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Understanding Tietze's Syndrome: A Rare and Often Overlooked Cause of Anterior Chest Wall Pain

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Abstract

Introduction: Tietze's syndrome is a rare, non-purulent arthropathy, most commonly associated with pain, swelling and tenderness in the 2nd and 3rd sternocostal junctions. Since its first description in 1921 many theories regarding the etiology and pathophysiology have been put forth, with a number of case reports and literature reviews. There is still, however, little agreement on the mechanisms underlying TS and diagnosis and treatment of TS remains challenging to clinicians to this day.

Aim: The purpose of this study is to review the existing scientific literature regarding the epidemiology, pathophysiology, clinical presentation, methods of diagnosing and managing Tietze's syndrome. Particular focus is placed on describing a comprehensive differential diagnosis of chest wall pain and outlining evidence-based treatment methods of TS.

Materials and methods: A comprehensive review of literature was conducted using the PubMed and Google Scholar databases. The following search phrases were used: „Tietze", „Tietze's Syndrome", „costochondritis", „Zespół Tietze'a". References were also screened in each article for additional sources.

Conclusions: Tietze's syndrome is a benign, self-limiting cause of chest wall pain that typically responds to simple symptomatic treatment but remains diagnostically challenging. The absence of standardized diagnostic criteria and limited epidemiological data often result in diagnostic

uncertainty and unnecessary investigations. Careful clinical assessment and appropriate follow-up are essential to ensure accurate diagnosis and effective management without overlooking alternative pathology. Increased clinician awareness and further clinical reporting are needed to reduce diagnostic delays and optimize care.

Keywords: Tietze's syndrome, musculoskeletal chest pain, chest wall syndrome, costochondritis

1. Introduction:

The first known description of Tietze's syndrome (TS) was provided in 1921 by prof. Alexander Tietze, assistant to Polish surgeon Jan Mikulicz-Radecki. In his paper " *Über eine eigenartige Häufung von Fällen mit Dystrophie der Rippenknorpel"* he describes the condition as "an initially painful, usually tender, prominence of one or more of the upper costal cartilages for which no specific etiology can be found" [1].

Most commonly, pain within the area of the 2nd and 3rd costal cartilages, as well as localized swelling and tenderness are described as the main symptoms of TS.
Our aim is to review the current scientific literature describing TS. The main objectives are:

- to outline the epidemiology, etiology and possible pathophysiological mechanisms behind TS
- to present a comprehensive description of clinical findings and diagnostic methods with the help of case reports in order to contextualize the approach
- to highlight the importance of a thorough differential diagnostic process
- to outline evidence-based treatment options for TS

2. Materials and methods

A comprehensive literature review was conducted using the PubMed and Google Scholar databases. To find data relevant to our study, we chose to use the following search terms: „Tietze's Syndrome", „Tietze", „costochondritis", „Zespół Tietze'a". Particular attention was paid to the similarities and differences in clinical presentations, as well as the range of

diagnostic and treatment methods described in case reports of patients diagnosed with TS. Additionally, the references described in the selected studies were screened for potentially useful sources. In an effort to obtain a deeper understanding of the topic, we decided to include not only case reports, but also literature reviews and cross-sectional studies.

3. Literature review

3.1. Epidemiology

Tietze's syndrome is a rare inflammatory disorder that most commonly affects individuals younger than 40 years of age [2,3]. The literature does not demonstrate a clear consensus regarding sex distribution. Several reports suggest an equal prevalence among men and women [3,4,5,6], whereas others indicate a slight female predominance [7]. No consistent occupational, racial, or geographic risk factors have been identified; however, sporadic case clustering has been described [4,8]. Epidemiological data on the prevalence of TS in the general population remain scarce [4,7]. The condition is likely underdiagnosed, as symptoms are often mild and self-limiting, potentially discouraging patients from seeking medical attention [5]. Limited awareness of the syndrome and its diagnostic criteria among clinicians may further contribute to underrecognition. Additionally, the true incidence may be underestimated because of diagnostic overlap with the clinically similar entity of costochondritis.

