

20-Year-Old Pathogenic “Active” Post-Surgical Scar: A Case Study of a Patient with Persistent Right Lower Quadrant Pain

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Five Learning Points:

- “Active Scar” tissue is an often overlooked cause of locomotor system pain’
- Active Scar” tissue can cause altered locomotor system function in the acute, subacute and chronic stages following the trauma that caused the initial scar formation. In this case, several decades had passed between the abdominal surgery that caused the scar and the onset of pain.
- Manual treatment of “Active Scar” tissue can effectively resolve the condition.
- Manual treatment of “Active Scar” tissue can be a very challenging technique, requiring special manual training skills for optimal results.
- Determining whether an “Active Scar” is a key etiological factor in a given case can be quickly determined utilizing gentle manual techniques, saving significant resources if performed in the early clinical stages of the condition. This case demonstrates how significant clinical resource was expended needlessly prior to assessing for and resolving the causative “Active Scar” condition.

Short description:

A 53-year-old male with right lower quadrant and back pain who failed to respond to prior multimodal trials of treatment. An “active scar” was ultimately isolated and the symptoms were subsequently ameliorated with manual scar release techniques.

Abstract:

Objective: To describe a case study in which a patient with persistent right lower quadrant and low back pain responded to manual therapeutic techniques directed to his “active scar”.

Clinical Features: The clinical progress of a 53-year-old male with pain in the right lower quadrant of the abdomen and low back, who previously failed several trials of multimodal treatment, is discussed.

Interventions and Outcomes: The prior diagnostic evaluations and therapeutic trials will be discussed. Then, an evaluation of the patient will be presented documenting pertinent clinical findings that led to the diagnosis of “active” scar tissue as the key etiological factor in the patient’s symptomatology. A trial course of treatment to release the “active” scar is explained, in addition to a more comprehensive strategy to allow for functional restoration and stability, in order to reduce the likelihood of recurrences.

Conclusions: This successful case study suggests that assessment and treatment of “active” scar tissue comprises an important component of the management of locomotor dysfunction and associated pain syndromes. The skills to isolate and release such scar tissue should be considered an important corollary to those manual methods commonly taught for other types of locomotor dysfunction.

Key Indexing Terms: Active scar, Soft Tissue, Mobilization, Myofascial Pain

Introduction

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

Over the past few decades, increasing emphasis has been placed on scar tissue becoming an etiological factor in varying types of pathologies. These pathologies vary significantly, from post-surgical epidural spinal adhesions causing tethered spinal nerve roots (2-4), to post-surgical, intra-abdominal scarring causing visceral (i.e. gastrointestinal, urological and gynecological) dysfunction (5-7), to increased risk of complications (e.g. additional trauma, such as rupture or tearing, of adhered vessels and organs during laparoscopic re-entry) during of subsequent intra-abdominal surgery (8-11), to 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 MRI 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. Lewit discussed scar as an etiological factor in locomotor system dysfunction, citing a clinical heritage dating back to the 1930's with Huneke et al (20). Travell and Simons discussed the impact of scar tissue with myofascial pain, stating, "In our experience, scar TrPs (Trigger Points) (in skin or mucous membranes) refer burning, prickling, or lightning like jabs of pain." (21). Unfortunately, Lewit notes that the therapeutic evolution of the literature on this topic has emphasized myofascial dysfunction, while the topic of pathologic scar tissue has been largely ignored. Therefore, he and Olsanska reported a series of 51 cases in 2004, in which post-surgical scar tissue was found to be the primary pain generator for a multitude of locomotor system pain syndromes (22).

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 move in harmony, i.e. all these structures have to stretch and to shift in concert. These complex interactions are frequently overlooked, but they are essential for the normal function of the muscles and joints. 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.

Following trauma, scar tissue formation is designed to essentially replace the tissue that was, or tissues that were, traumatized. Under physiological circumstances, the scar will indeed perform this task and the various healed tissue layers will be once again individually intact, respectively returned to their organized layers (23-24). Unfortunately, there are times when this optimal response does not occur, and clinical complications may ensue. 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 due to compromised tissue tensioning. This faulty afferentation can cause subsequent faulty efferentation, 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: increased dermal skin drag (due to moisture from sweating, increased sudomotor activity), decreased skin stretch (Fig.1) compared to the healthy (i.e. contralateral) side, and the skin fold tends to be thickened and tender when pinched (Fig.2). Flat scars do not move freely against the underlying bone. Post- surgical 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.

Utilization 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 practically no resistance is encountered. By our definition, the barrier is reached (engaged) at the first point of slight resistance. This definition implies that the physiological barrier is soft; it easily gives and can be “sprung”. Very gentle digital movement must therefore be utilized, which allows the first barrier of resistance (i.e. the “barrier”) to be palpated, and then the resistance gradually increases under physiological circumstances. This progressive tensioning is analogous with that of loading a spring, ergo the term “springing the barrier”. (24) However, in the presence of a pathological barrier, there is a restricted free range and the barrier springs very little once it is engaged. Treatment involves engaging the pathological barrier and waiting: after a short delay, a release gradually occurs until the normal (i.e. physiological) barrier is restored (Fig.4).

