THE JOURNAL OF
SEXUAL MEDICINE

Vulvodynia: Definition, Prevalence, Impact, and Pathophysiological Factors

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ABSTRACT

Introduction: Vulvodynia constitutes a highly prevalent form of chronic genital pain in women, and current information regarding its definition, prevalence, impact, and pathophysiologic factors involved is needed.

Aim: To update the scientific evidence published in 2010 from the Third International Consultation of Sexual Medicine pertaining to the definition, prevalence, impact, and pathophysiologic factors of women's sexual pain.

Methods: An expert committee, as part of the Fourth International Consultation of Sexual Medicine, comprised of researchers and clinicians from biological and social science disciplines, reviewed the scientific evidence on the definition, prevalence, impact, and pathophysiologic factors related to chronic genital pain.

Main Outcome Measures: A review of the definition, prevalence, impact, and pathophysiological factors involved in vulvodynia.

Results: Vulvodynia is a prevalent and highly impactful genital pain condition. Numerous factors have been implicated in its development and maintenance.

Conclusion: What is becoming increasingly apparent is that it likely represents the end point of different factors that can differ from patient to patient. Longitudinal research is needed to shed light on risk factors involved in the expression of vulvodynia, as well as in potential subgroups of affected patients, in order to develop an empirically supported treatment algorithm.

Key Words: Vulvodynia; Sexual Pain; Vestibulodynia; Prevalence; Definition; Pathophysiologic Factors; Dyspareunia

DEFINITION

Reports of sexual pain are highly prevalent in postmenarchal women, with pain sites including the vulvar, vaginal, cervical, and deep pelvic areas. Vulvodynia, or chronic vulvar pain, is a specific pain disorder that appears to have been reported by women for centuries. However, accurate descriptions of vulvar pain have only recently been defined. The most recent nomenclature (http://www.isswsh.org/news/190-2015-consensus-terminology-and-classification-of-persistent-vulvar-pain) was developed in April 2015 at a vulvar pain and vulvodynia consensus conference that was sponsored by the International Society for the Study of Vulvovaginal Disease, the International Society for the Study of Women's Sexual Health, and the International Pelvic Pain Society. The new nomenclature (referred to as the "2015 classification") proposes two main categories of chronic vulvar pain: vulvar pain related to a specific disorder (eg, inflammatory, neoplastic, traumatic) and vulvodynia, which is idiopathic vulvar pain of at least 3 months' duration. The 2015 classification also uses a pain-based system to characterize vulvodynia based on pain location (eg, localized, generalized, mixed), situations that elicit the pain (ie, upon contact, spontaneous, or mixed), temporal pattern (eg, intermittent or constant), and onset (ie, primary or secondary). The 2015 classification adds a list of potential associated factors for

Received September 15, 2015. Accepted December 20, 2015.

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http://dx.doi.org/10.1016/j.jsxm.2015.12.021

Table 1. Pathophysiologic Factors

Vulvar (neuro-proliferative)
Comorbid conditions
Central nervous system
Genetics
Myofascial and muscular
Hormonal
Embryologic and congenital
Inflammatory

vulvodynia (eg, musculoskeletal, neuro-proliferation, associated comorbidities, psychosocial factors) that act to acknowledge that vulvodynia is likely not one disease but a constellation of symptoms of several (sometimes overlapping) disease processes.

