Twenty-Year-Old Pathogenic "Active" Postsurgical Scar: A Case Study of a Patient With Persistent Right Lower Quadrant Pain

Alena Kobesova, MD,[a] Craig E. Morris, DC,[b,c] Karel Lewit, MD, DSc,[a] and Marcela Safarova, PT[a]

Abstract

Objective: This case study describes a patient with persistent right lower quadrant and low back pain who experienced relief after manual mobilization techniques of an old appendectomy scar.

Clinical Features: A 53-year-old man with pain in the right lower quadrant of the abdomen and low back had previously failed several trials of multimodal treatments. He had an irritated old appendectomy scar in the right lower quadrant. Degenerative disk findings were also noted in the upper lumbar spine.

Interventions and Outcomes: Manual mobilization of the superficial and deep layers of the scar tissue was applied. The patient experienced an immediate pain reduction after the first treatment. Nine treatments in total were administered to the patient.

Conclusions: Assessment and treatment of "active" scar tissue may comprise an important component of the management of locomotor dysfunction and associated pain syndromes. (J Manipulative Physiol Ther 2007;30:234-238)

Key Indexing Term: *Myofascial Pain Syndromes; Musculoskeletal Manipulations*

The clinical emphasis on scar tissue has undergone an interesting evolution over the past century. From the surgical perspective, the focus on scars emphasized successful postsurgical formation to prevent clinical complications, such as infection, by ensuring that the dermal and subdermal layers became permanently sealed to avoid both hemorrhage and infection. Once the scar was morphologically established, surgeons felt that their work was essentially completed. This perspective persists among some surgical specialties in various regions. For example, Lewit and Olsanska[1] stated that the topic of scar tissue complications following the initial healing phase were currently of no clinical interest to surgeons in the Czech Republic.

Over the past few decades, increasing emphasis has been placed on scar tissue as an etiologic factor in varying types of pathologies. Such pathologies may include the following: postsurgical epidural spinal adhesions causing tethered spinal nerve roots,[2-4] postsurgical intra-abdominal scarring causing visceral (ie, gastrointestinal, urologic, and gynecologic) dysfunction,[5-7] increased risk of complications (eg, rupture or tearing of adhered vessels and organs during laparoscopic reentry) during subsequent intra-abdominal surgery,[8-11] and extremity cases in which various scar-related entrapment syndromes ensue.[12-14] Some of these conditions have come to light following technological advances in diagnostics, such as ultrasound for the localization of abdominal wall adhesions[15] or magnetic resonance imaging with contrast for epidural scarring.[16-18] Others have become more commonly addressed with newer surgical technologies, such as with the less invasive laparoscopic procedures.[19] In any case, these clinical scenarios demonstrate a gradual increase in the clinical interest in the topic of scar tissue.

Additional interest in scar tissue as a source of other types of dysfunction has been rarely addressed in the literature. Huneke[20] discussed scar tissue as an etiologic factor in locomotor system dysfunction, citing a clinical heritage dating back to the 1930s with Huneke et al. Simons et al[21] discussed their impression of the impact of scar tissue

[a] Second Medical Faculty, Rehabilitation Clinic, University Hospital Motol, Charles University, Prague, Czech Republic.
[b] Professor of Clinical Sciences, Cleveland Chiropractic College, Los Angeles, Calif.
[c] Director, FIRST Health Clinic, Torrance, Calif.
Submit requests for reprints to: Alena Kobesova, MD, Department of Rehabilitation, University Hospital Motol, V Uvalu 84, 15006 Prague 5, Motol, Czech Republic.
(e-mail: *alena.kobesova@lfmotol.cuni.cz*).
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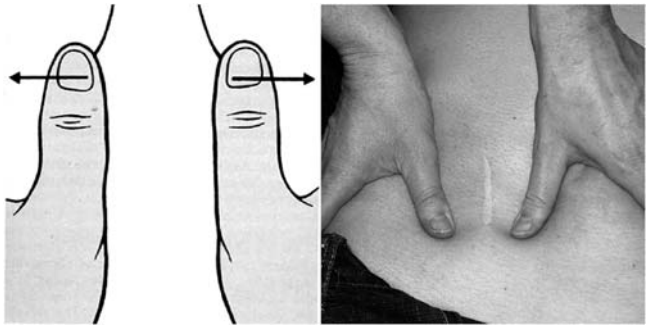

