

## Anterior Chest Wall Syndrome in Stick and Ball Sports. What's in a Name?

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**Received:** January 02, 2026; **Published:** February 02, 2026

### Abstract

Anterior chest wall syndrome (CWS) is a frequently encountered cause of chest pain in both acute and non-acute medical settings. In the acute context, clinical priorities appropriately focus on excluding potentially life-threatening cardiovascular and pulmonary conditions, particularly acute coronary syndromes. Once these diagnoses are excluded, alternative causes of anterior chest pain must be considered.

Musculoskeletal disorders represent the most common non-cardiac cause of anterior chest wall pain. Less frequent non-musculoskeletal causes, such as gastro-oesophageal reflux disease, may also contribute but are comparatively uncommon. Pathology affecting the sternum and costochondral articulations is a recognised source of symptoms, and a range of nomenclature has historically been applied to these conditions. Tietze syndrome, first described over a century ago, represents a distinct clinical entity within this spectrum, typically involving unilateral pain and tenderness at the second and third costochondral junctions. In sporting populations, CWS is particularly prevalent, especially in bat- and ball-based sports where repetitive upper-limb, trunk rotation, and rib-cage loading occur. The condition frequently follows a relapsing course, attributed to ongoing mechanical stress and hypersensitisation of musculoskeletal and neural structures.

The diagnosis of musculoskeletal anterior chest wall syndrome is primarily clinical, based on history and physical examination. Radiological investigations frequently fail to identify the underlying pathology and are mainly useful in excluding alternative diagnoses.

Although generally benign and self-limiting, accurate recognition of ACWS is essential to prevent unnecessary investigations and to guide appropriate activity modification, particularly in athletic individuals.

**Keywords:** *Anterior Chest Wall Syndrome; Tietze Syndrome; Costochondritis; Bat and Ball Sports; Tennis; Activity Modification; Neural Sensitisation*

### Introduction

Chest pain is a frequent complaint in medical practice, in the emergency and non-acute settings. The causes are diverse, including a broad spectrum from life threatening cardiovascular diseases, such as myocardial infarction to benign causes such as Anterior Chest Wall Syndrome [CWS]. This painful condition of the anterior chest wall can be caused by a musculoskeletal disorder (MSK) and associated with tenderness of the chest wall. However, it is not a benign condition and can cause great morbidity to sufferer impacting on daily activities, a cause of emotional distress and anxiety [1]. In the emergency department, an estimated 20 - 50% of non-cardiac chest pain is due to a musculoskeletal cause [2]. Gastroesophageal reflux and hiatus hernia can also be the cause of chest pain symptoms which are non-cardiac and non MSK (See table 1).

<p><b>Common causes of non-cardiac chest pain (NCCP)</b></p> <ul style="list-style-type: none"> <li>• Musculoskeletal (20-50%): Tietze syndrome, costochondritis, chest wall pain.</li> <li>• Gastrointestinal (up to 50% of NCCP cases): GERD, <u>oesophageal spasms</u>.</li> <li>• Neurology, intercostal neuropathy</li> <li>• Psychiatric: Anxiety/panic disorders.</li> <li>• Pulmonary: Pleurisy, pneumonia, pulmonary embolism.</li> <li>• Cardiac: Ruled out first, but stable angina/coronary vascular disease must be excluded.</li> </ul>
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**Table 1:** Common causes of non-cardiac chest pain (NCCP).

Despite musculoskeletal conditions such as CWS being a common reason for visits to the emergency room. Establishing their aetiology can be a secondary issue as other pathologies such as angina pectoris, pleurisy, and other serious cardiopulmonary conditions are out ruled, due to similar presentation. In the main the emergency room will in the first instance out rule potentially serious conditions such as cardiopulmonary disease. Once these emergency conditions are ruled out the specifics and aetiology of the chest pain is often left unresolved.

Non-cardiac chest pain is common with Musculoskeletal chest pain frequently observed and has a variety of causes of CWS: Tietze Syndrome, Costochondritis, rib fractures, inter costal neuropathy and diseases of the sternum are all encountered (See table 2). Clinical assessment is the corner stone in diagnosis to identify underlying MSK diseases after an acute coronary syndrome or similar, medical emergency conditions have been ruled out. In many instances of CWS diagnostic tests, such as radiology fail to reveal the cause of the Musculoskeletal pathology [2].

