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Vulvar Vestibulitis Syndrome: A Clinical Approach

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Vulvar vestibulitis syndrome (VVS) is a heterogeneous, multisystemic, and multifactorial disease and is one of the leading causes of dyspareunia in fertile women. As a multisystemic disease, it involves the mucous structure of the vulvar vestibule and the immune, muscular, vascular, and nervous systems, including pain fibers and centers. As a multifactorial disease, its etiology is complex, involving biological, psychosexual, and relational factors. In this article, we discuss the progression of the disease and the impact of an often lengthy delay between the onset of symptoms and a correct diagnosis. Moreover, despite documented improvements from available treatments, VVS becomes a chronic disease unless it is diagnosed early and an integrated, pathophysiologically oriented treatment is offered in an experienced center. Health care providers would therefore benefit from approaching the condition within a pain management framework focused on the woman's chronic pain, the impact on the couple's relationship, and any associated psychological sequelae.

Vulvar vestibulitis syndrome (VVS) is a heterogeneous, multisystemic and multifactorial disease and is one of the leading causes of dyspareunia in women of fertile age (Baggish & Miklos, 1995; Bergeron, Binik, Khalife, & Pagidas, 1997; Friedrich, 1987; Graziottin, 2001). VVS has been described as a clinical disease characterized by three symptoms and signs: (a) severe pain on vestibular touch or attempted vaginal entry; (b) tenderness when pressure is localized within the vestibule; and (c) physical findings confined to vestibular erythema of various degrees (Friedrich, 1987). Revised criteria

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have been proposed (Bergeron et al., 2001), but the diagnosis is still based on Friedrich's criteria. VVS is one aspect of "vulvodynia," a clinical condition well described by the International Society for the Study of Vulvovaginal Diseases. Understanding the pathophysiology involved in the diagnosis of VVS so that optimal treatment recommendations may follow currently is a challenge for clinicians and researchers in the field of female sexual medicine and is the topic of a forthcoming article from these authors. The focus of this article is on the optimal management of chronic pain in women with VVS, on psychological sequelae that women may experience, and on relationship factors arising from the condition. These aspects are critical to address if there is to be a shift from pain to pleasure; in other words, the ideal endpoint in our intervention is the reacquisition of a satisfying sexual experience for the woman and her partner. A brief summary of key pathophysiological concepts may help to design an optimal medical and psychosexual intervention in a stepped-care multidimensional model.

THE NATURAL HISTORY OF VVS

The concept of *natural history* is basic in understanding the spontaneous evolution of a disease and to designing the stepped-care rationale of a medical and psychosexual intervention. Natural history highlights a number of key concepts that apply to VVS as well, as illustrated in Figure 1. First, the number and type of factors that affect the clinical picture change and increase over time, particularly in chronic diseases that involve multiple biological systems, for example, nervous (including the pain system, a key protagonist in VVS), vascular, immune, and muscular. Second, the impact the disease has on the affected person increases over time, with complex adaptation of and changes in her psychological well-being and coping style, as well as in couple and family dynamics. This is true also for diseases with an initial strong biological base, like VVS, particularly when burning pain becomes a hallmark feature of the disease. Third, predisposing, precipitating, and maintaining factors in VVS should be recognized and addressed. Metaphorically speaking, the natural history of a disease is like a movie, with the diagnosis being just one segment of the story. The protagonists of that scene may be very different from those present at the beginning of the story. Moreover, the possibility of changing the ending of the story is greater at the beginning and decreases progressively over time, particularly if one hopes for a happy ending. This holds strongly true in VVS, where the shift from nociceptive pain (a condition in which ongoing acute tissue damage leads the organism to withhold and defend itself) to neuropathic pain (a condition wherein pain is generated within the pain fibers and centers themselves) is not only the hallmark of the disease but also its most challenging feature (Figure 2; Graziottin & Vincenti, 2002). The proliferation of introital nociceptive fibers is well

