The Art and Science of Kinesiology

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 spams, 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 range of motion of the left hip joint was restricted; all other hip joint ranges were within normal limits.

Evaluation of breathing showed a tendency for the patient to hold his anterior abdominal wall taut as he breathed in and out.

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 psoas major would tighten (Muscolino, 2011). The patient's anterior abdominal pain pattern did not fit the usual referral pattern of psoas major TrPs, however, his local pain at the psoas major abdominal belly itself is certainly consistent with the discomfort experienced with a tight muscle.

Although anterior abdominal wall tightness and TrPs are generally not common, given the patient's history of Crohn's disease, tightness and TrPs in this region of the body would be more likely to occur due to both the long-standing chronic abdominal pain as well as the abdominal wall engagement to assist in vomiting, a common effect of Crohn's disease (Simons and Travell, 1999). The patient's referral pattern was also typical given the location of the TrPs. Rectus abdominis TrPs typically cause nausea. The region of abdominal pain that the patient was experiencing also matches the usual referral zone for the rectus abdominis. And anterior abdominal wall TrPs also often refer to the testicular region (although these TrPs are usually located more laterally than the patient's) (Simons and Travell, 1999).

Treatment:
A myofascially-oriented treatment plan was designed. It consisted of one-hour treatments employing multiple modalities applied directly to the abdominal wall, and was recommended at a frequency of twice per week. The primary goal of treatment was to alleviate the PM tightness and RA TrPs. Modalities used in each session were ultrasound, electrical muscle stimulation, moist heat, soft tissue manipulation, and stretching, usually performed in that order. The patient was also given postural advice, breathing exercises, and stretches to be done at home.

Ultrasound was used to warm the tissues, aid in breaking patterns of adhesions that likely formed due to the patient's lack of mobility for many months, and begin to decrease the stiffness of the TrPs (Draper, Mahaffey, Kaiser, Eggett, & Jarmin, 2010). Electrical muscle stimulation was employed in the tetanized mode to stimulate the Golgi tendon organ (GTO) neurological reflex for the purpose of decreasing muscle tone (Kandal, Schwartz & Jessell, 2000). A hydrocollator moist heating pack was employed to further warm the tissues, soften the fibrous fascial connective tissues, and prepare it for soft tissue manipulation and stretching (Matteini et al., 2009). Soft tissue manipulation was done to break adhesions, increase local blood circulation, and loosen muscle tone (Bucci, 2012; Chaitow & DeLany, 2000; Simons and Travell, 1999). And stretching was done at the end of each visit to lengthen and stretch the RA and PM (Muscolino, 2009; Armiger & Martyn, 2010).

Home care consisted of using moist heat followed by a hip flexor stretch. The patient was instructed to perform a lunge forward with the right foot while keeping the left foot behind (Figure 1); an alternative home stretch was also recommended in which the patient's left knee could be resting and stabilized on the floor instead. A stretch to the anterior abdominal wall was also recommended in which the patient was instructed to lie supine over an exercise ball with his trunk in extension (Figure 2). Due to the patient's tendency to hold the abdomen taut, a breathing exercise in which the patient was directed to fully relax the abdomen and let it rise during inspiration, and then to mildly/moderately contract the abdominal wall during expiration was recommended. The patient was asked to perform these stretches/exercises 2-3 times per day. For the stretches, the patient was asked to perform 10 repetitions of each stretch, each one held

for approximately 2-3 seconds, followed by a longer-held repetition held for 10-20 seconds.

Figure 1 Stretch of the left hip flexor musculature.

Classic soft tissue manipulation technique for TrP therapy is ischemic compression or sustained compression, in which compression is applied directly to the TrP and held for a sustained period of time (Simons & Travell, 1999; Davies, 2001). However, the author chose to employ deep stroking massage instead. Deep stroking is performed by performing multiple short deep strokes, approximately one inch (2.5 cm) in length, across the TrP. They are performed at a rate of 1-2 seconds per stroke and applied for approximately 30-60 seconds. The strokes can be done in any direction, but at least some should be directed along the length of the muscle fibers that contain the TrP (Davies, 2001).

This treatment approach was based on the premise that deep stroking massage would likely be most effective at increasing local circulation, which would be needed to break the TrP sliding filament contraction mechanism (Simons & Travell, 1999; Davies, 2001). Research has continued to support the "energy crisis hypothesis," which states that TrPs are caused and perpetuated by local ischemia (a lack of arterial blood supply, that results in a deficiency of oxygen to the muscle cells). Ischemia results in an inability to make sufficient amounts of adenosine triphosphate (ATP) molecules. Without ATP, it is not possible to supply the energy needed to break the actin-myosin cross-bridges formed during the sliding filament mechanism and to reabsorb calcium back into the sarcoplasmic reticulum. Without these two crucial steps, actin-myosin cross-bridges cannot be broken, resulting in a perpetuation of the TrP. Hence, a *crisis of energy*. In other words, ATP deficiency is posited to be the mechanism for the formation and perpetuation of myofascial TrPs; therefore eliminating ischemia by increasing local blood circulation would be the logical treatment approach (Muscolino, 2009).

Figure 2 Stretch of the anterior abdominal wall. Courtesy of Joseph E. Muscolino (artist: Giovanni Rimasti).