<ul style="list-style-type: none"> <li>• Tietze syndrome: is an inflammatory condition that is characterized by localized swelling, which can be warm and red, typically affecting a single costal cartilage (most often the second or third rib). It is often associated with sporting activity.</li> <li>• Costochondritis: The most common cause, involving inflammation of the costochondral junctions, but without visible swelling. It usually affects multiple joints, most often the second to fifth costochondral junction. It can be associated with Cardiothoracic surgery.</li> <li>• Sternalis syndrome: Pain originating from an accessory sternalis muscle, often exercise-induced, which responds well to a specific type of injection.</li> <li>• SAPHO syndrome: A rare, chronic inflammatory disorder with both bone (osteitis, hyperostosis) and skin manifestations, heavily involving the anterior chest wall, especially the sternoclavicular region.</li> <li>• Intercostal neuralgia: neuropathic pain along the ribs, chest, or upper abdomen caused by inflammation/injury to intercostal nerves.</li> </ul>
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**Table 2:** Anterior chest wall syndrome (CWS) MSK types are differentiated primarily by the presence or absence of swelling.

Therefore the cornerstone of diagnosing the musculoskeletal condition is the history and clinical examination. Anterior chest wall and shoulder girdle stress is commonly observed in stick-and- ball sports requiring repetitive trunk rotation and upper limb acceleration. Such as Tennis, Baseball, Cricket and Hurling [3,4].

**CWS nomenclature**

Nomenclature for describing CWS is varied and there are various words used to describe the condition of CWS or non-cardiac chest pain. Shakespeare’s famous line from Romeo and Juliet comes to mind. “What’s in a name? That which we call a rose/By any other name would smell as sweet”, The nomenclature is simply a description of the clinical entity. Musculoskeletal related or simply of non-cardiac aetiology. The different labels don’t define the true essence, but rather an indication of pathology. As Juliet argues Romeo’s Montague surname shouldn’t matter because he is the same person, regardless of his family’s name. Similarly various names are used to describe a pain in the chest of non-cardiac origin. CWS can be described as one entity-the chest wall syndrome-but many doctors will use selective terminology depending on points of maximum tenderness on the chest wall and the presence of absence of swelling. Tietze syndrome and costochondritis are the most encountered MSK causes of CWS. Both affect the costochondral junction, but have subtle aerological and clinical findings.

**Tietze syndrome and costochondritis**

Tietze syndrome is a nonsuppurative inflammatory condition characterized by chest pain and swelling at the costochondral junction [5]. Tietze syndrome is a painful but benign condition characterized by localized swelling, most commonly unilaterally at the upper costosternal or costochondral joints. However, the sternoclavicular joint can also be involved.

Tietze syndrome can be confused and conflated with costochondritis. Tietze syndrome is differentiated from costochondritis by swelling of the costal cartilages, which does not appear in costochondritis. Additionally, costochondritis affects the 2<sup>nd</sup> to 5<sup>th</sup> ribs while Tietze syndrome typically affects the 2<sup>nd</sup> or 3<sup>rd</sup> rib (See table 3). The pain and swelling associated with Tietze syndrome is typically chronic and intermittent and can last from a few days to several weeks [6]. Tietze syndrome is commonly associated with a sporting population and particularly bat and ball athletes, where Costochondritis is associated with a variety of situations such as respiratory conditions and surgery.

Although frequently conflated, the two conditions have important differences.

**Similarities**

- Both cause anterior chest wall pain
- Both may follow coughing, physical strain, or minor trauma
- Both commonly affect physically active individuals or athletes

**Key Differences**

Feature	Tietze’s Syndrome	Costochondritis
Incidence	Rare	Common
Typical Age Group	All ages	All ages
Number of Sites	Usually one or two levels	Multiple levels (2 <sup>nd</sup> - 6 <sup>th</sup> ribs)
Location	Predominantly 2 <sup>nd</sup> - 3 <sup>rd</sup> costochondral junction Unilateral to Tietze bilateral to costochondritis aetiology	2 <sup>nd</sup> - 6 <sup>th</sup> ribs, wider distribution
Swelling	Present and clinically identifiable	Typically absent
Aetiology	Often post-infective (respiratory illness) or idiopathic	Frequently traumatic or atraumatic strain
Association with Sports	Reported in tennis, golf—due to sternocostal torque and acceleration-deceleration forces	Commonly associated with repetitive strain but less strongly linked to upper rib focal swelling

