Treatment of a female collegiate rower with costochondritis: a case report

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Rib injuries are common in collegiate rowing. The purpose of this case report is to provide insight into examination, evaluation, and treatment of persistent costochondritis in an elite athlete as well as propose an explanation for chronic dysfunction. The case involved a 21 year old female collegiate rower with multiple episodes of costochondritis over a 1-year period of time. Symptoms were localized to the left third costosternal junction and bilaterally at the fourth costosternal junction with moderate swelling. Initial interventions were directed at the costosternal joint, but only mild, temporary relief of symptoms was attained. Reexamination findings included hypomobility of the upper thoracic spine, costovertebral joints, and lateral ribs. Interventions included postural exercises and manual therapies directed at the lateral and posterior rib structures to improve rib and thoracic spine mobility. Over a 3-week time period pain experienced throughout the day had subsided (visual analog scale – VAS 0/10). She was able to resume running and elliptical aerobic training with minimal discomfort (VAS 2/10) and began to reintegrate into collegiate rowing. Examination of the lateral ribs, cervical and thoracic spine should be part of the comprehensive evaluation of costochondritis. Addressing posterior hypomobility may have allowed for a more thorough recovery in this case study.

Keywords: Costochondritis, Joint mobilization, Rib, Thoracic spine

Chest and rib injuries have a high prevalence (26%) among female rowers.[1–3] Pain which is localized to the costochondral or costosternal joints is typically associated with the diagnoses of costochondritis or Tietze's syndrome. There is a higher prevalence in females both in general[4,5] and athletic populations such as rowing.[1] The diagnosis is usually based on clinical symptoms[2] and imaging studies offer little value.[6] These two conditions are relatively similar with the exception that costochondritis exists without swelling, heat, or erythema.[2] Pain can be provoked with upper extremity movements, most commonly shoulder horizontal adduction.[2] Symptoms may be recurrent[5] and persist for months,[7] but are thought to typically resolve within 1 year.[4,8]

Costochondritis does not have an associated known etiology, but is thought to be due to inflammatory conditions, trauma, or insidious onset.[2] Limited reports of proposed mechanisms of injury include pull of surrounding musculature,[3] repetitive arm adduction,[3] and hypomobility of posterior spinal structures.[3,9,10] Costochondritis is thought of as a self-limiting condition[2] allowing individuals to continue athletic participation as symptoms allow. Conservative management is usually symptomatic[2,3,11] and includes reassurance,[2] oral analgesics,[2] and local injections.[5,7] Cases in which symptoms do not dissipate with typical conservative management can present challenges for the patient and clinician. Patient reevaluation and attempts to further identify the underlying cause of symptoms may be necessary.

Recent clinical suggestions[12] and case reports have included the use of manual therapy interventions directed at the thoracic spine in the management of rib injuries.[3,9,10] The rib and associated thoracic vertebral segment can be described as a fixed ring analogous to a hula hoop. Movement and stress applied at one portion can be transmitted through the entire ring. For example, during thoracic flexion the posterior rib rotates anteriorly (internal torsion) and elevates while the anterior portion of the rib translates inferiorly.[13] Similar types of coupled motions occur with rotation and side-bending as well.[13]

Unfortunately the potential mechanical explantation for the cause of costochondritis is limited.[2,10] Altered thoracic spine and rib mobility may be a factor associated with the development of costo-

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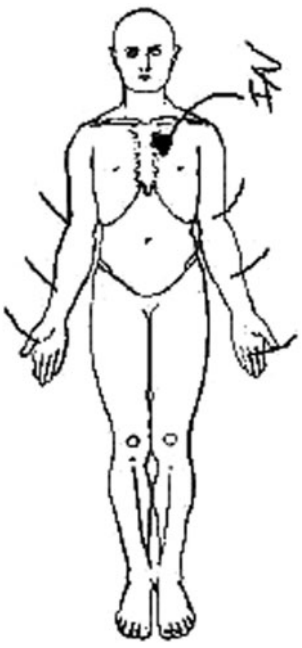

Figure 1 Pain diagram. Pain was localized to the left lateral aspect of the sternum in the area of the costosternal joints.

chondritis and identification of the underlying cause of costochondritis is necessary for appropriate management. The purpose of this case report is to provide additional insight into the examination, evaluation, and treatment of persistent costochondritis in an elite athlete as well as propose an explanation for chronic dysfunction by examining rib and thoracic spine osteokinematics and arthrokinematics.

