Female Sexual Dysfunction: Medical and Surgical Management of Pelvic Pain and Dyspareunia

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Podcast: AUA on female sexual dysfunction

1. Introduction

With the evolving language regarding gender identity and sexual orientation, the authors feel it is important to first define gender and sex. Gender refers to the attitudes, feelings, and behaviors that a given culture associates with a person's biological sex. Sex is defined as either of the two main categories (male and female) into which humans and most other living things are divided on the basis of their reproductive functions. The authors of this chapter will use the terminology “Female Sexual Dysfunction” as defined in the DSM-V [1] to describe anyone assigned female at birth or anyone with female internal or external genitalia.

Genito-Pelvic Pain/Penetration Disorder (GPPPD), previously known as dyspareunia, is defined as persistent or recurrent symptoms with one or more of the following for at least 6 months: marked vulvovaginal or pelvic pain during penetrative intercourse or penetration attempts, marked fear or anxiety about vulvovaginal or pelvic pain in anticipation of, during, or as a result of penetration, and marked tensing or tightening of the pelvic floor muscles during attempted vaginal penetration. **Sexual pain disorders[1] of the vulva and vagina are quite common and are within the scope of a general urology practice, particularly since many urologic procedures may include sexual pain as potential adverse events.**

There are many organic causes of vulvovaginal sexual pain. In this manuscript we will discuss 4 common causes of dyspareunia and genital pain.

- Genitourinary syndrome of menopause (see AUA Core Curriculum: **Genitourinary Syndrome of Menopause**)

- Vulvodynia/Provoked vestibulodynia
- Clitorodynia/Clitoral phimosis
- Pelvic Floor Dysfunction/Hypertonic pelvic floor muscles (previously known as vaginismus)

1.1 Epidemiology

Prevalence estimates for sexual pain in cisgender women ranges from 7-32%. The wide range of values is driven in part by age-related changes and differences in how questions on sexual pain are asked.[2,3,4,5]

Within a biopsychosocial model of disease, dyspareunia can be associated with an array of psychological factors in addition to biological factors. Psychological factors that may result from or contribute to dyspareunia include anxiety or guilt about sexual activity and/or intercourse, distress related to past sexual experiences, inexperience and lack of sexual education around adequate pre-coital stimulation or positioning, fear of pain with penetration, and severe stress. It is important to note that sexual pain is not always a result of psychological issues or sexual trauma even when visual inspection of the vulva does not reveal obvious pathology. [6,7] Currently, there is limited information regarding the prevalence amongst racial groups.

2. Genitourinary Syndrome of Menopause (GSM)

2.1 Epidemiology

With menopause, loss of estrogens and androgens leads in most cases to vaginal and vulvar tissue atrophy. This causes not only genital symptoms (e.g. dryness, burning, and irritation) and sexual symptoms (e.g. lack of lubrication, discomfort or pain, and decreased libido and difficulty with arousal and orgasm), but also urinary symptoms (e.g. urgency, dysuria, and recurrent urinary tract infections).These symptoms are known as the Genitourinary Syndrome of Menopause (GSM), a term which has replaced the earlier used vulvovaginal atrophy and atrophic vaginitis. Symptoms increase in severity over time, and do not improve without treatment. [8,9,10,11] While GSM is associated with the changes that occur during menopause, it can also occur with other hormonally depleted states including patients who are breast feeding, taking oral contraceptives, receiving adjuvant hormonal deprivation therapy for breast cancer or gender-confirming treatment, and women with thyroid disorders or pituitary tumors.