**Table 3:** Tietze’s syndrome vs costochondritis [Clinical differences].

### Rarer causes of anterior chest wall syndrome (CWS)

#### Rarer cause of MSK chest pain include

Sternalis syndrome is a rare cause of chest pain from a hyperactive or spasming accessory muscle (sternalis muscle) located over the sternum, often mistaken for cardiac issues. It causes localized, sometimes exercise-induced, sternal pain that may radiate when palpated. Hyperactivity or spasm of the abnormal, accessory sternalis muscle is found in about 7 - 8% of the population [7], and is considered to be the cause of the pain. Treatment for this rare cause of chest pain range from rest and physical therapy to Botox injections for severe cases.

SAPHO syndrome (Synovitis, Acne, Pustulosis, Hyperostosis, Osteitis) is a rare inflammatory disorder causing bone, joint, and skin issues, characterized by sterile bone inflammation (especially the chest wall), severe acne, pustules, and bone hardening (hyperostosis). It's an autoinflammatory condition, starting in childhood or adolescence, and can be underdiagnosed due to its varied presentation, sharing features with other conditions like chronic nonbacterial osteomyelitis [8].

Intercostal neuropathy (neuralgia) Intercostal neuralgia is a local nerve pain that affects the area below the ribs and causes chest pain. It is caused by a variety of conditions. Post thoracotomy pain due to irritation of the intercostal nerve is frequently reported post thoracic surgery. Post herpetic neuralgia can also cause Intercostal neuralgia His type of non-cardiac chest pain can also occur in pregnancy due to local compression. Symptoms include sharp, burning, or stabbing pain (band-like), numbness, and tingling, often worsened by movement, coughing, or sneezing.

In practice, multiple names have been used to describe this syndrome of musculoskeletal or non- cardiac chest pain, such as Tietze Syndrome, costochondritis, anterior chest wall syndrome, atypical chest pain, musculoskeletal chest pain syndromes. The variety of nomenclature probably reflecting the poor understanding of this condition [9].

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. It is a common and benign condition and common in the sporting community. Despite a diagnosis of CWS there are often lingering doubts among certain patients. Even in the face of normal coronary angiograms, 44% of patients with non cardiac chest pain with evidence of CWS still believed they suffer from an underlying cardiac disease and 50% reported limitations in performing their daily activities [10]. Therefore, a primary focus on ruling out cardiovascular disease in patients with NCCP may result in over testing without improving the patients' confidence. This issue can be surmounted if the CWS is successfully treated.

### Anatomy and relationships of the costochondral junction (2nd and 3rd ribs) as associated with Tietze syndrome

The costochondral junction (CCJ) is the transitional region where the osseous rib meets the hyaline costal cartilage. It is a primary cartilaginous joint (synchondrosis) without a synovial cavity and functions as a flexible but stable unit in thoracic wall mechanics, respiratory excursion, upper-limb kinetic chain movement (e.g. golf, tennis), and protection of mediastinal structures.

The 2<sup>nd</sup> and 3<sup>rd</sup> CCJs are of clinical relevance, being the most commonly symptomatic in Tietze syndrome and costochondritis.

- The second rib is longer and more curved than the first, with a thin, flat body and a well-defined angle.
- The third rib is similar but slightly shorter and less oblique.
- The costal cartilage of each rib extends medially and inserts into the sternum (rib 2) or into the costal margin (rib 3 → sternum via a direct articulation).

The costochondral junction is a firm fibrocartilaginous interface where the rib's periosteum blends gradually with the perichondrium of the costal cartilage. It is avascular relative to bone, relying largely on the perichondrial surfaces and adjacent muscular attachments for vascular support.

### 2<sup>nd</sup> costal cartilage

The second costal cartilage articulates with the manubriosternal synchondrosis at the sternal angle (Angle of Louis). This forms a true synovial joint between the cartilage and sternum (sternocostal joint). Because it straddles the manubrium and body of the sternum, it undergoes complex stress loading, especially in activities producing thoracic rotation or scapulothoracic acceleration (e.g. tennis forehand, golf swing).