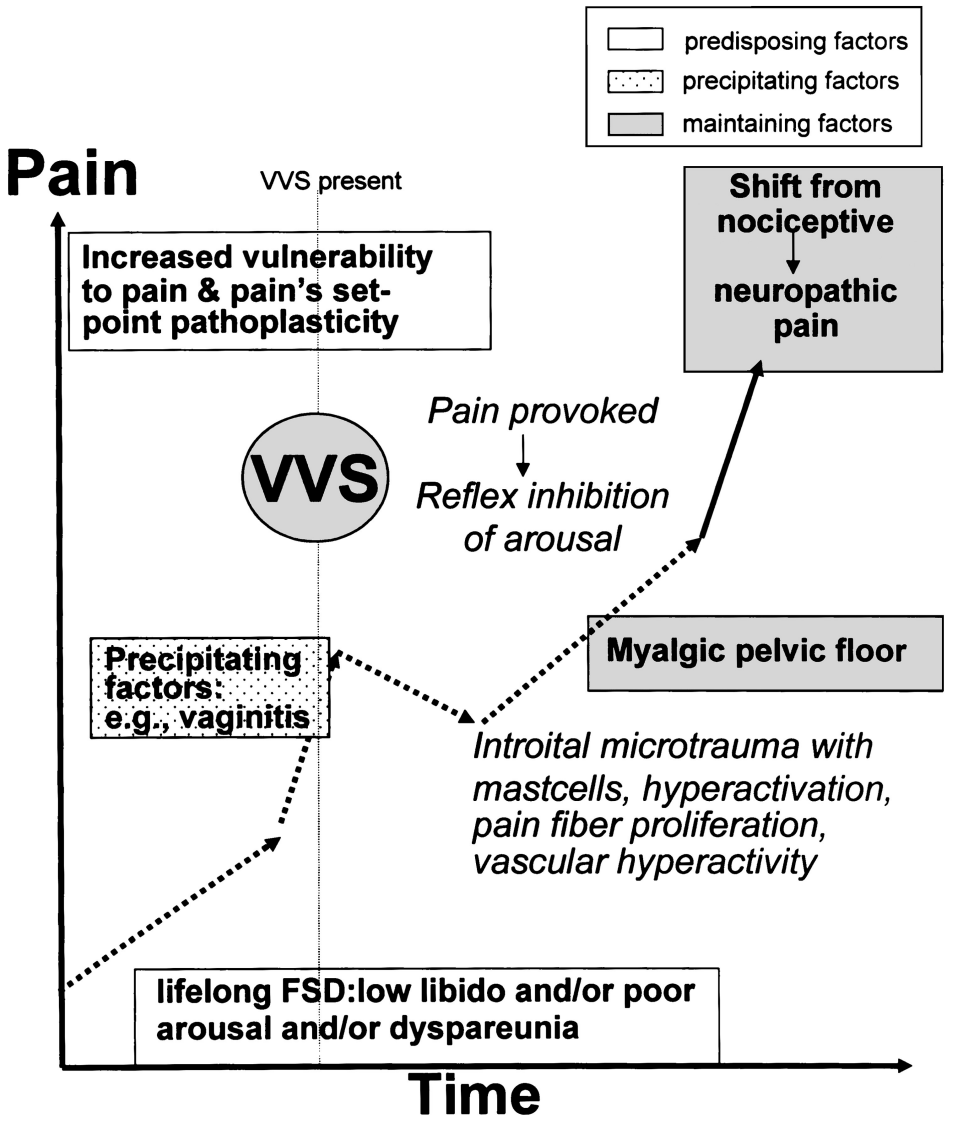

FIGURE 1. Natural history of VVS over time, showing the presence of different concomitant etiologies: (a) *predisposing factors* (e.g., lifelong female sexual dysfunction (FSD), including vaginismus as well as pain vulnerability and personality characteristics); (b) *precipitating factors* (e.g., recurrent vaginitis, candida); and (c) *maintaining factors* (e.g., lifelong or acquired hypertonic pelvic floor; lifelong or acquired low libido, poor arousal, and dyspareunia; acquired shift from nociceptive to neuropathic pain).

documented in VVS (Bohm-Starke, Hilliges, Blomgren, Falconer, & Rylander, 2001; Bohm-Starke, Hilliges, Brodda-Jansen, Rylander, & Torebjork, 2001; Bohm-Starke, Hilliges, Falconer, & Rylander, 1999). The concept of *neuropathic pain* is gaining increasing attention in the scientific literature (Baron, Levine, & Fields, 1999; Baron, Schattschneider, Binder, Siebrecht, & Wasner,