The importance of using a pain-based system to characterize vulvodynia is evident in research. For example, information regarding pain location and eliciting situations has led to the appreciation of different subtypes of vulvodynia. For example, provoked vestibulodynia (PVD; formerly called *vulvar vestibulitis syndrome, focal vulvitis, vestibular adenitis,* and *focal vestibulitis vulvae*) is characterized as localized provoked pain at the vaginal vestibule, whereas generalized vulvodynia (formerly termed *essential* or *dysesthetic vulvodynia* and *burning vulva syndrome*) is characterized by unprovoked, diffuse vulvar pain affecting the entire vulvar area.[1] At this point, it is not known whether provoked and unprovoked vulvar pain have overlapping or distinct pathophysiologies; however, there is agreement in the literature that vulvodynia can be caused by different factors. In addition, studies on PVD have indicated that pain onset might be an important factor to consider. The issue of whether the pain has been present since the patient's first episode of vaginal penetration (ie, lifelong or primary PVD, referred to as PVD1) or after a period of pain-free activities (ie, acquired or secondary PVD, referred to as PVD2) can influence pain sensitivity (eg, Sutton et al[2]) and treatment outcome (eg, Heddini et al[3]; for a review of distinct and overlapping factors in PVD1 and PVD2, see Pukall[4]).

In this article, *genital pain* refers to the report of any kind of genital or abdominal pain (eg, from vulvodynia, chronic pelvic pain, or undiagnosed conditions); *vulvodynia* refers to the general condition of idiopathic, chronic vulvar pain; *provoked vestibulodynia* refers to provoked vestibular pain; and *dyspareunia* describes a common symptom of many genital pain conditions (including, but not limited to, vulvodynia; eg, chronic pelvic pain and pain during penetrative sexual activities).

PREVALENCE

Prevalence studies of vulvodynia have indicated that it is prevalent, with lifetime estimates ranging from 10% to 28% in reproductive-aged women in the general population.[5–8] A recently published study by Harlow et al[9] indicated that 8% of women 18 to 40 years old reported a history of vulvar burning or pain upon contact that persisted longer than 3 months and that limited or prevented intercourse. These researchers also replicated previous work demonstrating that women of Hispanic origin were more likely to develop vulvar pain symptoms compared with white women.[8,10]

IMPACT

A recent non-probability survey indicated the costs of vulvodynia in the United States to be 31 to 72 billion dollars annually.[11] This staggering amount includes direct health care costs (eg, insurance payments and out-of-pocket expenses), direct non-health care costs (eg, transportation), and indirect costs (financial loss owing to medical leave from work and employer payments to patients for medically related work loss). However, this figure does not taken into account the very significant psychological burden of vulvodynia.

PATHOPHYSIOLOGIC FACTORS

Numerous factors have been suggested to play a role in the initiation and/or maintenance of vulvodynia (Table 1). These factors are interdependent and likely act within a cyclical model[12]; however, the direction of causality is not clear given the lack of prospective, longitudinal studies.

Vulvar (Neuro-Proliferative) Factors

Although hypersensitivity of the vulvar vestibule is one of the defining characteristics of vulvodynia—in particular PVD—the underlying mechanism of this allodynia was not elucidated until 1998. In that year, Weström and Willén[13] and Bohm-Starke et al[14] used immunohistochemical (IHC) staining to visualize an increase in the density of nerve endings in the vestibular endoderm of women with PVD who had undergone vulvar vestibulectomy compared with controls. Bohm-Starke et al[15] followed up their original research with an additional study that identified these increased nerve endings as nociceptors. They postulated that the neuro-proliferation of these nociceptors could explain the perceived allodynia. Other research teams have since confirmed the findings of these initial studies (eg, Tympanidis et al,[16] Halperin et al,[17] Leclair et al,[18] Goetsch et al[19]). Increased innervation has implications for increased sensitivity, and the phenomenon of increased sensitivity has been documented in women with PVD. For example, a quantitative sensory testing study conducted by Pukall et al[20] found that women with PVD were more sensitive to punctate tactile and pain stimuli applied to four vestibular sites compared with control women. A similar pattern of findings in response to various forms of stimulation (eg, thermal and pressure pain) has been reported by this and other groups (eg, Bohm-Starke et al,[21] Giesecke et al,[22] Pukall et al,[23,24] Sutton et al,[25] Smith et al,[26] Heddini et al[27]). Interestingly, the heightened sensitivity does not appear to be limited to static stimuli[28] or the vestibule (eg, forearm[20,29,30]). Research conducted by Heddini et al[27] implicated