3.5. Sexual pain disorders

Various degrees of dyspareunia are reported by 15% of coitally active women, and 22.5–33% of postmenopausal women. Vaginismus occurs in 0.5–1% of fertile women. However, mild hyperactivity of the pelvic floor, that could coincide with grade I or II of vaginismus may permit intercourse causing, though, coital pain [64].

Pathophysiology

Vaginal receptiveness is a prerequisite for intercourse, and requires anatomical and functional tissue integrity, both in resting and aroused states. Normal trophism, both 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 immune response are all considered necessary to guarantee vaginal ‘habitability’ and sexual responsiveness. Vaginal receptiveness may be further modulated by psychosexual, mental and interpersonal factors, all of which may result in poor arousal with vaginal dryness [65].

Fear of penetration, and a general muscular arousal secondary to anxiety, may cause a defensive contraction of the perivaginal muscles, leading to vaginismus [66]. This disorder may also be the clinical correlate of a primary neurodystonia of the pelvic floor, as recently proven with needle electromyography [41]. It may be so severe as to prevent penetration completely. The defensive pelvic floor contraction may also be secondary to genital pain, anal or bladder pain, of whatever cause.

Etiology

Dyspareunia is the common symptom of a variety of coital pain-causing disorders (Box 2). Vulvar vestibulitis is its leading cause in fertile age. The diagnostic triad is: 1) severe pain upon vestibular touch or attempted vaginal entry; 2) exquisite tenderness to cotton-swab palpation of the introital area (mostly at 5 and 7, when looking at the introitus as a clock face); 3) dyspareunia. The reader is referred to other publications for a more detailed analysis of different etiologies of coital pain [65,67].

Vaginismus is a painful spasm of pelvic floor muscles (levator ani) around the vagina. When mild, it makes intercourse painful, thus contributing to introital dyspareunia. Microabrasions secondary to the intercourse in dry conditions and with a tightened elevator ani, may contribute to a chronic vestibular inflammation leading to vulvar vestibulitis. When severe, it makes intercourse impossible: it is then the most frequent female cause of unconsummated marriage. It may express the local muscular correlate of a systemic muscular tension, secondary to a general systemic arousal due to the phobic attitude. Or it may be the expression of a local myogenic hyperactivity of the levator ani, isolated or secondary to genital, anal or bladder pain [65,67].

Pathophysiology

From the pathophysiologic point of view, vulvar vestibulitis involves the up-regulation of: a) the immunological system, ie of introital mast-cells (with hyperproduction of both inflammatory molecules and nerve growth factors (NGF) [10]; b) the pain system, with proliferation of local pain fibers induced by the NGF, which
contributes to neuropathic pain; c) hyperactivity of the levator ani, which can be antecedent to vulvar vestibulitis, or secondary to the introital pain. Hyperactivity of the pelvic floor may be triggered as well by non-genital, non-sexual causes, such as urologic factors (urge incontinence, when tightening the pelvic floor may be secondary to the aim of reinforcing the ability to control the bladder), or anorectal problems (anismus, hemorrhoids, rhagads).

**Box 2.** Leading Biological etiologies/risk factors of Dyspareunia (introital/superficial and deep).

<table>
<thead>
<tr>
<th>INTROITAL DYSpareunia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Inflammatory, with or without infection</strong></td>
</tr>
<tr>
<td>C.D: vulvar vestibulitis, vulvitis, vaginitis, post-coital cystitis, interstitial cystitis</td>
</tr>
<tr>
<td><strong>Hormonal</strong></td>
</tr>
<tr>
<td>C.D: vulval-vaginal atrophy/dystrophy, vaginal dryness</td>
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<tr>
<td><strong>Muscular</strong></td>
</tr>
<tr>
<td>C.D.: pelvic floor myalgia, with tender and/or trigger points</td>
</tr>
<tr>
<td><strong>Iatrogenic</strong></td>
</tr>
<tr>
<td>C.D: side effects of perineal surgery, genital radical surgery for cervical cancer or pelvic radiotherapy</td>
</tr>
<tr>
<td><strong>Neurologic</strong></td>
</tr>
<tr>
<td>C.D: neuropathic pain; or associated with a specific neurologic disease such as multiple sclerosis</td>
</tr>
<tr>
<td><strong>Psychiatric</strong></td>
</tr>
<tr>
<td>C.D: anxiety, depression</td>
</tr>
<tr>
<td><strong>Gastrointestinal</strong></td>
</tr>
<tr>
<td>C.D: ulcerative colitis, irritable bowel syndrome</td>
</tr>
<tr>
<td><strong>Vascular</strong></td>
</tr>
<tr>
<td>C.D: vaginal dryness (secondary to atherosclerosis, hypertension, diabetes)</td>
</tr>
<tr>
<td><strong>Immunological</strong></td>
</tr>
<tr>
<td>C.D: lichen sclerosus, Sjogren’ syndrome</td>
</tr>
<tr>
<td><strong>Anatomical</strong></td>
</tr>
<tr>
<td>C.D: according to the physical finding, such as a rigid, fibrotic hymen</td>
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</table>