Deep stroking massage is advocated in the *Second Edition of Simons'* and Travell's Travell and Simons' *Myofascial Pain and Dysfunction – The Trigger Point Manual – Volume 1. Upper Half of Body*, published in 1999. "The technique of deep-stroking massage… is probably the most effective way to inactivate central TrPs, when using a direct manual approach… The rationale is clear." (Simons & Travell, 1999, p. 141). Deep stroking massage is also advocated in Davies' Self-Treatment of Trigger Points (Davies, 2001, p. 38-39). The mechanism of performing multiple deep strokes compared to only a few sustained compressions would better promote the physical flow of blood into the TrP (Muscolino, 2009).

Figure 3 Stretch of the left psoas major.

Two approaches to stretching the PM were employed by the author/physician: one with the client supine and lying at the side of the table with the left thigh hanging off the side into extension, and the pelvis stabilized by pressure against the contralateral anterior superior iliac spine; and the other with the client at the end of the table and hugging the right thigh into the chest to stabilize the pelvis as the left thigh was brought down into extension (Figure 3). Stretching the RA was done with the client prone and performed using contract relax (CR) stretching technique. (CR is also known as post-isometric relaxation [PIR] or proprioceptive neuromuscular facilitation [PNF] technique.) The physician lifted the patient's trunk into extension (by grasping on the patient's arms), asked the patient to take in a breath, and then breathe out as he tried to flex his trunk against the resistance of the physician for approximately 5-10 seconds. The patient was then instructed to relax and was stretched farther into extension. This was repeated for a total of four repetitions (Figure 4). For the last two repetitions, right lateral flexion was added to the extension position of stretch to increase the stretch of the left side of the anterior abdominal wall. The classically accepted premise for CR stretching technique is that it engages the GTO reflex as a neural inhibition reflex to relax the target musculature beyond that which would be obtained with a mechanical stretch alone (McAtee & Charland, 1999; Muscolino, 2009). However, the GTO reflex as the underlying neurologic mechanism of CR stretching has recently been called into question (Chalmers, 2004).

Figure 4 Rectus abdominis stretch.

Progress:

Results from the aforementioned treatment approach were steady. After two weeks of treatment (four sessions), the patient reported that he was "definitely beginning to feel better." After another two weeks, he reported the pain to be a fairly consistent level of 6; and he felt able to attend a four-week sleep-away summer camp. Sleep-away camp necessitated a break in treatment. Although there were risks in attending the camp as well as suspending care for four weeks so early in the treatment plan, the patient felt able and his parents felt that affording him the opportunity to resume his activities of life would be beneficial. He was advised to rest often and participate only as he was comfortably able. Loss of care for a month did result in a temporary loss of improvement, but treatment recommenced and the patient began to improve once again.

By September, the patient's own estimation was that his RA TrPs were approximately 35% improved and that his PM was approximately 65% improved. Objective evaluation of the tightness and stiffness of the musculature by the author estimated the improvement at 10-20% improvement for the RA, 40-50% improvement for the abdominal belly of the PM, and 80% improvement for the distal belly/tendon of the PM. With the start of his junior year at high school that September, the patient began to attend classes again, beginning at first with a few days per week, with the goal of gradually attending full time.

The physicality of attending school was difficult and clearly slowed the progress that the patient had been making, so the author decided to recommend TrP injections by a local physiatrist. In conjunction with the author's care, the patient had a total of six TrP injections into his RA over the course of the next three months. Each injection consisted of 20 mg of methylprednisolone and 2.5 cc of 1% lidocaine, followed by a 20 cc normal saline flush. By January of 2010, the patient was 90% improved and able to reintegrate into attending classes full time at school. Frequency of care was gradually lessened. By early spring, the patient was coming in for treatments once per week (he also had one further TrP injection during that time); by summer, care had dropped to one visit every two weeks. During this time, the patient continued to improve and by the start of his senior year in September of 2010, he was functionally 100% and pain-free. His care dropped to two visits in September and then once per month in October, November, and December, at which time, the patient was released from care.

All told, six months of consistent hands-on care along with physical therapy modalities and TrP injections were required to stabilize the patient's condition and return him to a functional life. Another six to eight months of manual care was necessary to completely resolve his symptoms. Diminishing frequency of care then continued for another three months to prevent the likelihood of a relapse of his condition.

Fast forwarding to the summer of 2012, the patient has completed his first year away from home at a major metropolitan university, pursuing an innovative interdisciplinary major. He made the Dean's list both semesters, was a member of the fencing team, and extremely active socially. He is participating in an internship at a not-for-profit in New York City during the summer, and will be spending his sophomore year abroad in London.

Conclusion:

This case study describes an unfortunate circumstance in which the patient's medical advisors were unaware of the possibility that at least some of his musculoskeletal and visceral pain had an underlying myofascial basis. As a result, instead of receiving swift and speedy care in early 2008 that might have quickly and efficiently stopped the progression of the patient's condition before it had a chance to become chronic and patterned into his body, his condition gradually worsened until his pain had become so severe that he was unable to function in his life. By the time that an appropriate myofascial approach was begun one year later in 2009, intense and repeated care was needed. This is not the type of case study that is so often heralded and celebrated in which a patient's condition is miraculously resolved in one or three sessions. But it is a case of young person's quality of life slowly and steadily being returned through consistent and dedicated myofascial care.

Figure credits:

Photography: Yanik Chauvin (yc@image-y.com)
Figure 2: Courtesy Joseph E. Muscolino (artist: Giovanni Rimasti)

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