2.2 Diagnosis

On exam patients may have thinning of the pubic hair, the labia majora can appear flattened and the labia minora may be resorbed or fused. The vaginal introitus may be narrowed limiting speculum exam and the vaginal epithelium can become pale and friable with loss of normal rugae.[12] See **Figure 1**

As the surrounding tissue atrophies, the urethra often protrudes. The urethra may develop a caruncle which is a portion of the urethral tissue extending beyond the meatal opening, a polyp or a full prolapse. Friction against the urethral tissue may cause pain with penetration. This can be managed

conservatively with vaginal estrogen and if not resolved the prolapsed portion of urethra may be excised. See **Figure 1**

Vaginal pH testing may be used to confirm the diagnosis and follow treatment. Normal vaginal pH ranges from pH 3.5-5.0. Vaginal pH levels often reach 5.5 or higher in women with GSM.[12]

2.3 Treatment

2.3.1 Topical

There are a range of treatment options for GSM. Vaginal moisturizers can be purchased over the counter and have been shown to maintain tissue integrity, elasticity, and pliability with regular use.

Vaginal lubricants are used “as needed”, typically for sexual activity, to temporarily alleviate vaginal dryness and prevent dyspareunia. Lubricants may have a water, silicone, or oil base. Water-based lubricants are effective but may require repeat applications as they may evaporate/absorb. Silicone-based lubricants are less prone to drying out but may be harder to clean and may have an unpleasant taste/odor. Oil-based lubricants may be effective but should not be used with latex condoms as the oil content will tend to lead to condom breakage. Ultimately the choice of lubricants should be based on patient and partner preference and may require experimentation.

Table 1 shows all the FDA approved options for treating GSM. Topical low-dose vaginal estrogen works locally to restore hormone levels within the tissue without significant systemic absorption. This typically results in rapid improvement of vaginal symptoms (vaginal dryness and/or dyspareunia) within 2 to 3 weeks.[13,14] Local vaginal estrogen is also recommended in the **AUA guidelines for recurrent UTI** in peri- and post-menopausal women who do not have a contraindication to estrogen therapy. Patients with active breast or endometrial cancer and those with a history of those cancers who are still on hormone depletion therapy should be discussed with the treating oncologist. The Women’s Health Initiative study showed the risk of cardiovascular complications and breast cancer increased in older women with combined systemic hormonal therapy but decreased in younger women who were close with menopause with systemic estrogen therapy alone.[15] Vaginal estrogen use was not associated with any increased risks of cancer or cardiovascular outcomes over an 18-year follow-up period.[16]

Vaginal DHEA suppositories are converted by enzymes in the vulva, vestibule and vagina to become estrogens and androgens while not affecting the endometrial tissue in the uterus. Vaginal DHEA suppositories are specifically approved for treatment of dyspareunia due to GSM and result in improvement in all domains of sexual function.[17]

Some small studies, which have included breast cancer patients not eligible for vaginal estrogen therapy, have shown topical vaginal testosterone may reduce dyspareunia and improve sexual function.[18,19]

2.3.2 Oral

Ospemifene is a selective estrogen receptor modulator (SERM), a systemic non-hormonal therapy

approved for the treatment of dyspareunia due to vulvovaginal atrophy. This is particularly helpful for patients who prefer an oral versus vaginal treatment. In clinical trials, ospemifene reduced pain with sexual intercourse and increased vaginal mucosal maturation as well as vaginal pH. Contraindications include estrogen-dependent neoplasia, active or prior venous thromboembolism, previous stroke, and active heart disease or prior myocardial infarction. The most commonly reported adverse reactions from ospemifene were hot flushes, vaginal discharge, and muscle spasms. This medication has not been adequately studied in women with breast cancer. [20]

Systemic estrogen therapy may also be used in the treatment of GSM when a patient has concomitant vasomotor symptoms, although patients may also need local estrogen replacement.[21]

