Myofascial and Visceral Pain Syndromes: Visceral-Somatic Pain Representations

Robert D. Gerwin

SUMMARY. Objective: To describe the relationship between viscero-somatic pain syndromes and myofascial pain syndromes [MPS].

Findings: Myofascial pain syndromes can be primary conditions or secondary. When they are secondary, they occur as a manifestation of another disorder. A regional pain referral from a visceral disorder can induce secondary MPS. Visceral disorders induce central sensitization with hypersensitivity and expansion in the number and size of receptive fields. Central sensitization is topographically organized in the spinal cord, being segmentally predominant at the level of the affected viscera. The associated MPS tend to be regional, but are related to the segmental innervation of the affected viscera. Regional MPS in turn can mimic visceral disease, or be the diagnostic sign of visceral disease. Cardiac disease, gastrointestinal disorders, hepatic and biliary disorders, irritable bowel syndrome and interstitial cystitis are some of the conditions in which MPS can occur secondarily or mimic.

Conclusion: Myofascial pain syndromes can occur as a result of visceral disorders, but can also mimic visceral disease. Visceral disease must be considered in the differential diagnosis of regional MPS. [Article copies available for a fee from The Haworth Document Delivery Service: 1-800-HAWORTH. E-mail address: <getinfo@haworthpressinc.com> Website: <http://www.HaworthPress.com> 2002 by The Haworth Press, Inc. All rights reserved.]

Robert D. Gerwin, MD, is Assistant professor of Neurology, Johns Hopkins University School of Medicine, Baltimore, MD and Medical Director, Pain and Rehabilitation Medicine, Ltd., Bethesda, MD.

Address correspondance to: Robert D. Gerwin, MD, 7830 Old Georgetown Road, Suite C15, Bethesda, MD 20814-2432 USA [E-mail: gerwin@painpoints.com].

INTRODUCTION

Visceral pain is one of the most common forms of disease-induced pain. Referred pain from viscera is likewise common, occurring in both pathologic conditions such as ureteral colic or myocardial infarction, and in nonpathologic conditions such as bowel or bladder distention. Any visceral disease can be manifest by referred pain [renal stone, gastroenteritis, appendicitis, myocardial infarction, pleurisy, pulmonary embolism, cystitis, gallbladder colic, prostatitis, etc.], and referred pain can be an important diagnostic feature. Pain can be referred from viscera to the skin or to muscle, or regionally, as to the low back where both skin and muscle are involved. An early description of a muscle or somatic component of pain that both resulted from visceral organ injury and mimicked the pain from such an injury were left pectoral muscle trigger points [TrPs] that occurred with acute myocardial infarction, and which were relieved by procaine injection into the trigger areas reported by Travell and Rinzler in 1952 (1). The clinical importance of referred pain syndromes associated with visceral disease relates to the diagnostic value of such syndromes, to the awareness that similar pain syndromes may mimic visceral disease, and to the therapeutic benefit of treating the myofascial component of pain in providing clinical relief. The same myofascial pain syndromes [MPS] that result from visceral disease, can also mimic visceral disease when they occur from other causes.

Visceral and somatic pain mechanisms share many common features. Visceral pain, for example, can be referred to the body wall. An initial response to visceral disease can be muscle tightness that is not necessarily painful, but can be a tight band or a muscle spasm (2, pp. 142-143). As in other pain-producing conditions, the referred pain may outlast the inciting pain, and remain a problem long after the original pain resolved. The referred pain syndrome may then be the only sign of a previous visceral disorder. When that is the case, it usually can be differentiated from an acute visceral condition by its persistent, dull quality. However, not all visceral pathology is acute or causes acute pain, and sometimes the residual chronic myofascial component can be sharp and severe, and appear acute, as if there is a flare-up of the underlying condition.

VISCERAL PAIN PHYSIOLOGY

Visceral referred pain shares the same central mechanisms as pain input from other structures in the development of neuroplastic changes that lead to hypersensitivity, allodynia, and expansion of receptive fields.

Hypersensitivity. Irritable bowel syndrome [IBS] is accompanied by altered visceral perception (3,4). Although studies through the mid-1990's failed to show an increase in general pain sensitivity, recent studies have shown that patients with IBS have both visceral and cutaneous hyperalgesia that is at least partly dependent on topographically organized spinal mechanisms [more pronounced in the lower extremities than the upper extremities] (5). This is reflected in the predominant localization of pain in the low back and pelvic region in persons with IBS.