3.2. Etiology and Pathophysiology

To date, there is no consensus regarding the precise etiology of Tietze's syndrome. Early hypotheses proposed by Prof. Tietze suggested an association with tuberculosis or malnutrition; however, these theories have long since been disproved and abandoned [1,5]. Contemporary literature most commonly implicates repetitive mechanical stress to the costal cartilage, resulting in small structural lesions commonly referred to as microtrauma [3]. Such microtrauma may arise from blunt chest injury [9,10], strenuous physical activity [11], or postoperative factors [12].

Several case reports support this hypothesis. Koubaa et al. described a patient who developed Tietze's syndrome following repeated blunt trauma to the sternocostal region [9], while Mettola et al. reported symptom onset after a motorcycle accident in a patient with a history of sports-related trauma [10]. Postoperative onset has also been documented, including a case reported

by Kumar et al. in which refractory symptoms developed following lumbar spine surgery performed in the prone position [12]. In addition, Uyanik et al. described symptom onset shortly after strenuous physical exercise [11].

Another proposed mechanism involves repetitive increases in intrathoracic pressure, as seen with chronic coughing, sneezing, or vomiting, which may contribute to costal cartilage stress and injury. Upper respiratory tract infections, both viral and bacterial, have also been implicated as potential triggering factors [2,3,13,14]. In a prospective observational study conducted in 2017, Boran et al. reported a seasonal predominance of cases during the winter–spring period and found that 91.7% of patients had experienced a respiratory tract infection within two weeks preceding symptom onset [7]. Earlier observations by Gill et al. described clusters of Tietze's syndrome in young soldiers with concurrent pneumonia, chronic bronchitis, or rheumatic fever [8]. More recently, Tietze's syndrome has been reported in association with COVID-19, including cases occurring shortly after asymptomatic or symptomatic SARS-CoV-2 infection [13,15].

An increased incidence of Tietze's syndrome has also been observed among patients with inflammatory conditions, such as psoriatic arthritis, suggesting a potential role of systemic inflammatory mechanisms in disease development [3].

The pathophysiology of Tietze's syndrome remains poorly understood. Clinically, the condition is characterized by painful, localized swelling and edema, most commonly involving the second or third sternocostal junction or the costochondral joints. Approximately 70% of cases demonstrate unilateral involvement, although bilateral disease has been reported. Less frequently, the sternoclavicular or xiphisternal joints may be affected [3].

Histopathological data are scarce and inconsistent. Cameron et al. reported increased vascularity and degenerative changes within the costal cartilage, including mucopolysaccharide-rich clefts, calcification, hypertrophy, and patchy loss of ground substance, resulting in a fibrillar microscopic appearance [16]. In contrast, Gill noted that affected cartilage was often difficult to distinguish from normal tissue on microscopic examination [8]. Other reports have described hypertrophic cartilage without inflammatory cell infiltration [17], whereas Landon observed chronic inflammatory cell infiltration in the surrounding soft tissue and ligaments despite normal-appearing cartilage [5].

Overall, available evidence suggests that Tietze's syndrome may represent a localized inflammatory response to mechanical or infectious triggers, superimposed on individual susceptibility, although definitive pathogenic mechanisms remain to be elucidated.

3.3. Clinical presentation

The clinical presentation of Tietze's syndrome is nonspecific and typically includes localized pain of the anterior chest wall, which may radiate to the shoulder, neck [13], or upper extremity [17,18]. The condition shows a predilection for the upper ribs, particularly the second and third costochondral junctions, and is unilateral in more than 70% of cases. Less frequently, the chondrosternal, manubriosternal, sternoclavicular [10,13,18], or xiphisternal joints are involved [4]. Symptom onset may be acute or insidious, progressing gradually over time [3].

Pain is typically exacerbated by trunk rotation, arm movement, deep inspiration, coughing, sneezing, or bending [2,6,9,11,18,19,20,21,22]. The area surrounding the affected joint may be tender on palpation. Localized swelling is a hallmark feature of Tietze's syndrome and serves as a key distinction from costochondritis [7]. The swelling may be accompanied by erythema and increased local warmth [2,11,12,14,19], although these signs are not universally present [9,21,23].

