



International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
Canada
+15878858911
editorial-office@sciformat.ca

ARTICLE TITLE	VULVODYNIA AS A CHRONIC VULVAR PAIN DISORDER: A NARRATIVE REVIEW OF PATHOPHYSIOLOGY, DIAGNOSIS, AND MANAGEMENT
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DOI	https://doi.org/10.31435/ijitss.2(50).2026.5312
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RECEIVED	16 March 2026
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ACCEPTED	26 June 2026
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PUBLISHED	30 June 2026
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VULVODYNIA AS A CHRONIC VULVAR PAIN DISORDER: A NARRATIVE REVIEW OF PATHOPHYSIOLOGY, DIAGNOSIS, AND MANAGEMENT

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ABSTRACT

Background: Vulvodynia is a chronic vulvar pain condition frequently associated with dyspareunia and significant impairment of quality of life in women of all ages. Despite increasing clinical awareness, diagnosis and management remain challenging due to its heterogeneous presentation and multifactorial etiology.

Aim: This review aims to evaluate the current understanding of the pathogenesis, clinical presentation, diagnostic approaches, and management strategies of vulvodynia, and to synthesize available evidence to support clinical practice and guide future research.

Methods: A narrative review of the literature was conducted using PubMed, Scopus, Web of Science, and Embase, focusing on publications from 2005–2025. Additional sources included reference screening and international society guidelines. Narrative and systematic reviews, meta-analyses, consensus statements, clinical guidelines, and relevant observational or experimental studies were included. Due to methodological heterogeneity, findings were synthesized narratively.

Results: According to the International Society for the Study of Vulvovaginal Disease, vulvodynia is defined as vulvar pain lasting at least three months without an identifiable cause. Current evidence suggests a multifactorial pathophysiology involving neurogenic, inflammatory, myofascial, hormonal, and psychosocial mechanisms, including central sensitization. Diagnosis relies on detailed history-taking, comprehensive physical examination, targeted pain assessment, and exclusion of other gynecologic, dermatologic, or neurologic conditions. Management increasingly emphasizes a multimodal, interdisciplinary approach combining patient education, pelvic floor physical therapy, psychological interventions, and pharmacological treatment.

Conclusions: Vulvodynia is a complex chronic pain disorder requiring individualized, patient-centered care. Interdisciplinary management tailored to underlying pain mechanisms and coexisting factors is essential to improve clinical outcomes and quality of life.

KEYWORDS

Vulvodynia, Vestibulodynia, Dyspareunia, Chronic Pelvic Pain, Central Sensitization, Pelvic Floor Dysfunction

CITATION

Agnieszka Dobrowolska, Adam Skrobarczyk, Zuzanna Drewniak, Marta Rząsa, Karolina Rycman, Martyna Sternik, Kamil Wendrychowicz, Filip Wróbel. (2026) Vulvodynia as a Chronic Vulvar Pain Disorder: a Narrative Review of Pathophysiology, Diagnosis, and Management. *International Journal of Innovative Technologies in Social Science*. 2(50). doi: 10.31435/ijitss.2(50).2026.5312

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1. Introduction

Vulvar pain and dyspareunia are frequently reported symptoms among women across a wide age spectrum. Despite the fact that many affected patients seek gynecologic evaluation, these complaints continue to present a substantial diagnostic and therapeutic challenge in clinical practice. Vulvodynia presents as vulvar pain, occurring either spontaneously or in response to contact, and may arise during attempted or completed penetration as well as in various sexual or non-sexual contexts [1].

Since 2003 specialists, and researchers have relied on terminology of the International Society for the Study of Vulvovaginal Disease (ISSVD) as a framework for diagnosing vulvar pain. This terminology emerged from years of discussion about the nature of idiopathic vulvar pain [2]. In 2015, a consensus meeting on the classification of pelvic pain was convened among the the International Society for the Study of Vulvovaginal Disease (ISSVD), the International Society for the Study of Women's Sexual Health (ISSWSH), the International Pelvic Pain Society (IPPS) and representatives of the American Congress of Obstetricians and Gynecologists (ACOG), of the American Society for Colposcopy and Cervical Pathology (ASCCP), and of the National Vulvodynia Association (NVA) [3]. The revised taxonomy clearly distinguishes vulvar pain attributable to a specific underlying condition - such as infectious, inflammatory, neoplastic, neurologic, traumatic, iatrogenic, or hormonal causes including the genitourinary syndrome, menopause - from vulvodynia, for which no definitive etiology can be identified. The consensus introduced the following definition of vulvodynia: vulvar pain of at least three months' duration, without a clearly identifiable cause, which may