### 3<sup>rd</sup> costal cartilage

The third costal cartilage articulates directly with the body of the sternum in a simpler plane synovial joint. It lies inferior to the 2<sup>nd</sup> cartilage and shares load transmission pathways with the upper ribs during respiration and trunk rotation. The interchondral ligaments between 2 - 3 and 3 - 4 also contribute to stability.

The intercostal nerves, branches of the ventral rami of T2 and T3, supply the costochondral junctions.

- Anterior cutaneous branches: Pierce the intercostal muscles and sternum-adjacent fascia, supplying skin overlying the sternum.
- Lateral cutaneous branches: Supply the lateral thoracic wall; clinically relevant due to referred pain patterns.
- Muscular branches: Supply intercostal muscles that insert onto the rib and cartilage, transmitting nociceptive input from periosteum, perichondrium, and muscle-tendon interfaces.
- Costal cartilage itself is aneural, but the perichondrium is richly innervated, explaining severe point tenderness in Tietze syndrome.

The 2<sup>nd</sup>-3<sup>rd</sup> costochondral region lies deep to:

- Pectoralis major (sternocostal head)
- Pectoralis minor (more lateral)
- Transversus thoracis (posterior surface of sternum)
- External/internal intercostal muscles.

The 2<sup>nd</sup> rib, uniquely, articulates across manubriosternal junction, explaining its clinical sensitivity and characteristic stress responses. These relationships explain why inflammation at the CCJ can mimic pleurisy, myocardial ischemia, or anterior mediastinal discomfort.

### Clinical presentation of Tietze syndrome

Tietze's syndrome is an inflammatory disorder of the anterior chest wall, characterised by localized pain and swelling at the costochondral junctions, most frequently involving the second and third ribs. Although often grouped with costochondritis, Tietze's syndrome is distinct in its rarity, typical focal swelling, and classical upper costochondral involvement [11,12]. The condition is self-limiting but may persist for months, with intermittent exacerbations. Diagnosis is clinical following the exclusion of cardiopulmonary and other serious pathology.

Tietze's syndrome is a benign but troublesome condition affecting the anterior chest wall. First described in 1921, it presents with localised pain and swelling at the perichondrium of the costal cartilages. The exact aetiology remains unknown. Although seen in both sexes and across a wide age range, it is comparatively rare relative to costochondritis. Clinicians frequently encounter diagnostic challenges due to the need to exclude potentially life-threatening cardiovascular or pulmonary pathology, resulting in diagnostic delay for affected individuals.

In 1921 - Alexander Tietze (1864-1927) described the condition based on cases of painful, non-suppurative costochondral swelling [13]. The publication established a distinct clinical entity from infectious or suppurative arthritis of the chest wall in regarding a peculiar cluster of cases with dystrophy of the costal cartilage. The original description differentiated the condition from chest wall infections and malignancy.

In discussing the aetiology over a hundred years ago Alexander Tietze tentatively suggested that the condition represented a dystrophic change in the cartilage analogous to the bone disease of nutritional origin which was prevalent in Germany in the early 1920's. It was considered it was a discrete entity differing from costochondritis which affected a larger number of costochondral levels and lacked the local swelling.

### Tietze syndrome in a sporting population

There are a number of MSK condition that have to be differentiated. Tietze's syndrome contrasts with costochondritis, particularly in the context of sport-related chest wall loading. The 2<sup>nd</sup> and 3<sup>rd</sup> CCJs are the highest-load segments during upper-thoracic rotation, shoulder protraction/retraction, and force coupling between the serratus anterior, pectoralis major, and intercostal muscles [14]. Their richly innervated perichondrium explains the exquisite tenderness found in inflammatory syndromes. Their unique relationships with the sternum and manubriosternal angle render them susceptible to microtrauma, sudden torsional loads, and repetitive stress. This is particularly the case when playing sports such as tennis, Golf or Hurling, where the repetitive rotational forces and abrupt scapulothoracic acceleration-deceleration can impose significant load on the anterior chest wall [15,16]. This may precipitate symptoms or exacerbate pre-existing costochondral irritation, especially in Tietze's syndrome where upper-rib torque is key driver of local injury.

