anal disorders are a group of diseases arising in and around the anal canal without malignant origin`. Mainly these are attributed to dietary habits, type of food, working pattern of the individual, the personality of individual, comorbidities and a variety of other factors. Regular bowel habit is one parameter of good health, especially in the Indian subcontinent, where ancient literature on health advocates the same. Even as a general rule, bowel habits are considered of paramount importance, giving an individual a sense of well-being. Regular bowel movements result from multiple factors influencing the physical characteristics of stools, actions of the anal sphincters, neurological and vascular supply of the sphincters, skin around the anal canal and so on. Any disturbance in these factors causes disturbances in the regular bowel movement either in timing or other parameters. The most significant of these disturbances is constipation which is perceived by many in very different ways. In the Rome II criteria for functional constipation, at least two of the symptoms listed below have to be present in more than one-quarter of defecations over at least three months in the last year (1).

1. Straining
2. Lumpy or hard stool
3. Sensation of incomplete evacuation
4. Sensation of anorectal obstruction or blockage
5. Manual manoeuvres to facilitate defecation
6. Fewer than three bowel movements per week
7. Insufficient criteria for irritable bowel syndrome

In most cases, one or more of the above symptoms is present, labelled as constipation. As can be made out, except the last two, the rest of these are 'subjective criteria' for defining constipation. Moreover, these symptoms do not point towards the aetiological background. Hence in clinical practice and interaction with patients, the term constipation is perceived subjectively by the patient and the management by clinicians is directed mainly towards the relief of symptoms. This article will explain that a basic understanding of the defecation process is paramount for patients and clinicians. A detailed history taking, understanding the patient's symptomatology, and education become part of the treatment process for early recovery and reducing recurrence.

II. Review of literature

Functional Anatomy and physiology of Anal Sphincter:-

Maintenance of continence without hampering the smooth, regular and painless passage of flatus and faeces at an appropriate time and place is the primary function of the anal canal. This is facilitated by intimately linked, somatic, intrinsic, and autonomic nervous systems. Two differently oriented muscular tubes, namely outer and inner, plays an important role. Whereas the outer tube is funnel-shaped, composed of skeletal muscle, and innervated by somatic nerves, the upper portion of this funnel is formed by the levator ani muscles. This sheet of muscle originates from the sacrum (posteriorly), the pubis (anteriorly), the pelvic sidewall (laterally) to the upper anus. Three groups of fibres of levators, namely the puborectalis (inner), pubococcygeus, and iliococcygeus muscles (posterolateral), form a funnel and the tip of this funnel ends as an external anal sphincter (E.A.S.). Although this voluntary muscle has been divided into three clinically and physiologically, it acts as a unit. Contraction of this muscle produces the anal squeeze examined during the digital examination. The external anal sphincter (E.A.S.) muscle is composed mainly of slow-twitch striated muscle fibres. Though it partially contributes to the resting anal pressure, its primary function is to generate the anal squeeze pressure during defecation. The E.A.S. is partly under voluntary control via Onuf's motor nucleus in the spinal cord through the pudendal nerve and the perineal branch of the fourth sacral nerve. (3, 4, 5)

The inner tube of the anal canal, the Internal Anal Sphincter (I.A.S.), is composed of visceral smooth muscle under the control of autonomic nerves and is a direct caudal continuation of the circular muscle layer of the rectum. These longitudinal muscles of the rectum also have an attachment to the perianal skin. These inner muscles of the anus are controlled by branches of the inferior rectal nerve and the perineal branch of the fourth sacral nerve. Its primary function is to contribute to the anal resting pressure, hence remains contracted during rest to provide a resting anal tone. (2) The sympathetic nervous system stimulates the I.A.S. and causes contraction of the internal sphincter. Other pelvic floor muscles do not have an active role in the defecation process; however, they help stabilize the pelvic floor during straining. Abdominal muscles are mainly used for voluntary straining (during defecation) to facilitate the passage of stool during constipation.

Defecation process:-

The defecation process is an amalgamation of various physiological reflexes, mechanical contribution by the sphincter mechanism and synchronous relaxation of voluntary and involuntary muscles of the anal canal. It starts with *the anal sampling reflex* consists of a regularly occurring, short-lasting relaxation within the upper anal canal, with a simultaneous contraction of the upper rectum and relaxation of the distal rectum. Thus, contents can be moved from the rectum into contact with the mucosa of the upper anal canal, and it is assumed that sensory receptors therein can determine the nature of the content (solid stool, liquid stool, or gas). After a short time, the anal pressure is normalized, and the content is forced back to the rectum (6).

Colonic mass movements bring faecal content into the rectum. This volumetric expansion of the rectum stimulates further contractions of the colon via a reflex mediated by the Enteric Nervous System (ENS) and by the *parasympathetic defecation reflex(colo-anal reflex).* This stimulates a phasic contraction and an increase in rectal tone, transforming the rectum's functional role from a capacious reservoir into a conduit.