Pain intensity and quality vary among patients and may be described as sharp, stabbing, or aching, occasionally forcing individuals to limit movement in an effort to reduce discomfort [14,24]. In some cases, pain may be pleuritic in nature and associated with subjective dyspnea [23]. Functional impairment, including reduced range of motion of the ipsilateral shoulder, has also been reported [13].

3.4. Diagnostic approach

Patients presenting with chest pain frequently seek care in cardiology clinics or emergency departments for fear of underlying heart disease. Even after exclusion of life-threatening conditions, symptoms may persist or remain unexplained, leading to repeated healthcare encounters, extensive diagnostic testing, and increased psychological distress [7].

The diagnosis of Tietze's syndrome is primarily based on clinical evaluation: a thorough history, physical examination, and the exclusion of alternative causes of chest pain. Initial evaluation

should prioritize ruling out serious and potentially life-threatening conditions, particularly acute coronary syndromes, as well as inflammatory diseases of the lungs and pleura [2]. Once serious cardiopulmonary and other relevant conditions have been excluded, the diagnostic focus should shift toward differentiating Tietze's syndrome from other causes of chest wall pain, most notably costochondritis, which shares similar symptomatology but lacks the characteristic localized swelling seen in Tietze's syndrome [2]. In addition to other common causes of chest pain, a psychogenic origin of chest pain should also be considered as part of the differential diagnosis [25].

3.4.1. Physical examination

A thorough physical examination is an essential element of the diagnostic process and should include careful assessment of chest wall tenderness and pain characteristics, including location, onset, radiation, and factors that exacerbate or alleviate symptoms. Systematic palpation of the anterior, lateral, and posterior chest wall is recommended. Applying gentle, localized pressure with a single finger may help accurately reproduce and localize the patient's pain [2].

The chest wall musculature, costochondral and sternocostal joints, and the cervicothoracic spine should be examined through palpation and range of motion should be assessed to better delineate the origin of pain [24].

As part of the initial diagnostic workup, the crowing rooster maneuver may be utilized as a helpful diagnostic tool. Although primarily described in the evaluation of costochondritis, this maneuver can help confirm a chest wall origin of pain and support differentiation from non-musculoskeletal causes of chest discomfort [20,26].

3.4.2. Laboratory findings

Laboratory findings in Tietze's syndrome are nonspecific. Case reports have documented mild inflammatory changes in some patients, including leukocytosis, elevated levels of C-reactive protein and ESR [2,11,12,13,27]. On the other hand, normal laboratory results with no

significant elevation of inflammatory markers have also been frequently reported [8, 18,21,23,24].

As part of the differential diagnostic workup, serological testing for rheumatoid factor and antinuclear antibodies is commonly performed to rule out rheumatoid arthritis and other seropositive inflammatory arthropathies. In patients with Tietze's syndrome, these markers are typically negative [11].

3.4.3. Imaging diagnostics

Plain radiography

Plain radiography is typically of limited diagnostic value in Tietze's syndrome, as findings are normal in the vast majority of cases [12]. No distinctive radiographic features have been consistently associated with the condition, and occasional calcification of the costal cartilages is considered to be nonspecific [4,5]. Rarely, subtle changes such as soft-tissue shadows over the affected area [8] or an apparent increase in the diameter of the involved costochondral junction have been reported [27].

Despite their limited sensitivity for detecting cartilage pathology, conventional radiological techniques, including standard X-rays and CT, remain useful for excluding alternative diagnoses such as osseous lesions or structural abnormalities [22].

Ultrasonography

Ultrasonography has been described as an effective imaging technique for identifying features consistent with Tietze's syndrome [3,6]. It is a readily available and noninvasive imaging method and, although sonographic findings may be subtle, some characteristic abnormalities of the affected costal cartilage and surrounding soft tissues can be appreciated. Typically, they include thickening of cartilage with poorly defined margins, increased echogenicity, and heterogeneity of the cartilage, often accompanied by edema of the adjacent soft tissues [28,29]. Comparative assessment with the unaffected, contralateral side is advised, as it can help in diagnostic confirmation [30].

