Introduction

Sternoclavicular joint infections (SCJI) constitute less than 1% of all joint infections. Infections of this joint have clinical significance for physicians of all specialties particularly primary care, emergency medicine, infectious disease, thoracic and orthopedic surgeons (1-4). In the primary care setting, it can present as a rash, while in the Emergency Room, it can present as chest pain radiating down the arm (3,4). Due to the ambiguity of presentation and low prevalence, the diagnosis of SCJI is often delayed. This causes infection to spread to the nearby tissues. So, it is important to understand the etiology, pathophysiology and treatment of SCJI. There are currently no standardized diagnostic and therapeutic algorithms for SCJI as defined in literature (1). We present the following article in accordance...
with the Narrative Review reporting checklist (available at http://dx.doi.org/10.21037/jtd-20-761).

The sternoclavicular joint (SCJ) lies in proximity to important structures such as the subclavian vessels and the phrenic nerve. Infections of this structure should be taken seriously and addressed immediately to prevent extension and damage to the valuable nearby structures. Treatment options for SCJI include IV antibiotics, incision and drainage, surgical debridement and/or en bloc resection depending on the severity and extension of the infection. Surgical resection with or without muscle flaps is often preferred over medical management for osteomyelitis of SCJI. In this paper, we explore the existing literature to understand the knowledge on the diagnosis and treatment methods of SCJI.

**Methods**

We searched English publications in PubMed with the phrases “sternoclavicular joint”, “sternoclavicular joint infections”, “septic arthritis of the sternoclavicular joint”, “osteomyelitis of sternoclavicular joint” and “surgical management of sternoclavicular joint infection”. We included clinical trials, case reports, case series, retrospective cohort studies, literature and systematic reviews. We excluded papers with non-infectious etiology of SCJ pathologies from this review. Our search yielded 346 papers. We included 95 papers between 2000 and 2019 in our literature review. In this paper, we will focus on the infectious pathologies of SCJ. Two types of infections need to be considered while evaluating SCJ infections: septic arthritis and osteomyelitis. Septic arthritis involves the direct invasion of the joint space by pathogens (5). Osteomyelitis involves infection of the bone that can occur due to hematogenous seeding or direct extension into the joint space (6). Septic arthritis can often convert itself to osteomyelitis due to delay in diagnosis or inadequate initial treatment.

**Discussion**

**Anatomy**

The sternoclavicular joint is a saddle type, diarthrodial or synovial joint that functions as the only articulation between the upper extremity and axial skeleton (7). It comprises of the lateral notch of the manubrium, the medio-inferior head of the clavicle, and the costal-cartilage of the first rib. These joints are covered by fibrocartilages on the outside and synovial membranes on the inside. There are four main ligaments that stabilizes the sternoclavicular joint namely, anterior and posterior sternoclavicular ligaments, interclavicular ligament and costoclavicular ligament. The anterior sternoclavicular ligament originates from the anteromedial surface of the first rib and inserts superiorly and laterally on the clavicle. The posterior sternoclavicular ligament originates lateral to the anterior fibers and inserts superiorly and medially on the clavicle. The interclavicular ligament joins the superomedial aspect of the clavicle to the upper sternum. This protects against inferior displacement of the medial clavicle. The costoclavicular or rhomboid ligament, consisting of two fascicles, joins the clavicle with the first rib. This provides stability to the sternoclavicular joint during elevation and rotation of the clavicle (7,8).

Blood supply to this joint comes from the internal thoracic artery and suprascapular artery. The medial suprACLavicular nerve (C3 and C4) and the nerve to the subclavius (C5 and C6) innervate this joint. The clavicular head of the pectoralis major, sternocleidomastoid and sternohyoid muscles attach to the medial clavicle anteriorly, posteriorly and inferiorly, respectively.

The bony articulation between the medial end of the clavicle and the manubrium is <50% of the surface area, making the joint potentially unstable (9). The stability is maintained by the anterior and posterior capsule. The anterior capsule prevents anterior subluxation, while the posterior capsule prevents both anterior and posterior subluxation of the SCJ. The sternoclavicular joint can glide up to 30 degrees antero-posteriorly, pivot up to 30 degrees supero-inferiorly and rotate up to 45 degrees axially.