Fig 1. *Skin stretch.*

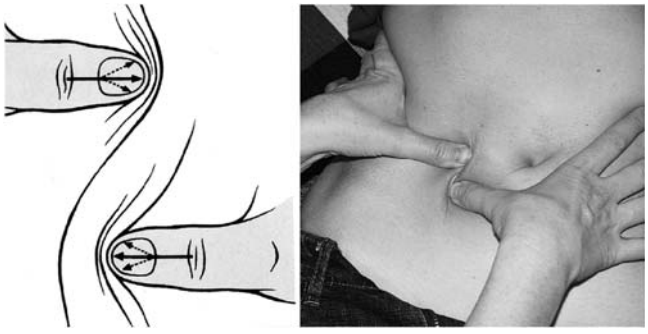

Fig 2. *Stretching a soft tissue fold.*

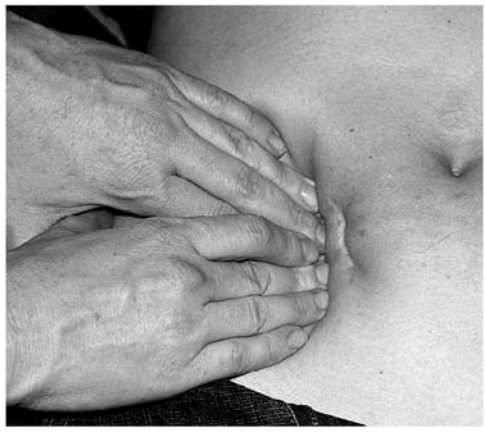

Fig 3. *Deep palpation of the abdominal cavity.*

in myofascial pain, stating, "In our experience, scar TrPs (trigger points) (in skin or mucous membranes) refer burning, prickling, or lightning like jabs of pain." The therapeutic evolution of the literature on this topic has emphasized myofascial dysfunction, whereas the topic of pathologic scar tissue has been largely ignored. Therefore, Lewit and Olsanska[22] reported a series of 51 cases in which postsurgical scar tissue was found to be the primary pain generator for a multitude of locomotor system pain syndromes.

Unfortunately, many clinicians fail to appreciate that movement of the trunk or the extremities not only involves muscles, bones, and joints, but also the soft tissues. Skin, fascia, ligaments, and tendons must stretch shift in concert. These complex interactions are frequently overlooked, but they are essential for the normal locomotor system function. Like the instruments of an orchestra, however, the functional sum is nevertheless composed of individual components. The various layers of the soft tissues must remain independent in a similar manner.

After trauma, scar tissue formation is designed to essentially replace the tissue that was, or tissues that were, traumatized. Under physiologic circumstances, the scar will typically perform this task and the various healed tissue layers will be once again individually intact and respectively returned to their organized layers.[23,24] Unfortunately, there are times when this optimal response does not occur, and clinical complications may ensue. A scar that fails to successfully establish independent layering of the tissues is defined as adhesive scar tissue. Adhesions are believed to alter the proprioceptive input of the region as a result of compromised tissue tensioning. This faulty afferent input can cause subsequent faulty efferent output, leading to a variety of complications such as protective postural patterns, increased neurovascular activity, and pain syndromes.[25-27] As such, the term *active* scar is designated to describe the ongoing additional neural activity associated with adhesive scar formations.

Active scar formation in the soft tissue can interfere with the elasticity and shifting movement of the various layers if the scars are dysfunctional. The clinical picture of such a scar is therefore similar to that of other soft tissue lesions in that there is increased dermal skin drag (due to moisture from increased sudomotor activity), there is decreased skin stretch (Fig 1) compared with the healthy (contralateral) side, and the skinfold tends to be thickened and tender when pinched (Fig 2). Flat scars do not move freely against the underlying bone. Postsurgical abdominal scars may even cause palpable resistance in the abdominal cavity (Fig 3), which must be distinguished from signs of possible visceral disease.

The diagnosis of an active or symptomatic scar is, however, only the first step in the clinical picture. The second is to assess its relevance, for even a symptomatic scar may not be relevant to the patient's problem. Its relevance can be only tested by the effect treatment of the scar has on the clinical condition.

Use of the barrier phenomenon in palpatory assessment is very important in the diagnosis and manual treatment of active scar tissue. Whether the clinician stretches or shifts the scar, there is always a free range in which little resistance is encountered. By our definition, the barrier is

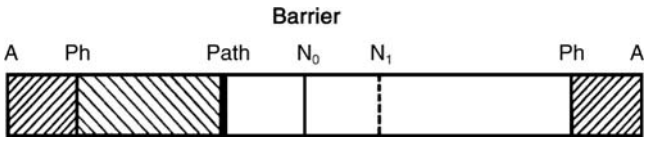

Fig 4. *The barrier phenomenon. A indicates the anatomical barrier; Ph, the physiologic barrier; N_0, the neutral point; N_1, the pathologically shifted neutral point.*

reached (engaged) at the first point of resistance. This definition implies that the physiologic barrier is soft; it easily gives and can be "sprung." Very gentle digital movement must therefore be used, which allows the first barrier of resistance to be palpated, and then the resistance gradually increases under physiologic circumstances. This progressive tensioning is analogous with that of loading a spring, giving rise to the term *springing the barrier*.[24] However, in the presence of a pathologic barrier, there is a restricted free range, and the barrier springs very little once it is engaged. Treatment involves engaging the pathologic barrier and waiting; after a short delay, a release gradually occurs until the normal barrier is restored (Fig 4).

