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REVIEW

Descending perineum syndrome: new perspectives

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Abstract The classical clinical profile of descending perineum syndrome (DPS) has been replaced by new pathophysiological, diagnostic, and therapeutic acquisitions. This paper will focus on trigger factors ranging from dyssynergic defecation to excessive straining, fecal incontinence against the backdrop of obstructed defecation, attendant rectal diseases, and therapy tailored to evolving stages of DPS.

Keywords Descending perineum syndrome · Obstructed defecation · Fecal incontinence · Pelvic floor

Introduction

As originally described by Parks et al. [1], descending perineum syndrome (DPS) is characterized by the ballooning of the perineum several centimeters below the bony outlet of the pelvis during straining. The classic clinical picture of DPS is usually limited to the proctologic compartment progressing from dyschezia to fecal incontinence: Obstructed defecation, often present for many years, evolves into impairment of fecal continence, considered as a late sign of the syndrome [2]. However, it is rational to think that perineal descent may simultaneously involve the anterior, middle, and posterior pelviperineal areas in women. Urogynecological structures and proctologic segments are thus all implicated [3, 4]. The report of complex anamnestic data derived from the simultaneous presence of urogynecological and proctologic symptoms obliges proctologists to evaluate the perineum as a whole, in search of rectal diseases associated with urogynecological diseases such as pelvic organ prolapse (POP).

Early trigger factors such as dyssynergic defecation, surfacing of fecal incontinence against the backdrop of obstructed defecation, attendant rectal and urogynecological diseases, and therapy tailored to DPS stage are all crucial new points that proctologists must be aware of in order to correctly diagnose and treat the DPS patient.

This paper will focus on new insights into the pathophysiology, clinical evolution, diagnosis, and therapy of DPS.

Pathophysiology

The term DPS is mainly descriptive since perineal descent upon straining is both the cause of the symptoms and the most obvious physical sign. Perineal descent, according to Park's definition, refers only to the superficial perineal plane, externally visible, but it is obvious that this external perineal descent is joined to the descent of the deep perineal plane, with the result that the anatomical muscular excursion is more complex and serious and involves the whole pelvic floor.

Excessive abdominal straining during bowel movements has been identified as the cause of progressive perineal descent: The recurrent straining against outlet obstruction impairs pelvic floor muscle tone until it disappears completely [1]. The whole pelvic floor descends, due to high intra-abdominal pressure, and becomes funnel shaped due to stretching of the puborectalis muscle. The postdefecation reflex is also impaired: Usually, a sharp contraction of the

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levator ani muscles repositions the pelvic floor after it drops during bowel evacuation, but excessive, straining-induced, muscular stretching impairs reflex muscular contractions and repositioning occurs when muscles become lengthened and loose. In this way, the perineum descends more and more as testified by levator ani muscle and ligament dysfunction. The decline in normal levator ani tone, induced by overstretching, results in an open urogenital hiatus, weakening of the horizontal orientation of the levator plate, and a bowl-like configuration [5]. Such anatomical arrangements are seen also in women with POP, and they are considered risk factors for impaired support for pelvic viscera [6]. In fact, the ballooning of the levator hiatus and increase in the levator plate angle are imaging findings characteristic of pelvic floor impairment in POP [7, 8]. The descending perineum is first mobile when the pelvic floor is in a normal position at rest, and then, it descends >3 cm during straining and bowel evacuation and afterward returns to its initial position. The descending perineum becomes fixed if defecography at rest shows a pelvic floor descent >3 cm which increases several centimeters during straining and evacuative maneuvers, and returns slowly or not returning to the starting line. Defecography images clarify the pathophysiology. A mobile descending perineum shows, at straining, a paradoxical puborectalis contraction that is simultaneous with excessive pelvic floor descent [5, 9]. Obstructed dyssynergic defecation occurs, and the patient triggers a more exaggerated effort to evacuate, an effort which then worsens perineal descent. The result is an excessive descent of the pelvic floor that becomes funnel shaped: Fecal material is forced out by straining vector powers inside the rectum which is deformed by this external muscular imprint. The rectum tries to empty itself but becomes invaginated resulting in first rectorectal and then rectoanal intussusception with a funnel-shaped infolding <10 mm. A fixed descending perineum, on the contrary, has lost its primary dyssynergic puborectalis contraction: The resting pelvic floor has a low basal position because the levator ani muscle has become atonic, and evacuative maneuvers worsen the situation by deepening the descent. The result is displacement rectocele [10] and/or rectoanal intussusception showing an infolding >10 mm [10]. Pathophysiology clarifies the multifaceted clinical aspects of DPS. The dyssynergic component plays a role in the early stages of the disease, inducing obstructed defecation. The organic descent of the hypotonic pelvic floor, combined with disease duration, advancing age, pudendal neuropathy, and sometimes uterine surgery, such as abdominal hysterectomy, explains the appearance of fecal incontinence [9, 11, 12]. Pudendal neuropathy is a pathophysiological sign of DPS. Recurrent trauma due to stretch injury to the pudendal nerves can occur during perineal descent, and this can lead to denervation and weakness of the external anal sphincter muscle [13, 14]; fecal incontinence may appear. Usually, fecal incontinence is the result of severe neurogenic damage in those patients with a long history of straining and this explains why fecal incontinence is a late sign of DPS. In some patients, rectal prolapse may be the last step [15, 16], because rectoanal intussusception may evolve into rectal prolapse, as suggested by several factors. Common findings in rectoanal intussusception and rectal prolapse include fixed descending perineum with anatomical derangement of the pelvic floor, radiological reports [17, 18] supported by Oxford Grading of rectal prolapse [19], similar manometric findings [16], pudendal neuropathy [20, 21], and, last but not least, a long history of impaired defecation. In particular, the pelvic floor in rectal prolapse exhibits diastasis of the levator ani hiatus, loss of posterior rectal fixation, and loss of a horizontal distal rectal segment, all anatomical derangements which are an expression of pelvic floor derangement.