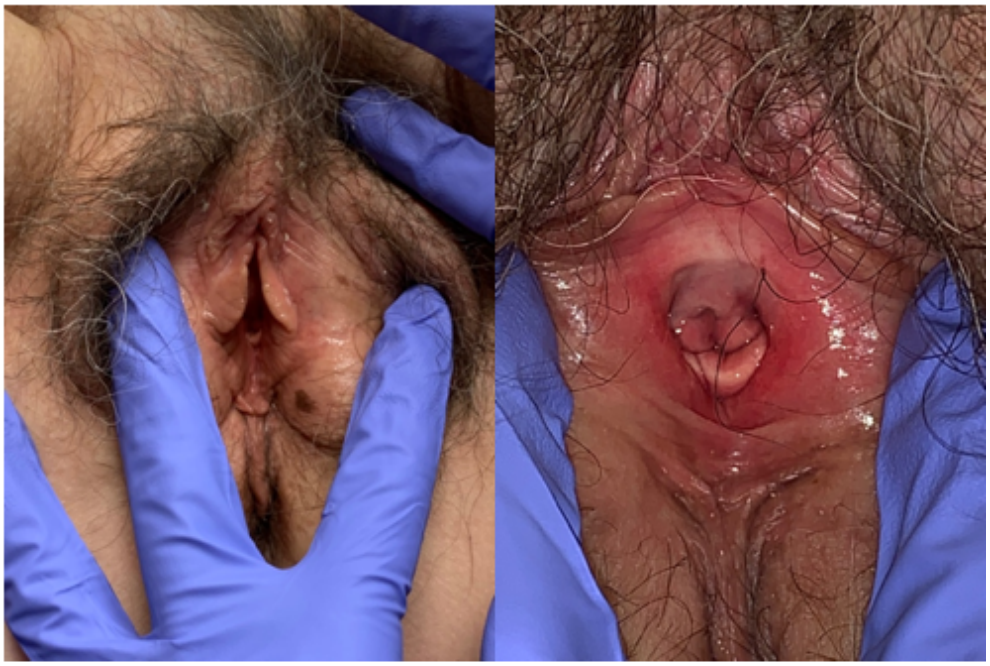
2.3.3 Non-medication treatments

Once a patient begins treatment for GSM she may benefit from pelvic floor physical therapy as well as regular use of vaginal dilators. Proper dilator use has been found to reduce pain with vaginal penetration by improving vaginal elasticity[22] and decreasing pain. Dilators are used in a sequential fashion and can most effectively be taught with the help of a subspecialized pelvic floor physical therapist. If specialized training is not possible, there are a number of internet guides available to help patients trying to learn dilator therapy.
https://www.mskcc.org/cancer-care/patient-education/how-use-vaginal-dilator.

CO2, Erbium laser and radiofrequency (RF) devices are thought to improve vascularization and connective tissue within the vaginal canal. They have been used to treat vaginal atrophy, dyspareunia, and vaginal laxity, however the FDA issued a recent notification urging caution in marketing these devices ahead of the scientific evidence. [23,24,25] A recent study looked at 6 month follow up after CO2 laser compared to use of conjugated vaginal estrogen cream. Results showed 85.8% of laser participants rated their improvement as “better or much better” and 78.5% reported being either “satisfied or very satisfied” compared to 70% and 73.3% in the estrogen group, respectively. The difference was not statistically different. [26]

Table 1: Pharmacologic treatments for genitourinary syndrome of menopause (GSM)

Treatment	Product Name	Dose
Vaginal Cream		
17-beta- estradiol cream	Estrace, generic	0.5-1 gm daily for 2 weeks then 0.5-1gm 1-3x per week
Conjugated equine estrogens cream	Premarin	0.5-1 gm daily for 2 weeks then 0.5-1gm 1-3x per week
Vaginal Inserts		
Estradiol vaginal tablets	Vagifem®, Yuvafem®,	10 mcg inserts daily for 2 weeks and then 2x per week
Estradiol soft gel capsules	ImVexxy®	4, 10 mcg inserts daily for 2 weeks and then 2x per week
DHEA (prasterone) inserts	Intrarosa®	6.5 mg capsules daily
Vaginal Ring		
17-beta-estradiol ring	Estring®	1 ring inserted every 3 months
SERM		
Ospemifene oral tablets	Osphena®	60 mg tablet daily



A B

Figure 1A: The 50% resorbed and thin labia minora. Figure 1B. None the protruding urethra, pallor and erythema of the vestibule. Patient's vaginal pH was 7.5 while the goal should be 4.5. Photos courtesy of Rachel S. Rubin MD.