<table>
<thead>
<tr>
<th>DEEP DYSpareunia</th>
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<tbody>
<tr>
<td><strong>Endometriosis</strong></td>
</tr>
<tr>
<td>C.D: endometriosis; invalidating chronic dysmenorrhea and/or deep dyspareunia</td>
</tr>
<tr>
<td><strong>Pelvic inflammatory disease (PID)</strong></td>
</tr>
<tr>
<td>C.D: Sexually Transmitted Disease (STD); adnexitis;</td>
</tr>
<tr>
<td><strong>Pelvic varicocele</strong></td>
</tr>
<tr>
<td>C.D: echographic diagnosis of usually left varicocele</td>
</tr>
<tr>
<td><strong>Iatrogenic</strong></td>
</tr>
<tr>
<td>C.D: iatrogenic vaginal shortening/narrowing;</td>
</tr>
<tr>
<td><strong>Neurologic</strong></td>
</tr>
<tr>
<td>C.D: acute pain elicited in the site of previous surgical abdominal incision (ACNES Abdominal Cutaneous Nerve Entrapment Syndrome);</td>
</tr>
<tr>
<td><strong>Referred abdominal pain</strong></td>
</tr>
<tr>
<td>C.D: fibromyalgia; myalgic pelvic floor</td>
</tr>
<tr>
<td><strong>Chronic pelvic pain</strong></td>
</tr>
<tr>
<td>C.D: same</td>
</tr>
</tbody>
</table>

LEGEND: C.D=Clinical Diagnosis
Adapted from Graziottin & Rovei [65]
Comorbidity with other sexual dysfunctions – loss of libido, arousal disorders, orgasmic difficulties, and/or sexual pain related disorders – is frequently reported with persisting/chronic dyspareunia.

Comorbidity between dyspareunia and other medical conditions is as well frequent and under-reported. For example, in the survey of Peters and coworkers, interstitial cystitis is associated with a significantly higher incidence of dyspareunia and fear of intercourse since the first intercourse, suggesting that the hyperactivity of the pelvic floor and sexual pain disorders are important contributors to the pathophysiology of bladder chronic inflammation and pain [68].

Clinical approach

The diagnostic work-up should focus on [65,67]:

- Physical examination: to define the ‘pain map’ (any site in the vulva, midvagina and deep vagina where pain can be elicited), as location of pain and its characteristics are the strongest predictors of type of organicity; pelvic floor trophism (vaginal pH), muscular tonus, strength and performance, signs of inflammation (primarily vulvar vestibulitis), poor outcomes of pelvic or perineal surgery (primarily episiotomy/rraphy), associated urogenital and rectal pain syndromes, myogenic or neurogenic pain and vascular problems and assess biological common denominators (such as the hyperactive pelvic floor) of medical comorbidities;

- Psychosexual factors, poor arousal and coexisting vaginismus; comorbidity with other sexual dysfunctions; investigation of any potential previous traumatic sexual experiences.

- Relationship issues

- Hormonal profile, if clinically indicated when dyspareunia is associated with vaginal dryness.

4. Current status of medical therapy

This latter professional is emerging as a key resource in addressing pelvic floor disorders, which are finally receiving the attention they deserve as key biological factors in the etiology of FSD. Pain is rarely purely psychogenic, and dyspareunia is no exception. Like all pain syndromes, it usually has one or more biological etiologic factors. Hyperactive pelvic floor disorders are a constant feature. However, psychosexual and relationship factors, generally lifelong or acquired low libido because of the persisting pain, and lifelong or acquired arousal disorders due to the inhibitory effect of pain, should be addressed in parallel, in order to provide comprehensive, integrated and effective treatment.

In tailoring of treatment, the physiotherapist has a crucial role, especially in sexual pain disorders, either lifelong or acquired, and in acquired desire, arousal or orgasmic disorders secondary to coital pain. A multimodal, individually tailored treatment (pharmacologic, physiotherapeutic, behavioural, psychodynamic, antalgic) is currently used for both dyspareunia and vaginismus.

4.1. Practical tips

Key points in the FSD diagnosis, preliminary to a well tailored treatment, should be:
Flibanserin are currently the most studied. They could cover the area on younger women with HSDD who cannot or would not take hormones to improve their desire and sexuality.

While the recent data supporting the therapeutic use of centrally acting agents, as a monotherapy for FSD is cautiously encouraging, there would seem to be tremendous promise for these centrally acting compounds to be integrated with treatment approaches that utilize other pathways in a multi-layered, individualised approach to care.

In parallel to this spring in the scientific research, there should be a parallel growth in physicians’ attitude to take care of FSD in the clinical setting. The goal is to give women and couples the full potential of a joyful sexuality in their life-span.

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