Clinical notes

Perineal descent involves the anterior, middle, and posterior pelviperineal areas in women. Urogynecological structures and proctologic segments are all implicated, and thus, POP may coexist with rectoanal intussusception, rectal prolapse, and rectocele [22]. A mix of obstructed defecation, fecal incontinence, and urogynecological symptoms may occur in varying degrees in relation to disease stage [4, 23]. Straining at stool, a sensation of incomplete evacuation and of anorectal obstruction/blockage, manual maneuvers to facilitate defecation, and loss of solid or liquid stool are mixed with vaginal symptoms (sensation of a bulge, heaviness) and urological symptoms (urinary incontinence, urgency, hesitancy, feeling of incomplete emptying). Several factors are associated with mobile or fixed DPS in women: aging, the number of vaginal deliveries and obesity [24]. All these conditions have a common denominator: impairment of the connective tissue network that envelops all organs of the pelvis and connects them loosely to the supportive pelvic musculature and pelvic bones. It is obvious that the result is a malfunctioning pelvic floor.

Diagnosis

Although a wide array of functional and morphologic disorders can be responsible for the signs and symptoms of DPS, a precise diagnosis is necessary before any therapeutic decision is made. A correct, objective, perineal examination must be combined with morphologic and functional diagnostic tests in order to have useful therapeutic information.

Objective perineal examination

The perineum must be evaluated as a whole using the Sims and lithotomy positions: Inspection of the anus and digital rectal examination must be associated with the examination of the external genitalia and vagina. POP must be graded by means of POP quantification [25], and rectal diseases must be evaluated in the context of a descending perineum with the possibility of associated urogynecological diseases. The old "perineometer," a device designed by Henry et al. [26] to provide objective measurement of perineal descent, and the new Perineocaliper® (Duchateau SA, Liège, Belgium) are complicated and impractical. Perineal descent can be measured clinically by the POP-Q system: The accompanying increase in the sagittal length of the levator hiatus and the distance from the urethra to the anus, such as detected in DPS, are approximated by the gh + pb parameter [25]. The parameter is strongly related to straining ($p < 0.01$) [23], and this confirms the pathophysiological importance of straining in DPS women.

Morphologic evaluation

A radiological diagnosis of DPS revolves around new imaging techniques. Dynamic pelvic magnetic resonance imaging (MRI) and transperineal ultrasound may be used in addition to traditional defecography.

Defecography

Descending perineum is identified when, on examining the patient in the left lateral position, the anal canal rapidly descends >3 cm when a straining effort is made [1]. Anorectal angle and pelvic floor descent values are significantly higher at rest and during evacuation ($p < 0.01$) in DPS patients than in controls [9]. Rectocele and rectoanal intussusception may be detected, and some DPS patients have a poor anorectal angle opening at evacuation. Puborectalis indentation is also a defecographic sign in about 40 % of DPS patients with obstructed defecation, confirming the hypothesis that pelvic floor dyssynergia may be a pathophysiological factor for mobile DPS. A recent paper has shown that constipated patients with an abnormal perineal position were more likely to have rectal intussusception and enterocele and that they also were older, had a significantly high hysterectomy rate and high body mass index when compared to constipated patients without descending perineum [27].

Colpocystodefecography was introduced in order to overcome the limitations of defecography which studies only the proctologic pelvic district [28]. Colpocystodefecography combines vaginal opacification, voiding cystography, and defecography. It assesses the entire female pelvis, including the pouch of Douglas, in one single procedure, by providing a view of the whole pelvis and simultaneously of the movements of pelvic viscera. Any associated urinary, genital, or anorectal abnormalities may be evidenced in women with DPS [22].

