Chest Pain in Focal Musculoskeletal Disorders

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Primary health care providers play an important role in the initial evaluation and treatment of patients with acute or chronic chest pain. As chest pain may have benign and life-threatening causes, it is imperative that clinicians have a thorough and structured approach to the evaluation of the patient with chest pain. The musculoskeletal system is recognized as a benign cause, but is sometimes not given the same systematic approach as when a more serious condition is suspected.

In patients with chest pain, the first priority for clinicians is to consider potentially life-threatening causes, but in primary care, the cause of pain may be benign in approximately 80\% of cases, of which musculoskeletal chest pain accounts for almost 50\%.\textsuperscript{1–3} In patients with noncardiac chest pain, a sizeable minority are never diagnosed or given a plan for follow-up.\textsuperscript{4–7} Nevertheless, as many as 75\% experience persistent or recurrent symptoms,\textsuperscript{8,9} and the lack of a diagnosis may result in depression,\textsuperscript{10} anxiety\textsuperscript{11} and a decrease in daily activity.\textsuperscript{8,10,12} In addition, noncardiac chest pain may lead to inappropriate and unnecessary investigations and management with associated further anxiety and time lost from work.\textsuperscript{6,13} In patients with noncardiac chest pain, conflicting evidence exists regarding mortality. Some studies have indicated an excellent prognosis for survival and a future risk of cardiac morbidity similar to that reported in the background population,\textsuperscript{14–16} whereas others have indicated much poorer outcomes.\textsuperscript{17–19} Therefore, it is imperative that the clinician has a current knowledge of the diagnostic approaches to patients with chest pain, especially musculoskeletal chest pain, to expedite diagnosis and appropriate management.

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thereby reducing unnecessary referrals to expensive clinical investigations and unnecessary patient anxiety.

**PREVALENCE**

**General Population**

The prevalence of musculoskeletal chest pain in the general population has recently been evaluated by 2 cross-sectional surveys. In Australia (2003), Eslick and colleagues\(^9\) defined musculoskeletal-like chest pain as chest pain worse on movement. Using self-report questionnaires, 35% (n = 76) of those reporting noncardiac chest pain also described pain that could be musculoskeletal in origin, equally distributed between female and male responders. In 2007, in a cross-sectional survey of more than 34,000 Danish twins by Leboeuf-Yde and colleagues\(^20\) the 1-year prevalence estimate of radiating pain from the thoracic spine to the chest wall was 5%. Women were more often affected, especially when they were in their late 40s and late 60s.

In the Copenhagen Heart Study (1978), questionnaire responders from an urban cohort with suspected stable angina pectoris (Box 1) were invited to a clinical examination.\(^21\) Of those accepting, 18% (n = 81) had musculoskeletal chest pain, more commonly seen in women than men (24% vs 11%, respectively).

**Primary Care**

In primary care, the prevalence of thoracic pain has been estimated to account for 1% to 3% of all contacts with general physicians, with musculoskeletal chest pain ranking as the most common diagnosis accounting for 21% to 49% of those patients with chest pain.\(^1\)\(^-\)\(^3\)\(^,\)\(^22\) The prevalence of musculoskeletal chest pain peaked at ages 21 to 40 years. Women were more often found to be affected than men.\(^2\)

**Coronary Care Units and Emergency Departments**

Several studies have looked at the prevalence of musculoskeletal chest pain in coronary care units and emergency department in patients with suspected acute coronary syndrome. In those patients without acute coronary syndrome, a nonspecific diagnosis of musculoskeletal chest pain has been evaluated in 4 studies.\(^6\)\(^,\)\(^14\)\(^,\)\(^23\)\(^,\)\(^24\) Musculoskeletal chest pain ranked from the most prevalent cause at 23%\(^6\) to the fourth most prevalent cause at 3%, following chest pain of cardiac undetermined origin, gastrointestinal origin, and pulmonary disease.\(^14\)