Patient characteristics

The patient was a 21 year old female (height= 185.4 cm; mass=63.0 kg) collegiate rower with multiple episodes of costochondritis over a 1-year period of time. The onset of symptoms occurred during the spring semester of her third academic/athletic year (February 2006) and persisted into the spring of her fourth academic/athletic year (May 2007). She predominately rowed starboard side which required repetitive thoracolumbar flexion, left rotation, and left side bending. Her primary complaint was pain at the left third and fourth and right fourth costosternal junctions, with her worst pain localized to the left third costosternal junction (Fig. 1). Moderate swelling was present over each of the associated costosternal joints. She recalled no specific trauma that precipitated the pain. Pain was rated using a visual analog scale (VAS) with worst pain (6/10) occurring with rowing, running, elliptical, deep breathing, weight training, and movements which required shoulder horizontal adduction. She indicated that pain was sometimes accompanied with an 'intense popping' sensation which would provide temporary mild relief of symptoms and she was able to reproduce this 'popping' with shoulder movement (horizontal adduction). Her past medical history included intermittent low back pain since her freshman year of high school.

Examination

Initial examination

Initial examination was performed late February 2006. The patient had full upper extremity active range of motion. She did demonstrate gross hypermobility (score 5/9) based on Beighton–Horan laxity scale.[14,15] Upper extremity manual muscle test for major muscle groups were all graded 5/5. Resisted shoulder horizontal adduction reproduced familiar pain. Upper extremity sensation to light touch was intact. Posture was observed in standing where both increased thoracic kyphosis (head forward, rounded shoulders) and increased lumbar lordosis were noted. Palpation of the affected area, anterior/posterior compression, and lateral compression all reproduced pain. Swelling was localized to the third and fourth costosternal junctions and a slight anterior protrusion of the left third and fourth costosternal joints was noted via palpation. Crepitus and cavitation were present around the costosternal joints with active shoulder horizontal adduction.

X-rays were negative for acute fracture or displacement (sternoclavicular and costosternal joints). Slight elevation of the left clavicle relative to the right was noted and thought to be due to muscle spasm. The patient continued participation in practice and intercollegiate races through the spring 2006 season. Interventions included stretching anterior chest musculature, therapeutic exercise targeting the pectoralis major, and modalities (electrical stimulation, pulsed ultrasound, iontophoresis, and cryotherapy) localized to the costosternal joints and celecoxib 200 mg one time per day. Prior to regional competition in May 2006 the patient received a fluoroscopic guided corticosteroid injection (0.25 cc 0.5% bupivacaine and 0.25 cc Kenalog) of the third costosternal joint. This reduced her symptoms (VAS 1/10) and she was able to compete in post-season races. Recommendations for the summer off-season included relative rest and general conditioning exercises.

Upon returning to the university in August 2006, the patient indicated she received approximately 1 month relief from the initial injection, but symptoms returned over the summer. Clinical findings were similar to previous examination which included pain and swelling localized to the third and fourth costosternal junctions. She resumed a rehabilitation program directed at the affected costosternal joint including pulsed ultrasound, cryotherapy, electrical stimulation, iontophoresis with dexamethasone, and Grade II anterior to posterior rib mobilizations. She also resumed celecoxib 200 mg one time per day.

Despite these measures her symptoms persisted and a bone scan (99mTc methylene diphosphonate)

was obtained at the end of September 2006. Results of the bone scan indicated no focal region of abnormal uptake and were negative for stress fracture. Four days later she received her second fluoroscopic guided corticosteroid injection (0.25 cc 0.5% bupivacaine and 0.25 cc Kenalog) of the third costosternal joint. Following injection, she resumed training for the fall 2006 season and remained asymptomatic for approximately 3 weeks. During this time she was also compliant with the rehabilitation program directed at the affected costosternal joint. Unfortunately, symptoms increased toward the end of the competitive fall season and she was not able to compete in any of the three races. During the winter off-season she was advised to avoid rowing and other exacerbating activities such as running and upper extremity resistance training.

The patient resumed training at the beginning of the spring 2007 semester. She continued to have symptoms localized to the third and fourth costosternal junctions and received a third fluoroscopic guided corticosteroid injection (0.25 cc 0.5% bupivacaine and 0.25 cc Kenalog) of the third costosternal joint. She was able to fully participate in preseason spring training, but symptoms returned approximately 3–4 weeks following injection. She continued conservative rehabilitation and also utilized a lidocaine patch 5%, applied 8–12 hours per day for approximately 1 month. This relieved local symptoms for approximately 10 days. She continued to row through the preseason as symptoms would allow. At the beginning of the competitive spring season she was unable to practice or compete secondary to pain which was rated at 6/10 with aggravating activities. Despite compliance with supervised rehabilitation, three local injections, oral analgesics, topical analgesics, and relative rest, only mild and temporary relief of symptoms was attained. Interventions were all directed at the site of reported pain.