have potential associated factors [4]. In addition to the diagnostic criteria necessary to establish the condition, vulvodynia is further categorized according to the location of pain (e.g., localized or generalized), its temporal pattern (e.g., intermittent or continuous), the factors that precipitate symptoms (e.g., provoked vestibulodynia or spontaneous), and the onset of symptomatology (primary or secondary) [5].

The exact prevalence of vulvodynia has not been fully established, current estimates suggest that 8 - 12% of women may be affected. However, given the condition's multifactorial nature and the frequently prolonged diagnostic process, the true proportion is presumed to be substantially higher [6]. In light of evolving definitions, increasing recognition of underlying pain mechanisms, and persistent challenges in diagnosis and management, a comprehensive narrative review is warranted. The aim of this review is to synthesize current evidence regarding the classification, pathophysiology, diagnostic approach, and contemporary management strategies for vulvodynia.

2. Methods

This narrative review was conducted to summarize and critically appraise current evidence on vulvodynia, including its definition, classification, pathophysiology, diagnosis, and management. The study selection process was predefined and transparent. A structured literature search was performed using the PubMed, Scopus, Web of Science, and Embase databases, focusing primarily on articles published between 2005 and 2025. The search strategy combined MeSH terms and free-text keywords, including "vulvodynia," "vulvar pain," "vestibulodynia," "dyspareunia," "chronic pelvic pain," "central sensitization," and "pelvic floor dysfunction."

Titles and abstracts of retrieved records were screened to identify studies relevant to the scope of this review. Full-text articles were subsequently assessed for eligibility based on predefined inclusion criteria.

Eligible publications included narrative and systematic reviews, meta-analyses, consensus statements, clinical guidelines, and original observational or experimental studies relevant to the topic. Foundational and landmark studies were included regardless of publication year to provide appropriate historical context, while priority was given to more recent literature to ensure clinical relevance. Articles were selected based on their relevance to the scope of the review and the quality of the scientific evidence regarding the definition, pathophysiology, diagnosis, and management of vulvodynia.

The literature was synthesized narratively due to heterogeneity in study designs, populations, and outcome measures, which precluded quantitative synthesis. Sufficient methodological detail regarding the search strategy, inclusion criteria, and synthesis approach is provided to ensure transparency and reproducibility of the review process.

3. Aims and research objectives

The primary aim of this narrative review is to summarize and critically evaluate the current state of knowledge regarding vulvodynia as a chronic vulvar pain condition. The specific research objectives are as follows:

- To present contemporary definitions and classification systems of vulvodynia proposed by international scientific societies.
- To analyze current evidence on the multifactorial pathophysiology of vulvodynia, including neurogenic, inflammatory, myofascial, hormonal, and psychosocial mechanisms.
- To review diagnostic approaches to vulvodynia, with particular emphasis on clinical evaluation, exclusion of alternative etiologies, and assessment of coexisting pain conditions.
- To summarize available evidence on pharmacological and non-pharmacological treatment strategies, highlighting multimodal and interdisciplinary management approaches.
- To identify existing knowledge gaps and outline directions for future research and clinical practice in the management of vulvodynia.

4. Results

4.1 Pathophysiology

According to the consensus of the International Society for the Study of Vulvovaginal Disease (ISSVD), vulvodynia is defined as vulvar pain lasting at least three months without a clearly identifiable cause, which may be associated with a range of potential contributing factors. Chronic vulvar pain is a condition that has affected women for many years. By the late 19th century, Dr. Thomas had reported cases of women presenting with “pronounced hypersensitivity of the nerves innervating the mucosal tissues of the vulva” [3, 7]. For many years, efforts have been made to elucidate the mechanisms underlying this pain disorder, however, vulvodynia does not constitute a uniform clinical entity, and no single pathophysiological process fully accounts for its development. Current evidence indicates a complex, multifactorial etiology involving concurrent neurogenic, immunologic, myofascial, and psychosocial dysfunctions [3].