In addition to its diagnostic utility, ultrasonography may be readily employed in the therapeutic setting by allowing image-guided corticosteroid injections into the affected area [6,30,31].

Computed Tomography

Computed tomography (CT) may demonstrate structural abnormalities of the costal cartilage and adjacent tissues in patients with Tietze's syndrome. Reported findings include enlargement of the affected costal cartilage, focal calcifications, edema in the surrounding soft tissue, a fracture-line-like appearance and bony sclerosis in proximity of the affected joints [28,30].

According to some authors, CT findings can be categorized into three main patterns: normal anatomy, focal cartilage enlargement, and ventral cartilage angulation [28,22].

Additionally, Koubaa et al. described a somewhat atypical CT finding: the presence of intra-articular air within the sternoclavicular joint space in a patient with TS [9].

Positron Emission Tomography

In several recent case reports, 18F-FDG PET/CT has been utilized in the diagnostic workup of TS. Increased FDG uptake has been reported at affected costochondral or sternoclavicular sites and has been often associated with prominent calcification on corresponding CT images [3].

Doudouh et al. described a case in which 18F-FDG PET/CT demonstrated focal hypermetabolic activity surrounding the sternoclavicular joint, with a maximum standardized uptake value (SUVmax) of 3,4. The increased uptake was localized beneath the cartilage and within the periarticular soft tissues surrounding the joint [18].

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is very effective for the evaluation of cartilage, joint, and bone marrow pathology and is therefore considered the imaging modality of choice in the diagnostic work-up of Tietze syndrome [10].

Characteristic MRI findings include focal or diffuse enlargement and thickening of the affected costal cartilage, typically demonstrated as increased signal intensity on TSE T2-weighted and STIR or fat-suppressed sequences. Additional features frequently observed are subchondral bone marrow edema and prominent contrast enhancement following gadolinium administration. Enhancement may involve the thickened cartilage, adjacent subchondral bone, joint capsule, and surrounding ligaments, reflecting active inflammatory changes [12,22,28,32,33].

Volterrani et al. reported MRI abnormalities in all (n=12) patients included in their study, with uniform detection of cartilage enlargement and thickening at the symptomatic site. Increased signal intensity within the affected cartilage on TSE T2-weighted and STIR or FAT-SAT sequences was observed in 10 of 12 patients and subchondral bone marrow edema in 5 patients. There was marked gadolinium enhancement of the thickened cartilage, subchondral bone marrow, and periarticular capsule or ligaments in 10, 4, and 7 patients, respectively [32].

In addition, edema of adjacent soft tissues, including the pectoralis major muscle can be observed, as described by Tan et al [13,22].

Dynamic contrast-enhanced (DCE) MRI has further contributed to the understanding of disease activity in TS. Kim et al. demonstrated that, when compared with healthy bone marrow, the affected subchondral bone marrow and capsular components show earlier and stronger enhancement, exhibiting a steep uptake slope and delayed wash-out. The authors presume such a pattern of enhancement to reflect angiogenesis related to mechanical stress–induced proliferative and inflammatory activity characteristic of TS [Kim].

3.5. Differential diagnosis

Chest pain is a frequent and diagnostically challenging complaint, with potential origins including intrathoracic structures, referred pain from the abdominal cavity or cervical spine, chest wall pathologies, systemic diseases, and psychogenic causes. Notably, pain may arise from nearly all thoracic structures except the lung parenchyma, which lacks sensory innervation [34].

The diagnosis of Tietze syndrome (TS) is one of exclusion and requires a systematic evaluation to rule out a broad spectrum of cardiopulmonary, gastrointestinal, infectious, inflammatory,

neoplastic, traumatic, and psychogenic conditions [6,11,17,23,34,35]. In primary care settings, chest wall syndromes constitute the most common cause of chest pain. In a large cohort study by Bösner et al. involving 1,212 patients presenting to general practitioners, chest wall syndrome accounted for 46.6% of cases and encompass a heterogeneous group of conditions, including costochondritis, costosternal syndrome, sternalis syndrome, Tietze syndrome, rib-tip syndrome, and xiphoidalgia.