3. Vulvodynia / Vestibulodynia

Provoked Vestibulodynia (PVD), previously known as vulvar vestibulitis syndrome (VVS), is a condition in which sexual pain originates from the endodermal embryologic tissue called the vestibule. Typically, patients will describe entrance dyspareunia but can also manifest as vulvar itching, burning, or discomfort. The vestibule is the mucosal surface circumscribing the opening to the vaginal canal, extending from Hart's line to the hymen, and it includes many gland ostia and the urethral meatus. The most common pathologies associated with PVD are an increased density of nerve endings (neuroproliferative) in the mucosa of the vestibule and hormonally mediated tissue changes [27]. Hormonal changes may result from infertility medications, treatment for endometriosis, surgical oophrectomy, and estrogen-containing hormonal birth control (pills, patches, vaginal rings) in pre-menopausal women.[27]

PVD can also be caused by hypertonic pelvic floor dysfunction (PFD) which occurs when the striated muscles that surround the vagina, urethra, and anus are in continued contraction (high tone)/spasm. Hypertonic pelvic floor muscles can also cause generalized genital, buttock, and pelvic pain; urethral pain; constipation; rectal fissures; urinary frequency, urgency, hesitancy, and incomplete bladder emptying. [27] See Section 4 below.

3.1 Diagnosis

PVD (as its name suggests) must be provoked by a stimulus to replicate the pain sensation.

This distinction between provoked pain and unprovoked pain is important in localizing the pathology to the vestibule itself versus a constant pain caused by a more proximal nerve disorder.

A simple diagnostic tool is a cotton swab test performed during vulvar examination. While a patient is in stirrups or with adequate exposure of the entire vestibule, a moistened cotton swab is used to touch the vestibular mucosa in a clockface pattern. A pain score out of 10 is solicited for each position along the clockface, and erythema or visible gland ostia are noted for each position. It is critical to examine the 12 o'clock region below the clitoris and above the urethral meatus as this area is included in the vestibule and may be a source of significant provoked pain.

Pain elicited in only the posterior vestibule (from 5 o'clock to 7 o'clock) and that is not present in the remainder of the vestibule may be due to pelvic floor muscle hypertonicity and is unlikely to represent a neuroproliferative or hormonally-mediated vestibulodynia.

3.2 Treatment

The treatment of dyspareunia depends on identifying the underlying cause. In the majority of cases, the pain is multifactorial and hence a multidisciplinary biopsychosocial approach is preferred. Sex therapy and/or cognitive behavior therapy can be useful in patients who have anxiety or other psychological issues related to their dyspareunia. Treatment modalities may include pelvic floor physical therapy, biofeedback, manual and electrical muscle stimulation, topical agents like anesthetics or capsaicin, pelvic floor botulinum toxin injections, pudendal nerve blocks, and pudendal neuromodulation. [6] A multimodal approach to therapy is often beneficial. Consideration should also be given to discontinuation of estrogen-containing hormonal contraceptives in women who are using these and in whom these may be a contributing factor. Discussion about options for progesterone only or non-hormonal versions of contraception as well as local compounded testosterone 0.1% and estradiol 0.03% in a methyl cellulose base are the mainstays for hormonally-mediated vestibulodynia. [28] An ultra-potent topical steroid clobetasol 0.05% ointment may be used with adequate patient education for proper application in vulvar dermatological conditions such as lichen sclerosus. [28]

In refractory cases, surgical intervention may be considered. Vestibulectomy surgery may be considered in cases of severe PVD with suspected neuroproliferation and increased density of nerve endings; this surgery involves complete vestibulectomy with right and left anterior repair and posterior repair with vaginal advancement flap for excision of inflamed, hypersensitive glandular tissue and increased nerve density in the vestibular tissue (**Figure 2**).[29]

This procedure was demonstrated to be highly effective in a randomized trial compared to both biofeedback and cognitive behavioral therapy.[30] Complete vestibulectomy should not be attempted without a complete understanding of vestibular anatomy and surgical technique; the procedure should only be performed by highly trained and experienced surgeons.