Vulvodynia is defined as pain without an identifiable cause, however, its development may involve a number of contributing factors and conditions that share common pathophysiological mechanisms and are likewise classified as pain syndromes, e.g.:

- Chronic fatigue syndrome,
- Chronic low back pain,
- Endometriosis,
- Fibromyalgia,
- Temporomandibular disorders[8][9]

In 2011, the Institute of Medicine released the Relieving Pain in America report, which underscored the significance of Chronic Overlapping Pain Conditions (COPCs). Ten COPCs were identified, which tend to co-occur in various combinations at rates higher than those observed in association with other chronic diseases. Each of these conditions may occur independently, however, a localized pain presentation is less common [10][9]. Individuals with conditions within this group often exhibit heightened sensitivity to stimuli, pain in response to normally nonpainful stimulation (i.e. allodynia), expansion of the receptive field, and other comorbid symptoms, including psychiatric disorders such as depression and anxiety disorders. [11]. An expanding body of evidence indicates that COPCs share common pathophysiological mechanisms involving peripheral and central sensitization that contribute to the development and amplification of pain. A growing number of studies further demonstrate that, statistically, a higher number of co-occurring COPCs is associated with greater pain severity in endometriosis [12].

Central sensitization (CS) is one of the possible mechanisms explaining the shared pathophysiology of COPCs and has also been investigated in the context of vulvodynia [13]. According to International Association for the Study of Pain, CS is, “Increased responsiveness of nociceptive neurons in the central nervous system to their normal or subthreshold afferent input” [13, 14]. The mechanisms underlying vulvodynia are not fully understood, however, features supporting a nociplastic pain phenotype include increased generalized pain sensitivity, brain MRI changes observed during vulvar pressure, and the occurrence of pain following sexual intercourse. This indicates that central sensitization may be involved in the pathophysiology of chronic pain, and that a deeper understanding of this process may be crucial for optimizing patient management and improving clinical outcomes [13].

At the peripheral level, evidence indicates that women with vulvodynia present with a higher density of intraepithelial nerve fibers in the mucosal tissue of the vulvar vestibule [15]. Additionally, painful regions are marked by an increased infiltration of inflammatory cells, including T and B lymphocytes and macrophages, which may play a contributory role in the neuroproliferative changes observed in this condition [15, 16].

Another proposed explanation for the development of vulvodynia focuses on the role of sex steroid hormone levels in women. Fluctuations or disturbances in the concentration of sex hormones may affect pain modulation mechanisms [17]. For instance, estrogen and progesterone deficiency in the postmenopausal period may be associated with decreased pain thresholds and an increased susceptibility to vulvodynia [18]. The potential influence of oral hormonal contraceptive therapy on the development of vulvodynia is currently under consideration. Research conducted to date does not provide a definitive answer as to whether the use of oral contraceptives prior to the onset of pain may have contributed to its development [17, 19].

Based on available scientific evidence, it can be postulated that vulvodynia may be associated with excessive tension of both superficial and deep pelvic floor muscles, as well as an impaired ability to relax these muscles during defecation or micturition [20]. Activation of the deep pelvic floor musculature may result in a lowered pain threshold and reduced pain tolerance [21]. Several factors potentially contributing to pelvic floor muscle overactivity have been identified, including a history of acute infection and prolonged sitting. These

factors may influence alterations in the function of the myofascial system [2]. However, it remains unclear whether abnormalities of the pelvic floor muscles and fascia represent a primary cause or a secondary consequence of chronic vulvar pain [21].