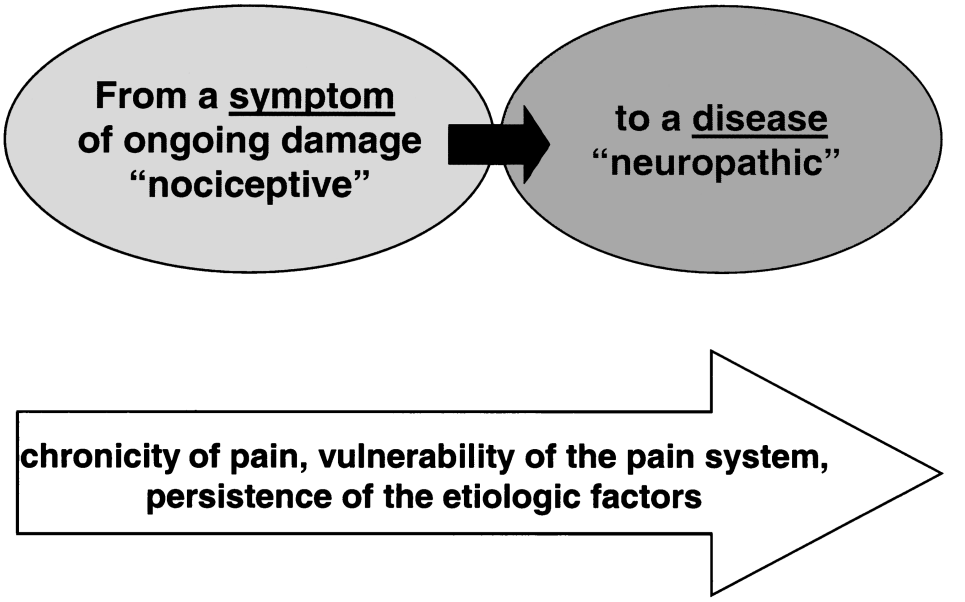

FIGURE 2. Descriptive model outlining the progressive shift from nociceptive to neuropathic pain when the persistence of etiological factors in vulnerability of the pain system leads to chronic pain.

2002; Bonica, 1990). The transition from nociceptive to neuropathic pain is key in VVS because it underlies the shift from a sexual pain disorder—where intercourse elicits and provokes pain—to a progressively pure pain disorder, which is self-maintained in spite of the avoidance of any further coital intimacy. Some women with chronic VVS report distress over noncoital activity, such as kissing or hugging their partner or even upon having an erotic dream or watching an erotic movie, when even mild genital arousal (without any direct genital contact) immediately elicits a worsening of the VVS pain. Finally, the time delay from disease onset to the moment of a clinical diagnosis is critical (as shown in Figure 1), because this may clarify which factors are implicated, which are still reversible, how the health care professional chooses between conservative versus invasive and surgical treatments, and how the prognosis may change.

As a multifactorial and multisystemic disease with a potentially chronic course, VVS requires attention to the biological, psychosexual, and relational cofactors in a clinically oriented way. Complex problems require complex solutions and, in terms of the tendency to search for "the" treatment, VVS is one condition in which this key conceptual mistake is common practice. There is no simple treatment that may address the complexity of the etiological factors in VVS and of their variable impact on the women's symptoms and on the couple's dynamics over time. Moreover, all proposed treatments

are designed to reduce and one hopes, eliminate pain. However, in a sexual pain disorder, this would be an incomplete outcome. A true therapeutic success takes place when the woman regains a satisfying sexual life.

THE CLINICAL DIAGNOSIS OF VVS

A tentative diagnosis of VVS should be considered when a woman reports superficial dyspareunia with introital contact, and a clinical examination reveals the three hallmark symptoms described by Friedrich. From the perspective of the practicing physician who most frequently encounters women presenting with such complaints, a focused clinical assessment helps to ensure an accurate diagnosis of VVS and helps to focus optimal management. We summarize the components of a thorough clinical assessment in nine categories that include both objective testing with a physical examination by the physician, as well as subjective assessment of the woman's self-reported experience.