### Diagnosis of Tietze syndrome

The hallmark of anterior chest wall syndrome is reproducible pain upon palpation of the affected area on the chest wall. There is no specific radiological finding:

- Symptoms: The pain is typically localized, can be sharp or aching, and may be aggravated by movement, deep breathing, coughing, or physical activity. It is often found on the left side, which increases patient anxiety about a heart condition.
- Diagnosis of exclusion: A diagnosis is made after life-threatening conditions (e.g., heart attack, pulmonary embolism) have been ruled out, especially in patients over 35 or with cardiac risk factors.
- Physical exam: The key to diagnosis is a physical exam where pressing on the specific costal cartilages or joints reproduces the pain.
- Imaging/Labs: In typical cases, lab results, ECGs, and chest x-rays are normal.

### Radiology investigation

- Conventional radiology is typically non-diagnostic, as there are no characteristic radiographic findings.
- Bone Scintigraphy [3 phase bone scan].
- Three-phase bone scanning may show increased uptake at the involved costochondral junction and can support the diagnosis in ambiguous cases.

### Diagnosis

Clinical diagnosis is the method of making a diagnosis of Tietze syndrome. The diagnosis is primarily clinical, made after exclusion of: A significant portion of clinical effort is often directed toward ruling out cardiovascular pathology before Tietze's syndrome is recognised.

Conditions which must be excluded in the differential diagnosis include:

1. Cardiovascular disease (angina, myocardial infarction, pericarditis).
2. Pulmonary disease (pleurisy, pulmonary embolism).
3. Musculoskeletal trauma (rib contusion or fracture, sternal injury).
4. Rheumatologic disorders (e.g. RA, seronegative arthropathies).
5. Neoplastic lesions (primary or metastatic involvement of costal cartilage).

### Management of Tietze syndrome

The condition is generally self-limiting and has a good prognosis, although it can recur. Playing with the condition or through pain usually results in longer term disability with Tietze Syndrome. Therapy should be directed towards:

- Activity modification and avoiding activities that stress the upper body and cause pain is recommended. Hence rest when the condition is active is a cornerstone of therapy.
- Reassurance and explanation of the benign nature of the condition.
- Non-steroidal anti-inflammatory drugs (NSAIDs).
- Local infiltration with anaesthetic and corticosteroid.
- Platelet-rich plasma (PRP) injections (used in selected cases).
- Hyaluronic acid injections (occasionally used).
- Intercostal nerve blocks, reserved for persistent or refractory pain.

Tietze's syndrome is self-limiting but may follow a relapsing course, with remissions and exacerbations a common pattern. The duration of a bout of Tietze syndrome is variable and can range from weeks to several months. In the sporting community recurrences are relatively common and probably inevitable due to the preconditioning of the autonomic and neurogenic sensitisation. A subset of patients appear to develop localized costosternal nociceptor hypersensitivity: In a similar to chronic tendon or enthesitis disorders. The intercostal nerve endings along the costochondral junction can become sensitised and therefore even minor inflammatory stimuli or cold exposure can "re-awaken" pain. This provides a neurophysiological basis for episodic pain recurrence even when swelling is minimal.

Relapse of Tietze syndrome can be triggered by viral or upper-respiratory illness with a direct association with Post-infectious and repetitive forced coughing. When the patient gets another viral or allergic respiratory flare, the mechanical impact of coughing can reactivate the previously inflamed costal cartilage.

Persistent low-grade inflammation of the costochondral junction is also a cause of a relapse of Tietze syndrome. After the acute swelling and pain settle in the costochondral junction low-grade peri-costal inflammation may remain histologically active. This residual inflammatory "smoulder" makes the costochondral cartilage more susceptible to re-activation with:

- Physical stress.
- Viral infections.
- Upper-respiratory tract inflammation resulting in vigorous coughing.
- Mechanical strain (golf, weightlifting, rowing, tennis and hurling).

Hence rest to allow full resolution of the condition is a cornerstone of treatment. Due to the mechanical vulnerability of the anterior chest wall the upper ribs (especially ribs 2-3) are subject to significant mechanical load during certain activities such as deep breathing, thoracic rotation, upper-limb abduction, sporting activities and chronic coughing. If the original episode led to cartilage oedema or micro-damage, that specific joint is more vulnerable to micro-trauma later. Resulting in the relapsing-remitting biomechanical pattern of injury. Golf and hurling have a similar propensity to injury and recurrence due to the nature of the acceleration deceleration pattern associated with striking a stationary or moving object which compromises the mechanically vulnerable costochondral junction.

