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Sexual pain disorders: Clinical approach

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ABSTRACT. Sexual pain disorders include dyspareunia and vaginismus. They may affect 10 to 15% of women between 18 and 59 years of age. Vulvar vestibulitis is the leading cause of dyspareunia in the fertile age. Etiology includes recurrent vaginal infections; hyperactivity of the mastcells; myalgic contraction of the pelvic floor; hyperactivity of the pain system, with shift from nociceptive to neuropathic pain, and neurogenically mediated inflammation. Iatrogenic factors may cause acquired dyspareunia. Loss of estrogens and related genital arousal disorders are the leading etiology of dyspareunia in the postmenopause. Special attention wll be dedicated to the strong association between urinary tract symptoms and dyspareunia – RR= 7.61 (4.06-14.26) – and genital arousal disorders – RR= 4.02 (2.75-5.89) – respectively. Psychosexual factors may contribute to sexual pain disorders. Inadequate coping modalities may worsen pain perception over time. A careful clinical approach, aiming at understanding and treating all the causes of sexual pain disorders, is mandatory if sexual pain is to be effectively addressed.
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INTRODUCTION

Dyspareunia is a comprehensive word, used when intercourse is characterized by pain, of different etiology (1-4). Vaginismus focuses on the muscular component of the disorder, namely the defensive contraction of the pelvic floor, that is usually psychogenically triggered by fear of penetration of whatever conscious or unconscious etiology (5). Vaginismus is still considered the clinical expression of fear of penetration, the muscular involvement being a more variable and inconsistent finding (see Graziottin et al., this issue). When severe, vaginismus interferes with penetration. It is the leading female cause of unconsummat-

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ed marriage. Lifelong vaginismus, when not severe enough to prevent penetration, may cause introital dyspareunia. Considering the main interest of the readers, this paper will focus on dyspareunia and associated urinary tract symptoms. Evidence of the increasing role of the pain system in the maintenance of the local inflammatory response (6), in the hyperalgic genital perception and in the lowering of the central pain threshold has dramatically changed the clinical approach to dyspareunia in the recent years. Implications for management will be reviewed.

DEFINITION AND PREVALENCE

"Dyspareunia defines the persistent or recurrent pain with attempted or complete vaginal entry and/or penile vaginal intercourse. Vaginismus indicates the persistent or recurrent difficulties of the woman to allow vaginal entry of a penis, a finger, and/or any object, despite the woman's expressed wish to do so. There is often (phobic) avoidance and anticipation/fear of pain. Structural or other physical abnormalities must be ruled out/addressed" (7). The "non coital sexual pain disorders", described as the "recurrent or persistent genital pain induced by non coital sexual stimulation", i.e. foreplay evoking or worsening clitoralgia or vestibular pain, formerly included in the classification of FSD, has been deleted from the latest version (8)

Ten to 15% of coitally active women in the fertile age (9), and 22.5-33% of postmenopausal women complain of various degrees of dyspareunia. Vaginismus is reported in average 0,5-1% of fertile women.

THE PATHOPHYSIOLOGICAL SCENARIO

Vaginal receptiveness is a prerequisite for intercourse. This ability requires anatomofunctional integrity of the many tissue components, both in resting and aroused state. Normal trophism, mucosal and cutaneous, adequate hormonal impregnation, lack of inflammation, particularly at the introitus, normal tonicity of the perivaginal muscles, vascular, connective and neurological integrity, and normal immunitary response are credited to be the necessary conditions to guarantee the vaginal "habitability" (2, 3). Vaginal receptiveness may be further modulated by psychosexual, mental and interpersonal factors: the woman's motivation to intercourse is key. Etiology of dyspareunia may be biological, psychosexual and context-dependent (1-8, 10-12) (Table 1). Usually multiple causes are present in the individual patient. Poor arousal, with vaginal dryness, may be a predisposing factor of biological etiology, or the consequent sign, of poor psychosexual arousal (13, 14). Fear of penetration, with a variable defensive pelvic floor contraction and general muscular arousal secondary to anxiety, may lead to vaginismus. It may be so severe as to prevent penetration at all. The defensive pelvic floor contraction may also be secondary to genital pain, of whatever cause. It may be triggered by pain in the introital area associated with: vulvar vestibulitis (1-3, 6, 14); recurrent vaginal infections (12); vulvovaginal atrophy, secondary to loss of estrogens and/or androgens (10); iatrogenic factors, such as poor outcome of episiorrhaphy, vulvar lasertherapy; overzealous colporrhaphy; posterior colporrhaphy, radical surgery for cervical carcinoma, pelvic radiotherapy (2, 3). Vulvar vestibulitis has three major symptoms: acute burning pain, most at 5 and 7, when loooking at the vaginal introitus like a clockface; reddening of the vestibular mucosa and dyspareunia (1). An almost constant finding is the associated contraction of the pelvic floor muscles (2, 3). Non-genital, non-sexual causes, such as anorectal problems (anismus, hemorroids, ragads) or urological factors (in association with urinary tract symptoms, cystitis first, urge incontinence

Table 1 - Etiology of dyspareunia. Many causes may overlap or be associated with coital pain with complex pathophysiologic interplay. The relative weight of each cause in the individual woman may change with chronicity of pain and progressive involvement of other pelvic organs.