Case Report

The patient was a 43-year-old man. In 1997, he was treated for gastroduodenal ulcer, with positive *Helicobacter pylori*, which resolved with antibiotics. He reported a history of chronic recurrent low back pain, each episode resolving on its own. The condition for which he sought treatment began in 2000, with pain noted in the right lower quadrant of his abdomen; he was repeatedly examined by a number of clinicians of varying specialties. Despite several evaluations, nothing pathologic was found. After playing golf on April 4, 2004, he experienced an excruciating exacerbation of his pain. The pain was located in the right lower abdominal quadrant and radiated into the right groin, testicle, and also to the anterior aspect of the right thigh. He was completely immobilized by his pain.

The patient was first examined at the neurologic department, where renal colic was ruled out. He was hospitalized on April 16 at the neurologic clinic of the University Hospital Motol in Prague, Czech Republic. After neurologic examination, numerous laboratory tests and specialty consultations were performed. The results of complete blood panel (differential blood count, erythrocyte sedimentation rate, HLA-B27, CRP, ASLO, latex, basic liver and renal parameters) and urine analysis (basic chemical analysis, urinal sediment, culture urinal) were negative, as were spinal tap, ophthalmologic examination, excretory urography, and urologic examination. After an internal examination, the conclusion was that the patient was experiencing recurrent abdominal colic of unknown etiology. No acute abdominal condition or any other condition requiring surgery was found upon surgical consultation. The psychiatric consultation revealed an uncharacteristic, acute stressful reaction to cumulative strain, secondary to pain associated with nociceptive neuropsychological and conversion mechanisms. Radiographs of the lumbosacral spine (anteroposterior and lateral views) showed degenerative spondylosis with degenerative osteophytes on the ventral aspect at L1 and L2 with narrowing of the L1-2 disk. Magnetic resonance imaging of the thoracic and lumbar spine revealed a slight disk protrusion at T6. Computerized tomography of the abdominal cavity revealed a small cyst in the right hepatic lobe and slight liver steatosis. Musculoskeletal findings included right-sided dysesthesia along the L1 dermatome, slight antalgic posture to the left (although no scoliosis was present on x-ray), pain in the thoracolumbar region with associated paraspinal and iliopsoas hypertonicity, antalgic posture, and gait. The diagnostic impression was that of L1 nerve root irritation on the right.

Subsequent treatment by the neurologic clinic consisted of the following oral medications: indomethacin, tramadol, paracetamol, pethidine, amitriptyline, and carbamazepine. Spasmolytic suppositories and drops were also provided, as were intravenous deliveries of natrium salicylate, guaifenesin, and trimecaine. Unfortunately, these courses of treatment demonstrated minimal benefit. After this comprehensive and costly diagnostic and therapeutic program, the patient was referred to our rehabilitation clinic for his persistent and unchanged symptomatology.

On examination, an antalgic posture (trunk deviation anterior and to the right, pelvic shift to the left) was noted. He walked with a limp, apparently protecting his right leg. Low back extension produced immediate pain in the low back and in the right groin region. Springing (posterior to anterior) the lumbar spine with the patient lying on his side revealed painful movement restriction at the L5/S1 segment. An unusually symptomatic appendectomy scar was observed. There was erythema surrounding this 20-year-old scar, which was tender even on gentle palpation, and demonstrated increased skin drag. The skin resisted springing, and hypesthesia with allodynia was noted. A pathologic barrier was found not only at the dermal layer but also in the subcutaneous tissues, mainly at both ends of the scar. In addition, deep palpation was painful, with palpatory resistance noted in the abdominal cavity.

Based upon our findings, a therapeutic strategy was established, which included a trial course of manual scar release procedures. Treatment began with gentle skin stretch, which was initially painful. After a few seconds, release was obtained, which the patient noted was associated with relief of his pain. We then obtained a release in the

deeper layers of the scar, including both the fascia and muscle. This was accomplished using the same gentle techniques: engaging the barrier in each layer with minimum force, then waiting at the barrier until full release was obtained. Again, the patient initially felt pain when releasing the deeper scar tissue, but it soon gave way to relief. The entire treatment took about 15 minutes.