### Tietze syndrome and tennis

Tietze syndrome, although often described as a self-limiting episode of anterior chest wall inflammation, is increasingly recognised in a minority of patients as a relapsing condition, particularly when the original costochondral injury has left the cartilage biomechanically vulnerable. In the game of tennis the costochondral structure is more exposed to injury as the ball is hit with rib cage torque in the service and varying forces during the rally when the player adjusts to the returning tennis ball depending on the stroke.

The upper costochondral joints-especially ribs 2 and 3-are placed under considerable rotational and torsional load during tennis, where forehand and backhand strokes generate rapid trunk rotation, rib-cage torque, and abrupt scapulothoracic acceleration. These movements can repeatedly stress a previously inflamed costal cartilage, reactivating low-grade inflammation or sensitised intercostal nociceptors.

### Intrinsic and extrinsic causes of CWS overuse injury

When returning a sportsman or woman to play, it is important to address intrinsic and extrinsic elements that may have been precursors to developing the overuse injury. In the case of Tietze syndrome. Intrinsic causes can be prior injury resulting in neuro sensitisation and often a persisting inflammation and potential for recurrence. Therefore, knowledge of prior pathology is important. Similarly extrinsic element such as equipment are also important to address. Evaluation of bat, club or racket is an important consideration when returning to play. Tennis rackets, hurls and shafts in golf clubs are critical elements to address before returning a player back to his activity. Hence getting advice from professional coach is also important to ensure that there is no equipment issue or biomechanical mistake being made in the stroke and shot making in any individuals who plays a racket or hitting sport.

In the case of a tennis player, assessing for the weight of the racket, the grip size and the tension in the strings is critical as well as ensuring serving style and ground stroke style is adequate and isn't placing unnecessary strain on the costochondral junctions.

In players with a history of heavy serving or high-velocity rotational play, even subtle asymmetry in shoulder-thoracic mechanics may lead to episodic flares of localised swelling and pain, giving the condition a relapsing-remitting pattern rather than a single monophasic course. Hence in the case of tennis players rest is critical and appropriate coaching of technique prior to returning to play will assist in preventing reoccurrence.

### Conclusion

Chest pain is a frequent complaint in the everyday medical practice. The causes are miscellaneous and varied, from life threatening diseases to benign musculoskeletal causes such as anterior chest wall syndrome (CWS). Chest wall pain can be a source of great morbidity and anxiety for patients. Various nomenclature is used to describe the MSK source of CWS [17]. While there is significant overlap in the different clinical entities, identifying the exact anatomical area of pathology is helpful in confirming the diagnosis and the treatment path [18-23].

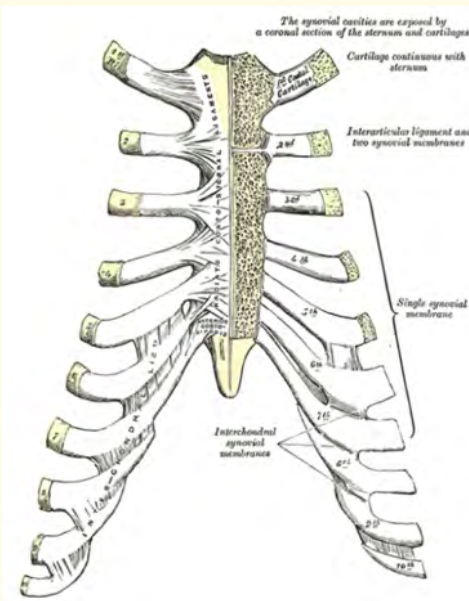


Figure 1: Anatomy of the bony anterior chest wall.