An increasing body of evidence also indicates that women with vulvodynia more frequently report a history of mood and anxiety disorders; some studies suggest that these conditions occur at rates up to twice as high as those observed in control populations [22]. Moreover, some studies suggest that negative sexual and relational experiences are considered factors that may modulate and perpetuate pain [23]. However, the origins may extend even further back, as it is hypothesized that early childhood trauma and insufficient psychosocial support during this period may constitute predisposing factors for the development of vulvodynia in adulthood [24]. All of these experiences, occurring in both early and later stages of life, may lead to long-term alterations in pain processing, stress regulation, and pelvic floor muscle function [22, 24]. Additionally, the association between vulvodynia and mental health disorders suggests a bidirectional relationship between pain and psychological health [23]. A range of psychiatric disorders may develop in women exposed to chronic pain that significantly interferes with daily functioning. This underscores the profound psychosocial impact of vulvodynia and the substantial psychological burden experienced by women living with chronic pain [25].

In a subset of patients, peripheral sensitization combined with psychological predispositions may lead to central sensitization and the development of chronic pain - Fig.1 [21].

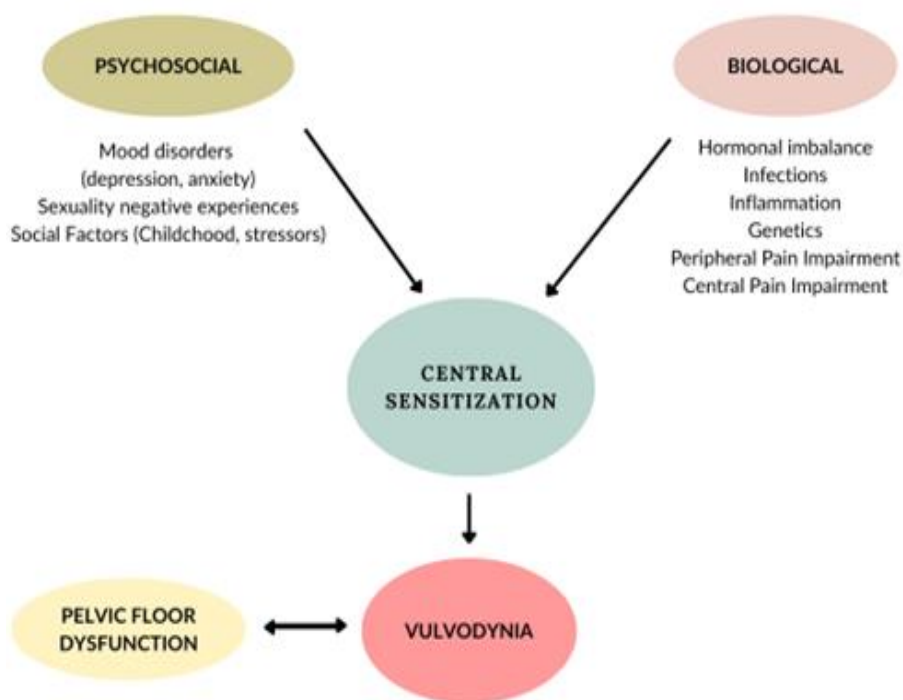


Fig. 1. Pathophysiology of vulvodynia

4.2 Diagnostics

A detailed medical history constitutes the cornerstone of the diagnostic process in vulvodynia, aiming not only to characterize the symptoms but also to identify coexisting factors as well as triggers or aggravating factors of pain. Vulvodynia is a diagnosis of exclusion, therefore, careful elimination of alternative etiologies is essential [26]. According to current criteria, vulvodynia is defined as vulvar pain persisting for at least three months in the absence of an identifiable cause [2]. A properly obtained medical history should include:

- Duration of pain,
- Pain characteristics (e.g. burning, stabbing, neuropathic pain),
- Pain localization,
- Coexisting pain syndromes (COPCs),
- Use of hormonal therapy, including oral contraceptives (OCs),
- Previously used medications,
- Aggravating and relieving factors,

- Past medical history,
- History of surgical procedures,
- Sexual history,
- Previous treatment modalities [26, 27].

The medical history should also include information regarding recurrent vulvovaginal infections, previous treatments for such infections, prior or current antibiotic use, and exposure to potential contact allergens, including cosmetic products, intimate hygiene agents, and sanitary pads. It is also important to inquire about a family medical history [28].