Immediately after treatment, the lower quadrant, groin, testicular, and low back pain disappeared. The patient was able to straighten up and to walk normally. The restriction at L5/S1 also seemed normalized. The following day, the patient was discharged from the hospital. Because of some residual pain in his lower right abdomen, he visited our clinic at regular intervals (1-2 times a week) for another 6 weeks for a total of 9 treatments. The scar was treated by application of hot packs followed by manual soft tissue treatment. In addition, stretching and mobilization of the dorsal fascia and mobilization of the lumbar spine was applied. The patient was taught how to independently treat himself with low back self-mobilization and by stroking and stretching the scar.

Discussion

An overriding question in this case is why the patient developed a pain syndrome 20 years after the appendectomy if the etiology was from the scar. First, it is hypothesized that active scars can remain dormant for extended periods because they are neither released (ie, normalized) nor sufficiently provoked. A variety of factors may come into play that, individually or in concert, could provoke this condition. These may include a minor trauma, such as a sudden turn, misstep, awkward or heavy lifting, or perhaps even a sneeze, which may have suddenly loaded and slightly torn the adherent scar. The patient might not remember such a commonplace incident if it was initially a painless event. Second, additional contracture of the scar can occur,[23] which would increase the tone and logically stimulate local afferents. Third, aging and conditioning factors may have occurred, such as weight gain, decreasing connective tissue tone, and compromises in intra-abdominal pressure. Although this is speculative, the authors of this article have indeed found that active scars can occur long after the occurrence of the trauma, although 20 years afterward, is clearly noteworthy.

The treatment of scar tissue for otherwise unexplained symptoms using local injections of analgesics such as novocaine dates back more than 60 years in German medical literature.[20,28,29] Some acupuncturists have applied dry needling, as did one of the authors (KL) of this article.[30] It is important to understand that the same diagnostic criteria, using the palpatory principles of the barrier phenomenon, apply to scar tissue as well as any other connective tissue.[31,32] It is therefore logical that the same therapeutic procedures suitable for soft tissue lesions may also be applied to a scar.

Differential diagnosis is particularly important if the painful resistance can be felt in deep structures, such as the abdominal cavity. There is, however, an important diagnostic criterion: if resistance is due to a scar, we can sense release after engaging the barrier and waiting for that release. In the presence of underlying pathology, this release is either short lived or does not occur at all. In such an event, clinical follow-up is mandatory.

Soft tissue lesions, such as scar tissue, can cause locomoter dysfunction. Locomoter dysfunction is concerned with motor programs, and a lesion of any link (including soft tissue) can affect the program as a whole, such as the posture, gait, and lumbar extension in our patient.[33] The diagnosis of the patient was particularly striking because the scar was obviously painful, with visible erythema that should have resolved within a few months following the appendectomy. This demonstrates a classic active scar. These clinical findings are not usually so evident. As with other soft tissues lesions, the clinical significance of a scar is revealed by palpatory assessment. In retrospect, this patient's symptoms are easily explained: the tender appendectomy scar was stretched by backward bending and left side bending and relieved by forward flexion and right side bending. This explains why the patient adopted an antalgic posture to reduce tension on the irritated active scar.

It is important for doctors to provide a differential diagnosis to rule out serious pathologic conditions. On the other hand, we should reduce wasteful, costly, and frequently unpleasant diagnostic procedures to a reasonable minimum. It is this critical balance that helps to define the art of manual medicine. Therefore, it is important to assess for and diagnose an active scar in cases such as this. Once isolated, the scar should be given a trial course of treatment before giving treatment to any other diagnosed lesion (ie, segmental movement restriction, myofascial trigger points, etc). Only in this way can we establish the clinical relevance of the active scar.[32,33] If the response is positive and the condition of the patient markedly improves, then the etiology of the condition appears to have been confirmed, and appropriate therapeutic follow-up can be started. If the improvement is permanent, we may discontinue further clinical management.

Conclusion

Assessment for and treatment of active scars should be part of the routine management of painful conditions of the locomotor system. This seems logical if there is frequent recurrence of symptoms not otherwise explained, if clinical findings do not sufficiently explain a patient's symptoms, or if a patient's complaints begin or significantly deteriorate when a scar is formed. In this case, a 20-year-old appen-

dectomy scar seemed to be related to a patient's low back pain and groin pain.

Practical Applications

- Active scar tissue may be an overlooked cause of locomotor system pain.
- Active scar tissue may cause altered locomotor system function in the acute, subacute, and chronic stages after the trauma that caused the initial scar formation. In this case, two decades had passed between the abdominal surgery that caused the scar and the onset of pain.
- Manual treatment of active scar tissue appeared to resolve the condition for this patient.
- Determining whether an active scar is a key etiologic factor in a given case can be quickly determined using gentle manual techniques, saving significant resources if performed in the early clinical stages of the condition.

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