Visceral pain or hyperalgesia is caused by mechanisms that are both shared with somatic pain mechanisms and that are unique to viscera (6). Tissue injury is not necessary in order to produce visceral pain. Non-damaging mechanical causes of visceral hyperalgesia include excessive distention or abnormal contraction of a hollow visceral organ, rapid stretching of the capsule of a solid organ such as the liver, and traction on ligaments and vessels. Visceral hypersensitivity to stimulation such as distention or traction is seen in visceral pain syndromes, and has been particularly well studied in IBS. Anoxia, ischemia or the accumulation of nociceptive substances, chemical irritants, and inflammatory conditions that release potassium, kinins, 5-hydroxytryptamine, histamine, or prostaglandins and that can cause tissue injury are biochemical initiators of visceral hyperalgesia. The referred pain is generally the same in both cases.

Central mechanisms of referred pain involving viscera. Central sensitization occurs at the level of the dorsal horn cell in the posterior horns of the spinal cord. Modulation of nociceptive dorsal horn cell activation occurs as a result of supraspinal facilitatory and inhibitory ascending and descending impulses.

The majority of dorsal horn cells that receive input from the viscera also receive input from receptors in the skin and/or deep tissues [viscerosomatic neurons] (7,8). Viscerosomatic convergence is the rule in visceral pain (6,9). Almost all dorsal horn cells driven by visceral input have additional somatic input (10,11). In the rat experimental model of artificial stone-induced ureteral colic, referred muscle hyperalgesia occurred early, was accentuated by repeated episodes of colic, and out-

lasted the presence of the irritant (12). Thus, muscle pain is commonly felt with visceral nociceptive activation of dorsal horn neurons.

The dorsal columns of the spinal cord transmit visceral pain information. Information carried in the dorsal columns affects pain awareness in humans. For example, midline dorsal column lesions are effective treatment for the relief of pelvic visceral pain (13). This has also been shown to be the case in animals for pain arising from colorectal distention, the duodenum, and the pancreas (14). The dorsal midline of the rat spinal cord contains both ascending tracts to the gracile and cuneate nucleus and descending corticospinal pathways. Ventrolateral cordotomies alone do not abolish visceral stimulation-evoked responses. Both the ventrolateral quadrants and dorsolateral funiculi are important for the transmission of visceral nociceptive information. Dorsal midline structures are important for input into the ventrobasal thalamus, whereas lateral spinothalamic pathways are more important for input into the ventrolateral medullary structures (15). The ventrolateral medulla is greatly involved in autonomic regulation. Visceral input through the lateral spinothalamic tracts to the ventrolateral medulla therefore plays an important role in autonomic reflex activity [such as change in heart rate], and is attenuated by lesions of the lateral column of the spinal cord, but not by midline dorsal lesions. However, the spinal cord dorsal column does not only transmit visceral nociceptive information. The dorsal columns have a more general function in pain, and, for example, have been shown to transmit information in peripheral nerve injuries like experimental mononeuropathy (16).

Sex and gender effects. Chronic visceral pain syndromes are more common in women than in men, and reflect the influence of hormonal factors on the algesic response both peripherally and centrally, the direct effect of estrogen, progesterone, and testosterone on organ function, and psychological and social factors [e.g., abuse history and gender role differences] (17,18). The effect of sex and gender on pain states is not simple, however, and changes with age as well as with sex and gender, but occurrence of pain states remains higher in women than in men in such conditions as abdominal pain, migraine, temperomandibular joint syndrome, and fibromyalgia syndrome [FMS] (19). Thus, IBS and interstitial cystitis [IC] are more common in women, and therefore, associated low back pain syndromes and pelvic floor MPS are more common in women than in men.

REFERRED PAIN AND REFERRED HYPERALGESIA
FROM VISCERA

In areas of referred pain from viscera, hypersensitivity often occurs. Visceral pain referred to the body wall is associated with muscle tenderness in the referred pain zone (6,20). Proposed mechanisms are dichotomizing or split sensory fibers, afferent-afferent interactions with orthodromic and antidromic impulses, and sympathetic reflexes to the skin causing fluid extravasation and edema. Sustained muscle contraction can occur as a result (21). Examples are hyperalgesia in the pectoralis major, the interscapular region and the forearm in myocardial infarction, lumbar muscle pain, groin pain, and flank pain in patients with ureteral colic, right upper abdominal quadrant muscle tenderness in patients with biliary colic, and lower abdominal and pelvic muscle tenderness in women with ovarian or uterine pain. The overlying cutaneous and subcutaneous tissues may also be hypersensitive. Trophic changes in the cutaneous zone of referred pain have been reported for experimental uterine inflammation in the rat (22), emphasizing the fact that functional changes also occur in the referred pain zone, from skin edema to TrP formation.

