

Topical review

Genital pain in women: Beyond interference with intercourse

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1. Introduction

Pelvic pain problems affecting women's sexual and reproductive health are poorly understood and often misdiagnosed or ignored [21]. In addition to their high prevalence rates, from 12% to 21% in community samples, these conditions can impinge on patients' sexual functioning, psychological well-being, and quality of life [1]. Recommendations for the classification of sexual pain in the upcoming *Diagnostic and Statistical Manual of Mental Disorders-V* suggest collapsing dyspareunia and vaginismus into a single diagnostic entity called *genito-pelvic pain/penetration disorder*, which comprises five elements: percentage success of vaginal penetration, pain with vaginal penetration, fear of vaginal penetration or of pain during vaginal penetration, pelvic floor muscle dysfunction, and medical comorbidity [6]. Noteworthy is the emphasis on the multidimensional aspects of sexual pain, which represents an improvement over previous classifications. Yet genital pain remains within the category of sexual dysfunction, despite accumulating evidence in favour of a pain conceptualization. Further, the diagnosis still focuses heavily on the interference of the pain with a single type of sexual activity, intercourse. These classification challenges are reflected in our search for etiological explanations, with proponents of biomedical causes on one side [8] and those adhering to psychosexual models on the other [17]. We propose an integrated model taking into account the interdependency of biomedical – including pelvic floor muscle dysfunctions – cognitive, affective, behavioural, and interpersonal factors in genital pain and associated sexual impairment. Specifically, this model acknowledges that the pain experienced by women goes beyond an interference with intercourse, affecting and being affected by other sexual and nonsexual aspects of their lives.

2. Biomedical factors

Up until recently, most biomedical hypotheses had been derived from poorly controlled studies. In the last 10 years however,

the quality of the research has greatly improved, although it has pertained almost exclusively to provoked vestibulodynia – a burning pain triggered by pressure applied to the vulvar vestibule. The following factors have been found to be more common in women with genital pain than in controls: early puberty and pain with first tampon use [23], a history of repeated yeast infections [3], early and prolonged use of oral contraceptives [11], polymorphisms in genes regulating inflammatory response [19], nociceptor proliferation and sensitization [9,10], and lower touch and pain thresholds in the vulvar area as well as other body locations [31]. Taken together, findings suggest that both peripheral and central mechanisms are at play in the onset and maintenance of genital pain. Pelvic floor muscle dysfunctions figure prominently among the mechanisms receiving increased empirical attention.

3. Pelvic floor muscle dysfunctions

Studies to date indicate that abnormality at rest is the dysfunction of the pelvic floor muscles (PFM) that seems of greatest importance in the pathophysiology of genital pain. Different terminologies have been used to describe this abnormality, namely overactivity [29], hypertonicity [34], spasm [33], and spasticity [25]. Such abnormality is hypothesized to close the vaginal hiatus and interfere with penetration [4]. PFM hypertonicity evaluated by digital palpation [33,34] as well as increases in resting electromyographic (EMG) activity [18] were found in women with vestibulodynia and vaginismus compared to controls. It has been suggested that women with dyspareunia may exhibit a protective-like defensive reaction of the PFM during attempted vaginal penetration [33,34]. Shafik and El-Sibai [36] confirmed that reaction by showing greater resting EMG activity in women with vaginismus during dilator insertion compared to asymptomatic women.

Some studies have also shown that women with dyspareunia present deficits in the PFM contractile capacities. Reductions in strength and endurance were observed in women with dyspareunia compared to controls using palpation [33,34] and EMG [20,33]. However, measurement issues limit the validity of these findings, as EMG is not suitable for inter-subject comparison [2] and digital palpation is criticized for being a subjective measure [7]. New methodologies such as dynamometry and ultrasound appear more promising [27,30].

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Whether PFM dysfunctions are a consequence or a cause of genital pain remains unknown. It has been hypothesized that genital pain and inflammation may provoke PFM dysfunctions by (1) destabilizing the PFM, inducing tensions and poor muscle control [20]; (2) triggering a defensive-like reaction toward pain during penetration attempts [33,34]; (3) intruding the underlying muscle tissues and causing hypersensitivity in the PFM [39]. Others have suggested that PFM dysfunctions may initiate an increase in mucosal sensitivity [40]. It is likely that a vicious cycle involving pain and further muscle dysfunctions is implicated, potentially compounded by the involvement of cognitive, affective, and behavioural factors.

4. Cognitive, affective, and behavioural factors

Research published in the last decade indicates that psychosocial factors may contribute to the onset of genital pain problems. In a study involving over 1400 adolescent girls, those complaining of dyspareunia of at least 6 months' duration were more likely to report past sexual abuse, fear of physical abuse, and trait anxiety in comparison to no-pain controls [26]. Additionally, the first study focusing on victimization in a general population adult sample showed that severe physical or sexual childhood abuse was linked to a 4- to 6-fold risk of reporting genital pain in adulthood [22]. These findings need not be interpreted solely in light of a sexual development perspective. Longitudinal work following individuals from birth to 32 years has shown that exposure to childhood maltreatment is linked to clinically significant biomarkers of inflammation in adulthood such as C-reactive protein levels, suggesting that inflammation may mediate the effect of adverse early life experiences on adult health [14]. Further, in a randomized treatment outcome study, pretreatment levels of erotophobia – the tendency to respond with negative affect to sexual cues – were found to account for 27% of the variance in a surgical outcome for vestibulodynia, independent of pretreatment pain [5]. Taken together, these results indicate that psychosocial factors may contribute to the development and persistence of genital pain.

