Managing Female Sexual Pain

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KEYWORDS

• Female • Sexual pain disorder • Genito-pelvic pain/penetration disorder • GPPPD

KEY POINTS

- Definition of GPPD.
- Etiology/differential diagnosis.
- Diagnosis.
- Treatment.

INTRODUCTION

With the evolving language regarding gender identity and sexual orientation, the authors believe that 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 based on their reproductive functions. The authors of this article 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.

Female sexual pain disorder or genito-pelvic pain/penetration disorder (GPPPD) is defined as persistent or recurrent symptoms with one or more of the following for at least 6 months:

1. Marked vulvovaginal or pelvic pain during penetrative intercourse or penetration attempts
2. Marked fear or anxiety about vulvovaginal or pelvic pain in anticipation of, during, or as a result of penetration
3. Marked tensing or tightening of the pelvic floor muscles during attempted vaginal penetration.[1]

GPPPD, previously termed dyspareunia and/or vaginismus, first debuted in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) in 2013. It is classified as either lifelong or acquired and ranges in the degree of distress from mild, moderate to severe. The estimated prevalence of GPPPD in the United States varies between 3% and 25% with causes differing by age group.[2] Women with GPPPD report significantly reduced quality of life and well-being. GPPPD can contribute to a decline in self-esteem, feelings of femininity and is associated with a negative body and genital self-image. GPPPD can pose a significant burden on the couple as well. The disordered cycle of pain can lead to fear, hypervigilance in sexual situations, or complete avoidance of sexual intimacy.[3–5]

Individuals with GPPPD often do not seek care despite the distressing nature of the disorder for a variety of reasons. These reasons include but are not limited to:

1. GPPPD was not previously regarded as a recognized disorder and instead viewed as a culturally taboo subject[6]
2. Feelings of shame and guilt by the individual because of cultural and personal stigmatization[6,7]

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3. Lack of appropriately trained health care professionals that do not make an inquiry about sexual health in routine care visits. Provider reluctance to discuss sexuality may stem from a sense that this is not a medical problem, personal discomfort with sexuality, or lack of training/knowledge/time.[8]

Patients with GPPPD who do present for treatment may not receive appropriate treatment/referral, which may lead to a further burden for the individual and partner as well as increased direct health costs. GPPPD symptoms are frequently multifactorial and require a multidisciplinary approach that assesses various factors including biological, psychological, sociocultural, interdependent pathophysiology, critical life events, and relational status.[9–11] A comprehensive biopsychosocial approach should be implemented for assessment and treatment. The treatment plans should focus not only on difficulties with vaginal penetration and muscle tightness associated with sexual intercourse but also the psychological factors that contribute to sexual dissatisfaction and couple dynamics.[12–14] Psychological factors that may contribute to dyspareunia include anxiety or guilt about intercourse, memories of distressing early sexual experiences, fear of penetration, unresolved anger, feelings of shame or guilt, and inadequate precoital stimulation.

Pathophysiology

Genitourinary Syndrome of Menopause: Genitourinary Syndrome of Menopause (GSM), previously described as vulvovaginal atrophy/atrophic vaginitis, is mediated by depletion of androgens and estrogens and subsequently decreased blood flow to the vagina and vulva. GSM is characterized by genital symptoms (eg, dryness, burning, and irritation), sexual symptoms (eg, lack of lubrication, discomfort or pain, and decreased libido and difficulty with arousal and orgasm), and/or urinary symptoms (eg, urgency, dysuria, and recurrent urinary tract infections).[15] GSM may occur in hormone-depleted states outside of menopause including breastfeeding, oral contraceptive use, adjuvant hormonal deprivation therapy for various disorders, gender-affirming hormone therapy, thyroid disorders, and pituitary tumors[16–18] (**Fig. 1**).