Case Report

Patient: P.A., a 43-year-old male, electrician/manager, married, non-smoker.

Past medical history: As a child he suffered from recurrent streptococcal tonsillitis; A case of chronic appendicitis resulted in appendectomy in 1984. In 1997 he was treated for gastro-duodenal ulcer with positive helicobacter pylori, resolved with antibiotics. He demonstrated a history of chronic recurrent low back pain, which resolved without any formal treatment. (It always resolved spontaneously, then he was pain free for a certain time, then he got another attack of LBP)

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 pathological was found. After playing golf on April 4th 2004, the patient 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.

He was first examined at the neurological department, where renal colic was ruled out. He was hospitalized on April 16th at the neurological clinic of the university hospital Motol in Prague. Following neurological examination, the following laboratory tests and specialty consultations were performed:

1. Complete blood (diff. 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)
– with negative results
2. Spinal tap (cerebrospinal fluid examination) negative results
 3. Ophthalmologic examination – negative results
 4. X-ray examination of the lumbosacral spine (AP and lateral views):
degenerative spondylosis with degenerative osteophytes on the ventral
aspect most prominent at L1 and L2 with narrowing of the L1/2 disc.
 5. MRI of the thoracic and lumbar spine: slight disc protrusion at T6
 6. CT of the abdominal cavity: small cyst in the right hepatic lobe, slight
liver steatosis
 7. Excretory urography: normal excretory renal function
 8. Conclusion after internal examination: recurrent abdominal colic of
unknown etiology
 9. Urological examination: normal findings
 10. Surgical consultation: no acute abdominal condition nor any other
condition requiring surgery
 11. Psychiatric consultation: uncharacteristic acute stressful reaction to
cumulative strain secondary to pain associated with nociceptive neuro-
psychological and conversion mechanisms.

Neurological 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 paraspinous and iliopsoas hypertonicity, antalgic posture and gait. The diagnostic impression was that of nerve root irritation L1 on the right.

The subsequent treatment from the neurological clinic consisted of the following medications - orally: Indometacin, Tramadol, Paracetamol, spasmolytic suppositories and drops, Pethidin, Amitryptilin, Carbamazepin ; intravenous: Natrium Salicylate, Guaifenesin, Trimecain. Unfortunately, these trial courses of treatment demonstrated minimal benefit.

Following this comprehensive and costly diagnostic and therapeutic program, the patient was referred to our rehabilitation clinic for his persistent and unchanged symptomatology on May 11, 2004.

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. Back bending (i.e. 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.

In addition, 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 due to dermal moisture; the skin resisted springing, while hyperesthesia with allodynia was also noted. A pathological 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 entailed 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. Even the restriction at L5/S1 was normalized. The following day, May 13th 2004, 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) until June 29th, a total of 9 times. 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. Also, the patient was taught how to independently treat himself with low back self-mobilization and by stroking and stretching the scar.

Discussion

A patient of 53 years was hospitalized in the neurological clinic for severe pain in his right lower abdomen, right groin, right testicle and low back. After exhaustive medical, surgical, X-ray and laboratory testing, and a failed trial of various medications he was referred to the rehabilitation department where the etiology of his symptoms, an active appendectomy scar from surgery 20 years prior, was revealed.

An over-riding question in this case is why the patient developed a pain syndrome 20 years following the appendectomy if the etiology was from the

scar? First, it must be remembered that active scars can remain dormant for extended periods because they are neither released (i.e. normalized) nor sufficiently provoked. Therefore, the answer to this question is uncertain, but a variety of factors may come into play here 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 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 paper 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 utilizing 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 of this paper (30). It is important to understand that the same diagnostic criteria, utilizing 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 can also be applied to 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 release. In 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 motor dysfunction. Motor function, and dysfunction, is concerned with motor programs, and a lesion of any link (including soft tissue) will affect the program as a whole (posture, gait, back bending in our patient) (33) The diagnosis of the patient was particularly striking, as 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 is stretched by back bending and left side bending and relieved by anteflexion (i.e. forward

flexion) and right trunk deviation (i.e. lateral flexion). This explains why the patient adopted an antalgic posture in order to reduce tension on the irritated active scar.

It is important for doctors to provide a differential diagnosis in order to rule out serious pathological 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 (i.e. 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 desist from 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 is mandatory if there is frequent recurrence of symptoms not otherwise explained: if the clinical findings do not sufficiently explain the patient's symptoms; if the patient's complaints begin or significantly deteriorate when the scar is formed.

The diagnostic criteria can include: Movement restriction of the skin and soft tissue is present in the vicinity of the scar. A hyperalgesic zone is present upon palpatory assessment. There is resistance against manual distraction and shifting. There is tenderness on palpation and resistance in the deeper tissue layers. The presence of post-surgical erythema of the scar long after it should have resolved. The presence of increased sweating (i.e. sudomotor activity) noticed by increased skin resistance to palpatory skin drag methods.

