

CHRONIC PELVIC PAIN

This complex disorder can involve multiple systems and require treatment on several fronts

For many decades, chronic pelvic pain has been discussed, investigated, and treated as if it were caused by 1) a disorder of one of the visceral organ systems in the pelvis (gastrointestinal, urologic, or reproductive), or of somatic structures (pelvic floor muscles, etc) or 2) psychological dysfunction (depression, past abuse, personality disorder, anxiety, sexual dysfunction, and so on). Now, these assumptions are beginning to change.

Thanks to recent clinical investigation and experience, we are gaining a more complex understanding of the interactions among organ systems and the interplay between visceral and somatic structures and their contributions

to pain. Understanding the interactions among these components should lead to more informed therapeutic approaches.

In this article, I focus on 2 common complaints that appear to have multiple components: vulvar vestibulitis and endometriosis.

I also explore the role of myofascial tissue in pelvic pain disorders.

Conspicuously absent from this discussion is any review of surgical technique—be it robotic, laparoscopic, or other minimally invasive surgery. As beneficial as these approaches are, in general, surgical details in the case of pelvic pain matter less than the need to integrate surgery with other aspects of treatment.

Vulvar vestibulitis is a chronic pain disorder

Zolnoun D, Hartmann K, Lamvu G, As-Sanie S, Maixner W, Steege J. A conceptual model for the pathophysiology of vulvar vestibulitis syndrome. *Obstet Gynecol Surv.* 2006;61:395–401.

Erythema and hypersensitivity of the predominantly posterior vulvar vestibule are now widely recognized as a common cause of introital dyspareunia, as well as pain during daily activities. In patients with vulvar vestibulitis, a cotton-tipped applicator touched to the posterior vestibule commonly elicits allodynia (pain in response

to a typically nonpainful stimulus).

Although vulvar vestibulitis often involves muscular contraction, it now seems likely that many cases labeled as vaginismus in the past were in fact more complex, attributable to what we increasingly understand as a neuro-inflammatory disorder, as Zolnoun and colleagues observe. Research suggests that the pathophysiology of vulvar vestibulitis involves abnormalities in 3 interdependent systems:

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How this view affects treatment

A modest literature search and extensive clinical experience suggest that pelvic floor muscular spasm can accompany vestibulitis, so treating the pelvic floor with biofeedback or physical therapy or both is helpful.¹ The presence of muscular spasm is sometimes interpreted to mean that muscle dysfunction is the primary disorder and the vestibular response secondary, but my clinical experience favors the opposite view: Vestibulitis happens first, and then the muscles become involved. For many patients, both aspects require treatment.

Vulvar vestibulitis is now often viewed as neuropathic pain—that is, the activation of local pain fibers that appears to be strikingly out of proportion to any demonstrable tissue damage. Overnight application of 5% lidocaine to the vestibule for 4 weeks or more has substantially reduced dyspareunia.² (Compounded preparations of pH-neutral media are often better tolerated than the commercially available medications, which tend to be mildly acidic.) A randomized, controlled trial of this approach is under way.

Multiple studies have reported high success rates (85–95%) for vestibuloplasty. Most surgeons seem to favor excision

of the posterior vestibule and posterior hymeneal ring, covering the defect with the leading edge of advanced vaginal mucosa. This surgery is sometimes the first treatment for vestibulitis, but usually is a last resort after other therapies have failed.

Few clinical studies have explored nonsurgical treatments. Tricyclic antidepressants, long used to treat chronic pain in general, have proved helpful in treating generalized vulvodynia, as well as the more localized syndrome of vestibulitis.³

Anti-epileptic drugs, including gabapentin, carbamazepine, and lamotrigine, have been used in other pain disorders, and have recently met with some success when used for vestibulitis.

Success of different treatments highlights complexity of vestibulitis

At first glance, it would seem puzzling that treatment of the pelvic floor muscles, medical therapy for neuropathic pain, and surgical revision of the posterior vestibule would all be beneficial. This finding makes more sense if vestibulitis is viewed as one example of the interaction of several systems.

Early studies suggest potential overlap between interstitial cystitis and vulvar vestibulitis, and there may be an association between vestibulitis and temporomandibular joint disorder, supporting the notion that these conditions are members of a family of neurosensory disorders that share a common genetic susceptibility.⁴

Focus on endometriosis implants may not fully address the disease

Practice Committee, American Society for Reproductive Medicine. Treatment of pelvic pain associated with endometriosis. *Fertil Steril*. 2006;86:S156–S160.

Varma R, Sinha D, Gupta JK. Non-contraceptive uses of levonorgestrel-releasing hormone system—a systematic enquiry and overview. *Eur J Obstet Gynecol Reprod Biol*. 2006;125:9–28.

Because endometriosis involves the ectopic presence of endometrium in the

pelvis or beyond, medical and surgical treatments have traditionally targeted the implants. However, American Fertility Society staging of the disease, based on a fundamentally oncologic model (volume and distribution), is poorly predictive of the 2 major clinical morbidities of endometriosis: pain and infertility. Hence, the

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mechanisms of pain remain obscure.