First, the physician must assess an accurate psychosexual history, focusing on the occurrence of any sexual dysfunction present before the onset of VVS. The woman's current sexual practices and the coexistence of any other sexual difficulties delineating life-long versus acquired desire, arousal, orgasmic, and satisfaction difficulties besides dyspareunia must be evaluated. If there are signs of a general defensive posture (defined as a dramatic increase in muscle tension, with avoidance of any genital contact, including avoidance of the examiner's hand) and a history of lifelong dyspareunia, this may point to a primary condition of vaginismus, cooccuring with the VVS (Van der Velde, Laan, & Everaerd, 2001).

Second, a detailed medical history must be undertaken that includes assessment of food and medication intake. In particular, any food intolerance, allergy, current hormonal treatment (e.g., oral contraceptive pill, hormone replacement therapy), and all previous systemic and genital treatments must be assessed and recorded.

Third, the physician should inquire as to previous vaginal infections or sexually transmitted diseases, such as candida albicans, gardnerella vaginalis, human papilloma virus (HPV), and herpes. The physician must test a possible current vaginal bacterial infection with a vaginal swab and subsequent cultures, especially if an ongoing infection is suspected.

Moving to the more qualitative aspects of the assessment, the fourth step involves determining the woman's perception of the characteristics and duration of her symptoms. This may involve taking a developmental perspective (i.e., the natural history approach) to trace the onset of VVS symptoms.

Fifth, a very careful physical examination should be recorded. Vaginal pH should be tested objectively with a vaginal stick for 10–15 s during the gynecological examination (Caillouette, Sharp, Zimmerman, & Roy, 1997).

Step six involves asking three basic questions about the quality of the pain: "Where does it hurt?," "When does it hurt?," and "What are the associated symptoms that you experience?." These three questions have been suggested to reliably indicate the relevant biological etiology of dyspareunia in women (Meana, Binik, Khalife, & Cohen, 1997) and will guide clinical recording in the next step.

The seventh step is the key feature of the examination: the making of an accurate "pain map"—the precise recording of any point in the external genitalia and mid- and deep-vagina where pain can be objectively elicited, in parallel with questioning (Graziottin, 2001; Graziottin, Nicolosi, & Caliari, 2001c). In 90% of cases an accurate gynecological examination may reproduce exactly the pain site and characteristics that the woman experiences (Bergeron et al., 2001). The physician does this through a careful gynecological examination, paying attention to any increase in pain perception in the vulvar and vestibular areas, at the mid-vagina, and at deep insertion. The precise location of pain, its onset, and its characteristics have proven to be the strongest predictors of its organicity (Meana et al., 1997). During the gynecological examination, the physician should also pay attention to the presence of other complicating factors, such as defensive levator ani contraction, that over time may become a cofactor of pain, leading to levator ani myalgia. This reflex contraction may be present from one's first sexual experience (i.e., lifelong), where vaginismus is present, or it may be acquired in response to pain, as an automatic defensive measure to prevent further penetration and consequent pain and tissue damage. The progressively intense myalgia that may develop further increases the generation and perception of pain and consequently prevents penetration. The presence of such factors would necessitate a more comprehensive, rehabilitative treatment aimed at relaxing the tightened pelvic floor. Next, using a Likert scale (from 0 = no pain, to 10 = worst pain ever), the physician should record bilateral quantification of the perception of the pain intensity. This is done bilaterally because higher pain perception is usually reported on the left side, possibly for postural reasons or because of sitting with crossed legs, which causes different tension in the two halves of the levator ani. Other medical conditions that could obiectively be diagnosed and that could be a cofactor of pain should also be recorded (e.g., painful episiorraphy, dystrophias, associated clitoralgia).

Finally, because sexual activity typically involves a partner, the status and quality of the relationship, focusing on the couple's affective, sexual, and interpersonal functioning, should be assessed. Given that aspects of a partner's functioning have been shown to affect sexual functioning of the woman (see, for example, Dennerstein, Lehert, Burger, Garamszegi, & Dudley, 2000; Kaplan, 1974), the presence of sexual or genital symptoms in the partner should be recorded as well.

PROGNOSIS

Few data are published on the prognosis of VVS in women following treatment. Even less is known about putative characteristics of the woman at the time of assessment that might suggest a particular prognosis. We would suggest an algorithm based on a tentative clinical categorization to guide prognosis.