**Pathology**

The SCJ is lined by fibrocartilage, not hyaline cartilage. The presence of this bulky central disc may predispose this joint to have the same susceptibility to infections as amphiarthrodial joints like the pubic symphysis and sacroiliac joint. These joints have limited mobility and are without a synovial lining (10). The joint capsule is unable to distend freely, which creates high intra-articular pressures. This facilitates dissemination of infections, possibly through lymphatics, into adjacent tissues (11).

The SCJ lies in proximity to important vasculature, nerves and organs in the thoracic cavity such as the great vessels of the superior mediastinum, trachea, esophagus,
vagus nerve and phrenic nerve. Hence, pathology of this joint should be managed carefully and promptly to prevent spread to these nearby structures (12). Pathologies of this joint include injury, infections, malignancy, rheumatoid arthritis, SAPHO (syndrome of synovitis, acne, pustulosis, hyperostosis and osteitis) (13), Tietze’s syndrome (14), Lemierre syndrome (15) and Friedrich’s disease (16). SCJI can often be mistaken with any of these other pathologies.

Medical history and physical exam

A detailed medical history is crucial in evaluating SCJ infections. Since SCJI frequently affects patients with pre-existing systemic diseases, an inquiry about arthritic diseases, diabetes, recent trauma, infections and hospitalizations is paramount. SCJI are mostly unilateral (95%), with the majority occurring on the right side (60%) (17). This difference in laterality becomes less apparent in IV drug users (18). Unilateral SCJI can often present with fever, joint swelling, warmth and immobility. Bilateral infections can present as a butterfly rash on the chest (19). Some unusual presentations have also been reported in literature, such as a pressure like chest pain radiating to the neck or shoulder. This pattern of pain can mimic a myocardial Infarction. Other unusual presentations include vocal cord palsy (20), dysphagia (21), cervical esophageal rupture (22), lower respiratory infection (23), breast abscess (24) and subclavian compression syndrome (25).

It is very rare that SCJI get diagnosed at the first visit. Diagnosis is often delayed, as the initial symptoms can be quite subtle (26). Reasons for the delay in diagnosis can be attributed to ambiguity in presentation, slow progression, and prior treatment with steroids (27). Slow progression can be attributed to the surrounding ligaments that reinforce the SCJ creating a relatively non-distensible synovial space. This makes large effusions extremely rare. The mean duration of symptoms for SCJI is approximately 14 days (28).

Risk factors

There are several risk factors for SCJ infections. The most common factors are an immunocompromised state, diabetes mellitus, end stage renal disease (29), intravenous drug use, trauma (30,31), presence of a central venous catheter (32), rheumatoid arthritis and intra-articular injections. Other risk factors include suprapubic catheter (33), crystalline arthropathies, radiation, cirrhosis, joint surgery, skin infections, malignancy (34), chemotherapy, breast cancer and radiation (35), tracheostomy (36), epidural block (37) and coronary angiography (38). However, 23% of cases are not associated with any predisposing factor (39).

SCJI can spread either hematogenously or from direct extension to contiguous structures. Sixty-two percent of patients with SCJI have bacteremia (40). It is theorized that immunocompetent individuals can clear transient bacteremia, but immunocompromised individuals fail to do so effectively. Transient bacteremia can lead to seeding of the SCJ and ultimately cause osteomyelitis of the joint (41,42). There are three stages for this process: invasion (days), suppuration (weeks), and possible sepsis (43). Contiguous spread, on the other hand, can occur from the joint directly to the skin, lung or other nearby structures. Delayed presentations will result in more extensive direct spread at the time of diagnosis.

Laboratory data

Laboratory workup includes complete blood count, erythrocyte sedimentation rate, c-reactive protein, rheumatoid factor, antinuclear antibody and human leukocyte antigen B27. Mean values, ranges and sensitivities

<table>
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<tr>
<th>Table 1 Range of lab values</th>
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<tr>
<td><strong>Range</strong></td>
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<tr>
<td>WBC (cells/µL)</td>
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<tr>
<td>ESR (mm/h)</td>
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<td>CRP (mg/dL)</td>
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WBC, white blood cell; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein.
of each lab test are reported in Table 1.