The diagnosis of vulvodynia should be considered in the presence of symptoms such as persistent vulvar pain described as burning or pressure - like, sometimes radiating to the thighs, buttocks, and abdominal region. The symptoms are not exacerbated by external factors, and the pain is typically constant. This clinical presentation predominates in postmenopausal women. The second group comprises younger women who report pain during sexual intercourse and tampon use, accompanied by a sensation that the vagina is “too small.” Symptoms such as burning and dysuria may persist for several hours after sexual intercourse [29].

After a detailed medical history has been obtained, a physical examination should be performed. If vulvodynia is suspected, assessment for allodynia (pain elicited by a normally non-painful stimulus) should be conducted using the cotton swab test. Gentle pressure with the cotton swab should begin on the thigh and proceed medially toward the labia majora, the interlabial sulcus, the clitoral hood, the labia minora, and the points within the vaginal vestibule corresponding to the 2, 4, 6, 8, 10, and 12 o'clock positions. The patient rates pain intensity on a 0–10 numerical rating scale (NRS). Vulvodynia may be classified as localized (when pain is confined to the vaginal vestibule) or generalized (when pain extends beyond the vestibular area) [29–31].

Examination of the vagina and cervix allows assessment of the mucosa (atrophy, erosions) and any features suggestive of infection. The character and quantity of vaginal discharge, as well as changes indicating an active infectious process, should also be evaluated. Assessment of the discharge enables preliminary identification or exclusion of vaginitis, such as candidiasis, bacterial vaginosis, or sexually transmitted infections [29]. Vaginal discharge can be evaluated by measuring vaginal pH and obtaining a sample for Gram staining and culture. Gram staining of the vaginal discharge allows assessment of markers of inflammation or their absence, including the presence of lactobacilli, clue cells, leukocytes, and active yeast forms. An acidic pH, the presence of lactobacilli, and the absence of clue cells support exclusion of bacterial vaginosis. A negative culture may be helpful in ruling out vulvovaginal candidiasis. In cases where sexually transmitted infections are suspected, nucleic acid amplification testing (PCR) for common sexually transmitted pathogens, including *Trichomonas vaginalis* and *Chlamydia trachomatis*, should be considered [32]. Additionally, any suspicious, non-healing, or ulcerated vulvar lesion requires oncologic evaluation, usually including biopsy, to exclude premalignant conditions such as vulvar intraepithelial neoplasia (VIN) or vulvar carcinoma [28].

Another integral component of the physical examination is assessment of the pelvic floor muscles. This evaluation includes assessment of resting muscle tone, symmetry, ability to relax, and the presence of tenderness to palpation and myofascial trigger points. Studies have demonstrated that increased pelvic floor muscle tone, referred to as high-tone pelvic floor dysfunction or myofascial pelvic pain, occurs significantly more frequently in women with vulvodynia than in the general population [21, 30]. Chronic activation of the pelvic floor muscles promotes the persistence of central pain mechanisms and peripheral sensitization, which may exacerbate symptoms and complicate treatment [27].

An alternative to the cotton swab test that provides more comprehensive clinical information is the use of the VAMP protocol. The gynecologist assesses four regions: the vulva (V) and anus (A) using cotton swab pressure, the pelvic floor muscles (M) through digital palpation of the levator ani muscle, and the periurethral area (P) by targeted palpation. The patient rates pain intensity using the NRS from 0 to 10, where 0 indicates no pain and 10 represents the worst imaginable pain:

- vulva (V) – pressure with a dry cotton swab applied to five points at the base of the hymenal ring,
- anus (A) – pressure applied to two points surrounding the anal margin,
- pelvic floor muscles (M) – bimanual vaginal examination using the index finger with lubricant, the finger is directed posteriorly and moved bilaterally along the pelvic floor, applying pressure to the most superficial muscle layers,
- periurethral area (P) – the index finger is rotated upward and laterally from the urethra, applying pressure to the periurethral fascia from external to internal on both sides.

The examination takes approximately two minutes and may provide valuable information to guide further therapeutic management. A VAMP score greater than 3 in at least one assessed region indicates an overactive state of the pelvic floor muscles [20, 21].