Reexamination

Reexamination of the patient occurred late March 2007 and was performed by a separate physical therapist in conjunction with the original treating clinician. Examination findings were relatively consistent with previous examinations. Additionally, adjacent structures were examined and included anterior, lateral, and posterior ribs, as well as the cervicothoracic spine. Hypomobility of the upper thoracic spine (T1–4) was noted with posterior to anterior glide of the spinous process. Unilateral posterior to anterior glide of the transverse process on the left side demonstrated more hypomobility than on the right. Hypomobility was also noted at the T3–4 costovertebral joints on the left side. Mobility of the lateral aspect of the rib cage was examined with passive accessory motion and restricted mobility of the third and fourth ribs was noted. Palpation of the left lateral third and fourth rib interspace revealed tenderness and spasm with inferior displacement of the third rib on the fourth. Posture was evaluated in standing with the patient demonstrating a forward head, rounded shoulder posture, with increased thoracic kyphosis and increased lumbar lordosis. Length of anterior chest and shoulder musculature was assessed and pectoralis major and minor tightness were noted. Pectoralis minor muscle fibrosis was also identified via palpation.

Clinical impression

Differential diagnosis included fracture (acute, stress), joint subluxation or displacement, rib contusion, muscle avulsion, and differentiation of referred pain from pleural or visceral structures, costochondritis, or Tietze's syndrome. Imaging included plain film X-rays which were negative for fracture or displacement of the sternum, ribs, or clavicle. Results of the bone scan indicated no focal region of abnormal uptake, thus negative for stress fracture. Rib contusion was not likely due to insidious onset and lack of specific trauma. Muscle avulsion of the pectoralis major or minor was not considered to be a likely diagnosis due to full pain-free upper extremity active range of motion and manual muscle testing which was 5/5. Referred pain from pleural or visceral structures was not considered likely due to the ability to reproduce pain with musculoskeletal movements.

A diagnosis of either costochondritis or Tietze's syndrome were considered as likely possibilities as both are associated with localized pain and tenderness over costosternal or costochondral joints. The diagnoses are usually clinical and imaging studies such as X-ray and bone scan are usually negative. Bone scan is not a specific test for costochondritis (sensitivity: 0.8; specificity: 0.0).[6] Costochondritis is described as a chronic condition that exists in the absence of swelling, heat, and erythema. Tietze's syndrome has a similar presentation, but is acute and signs include swelling, heat, and erythema. This case had aspects of both conditions. The chronic nature of the case, although in the presence of swelling, was thought to be in line with costochondritis. Although swelling was present, this was thought to be attributed to the severity and chronic irritation of the costosternal joint.

Intervention

Based on additional evaluation findings from the reexamination the intervention program was revised. We recommended that the patient continue to reduce tissue stress via relative rest and minimize self-manipulation of the affected joints. The revised rehabilitation plan was directed at addressing underlying impairments which were thought to contribute to anterior hypermobility of the costosternal joints. The program addressed muscle tightness, posterior

thoracic and rib hypomobility, specifically costovertebral and facet joints, and postural correction.

Interventions to address pectoralis minor fibrosis included continuous ultrasound followed by sustained pressure to the pectoralis minor muscle belly, five repetitions each held for 30 seconds. Lateral rib and posterior spinal hypomobility was addressed with joint mobilization/manipulation. Superior mobilization of the left lateral ribs was performed in sidelying in conjunction with respiration. Since we perceived the third rib was inferiorly displaced on the fourth rib a superior glide of the third rib was performed during exhalation. Upper thoracic extension mobilization was performed in supine. Grade IV mobilizations were performed, two sets of 15 repetitions, with a mobilization wedge. Regional upper thoracic Grade V mobilization was also performed in supine using previously described methods.[16] This manipulation was intended to target the upper thoracic region, as the technique is not likely to target one specific segment.[17] Therapeutic exercises included postural correction, cervical stabilization, and scapular stabilization exercises. Initial exercises included chin tucks on foam roller, two sets, 30 repetitions and prone shoulder horizontal abduction on a stability ball, two sets, 15 repetitions.

Outcome

Three weeks after the reexamination (April 2007) and revised rehabilitation program the patient reported the pain experienced throughout the day had subsided (VAS 0/10). With the exception of injections, the patient had not experienced this degree of pain relief since the initial onset of symptoms 1 year previous. She was able to resume running and elliptical aerobic training with minimal discomfort (VAS 2/10) and began to reintegrate into collegiate rowing. At this point there was approximately 1 month left in the rowing season. Unfortunately she was unable to completely return to collegiate rowing at a performance and fitness level which was consistent with other varsity rowers.

