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INVITED COMMENTARY

Understanding Multisymptom Presentations in Chronic Pelvic Pain: The Inter-relationships Between the Viscera and Myofascial Pelvic Floor Dysfunction

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Abstract Patients presenting with chronic pelvic pain frequently complain of multiple symptoms that appear to involve more than one organ system, creating diagnostic confusion. The multisymptom presentation of chronic pelvic pain has been frequently described. This article describes four proposed explanations for the clinical observation of multisymptom presentations of patients with chronic pelvic pain. These include the concepts of viscerovisceral convergence; viscerosomatic convergence; hypertonicity of pelvic floor muscles creating visceral symptoms along with somatovisceral convergence; and central sensitization with expansion of receptive fields.

Keywords Viscerovisceral convergence · Viscerosomatic convergence · Somatovisceral convergence · Central sensitization · Pelvic floor muscle dysfunction · Myofascial pain

Introduction

Patients presenting with chronic pelvic pain frequently complain of multiple symptoms that appear to involve more than one organ system, creating diagnostic confusion. The multisymptom presentation of chronic pelvic pain has been frequently described. In a study of women with interstitial cystitis/painful bladder syndrome, 93% experienced pelvic pain, 70% experienced dyspareunia, 60% experienced vulvodynia, 52% experienced constipation, 49% experienced irritable bowel syndrome, 17% experienced fibromyalgia, and 41% experienced stress urinary stress incontinence [1]. This article describes four proposed explanations for the clinical observation of multisymptom presentations of patients with chronic pelvic pain. These include the concepts of viscerovisceral convergence; viscerosomatic convergence; hypertonicity of pelvic floor muscles creating visceral symptoms along with somatovisceral convergence; and central sensitization with expansion of receptive fields.

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Viscerovisceral Convergence Creates Multisymptom Presentations

Pathway A on Fig. 1 represents viscerovisceral convergence, or innervation between viscera that converge in the dorsal horn of the spinal cord, for which pathology in one organ can create pathology in another organ. Antidromic propagation of an impulse refers to the impulse moving in a direction that is the reverse of normal. An example would be nociceptive stimulation of a primary afferent that travels to the central nervous system (CNS) as expected, but then, in addition to providing the afferent information, an impulse is propagated in the reverse, acting as an effector neuron such that inflammatory mediators are then released back into peripheral tissue or, in this case, a different visceral organ. This results in multiple symptoms experienced by the patient that appear to derive from more than one organ. Berkley [2], in her publication from 2005, summarizes her work over 35 years on the coexistence of visceral disease, such as dysmenorrhea and endometriosis, with other conditions such as interstitial cystitis (IC) or irritable bowel syndrome (IBS). In one rat model, colon inflammation produced signs of inflammation in otherwise healthy tissue of the bladder and uterus. Another model

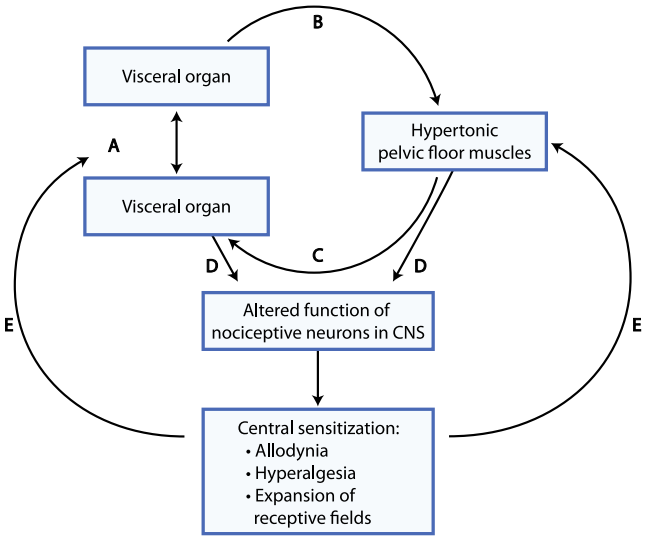

Fig. 1 Interconnected relationships between the viscera, pelvic floor muscles/myofascial structures, and the central nervous system create the multisymptom presentation of chronic pelvic pain. Viscerovisceral, viscerosomatic, and somatovisceral convergence are depicted by pathways *A*, *B*, and *C*. *D* represents nociceptive inputs from viscera and/or myofascial sources leading to central sensitization. *E* represents central sensitization creating pain perception in either myofascial structures (ie, fibromyalgia) or a visceral organ (ie, irritable bowel syndrome). All can occur in a single patient, contributing to multiple seemingly unrelated symptoms. *CNS* central nervous system

demonstrated how rats with endometriosis as the primary site of visceral dysfunction produced secondary visceral dysfunction in the form of reduced volume voiding thresholds in the bladder. She concluded the convergence of shared innervations between different organs within the CNS explains the co-occurrence of these painful conditions. Brinkert et al. [3] also concluded that viscerovisceral convergence explained how intestinal hypersensitivity was induced by severe menstrual pain.