Vestibulodynia: Vestibulodynia (VD), previously known as vulvar vestibulitis syndrome, is defined as vulvar vestibule pain lasting more than 3 months unrelated to infection, skin disorders, or other identifiable factors. It is often characterized as burning, stinging, itching, pain with penetration, and/or rawness of the vestibule/vagina. The most common factors associated with VD are hormonal changes, hypertonic pelvic floor dysfunction (PFD), and/or an increased number of nerve endings in the mucosa of the vestibule termed neuroproliferative VD.[19]

Clitorodynia: Clitorodynia is a form of vulvodynia localized to the clitoris. It is characterized by frequent and intense pain episodes that can be either provoked or unprovoked. Clitorodynia may cause significant impairment in both activities of daily living and sexual activity. It may be associated with other chronic pain disorders, lichen sclerosus, multiple sclerosis, pelvic surgery, and vaginal delivery.[20] Physical examination may show clitoral adhesions, phimosis, balanitis, or skin changes associated with lichen sclerosus.

Pelvic Floor Dysfunction: PFD is an umbrella term encompassing the constellation of symptoms secondary to the nonrelaxing and spastic pelvic

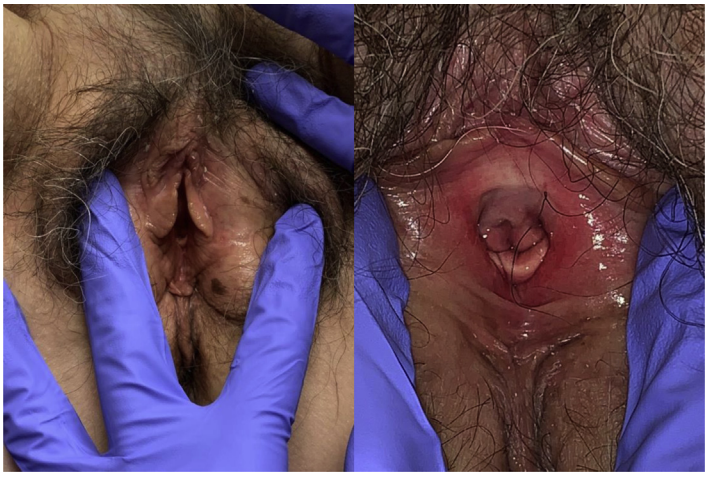

Fig. 1. (A) The 50% resorbed and thin labia minora. (B) The protruding urethra, pallor, and erythema of the vestibule. Patient's vaginal pH was 7.5, whereas the goal should be 4.5. Image courtesy of Rachel Rubin, MD, NW, Washington.

floor musculature. The presenting symptoms of PFD depend on the particular muscle(s) in spasm. Common manifestations include dyspareunia, voiding dysfunction, incontinence, urinary retention, constipation, and dyschezia.

Anatomy/Physiology

A detailed understanding of female genital anatomy is crucial in providing comprehensive evaluation and optimizing treatment. The anatomic female sexual organs include the clitoris (glans and crura), labia major and minora, the vulvar vestibule, the vagina, the cervix, the vestibular bulbs, and the pelvic floor muscles.

Vulva

The vulva is a complicated anatomic structure intricately involved in the sexual response cycle. It includes the mons pubis, labia majora, labia minora, vestibule, and clitoris.

Vestibule

The vulvar vestibule is located between the labia minora and the hymen. This tissue originates from endoderm and is hormonally regulated by testosterone.[21] The medial border is the hymen and lateral border is "Hart's Line," which marks the change from the vulva skin to the smooth transitional skin of the vulva located inside the labia minora. The vestibule contains the external urethral meatus, the openings of the two greater (Bartholin's) glands, and the perivestibular/Skeene's glands (respectively analogous to Cowper's glands and glands of Littre in male anatomy). The vulvar vestibule can become painful due to changes in hormones (ie, low androgen states like menopause or use of oral contraceptive pills), inflammation, muscle hypertonicity, or more rarely congenital neuroproliferation.[22] The vestibule is an area of significance in patients presenting with vulvar pain and GPPPD (Fig. 2).