The next most common cause of chest pain was coronary artery disease (14.8%), followed by psychogenic disorders (9.5%), upper respiratory infections (8.1%), hypertension (4.0%), gastroesophageal reflux disease (3.5%), trauma (3.2%), and other causes [25].

A wide range of chest wall disorders may mimic TS and should be carefully considered. These include costochondritis, rib trauma, benign and malignant neoplasms, slipping rib syndrome, inflammatory and degenerative arthritis, painful xiphoid syndrome, traumatic or overuse-related muscle pain, precordial catch syndrome, intervertebral disc disease, seronegative spondyloarthropathies, multiple myeloma, chest wall abscesses, spinal cord pathologies (e.g. spinal tumors), thoracic nerve disorders (e.g. herpes zoster), fibromyalgia, psychogenic pain, and referred pain from the cervical spine [3,4,12,13,36]. Importantly, there have been multiple case reports documenting patients with lymphoma initially misdiagnosed as TS. This proves the necessity of thorough evaluation and vigilance for malignant causes [35,36,37].

Costochondritis

Costochondritis is a relatively common cause of anterior chest wall pain and is more frequently encountered than TS. First described by Carabasi et al. in 1962, it typically affects multiple costochondral junctions unilaterally—most often the second to fifth ribs—in approximately 90% of cases. Most authors report a predominance in individuals over 40 years of age [4,26]. Clinically, patients present with localized pain and tenderness at the costochondral or chondrosternal articulations, or occasionally at the xiphoid process, without the visible swelling or inflammatory signs characteristic of TS [24]. Pain may range from dull and aching to sharp or pressure-like and is often exacerbated by upper limb movement, deep inspiration, or physical exertion. Management is conservative and includes analgesics, nonsteroidal anti-inflammatory drugs, local heat or ice application, physiotherapy, and, in refractory cases, local corticosteroid or sulfasalazine injections. Lastly, costochondritis may coexist with other conditions such as

seronegative arthropathies, cervical strain syndrome, coronary artery disease, or fibromyalgia [2,4,24,26].

Slipping Rib Syndrome

Slipping rib syndrome is a rare cause of chest wall pain resulting from hypermobility of the 8th, 9th, or 10th costal cartilage. Patients may report sharp pain accompanied by a clicking sensation or abnormal movement, which can sometimes be reproduced by manual manipulation of the rib cage. The condition is often post-traumatic. Initial management is conservative with oral analgesics, while persistent cases may require intercostal nerve blocks or surgical excision of the affected cartilage [4].

Neoplasm

Both primary and secondary neoplasms of the chest wall must be considered in the differential diagnosis of TS. Secondary involvement most commonly results from local growth of breast or lung carcinoma or from metastases originating from the lung, breast, thyroid, kidney, or prostate. Primary chest wall tumors are rare and include osteochondroma, chondrosarcoma, multiple myeloma and others. These entities may present with localized pain, swelling, tenderness, or pathological fractures and often require surgical resection as part of management [4,36,38].

Sternoclavicular joint pathologies

In cases presenting with sternoclavicular joint involvement,Edwin et al. propose a management algorithm to exclude traumatic injury, septic arthritis, osteoarthritis, inflammatory arthropathies (including rheumatoid arthritis and seronegative spondyloarthropathies), crystal-deposition arthropathies, SAPHO syndrome, condensing osteitis, Friedrich’s disease, and other rare conditions. The authors emphasize the importance of thorough clinical examination, as laboratory tests and imaging studies may show no abnormalities [39]. Additional causes of pain and swelling at the medial end of the clavicle include osteomyelitis (secondary to tuberculosis or pyogenic infection), rheumatologic diseases, and bone tumors such as osteoid osteoma [22].

Pediatric chest pain

In pediatric populations, musculoskeletal causes represent the most common etiology of chest pain. Kassab et al., in a cohort of 3,700 children, reported musculoskeletal pain in 46.3% of cases, followed by psychogenic (28.4%), idiopathic (11.6%), respiratory (7.3%),

gastrointestinal (3.1%), and miscellaneous causes (2.3%). Among musculoskeletal etiologies, precordial catch syndrome, Tietze syndrome, and costochondritis were identified [40]. Precordial catch syndrome, also known as Texidor's twinge, is a benign condition characterized by sudden-onset, sharp, well-localized precordial pain lasting seconds to minutes and exacerbated by deep inspiration. There is no associated swelling or erythema, and reassurance is typically sufficient. It accounts for up to 80–90% of non-traumatic chest pain in children and adolescents [40,41].