MRI

MRI is an excellent tool for understanding the complex anatomy of the pelvic floor and for assessing pelvic floor disorders. The urethra, uterus, vagina, rectum, anal canal, pelviperineal muscles, perineal body, and supportive elements of the endopelvic fascia are all easily identified. Static MRI provides imaging of the morphology of the pelvic floor with the patient at rest. Dynamic MRI means that the pelviperineal contents are imaged dynamically, i.e., with the pelvic floor at rest, during squeezing, straining, defecation, or urination. It offers a perfect image of what is happening in women affected by DPS. The HMO system was developed for grading pelvic floor abnormalities: It distinguishes between pelvic organ prolapse and pelvic floor relaxation, distinct but often coexisting pathologies [29]. The degree of pelvic floor relaxation is measured with two reference lines: the H line, which represents hiatal widening and extends from the inferior plane of the symphysis pubis to the posterior wall at the anorectal junction, and the M line, which represents hiatal descent and extends perpendicularly from the pubococcygeal line to the posterior end of the H line. The H and M lines tend to elongate with pelvic floor relaxation, representing, respectively, levator hiatal widening and levator plate descent, and both markers of DPS, when altered, may be easily detected.

Transperineal sonography

Pelvic floor anatomy may be ascertained by means of transperineal sonography (TS) [30]. Standard images of the pelvic floor are obtained during resting state, squeezing, and straining from longitudinal and axial planes by placing the transducer directly on the perineal body between the vagina and anus; the standard measurements are taken from the final fixed images. The biggest limitation of transperineal sonography is the absence of defecation and micturition, but it is an initial outpatient diagnostic imaging modality that gives useful information about the anal sphincters, bladder neck position, and levator function. A recent study stated that multicompartment TS identifies more conditions than those diagnosed through clinical urogynecological assessment but it neither changes the initial surgical management nor the management at 1-year follow-up, and therefore, TS should not be substituted for clinical assessment [31].

Functional evaluation

After a morphologic evaluation, it is necessary to have a functional evaluation that may give useful information about anorectal function.

Anorectal manometry

In DPS patients, anorectal pressures are impaired. DPS women with obstructed defecation have anal pressure values similar to those of the controls, whereas patients with fecal incontinence have lower anal pressure values than those with obstructed defecation and controls [9]. These data underline the progression of defective anal sphincter function which is significantly related to fecal incontinence. A significant impairment of the amplitude of maximal voluntary contraction may be detected in DPS women, independently from defecation disorders, when compared to controls ($p < 0.001$) [9]. It is the obvious expression of the negative influence of pudendal neuropathy on external anal sphincter function. A recent paper demonstrated that three-dimensional high-resolution anorectal manometry, in addition to the usual measurements of anal canal and rectal functions, can simultaneously provide topographical data [32]. Excessive perineal descent may be detected, defined as the downward movement of the anal high-pressure zone during straining. There is a high positive correlation between defecographic and manometric measurements (Spearman's correlation: 0.726).

Neurophysiological tests

Fecal incontinence is an anorectal disorder relevant for neurophysiological examination. External anal sphincter electromyography, motor-evoked potentials, somatosensory evoked potentials, and sacral anal reflex latency measurement are currently available to evaluate neurogenic anorectal disorders. Sacral reflexes can be used to investigate sensorimotor pathways within the S2–S4 segment of the spine [33], a segment that is the origin of the pudendal nerve. Excessive perineal descent can result in an increase in the length of the pudendal nerves by as much as 20 %, an amount sufficient to cause neuropathy. The relationship between perineal descent and pudendal nerve damage has been proved [13, 34], and the traditional "entrapment and stretch" theory of pudendal neuropathy has been supported [14]. Sacral reflexes are altered by pudendal nerve lesions: For this reason, to understand the neurophysiological significance of perineal descent, sacral anal reflex latency measurement is mandatory in DPS patients with fecal incontinence [35].

Therapy

DPS therapy is not well defined because of the separation between gynecological and proctologic specialists: Each group deals with a single pelvic area, and there is no complete DPS overview. Therefore, therapy is often administered as part of the individual competence of a single specialist, whereas occasionally a beneficial effect may be obtained from treating adjacent pelvic/perineal districts. This helps to explain why there is no standard DPS therapy that is universally accepted.