In conclusion, vulvodynia should be diagnosed only after careful exclusion of alternative causes of vulvar pain. Failure to recognize underlying infections, inflammatory dermatoses, or pelvic floor dysfunction may lead to misdiagnosis and inappropriate management. Comprehensive diagnostic evaluation remains the cornerstone of evidence-based care for patients presenting with chronic vulvar pain [2].

4.3 Management

Management of vulvodynia should be multimodal and individualized to each patient's needs. Because multiple pain mechanisms often coexist, a thorough clinical history and comprehensive examination are essential. In practice, optimal care involves integrating patient education with both pharmacologic and nonpharmacologic treatment strategies [28, 31].

The therapeutic process should begin with patient education regarding vulvodynia. Patients should be informed about factors that may exacerbate irritation and pain, such as perfumed sanitary pads and strongly scented detergents, and advised on strategies to reduce friction and mechanical trauma to the vulvar area [26].

In most patients, increased pelvic floor muscle tone is present and may both provoke and perpetuate pain and dyspareunia. In such cases, specialized urogynecologic pelvic floor physical therapy may provide the greatest benefit by promoting muscle relaxation, addressing myofascial trigger points, and facilitating appropriate functional use of the pelvic floor musculature [31]. The goals of physical therapy also include improving body awareness and neuromuscular control. Evidence suggests that regular therapeutic exercises lead to significant pain reduction and improvement in sexual function [33][31][34].

Another essential component of nonpharmacological management is the patient's participation in cognitive-behavioral therapy, interventions specifically targeting chronic pain, and, when indicated, sex therapy (e.g., management of dyspareunia, graded exposure, and techniques aimed at reducing pain-related anxiety). Vulvodynia has a substantial negative impact on quality of life, and factors such as fear of pain and mood disturbances secondary to chronic pain may further exacerbate symptoms [26][35].

Pharmacological treatment can be divided into topical, oral, and intralesional therapies, as well as pudendal nerve blocks and botulinum toxin injections [36]. The most commonly used topical agent Lidocaine 2-5%, applied as an ointment or gel, used either immediately before intercourse or for prolonged use. It is employed as a strategy to reduce allodynia and peripheral hypersensitivity [29]. Topical estrogen therapy - often used in combination with lidocaine, has been shown to alleviate the adverse effects of vulvovaginal atrophy on sexual health in postmenopausal women. The impact of hormonal treatments in women prior to menopause has not yet been fully characterized [1, 31]. Sometimes, 1% hydrocortisone creams are occasionally used to reduce inflammation; however, prolonged treatment may lead to atrophy of the vulvar skin and mucosa [31].

When topical therapy fails to provide adequate symptom relief, oral medications are frequently prescribed. Agents used in the management of chronic neuropathic pain include oral tricyclic antidepressants (TCAs) - most commonly amitriptyline, serotonin - norepinephrine reuptake inhibitors (SNRIs), selective serotonin reuptake inhibitors (SSRIs), and antiepileptic drugs such as gabapentin and pregabalin. Although amitriptyline is not formally approved for this indication, it is commonly used in the treatment of vulvodynia and has demonstrated favorable analgesic effects in studies conducted to date. In contrast, the clinical efficacy of the other aforementioned pharmacologic agents in vulvodynia has not yet been fully established [1, 31].

Several studies have explored the use of botulinum toxin type A for the reduction of pelvic floor muscle hypertonicity in patients unresponsive to pelvic floor physical therapy. Although its efficacy in the treatment of vulvodynia has not been conclusively demonstrated, it continues to be employed in the management of chronic pain, and further investigation is ongoing [1].

Additional modalities used in the management of chronic pain include electrical nerve stimulation - particularly in women with localized vulvodynia, treatment with platelet - rich plasma injections, and, in selected severe cases, surgical intervention in the form of vestibulectomy [37].

Vulvodynia is a condition with a complex pathophysiology and heterogeneous clinical presentations among women. Its management requires an individualized, stepwise, and multidisciplinary approach based on pain phenotyping and the identification of coexisting factors. Effective treatment most often necessitates a combination of pharmacological and nonpharmacological interventions. Collaboration among multiple specialists and careful monitoring of therapeutic outcomes are frequently essential to achieve optimal results [28, 29, 31].

