Failure to Diagnose Pain of Muscular Origin Leads to Unnecessary Surgery

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ABSTRACT

Three patients who were scheduled for surgical procedures for a variety of diagnoses are presented. Each of the patients presented with pain that was interpreted as a result of an operable lesion. None of the patients was assessed for a possible muscular etiology of their pain prior to being evaluated at the author’s pain center. In all three patients, muscles were identified that replicated their pain. Each patient received treatment to his or her pain-producing muscles. None of the contemplated surgeries was performed.

Key Words. Chronic Pain; Muscle Pain; Trigger Points; Myofascial Pain Syndrome; Rotator Cuff; HNP; Epididymitis

Background

Chronic pain has been defined as pain that lasts longer than generally expected from a similar condition. Low back pain is generally considered chronic after 3 months [1]. Chronicity in other parts of the body generally is considered when pain persists after a reasonable healing period following an acute inflammatory event such as infection or trauma [2,3]. Metabolic causes, such as diabetes mellitus, may produce nerve damage that results in persistent pain [4]. The assumption in most persistent pain problems is that the pain will not be relieved without interrupting the neural circuitry perpetuating the pain or by removing the “pain generator” [5-7]. In each of the cases presented here, a pain generator was postulated, and a surgical intervention to remove the generator was planned. The patients had long-standing significant incapacity and were willing to undergo surgery.

Case 1

Diagnosis: Testicular pain. Planned treatment: Orchiectomy.

A 28-year-old married business owner with a 10-year history of left testicular pain since the age of 18 was evaluated prior to proceeding with a recommendation to surgically remove the left testicle. He had been a soccer player and in 1991 had developed pain during the course of play without any obvious injury. In 1996, he underwent varicocele surgery without any relief of his pain. In 1999, he had an exploratory surgery of the left testicle for persistent pain with removal of the appendix epididymitis and ablation of sensory nerve fibers. Postoperatively, he experienced a transient decrease in pain but, after a short period, the pain recurred.

In 1998, while defecating, his testicular pain was complicated with the onset of a stiff sensation in the left hip, which progressed over the next month to the left low back and posterior thigh. He reported ribbon-like stools and a hard lump in the left side of his rectum. He was referred to a gastroenterologist, and a sigmoidoscopy was performed without significant findings. He was diagnosed with spastic colon secondary to stress. Bulking agents and antispasmodics were ineffective.

An orthopedic consultation in July 1998 opined synovitis of the left hip. Physical therapy was prescribed, which was ineffective. In November 1998, a neurological consultant ordered a magnetic resonance imaging (MRI) scan of the lumbosacral spine, which found a bulge at L5/S1 that was thought to be unrelated. Electromyogram and nerve conduction velocities were within normal limits. Computerized tomography of the abdomen and pelvis were within normal limits. Urological consultation in January 1999 resulted in removal of the appendix epididymitis.

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mis as noted above. Chiropractic care was ineffective. An orchiectomy of the left testicle was planned.

Physical Examination
The patient was seen on January 15, 2001, and physical examination revealed a finger-to-floor measurement of 10 inches with knees together and legs erect. Straight leg raising was positive at 65 degrees on the left and 45 degrees on the right with complaints of pain in the hamstrings and buttocks. Examination of abdominal strength, hip flexors, and back muscle extensors revealed the patient to be strong. Examination of the musculature revealed trigger points in the left tensor fascia lata, gluteus maximus, piriformis, pectineus, lower abdominal obliques, and lumbar paraspinals. Neurological examination was unremarkable. The impression was of low back and hamstring stiffness and multiple trigger points in the muscles that were identified.