Three aspects of endometriosis deserve comment here:

- Successful treatment with a gonadotropin-releasing hormone (GnRH) agonist does not necessarily mean endometriosis is present
- The levonorgestrel-releasing intrauterine system (LNG-IUS) eases pain, suggesting uterine involvement
- Surrounding organ systems appear to contribute to the disease.

Is a presumptive diagnosis accurate?

When treatment of pelvic pain with first-line agents such as NSAIDs or oral contraceptives fails, it is common practice to make a presumptive diagnosis of endometriosis and administer a GnRH agonist. If pain is relieved, it is assumed that endometriosis was the cause, but data do not support this conclusion. In the study most often cited in support of preemptive GnRH-agonist treatment without laparoscopic diagnosis,⁵ women with and without endometriosis experienced pain relief with equal frequency. The ASRM concluded in its recent guideline that relief of pelvic pain in response to a GnRH agonist does not make the diagnosis of endometriosis. This agent interrupts the hypothalamic–pituitary–ovarian axis, causing hypoestrogenism and amenorrhea, and may alter pain by

- reducing contractility of intestinal muscle
- eliminating the physiologic perimenstrual rise in pain sensitivity
- quieting uterine contractions.

How myofascial tissue contributes to pelvic pain

Tu FF, As-Sanie S, Steege JF. Prevalence of pelvic musculoskeletal disorders in a female chronic pelvic pain clinic. *J Reprod Med.* 2006;51:185–189.

More than 20 years ago, Lipscomb and colleagues⁸ identified pelvic floor dys-

Evidence suggests a uterine link

According to Varma and colleagues, the easing of endometriosis-related pain with the LNG-IUS suggests that the uterus itself—as opposed to the peritoneal implants—plays an important role. (Women with chronic pelvic pain, with or without endometriosis, have increased nerve-fiber density in the lower uterine segment.⁶) Therefore, the benefits of progestins may be at least partly attributable to their quieting effect on uterine contractility, in addition to their direct impact on endometriosis implants, and therapies thought to target implants may relieve pain in part through their impact on the uterus.

In a randomized trial, the LNG-IUS relieved endometriosis-related pain as effectively as depot leuprolide.⁷ Given that this device may remain in place for 5 years, it offers substantial benefit.

What interstitial cystitis may reveal about endometriosis

Clinical evidence suggests that endometriosis and disorders of surrounding visceral systems (eg, interstitial cystitis and irritable bowel syndrome) share some morbidities. Assuming these associations are validated by epidemiologic investigations, the common denominator may be vulnerability to inflammation (or a deficit in counterinflammatory systems), non-specific stress responses to the primary illness, genetically determined deficits in neuromodulation of nociceptive signals reaching the spinal chord,⁴ and other mechanisms awaiting discovery.

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function as an important component of pelvic pain in women, and Slocumb⁹ described abdominal wall trigger points as another. We continue to gain appreciation of myofascial contributions to

pelvic pain, although systematic study is lacking.

Tu and colleagues retrospectively studied the records of 987 women who presented to a chronic pelvic pain clinic for evaluation. Single-digit, intravaginal palpation revealed tenderness in the levator ani and piriformis muscles in 22% and 14% of women, respectively. Tenderness at these sites was associated with a higher total number of pain sites, previous surgery for pelvic pain, higher scores on the Beck Depression Inventory and McGill Pain Inventory, and worsening pain with bowel movements.

Muscles that are tender to palpation may, on occasion, be the prime movers in a pain syndrome, but my experience suggests that muscle problems often develop secondary to some other condition. For example, a woman with endometriosis may, over time, develop pelvic floor dysfunction as an important part of her dyspareunia, as a reaction to the tenderness in the posterior cul-de-sac. In a similar manner, muscle dysfunction may follow in the wake of pelvic infection or uterine enlargement, or after gynecologic surgery.

Suboptimal response to generally effective treatments is a common clue to the presence of myofascial and other factors. In this circumstance, rather than escalating treatment (eg, by operating repeatedly to treat endometriosis), the gynecologist should broaden the clinical inquiry by palpating the pelvic muscle groups during physical examination.

Can myofascial pain be treated?

Physical therapy has moved enthusiastically into the area of pelvic pain in general. Many clinicians and physical therapists have begun to look beyond the pelvic floor and recognize contributions from the hip external rotator muscles (piriformis, obturator), the sacroiliac joints, and the abdominal wall muscles. These muscle groups seem to communicate with each other at times. For example, palpation of the pelvic floor

may refer pain to the ipsilateral lower abdominal wall, and palpation of the sacroiliac joint, which may be painful itself, may also refer pain to the corresponding anterior lower quadrant. The gynecologist can readily screen for these dysfunctions, with treatment provided by the physical therapist. Follow-up by the gynecologist then permits integration of all medical, surgical, and physical therapy.

Systematic studies are needed

At present, comparisons across treatment centers are complicated by variations in clinical assessment techniques. For example, the literature describing how the bladder contributes to pelvic pain often fails to describe assessment techniques for disorders in other systems (gastrointestinal, pelvic floor, etc). When increased bladder sensitivity is then demonstrated, the reader is left to wonder whether it is the prime mover in the problem or an epiphenomenon, secondary to some other disorder. ■

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Suboptimal response to generally effective treatments suggests myofascial factors may be contributing to pelvic pain