**Imaging**

Diagnostic workup includes imaging like x-ray, computed tomography and magnetic resonance imaging if more soft tissue detail is needed. Only 20% of cases of osteomyelitis have abnormal radiographic findings by the second week of symptoms. Inflammatory factors associated with infections accumulate in the joint space increasing the diameter of the joint space. This increased diameter is in contrast to typical osteoarthritis and joint degeneration, in which the joint space is narrowed due to loss of cushioning between bones. If the average joint distention is 14 mm (range, 10–20 mm), it is more likely to be infection. If the distension is 5 mm (range, 3–8 mm), it is more consistent with degeneration (44).

A chest X-ray is usually the first step in diagnosis. It may show joint space distension and soft tissue shadow widening. It usually takes 10–12 days for soft tissue swelling to be evident on a typical X-ray. Demineralization and bony destruction tend to appear much later.

Ultrasound (US) is another diagnostic modality that can be used and is a safe diagnostic test to use during pregnancy (45). Infection is less likely if the distance of the joint capsule from the bone is less than 3 mm (46). However, the sensitivity for US is low (47). US-guided aspiration can also be performed if an obvious fluid collection is seen (48).

Computed tomography (CT) scan has an 83% sensitivity in diagnosing SCJI. CT-guided aspirations are safe and have yielded positive cultures in more than 50% of cases (49). CT can further elucidate the extent of anatomical involvement but cannot show soft tissue involvement.

Magnetic resonance imaging (MRI) is almost 100% sensitive but has low specificity. It yields a faster diagnosis than other imaging modalities. The earliest changes can be seen on MRI within 1–2 days, whereas, other modalities rarely show changes before 10 days. MRI can show capsular distension (>5 mm), extracapsular fluid collection, periarticular muscle edema and bone erosion. Osteomyelitis is seen as a hypointense signal on T1-weighted images and hyperintense signal on T2-weighted images. Even though MRI is sensitive in diagnosing SCJI, it is usually not obtained in the initial work up period. On MRI appearance SCJI can be misinterpreted as a bony metastasis (50).

A triple phase technetium bone scan, positron emission tomography (PET) scan and skeletal scintigraphy are other modalities which are rarely needed to diagnose SCJI. A technetium bone scan can show radiologic changes within hours of onset of infection. The first two phases show hyperemia. PET scan shows increased fluorodeoxyglucose uptake (51). Skeletal scintigraphy may show increased radiotracer uptake in the sternum and sternoclavicular regions.

**Infectious etiology**

The most common infectious organism that causes SCJI in the general population is *Staphylococcus aureus*. Blood cultures in SCJI tend to be positive in 65% of cases. *S. aureus* is found in 44–67% of all septic arthritis cases and in 1.3% of the patients with tuberculosis (52). Endocarditis complicated by Staphylococcus pneumonia can also cause SCJI (53).

*Streptococcus pneumoniae* and *Streptococcus pyogenes* are other common causes of SCJI (54). These throat and lung infections can lead to lung abscesses which can spread to the SCJ in both immunocompetent and immunocompromised patients (55). These infections tend to be severe in immunocompromised patients and post-surgical patients complicating the course of treatment (56). Other species of *Streptococcus* such as *Streptococcus viridans* or *Streptococcus sanguinis* have been reported to cause SCJI after mis-placed tracheostomy tube in the pre-tracheal space in a patient with laryngeal cancer (57,58). Two reports of *Streptococcus agalactiae* have been reported in previous literature as well. One of the reports detailed a patient with uncontrolled diabetes, while the other report highlighted a superinfection after *Mycobacterium tuberculosis* (MTB) empyema (59,60). *Haemophilus*, *Aggregatibacter*, *Cardiobacterium*, *Eikenella* and *Kingella* (HACEK) organisms from the oral cavity have also been known to colonize the SCJ even in children (61,62).

SCJI with *Mycobacterium tuberculosis* is very rare (<5%) (63). *Mycobacterium tuberculosis* can spread both hematogenously and contiguously from an apical pulmonary tuberculous focus (64). An unusual presentation of *Mycobacterium tuberculosis* SCJI was reported in a patient after malaria infection (65,66).