5 Discussion

This narrative review highlights vulvodynia as a complex chronic pain condition characterized by heterogeneous clinical presentations and a multifactorial pathophysiology. The reviewed evidence demonstrates that vulvodynia cannot be explained by a single etiological mechanism. Rather, it arises from the interaction of peripheral and central pain processes, pelvic floor dysfunction, hormonal influences, and psychosocial factors [1, 3]. These findings place vulvodynia within the broader framework of chronic overlapping pain conditions and nociplastic pain syndromes, providing a clinically meaningful context for understanding symptom persistence and treatment resistance.

From a pathophysiological perspective, increasing evidence supports the role of central sensitization and altered pain processing in a subset of patients, alongside peripheral mechanisms such as neuroproliferation and local inflammation [2, 10]. Importantly, these mechanisms are not mutually exclusive and may coexist within the same individual, which partially explains the variability in clinical presentation and therapeutic response [1, 36]. The reviewed literature consistently emphasizes that failure to recognize this complexity may lead to underdiagnosis or ineffective, single-modality treatment approaches [2].

Diagnostic strategies discussed in this review underscore the importance of a thorough medical history, careful physical examination, and exclusion of alternative gynecologic, dermatologic, infectious, and neurologic conditions [29]. The incorporation of targeted pain assessment and evaluation of pelvic floor muscle function is particularly relevant, as myofascial dysfunction appears to play a significant role in symptom generation and maintenance in many patients [29]. Placing diagnostic findings within a biopsychosocial framework allows clinicians to better contextualize symptoms without attributing pain solely to psychological factors, thereby supporting a comprehensive and balanced diagnostic approach.

With regard to management, the evidence reviewed consistently supports an interdisciplinary and multimodal treatment strategy. Nonpharmacological interventions, including patient education, pelvic floor physical therapy, and psychological therapies, are increasingly recognized as essential components of care and should be considered first-line approaches in many patients [1, 29]. Pharmacological treatments may provide symptom relief in selected cases, however, current evidence does not support the existence of a universally effective medication, reinforcing the need for individualized treatment plans based on pain mechanisms and patient-specific factors [36]. Surgical and other invasive interventions should be reserved for carefully selected patients after failure of conservative management strategies.

Several limitations of the current evidence base should be acknowledged. Much of the available literature consists of observational studies, small clinical trials, and expert consensus statements, and there is considerable heterogeneity in diagnostic criteria, outcome measures, and study populations. These factors limit direct comparison between studies and preclude quantitative synthesis. Rather than representing fatal limitations, these gaps highlight important opportunities for future research, including the development of standardized diagnostic tools, refinement of phenotypic classification, and the design of longitudinal and mechanism-based therapeutic studies [2, 27, 29, 31].

6 Conclusions

Vulvodynia is a chronic vulvar pain disorder characterized by heterogeneous clinical presentation and complex, multifactorial pathophysiology. Available evidence indicates that the condition arises from the interaction of peripheral and central pain mechanisms, including central sensitization, neuroinflammatory processes, pelvic floor muscle dysfunction, as well as hormonal and psychosocial factors. Conceptualizing vulvodynia within the framework of chronic overlapping pain conditions facilitates a more comprehensive understanding of symptom persistence and variability in treatment response.

Accurate diagnosis requires a comprehensive clinical evaluation, including detailed history-taking, exclusion of alternative causes of vulvar pain, targeted pain assessment, and evaluation of pelvic floor muscle function. Failure to recognize coexisting pain mechanisms may result in misdiagnosis and suboptimal management.

Current evidence supports a multimodal, interdisciplinary approach as the cornerstone of vulvodynia management. Nonpharmacological interventions - particularly patient education, pelvic floor physical therapy, and psychological therapies - play a central role, while pharmacological treatment may provide benefit in selected patients. Limitations of the existing literature underscore the need for further research focused on standardized diagnostic frameworks and the development of targeted, mechanism-based therapeutic strategies.

Authors' contributions: All authors have read and approved the final version of the manuscript.

Conflict of interests: The authors declare no conflict of interest.

Funding: No funding was received to conduct this study.

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