Another reflex is the *recto-anal inhibitory reflex* (RAIR), which mediates relaxation of the I.A.S. during rectal distension. (10) It is a local reflex executed through intramural nerve fibres. It is absent in Hirschsprung's disease. The distension of the rectum stimulates the recto-anal inhibitory reflex, leading to a relaxation of the I.A.S. (7, 8). In addition, there may be a *direct colo-anal reflex*, whereby the I.A.S. is relaxed simultaneously with the colonic mass movement. (9) The pubo-rectalis muscle, which is a sling around the rectum and acts as an external valve, voluntarily relaxes to increase the angle. Simultaneously, the external sphincter also relaxes to open the anal canal. The control over the defection process is partially achieved by voluntary contraction of the E.A.S. and pubo-rectalis muscle. If this is exercised, the defecation reflex will gradually subside and the rectal compliance increases. However, the outcome depends upon the strength of reflex contraction, the volume of stool in the rectum, stool consistency, and the neurological status of the reflex mechanism of the rectum. (10)

Anal Continence and Normal Bowel Movements:-

Principally, the same factors are involved in achieving normal continence and normal bowel function. The key factors causing anal incontinence and constipation/ obstructed defecation are mechanical forces exerted by the sphincters and physiological in the form of reflexes. Mechanical lesions or atrophy of I.A.S. and or E.A.S., low volumes (low compliance) of the rectum, excessive motility of the colon, short transit time or incomplete evacuation causes *incontinence,* whereas Lack of recto-anal inhibitory reflex, high volume threshold of the rectum, high compliance/capacity of the rectum, decreased colonic motility, failure to relax pubo-rectalis muscle and slow transit/difficult evacuation causes constipation.

The commonly encountered, though benign, anal disorders have one thing in common: constipation. Constipation is invariably present in the anal fissure, haemorrhoids, and anal fistula cases; let us review the aetiopathogenesis of each.

Pathophysiology of anal disorders

Anal Fissures:-

Though the exact cause of fissure-in-ano has not been proved, the etiological relation is drawn with the anatomical configuration of sphincter mechanism, traumatic insult of the anal mucosa (chemical/mechanical) and ischemia due to poor blood supply. Generally, most patients presenting with an anal fissure relate a history of large, hard stool precipitating trauma after an attempt to defecate forcefully; sometimes, frequent bowel movements associated with diarrhoea are the initiating event. History of instrumentation like insertion of a rectal thermometer, enema tip, procto-scope used to examine the rectum or anus, or even traumatic anal intercourse is also related to the onset of fissure symptoms. (11, 12, 13).

Studies revealed that patients with anal fissures had elevated resting anal sphincter pressures when compared with normal controls. However, these studies could not conclude whether the abnormally high resting pressure resulted from an anal fissure or was predisposed to its development. (14). More recently, studies were using anal manometry and colour doppler study and demonstrated the relation of anal fissure with poor vascularity and showed that lesser blood flow to the anoderm of the posterior midline as compared to other areas of the anal canal. The study also found that a rise in resting anal pressure is directly proportional to a reduction in midline anodermal blood flow. These findings led them to postulate that ischemia may be responsible for the severe pain associated with anal fissures and may contribute to their failure to heal. (12, 14, 15, 16).

Haemorrhoids:-

Enlargement and pathological change in haemorrhoidal tissue on either side of the dentate line resulting in symptoms of the "hemorrhoidal syndrome", which includes parts or complete protrusion of haemorrhoids, fresh, voluminous and mostly painless bleeding per anus. Proposed etiological factors for these changes include constipation, prolonged straining, pregnancy, and derangement of the internal sphincter. These conditions work towards stretching and slippage the hemorrhoidal tissue due to a downward pressure against an inadequately opened internal sphincter. The overlying skin or mucosa that is partially stuck to the faecal matter gets stretched and creates a potential space for additional fibrous and sinusoidal tissue development. The extra tissue occupies space towards the anal canal cavity, and this cycle repeats itself during the subsequent defecation. This extra tissue tends to move toward the anal verge (prolapse), making it susceptible to injury (bleeding), especially during hard stool or getting trapped in the involuntary internal sphincter (acute prolapse/ thrombosed piles). (17, 18).