Discussion

Typically costochondritis is thought to be a self-limiting condition which spontaneously resolves with reassurance and relative rest.[2] For the general population these measures may provide adequate relief of symptoms. For elite athletes, reassurance, relative rest from activity, formal rehabilitation program, and medical interventions such as joint injection may not resolve symptoms to allow an athlete to resume/continue competitive training. Identification of the underlying causative factors and impairments relative to chronic costochondritis is necessary to appropriately manage this condition.

The rib and associated thoracic vertebral segment can be described as a segmented ring with mobile segments/articulations of the vertebrae, ribs, and sternum. Movement and stress applied at one portion can be transmitted through the entire ring. Thoracic flexion is coupled with posterior rib elevation and anterior rotation (internal torsion) which translates the anterior portion of the rib inferiorly.[13] Thoracic rotation causes the contralateral rib to anteriorly rotate (internal torsion) and the ipsilateral rib to posteriorly rotate (external torsion).[13] Lateral or side bending causes the lateral margin of the rib to approximate and the contralateral lateral margin to separate.[13]

The individual in this case report performed repetitive thoracic flexion, left rotation, and side bending associated with rowing starboard side. The osteokinematics associated with this movement pattern include anterior rib movement in an inferior direction coupled with anterior rotation (internal torsion) and lateral rib approximation.[13] This places additional stress on the anterior portion of the rib at the costosternal and costochondral joints which are attached to a relatively stable sternum.

It is important for clinicians to recognize structures distant to the site of pain may contribute to dysfunction. It is common to examine distal and proximal joints in the extremities, but examination of distant structures associated with rib pathology is not as commonly described in the literature.[9,10] This case report proposes a mechanistic rationale in which hypomobility and tightness of posterior spinal structures may place additional stresses at the anterior joints of the chest wall, which may manifest as hypermobility. Solely directing interventions at the site of pain and swelling may not fully abate symptoms related to costochondritis. Addressing hypomobility of posterior spinal structures and reinforcement of correction via postural reeducation and exercise can decrease loads placed on the joints of the anterior chest wall, which may have allowed for a more thorough recovery in this case study.

Clinical examination findings included slight anterior prominence of the left third and fourth costosternal joints, restricted mobility of the third and fourth ribs with inferior displacement of the third rib on the fourth, hypomobility of the T3–4 facet joints into extension, and hypomobility of the third and fourth costovertebral joints on the left side. These findings are logical due to the repetitive pattern of the rowing stroke performed thousands of times each practice. We hypothesized that as the posterior thoracic facet joints and costovertebral joints became hypomobile, the relative motion was regained anteriorly, via hypermobility, at the costosternal joint. Pain and swelling at the costosternal joints were potentially related to repetitive tissue stress. Reducing stress on anterior tissues by restoring posterior thoracic mobility provides plausible rationale for intervention outcomes.

The use upper thoracic mobilization and manipulation for the treatment of rib dysfunction is not a new concept. It has previously been described for the treatment of costochondritis in case report format as an adjunct therapy for a physiotherapist,[10] a photographic processor[10] and a volleyball player.[9] The first report[10] suggested the underlying cause was related to neurogenic inflammation whereas the other[9] briefly discussed the potential contribution of hypomobile costovertebral and costotransverse joints. Thoracic and rib joint mobilization has also been utilized in the treatment of rowers with rib stress fracture.[18,19] These reports also suggested that rib stress fracture symptoms may have been related to hypomobility of the thoracic spine and costovertebral joints. Based on the findings associated with this case report, as well as our clinic experience with the management of both costochondritis and rib stress fractures, we are in agreement with previous reports[3,9,10] that hypomobility of the thoracic spine and costovertebral joints may contribute to anterior thoracic cage pathology.

Limitations

Results of this case report should be approached with caution due to the nature the single subject design and limited reliability and validity of examination methods such as spinal joint mobility assessment.[20,21] The timing of symptom resolution during the last month of her fourth year of collegiate rowing also made it difficult to determine if symptoms would have remained diminished should the patient have returned to her previous level of training and competition. Since the patient graduated from the university and was not a national team candidate, she concluded her rowing career. Thus, we were also unable to determine if pain would have returned upon resuming rowing during the following fall season. Finally, a causal relationship between interventions and symptom abatement cannot be made. It is possible that symptoms decreased as a result of natural progression of costochondritis.[9,10] Further research is necessary to better determine the relationship between the thoracic spine and posterior and anterior rib articulations.

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