Vulvodynia management

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Abstract

Vulval pain is a common problem encountered in many clinical settings involving women's health. Many women with vulval pain will have no apparent cause and a diagnosis of vulvodynia should be considered. Vulvodynia is defined as vulval discomfort, most often described as burning pain occurring in the absence of relevant visible findings, or a specific, clinically identifiable neurologic disorder. Before vulvodynia is diagnosed patients presenting with vulval pain need a careful history and clinical examination to avoid missing subtle relevant dermatological conditions of the vulva. Women with vulvodynia form a diverse group with different levels of symptoms, experiences and expectations of treatment. Clinicians not familiar with assessment and management with vulvodynia should refer onto secondary level care or specialist clinics. When making a diagnosis of vulvodynia clinicians should identify subtypes of vulvodynia and explore the key treatment needs of each patient. Based on current evidence, the prognosis for many women with vulvodynia is hopeful if an early diagnosis can be made and correct, individualized treatment given.

Keywords vestibulodynia; vulval pain; vulvodynia

Introduction

Vulval pain is a common clinical problem in both primary and secondary level care.

The prevalence of women with vulval pain in the UK remains unknown, however, recently community based studies in the US suggest that up to 8% of women fulfil the criteria for vulvodynia and that the lifetime prevalence of vulvodynia is 25%. As yet no incidence studies have been carried out but it is estimated that 2% of women per year develop vulvodynia. The prevalence might be underestimated as many women are often misdiagnosed as having recurrent candidiasis. As more vulval clinics have become established in the UK an increasing proportion of referrals to these clinics are women with vulvodynia. Vulval clinics focus on the complicated cases requiring multidisciplinary input by genitourinary medicine physicians, dermatologists and gynaecologists. Sadly this area of women's health is poorly developed in the UK, but offers an excellent opportunity for patient benefit, clinician satisfaction and research.

Vulvodynia – an evolution of classifications

Women with vulval pain with no apparent explanation have been documented in the literature for more than a century. One of the first accounts was by Skene in 1889 who noted that the external clinical appearances were often normal, but when the 'the examining finger comes into contact with the hyperaesthetic part (of the vulva), the patient complains of pain which is sometimes so great to cause her to cry out'. Kelly in 1928 also described 'exquisitely, sensitive, deep red spots in the mucosa of the hymenal ring as a fruitful cause of dyspareunia'. Since then scant reference has been made to vulval pain. In the 1970s many loose, ambiguous definitions were introduced which probably delayed progress in researching effective treatments and the literature was confusing. The terms, focal vulvitis, erythematous vulvitis en plaque, burning vulva syndrome and vestibular adenitis were introduced, and it was likely that they were all describing the same condition. In 1985 a specific task force was instituted by the International Society for the Study of Vulval Diseases to deal with the problem of recognition and therapy for these patients. The task force consisted of gynaecologists, dermatologists and venereologists all interested in managing women with vulval diseases. The term vulvodynia was introduced which has now been adopted for general clinical use in the UK. Vulvodynia and its subsets described women with *chronic vulval discomfort characterised by burning, stinging, rawness or irritation*. The terminology is potentially confusing as vulvodynia was originally described as having subsets including both infective and dermatological diagnoses. These included vulval dermatoses (e.g. lichen sclerosus), vulval vestibulitis, vestibular papillomatosis, dysaesthetic (formerly essential vulvodynia) and cyclical vulvitis. Friedrich in 1987 defined vulval vestibulitis and included three criteria for diagnosis: (1) severe pain on vestibular touch or attempted vaginal entry, (2) tenderness to pressure localized within the vestibule and (3) the physical findings of erythema confined to the vestibule. The swab test suggested by Friedrich was a useful way of demonstrating tenderness within the vestibule. This involved the clinician touching different areas of the vestibule area gently using a cotton tipped swab. Although the use of Friedrich's criteria has become outdated (because of doubt over the importance of erythema as a finding), the use of the swab test to demonstrate tenderness remains popular amongst clinicians.

More recently the classification of vulvodynia has changed to incorporate other subgroups outlined in Table 1 which takes into account of pain localization and whether it is provoked or not. The term vulval vestibulitis has been replaced by the term vestibulodynia as a form of localized provoked pain. Vulval vestibulitis was misleading as it implied an inflammatory process in the skin for which is there is no evidence. There is a preference amongst some UK clinicians to refer to patients as having 'vulval pain syndromes' which is not unreasonable as patients are analogous to other chronic pain patients and often have a syndrome of symptoms.

Vulvodynia – what is the cause?

A cause for symptoms remains elusive but is likely to be multifactorial. Difficulty in determining an exact cause relates to a long history of symptoms prior to a diagnosis and other factors which may have protracted symptoms such as topical treatments (e.g. inappropriate medications), psychological and psychosexual factors. A history of genital tract inflammation most often vulvovaginal candidiasis is the single most consistently reported

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ISSVD classification of vulval pain

Vulval pain related to a specific disorder

- Infectious (e.g. vulval candidiasis, herpes, etc)
- Inflammatory (e.g. lichen sclerosus, lichen planus, etc.)
- Neoplastic (e.g. Vulval intraepithelial neoplasia (VIN), squamous cell carcinoma, etc.)
- Neurologic (e.g. herpes neuralgia, etc.)