Figure 2: Complete vestibulectomy with left and right anterior vestibulectomy and posterior vestibulectomy with vaginal advancement flap reconstruction

Recommend AUA podium session video: **Complete Vestibulectomy: Surgical Technique and Operative Outcomes**

4. Clitorodynia / Clitoral Adhesions / Clitoral Phimosis

The clitoris is the anatomical homologue of the penis. Like the penis, the clitoris has a prepuce, glans, coronal sulcus, corporal bodies, and crura. However, literature focusing on the clitoris and its associated pathologies is minimal compared to the penis.

4.1 Epidemiology

It has been reported that clitoral phimosis may be present in 22-33% of people with a vulva and may cause clitorodynia, anorgasmia, muted orgasm, or aversion to touch.[31] Risk factors included a history of sexual pain, yeast infection, urinary tract infection, inadequate education regarding hygiene practices, blunt perineal or genital trauma, lichen sclerosus, low calculated free testosterone, and other sexual dysfunctions including persistent genital arousal disorder.

4.2 Diagnosis

Physical exam may show an adhered clitoral prepuce to the glans. Severity of the adhesions can be described as mild, moderate and severe. Symptoms of pain or hyposensitivity should be elicited. Signs of thick, white lichenoid changes should be identified and biopsied if needed.

4.3 Treatment

Treatment includes aggressively managing lichen sclerosus (when biopsy proven) with potent topical

steroid clobetasol 0.05% ointment and traction to reduce phimosis.

Surgical treatment includes office-based lysis of adhesion after local anesthetic (**Figure 3**), or surgical dorsal slit procedure.

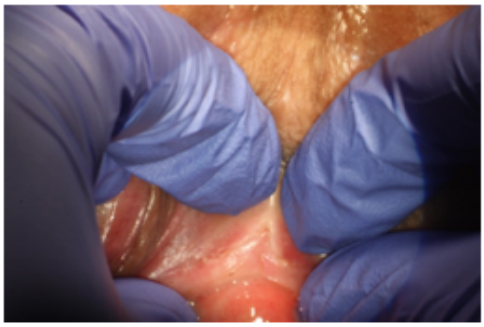


Figure 3A Phimosis of the clitoris.

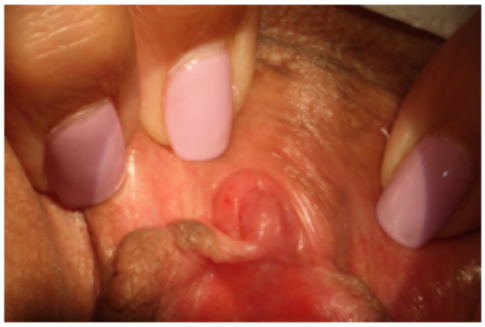


Figure 3B post clitoral lysis of adhesions

Figure 3: Photos courtesy of Rachel Rubin MD

5. Pelvic Floor Dysfunction

5.1 Introduction

The pelvic floor encompasses all structures within the pelvic cavity, including the muscles.[32] Much like any other muscle group in the body, these muscles can become hypertonic and spastic. Muscle tension decreases the range of motion of the muscle body, leading to “poor contractile force followed by slow incomplete release of tension” and causing disability and pain.[33] Pelvic floor dysfunction (PFD) refers to the constellation of symptoms secondary to the nonrelaxing pelvic floor musculature. PFD presentation depends on the particular muscles in spasm. Manifestations include dyspareunia, voiding dysfunction, incontinence, urinary retention, constipation, and dyschezia. Because of this variable symptomatology, PFD masquerades under many names, based on the organ or function primarily affected: levator myalgia, myofascial pelvic pain syndrome, coccygodynia, urethral / anal sphincter dyssynergia, vaginismus, proctalgia fugax and pelvic floor spasm. [34] All refer to the nonrelaxing (or high-tone) pelvic floor.

5.2 Epidemiology

The prevalence of PFD is estimated between 14 and 78% of women presenting with chronic pelvic pain.[35] Dyspareunia occurs in 63-71% of patients with PFD.[36,37] Many mechanisms are suggested, but all typically start with an incipient insult or injury that causes a vicious cycle of muscle spasm and pain, leading to C-fiber activation, release of substance P, mast cell activation and histamine release; the end result is further tissue inflammation and nerve injury. **Table 2** summarizes some common initial insults leading to the PFD cycle.

