

Abdominal Wall Trigger Point Case Study

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Summary:

Myofascial trigger points (TrPs) are posited to be an element in the etiology of both musculoskeletal and visceral pain. However, the recognition of TrPs as a causative factor in a patient's pain presentation varies amongst physicians and therapists. When myofascial pain syndrome is responsible for a patient's condition and is not recognized by the patient's medical advisors, the patient may be put through a plethora of testing procedures to find the cause of the patient's pain, and placed on a number of prescription medications in an effort to treat the patient's symptoms. The case review presented here involves a patient with severe anterior abdominal pain, confounded by a history of Crohn's disease, who experienced a long and difficult medical journey before a diagnosis of myofascial pain syndrome was made.

Introduction:

The concept of myofascial trigger points as a possible element in the etiology of pain is not new. Pioneering work by Janet Travell MD, David Simons MD, and Raymond Nimmo DC back in the 1950s began to introduce the concept that a focal point of muscle contraction, eventually termed a trigger point (TrP), could be responsible not only for local pain in the region of the TrP, but also referred pain to a location distant from the TrP (Simons & Travell, 1999;

Nimmo & Vannerson, 2001). With the publication of the first edition of *Travell and Simons' Myofascial Pain and Dysfunction – The Trigger Point Manual – Volume 1. Upper Half of Body* in 1983, the concept of myofascial TrPs as the cause of a patient's pain reached wide audiences. Although the TrP model is not universally accepted, notably some argue that it is a region of secondary hyperalgesia of peripheral nerve origin (Rickards, 2006; Quintner & Cohen, 1994; Butler, 2000), the validity of TrP mediated pain is now

fairly well accepted and incorporated into most fields of manual and movement therapies, including massage therapy, physical therapy, osteopathy, and chiropractic.

Following is the patient case study of an adolescent male who experienced chronic severe lower left abdominal pain and nausea as the result of abdominal wall TrPs. What distinguishes this case study is not only the severity of the symptoms, but also the long and difficult “medical journey” that the patient experienced before a diagnosis of myofascial pain syndrome was made.

Presenting Complaints:

The patient, age 16 years and nine months, presented for the first time to the author’s office in May of 2009, complaining of constant severe lower left abdominal pain and nausea. The patient stated that the pain began approximately one year before during the summer of 2008; there was no specific precipitating trauma or event. He was 5’ 6” tall, weighed 160 pounds, and is right-hand dominant.

On a pain scale of 0-10, in which 0 is the complete absence of pain and 10 is the worst pain that can be imagined, he reported the pain as ranging from a 7.5 to 9, with 7.5 being the pain on a good day, and 9 as the pain on a bad day. He described the quality of the pain as a dull pressure, never sharp. He also experienced occasional mild left testicular pain that he related to the abdominal pain. No other pain radiation was experienced.

Bending forward and sitting increased the abdominal pain, as did prolonged standing (longer than approximately 20

minutes) and lack of sleep. Lying down relieved the pain. Temporary relief was also afforded by the use of moist heat and walking on a treadmill for short periods of time (approximately 5-10 minutes).

Regarding systemic functioning, he denied any problems such as fevers, night sweats, unexplained weight changes, visual disturbances, chest pain, rash, numbness in the hands or feet, focal weakness or loss of muscular control, or difficulty swallowing, breathing or walking.

Past History:

The patient’s case was confounded by the fact that he had a history of abdominal pain and nausea caused by Crohn’s disease. He was diagnosed with Crohn’s disease in June of 2005, and immediately put on medication with initial success. When a more severe flare-up occurred, his medication was changed to Humira, an immunosuppressant that works to decrease swelling and inflammation. Humira is often prescribed for moderate to severe arthritic conditions as well as Crohn’s disease (www.Humira.com).

The patient’s symptoms of Crohn’s disease were well under control by 2008 when during the spring of that year, he began to experience left-sided abdominal pain and nausea. The pain was moderate in intensity at first, but by autumn of that year, his pain and nausea had become so severe that he was no longer able to attend school and had to remain home lying in bed or on the couch for most of the day.

The patient’s past history was negative for fractured bones, car accidents, or any incidents of physical trauma to his trunk. Other than having his tonsils removed as

a child, he had no history of surgical procedures as of the onset of pain in 2008.

The patient's social and academic history showed an active and intellectually inquisitive adolescent. Prior to pain onset in 2008, he was elected president of his high school's Future Business Leaders of America, was active in the political arena, and excelled academically in school.