Once the diagnosis of such active scar is established, the scar should be treated with a trial course of care. Manual therapy of active scars consists of releasing the skin and the subcutaneous tissue. Utilizing gentle digital pressure to deep structures where a tissues fold can neither be formed nor stretched. Treating large flat scars adhering to the bone similarly to fascia. The additional use of hot, which can be preformed by the patient independently. Teaching the patient to perform self-treatment and independent exercises.

It is most important to start the patient's treatment by treating the scar first, to be able to assess the effect on the patient's condition. If the effect is clearly positive, we may avoid unnecessary and costly diagnostic and therapeutic procedures, if we can follow up the patient for a sufficient length of the time. Manual treatment of such scars is noninvasive, almost painless and without any risk of known side effects.

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Illustrations:

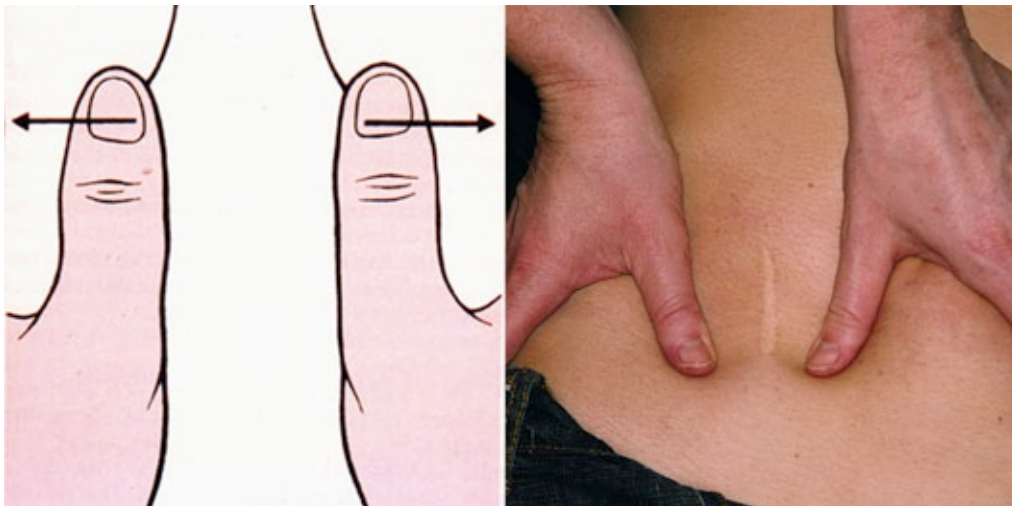


Fig. 1: Skin stretch

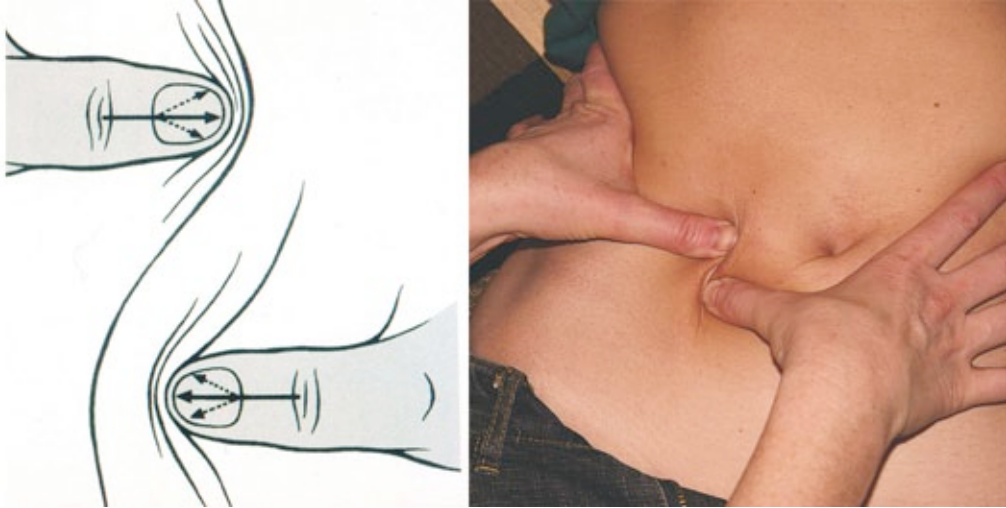


Fig. 2: Stretching a soft tissue fold



Fig. 3: Deep palpation of the abdominal cavity

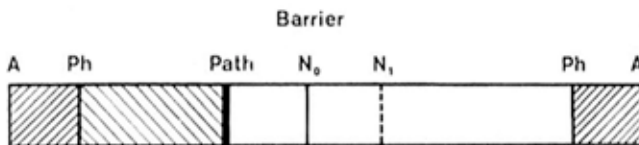


Fig. 4: The barrier phenomenon: A the anatomical barrier, Ph the physiological barrier, No the neutral point, N1 the pathologically shifted neutral point.