Table 2: Common PFD Mechanisms
Local Trauma / Injury
Vaginal deliveries
Pelvic floor surgery
Infection
Prolonged muscle overload
Competitive athletes
Volitional urine / Stool withholding
Nervous system changes
Hyperalgesia
Central sensitization
Excitatory neurotransmitter upregulation
Visceral, somatic cross-communication
Comorbid pain disorders
TMJ
Vulvodynia
Fibromyalgia
IBS
Endometriosis

Migraine headaches

5.3 History

Because more than one pain generator is often present, a thorough evaluation is necessary to ensure all contributing factors are addressed. Pain secondary to PFD can be episodic – many patients will have worsening pain during times of stress due to involuntary muscle clenching causing an increase in muscle tension. The pain may be described as aching, soreness, stabbing, throbbing, heaviness, or pressure. **Table 3**[38] summarizes some important elements of the history.

Table 3: Important Elements of History

Key Clinical Question / Patient Complaint		Possible Etiology / DDx IN ADDITION TO PFD
Sexual function: Pain with penetration (dyspareunia)	Initial penetration	Hormonally mediated tissue changes (estrogen/testosterone) Perineal scarring – vaginal laceration during childbirth, episiotomy, prior vaginal surgery Vestibulodynia Genitourinary syndrome of menopause
	Deep penetration	Endometriosis Pelvic congestion syndrome Painful bladder syndrome
Urinary function: urgency, frequency, dysuria		Untreated UTI Interstitial cystitis / painful bladder syndrome
Suprapubic or abdominal pain		IBS Diverticulitis Pelvic inflammatory disease
Bowel function: constipation, dyschezia, incomplete evacuation of stool, bloating, splinting posterior vagina, anal digitation, incomplete evacuation, blood in stool		Rectocele Shortened puborectalis muscle (tight anorectal angle) Hemorrhoids Colorectal malignancy
Worsening pain with prolonged sitting; “pain is best at night when lying flat”		Pudendal neuralgia

Some historical elements suggestive of PFD include a history of competitive athletics, urinary hesitancy that temporarily improves with urethral dilation in spite of a cystoscopy negative for urethral stricture, recurrent urinary tract infections with negative cultures (particularly if these occur primarily around times of stress), and low back and lower abdominal wall pain, tailbone pain, and hip pain concerning for hip labral tear. Onset after a vaginal delivery, especially if laceration or episiotomy occurred, after pelvic surgery, or a history of emotional, physical, or sexual abuse can also be associated with PFD.

5.4 Diagnosis

Thorough examination is diagnostic for PFD. In women experiencing painful pelvic symptoms, a gentle examination of the pelvic floor, frequently assessing for patient discomfort, is critical to both diagnosis and rapport building.

5.4.1 External Examination

Table 4 summarizes key points of the external musculoskeletal examination for PFD.

Table 4: External Musculoskeletal Examination

Examination Findings	Etiology / Cause / Importance
Lumbar lordosis	Muscular imbalances in the lumbopelvic / hip complex
Avoidance of sitting: often standing when physician enters the room; if sitting, patient with exaggerated lean to one side (away from affected side)	Pudendal neuralgia - compression of the pudendal nerve by obturator internus or piriformis muscles – patient posture avoids pudendal nerve compression and worsening of pain symptoms
Painful nodules around abdominal wall muscles or scars – especially if they elicit or reproduce patient's pelvic symptoms	MTrPs of abdominal wall *Refer to pelvic floor physical therapist*
Pain with palpation of sacroiliac joint	SI joint instability
Positive Fitzgerald's test – pain with hip manipulation	Hip labral tear – pain radiates into vaginal side wall; compensatory pelvic muscle changes can also cause pelvic floor spasm *Refer to orthopedic surgeon*

