Sternoclavicular septic joint arthritis is a relatively rare infection. Clinical recognition may be hampered by minor signs and symptoms and the tendency of sternoclavicular joint pain to be referred to the shoulder. In advanced cases, sternoclavicular joint sepsis can progress to descending mediastinitis. Prompt recognition and treatment is critical given the potentially life-threatening consequences of this infection.

This article presents an elderly woman with a neck and retrosternal abscess secondary to a septic sternoclavicular joint. Her symptoms were originally attributed to a torn rotator cuff. This article demonstrates the varied presentation of sternoclavicular joint pathology in the evaluation of shoulder pain and presents one scenario in which misdiagnosis could have had fatal consequences.

**CASE REPORT**

A 67-year-old woman presented with left shoulder pain, with symptom onset 5 days prior to presentation. The pain was initially responsive to rest, heat, and non-steroidal anti-inflammatory medications, but progressed to constant, throbbing pain. The pain radiated into the left arm and jaw. No history of shoulder trauma was reported, however the patient recalled carrying heavy groceries with her left arm on the day prior to symptom onset. Medical history was significant for diabetes mellitus, end-stage renal disease, mild degenerative cervical spine disease, and depression.

Physical examination revealed an obese, afebrile patient with normal vital signs. The left shoulder and sternoclavicular joint were nonerythematous without increased warmth or palpable masses. Diffuse tenderness to palpation was noted throughout the left shoulder, supraclavicular area, neck, and arm. The patient refused to actively move the left upper extremity. Passive shoulder motion was decreased in all directions, and marked weakness with resisted external rotation and abduction was noted. Hawkins and Neer impingement maneuvers could not be adequately assessed secondary to discomfort. Sensory and vascular examinations were normal.

Significant laboratory studies were white blood cell count 16,100 cells/mm³, and erythrocyte sedimentation rate 136 mm/h. A tuberculosis skin test was negative. A chest radiograph and urinalysis did not reveal the source of infection.

Plain radiographs of the left shoulder and clavicle were unremarkable. Magnetic resonance imaging (MRI) of the cervical spine revealed only degenerative changes. A shoulder MRI revealed degenerative changes of the acromioclavicular joint and a complete tear of the supraspinatus tendon without retraction (Figure 1). Fluid accumulation was noted in the subacromial and subdeltoid bursae and biceps tendon sheath.

The patient was admitted to the general ward. MRI showing full-thickness tear of the supraspinatus tendon (arrow).

**Figure 1**

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By the third day after admission, a subtle soft-tissue prominence became apparent over the length of the left clavicle and into the left supraclavicular fossa. Subsequent ultrasound revealed a hypovascular intramuscular soft-tissue mass in the left neck. Non-contrast computed tomography (CT) revealed a 2.0 x 2.7-cm soft-tissue mass within the left strap muscles of the neck, which extended inferiorly to involve the sternoclavicular joint and the periosteum of the clavicle. An enlarged 2.0 x 2.4-cm lymph node was also noted on the left side of the neck. Magnetic resonance imaging revealed left sternoclavicular joint involvement as well as several ribs and a fluid collection that descended posterior to the sternum to the pulmonary artery (Figure 2). Fine needle fluid aspiration collection yielded purulent material with no atypical cells. An incision and drainage of the same abscess liberated 4 cc of frank pus and was accompanied by moderate symptomatic relief.

Biopsy and blood cultures were positive for methicillin-sensitive Staphylococcus aureus. A 6-week course of intravenous oxacillin was started. The patients’ symptoms had significantly reduced 2 months after completing the course of antibiotics, although some weakness in the left arm with overhead activities was noted.

**DISCUSSION**

Sternoclavicular joint infection is uncommon and accounts for <10% of cases of septic arthritis. Predisposing factors include intravenous drug use, rheumatoid arthritis, liver disease, alcohol abuse, diabetes mellitus, renal failure, malignancy, steroid therapy, or infection at another site. Direct spread from a subclavian central venous catheter has also been documented, and reports exist of infection in previously healthy hosts.

*Staphylococcus aureus* appears to be the most common pathogen, although infections have been caused by numerous organisms including tuberculosis. Sternoclavicular joint infection leads to abscess formation in as many as 20% of patients. Few signs or symptoms are often present to suggest mediastinal spread, but mediastinitis can be fatal with septic shock as a major cause of death.

As this case demonstrates, sternoclavicular joint septic arthritis can be a difficult clinical problem. Septic sternoclavicular joint does not have a uniform presentation and can initially be mistaken for a glenohumeral process. Pain due to sternoclavicular joint disease may be insidious in onset and referred to various ipsilateral anatomical locations, including the shoulder, neck, jaw, or elbow, and can easily be confused with intrinsic shoulder or cervical spine pathologies. The pattern of pain has also been reported to mimic myocardial ischemia and lung disease. Swelling and erythema over the sternoclavicular joint may be absent, and fever and leukocytosis are not always present. In a patient with renal disease, an isolated elevated erythrocyte sedimentation rate is also not specific, as 20% of patients with end-stage renal disease will have extreme elevations to >100 mm/h.

The pathogenesis of sternoclavicular joint septic arthritis may be variable. Direct injury, bacteremia, and direct spread from the subclavian vein have all

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*Figure 2: MRI showing soft-tissue swelling around the left clavicular head (arrow) (A). Further caudally, an abscess was revealed (arrow) medial to the left jugular vein and lateral to the left thyroid lobe (B).*
been postulated as potential mechanisms for initial seeding. Wohlgethan et al speculated that the dense fibrous joint capsule is not easily ruptured and spreading is through lymphatic dissemination. However, Chen et al believed that infection spread through a ruptured capsule; superior capsular rupture would lead to erythema, swelling, and a neck abscess, whereas inferior rupture would lead to retrosternal spread indicated only by vague anterior chest pain. However, this case demonstrates both neck and retrosternal involvement with lymphadenopathy and no erythema on examination.

The treatment of sternoclavicular infection is controversial. Most early cases appear to respond well to conservative measures; however, development of osteomyelitis or the formation of a retrosternal abscess are generally considered indications for surgery. Successful resection of the manubrium and medial clavicle with wound healing by secondary intent have been documented. Aggressive en bloc resection of the joint and surrounding bone and infected soft tissues with muscle flap coverage of the defect also appears to be well-tolerated with remarkably little disability. In this case, initial aggressive surgical treatment was contraindicated given the patient’s comorbidities, and the patient responded favorably to medical therapy.

To our knowledge, no other report exists in the literature of a patient with a concomitant rotator cuff tear and sternoclavicular infection. Although no causative or etiological connection exists between the two pathologies, this case demonstrates that concurrent intrinsic shoulder disease may delay the diagnosis of a serious sternoclavicular joint infection.

**REFERENCES**


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**Case Report**

Septic sternoclavicular joint does not have a uniform presentation and can initially be mistaken for a glenohumeral process.