Clitoris

The clitoris consists of spongy vascular tissue that engorges with blood during arousal. The body of the clitoris is surrounded by the tunica albuginea and consists of two paired corpora cavernosa composed of trabecular smooth muscle and lacunar sinusoids. The glans clitoris is the component of the clitoris that is, visible but often covered by a "hood" of preputial skin, particularly in the unaroused state. The preputial skin can develop phimosis and/or balanitis, which may contribute to clitoral pain (clitorodynia) and/or anorgasmia.[23]

Vagina

The vagina is an elastic, muscular canal that extends from the vulva to the cervix. The vaginal wall consists of three layers: (1) an inner mucous type stratified squamous cell epithelium supported by a thick lamina propia, that undergoes hormone-related cyclical changes, (2) the muscularis, composed of outer longitudinal smooth muscle fibers and inner circular fibers, and (3) an outer fibrous layer, rich in collagen and elastin, which provides structural support to the vagina.[24] It serves a multitude of functions in response to hormonal changes and plays a vital role in the reproductive system and sexual pleasure. The vaginal mucosa contains a high concentration of estrogen and androgen receptors.[25]

Pelvic floor

The female pelvic floor muscles support the bladder, uterus, and colon and play a vital role in sexual function as well as bladder and bowel control. A clock face can be used as a reference when describing the location of the pelvic structures.

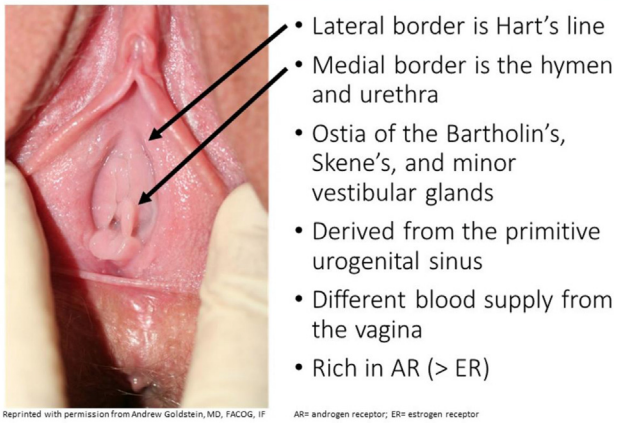

Fig. 2. The vestibule. Courtesy of Irwin Goldstein, MD, San Diego, California, with permission

The 12- and 6-o'clock positions correspond to the anterior and posterior midline, or pubic symphysis and anus, respectively. (10) The obturator internus and externus can be palpated by sweeping from the pubic ramus downward 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 spines serve as the anatomic marker for the pudendal nerve, which runs approximately 2 cm posteromedial to the ischial spine and innervates the clitoris, vulva, and anus.[26]

Lymphovascular

Arterial

The internal pudendal artery is the dominant blood supply to the female external genitalia. The internal pudendal artery is a branch of the internal iliac artery. The labia majora is also supplied by the superficial external pudendal artery, a branch of the femoral. This anatomic distinction is important to note as the dual blood supply makes it a useful flap in reconstructive surgeries.[27]

Venous

The venous drainage of the external female genitalia is via the external and internal pudendal veins. The external pudendal vein drains to the great saphenous vein, which in turn drains into the femoral vein and from there to the external iliac vein after ascending past the inguinal ligament. The internal pudendal vein drains back into the internal iliac vein. Both the external and internal iliac veins will ascend and merge to form the common iliac veins, which merge to form the inferior vena cava.[27]

Lymphatic

The lymphatic drainage of the external female genitalia is primarily by the superficial inguinal lymph nodes; the exception to this is the clitoris, which drains toward the deep inguinal lymph nodes. The superficial and deep inguinal lymph nodes conjoin and drain into the common iliac lymph nodes. All of this lymph will ascend toward the inferior part of the thoracic duct known as the cisterna chyli.[27]