Course of Treatment
After discussion with the patient, a plan of treatment was agreed upon, which involved injecting the involved muscles starting with the most painful muscles first. The muscles were injected according to the technique espoused by Hans Kraus and modified by the author [8-10]. In this technique, only one muscle is injected during a treatment session. Because of the extensive needling of an indexed muscle (based on the clinical observation that injecting the origin and insertion as well as the belly of the muscle will result in lasting relief of pain without the need to reinject the previously painful muscle), the patient frequently experiences substantial discomfort following an injection session. Therefore, the technique calls for 3 days of a structured physical therapy protocol following each muscle injected. The protocol consists of 20 minutes of rhythmic (2 seconds on, 2 seconds off) neuromuscular stimulation producing a contraction of the injected muscle, followed by a limbering exercise. If pain and stiffness are encountered during the exercise, ethyl chloride spray is applied to the stiff/painful muscle, and it is moved again until maximum reduction of pain and/or stiffness is achieved. The technique for muscle injection involves needling the entirety of the origin and insertion of each involved muscle. Since the entire technique is so different from the usual technique, called trigger point injections (TPIs), the author suggests that the technique be called muscle softening injection therapy or the Marcus Method (patent pending). Due to the painful nature of the treatment, patients are premedicated with intravenous meperidine and given postinjection prescription analgesics. The area to be injected is infused subcutaneously with 5 mL of 1% lidocaine. The injectate is also 5 mL of 1% lidocaine. The total amount of lidocaine injected, 10 mL, provides some added comfort for the patient during the TPI, but is low enough to not easily produce a seizure and is well below the dose one would use to treat neuropathic pain with a lidocaine infusion [11]. In the second week of treatment, after injections of the entirety of the left gluteus maximus and left piriformis, the patient’s testicular pain was eliminated. Over the course of the next year, there was recurrence of testicular discomfort. One year later (January, 2002), he has multiple days each week with no testicular pain. On days with pain, the intensity of which the patient rated as 2-7 out of 10, the pain is managed with ibuprofen and does not interfere with walking. Prior to injections, the pain intensity was rated at 8-10 out of 10 every day and prevented walking.

Commentary
Incapacitating painful symptoms may provide the impetus to do drastic interventions to help the patient. Unfortunately, these interventions, where not clearly indicated, may at best be ineffective and costly, and at worst, productive of more pain and suffering. Irreversible procedures for pain treatment should not be undertaken without considering a possible contributing muscular etiology for the pain. Although it is not in our intellectual armamentarium to consider primary muscle pain as the cause of arcane pain symptomatology, cases such as this young man should alert us for the need to expand our evaluation and treatment paradigms to include primary muscle pain.

Case 2
Diagnosis: HNP with S1 nerve compression. Planned treatment: Decompressive laminectomy
A 35-year-old woman, status post-left L5-S1 laminectomy in September of 1999, presented with left low back pain and sciatica on October 17, 2001. She had achieved moderate relief of her low back pain and sciatica following the surgery in 1999, but she fell onto her back in June of 2000 with recurrence of severe low back pain and sciatica. An MRI showed S1 nerve root compression, and in light of her history and failure to respond to physical therapy and chiropractic care, surgery to decompress the S1 nerve root was scheduled for December 2001. The author saw her after her treating physi-
Case 3

Diagnosis: Torn rotator cuff. Planned treatment: Rotator cuff repair.

A 58-year-old man was seen in consultation on October 15, 2001 for severe right shoulder pain prior to an anticipated rotator cuff repair. The signs and symptoms included restriction in range of motion in the right shoulder with forward elevation at 20 degrees, abduction at 20 degrees, and with almost no ability for internal or external rotation. MRI showed a full thickness tear in the supraspinatus tendon and the patient was told that without surgery he would have no use of his right shoulder. He was told that postoperatively he would have a 10-month recovery and that he would probably only regain 80% of his former function. On physical examination, utilizing electrical provocation of the right shoulder girdle, the patient had severe pain in the right supraspinatus, deltoids, trapezius, biceps, and coracobrachialis muscles. Electrical provocation consists of stimulating the suspected muscle, through a roving electrode (with a stationary electrode grounded near the muscle being stimulated), with an electrical current strong enough to elicit a contraction in the chosen muscle. The author has found that muscles that will require needling will be persistently painful with repeated electrical provocations, whereas a muscle that is not persistently painful but becomes more tolerant of the stimulus, will respond to noninvasive treatments.