Vulvodynia

- Generalized
  - Provoked (sexual, nonsexual, or both)
  - Unprovoked
  - Mixed (provoked and unprovoked)
- Localized (vestibulodynia, clitorodynia, hemivulvodynia, etc.)
  - Provoked (sexual, nonsexual, or both)
  - Unprovoked
  - Mixed (provoked and unprovoked

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Table 1

feature reported by patients. Many patients recall an acute attack with the onset of symptoms and many complain of repeated attacks of candidiasis prior to an accurate diagnosis, however, many studies rely on self-reporting and confirmatory microbiology is rarely documented. Colonization rates of candida in patients with provoked pain are not increased compared to the general population.

Irritant dermatitis usually causes vulval inflammation and settles once the irritant is removed and the skin begins to heal. Although irritancy is unlikely to be responsible for initiating symptoms, it may possibly protract symptoms against a background of vulval pain. Multiple use of topical agents on the skin of women with vulvodynia is common and these are many potential irritants that can come into contact with the skin including prescription based treatments (e.g. antifungals), over-the-counter preparations, soaps, bubble-baths and scented hygiene sprays. Irritancy from topical medications is commoner on the vulva compared to skin elsewhere as the stratum corneum of the vulval skin functions less efficiently as a protective barrier. Many women complain of being allergic to many products and there is an increased background incidence of atopy within the group as a whole.

Psychological and psychosexual morbidity are common in women with vulvodynia, but it remains debatable as to whether these factors could be responsible for the onset of symptoms. Certain personality traits of women with vulvodynia suggest a proneness to stress and anxiety, which may ultimately, influence pain perception and symptoms. Some studies link a stressful period in the women's life such as marital disharmony and suggest that poor arousal could result in reduced lubrication during sexual intercourse leading to vulvo-vaginal irritation and a cycle of irritative vulval symptoms. Several studies have failed to show high rates of previous unpleasant sexual experiences or sexual and physical abuse compared to controls.

A hormonal basis for symptoms has been suggested but the data from the literature is conflicting. Taking the oral contraceptive pill has also been linked to an increased relative risk of developing vulval pain with an 11-fold increased relative risk if the pill was started before the age of 17 years.

Hypertonicity in the levator ani muscles when the vulval/vestibular area is touched is common and is often seen as a protective guarding response. This hypertonicity has been objectively measured with pelvic floor muscle electromyography and patients with provoked vestibulodynia demonstrate levator ani instability, poor muscle recovery after a contraction and elevated resting baseline tension when there was no attempt to provoke pain. Whether pelvic floor muscle tension is responsible for the perpetuation of symptoms remains to be answered, but treatment with biofeedback therapy to overcome levator hypertonia does have promising results.

Vulvodynia — a chronic pain issue?

Allodynia (pain on touch) and hyperalgesia (excessive sensitivity), both seen with vulvodynia, can possibly be explained by both central and peripheral nervous system adaptations. Repeated activation of skin nociceptors (C-fibres) leads to changes both within the peripheral and central nervous system. Normally, afferent fibres from nociceptors synapse within the grey horn of the spinal cord in an area reserved for pain stimuli (laminate II), before synapsing with higher cortical areas giving the perception of pain. In chronic pain, afferent fibres responsible for innocuous stimuli (A-beta fibres) sprout into the laminate of the grey horn where pain fibres normally end. Stimuli such as touch and pressure therefore elicit pain at a cortical level through these changes within the grey horn of the central nervous system, so-called *central sensitization*. Changes in the peripheral nervous system may also be involved. Cutaneous hyperalgesia is a protective response allowing damaged tissues to heal and is mediated though an increase in sensitivity of the skin nociceptors through neuropeptide release such as Substance P and calcitonin gene regulatory peptide. Their release leads to increased sensitivity of cutaneous nociceptors called *peripheral sensitization*, which has been demonstrated among women with vulval vestibulitis, where Substance P levels have been shown to be higher within the vestibule than controls. It has long been assumed that inflammatory pain can lead to neuropathic pain, but this has never been proven in vulvodynia. Recent animal studies, however, have confirmed that an allodynia response can be generated in the vulva secondary to repeated attacks of vulvovaginal candidiasis independent of tissue inflammation. After three attacks of treated vulvovaginal candidiasis, 40% of mice tested showed a sustained allodynia response. The vulval skin of mice examined after treatment for vulvovaginal candidiasis demonstrated neuronal proliferation in the absence of inflammation. In humans some studies have shown an increase in the intraepithelial nerve fibre density among women with provoked pain which fits into the inflammatory to neuropathic pain model. Central and peripheral sensitization may be responsible for the perpetuation of symptoms once the original tissue 'trauma' has resolved and also explain why tricyclic antidepressants, which influence pain perception centrally, help alleviate symptoms in women with unprovoked vulvodynia. The message is clear, early