Differential Diagnosis of Pelvic Floor Dysfunction and Vulvar Pain

Mary Christine Knutson, MN, ANP

Introduction

Vulvovaginal problems and vulvar pain are common patient complaints. Patient complaints may suggest urologic, dermatologic, dysesthetic, vaginal, or colorectal problems. The pelvic floor is a complex structure, composed of integrated local pelvic support muscle groups and nerves as well as the general postural support system. The astute clinician can correctly diagnose and offer appropriate therapies more successfully after systematic assessment of the entire vulvovaginal region. Susan Kellogg-Spadt, PhD,[1] of The Pelvic Floor Institute, Philadelphia, Pennsylvania, discussed the differential diagnosis of pelvic floor dysfunction (PFD) and vulvar pain.

Pelvic Floor Dysfunction

PFD has traditionally been described as resulting from laxity or poor tonus of the pelvic floor musculature and/or ligaments. Damage of this nature usually results from aging, straining, or trauma and causes urinary or fecal incontinence and organ prolapse. More attention is focused now on identifying and understanding so-called "high-tone" PFD, a product of hyperclonic or spastic musculature, leading to symptoms such as urinary urgency-frequency, constipation, vulvar pain, dyspareunia, and orgasmic dysfunction.[1] High-tone PFD has been identified as a frequent source of overactive bladder syndrome and interstitial cystitis for at least 25 years.[2] The hypertonus can become so severe as to cause urinary retention severe enough to secondarily result in vaginal obstruction and even hematocolpos (an accumulation of menstrual blood in the vagina).[3]

Assessment for high-tone PFD consists of a thorough evaluation of external pelvic floor trigger points, internal pelvic floor muscles, and identification of any postural dissymmetry as a contributing factor. Hypertonus, and especially painful interstitial cystitis and urethral syndrome with or without pain, is often responsive to physical therapy and massage.[4] Therapy may be external, as in muscle training or home exercise programs, or internal, via massage of the ligaments and biofeedback or electrical stimulation. Symptoms are reduced as muscle tone normalizes.[1]

Evaluating Vulvovaginal Pain

A focused patient history and meticulous examination of the fragile vulvar/introital structures are essential in the evaluation of vulvar pain. Aside from an accurate description of symptoms, history should include questioning regarding the use of common vulvar irritants such as: adhesive of minipads; additives in tampons; dryer fabric softener sheets; detergents; deodorants; bleaching or dying agents used in toilet tissue and clothing; ingredients of creams, suppositories, and topical products (cetyl alcohol, propylene glycol, methyl paraben, benzylkonium chloride, neomycin), and others.[1]

Kellogg-Spadt encourages colposcopic examination of the vulva and introitus to follow gross examination. Even very small areas of erythema, ectasia, or microfissures could help explain symptoms. Take biopsies or cultures of any areas of concern. Microscopy should not be relied upon to completely rule out the presence of *Candida* or bacterial vaginosis (BV), since it has been

shown to be only about 50% sensitive. Clinicians should culture for these as well, using saline-moistened swabs and sampling from the labial folds and the external surface of the anal area. When sampling the vagina, use a saline-moistened swab to obtain a sample from the anterior vaginal wall, avoiding the posterior vaginal pool.

Recurrent vulvovaginal candidiasis is more common than many clinicians recognize and non-*Candida albicans* species may recur more readily due to treatment with a narrow spectrum of coverage.[5-7] Candida treatments with wide-spectrum coverage usually are better at preventing recurrent non-*C albicans* infection. These include: terconazole 0.4% cream given for 7 days, then weekly[8]; butaconazole nitrate 2% cream given every 5 days for 3-5 doses[1]; boric acid powder capsules or suppositories 600 mg 1-2 times daily for 14 days[1,9]; flucytosine for *C glabrata* (flucytosine is indicated for "serious infections" [eg, "septicemia, endocarditis, and urinary tract infections"]).[9,10]

Similar to vulvovaginal candidiasis, BV can recur. BV has a notoriously frustrating history and only in recent years has been correlated with premature rupture of membranes, preterm labor, increased susceptibility to urinary tract infections, postoperative infections, and HIV acquisition.[5] Suppression therapy has been successful in reducing recurrence from 83% to 33%.[1] For treatment and suppression, use metronidazole gel, 0.75%, 5 g vaginal applicator at bedtime for 10 days, then twice weekly for 12 weeks.[1]

The onset of vulvar vestibulitis syndrome (VVS), which affects as many as 1 in 15 women, may be associated with recurrent vulvovaginal candidiasis or recurrent bacterial vaginosis. It may also be a cofactor in other illnesses or syndromes such as irritable bowel syndrome or autoimmune disorders and is a major cause of vulvar and sexual pain. VVS presents as a localized vulvar dysesthesia. It is diagnosed by a touch test; that is, the typical pain is elicited by touching the ostia of the minor vestibular glands and/or the Skene's glands. Touch on other perineal sites or the clitoris does not produce pain. There may also be small focal areas of erythema at the ostia.

Vulvar vestibulitis will resolve spontaneously up to one third of the time, but it can be especially frustrating to treat due to the poor understanding of its etiology.[11,12] Traditional treatments have included tricyclic antidepressants, topical steroid creams, topical anesthetics, and even surgery to remove the affected skin.[1,11] Innovative treatment options include: topical application of 4% cromolyn cream daily[13]; 0.2% nitroglycerin cream before vaginal penetration[1]; 0.2% atropine cream twice daily[1]; 0.025% capsaicin cream daily[1]; and methylprednisone/lidocaine injections.[14] Some success has been reported with physical therapy, internal massage, and biofeedback as well.[1,15]

A more generalized vulvar dysesthesia, vulvodynia, has an even more poorly characterized etiology and often goes unrecognized and treated. This is a diagnosis of exclusion. In a population-based survey of women in the Boston area, 16% of respondents reported histories of vulvar pain lasting 3 or more months. In addition, nearly 40% of these women did not seek treatment, and, of those who did, 60% saw 3 or more clinicians, "many of whom could not provide a diagnosis."[16] The pain described in generalized vulvar dysesthesia is burning, often constant, and is not limited to the introital area. There is usually no outward clinical sign associated with it. Misdiagnosis and ineffective treatments often leave women feeling depressed and desperate. Tricyclic antidepressants and psychotherapy have been the most prescribed therapy.[1,11,17] Innovations in treatment include: gabapentin (indicated for postherpetic neuralgia),[9,17,18] topiramate (indicated only for management of seizures),[9] and topical ketamine compounds. Other potentially helpful interventions include: acupuncture, internal massage, trigger point injections, or nerve blocks.[1,17]

Recommendations for Practice

Meticulous clinical histories and observations are called for when evaluating any complaint of vulvar pain, as these complaints may be associated with urogenital conditions and PFD. Rule out pelvic floor injury or dysfunction, infections, and inflammatory processes before assuming a neurogenic cause of vulvar pain. Determining the cause of vulvar pain can be elusive and often frustrating for the clinician, but it is "real" for the patients who must cope with the limitations pain places on the quality of their lives. When a treatment isn't working, the diagnosis must be reconsidered. Fortunately, more options are available for the clinician and patient who are willing to work together over time for an improvement in symptoms.

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