In addition, 3 studies have specifically defined subgroups and diagnostic procedures that result in higher prevalence of musculoskeletal chest pain (almost 30% in patients without acute myocardial infarction).\(^5\)\(^,\)\(^25\)\(^,\)\(^26\)

**Outpatient Settings and Patients with Suspected Stable Angina Pectoris**

In patients referred for coronary angiography, 11% to 18% have been classified as having musculoskeletal chest pain.\(^27\)\(^,\)\(^28\) In patients with normal coronary arteriography these figures increase to 23% to 35%.\(^29\)\(^,\)\(^30\)

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**Box 1**

**Definition stable angina pectoris**

A clinical syndrome characterized by discomfort in the chest, jaw, shoulder, back or arm. It is typically aggravated by exertion or emotional stress and relieved by rest or nitroglycerine.

DEFINITIONS

The musculoskeletal system is recognized as a possible source of pain in patients with chest pain. However, a confident diagnosis of musculoskeletal chest pain can be difficult to establish because no reference standard exists to verify this diagnosis. The broad term musculoskeletal chest pain encompasses pain from many different musculoskeletal sources and with many proposed mechanisms, including traumatic, nontraumatic, inflammatory, and noninflammatory origins, but definitions and terms overlap (Box 2). To complicate matters there are only a few Medline subheadings (MeSH) that adequately differentiate aspects of musculoskeletal chest pain to guide clinicians and researchers in efforts to gain knowledge in this area.

For all of these conditions, with the exception of degenerative pathology of the spine, psoriatic arthritis, spondyloarthropathies, and stress fracture, the diagnosis is essentially based on history and clinical examination findings with the use of investigations to exclude other conditions, rather than to confirm the diagnosis. This article focuses on the clinical features of the focal musculoskeletal disorders most commonly diagnosed as causes of musculoskeletal chest pain. However, only segmental dysfunction has been evaluated in prospective clinical trials by chiropractors. The generalized pain syndromes of fibromyalgia and fibrositis are addressed elsewhere in this issue.

<table>
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<th>Box 2 Subgroups of musculoskeletal chest pain</th>
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<td>Cervicothoracic angina</td>
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**Tietze Syndrome**

Tietze syndrome was first described in 1921 by the German surgeon Alexander Tietze (1864–1927). Even though the syndrome has frequently been described in the literature, true Tietze syndrome is probably rare. Tietze syndrome is characterized by the presence of painful swelling of the costal cartilages caused by a benign inflammation. Although similar, Tietze syndrome is not identical to costochondritis, which also affects the costal cartilages. The inflammation and swelling of the costal cartilages seen in Tietze syndrome are absent in costochondritis. Tietze syndrome affects younger people of either gender with a predilection for the upper ribs, particularly second and third costochondral junctions. Lesions are unilateral and single in 70% to 80% of cases. If multiple, lesions usually affect neighboring articulations on the same side. Onset may be insidious, and heat and erythema may be present. It is now recognized that in Tietze syndrome the presence or absence of swelling is only an indicator of the severity of the condition. Although the true causes of Tietze syndrome are not well understood, it often results from a physical strain or minor injury, such as excessive coughing, vomiting, or impacts to the chest. The syndrome was at one time believed to be associated with, or caused by, a viral infection acquired during surgery, but this is now known not to be the case. Diagnosis is based on clinical grounds after exclusion of other conditions. Routine investigations do not show any abnormalities, but bone scintigraphy and ultrasound are suggested as screening procedures. Pain usually has a self-limited course within weeks to months, but may in cases become more chronic. Suggested treatments consist of reassurance, local application of heat and use of nonsteroidal antiinflammatory drugs (NSAIDS). Local steroid or lidocaine injections may be indicated in refractory cases, but there are no formal studies to support the evidence of a potential treatment effect of any of the suggested therapies.