Neuroanatomy and Physiology

Autonomic Nervous System

Sexual response is the result of coordinated activity of the parasympathetic, sympathetic, and somatic nervous system. The reproductive organs receive preganglionic parasympathetic innervation from the sacral spinal cord, sympathetic innervation from the outflow of the lower thoracic and upper lumbar spinal cord segments, and somatic motor innervation from α-motor neurons in the ventral horn of the lower spinal cord segments.[28] During genital stimulation, afferent somatic sensory endings in the dorsal roots of S2–S4 are relayed centrally to the somatic sensory cortex. This is the region of the cerebral cortex concerned with processing sensory information from the body surface, subcutaneous tissues, muscles, and joints. Activation of the somatic sensory cortex leads to decreased sympathetic input and increased parasympathetic activity via the pelvic nerve causing a release of nitric oxide resulting in vasodilation. This in turn leads to a rise in clitoral cavernosal artery inflow and increased clitoral intracavernosal pressure. Increasing pressure leads to clitoral engorgement, extrusion of the glans clitoris from the prepuce, and enhanced sensitivity. Increased blood flow to the vagina leads to vasocongestion within the vaginal submucosa, increasing oncotic pressure, which leads to the production of a fluid transudate (lubrication) that passes into the vaginal lumen via aquaporins located in the vaginal mucosa.[29,30]

The lumbar sympathetic pathway to the sexual organs originates in the thoracolumbar segments (T11–L2) and reaches the target organs via the corresponding sympathetic chain ganglia and the inferior mesenteric and pelvic ganglia in the hypogastric nerve. The sympathetic nervous system produces rhythmic smooth muscle contractions of the vagina during orgasm.

The somatic nervous system is composed of the pudendal nerve (S2–S4). The pudendal nerve reaches the perineum through Alcock's canal and provides sensory and motor innervation to the external genitalia. Excitatory input leads to muscle contraction of the bulbocavernosus and ischiocavernosus muscles that accompany orgasm.[31]

Hormonal Physiology

Estrogens (E) are the primary "female" sex steroids and act by binding to widely distributed estrogen receptors within the body.[32,33] Three estrogens are naturally produced in the female body.[34] In premenopausal women, 17β-estradiol (Estradiol or E_2) produced by the ovary is the estrogen present in the largest quantity; circulating estradiol levels fluctuate from 40 to 200–400 pg/mL across the menstrual cycle.[35] Owing to its high affinity for the estrogen receptor, it is the most potent of the three estrogens. Estrone (E1) is a less potent estrogen that can be synthesized via E2 by the enzyme 17-beta hydroxysteroid dehydrogenase or through the conversion of androstenedione in

adipose tissue via aromatase. The third endogenous estrogen, estriol (E_3), is also a metabolite of estradiol in the periphery. Estriol is the principle estrogen produced by the placenta during pregnancy but is found in smaller quantities than either estradiol or estrone in nonpregnant individuals. In postmenopausal women, estradiol levels drop to less than 20 pg/mL. The ovary ceases producing estradiol, but the adrenal gland continues making androstenedione, the immediate precursor to estrone, so the levels of estrone remain unchanged despite the dramatic fall of estradiol.[36]

At the target organ, estrogens diffuse into the cell and through the nuclear membrane. Inside the nucleus, estrogens attach to an estrogen receptor to form a ligand-receptor complex, which binds to DNA and initiates gene transcription. Gene transcription in turn leads to the production of specific proteins that trigger estrogenic effects in the target tissue (eg, maintenance of tissue integrity and thickness in the vaginal wall).[37]

Androgen receptors are also common and extensive throughout the female genitourinary tract. Androgens (ie, dehydroepiandrosterone [DHEA], androstenedione, and testosterone), which are produced in the adrenal cortex and ovaries, are necessary precursors for the biosynthesis of estrogens and play important direct roles in the physiology and homeostasis of the vagina.[38] Their production is significantly greater than that of estrogens in premenopausal and postmenopausal women.[39]

EVALUATION

History

Providers should provide an open and compassionate approach with patients when addressing sexual health to increase patient's comfort on such a sensitive subject matter. The most important goal during the introductory discussion is to provide validation of the patient's pain and distress. This will aid in establishing rapport and trust between the patient and provider. This should be done while the patient is fully dressed and ideally not in an examination room.