Type one could be described as "Low-risk VVS patients with good prognosis." Aspects of their detailed clinical assessment that might suggest such a prognosis would include: (a) VVS duration of less than 1 year; (b) a normal systemic pain threshold; (c) no history of invasive treatments; (d) no difficulties with sexuality before the VVS symptoms; (e) demonstrable self-efficacy and coping strategies; and (f) a positive relationship with a supportive partner.

Type two could be described as "High-risk VVS patients with questionable prognosis." Aspects of their clinical assessment that might signal such a prognosis would include: (a) VVS duration of more than 1 year; (b) chronic candida or candida treatment resistance; (c) a history of vulvar or vestibular HPV laser treatment; (d) hyperalgesia (local and systemic); (e) sexual difficulties present before the onset of VVS; (f) depressive symptoms; (g) anxiety symptoms, which might include fear of sexual intimacy or avoidant behavior; and (h) being single or in a troubled relationship.

A third type of prognostic categorization could be described as "High-risk VVS patients deserving systemic and local treatment, in addition to psychosexual support." This classification would be considered when there is: (a) pain persisting after electroanalgesia, electromyographic biofeedback, or vestibulectomy (Graziottin & Vincenti, 2002); (b) burning pain that is independent from intercourse; and (c) high personal or couple distress.

TREATMENT GUIDELINES

Given the complex etiological and pathophysiological processes involved in VVS, treatment requires a therapeutic approach that combines attention to the biological as well as psychosexual domains (Figure 3).

Medical Approach

Physicians must address biological precipitants and treat any associated genital disease or infection, muscle tension of the pelvic floor, as well as pain. If there is a precipitating infectious disease, such as candida or gardnerella (Faro, 1996; Horowitz, 1991; Paavonen, 1995) or, less frequently, HPV or herpes, we suggest the following treatments. When present, chronic candida