Treatment History:

Because the onset of pain in 2008 was first thought to be due to an exacerbation of the Crohn's disease, the patient's parents brought him to his gastroenterologist for assessment and treatment. After a full workup, it was determined that his Crohn's disease was stable and not causing his symptomology. Further, the abdominal symptoms that he was now experiencing were somewhat different in quality than the symptoms that he had experienced from the Crohn's disease in the previous few years. Although he had experienced abdominal pain before, it had been more causally related to intestinal dysfunction, indicated by vomiting and diarrhea. Now, the pain was constant and largely unrelated to occasional intestinal dysfunction.

What ensued next was a continuing procession of physicians, tests, and medications, with little or no relief. Between the spring of 2008 and May of 2009, he had blood tests and CT scans, ultrasounds, and MRIs of his thoracic and lumbar spines. He even had exploratory abdominal laparotomy. None of these procedures shed any light on the cause of his chronic abdominal pain. Left with no clear diagnosis, his physicians prescribed

medications for his symptoms as well as for whatever etiology they felt might be causing the pain and nausea.

By May of 2009, he was taking the following prescription medications: Humira as an immunosuppressant, Tramadol as an analgesic, Librax for possible intestinal spasms, Cymbalta for a possible pinched nerve, and Prevacid and Zantac for nausea. He had also been given and had since discontinued Lyrica, Nortriptyline, a Medrol Dosepack (prednisone), and oxycodone. Of all these medications, the only ones that offered any symptomatic relief, albeit partial and temporary, were the Medrol Dosepack and Oxycodone.

Due to the lack of physical findings, his medical physicians recommended biofeedback, which provided no relief, and weekly counseling with a psychologist, as well as visits to a psychiatrist approximately once per month. His consultation at a nationally renowned pain center assessed him as having "functional and organic abdominal pain, anxiety and school avoidance behavior" with a "mild musculoskeletal component." The pain center recommended "Extensive counseling on coping and on the mind-body experience as it is established in the literature." In effect, they stated that the patient's condition was largely if not solely psychosomatic.

Status – May 2009:

As of the spring of 2009, the patient's medical care had reached a dead end. No discernable physical cause(s) had been found for his pain and nausea. The medications were being continued but his medical physicians were blaming his condition on his mental and emotional

state. Other than copious amounts of drugs to manage the pain and nausea, psychological counseling was the treatment direction that was being recommended. Throughout this experience, the patient's parents, both well educated, but not in the medical field, kept asking the physicians if there was anything else that could possibly be causing their son's condition. They were repeatedly told no. It was in desperation that the patient's mother contacted the author, a chiropractic physician specializing in soft tissue treatment, asking if there might be something that could be done.

Physical Exam – May 2009:

Upon postural exam, the patient showed bilateral overpronation of the feet on weight-bearing, with the right side exhibiting greater pronation than the left. His right iliac crest was slightly lower than the left. His right shoulder was slightly/moderately higher than the left. He had a mild/moderate hyperlordotic lumbar spine, decreased thoracic kyphotic spine, and a moderate forward-head posture, with hyperlordosis of the upper cervical spine.

Active ranges of motion of the trunk were full in all six cardinal directions (flexion, extension, right and left lateral flexions, and right and left rotations); abdominal pain increased slightly with active flexion and active extension. Passive extension

Upon palpation, his lumbar and thoracic paraspinal musculature exhibited normal tone. However, his left psoas major (PM) belly in the abdominal region was markedly tight, as was the distal PM belly/tendon in the proximal thigh (immediately distal to the inguinal ligament). He also had two distinct TrPs in his left rectus abdominis (RA) approximately one inch lateral to the umbilicus, one slightly inferior to the umbilicus, the other slightly superior. Further, palpation of the PM in the abdomen and the RA TrPs, recreated the characteristic pain pattern that he had been experiencing for the past year, with the RA TrPs being the most severe and reliably able to reproduce his characteristic pain. Based on the patient's previous assessment and treatment history and the findings of the physical exam, he was assessed with myofascial pain syndrome due to TrPs in his left RA and PM.

Discussion:

Tightness of and TrPs in the psoas major are quite common. Given that the psoas major is a flexor of the thigh at the hip joint (Neumann, 2010), it would be slackened and shortened when a person is sitting in a chair because of the posture of hip joint flexion. If a person spends large amounts of time seated, which is certainly very common in our age of computers, by the principle of adaptive shortening, hip flexors, including the

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