Open-ended questions, affirming statements, and reflective listening should be used to obtain a comprehensive history. Generalizing statements are helpful tools to engage the patient; an example of this would be "Many women with (a specified condition) experience (a specific or generalized issue with sex); Do you experience this?". The use of validated self-reported questionnaires such as the Female Sexual Function Index, the McGill Pain Questionnaire, or the Patient-Reported Outcomes Measurement Information System (PROMIS) vulvar discomfort scale can also provide objective information when quantifying pain and the impact it has on the patients' life.[40] A focused sexual history should review the following:

1 .Pain characteristics (location, duration, exacerbating factors, alleviating factors)
2. Associated symptoms such as bowel, bladder, or musculoskeletal symptoms
3. Sexual activity and behavior
4. Past medical history
5. Surgical history
6. Medication history (use of hormonal birth control, SSRIs, etc)
7. Mental health history
8. Obstetrics and gynecologic history including onset of menstruation, characteristics of periods, and onset of menopause
9. History of physical or sexual abuse
10. Previous interventions

Physical Examination

Many objective findings on physical examination can be seen in a patient with sexual complaints. A full vulvar and vaginal examination will aid in finding the proper treatment for the patient. Important anatomy to assess includes:

- Labia majora and minora.
- Clitoris, including glans and hood.
- Urethra and periurethral glands.
- Vestibule.
- Vaginal vault.
- Cervix.
- Pelvic floor levator ani muscles.

Important factors to evaluate in the focused genital examination include:

- Distribution of hair.
- Symmetry and size of genital tissues.
- Evidence of atrophy or stenosis.
- Areas of provoked pain.
- Color uniformity, erythema, or other discoloration.
- Visible lesions, excoriations, or scars.
- Pelvic organ prolapse.
- Pelvic floor muscle tone and voluntary control.
- Presence of vaginal discharge.
- Presence or absence of vaginal rugae.

Each examination should begin with an explanation of each step and a real-time discussion of the examination findings with the patient. The examination should include an external and internal musculoskeletal evaluation, external visual and sensory examination, and a bimanual examination

if tolerated by the patient. It is empowering for patients to visualize their anatomy either with a vulvoscope or mirror as a means to better understand and feel comfortable with their anatomy. The external musculoskeletal examination should include evaluation of posture/gait, symmetry/asymmetry, palpation of abdominal, gluteal, back, and lower extremity muscles. Assessment should include areas of tension and/or pain, muscle strength, range of motion, sensation, and reflexes.

Examination of the vulva is performed by inspecting the external genitalia, perineum, perianal areas, and the mons pubis. The clinician should evaluate for signs of infection, trauma, atrophy, fissures, and dermatologic changes. A cotton swab should be used to assess allodynia and/or hyperalgesia by light palpation of the vulvar structures. The vestibule should be examined making sure to note areas of hyperemia or erythema and assessment/presence of the periurethral and perivestibular glands. A sensory test should be performed using a cotton swab to palpate the vestibule in 7 anatomic sites (12 o'clock, 1 o'clock, 3 o'clock, 5 o'clock, 7 o'clock, 9 o'clock, and 11 o'clock).

The internal pelvic muscles should be examined through the vagina. The examination should start with light palpation for general tone, then deeper pressure to assess for trigger points, which are hallmark diagnostic indicators of PFD. Using the index finger, the examiner can palpate the lateral, anterior, and posterior walls of the vagina, the urethra, and pelvic floor muscles assessing for tone, tenderness, or involuntary contractions. 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.[41] If pain or hypertonic muscles are noted during this examination, a pelvic floor physical therapy referral may be warranted.

A bimanual examination should be performed to evaluate the uterus and adnexa for any masses or tenderness. Internal examination of the vagina and cervix should follow using a warmed small-sized Grave's or Pederson speculum. The speculum should be inserted slowly ensuring to avoid the urethra or vestibule as these areas can provoke pain. During the speculum examination, the internal vaginal tissue, cervix, and vaginal secretions are examined. Cultures or biopsies can be collected at this time to rule out infections, dermatoses, or abnormal cellular dysplasia that can cause dyspareunia or vulvodynia.