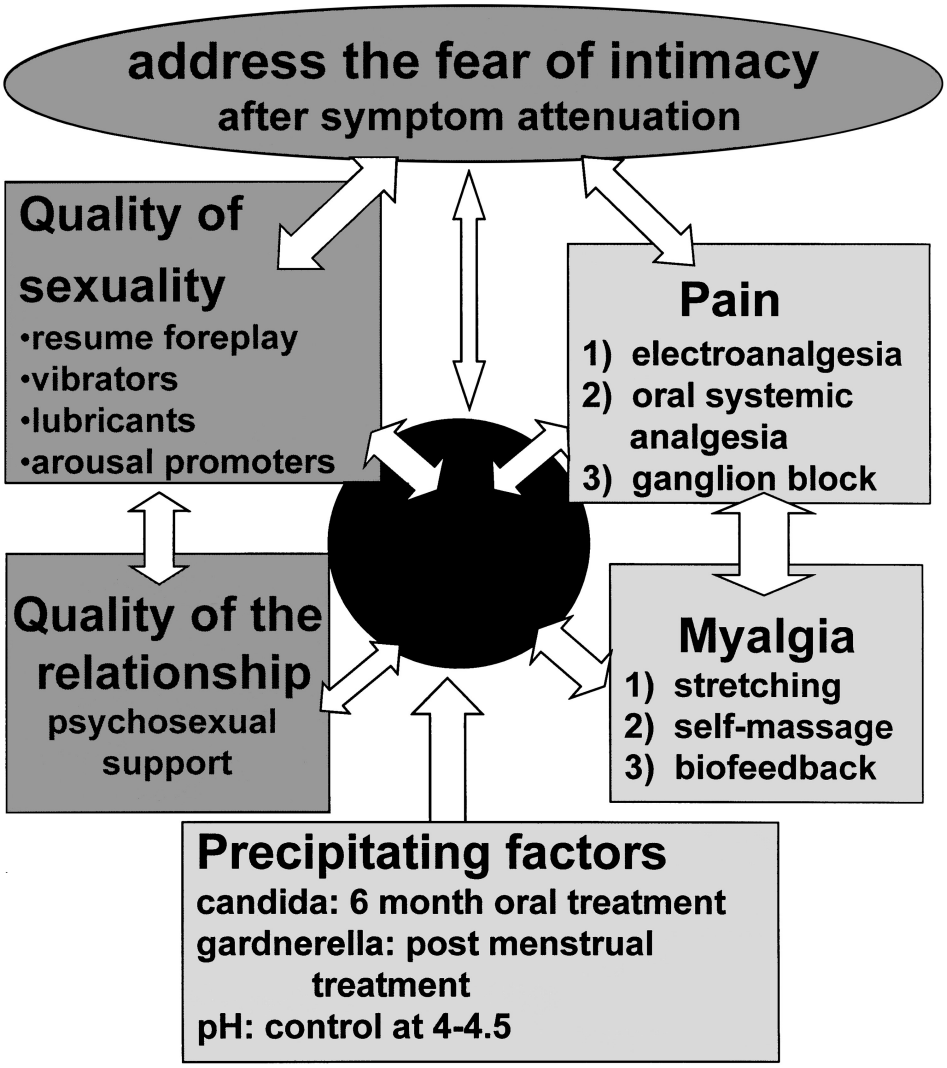

FIGURE 3. The therapeutic approach to VVS combines medical (light gray boxes) and psychosexual (dark gray boxes) approaches.

is to be treated with itroconazole, 200 mg orally a day for 3 days (or fluconazole 100 mg orally a day for 2 days) every 2 weeks for 3 months, then once a month for another 3 months. The partner should also be treated for the first month. This treatment is aimed at reducing the recurrent episodes of candida that might perpetuate the vaginitis and the mast-cell hyperactivation. In a review of the cases seen in our center in Milan, Italy, a history of chronic vaginal candida infections were reported in 58.1%, and in 29.0%, this incidence was confirmed upon presentation to our center (Graziottin, Nicolosi, & Caliari, 2001a). According to some authors (Mariani, 2002), patients with a

positive culture for candida and symptoms flaring with menses are thought to be better categorized as "cyclic vulvitis" (McKay, 1992) or "atypical" candidiasis and are thus removed from the classification. However, when recurrent candida is associated with the specific triad of VVS features, the condition should be addressed in parallel with VVS treatment.

Recurrent gardnerella or haemophilus infection (Paavonen, 1995), reported in 33.9% of our patients (Graziottin et al., 2001a), is usually associated with a vaginal pH of 5. The physician may obtain a lower pH with vaginal tablets of boric acid (300 mg) once a day for 10 days every month (following menstruation). Topical estrogen may be prescribed if the patient has a persisting amenorrhea with vaginal dryness as an associated symptom. Or the physician may prescribe long-acting polymeric acid gel, delivering H+ (and thus lowering the pH) twice a week. However, topical treatments should be avoided in the acute phase of the disease because a vaginal/vestibular hyperreactivity to almost all topical compounds is usually reported.

The physician should address the reactive myalgic tension of the pelvic floor by teaching the patient (a) how to voluntarily relax the pelvic floor muscles; (b) how to self-perform an accurate stretching of the pelvic floor muscles (Graziottin, 2001), in addition to self-massage, for 5 min twice a day with a medicated oil (e.g., Saint John's Wart); and (c) how to perform circular massage at the tender points on the painful muscle (mid vagina, at the insertion of the levator ani on the ischiatic spine). These simple and effective techniques are especially beneficial for patients who are unable to travel long distances for weekly physiotherapy or electromyographic biofeedback treatment (Graziottin, 2001). Self-massage and stretching are "homemade," cost-free, and empowering for the woman to feel progressively more aware of her defensive contraction and in control of at least one component of her pain. We encourage all of our patients to do their massage on a regular basis. If the partner is present and the woman is accepting, the partner can be taught to recognize the different levels of contraction and relaxation and, in the final phase of treatment, to incorporate gentle massaging of the pelvic floor during foreplay.

We also recommend physiotherapy treatment, consisting of two sessions of general relaxation and postural changes and eight sessions of levator ani surface electromyographic biofeedback, with self-insertion of a small single user s-EMG sensor into the vagina (Glazer, Rodke, Swencionis, Hertz, & Young, 1995; McKay, Kaufman, Doctor, Berkova, & Glazer, 2001).

