Chronic Infections of the Chest Wall

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KEYWORDS

• Infection • Chest wall • Osteomyelitis • Sternoclavicular joint • Tuberculosis • Granuloma

KEY POINTS

- Chronic chest wall infections may occur in soft tissue, cartilage, and bone. Infectious pathogens may be bacterial, fungal, or parasitic.
- They may present as a discrete mass and can initially be mistaken for a neoplasm, a superficial infection, or a draining sinus.
- Effective management of chronic chest wall infections ranges from antibiotic administration to wide surgical resection of all devitalized tissue and subsequent coverage with well-vascularized soft tissue.
- Secondary sternal osteomyelitis is associated with complications of median sternotomy.
- Infectious seeding of the sternoclavicular joint is typically via hematogenous route.

INTRODUCTION

Chronic chest wall infections are rare, and those necessitating surgical consultation are even more uncommon. They result from direct inoculation of the chest wall or from contiguous or hematogenous spread from infected tissue, or from previous chest wall trauma or instrumentation. Chronic chest wall infections may occur in soft tissue, cartilage, and bone. Infectious pathogens may be bacterial, fungal, or parasitic. The diagnosis of chronic chest wall infections is difficult because of subtle, nonspecific signs, symptoms, and presentations, and because of their clinical rarity. They may present as localized chest wall pain, a discrete mass initially mistaken for neoplasm, a superficial infection, or a draining sinus. Effective management of chest wall infections ranges from antimicrobial administration to wide surgical resection of all devitalized tissue and subsequent coverage with well-vascularized soft tissue. Treatment depends on the type, magnitude, and location of the infection. Prompt timing in the diagnosis and treatment is important to minimize associated morbidities. This article presents a systematic review of chronic chest wall infections, and their associated work-up and management.

Chronic chest wall infections are typically nonnecrotizing and associated with lower morbidity than their more acute and necrotizing counterparts. They may often respond to nonsurgical management, but knowledge of their presentation and management is important to thoracic surgeons. Because of the chronic nature of the infection and subsequent prolonged inflammation, wound healing associated with chronic chest wall infections may result in differing degrees of fibrosis, aiding in the structural integrity of the chest wall if surgical therapy is needed. This is in contrast to their acute necrotizing infection counterparts, where the rapid onset of the infection does not allow for underlying fibrosis. If radical surgical debridement is required for chronic chest wall infection management, blood supply and

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resulting wound healing can be compromised as a result of the fibrosis. Common and rare chronic chest wall infections are discussed next.

CARTILAGE AND BONE INFECTIONS

Costochondral Infection

Infections of the costal cartilage, spanning the boney rib to the sternum, occur because of inoculation of the tissue by bacteria, or less commonly fungi. Most costochondral infections result from surgical intervention. However, costochondral infections may occur subsequent to penetrating trauma or via hematogenous spread from other sources of infection within the body. Most iatrogenic costochondral infections follow median sternotomy for cardiac surgery. In addition, thoracotomy and tube thoracostomy have been identified as inciting surgical trauma resulting in costochondral infections. Infection of the xiphoid cartilage may result in spread of infection bilaterally to the chest wall and costal cartilages.

Costochondral infections typically present as progressive discomfort, or pain of the anterior chest wall over time accompanied by localized tenderness on palpation, low-grade fever, leukocytosis, overlying erythema, and rarely, a draining sinus. In situations where the patient has undergone prior surgery or trauma to the area, nonunion of the involved ribs may result in instability beyond the expected time required for routine surgical healing. Wounds healing via secondary intention may result in a narrow, granulating wound with the underlying infected cartilage persistently exposed. As the overlying soft tissue and skin granulates with time, the chronically infected underlying cartilage serves as a nidus for a draining sinus.

Diagnosis of costochondral infection is made through a combination of clinical suspicion caused by a compelling history, prolonged duration of symptoms, physical examination, and imaging to guide local therapy. On interview with the patient, one can often elicit a history of discreet chest wall trauma, such as surgery. This is often followed by a prodromal period of chest wall pain on exertion, progressing to pain with minimal activity and respiration. Accompanying fevers, if any, are often low grade. Physical examination demonstrates tenderness over the chest wall adjacent to the nearby infection. Laboratory work-up may be notable for mildly elevated leukocytosis and elevated erythrocyte sedimentation rate and C-reactive protein. MRI is the imaging modality of choice because it may demonstrate tissue edema not well visualized on computed tomography (CT) scan.

Treatment of costochondral infection is a combination of the use of targeted antimicrobials and surgical resection of the infected connective tissue. Given the difficulty in identification of causative pathogens of most costochondral infections, broad-spectrum antibiotics targeted at gram-positive bacteria (eg, *Staphylococcus aureus* and *Staphylococcus epidermidis*) should be instituted. Surgery is indicated when symptoms persist, or findings on physical examination or radiographic imaging are compelling of infection refractory to antibiotics alone. Surgical treatment involves resection of the infected cartilage and surrounding infected soft tissue. Because the seventh through tenth costal cartilages are contiguous, infection involving any part of this costal cartilage typically requires resection of the entire costal arch unilaterally to achieve control. Samples of infected tissue should be sent for Gram stain and culture at the time of debridement.

Management of chest wall defects resulting from wide resection of costochondral infection depends on the extent of the defect. In the authors' experience, most defects resulting from treatment of costochondral infections are not full thickness with most of the skin, intercostal muscle, and perichondrium preserved. The resulting defects are managed with local soft tissue transposition, wet-to-dry dressings, or negative-pressure dressings with ongoing antibiotic administration.

Tubercular abscesses

Classically, abscesses are thought to occur in acute spectrum of infections. Additionally, extrapulmonary tubercular infections account for a minority of tuberculosis infections. Tuberculosis infections of the chest wall account for only 10% of extrapulmonary tuberculosis infections and are typically chronic infections.[1] However, a resurgence of tuberculosis in recent decades, largely as a result of proliferation of immunosuppressive conditions combined with immigration of people from developing nations to North America, has resulted in this being a more common entity of chronic chest wall infections.[2]

Tubercular abscesses of the chest wall have a strong predilection to the sternal margins. However, they may additionally involve ribs, costochondral junctions, costovertebral joints, and the vertebrae. Predilection to the sternal margin has been suggested to be the result from internal mammary lymph node infections secondary to primary pulmonary involvement. The infected lymph nodes then caseate, erode through the chest wall, and result in visible "swelling." The subpleural collections of this caseating material from the infected, necrosed lymph nodes are known