**Costochondritis**

Costochondritis is used interchangeably with costosternal syndrome and chest wall syndrome, and definitions are not consistent. These syndromes are all characterized by pain and local tenderness at costochondral or chondrosternal articulations, or even at the xiphoid process, but without the inflammation and swelling seen in Tietze syndrome. It is presumably a much more common cause of chest pain affecting as many as 30% of presentations in emergency departments. In primary care, costochondritis has been reported in 42% of patients with musculoskeletal chest pain, but as with Tietze syndrome, reports of prevalence are mostly based on cases, and the exact prevalence is difficult to evaluate. Little is known of the pathogenesis, but in a subgroup of patients with costochondritis, an association with seronegative spondylarthropathies has been recognized, and costochondritis has been seen as the presenting feature in such conditions. In others, repetitive physical activity has been reported as a precipitating factor. Pain may be provoked at rest, during movement of the ribcage or related to breathing. It most often has a self-limited course, but recurrences are common as are prolonged cases. Treatment includes reassurance, and manual therapy has been shown to be beneficial in some cases. Injections with local anesthetics for immediate response and sulphasalazine for prolonged effect have been suggested as beneficial treatment options.

**Muscular Tenderness**

Along with costochondritis, muscular tenderness of the intercostal and pectoral muscles may be one of the most common causes of musculoskeletal chest pain. In primary care, intercostal tenderness has been reported to be the most common origin...
of pain, comprising almost 50% of all patients with chest pain.\textsuperscript{2} Others have reported pectoral tenderness to be the second most common cause of musculoskeletal chest pain accounting for approximately 25\% of cases and intercostal tenderness accounting for only 9\%.\textsuperscript{3} Another study in a hospital setting examining patients referred for coronary angiography with stable angina pectoris found up to 98\% of all patients with an overall diagnosis of chest pain from the musculoskeletal system to have chest wall tenderness.\textsuperscript{27}

The cause has been attributed to a history of unaccustomed or excessive activity, such as lifting, painting a ceiling, chopping wood, coughing or exertion of undertrained muscles. Similarly a state of tension or anxiety can produce excessive tension. Onset may be either gradual or sudden. The patients will report localized pain and tenderness over the strained muscles. Pain is made worse by maneuvers that tense or stretch the muscles.\textsuperscript{35}

Within different clinical disciplines (eg, rheumatology, cardiology, or chiropractic), these clinical conditions of muscular tenderness are diagnosed with manual palpation (\textbf{Figs. 1 and 2}). However, the manual palpation methods have rarely been evaluated in clinical settings. Only 1 study, to our knowledge, has evaluated the reliability of palpation for tenderness of the chest wall and found great variation between examiners when palpating for tenderness.\textsuperscript{27} This may hamper clinicians’ ability to diagnose and classify part of the potential muscular component of chest pain.

\textbf{Segmental Dysfunction of the Neck and Thoracic Spine}

Coming from the posterior aspect of the chest wall or neck, segmental dysfunction of the spine is perhaps one of the most under-diagnosed causes of musculoskeletal chest pain.\textsuperscript{43} The term “segmental dysfunction” refers to a disturbance of function affecting quality and range of motion of spinal segments without structural change. The definition embodies disturbances in function that can be represented by decreased or aberrant motion.\textsuperscript{44} Segmental dysfunction in the lower cervical (C4 to C7) and upper thoracic spine (Th1 to Th8) may cause pain referred to the anterior aspects of the chest wall.\textsuperscript{27,45} This is because dysfunctional spinal segments tend to refer pain to a zone corresponding to the distribution of the segmental innervations of the deep structures.\textsuperscript{46} Excessive strain in the spinal joints after trauma, effort, or false movement may lead to this abnormal firing of nociceptive structures.\textsuperscript{43} Segmental dysfunction may be present with or without degenerative pathologies of the spine.

\textbf{Fig. 1.} Manual palpation of the anterior chest wall using soft contact with the index or middle finger contact and with the clinician placed behind the patient.
Segmental dysfunction has been reported to account for between 14% of patients with musculoskeletal chest pain and 29% of all patients admitted with acute chest pain suspected of acute myocardial infarction. In a population with chronic chest pain admitted for coronary angiography at a cardiology department, 18% of the population was found to have a clinical syndrome of chest discomfort originating from the cervicothoracic spine and thorax, called cervicothoracic angina (CTA). Using the same definition in a population of patients with acute chest pain and no clear diagnosis at initial presentation, the prevalence is currently being evaluated in a PhD thesis, the results of which will be communicated elsewhere.