A) Biological

1) superficial/introital and/or mid-vaginal dyspareunia
- infectious: vulvitis, vulvar vestibulitis, vaginitis, cystitis
- inflammatory, with mastcell's up-regulation
- hormonal: vulvo-vaginal atrophy
- anatomical: fibrous hymen, vaginal agenesis
- muscular: primary or secondary contraction of levator ani muscle
- iatrogenic: poor outcome of genital surgery; pelvic radiotherapy
- neurologic, inclusive of neuropathic pain
- connective and immunitary: Sjogren's syndrome
- vascular

2) deep dyspareunia
- endometriosis
- pelvic inflammatory disease (PID)
- pelvic varicocele
- chronic pelvic pain and referred pain
- outcome of pelvic or endovaginal radiotherapy
- abdominal nerve entrapment syndrome

B) Psychosexual
- co-morbidity with desire and /or arousal disorders, or vaginismus
- past sexual harassment and/or abuse
- affective disorders: depression and anxiety
- catastrophism as leading psychologic coping modality

C) Context or couple related
- lack of emotional intimacy
- inadequate foreplay
- couple's conflicts; verbally, physically or sexually abusive partner
- sexual dissatisfaction and consequent inadequate arousal
- poor anatomic compatibility (penis's size and/or infantile female genitalia)

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second) are often associated to the levator ani contraction (13). Latent classes of sexual dysfunctions by risk factors in women indicate that urinary tract symptoms have a RR= 4.02 (2.75-5.89) of being associated with arousal disorders and a RR=7.61 (4.06-14.26) of being associated with sexual pain disorders, according to the epidemiological survey of Laumann et al. (9). Endocrine, infectious, muscular, vascular, nervous (particularly pain associated) and psychosexual factors may variably contribute to the individual sexual pain disorder. In chronic dyspareunia, particularly when associated with vulvar vestibulitis or interstitial cystitis (IC), pain becomes prominent in 90.3% of patients with vulvar vestibulitis and 93.6% of classic IC patients report various degrees of pain. Of those with pain, 80.4% have pain in the lower abdomen, 73.8% in the urethra, 65.7% in the lower back and 51.5% in the vestibular/vaginal area. (13). Intermittent flares may occur premenstrually, both in IC and VV patients. Hormonal modulation of the mastcell's hyperactivity associated with chronic pain is the most plausible factor. Hormones may trigger the release of histamine, substance P, bradichynine, and other pro-inflammatory substances from the vescicules packed in the mastcells. Whatever the agonist stimulus – infectious, inflammatory, neurogenic, mechanic, hormonal or chemical – that triggers the mastcell's release and up-regulation, the common outcome is the maintenance and worsening of local inflammation and tissue damage, with parallel up-regulation of the pain system, mediated by the mastcell's production of Nerve Growth Factor (NGF). Current understanding of the pathoplasticity of the pain systems indicate that the shift from nociceptive pain (indicator of threatened or ongoing tissue damage) to neuropathic pain (generated in up-regulated fibers and centers of the pain system) is the common denominator of most of pelvic pain syndromes, inclusive of dyspareunia (2, 3, 6, 11, 13). Three major steps have been described: 1) neurogenic inflammation, as a result of prolonged noxious stimuli, with afferent nerves firing antidromically (backward down the sensory nerve); 2) "cross-talk" of pelvic viscera with shared innervation, together with the increasing recruitment of usually "silent" unmyelinated fibers (A-delta and

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C-fibers), the most important carriers of pain signals. This recruitment may explain the increasing hyperalgesia whilst the cross-talk seems to be a further contributor of the progressive involvement of different pelvic organs in the increasing pain perception; 3) development of viscero-muscular reflex, which results in muscular instability and a hypertonic contracted state within the muscles of the pelvic floor (13). This results in a decrease in muscle function and in the development of myofascial trigger points and myofascial pain with the development of new pain generators (13).