Noncardiac chest pain is a psychophysical disorder that has an organic cause like gastroesophageal reflux disease or a psychological disorder like anxiety and depression. Patients with noncardiac chest pain have a lower pain threshold to forearm ischemia and electrical skin stimulation than coronary artery patients, similar to other types of visceral pain syndromes (23).

Pain from liver and gallbladder disease is often referred to the right shoulder. The referral pattern from diaphragmatic irritation is mediated via the phrenic nerve that provides motor and sensory innervation to the diaphragm as well as to mediastinal and pleural tissues. The phrenic nerve is derived from C3-C5, so that pain referral to the shoulder in a C4-C5 distribution is really a segmental pain referral. Trigger points and a regional MPS affecting the shoulder that looks like an impingement syndrome or frozen shoulder can occur in persons with hepatic or gallbladder disorders. In addition, a local abdominal muscle wall myofascial syndrome can occur. In one instance, a 60-year-old woman presented with shoulder pain and restricted abduction and internal rotation of the arm of several weeks duration, seemingly the result of heavy household cleaning. Treatment of TrPs points in the shoulder muscles, including the infraspinatus, the subscapularis, the latissimus dorsi, and the trapezius muscles relieved her pain. There was no abdominal or right

upper quadrant [RUQ] pain or tenderness. Within two weeks she developed right abdominal wall pain following further heavy physical household cleaning, again with no RUQ tenderness or evidence of hepatic enlargement. Local treatment of abdominal oblique muscle TrPs again eliminated her pain. The shoulder and flank pain recurred within two weeks, and this time she had RUQ tenderness made worse with deep breathing and an enlarged liver. Initial investigation showed normal gallbladder and pancreatic function and appearance. She had massively enlarged congenital hepatic cysts, one of which contained 1.5 liters of fluid. Drainage of the cysts resolved her regional MPS.

Irritable Bowel Syndrome. The IBS and FMS are both common hyperalgesic syndromes, and overlap to a considerable degree. Patients with IBS show visceral hyperalgesia and cutaneous allodynia/hyperalgesia indicative of a widely distributed yet topographically organized central hypersensitivity, being more pronounced in the lower than in the upper extremities (5). The overlap between FMS and IBS is considerable, with 70 percent of FMS patients reporting chronic visceral pain and 65 percent of IBS patients having primary FMS in one study (24). The FMS is associated with hypervigilence, or hypersensitivity to stimuli of different types (25-27). Patients with IBS show hypervigilence similar to FMS patients, but show somatic hyperalgesia only when there is comorbid FMS (28). Patients with IBS show an increased vigilance towards expected aversive events, and a decreased tolerance for such stimulation, as well as hyperalgesia following a series of painful sigmoid colon distentions in contrast to controls. One postulated mechanism to explain these observations is that the pain modulatory system is altered. In support of this finding is the greater efficacy of fentanyl in attenuating the perception of phasic rectal distention and discomfort ratings to rectal fixed stimuli in IBS patients compared to controls, suggesting that IBS patients have a diminished release of endogenous opioids in response to visceral aversive stimulation (29).

Chronic pelvic pain and urologic disease. Vecchiet and Giamberardino have studied hyperalgesia in referred pain zones from urinary colic extensively (21,30). Hypersensitivity was found to be greatest in muscle in the referred pain zone. There was persistent hypersensitivity in one of three layers of the body wall in 90 percent, most predominantly in muscle, but also in skin or subcutaneous layers, and in all three layers in 25 percent of subjects. This is also true in biliary colic and dysmenorrhea. Persistent abdominal wall pain after ureteral colic can be relieved by TrP injections. Likewise, lateral abdominal wall MPS looks like ureteral colic.

Chronic pelvic pain [CPP] and IC remain enigmas and are frustrating conditions to treat (31). Interstitial cystitis is only one of a group of painful bladder-related conditions that include a variety of bladder infectious diseases, including chlamydia, drug-induced cystitis [e.g., cyclophosphamide], and sarcoid, that can cause low back and pelvic region MPS. When an organic cause for CPP is not readily found, surgery may be done to find a cause. The pain of IC is usually a moderate to severe, constant pain that is dull and aching in character. There may also be a sharper, stabbing like pain and spasm. The pain is worsened by voiding, intercourse, exercise, and by tight clothing. Pain is experienced in the perineal region [vagina, urethra] and in the supra pubic region. Fifty percent of persons with IC report having been abused as a child. In these persons, the learned behavior of voluntary sphincter control is not learned properly, causing pelvic floor muscle dysfunction. This can result in the development of TrPs that can in turn induce bladder pain, and sphincter spasm. In males, intra pelvic TrPs can cause pain like prostatitis (31). Treatment of the TrPs can decrease pain and improve bladder function. Medullary sponge kidney [MSK] is a possible cause for costo-vertebral angle [CVA] MPS. Little is written about pain from this condition, but it is associated with hematuria, red blood cell casts, and recurrent stone formation. We do not know if a patient with MSK and CVA pain and no other cause for musculoskeletal pain has MSK unrelated to MPS, or a renal-origin pain referred to the CVA.