Treatment of the pain itself is dependent on the severity and on the degree of impairment in daily life. Pain may best be addressed with surface electroanalgesia to modulate residual vestibular pain, when mucosal integrity is maintained (Nappi et al., 2003). We recommend mood modulation with a low dose selective serotonin reuptake inhibitor when unremitting depressive symptoms are complicating the condition. Systemic oral analgesia is gaining increasing attention, because research indicates a significant lowering of the

systemic pain threshold in these women (Pukall, Binik, Khalife, Amsel, & Abbott, 2002). Systemic treatment may include: (a) tricyclic antidepressants aimed at modulating the serotonin and epinephrine imbalance associated with persisting pain (Mariani, 2002; McKay, 1993) or (b) anticonvulsants, such as gabapentin, aimed at raising the threshold for the amount of stimuli needed for nerves to fire, thus raising the central pain threshold (Graziottin & Vincenti, 2002).

Presacral anesthetic block of the ganglion impar has recently been proposed as an effective conservative second-line treatment when all previous treatments have failed (Graziottin & Vincenti, 2002). To the best of our knowledge, this technique, proposed by Plancarte and colleagues for the treatment of neuropathic pain in patients with recurrent pelvic cancer (Plancarte, Amescua, & Patt, 1990) has not been reported so far in the clinical literature on VVS. This technique is used in our Milan, Italy, center when genital pain of VVS is resistant to all the above-mentioned treatments, and when the characteristics of pain become unbearable and unremitting day and night, thus preventing activities of daily living and having significant effects on mood and well-being. It requires an experienced and skilled anaesthetist with specialized training.

Vestibulectomy has been shown to be an effective treatment for VVS (Bergeron et al., 2001; Schneider, Yaron, Bukovsky, Soffer, & Halperin, 2001; Weijmar et al., 1996) after more conservative pain treatments have failed. The rationale is to remove the mucosal tissue with nerve proliferation and hypersensitity in order to restore a more normal perception. The inadequacy of our knowledge of pathophysiology of VVS suggests limiting the surgical option to later in treatment, particularly when outcome measures are somewhat questionable (Marin, 2001). Moreover, we examine a number of postvestibulectomy patients with persisting and even worsening pain, suggesting that clinicians should pursue more conservative and systemic treatments to keep pain under control before considering surgery.

Psychosexual Approach

The psychosexual domains require a detailed clinical assessment by a physician with some knowledge of psychological processes and how these might interface with medical issues. Following a significant reduction in the clinical signs and symptoms of VVS, the second part of treatment is to be initiated—defined as the reexperience of sexual satisfaction, and one hopes, of coital pleasure. However, the psychosexual intervention should be timed according to the diagnosis; for example, if a woman reports a concomitant story of trauma or an abusive partner that is impacting her coping ability, this deserves attention in parallel with medical treatment. The role of psychosexual factors in contributing to the vulnerability of VVS is increasingly acknowledged (Brotto, Basson, & Gehring, 2003; Graziottin, Nicolosi, & Caliari

2001b; Jantos & White, 1997; Meana et al., 1997; Sackett, Gates, Heckman-Stone, Kobus, & Galask, 2001; van Lankveld, Weijenborg, & ter Kuille, 1996). In our VVS series, 22.7% of patients reported lifelong low libido, 58.1% reported acquired low libido, and the remaining reported nonproblematic sex drive; 17.7% acknowledged lifelong arousal disorder, 50% reported acquired arousal disorder and the remaining reporting unproblematic mental and genital arousal, at least during foreplay. Twenty-nine percent of patients reported lifelong dyspareunia (well addressing the issue of a likely primary vaginismus as well as comcomitant low libido and/or arousal disorders), 61.3% reported acquired dyspareunia, and the remaining 9.7% reporting a recurrent dyspareunia from the beginning of their sexual life, with periods of remission prior to the current persisting problem. Nearly eighteen percent of patients reported lifelong orgasmic disorders, 40.3% reported acquired orgasmic disorders (most because of coital pain), and 41.9% reported no changes in their orgasmic potential during foreplay (Graziottin, Nicolosi, & Caliari, 2001b). Non-penetrative sexual abuse was reported by 29.2% of our VVS patients and penetrative abuse by 6.5% (Graziottin, Nicolosi, & Caliari, 2001b). Poor arousal may indeed result from introital mechanical microabrasion due to the vaginal dryness and to the tightened pelvic floor that squeezes and narrows the introitus.