TREATMENT

Genitourinary Symptoms of Menopause

The primary goals for treating GSM are alleviation of symptoms, restoring vaginal pH, and preventing recurrent urinary tract infections. A multimodal approach can be used to optimize results.

Local Vaginal Therapy

Low-dose vaginal estrogen

Life-long low-dose vaginal hormone therapy is the mainstay treatment for GSM (eg, vaginal creams, intravaginal tablets, or intravaginal rings). It is recommended by the American Urologic Association for the treatment of recurrent urinary tract infections in premenopausal and postmenopausal women as data show it prevents urinary tract infection by restoring vaginal flora and pH.[42] Topical low dose vaginal estrogen restores hormone levels within the tissue without significant systemic absorption. This results in rapid improvement of vaginal symptoms within 2 to 3 weeks but may take 2 months for maximal benefit. Systemic estrogen therapy can be considered in addition to topical estrogen if there are concomitant vasomotor symptoms. Its use as monotherapy, however, has not been shown to treat symptoms of GSM. The American College of Obstetricians and Gynecologists recommends the use of nonhormonal options as the first choice for the treatment of vaginal atrophy in women with current or a history of estrogen-dependent breast cancer. Vaginal estrogen therapy is deemed appropriate for patients with a history of estrogen-dependent breast cancer who are unresponsive to nonhormonal remedies but only after a thorough discussion of risks and benefits with their oncologist.[43]

Androgen/testosterone

Vaginal DHEA suppositories (Intrarosa) are FDA approved for the treatment of moderate to severe GPPPD. DHEA is converted by enzymes in the vulva, vestibule, and vagina into estrogen and testosterone. This results in significant improvements in vaginal epithelial cells and integrity, vaginal pH, parabasal cells, increased vaginal secretions, all without affecting serum levels of estradiol and testosterone or endometrial tissues.[44,45]

Lubricants and moisturizers

Several over-the-counter vaginal lubricants (water-, silicone-, or oil-based) and moisturizers are commonly used for supplemental/symptomatic treatment of postmenopausal women with vulvovaginal symptoms. Moisturizers are used as daily therapy and lubricants are used as needed

typically for sexual activity but can be used independently of sexual activity. Patients should choose a product that is physiologically most similar to natural vaginal secretions and will not disturb the vaginal pH.[46] Water-based lubricants are often preferred over oil-based lubricants as they are nonstaining and associated with fewer genital symptoms, although they often need to be reapplied. Caution should be used with oil-based lubricants as this can lead to condom breakage.[47]

Oral Therapy

Selective estrogen receptor modulators

Selective estrogen receptor modulators (SERMs) are systemic nonhormonal therapy delivered orally. Ospemifene is the only SERM approved by the US Food and Drug Administration (FDA) for the treatment of moderate to severe dyspareunia.[48] Several studies have shown that it increases vaginal maturation index and lubrication and normalizes vaginal pH. Common side-effects include hot flashes, vaginal discharge, and muscle spasm. Contraindications include estrogen-dependent neoplasms, history of venous thromboembolism, previous stroke or MI, or active heart disease (see **Table 1**).[48,49]

Nonsurgical/Nonmedical Treatments

Vaginal dilators

Women with GSM may see benefit from gentle stretching of the vagina with the use of lubricated, sequential dilators. Dilators have been shown to increase vaginal elasticity, which in turn decreases pain with penetration. Pelvic floor muscle therapy may also be useful in patients with nonrelaxing or high-tone pelvic floor muscle dysfunction triggered by painful sexual activity related to GSM.[50] Patients and providers can find pelvic floor physical therapists through numerous online resources including https://aptapelvichealth.org/.

Lasers: fractional CO_2 laser or erbium:YAG laser can be used as a nonhormonal treatment option for GSM.[51] Several small studies have shown restoration of the vaginal epithelium, increase in premenopausal vaginal flora, and subjective improvement in symptoms of GSM, including lower urinary tract symptoms.[52] At this time, these therapies are not FDA approved and should not be considered a standard of care. Readers interested in more detail on these therapies are referred to the chapter in this volume on energy-based and pharmaceutical treatments for sexual concerns in women.