The urogenital [pelvic] floor is innervated by the sympathetic, parasympathetic, and somatic nervous systems. The sympathetic and parasympathetic [pelvic splanchnic nerve] nervous system input is through the inferior hypogastric plexus. Somatic innervation is through the sacral spinal cord. The pudendal nerve receives sympathetic fibers in addition to somatic nerves. It innervates the penis or clitoris, the anal canal, the urethral sphincter, and anterior perineal muscles. The posterior pelvic floor musculature is innervated by the coccygeal plexus. There is an overlap of pelvic splanchnic nerve and pudendal nerve afferent input in the spinal cord, such that stimulation of one area of the urogenital floor can influence the output to another area (32). Thus, persons with urogenital pain syndromes complain about bowel and bladder dysfunction, sexual dysfunction, and show increased pelvic floor muscle tone or develop pelvic floor TrPs, and a global dysfunction of the pelvic region is often seen clinically [IBS, IC, dyspareunia]. The urogenital pain syndromes that are commonly seen include vulvodynia [which is associated with a profound hyperalgesia as shown by the stabbing neuropathic-like pain associated with touching the vulva with a

moist cotton swab], testicular pain [orchialgia], urethral syndrome [urgency, frequency, dysuria, and regional pain, similar to IC], and prostatodynia [accounting for 30 percent of patients with prostatitis, often associated with pelvic floor muscle pain] (32).

Referred pain from experimental bladder distention in female volunteers is to the suprapubic region. Repeated urinary bladder distention results in an increase in perceived painful sensations [hypersensitivity], a decrease in the intravesicular pressure required to produce discomfort [a lowering of pain threshold], and a progressive increase in cardiovascular responses [pseudo-affective reflex] (33). This correlates with the reports of urgency and discomfort reported in persons with IC in response to low intensity bladder distention. The study showed a progressive sensitization for perception of pain and for autonomic responses, although somatic [muscle wall] sensitivity was not measured. A similar finding of progressive sensitization was seen in studies of colorectal distention. It would be interesting to know if repeated bladder distention or persistent IC would lead to progressive somatic sensitivity and a lowered threshold for muscle tenderness. Could there be a reciprocal role for IC and IBS in the production of muscle wall tenderness in FMS and MPS, each leading to central sensitization that makes both the visceral symptoms worse and the muscle tenderness greater?

These questions were addressed in part in a study of sensory changes during the ovulatory phase of the menstrual cycle in healthy women (34). Pressure pain threshold, heat pain threshold, and tactile threshold were all reduced in the referred pain zones of the abdomen and low back in females during all phases of the menstrual cycle. The thresholds were also further reduced during the ovulatory phase of the cycle. A lowered threshold to pressure pain over referred pain zones in the abdominal wall and the back, but not to regions outside of the referred pain zone, supports the general notion that MPS are induced by visceral pain, but does not indicate that the visceral pain can be made worse by the TrP.

The complexity of the relationship between the pelvic floor musculature and visceral function in visceral CPPs is illustrated by a patient with a history of sexual abuse as a child, and chronic urinary dysfunction with impaired control of voiding [pressure, pain, urgency, retention] and chronic rectal and anal pain with poorly formed stools. The patient had severe pelvic floor MPS with active TrPs in all of the pelvic-related muscles [gluteal muscles, piriformis, hamstrings, psoas, quadratus lumborum, abdominal obliques, and levator ani]. Treatment was directed towards the inactivation of the TrPs in the muscles that could be reached externally, and also towards the levator ani treated in-

ternally. Biofeedback was used to improve the patient's awareness of muscle tension. Voiding changed to become more controlled, with a more predictable stream, complete emptying, and less urgency, and decreased frequency. The stool became better formed and less painful to evacuate. The presence of TrPs contributed to the visceral dysfunction that improved when the TrPs were inactivated.

CONCLUSION

Visceral pain states commonly refer pain to the axial or extremity muscle, creating a regional MPS. Similar regional MPS resulting from other causes can look identical to visceral pain-induced MPS. Therefore, visceral disorders are part of the differential diagnosis of regional MPS. A thorough history, physical examination, and a high degree of suspicion are required to distinguish a primary MPS from a secondary one.

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