Therapy, restricted to the proctologic area, must be tailored on the DPS stage. Rehabilitative treatment using biofeedback and pelviperineal kinesitherapy may be considered the first-line option in treating mobile DPS in patients with symptoms and signs of obstructed defecation who have not responded to simple dietary change or medication. Pelviperineal kinesitherapy is a type of muscular training that is selectively aimed at the levator ani muscles. Biofeedback is an operant conditioning method for the defecation reflex, which consists of pelvic-floor-strengthening exercises together with visual/verbal feedback training. Rehabilitative results can be good, and the patient's obstructed defecation syndrome score can improve significantly after rehabilitation [36]. The extent of perineal descent appears to be the predictor of response to retraining because patients who respond to pelvic floor retraining and biofeedback have less perineal descent (mean 3.3 cm) than those who do not respond (mean 4.9 cm) [4]. Literature data suggest that rehabilitative treatment can also play a role in DPS patients with symptoms of fecal incontinence: The continence improves, and the Wexner incontinence score significantly decreases [37, 38]. However, some patients do not respond to rehabilitation and such failures may depend on both the disease stage and the severity of combined rectal disease. Therefore, a surgical option might be necessary.

Surgery, for fixed DPS and rehabilitation failures, should be aimed at correcting pelvic floor impairment. The correction is made directly by means of levatorplasty techniques or indirectly by surgical procedures involving mesh or resection that are aimed at rectal diseases. In 1982, Nichols used a retrorectal levatorplasty combined with colporrhaphy to treat an uncommon type of genital prolapse characterized by the descent of the anus and sagging of the levator plate associated with severe constipation [39]. The results were fair, but this was an isolated report. After several years, Boccasanta et al. [40] combined a single-stapled transanal prolapsectomy with perineal levatorplasty, concluding that it was a satisfactory treatment for patients with descending perineum,

intussusception, and rectocele. More recently, Beco proposed retroanal levator plate myorrhaphy (RLPM) in selected cases of DPS with a positive anti-sagging test [3]. Levator plate myorrhaphy is carried out between the coccyx and anorectal junction until sagging is eliminated (checked by rectal examination).Usually, two to four stitches (with some "figure eight" stitches if possible) are necessary to completely suppress the sagging. The objective evaluation of the position of the levator plate and anal margin at rest and during straining after RLPM showed that the reduction in perineal descent was about 1.08 cm and that obstructed defecation and fecal incontinence improved in 87.5 % of patients. Also, all the urogynecological symptoms associated with DPS (stress urinary incontinence, frequency, urgency, dysuria, dyspareunia, and perineodynia) were cured. Nevertheless, there are no randomized trials conducted on levatorplasty techniques. In the past few years, the target of surgery has not been the pelvic floor but the attendant rectal disease. Surgical procedures involving mesh or resection correct only rectal diseases, and repositioning of the rectum indirectly causes the descending perineum to rise. Laparoscopic ventral mesh rectopexy has gained popularity in Europe as a treatment for full-thickness external and internal rectal prolapse. This procedure has been shown to achieve acceptable anatomical results with low recurrence rates, few complications, and improvements in both constipation and fecal incontinence [41, 42]. Recently, Lundby et al. commented on this technique saying that: (1) Only level 3 evidence exists; (2) special attention should be paid to possible mesh-related complications and long-term sequelae that could have a significant impact on quality of life; and (3) a clear correlation between surgical correction of the anatomical abnormalities and improvement in obstructed defecation syndrome has not been demonstrated [43]. He concluded that it is time for a critical appraisal. Laparoscopic Pelvic Organ Prolapse Suspension (POPS) has recently been suggested for women with genital prolapse and obstructed defecation syndrome [44]. Preliminary results of this technique have shown that treatment of POP is successful and that the patient's obstructed defecation score fell significantly. However, there are no randomized trials that actually validate this technique. Resective surgery (stapled transanal rectal resection (STARR)-TRANSTAR [45], Delorme operation [46]) is used for rectoanal intussusception and rectocele. Fecal incontinence and obstructed defecation are significantly reduced. Only one randomized trial compared Delorme to STARR: The overall incidence of postoperative complications was low and similar between the two groups of patients, and a significant improvement in symptoms was obtained with both techniques [45].

Conclusions

The profile of DPS has changed a lot since the original description by Parks. New pathophysiological reports suggest a possible evolutionary pathway in obstructed defecation: Pelvic floor dyssynergia might evolve with age into DPS, and fecal incontinence could be the last step. The diagnostic approach must be tailored on different anamnestic and objective clinical remarks. Therapy is oriented to conservative management or surgery on the strength of accurate definition of DPS stage and concomitant diseases. Only by conducting large randomized trials will we learn whether rehabilitative and surgical procedures stand the test of time and have a permanent role in the therapeutic armamentarium for DPS.

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval This paper is a review and not research involving human participants and animals.

Informed consent Informed consent was obtained from all individual participants included in the study.

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