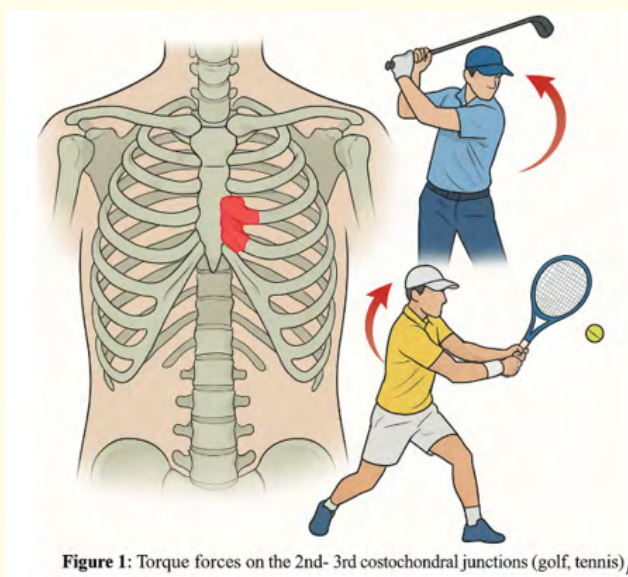
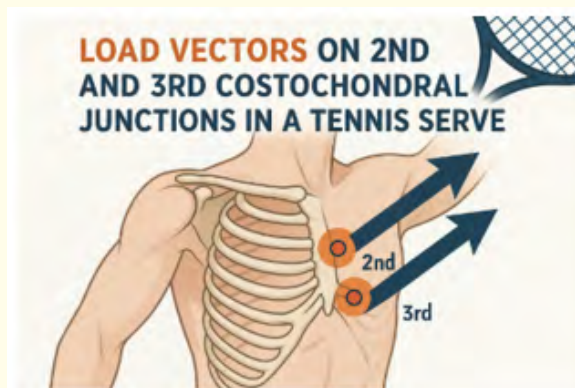
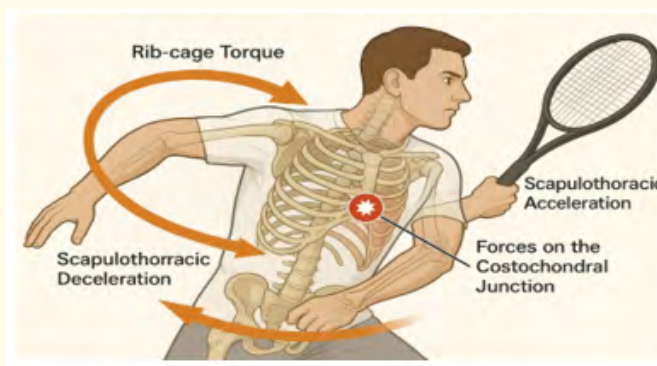


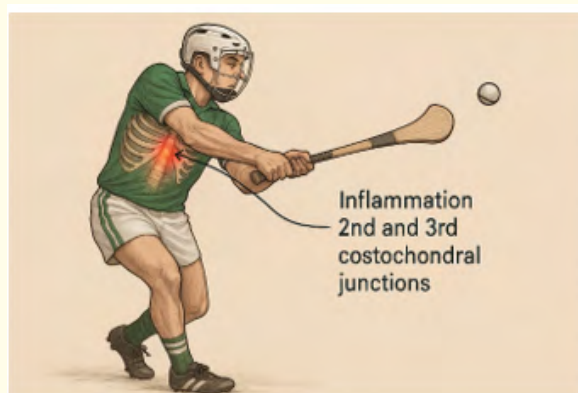
Figure 1: Torque forces on the 2nd- 3rd costochondral junctions (golf, tennis)/



**Figure 2:** Torque forces on the 2<sup>nd</sup> and 3<sup>rd</sup> costochondral junction during bat and ball sports such as golf and tennis.



**Figure 3:** Tennis serve and ground shots.



**Figure 4:** Striking a slitter with a hurl putting stress onto the costochondral junctions.

This painful condition of the anterior chest wall can be caused by a variety of musculoskeletal disorder that are associated with tenderness of the chest wall. However, while CWS is a benign condition it can produce impairment in patients' daily activities, create emotional distress and be a cause of anxiety.

Tietze's syndrome is a distinctive cause of anterior chest wall pain, characterised by focal swelling in the upper costochondral joints. The characteristic symptoms are tenderness, pain and oedema involving usually the second and or third costochondral joints on one side. Diagnosis of Tietze's syndrome is based on physical examination (increase of palpation tenderness in the affected joint). Accurate diagnosis relies on clinical assessment and the exclusion of cardiopulmonary and other serious pathology. Conservative therapy remains the mainstay of management. Differentiation from costochondritis can be achieved by the differences in anatomical distribution, presence of swelling, and clinical trajectory. Certain sports and activities are at risk of developing Tietze's Syndrome such as golf, hurling, rowing and tennis as the anatomical structure is liable to injury due to the rotational forces place on them due to the sporting activity. Awareness of sport-related biomechanical factors and addressing these may assist in understanding symptom exacerbation in active individuals.

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**Volume 17 Issue 2 February 2026**

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