Viscerosomatic Convergence Creates Multisymptom Presentations

Pathway B on Fig. 1 represents how pathology in a visceral organ can create muscle hyperalgesia and dysfunction and, thus, symptoms beyond what would be expected from visceral pathology only. Guarding reflexes are described as an upregulation of sacral reflexes leading to muscle tightening and pelvic floor dysfunction due to chronic inflammation or pain from syndromes such as endometriosis, ulcerative colitis, or recurrent urinary tract infections [4]. Viscerosomatic convergence is the concept that noxious afferent signals from visceral disease converge with somatic afferents in the spinal cord. Signals then are sent out to somatic structures (again, away from the spinal cord antidromically via somatic afferents) that share the same dermatome as the visceral organ. Clinically, this process was used to explain muscular hyperalgesia in the rectus abdominis in patients with dysmenorrhea and endometriosis [5]. Wesselmann and Lai [6] conducted a study in rats that demonstrated the production of neurogenic inflammation in muscles of the trunk, perineum, thighs, saddle area, and proximal tail in response to induced uterine inflammation. In another study, rats with endometriosis and rats with sham endometriosis were implanted with kidney stones as a nociceptive stimulus. The rats with endometriosis experienced increased muscle hyperalgesia compared to rats with sham endometriosis [7]. Guarding reflexes and viscerosomatic convergence are two proposed mechanisms to explain how visceral pathology can cause myofascial dysfunction and, thus, to explain why a patient may present with symptoms beyond those expected from a single visceral source.

Hypertonicity of Pelvic Floor Muscles Creates Visceral Symptoms

Pathway C on Fig. 1 represents how pelvic floor muscles can create visceral symptoms. This occurs both by direct mechanical compression of organs by tight, shortened muscles and through somatovisceral convergence. A hypertonic pelvic floor is defined by pelvic floor muscles that are short, painful, and weak. This is opposed to the more commonly recognized atrophied, weak, hypotonic pelvic floor muscles of normal length recognized to be associated with urinary and fecal incontinence, but usually not pain. Myofascial trigger points are hyperirritable spots, or contraction knots, usually within a taut band of skeletal muscle or in the muscle fascia, that are painful on compression and, thus, shorten muscles and create this hypertonicity [8]. Many visceral conditions, such as IC/painful bladder syndrome (PBS), IBS, and endometriosis, now are recognized to coexist with hypertonic, short, tight pelvic floor muscles. Normal bladder emptying is dependent on detrusor or bladder contractions. Short, weak pelvic floor muscles have limited ability to inhibit detrusor contractions, resulting in urinary urgency, frequency, and stress incontinence. Additionally, tight, hypertonic pelvic floor muscles that do not properly relax contribute to constipation and delayed voiding [9]. Weiss [10] also has observed that when pelvic floor muscles become hypertonic or develop spasm, the result is symptoms in the penetrating viscera.

Somatovisceral convergence occurs as noxious afferents from muscle and somatic structures converge with visceral afferents in the spinal cord, and again, inflammation/impulses are transmitted antidromically down to the peripheral end-organ. This was demonstrated in a study by Doggweiler et al. [11], in which somatic afferent C-fibers were stimulated by injection of pseudorabies virus into a tail muscle of their rat

model. Impulses were shown to then transmit to the spinal cord. This stimulated the release of inflammatory mediators in the periphery, the bladder in this case, creating a hemorrhagic cystitis. They conclude that this model exemplifies how somatic afferents may create neurogenic inflammation in the bladder of patients with IC. This is an example of the creation of true inflammation in a completely different structure from the original nociceptive source. The next section will describe the process of how increased perception of pain can be created in a different structure or structures than the original nociceptive stimulus. This later process is termed central sensitization.