5.4.2 Internal Examination

A standard examination is performed with the patient in low lithotomy or stirrups using a single well-lubricated digit.[39] The clinician should examine for any scars or incisions, which may indicate etiology. Assessment of the superficial muscles (bulbospongiosus, ischiocavernosus, superficial transverse perineal muscles) is the first step. The obturator internus and externus can be palpated by sweeping from the pubic ramus inward (caudal to cranial) along the muscle belly behind the pubic ramus at 1- and 11-o'clock. At 3- and 9-o'clock, the levator ani complex is present, and at 5- and 7-o'clock, the iliococcygeus muscle (distal) is palpable. Approximately a finger-length depth into the vagina around 4- and 8-o'clock, the ischial spines are palpated as bony prominences. The ischial spine is the attachment of the fan-shaped sacrospinous ligament that runs through the coccygeus muscle; the ischial spine is also the anatomic marker for the pudendal nerve, which courses approximately 2 cm postero-medial to the spine. [40]

The muscle examination should start with light palpation for general tone, then deeper pressure to assess for trigger points which are hallmark diagnostic indicators of pelvic floor dysfunction. Clinical criteria that indicate presence of a trigger point include (1) a palpable taut band, (2) an extremely tender nodule in the taut band, (3) ability to reproduce the pain with palpation of the tender nodule, and (4) painful limit to stretch or full range of motion. [35] The degree of tension can be quite marked on examination.

5.4.3 Additional Diagnostic Testing

The patient's history and physical examination may warrant judicious use of additional diagnostic tests, but expensive testing is not necessary to make the diagnosis of PFD. Pelvic ultrasound is indicated for pressure, pain, and bloating. Anorectal manometry may be useful for patients with primarily defecatory symptoms. Any history of **gross hematuria** merits cystoscopy and appropriate upper tract study; bloody stool warrants rectal examination and colonoscopy. Cystometry and/or urodynamics can be useful in patients with comorbid urgency and frequency.

6. Treatments

Multimodal treatment plans directed at the patient's underlying etiology are most successful at alleviating and resolving symptoms of PFD. The cornerstone of management is pelvic floor rehabilitation with neuromuscular education, ideally by a physical therapist with extensive pelvic floor training. Many other therapies have been described but lack level 1 evidence supporting their use, and the secondary end point of dyspareunia is often not reported.[41] It is also critical to eliminate other sources of genitopelvic pain that may be contributing to chronic contraction of pelvic floor muscles and hypertonicity. In cases of secondary pelvic floor dysfunction, treatment of only the pelvic floor muscles may be less successful as the stimulus for contraction is ongoing.

6.1 Pelvic floor physical therapy

Pelvic floor physical therapy (PFPT) uses manual techniques of massage, myofascial release, joint

mobilization, strain-counter-strain, and trigger point massage to achieve lasting pain relief. Manual physical therapy is the foundation of management of PFD because it is truly effective – 59 to 80% of patients with pelvic pain have marked or complete relief of symptoms. [41] Thiele originally described transrectal digital release of spastic pelvic floor muscles in 1963[42], and Thiele massage works by application of digital pressure to elongate and relax the muscles, restoring their normal tone.

An online resource to find local pelvic floor physical therapists can be found at **www.womenshealthapta.org.**

6.2 Trigger Point/Levator Injections

Tender myofascial trigger points and the spastic pelvic floor can both be targeted with local injections to assist in relaxation. No particular combination of medications has been shown to be superior; however, many studies involve a combination of both local anesthetic (e.g. bupivacaine) and steroid (e.g. triamcinolone). Bupivacaine is FDA-approved for intramuscular infiltration due to lower myotoxicity risk than other injectable anesthetics; it also has a longer half-life, allowing for a prolonged improvement in pain. Triamcinolone (and all steroids) improve myalgia. The combination is very effective at temporarily improving pain when injected into MfTP.

A systematic way to address all areas of pelvic floor muscle spasticity is injections at a template of 1-, 3-, 5-, 7-, 9-, and 11-o'clock positions, both proximally (towards the ischial spine) and distally (behind the pubic ramus) [43].