Utilization of a ketamine and butambine topical analgesic reduced the pain and improved the range of motion in the shoulder, suggesting that the torn rotator cuff was not the sole reason for the patient’s dysfunction and that injecting the painful muscles could potentially be helpful. The patient received TPIs and post-TPI physical therapy, with total relief of his pain and total restoration in range of motion by the end of the third week of treatment. When the MRI was reexamined, it was interpreted as a buttonhole full-thickness tear, and the patient was told that in the future he should anticipate that he would have additional problems. Three weeks after the injections to the right shoulder, the patient fell onto his shoulder, suffering bruising throughout the shoulder but with no residual restriction in range of motion. The bruising resolved, and he remained with total range of motion and pain free for 3 months until he experienced an acute exacerbation of pain and stiffness in the right shoulder and arm, primarily in the right biceps, rotator cuff, and trapezius. Physical examination revealed tenderness in the biceps, trapezius, and subscapularis, however, electrically simulating these muscles only produced a painful contraction in the biceps and subscapularis, which had not been injected previously. The right biceps and then the subscapularis were injected on consecutive days with 3 days post-injection follow-up physical therapy for each muscle injected, with complete relief of pain and restoration of full range of motion at the time of writing this paper, 2 weeks after the additional treatment.

Pain in the shoulder is frequently diagnosed as a result of a rotator cuff tear or an impingement syn-
drome. A simple examination of the shoulder that includes range of motion and identification of tender muscles could uncover a muscular etiology that is easily treatable and avoid an unnecessary surgery.

Discussion

In spite of near miraculous advances in basic sciences and clinical medicine in our understanding of basic disease processes, we remain relatively nihilistic in our pursuit of the cause of chronic pain, particularly low back pain—“The actual causes of low back pain are unknown... There is nothing that physical examination can diagnose.” [14]

The concept of pain as the disease, and the underlying cause as relatively unimportant has a compassionate basis. If we can diminish the suffering of our patients and manage the pain, we have substantively helped them. Discouraging additional tests and procedures is thought to be a humanistic approach, since the likelihood of finding and alleviating the cause of the pain is so small.

Our confusion, regarding the causes of chronic pain syndromes, occurs while we cannot agree upon a rational method to assess and treat the possible contribution of the largest organ system in the body—the muscles.

The cases presented here are only dramatic examples of what I commonly encounter in my office, where diagnostic findings other than muscles [12] have been postulated as the cause of the pain complaints. Although low back pain is the most commonly acknowledged conundrum in our quest to understand chronic pain, the cases presented suggest that the problem may indeed be more widespread.

Each of the patients presented was evaluated utilizing a structured physical exam to determine the existence and type of muscle dysfunction that could produce the presenting pain complaint. The details of the examination are based upon a multidisciplinary project at Columbia University in the 1950s spearheaded by Hans Kraus, MD, to identify muscular causes for common pain problems [15-17]. The project found that muscle tension, deficiency (weakness and/or stiffness), spasm (involuntary painful contraction restricting range of motion), and trigger points (tender points in the muscle that were nonpainful with lack of activity but would be consistently painful, oftentimes with radiation of pain suggestive of radicular pain, with more than sedentary activity) could all produce primary muscle pain. Exercise programs were developed, which addressed tension, weakness, and stiffness causing pain in the upper and lower body. A specific protocol for spasm (electrical stimulation of the muscle in spasm to achieve, first, a tetanic contraction that would fatigue the muscle, followed by a rhythmic contraction, and then limbering facilitated with ethyl chloride spray) was created in 1968 following discussions with Arthur Abramson, MD, then Chief of PM&R at Albert Einstein College of Medicine [18]. Abramson, a paraplegic as a result of multiple sclerosis, experiencing frequent painful muscle spasms, had discovered that applying a tetanizing electrical current resulting in a strong contraction of the painful muscle followed by a sinusoidal surge current producing a rhythmic contraction, frequently would relieve the spasm. Kraus successfully applied this technique to patients with spasm in otherwise healthy muscles.