Anorectal Abscess and Fistula in Ano:-

As far as etiopathogenesis is concerned, I would like to keep these two disorders in one category as the common culprit appears to be chronic constipation. Fistula-in-ano and anorectal abscesses share a common cause and differ only with respect to timing. The abscess represents the acute phase, whereas the fistula represents the chronic phase of the abscess. Ninety per`` centage of abscesses results from nonspecific crypto-glandular infection. The crypto-glandular theory proposed by Parks (19) suggests that abscesses result from obstruction of the anal glands and ducts, causing the accumulation of secretions within the gland, followed by infection of the accumulated secretions and presents as follows: abscess. Either surgical treatment of abscess (incision and drainage) or spontaneous rupture may drain the abscess, but the persistence of anal gland epithelium in part of the abscess cavity between the crypt, blocked part of the duct and external opening towards skin created either surgically/spontaneous rupture) leads to the formation of a fistula. (20)

Effect of Straining on the anorectal pressure gradient

During recto-anal inhibitory reflex, which is a natural part of the defecation mechanism, maximum pressure inside the anal canal during distension and relaxation is attained up to 40 and 10 cm of H2O, respectively. However, this pressure rises to 124 cm of H2O and 97 cm of H2O during sudden and sustained

straining, respectively. (21, 22) This extra pressure exerted over the pelvic floor, and delicate anorectal musculature may cause structural disturbances. These structural disturbances are a tear in anal mucosa, stretching of overlying skin or mucosa and presenting as acute anal fissure, haemorrhoids and so on. The onset of this event is a vicious cycle that repeats the sequence and makes an acute event into a chronic one. This perpetual high resting anal pressure also interferes with the compliance of the internal sphincter and further disturbs the sphincter and reflex mechanism.

III. Discussion

This review of the literature points towards few salient and consistent features. Firstly constipation, either subjective (as perceived by the individual) or objective (as appreciated by the clinician), is the most common feature found amongst anal disorders. Secondly, straining is the common and most initial response by the individual to constipation. Thirdly the anal musculature and physiological reflex mechanism are interdependent in facilitating the primary function of the anal canal that is defecation. Fourthly and most importantly, the process of defecation is reflex dependent, and active straining is not part of it at all.

The functioning of the anal canal is based on various parameters. These are namely the characteristic of stool, bowel training, and personality of an individual (23) intactness of various anal and rectal reflexes, which in turn dependent upon the intactness of nerve pathways and central nervous system and last but not the least, local factors of anal canal like musculature, mucosa and sphincter mechanism. As we can see, management of commonly encountered anal disorders like fissure and haemorrhoids is targeted towards the local factors of the anal canal and partly addressing constipation in a particular case. The chronology of events in the defecation process is an essential part of managing anal disorders. Most of the disorders arise due to constipation; however, the term constipation itself needs to be well understood before labelling it as a symptom. Though the definition of constipation involves frequency and consistency of defecation as mentioned by the patient, it does not address the details of the defecation process.

At the time of presentation, most of the patients are already suffering from constipation and used straining to relieve constipation. Poor understanding of the defecation process, inability to relax sphincter mechanism, changes the bowel schedule, which is considered constipation. This prolongs the transit time of faecal matter and makes it dry and hard. Hence, this mild disturbance in bowel schedule is perceived as constipation by the individual, who later resorts to straining while defecation only to end up causing fissure/haemorrhoids or aggravating those already there.

The perception of rectal fullness and the process of straining to empty the rectum appears to be the main culprit for anal disorders, mainly fissures and haemorrhoids. Let me explain this. As mentioned above, the defecation process is dependent upon many reflexes and synchronization of each event that completes the defecation. Hence, there is no role of straining in defecation. The majority of the process is dependent on the contraction of the smooth muscles and intact reflex mechanisms. The voluntary contraction of the abdominal musculature may increase the intra-abdominal pressure, but it does not act selectively on the faecal matter only. Straining causes a rise in intra-abdominal pressure and intra-rectal pressure, which is against the closed internal sphincter. The I.A.S. is under involuntary control and fails to relax. This pressure is transferred all over the pelvic floor, and it is the weakest point that is the anal canal. This 2-3 times extra pressure is significant enough to inflict structural damage to soft tissue in the anal canal. Prolonged and repeated straining causes chronic rise in venous pressure in the pelvic region and prolapse of the sinusoidal venous plexus in the additional space created in the anal mucosa due to stretch. This chronic rise in pressure in the anal canal also pushes faecal matter in the anal glands at crypts. This retrograde spread of faecal matter and subsequent infection of the anal gland causes the formation of anal/ anorectal abscesses and fistulas. The persistent rise in pressure gradient in the anal canal does not allow the fistula tract to heal as the pressure within the canal ensures unidirectional flow in the track and does not allow it to heal.

IV. Conclusion

Anal disorders are the most common entities in a routine surgical out-patient department. Constipation forms a common symptom across common disorders like fissure, haemorrhoids and fistula and management is directed towards anatomical defects. As defecation depends on the integrity and synchronization of the reflex mechanism (in addition to the consistency of stool), treatment only for consistency may not be very effective. Straining during defecation is a typical response by individuals. It creates a significant rise in intra-rectal/intra-anal pressure causing structural changes in the soft tissue of the anal canal. This forms the basis of the formation of the fissure (traumatic), haemorrhoids and fistula-in-ano. However, there is a study requirement to see the exact prevalence of straining amongst the community and its relevance with the precipitation of anal disorder. It is also necessary to establish the relation between straining and the healing process of a particular anal disorder.

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