VD treatment

The treatment of VD requires a multidisciplinary biopsychosocial approach as most cases are multifactorial. In terms of medical management, hormone therapy with vaginal estrogen and testosterone are the mainstay of medical management for hormonally mediated VD; options include DHEA vaginal inserts or compounded estrogen/testosterone creams. We recommend compounded estradiol 0.03% and testosterone 0.1% in a methyl cellulose or versa base applied to the vestibule 1 to 2 per day. Studies have shown

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 g daily for 2 wk, and then 0.5–1 g 1–3× per wk
Conjugated equine estrogens cream	Premarin	0.5–1 g daily for 2 wk, and then 0.5–1 g 1–3× per wk
Vaginal Inserts		
Estradiol vaginal tablets	Vagifem®, Yuvafem®,	10 mcg inserts daily for 2 wk, and then 2× per wk
Estradiol soft gel capsules	ImVexxy®	4, 10 mcg inserts daily for 2 wk, and then 2× per wk
DHEA (prasterone) inserts	Intrarosa®	6.5 mg capsules daily
Vaginal Ring		
17-beta-estradiol ring	Estring®	1 ring inserted every 3 mo
SERM		
Ospemifene oral tablets	Osphena®	60 mg tablet daily

improvement in 50% of people with VD symptoms over 12 weeks.[53] Discontinuation of oral hormonal contraception is a consideration if the onset of symptoms is linked to the initiation of this treatment. Patients should be counseled on long-acting reversible contraception, which may have lesser hormonal effects on the vestibule.[54]

Cognitive behavioral therapy and sex therapy facilitated by a licensed professional play a key role not only in the treatment of VD but also in all causes of GPPD. Resources for sex therapy include the American Association of Sex Educators, Councilors, and Therapists (www.aasect.org/) and the Society for Sex Therapy and Research (www.sstarnet.org/).

Topical steroids may be indicated in cases of VD associated with dermatologic conditions, that is, Lichen Sclerosus. Pelvic floor physical therapy, biofeedback, OnabotulinumtoxinA injection, compounded vaginal valium suppositories, pudendal nerve blocks, and neuromodulation are other adjuvant therapies that have been shown to be beneficial in the treatment of some cases of VD.[41] In refractory cases or for those with persistent pain despite correction with hormone therapy, a vulvar vestibulectomy with vaginal flap advancement, may be offered. A high success rate can be expected for appropriately selected patients cared for by an experienced surgeon (Fig. 3).

Clitorodynia treatment

The treatment for clitorodynia is determined by the underlying cause. If clitorodynia is secondary to phimosis or lichen sclerosis, potent topical steroids may be used for management.[20,55] Surgical treatment can be offered if the pain is associated with correctable physical examination findings such as clitoral pearl or adhesions refractory to medication. Either office-based lysis of adhesions or surgical dorsal slit may be considered dependent on patient tolerance and severity of disease (Fig. 4).

Pelvic floor rehabilitation

Pelvic floor rehabilitation (PFR) is most typically a multimodal treatment plan developed in consultation with a licensed physical therapist with expertise in the management of PFD. The mainstay of PFR is pelvic floor physical therapy, which includes manual techniques of massage, myofacial and trigger point release, and joint mobilization. Adjunct therapies include trigger point injections with steroids or OnabotulinumtoxinA for pelvic floor tightness or spasticity, intravaginal diazepam