Trigger point treatment was based on the work of Max Lange, MD, [19] and evolved over the years into the concept of treating the entire muscle found to have the trigger points by needling the origin and insertion of the painful muscle. The skin over the identified muscle is prepared with alcohol and iodine to establish a sterile field. This skin is then infiltrated with 5 mL of 1% lidocaine thru a 25-gauge, 5/8-inch needle. The subcutaneous infiltration as well as the actual trigger point injections are facilitated by spraying the point of skin to be injected with ethyl chloride spray prior to inserting the needle. The muscle is then injected through a needle chosen by the size of the muscle. For example, when injecting the glutaeus maximus, a 20-gauge, 3 1/2-inch needle is used to allow complete penetration of the muscle down to the bony insertion, whereas a 23-gauge, 1 1/2-inch needle is used for the rhomboids. The initial penetration is at one end of the muscle. Each penetration involves multiple placements of the needle by partially withdrawing the needle so that as much of the area reachable from the skin insertion site can be needled per insertion. The path along the course of the muscle leading to the muscle’s insertion is then needled in the same fashion. In the course of needling the muscle, only 5 additional mL of lidocaine are injected. Following the injection, the area is covered with a cold pack to minimize bleeding and swelling.

This is in contrast with other trigger point injection paradigms where the needling of one or more specific points is emphasized [20]. Since the technique involves a great deal of needling of the treated muscle, the patient frequently experiences pain and stiffness on at least the first postinjection day. Postinjection physical therapy, which is an integral part of the injection protocol, reduces pain...
and enhances flexibility in the stiff and painful injected muscle.

The difficulty in deciding whether a muscle has a true “trigger point” is described in a number of published papers [21-24]. The method used in the cases above to determine the presence of the trigger points is based on a technique developed by the author. When a trigger point is suspected, by history and physical examination, an electrical stimulus strong enough to cause a muscular contraction is applied with a roving electrode to the generalized painful area. The author has discovered that a muscle requiring needling in order to eliminate its pain and tenderness (in contrast to one requiring only physical therapy modalities) or a muscle that will be relieved with needling of another adjacent muscle with trigger points will be experienced by the patient as painfully uncomfortable when it is made to passively contract with an electrical stimulus, whereas a muscle without trigger points (i.e., not requiring needling) will not contract painfully. In each of the cases above, specific muscles were identified as the cause of the patient’s pain. Thoroughly injecting the identified muscles on 1 day followed by 3 days of neuromuscular stimulation, producing a rhythmic contraction of the injected muscle, and using gentle limbering exercises, resulted in dramatic reduction or elimination of pain and restoration of function without the need, to date, for follow-up injections to sustain the result.

**Conclusion**

These cases demonstrate that diagnosing the etiology of a painful symptom without considering a specific muscular etiology may result in costly, unnecessary, and inappropriate tests, procedures, and surgeries. The mere demonstration of some pathology, for example, torn rotator cuff, epididymitis, or herniated intervertebral disc with nerve root compression, without also looking for a coexistent and specific muscular etiology for the pain, produces a skewed perspective. It may be that an abnormality exists that could account for the painful symptom, but the abnormality may be connected, if at all, to the initiation of the patient's pain but not necessarily to its perpetuation. If this is the case, correcting an initiating factor may not address the patient's pain even though an apparent etiological factor has been eliminated.

The diagnosis of nonspecific pain, for example low back pain, should be avoided since it provides license to assume that any radiological finding can be the source of the pain, and therefore, justifies the myriad treatments for elimination or palliation of the pain [25]. Perhaps the poor ability of the standard physical examination to predict treatment outcome for painful syndromes [26-28] is in part a function of the absence of a universally agreed and administered structured protocol to evaluate and treat pain of muscular origin.

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