Contrary to clinical lore, pain and sexual impairment may be distinct and partially independent phenomena, as shown by the lack of significant correlation between pain and sexual function, as well as pain and sexual satisfaction, in a recent study focusing on women with vestibulodynia [35]. It may be more fruitful to search for different predictors of these two critical outcomes. One such study indicated that higher levels of hypervigilance, fear of pain, and catastrophizing were significant predictors of increased pain in this population, whereas greater anxiety and avoidance were associated with poorer sexual function. However, results also showed that lower levels of self-efficacy were related to worse pain and sexual function, suggesting that some variables may affect both outcomes indiscriminately [15].

Because research examining the relation between psychosocial factors and genital pain is still in its infancy, the majority of studies have been descriptive and cross-sectional. Such designs do not elucidate the extent to which pain intensity may drive patients to fear and avoid threat or whether fear and anxiety precede the experience of pain. The fear-avoidance model – whereby an initial pain experience possibly caused by an injury may be interpreted as threatening (catastrophizing), leading to fear of pain and to avoidant behaviours, which in turn lead to hypervigilance followed by disability (in this case, sexual dysfunction), disuse (potential reduction of the sexual repertoire), and depression – may provide a useful framework for integrating findings concerning muscular reactivity and psychological factors [38], especially when expanding the focus from intraindividual to interpersonal variables.

5. Interpersonal factors

Surprisingly, relationship variables have been widely neglected in dyspareunia and vaginismus. Although there appear to be no differences in self-reported dyadic adjustment [28] and no association between dyadic adjustment and pain in women with dyspareunia [16], interpersonal factors remain relevant given that partners both elicit and witness women's pain experience. Recent studies have identified key relational variables, including attachment orientation, attributions, and partner responses that underscore the need for further research in this area [16,24].

Communicating pain to a significant other may serve to evoke empathic responses, assistance, or to maximize proximity to one's partner [37]. In turn, partner responses to expressions of pain may reinforce and perpetuate patients' pain experience [13,35]. In women with vestibulodynia, higher solicitous partner responses were associated with greater pain during intercourse [16,35]. Partner solicitousness may encourage avoidance of sexual activity and enhance negative cognitive-affective factors, which in turn heighten pain. Greater solicitous and lower negative partner responses were also associated with more sexual satisfaction in women [35]. Partner sensitivity to women's pain may impact sexual satisfaction because couples may be more or less likely to focus on alternative sexual activities to penetration or on the emotional benefits of sexual activity [35].

What type of partner response will minimize women's pain intensity, but maximize her sexual satisfaction? Facilitative responses, in which the partner encourages the patient's efforts at coping with the pain, warrant further investigation as they are associated with less patient disability in chronic pain populations [32]. In women with genital pain, facilitative responses may promote engaging in sexual activity by focusing on less painful activities or by expressing affection and pleasure during or after sexual activity.

Recent calls in the literature have lobbied for moving beyond cognitive-behavioural models of pain interactions in couples to include intimacy models [12]. Cognitive-behavioural models are limited in their focus on pain ratings and descriptions of the pain, often failing to capture pain-related distress, which can be another facet of verbal pain behaviours [12]. In contrast, intimacy models emphasize emotional disclosure and subsequent validating or invalidating partner responses. While cognitive-behavioural models view partner responses as reinforcing pain behaviours, intimacy models assert that these may enhance or disrupt intimacy and emotion regulation in couples [12]. It may be that the association between women's genital pain and disclosure about the pain will vary according to the partner's level of emotional validation, which in turn may contribute to pain persistence. Researchers will benefit from drawing on several models of interpersonal processes in order to better understand the role of dyadic interactions in couples with genital pain.

6. Conclusion

Fragmentation of scientists into specific disciplines, ongoing classification issues, and lack of sound research designs all impede progress concerning the identification of etiologic factors and the development of tailored interventions for women with genital pain. Empirical evidence to date suggests the existence of multiple etiologic pathways leading to the development and persistence of genital pain. We suggest that initial biomedical/mechanical trauma to the genitalia may trigger inflammatory processes, pelvic floor muscle dysfunctions, and other local changes leading to nociceptor sensitization and further peripheral and central alterations in pain processing. The experience of pain combined with a lack of proper diagnosis and treatment by health professionals [21] may interact

with individual predispositions and generate varying degrees of psychological distress in patients and their partners. In turn, cognitive, behavioural, affective, and interpersonal factors may modulate pain intensity as well as associated sexual impairment, as per the fear-avoidance model [38], such that not all women who have an initial experience of pain are at risk of suffering from a persistent condition, or of developing sexual dysfunction. Further, predictors of the pain experience can be different from those of the negative sexual and psychological symptomatology. Genital pain may be best conceptualized as a multidimensional persistent pain condition that constitutes the endpoint of multiple etiologic trajectories and whose understanding goes beyond its interference with intercourse.

Further progress in the genital pain field demands an expansion of our research methodologies. First, future research should include both members of the couples in order to examine the dynamic interactions of interpersonal processes on pain and associated psychological distress and sexual impairment. Second, it is important to venture beyond the study of clinical samples, which are probably biased in the direction of increased symptomatology. Third, researchers should use prospective and experimental designs such as observational and experience processing methods to tease apart the temporal order of the associations among women's behaviours, partner responses, pain, and psychosexual variables. Fourth, biomedical factors such as pelvic floor muscle dysfunctions should be investigated using better validated measurements to understand further their implication in the pathophysiology of genital pain. Such studies will contribute to the development of more refined, biopsychosocial etiological models of genital pain and will inform the development of targeted interventions.

Conflict of interest statement

None of the authors has a conflict of interest.

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