The prospective clinical study of patients with chronic chest pain by Christensen and colleagues has been able to establish a few clinical characteristics that could differentiate patients with CTA and patients without. The different pain descriptors appeared with similar frequencies in the 2 groups, except for sharp pain, which was more frequent in the patients positive for CTA, who also had their symptoms for a shorter time and with less frequent episodes. Physical activity provoking pain was significantly less frequent in the CTA-positive group, and they suffered more often from self-reported neck pain, thoracic spine pain, and shoulder-arm pain. In contrast to common beliefs, movement of the thorax was seldom found to provoke the chest pain in patients with chronic chest pain (8%, P = .06). In the study by Christensen and colleagues, systematic classification of patients according to international guidelines for type and severity of angina pectoris appeared to be an important indicator for distinguishing patients with and without CTA. Type of angina pectoris was classified according to Diamond and Forrester into classes of typical, atypical,
**Box 3**

Clinical features with significant differences in frequency between CTA-positive patients and CTA-negative patients with chronic chest pain

CTA-positive patients are more likely to have

- Sharp pain
- Shorter duration of pain
- Neck pain
- Thoracic spinal pain
- Shoulder-arm pain
- Noncardiac pain and atypical angina

CTA-negative patients are more likely to have

- More frequent episodes of pain
- Pain related to physical activity
- Typical angina
- Higher CCS class of angina severity


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**Fig. 3.** Patterns of referred pain from deep somatic structures of the thoracic and lumbar spinal segments based on experiments by Kellgren et al and Feinstein et al in which spinal and paraspinous structures were injected with hypertonic saline. (From Dvorak J, Dvorak V. Manual medicine diagnostics. Stuttgart (NY): Georg Thieme Verlag; 1990; with permission.)
and noncardiac chest pain, whereas severity was graded in 4 categories in accordance with the Canadian Cardiovascular Society (CCS). Noncardiac pain and atypical angina pectoris were significantly more frequent in the CTA-positive group. In addition, there was a significant trend for generally higher CCS class in the CTA-negative group.

Pain caused by segmental dysfunction has been reported to be worst at rest or after prolonged sitting and with spinal rotation, but pain worst at rest has only been reported in approximately one-third of patients with chronic chest pain and does not seem to be a discriminatory factor. Unlike in ischemic heart disease, activity may relieve pain. However, the opposite scenario with pain on physical activity and relief by rest may also be the case in up to 50% of patients with CTA. Paraspinal muscular tenderness is often present together with spinal joint dysfunction, and sometimes pain may be reproduced by palpation of the spinal joints and related structures (Fig. 4). Excessive strain (acute or repeated) in such joints after trauma, effort, or false movement may lead to this abnormal firing of nociceptive structures.

Benefits of spinal manipulative treatment (SMT) have been evaluated in a nonrandomized clinical trial comparing patients with CTA and treated with SMT with untreated patients without CTA. Approximately 75% of patients with CTA reported improvement in pain and in general health after treatment, compared with a statistically significant smaller proportion of 22% and 25% of patients without CTA. However, the design of this nonrandomized trial had several limitations and the value of SMT was not fully elucidated.

**Cervical Angina**

Cervical angina has been defined as chest pain that resembles true cardiac angina but originates from cervical discopathy with nerve root compression. Cervical angina has been described by different investigators in the past 70 years. Patients with cervical angina may be considered in an end stage of a progressing pathology in which degenerative changes of the spine have led to discopathy. Unlike most other focal musculoskeletal chest pain syndromes, the diagnosis can be confirmed by magnetic resonance imaging or radiographic findings. The patients require specialist attention for further evaluation and management.