Medical ("organic") factors – too often underevaluated in the clinical setting – are therefore the most frequent and important causes of dyspareunia and chronic pelvic pain. They may interact with psychosexual factors. A thorough medical evaluation is therefore mandatory.

THE DIAGNOSTIC WORK-UP

Clinicians should investigate sexual pain disorders with an empathic attitude, respect and kindness. Women suffering from sexual pain disorders have usually been neglected for years, treated as neurotic, referred to mental health providers, and/or substantially denied of having something deserving medical attention other than psychiatric. For many of them, to be understood in the reality of pain is the first key step to begin a constructive, effective and rewarding relationship with the physician (11). The clinical history and diagnostic work-up in sexual medicine, unfortunately unaddressed in the formal medical training as well as in the specialty one, will be presented in detail, to ease the clinical approach of the caring physician (2, 3, 11).

1) "Was the coital pain you are experiencing present from the very beginning of your sexual life ("lifelong") or did it appear after months or years of sexual well being ("acquired")? If lifelong, were you afraid of feeling pain before your first intercourse? If lifelong, dyspareunia might usually be caused by vaginismus and/or coexisting, life-long low libido and arousal disorders. Do you remember urinary tract symptoms (enuresis, urge incontinence, giggle incontinence, cystitis) preceding your sexual pain disorders? Or did such symptom(s) appear in concomitance of, or after, the beginning of the coital pain (dyspareunia)" (11, 13-15)?

2) "If the sexual pain is acquired, do you remember the situation or what happened when it started?"

3) "Is it present in any situation and/or with different partners ("generalized"), or is it limited to specific situations ("situational")?"

4) "Where does it hurt? At the beginning of the vagina, in the mid vagina or deep in the vagina?" Ask again the question while you are gently examining the patient to record her "pain map" (2, 3, 11). Location of the pain and its onset within an episode of intercourse are the strongest predictors of presence and type of organicity:

- Introital dyspareunia may be more frequently caused by poor genital arousal (of biological and/or psychosexual etiology), vulvar vestibulitis, vulvar dystrophia, painful outcome of vulvar physical therapies, perineal surgery (episiorrhaphy, colporrhaphy, posterior perineorrhaphy), pudendal nerve entrapment syndrome and/or pudendal neuralgia, Sjogren syndrome or painful outcome of female genital mutilation (FGM) (2, 3).
- Mid vaginal pain, acutely evoked during physical examination by a gentle pressure on the bilateral sacro-spinous insertion of the elevator ani muscle, is more frequently due to levator ani myalgia, the most frequently overlooked biological cause of dyspareunia, either primary (and possibly concomitant to vaginismus) or secondary to persistent coital pain and/or recurrent or chronic cystitis or anorectal painful syndromes (2, 3, 11-15).

- Deep vaginal pain may be caused by or associated with: endometriosis; pelvic inflammatory disease (PID; chronic pelvic pain; varicocele; adhesions, referred abdominal pain, outcomes of radiotherapy and abdominal cutaneous nerve entrapment syndrome (2, 3).

5) "When do you feel pain? Before, during or after intercourse?"

- Pain before intercourse suggests a phobic attitude towards penetration, usually associated with vaginismus, and/or the presence of chronic vulvar vestibulitis.
- Pain during intercourse is more frequently reported. This information, combined with the previous – "where does it hurt?" – proves to be the most predictive of the organicity of pain.
- Pain after intercourse indicates that a mucosal damage was provoked during intercourse, possibly because of poor arousal, contributing to or secondary to the pain associated with vulvar vestibulitis, pain and defensive contraction of the pelvic floor.

6) "Do you feel other accompanying symptoms, vaginal dryness, pain or paresthesias in the genitals and pelvic areas? Or do you suffer from cystitis symptoms 24-72 hours after intercourse or from other urinary symptoms?"

- Vaginal dryness, either secondary to loss of oestrogen and/or to poor genital arousal may coincide with dyspareunia.
- Clitoralgia and/or vulvodynia, spontaneous and/or worsening during sexual arousal may be associated with dyspareunia and hypertonic pelvic floor muscles.
- Post coital-cystitis should suggest the presence of a concomitant bladder vulnerability, a subclinical interstitial cystitis, a hypertonic pelvic floor muscles and/or a hypoestrogenic condition: it should specifically be investigated in fertile patients complaining of bladder symptoms first, and/or of dyspareunia (13, 14). It deserves to be investigated and treated in the post-menopause and senium as it may benefit from vaginal Estrogen Replacement Therapy (14, 16) and a specific rehabilitation of the pelvic floor aimed at relaxing the myalgic perivaginal muscles.
- Vulvar pruritus, vulvar dryness and/or feeling of a burning vulva should be investigated, as they may suggest the presence of vulvar lichen sclerosus, that may worsen the introital dyspareunia. Neurogenic pain may cause not only dyspareunia but also clitoralgia. Eye and mouth dryness, when accompanying dyspareunia and vaginal dryness, should suggest Sjogren's syndrome, a connective and immunitary disease.