*Mycobacterium kansasi* is another uncommon aquatic organism known to cause SCJI primarily in immunocompromised individuals. One fatal case was reported in a Caucasian female with Waardenburg syndrome complicated by diabetes mellitus, a learning disability, and 15 previous surgeries for deformities of her foot (67).

*Pseudomonas aeruginosa* is a gram negative organism responsible for 10% of SCJI in intravenous drug users,
sickle cell disease and thalassemia (68). *Escherichia coli* is responsible for about 5% of SCJI. An unusual case of *Escherichia coli* was reported after drainage of a Bartholin gland cyst (69). *Brucella*, *Pasteurella* and *Coxiella* are additional causes of SCJI in populations that work with animals (70,71). *Salmonella typhi* is known to cause SCJI in sickle cell patients (72). Other species of *Salmonella* like *Salmonella enterica* serotype D can also cause SCJI (73).

Another source of SCJI is disseminated gonorrhea. There is increased risk for such infections in pregnancy due to an immunocompromised status (74). *Propionibacterium* and *Corynebacterium* are also other unusual organisms to cause SCJI (75,76). *Propionibacterium* can form biofilms on the articular joint. These biofilms delay the clearing of infections and may result in the need for multiple surgeries to debride the joint. *Candida* is known to be a fungal source of SCJI in HIV patients. Since SCJI can be caused by a myriad of pathogens, cultures of the pathogens are necessary for targeted antibiotics treatment of SCJI.

**Medical management**

Medical management is the first step in addressing SCJI. Soft tissue infections such as thrombophlebitis, chest wall cellulitis, myositis and septic arthritis respond well to appropriate medical therapy alone (77). IV antibiotics are considered first line. The type of antibiotic is chosen based on the suspected organism and route of infection of the joint. A broad-spectrum antibiotic is usually utilized as the initial treatment, and then replaced by a narrower choice depending on culture and susceptibility results. The most common antibiotics used are cephalosporins, vancomycin and fluoroquinolones (78). Intravenous antibiotics can be switched to oral ones either on discharge or as deemed appropriate by treating physicians. Symptoms usually resolve within weeks to months. Patients should be followed for an extended period of time to ensure complete resolution of infection (79). Antibiotic duration varies based on the severity of infection.

**Surgery**

Surgical management is preferred for extensive SCJI with bony involvement, abscesses, and periarticular fluid collection (80). *En bloc* resection is the preferred surgical procedure and has a much better chance of resolving the infection compared to simple debridement or piecemeal resection (81). Once the resection is performed, a wound vacuum dressing can be placed for weeks to facilitate granulation tissue formation and allow the wound edges to contract. Hyperbaric oxygen therapy is another option for delayed closure (82).

Placement of calcium sulfate beads impregnated with antibiotics has been used with varying success. Vancomycin and gentamicin loaded calcium sulfate beads are used to fill the defect. The beads get completely resorbed within weeks without increasing serum calcium levels (83,84).

When a large defect remains, various options exist for muscle flap reconstruction (85). The pectoralis major can be used and receives its neurovascular supply from the thoraco-acromial bundle (86). Latissimus dorsi flaps are also an option. Use of muscle flaps are associated with a higher need for blood transfusion and longer hospital stays (10.5 vs. 5.5 days) however (87-89).

Postoperative length of stay can be quite long, ranging between 5 and 40 days depending on the type and extent of surgery (90). Postoperative risks include empyema, pleural effusion, abscess, mediastinitis and sepsis (91-93). Sepsis is another life-threatening complication of SCJI (94,95). Mortality rate from SCJI sepsis ranges roughly from 8% to 15%.

**Summary**

SCJIs are rare but serious joint infections secondary to hematogenous or contiguous spread from a nearby source. SCJIs can occur after trauma, central venous catheter placement or intravenous drug use. Early detection and intervention are the goals of treatment of SCJIs. Patients presenting with symptoms such as fever, rash, tenderness to palpation on the joint should be evaluated for SCJI by obtaining appropriate imaging studies. Surgical intervention should be taken early in the course if antibiotic management is not effective. Patients should be monitored for postoperative developments of mediastinitis and sepsis.

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References