![Fig. 4. Manual palpation of thoracic spine motion. The patient is guided by the clinician through movements of rotation and/or side bending by a gentle push of the forearm of the clinician’s indifferent hand. At the end of range of motion, the clinician’s palpating hand gently applies extra force over the spinal joints to assess joint movement.](image)
**Slipping Rib**

Slipping rib, also called rib-tip-syndrome, is a less known source of mechanical rib pain, accounting for approximately 5% of musculoskeletal chest pain cases in primary care.\(^3\,\text{,}\,35\,\text{,}\,36\) Slipping rib is attributed to loosening of the fibrous attachments binding the lower costal cartilages to one another allowing a rib tip to curl upwards and over-ride the inner aspect of the rib above, causing pressure on the intercostal nerve between. The disorder is most likely traumatic in origin, as many patients recall past injury to the affected side. Onset is insidious, with intermittent unilateral pain in the lower margin of the ribcage. A painful click is sometimes felt over the tip of the costal cartilage involved with certain movements. The costal cartilage involved is tender and moves more freely than normal on palpation. Remission is slow and pain may linger for several months. Beneficial therapies include reassurance and mild analgesics.

**Mechanisms of Visceral and Somatic Chest Pain**

Chest pain may be broadly categorized as visceral or somatic in origin. Visceral pain includes pain from structures including the heart, esophagus, stomach, and so forth. whereas somatic pain includes pain from the musculoskeletal structures, dermal tissues, and the coverings of major organs. The depth of the tissue in somatic pain tends to determine if it is superficial somatic (skin, tendon sheaths, periosteum, superficial fasciae) or deep somatic (muscle, fasciae, tendons, joint capsules, ligaments, periosteuem).\(^59\)

Using cardiac pain as illustrative of the mechanisms of visceral pain, cardiac pain is transmitted by afferent sympathetic nerve fibers and vagal nerve fibers.\(^59\) This visceral type of pain is mediated by free nerve endings that have receptors located in the mucosa, muscle, and serosa of the heart and can be stimulated chemically or mechanically.\(^60\,\text{,}\,61\,\text{,}\,62\) The sympathetic nerves have cell bodies in the dorsal root ganglia and synapse on the interneurons in the dorsal horn of the spinal cord. Interneurons that receive visceral pain are called viscerosomatic neurons. They also receive somatic afferent input from skin, tendons, and muscles. The visceral pain from the heart is transmitted via the 4 to 5 upper thoracic spinal segments as well as some cervical segments. The sensory impulses from the viscera of the thorax and from the body wall (muscles, skin, joints), however, share the same spinal segments, making the differentiation of causes and diagnosis of chest pain difficult in some cases.\(^63\,\text{,}\,64\) The convergence of visceral and somatic pain fibers on the same interneurons in the spinal cord might explain why visceral pain is often referred, that is, why it is often perceived in somatic areas remote from the involved organ.

Therefore, pain arising from different organs, such as the chest wall, esophagus, or the heart may be indistinguishable as may pain arising from the spine.\(^64\) The ascending pathways to the brain project through the anterolateral system of the spinal cord to the medulla, midbrain, and thalamus. In the medulla, visceral pain pathways interact with the reticular formation, mediating arousal and autonomic responses to pain. In the midbrain, projections to the periaqueductal gray matter are important for descending modulation. From the thalamus, visceral pain input is relayed to areas of the cortex, as demonstrated in man using positron emission tomography, where it is decoded as a painful sensation.\(^65\)

Using musculoskeletal pain as illustrative of the mechanisms of somatic pain, sensory receptors in musculoskeletal structures are characterized by an ability to respond to a particular stimulus with a relative insensitivity to certain other stimuli.
Nociceptors are sensitive to stimuli that are potential noxious. Mechanical, chemical, and thermal stimuli may cause nociception. The nociceptors can be sensitized by a wide variety of mechanisms that can lower the threshold from nociceptive stimuli and make it possible for nonnociceptive stimuli to be registered as pain-producing stimuli. The pain impulses are mediated by afferent nerve fibers linking the peripheral nociceptor with the spinal cord. The primary afferent fibers enter the dorsal horn of the spinal cord and synapse with second-order neurons. The ascending pathways have been described previously.