In parallel with an individual approach, couple treatment should be offered to VVS patients, with diagnostic and therapeutic components being carried out by an experienced psychiatrist or psychotherapist with sexological training. Key points of the psychosexual intervention are: (a) psychodynamic/interpersonal treatment when lifelong issues are evident (e.g., previous harassment or abuse, poor couple differentiation [Schnarch, 2000], poor coping attitude with a catastrophizing approach to life difficulties and VVS), or concomitant depression is present; (b) brief behavioral therapy when a lifelong vaginismus is diagnosed, or when erotic aversion, sexual inhibition, or poor sexual skills are features of the woman or the couple; (c) clitoral vibrators, a clitoral therapy device, or vasoactive drugs to improve genital arousal when pain has relieved and the focus is on resuming sexual intimacy. The psychosexual intervention should be integrated with tailored pharmacologic management (if necessary) of depression or anxiety when they are diagnosed as significant cofactors in low libido, poor arousal, and of a general tendency to sexually avoidant behavior, because of the persisting fear of pain. The aim of the psychosexual and pharmacologic intervention is for the woman to regain a serene and satisfying sexual intimacy.

On the basis of these recommendations, we can identify two subgroups of patients, with a full spectrum of intermediate characteristics in between. At one end might be women (or couples) who have had a disappointing sexual life from the beginning of their sexual experience. In these patients, curing VVS is a preliminary part of a more-complex treatment aimed at significantly improving the whole sexual experience. We recommend

psychotherapeutic and sexual support, with a treatment aimed at reexploring the individual's pleasure capacity, addressing individual and couple foreplay first, with intercourse being reintroduced only in the final sessions. Clitoral vibrators, and lubricants may all prove useful in increasing genital arousal and experiencing in women, although the empirical data here are scant. Another type of presentation might be the woman with a full pleasurable orgasmic sexual experience who had an acquired dyspareunia of variable intensity before VVS was diagnosed. For these patients the normalization of the vaginal/vestibular area usually may lead to a rapid return to a normal sex life unless the symptom duration has been so long as to impair the relationship and any remaining sexual intimacy.

With respect to psychosexual support of the couple, the very first therapeutic step is to explain to the partner what VVS is from the medical point of view, its strong biological nature and pain that is generated in the affected tissue and not "in her mind." An accurate and simple explanation of the medical basis of VVS may have a profound impact on the partner's attitude, which may shift from variably unsupportive (after months or years of being told that this was a psychogenic pain, that everything was fine, and that she was just refusing to have intercourse), to understanding, and supportive. One may show the partner (with the woman's permission) the pain map and the objective sites of pain in a nonsexual state, and how she relaxes the tightened pelvic floor, all of which may have profound effects on the couple dynamics. Explaining to the couple the rationale of a complex and gradually effective therapeutic approach helps them give meaning to their chronic suffering, provides direction for their coping efforts, and encourages new hope. A specific psychosexual intervention for the partner may be necessary if he/she suffers from a sexual dysfunction that was present before VVS or appeared during the course of it. Loss of libido or avoidance of intercourse in the male partner for fear of causing further pain to the female partner are common, but rarely addressed aspects of VVS. The longer the chronicity of VVS, the higher the likelihood of a mutual disinterest in coital intimacy. After the physician has cured pain, the pursuit of pleasure requires a committed therapeutic approach. An often unconsidered etiological factor in the maintenance of pain is resuming coital activity despite a lack of sexual arousal. This is likely the most reliable way of causing further microtrauma to the vulnerable vulvar vestibule, restarting the vicious cycle that maintains chronic coital pain.

CONCLUSION

VVS is a heterogeneous, multisystemic and multifactorial disease. An accurate clinical diagnosis of different biological, psychosexual and relational factors that may act as either sexual vulnerabilities, precipitating, or reactive factors, should be carried out to establish the treatment that may best address the

etiologic complexity of VVS for an individual woman. The physician should recommend different treatment strategies according to the etiologic factors that come into play, the individual risk profile, and any context-dependent factors (e.g., distance from the referral center) that may make full treatment adherence difficult. A better understanding of the pathophysiology of the disease is necessary if we are to shift from a pragmatic, symptom-oriented approach to a more complete, pathophysiologically oriented one. On a final note, pain is almost never completely psychogenic, and VVS is no exception. Patients should no longer be told "the pain is all in your head." Providing understanding and respect to the emotional needs of women who experience this painful sexual condition is a crucial first step in an effective therapeutic alliance.

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