A typical set up for pelvic floor injections includes the following:

1. 7 inch 22 gauge Quincke spinal needle – the cutting bevel allows for penetration into the muscle while the Luer lock connection at the hub attaches to a hypodermic syringe
2. 140 mm curved Killian (Von Eicken) antral irrigation cannula – this metal cannula is designed for irrigation of nasal pathology; however, its curved shaft permits passage of the Quincke spinal needle to permit blind / visionless injections to the pelvic floor under digital guidance. When the 7 inch needle is "hubbed", the muscle is penetrated to 2 cm. One can also use an Iowa trumpet needle guide for pudendal nerve blocks; its straight design may be too painful to easily maneuver through a spastic pelvic floor. **Figure 4** shows the Killian antral irrigation cannula and its gentle curvature.

Figure 4: 140 mm Killian antral irrigation cannula

3. 30 mL Luer lock syringe – larger syringe (such as 60 mL) may be more difficult to compress while injecting; 30 mL size gives good control
4. Lubricating jelly
5. 4 x 4 gauze sponges

6. Tampon
7. Povidone / iodine prep to cleanse the vagina prior to injection
8. Medication – common combinations include bupivacaine or ropivacaine with or without triamcinolone or onabotulinumtoxinA – 30 mL total volume

Once the setup is complete and the vagina is prepped, one can proceed with transvaginal digitally guided injections. At the 1- and 11-o'clock positions, the obturator internus and externus are targeted; because of their relatively superficial location, the needle only needs to be advanced 1 cm into the muscle (and injecting too deep risks anesthetizing the obturator nerve). Each injection (proximal and distal) should use approximately 3-5 mL of anesthetic cocktail. At 3- and 9-o'clock, the levator ani complex is targeted proximally and distally in a similar fashion but inserting the needle to 2 cm depth. At 5- and 7-o'clock, the iliococcygeus muscle and pudendal nerve are injected. The proximal injection just off the ischial spine targets the pudendal nerve at 2 cm depth, but the distal injection must be more shallow (1 cm deep) otherwise the medication enters the ischiorectal fossa fat.

OnabotulinumtoxinA has also been injected into spastic pelvic floor musculature to produce muscle paralysis and relieve the spasticity of PFD. Current literature shows mixed results but is limited by small sample sizes and short duration of follow-up. The use of onabotulinumtoxinA for PFD is not FDA-approved; its use is off-label.[44]

6.3 Pharmaceutical Management

Intravaginal diazepam suppositories are commonly used for relief of PFD. By relaxing the pelvic floor spasm, there is symptomatic improvement in PFD. Its use is also relatively safe; Carrico and Peters measured low serum diazepam levels after administration[45] but due to its prolonged half-life, gradual accumulation will occur with chronic daily doses. Because[46] of this, some physicians advocate for on demand use prior to physical therapy or intercourse.

Other cocktails of vaginal medications are also in use, including ketamine and baclofen.

6.4 Neuromodulation

Neuromodulation is another off-label minimally invasive option available for management of chronic refractory pelvic pain. In theory, by stimulating afferent nerve roots, the pelvic floor spasm decreases[47]. Recent meta-analysis shows consistent reduction in pain symptoms in patients with chronic pelvic pain[48]; Everaert et al reported significant improvement in dyspareunia in 14/26 patients after sacral neuromodulation.[49]

6.5 Other Conservative Treatments

Cognitive behavioral therapy, relaxation and mindfulness therapy, and TENS units can be used as adjunctive therapies to the above interventions.[50]

PFD is a common source of dyspareunia and pelvic pain and also commonly misdiagnosed as interstitial cystitis. It is essential to keep PFD on the differential diagnosis for pelvic pain syndromes

in all patients but particularly for women with dyspareunia. A multimodal treatment regimen with physical therapy as the center is very effective at complete pain relief.

Conclusion

Urologist can play a pivotal role in the treatment of pelvic pain and vulvar genital pain. Using topical hormones, pelvic floor physical therapy and behavioral therapy referrals as well as procedural and surgical interventions it is possible to help patients find relief from their genitourinary pelvic pain.

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