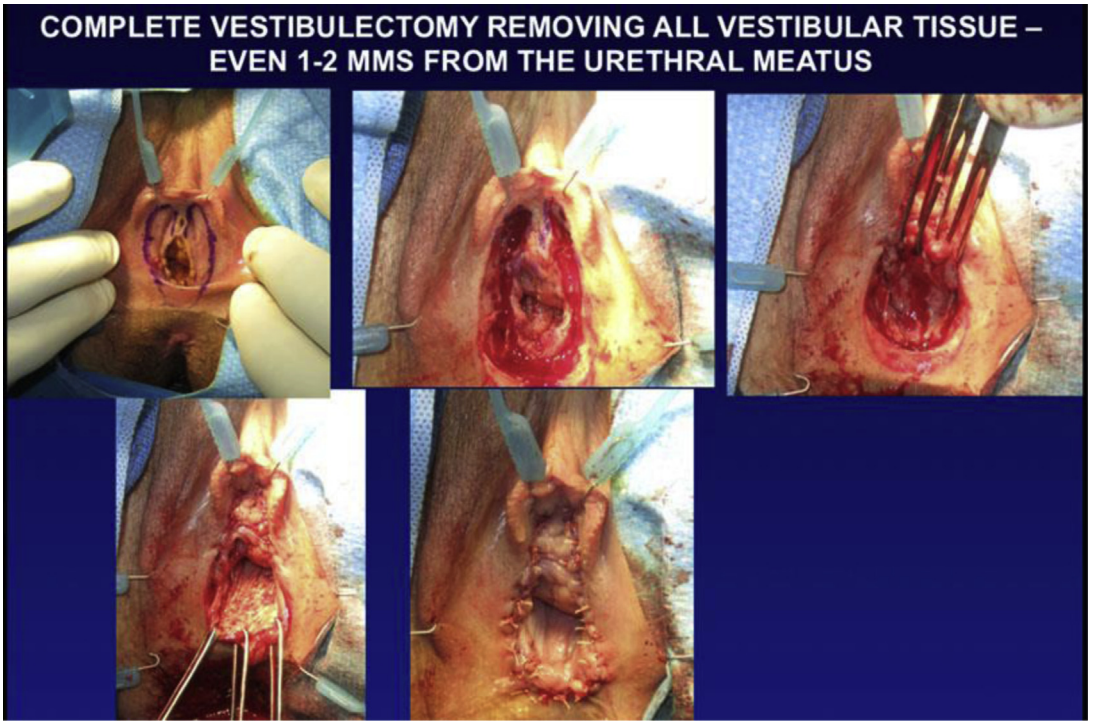

Fig. 3. Complete vestibulectomy with left and right anterior vestibulectomy and posterior vestibulectomy with vaginal advancement flap reconstruction. (Photos courtesy of Irwin Goldstein MD)

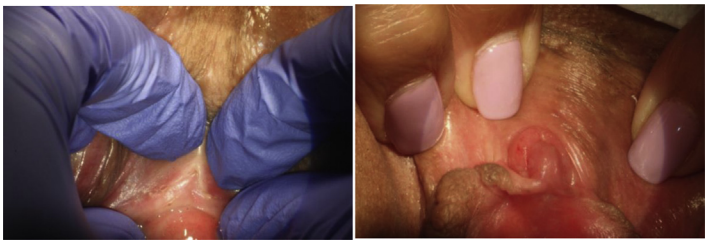

Fig. 4. Phimosis of the clitoris and clitoral lysis of adhesions. Courtesy of Rachel Rubin, MD, NW, Washington.

suppositories, transcutaneous electrical nerve stimulation, and neuromodulation.

SUMMARY

GPPPD is a complex often multifactorial disorder that significantly impacts patients' physical health, mental health, and overall quality of life. Owing to the complex nature of sexual response, GPPPD is typically best managed using a multidisciplinary approach that includes medical, surgical, behavioral, and musculoskeletal interventions.

CLINICS CARE POINTS

- Female sexual pain disorder or genito-pelvic pain/penetration disorder (GPPPD), previously known as dyspareunia, is defined as persistent or recurrent symptoms with one or more of symptoms for over 6 months.
- The estimated prevalence of GPPPD in the United States varies between 3% and 25% with causes differing by age groups.
- GPPPD is frequently multifactorial and requires a multidisciplinary approach that includes biological, psychological, sociocultural, and relational factors.
- An understanding of female genital anatomy is essential to provide comprehensive evaluation and optimize treatment outcomes for women with sexual concerns.

DISCLOSURE

The authors have nothing to disclose.

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