Different musculoskeletal structures of the spinal region have been characterized as pain producing in human studies from the mid-twentieth century using irritant injection solutions. Kellgren outlined the distribution of pain from ligaments, which was different from the well-known dermatomes. He found that fascia, periosteum, and tendons give rise to pain of segmental distribution. Feinstein outlined the distribution for pain and tenderness when the interspinous ligament is irritated. Several spinal segments referred pain to the anterior chest wall. Furthermore, the thoracic zygapophyseal joints at segments T3 to T9 can cause intense areas of provoked pain one segment inferiorly and slightly lateral to the joint injected and similar pain referred toward the anterior chest wall. Large and more recent studies of patterns of musculoskeletal referred pain do not exist.

**DIAGNOSTIC METHODS**

Most musculoskeletal chest pain syndromes are essentially clinically diagnosed without reference standards to verify the diagnoses. The cornerstone to diagnosis is manual palpation of pain and motion of muscles and joints of the chest wall and cervicothoracic spine. As a result, the syndrome is difficult to confirm and susceptible to interobserver variation. The interobserver variation of manual palpation of the spine has been evaluated several times. Results indicate that although there is a high degree of interobserver variability in palpation of spinal motion and of anterior muscular tenderness of the chest wall, the degree of interobserver variability in palpation of tenderness and assessment of patients based on full clinical evaluation is limited to a clinically acceptable level.

The issue of verifying clinical diagnosis, especially spinal motion and pain, without reference standards has been addressed through various indirect measures. Results lend support to the validity of palpation in detecting spinal segmental dysfunction. One study has used myocardial perfusion scintigraphy as a proxy measure in patients with suspected stable angina pectoris referred for coronary angiography. The results suggest that an experienced clinician can fairly convincingly identify a subset of patients with suspected angina pectoris as having segmental dysfunction. Diagnosis was based on a 4-step approach encompassing a combination of palpation of the chest wall, neck and thoracic spin (see Figs. 1, 2 and 4), classification of type (more common among noncardiac chest pain), and severity of angina (more frequent with a CCS grade 1), the presence of neck pain, sharp pain, and pain relieved by rest. Additional indirect support for the diagnosis of segmental dysfunction in this patient subset came from improvements in pain and general health with a trial of manual therapy.

**IMPLICATIONS FOR CLINICAL PRACTICE**

The implications of the material discussed in this article for clinical practice are summarized in Box 4.
SUMMARY

Despite being a recognized and frequent source of chest pain, focal musculoskeletal disorders remain poorly understood. Nevertheless, the small amount of available research indicates that a detailed case history with emphasis on pain characteristics, precipitating, provoking and relieving factors, and classification of patients according to international guidelines of angina pectoris should be used in combination with a systematic, manual palpation of the spine and chest wall to positively diagnose a focal musculoskeletal cause of chest pain. Appropriate conservative treatment approaches, including manual therapy and mild analgesics, can be initiated to treat the sometimes disabling cause of the disorders.

REFERENCES


Box 4

What can the clinician do?

- Take a systematic case history to identify cases of excessive strain (acute or repeated) that may indicate a musculoskeletal origin of pain; pay attention to traumatic origin of the chest pain
- Use internationally accepted classification systems of angina pectoris (ie, according to CCS and Diamond and Forrester) to identify those patients with low risk of ischemic heart disease
- Do a systematic and structured palpation of the cervical and thoracic spine and the anterior and posterior aspect of the thorax to identify signs of inflammation, and to identify segmental dysfunction and muscular tenderness
- Perform a clinical examination including neurologic examination for sensory disturbances, muscular strength, and peripheral reflexes of the upper and lower extremities to rule out nerve root compression. Look for swelling, heat, and erythema of the costosternal joints
- Possible referral for manual assessment (chiropractor) if the clinician does not have the palpation and treatment skills to clarify a musculoskeletal diagnosis
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