Central Sensitization Creates Multisymptom Presentations

Central sensitization refers to the process that occurs in the CNS that results in long-term changes in the perception of pain. Additionally, it can lead to the expansion of receptive fields and, thus, pain being perceived as coming from structures beyond that of the original noxious stimulus. Alterations in the perception of pain include the development of hyperalgesia, a pain response that is exaggerated and prolonged in response to a noxious stimulus, and allodynia, where a nonpainful stimulus causes pain. Expansion of receptive fields occurs when afferents that enter the dorsal horn branch to synapse with dorsal horn cells in spinal cord segments above, below, and contralaterally. This expands the structures that are perceived to be a source of pain. Persistent nociceptive input from any source has the potential to cause central sensitization, including visceral or myofascial sources (see pathway D on Fig. 1). The role of muscles potentially creating this change in the CNS was referenced in Latremoliere and Woolf's [12] review article on central sensitization, which describes muscles and joints as being a source of nociceptor afferents that produce a longer-lasting central sensitization than that produced by afferents that innervate the skin. Once central sensitization has occurred and nociceptive neurons in the spinal cord and brain have changed and are incorrectly processing afferent information, pain can be perceived by the patient in other visceral organs or myofascial structures without any tissue damage or pathology at these peripheral sites. This hyperalgesia and allodynia can clinically manifest as IBS, IC, vestibulodynia, or fibromyalgia, as in pathway E on Fig. 1. The development of central sensitization and potentially widespread changes in pain perception is believed to explain why so many overlapping syndromes such as those already mentioned occur concurrently in the same patient. In the same way, it is another explanation for why patients with chronic pelvic pain present with multiple symptoms that appear to derive from multiple organ systems and myofascial structures.

A Proposed Mechanism of How Trigger Points in Pelvic Floor Muscles Create Changes in the Central Nervous System

Damage to the motor end plate within a muscle fiber leads to release of acetylcholine, depolarization, contraction of actin and myosin, development of a contraction knot (the trigger point), and resulting local ischemia [8]. Ischemia and a decreased pH activate local muscle nociceptors and a release of multiple neuropeptides and inflammatory mediators such as substance P, calcitonin gene-related peptide (CGRP), bradykinins, and others. This increased chemical/inflammatory milieu leads to increased excitability of the nociceptive neurons in the dorsal horn of the spinal cord (ie, CGRP stimulates N-methyl-D-aspartate receptors on the dorsal horn nociceptor, which opens Ca^{+2} ion channels and, thus, leads to depolarization), resulting in the increased synaptic efficiency characteristic of hyperalgesia/allodynia: in other words, central sensitization [12].

Conclusions

Patients presenting with chronic pelvic pain commonly complain of many seemingly unrelated symptoms that can create confusion as to the source of their pain. A helpful clinical approach to establish an accurate diagnosis and to create an appropriate treatment plan is to think of three possible scenarios. The first scenario is that the patient's source of pain is visceral only. For example, a primary diagnosis of endometriosis via viscerovisceral convergence and central sensitization may exhibit symptoms suggesting vestibulodynia and IBS in addition to typical symptoms of endometriosis. The second clinical scenario would be a patient presenting with both visceral and myofascial pain. This is commonly the case for patients presenting with IC/PBS, IBS, or endometriosis and coexisting hypertonus of the pelvic floor/abdominal wall muscles. These patients also could present with multiple symptoms for the reasons described above. The third and most under-recognized scenario is that of a myofascial source of pain only. In this situation, a patient may have a source of myofascial dysfunction (eg, a leg-length discrepancy), a fall injuring the tailbone or pelvis, or even a previous surgery. These triggers can create hypertonicity and trigger points in the abdominal wall, low back, or hip-girdle muscles and fascia that can perpetuate pelvic floor muscle tightness and dysfunction. This can create the multisymptom presentation in and of itself via direct compressive effects, somatovisceral convergence, and central sensitization. Many women with chronic pelvic pain present with pain that appears to be out of proportion to physical

examination findings (hyperalgesia), or pain on examination where it would not be expected at all (allodynia). It can be very easy to attribute these symptoms to malingering, drug-seeking behavior, or affective disturbance. It is of critical importance to understand how these characteristics are explained by the four mechanisms described above and why a multisymptom presentation has tangible and real explanations. When this is not understood, many of these patients leave an encounter with a provider feeling like their condition is "all in their head," or that they are seeking secondary gain. Many times, this will heighten their anxiety or depression and further exacerbate not only their myofascial dysfunction, but also central sensitization and pain. Thus, a multidisciplinary approach to treatment of this complicated syndrome is most effective. Identification of and treatment of all visceral and myofascial dysfunctions are critical. Additionally, it is important to recognize that many factors, such as poor sleep, nutritional deficiencies, catastrophic thinking, abuse histories, and emotional stress, can exacerbate and perpetuate myofascial dysfunction and the development of central sensitization, making treatment of these issues essential.

Disclosure No potential conflicts of interest relevant to this article were reported.

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