7) "How intense is the pain you feel?" Focusing on the intensity and characteristics of pain is a relatively new approach in addressing dyspareunia issues. A shift from nociceptic to neuropathic pain is typical of chronic dyspareunia, as briefly indicated above; treatment may require a systemic and local analgesic approach (see Vincenti and Graziottin, this issue).

The accurate clinical history will help the physician to focus his/her following diagnostic steps.

1. Physical accurate examination to define the "pain map", inclusive of pelvic floor trophism, muscular tonus, signs of inflammation, elicited pain in the urethral and/or trigonal area, poor outcome of pelvic surgery, associated urogenital and rectal pain syndromes, myogenic or neurogenic pain, vascular problems (11). All medical factors contributing to dyspareunia in the individual patient should be described, explained to the woman (and partner!) and addressed with a multimodal approach (see Vincenti and Graziottin, this issue). Intensity of pain elicited in each point during the medical examination should be scored with an analogic scale, from 0 to ten (worst ever) and recorded in the medical chart.
2. Hormonal profile should be tested, if amenorrhea, of whatever etiology, is re-

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Table 2 - Treatment of dyspareunia.

- Address predisposing, precipitating and maintaining etiologic factors, either biological and/or psychosexual, taking a collaborative approach where possible
- Treat recurrent vaginitis and/or cystitis or other associated urinary tract or proctologic symptoms
- Restore normal vaginal trophism with topical estrogens first (always record the vaginal pH!)
- When a defensive contraction of the pelvic floor is diagnosed, teach relaxation techniques, focused on the pelvic floor muscles, inclusive of local vaginal self-massage.
- When myalgia of the pelvic floor is diagnosed with tender and/or trigger points on the levator ani, either physiotherapy and/ or electromyographic vaginal biofeedback may significantly reduce both the contraction and the associated muscular pain.
- If VVS is diagnosed, treat the pertinent predisposing, precipitating and maintaining factors
- Depending on the nature of pain (nociceptive or neuropathic), address the pain disorder with topical electroanalgesia or specific systemic and local antalgic treatments; vestibulectomy is to be reserved to chronic VVS that is not responsive to the above treatments

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ported (10). The recording of the vaginal pH during the medical examination may immediately give a measure of vaginal estrogenization and associated ecosystems (12). Increase pH is likely in postmenopausal years, from the normal pH 4 typical of the fertile age, to pH 7 of the late menopause (12). This shift may be associated with both genital arousal disorders with vaginal dryness and/or dyspareunia and recurrent vaginal and bladder infections by colonic germs. Different estrogenic preparations may improve vaginal and bladder symptoms. Controlled prospective randomized trials show that topical vaginal estrogens (17 beta-estradiol vaginal tablets, twice a week) may significantly improve symptoms of vaginal dystrophy, dyspareunia and urinary tract symptoms as well (16).

3. Psychosexual factors, poor arousal first, should as well be investigated (see Leiblum, this issue).
4. Marital issues, particularly in chronic dyspareunia, deserve special attention.

A multimodal approach aimed at curing different causal factors is mandatory to address dyspareunia and associated co-morbidities, both with other FSDs and/or other medical conditions, urological first. Relaxation of the contracted pelvic floor is a common step in most dyspareunia cases (17). Key steps of the multimodal treatment are summarized in Table 2 (2, 3, 10, 11, 14, 16-19). (See also Vincenti and Graziottin, this issue, for the specific intervention in chronic neuropathic pain associated with Vulvar Vestibulitis and dyspareunia).

CONCLUSIONS

Pain is rarely purely psychogenic. Dyspareunia is no exception. Like all pain syndromes, usually it has one or multiple biological etiological factors. Psychosexual factors, mostly low libido, lifelong or acquired, because of the persisting pain; lifelong vaginismus, when a phobic component is prominent; and genital arousal disorders, lifelong or acquired, due to the inhibitory effect of pain and/or loss of estrogens, should be addressed in parallel, in order to give a comprehensive, integrated and more effective treatment. Urinary tract symptoms, either preexisting or secondary to dyspareunia and/or genital arousal disorder, should be addressed in parallel, to cure the complex co-morbidity, both between FSD and between FSD and associated medical conditions, urological first.

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