Lymphoma

Lymphoma represents a critical and potentially misleading differential diagnosis, as several case reports describe presentations closely resembling TS, including young age, unilateral parasternal pain, tenderness, and swelling. These patients often fail to respond to nonsteroidal anti-inflammatory drugs or corticosteroid injections. Biopsy is ultimately decisive in establishing the diagnosis of lymphoma [35]. Cipoletta et al. describe a 26-year-old woman with rapidly progressive swelling of the second costochondral joint, initially diagnosed with TS and treated with 1800 mg/day Ibuprofen without improvement. The authors point to features prompting further investigation: rapid mass enlargement, systemic symptoms, clinical deterioration, lack of response to first-line therapy, and ultrasonographic findings suggestive of an aggressive process. The diagnosis of diffuse large B-cell lymphoma was confirmed by biopsy and FDG-PET/CT. Uthman et al. recount a similar case of a 26-year-old female patient presenting with a history of right upper parasternal pain, which responded well to NSAIDs but recurred shortly after the medication was discontinued. A few months later she developed a bulging mass over the affected site. Biopsy confirmed the diagnosis of Hodgkin's lymphoma [37]. Fioravanti et al. suggest that frequent sternal involvement in Hodgkin lymphoma may be related to proximity to thoracic lymphatic pathways or direct invasion from mediastinal disease [36].

Psychogenic chest pain

Psychogenic chest pain is more frequent in women and is characterized by variable symptom location and intensity. Commonly, patients present with other somatic (e.g. headache, fatigue, constipation), and psychiatric (e.g. anxiety or depression) symptoms. Laboratory and imaging studies are typically normal. Careful history taking aimed at identifying psychosocial stressors is essential, particularly in adolescent patients [4,41].

3.6. Treatment

Management of Tietze syndrome (TS) is mainly conservative, as the condition is generally self-limiting. Treatment is primarily aimed at pain control and reduction of local inflammation. Less frequently, refractory cases may occur and require interventional or, rarely, surgical treatment.

Conservative Management

First-line therapy consists of nonsteroidal anti-inflammatory drugs (NSAIDs), analgesics, and local supportive measures. Conservative treatment options include oral or topical NSAIDs, application of local heat, activity modification, and reassurance. In mild cases, radiant heat or warming wraps and compresses applied to the affected area may be sufficient to achieve symptom relief [2,5,23]. Manual therapy and physiotherapy-based approaches have also been described as adjunctive treatments [24].

Several NSAID regimens have been reported in the literature. Uyanik et al. describe successful symptom control with one month of oral and topical NSAIDs [11]. Boran et al. administered diclofenac 100 mg/day or flurbiprofen 200 mg/day for a minimum of 2–3 weeks, reporting pain resolution in 87% of patients within three weeks, although localized swelling frequently persisted; recurrence occurred in 12.2% of cases [7]. Other proposed regimens include naproxen sodium 220 mg once or twice daily, ibuprofen 200 mg (2–4 tablets) administered two to three times daily, or flurbiprofen 100 mg twice daily for approximately three weeks [38]. Shorter treatment courses have also been effective, with symptom resolution reported after one week of ibuprofen 600 mg three times daily [15]. In cases of severe pain, adjunctive measures such as warm compresses and short-term opioid use for breakthrough pain have been described [23].

Selective cyclooxygenase-2 inhibitors may also be effective. Mettola et al. reported successful treatment with etoricoxib 90 mg/day for three months in combination with physical therapy, followed by maintenance therapy with etoricoxib 60 mg as needed, with sustained symptom resolution [10].

Systemic corticosteroids have been used in selected patients, particularly in cases with pronounced inflammatory features. Various treatment protocols have been proposed, including short courses of oral prednisolone alone or in combination with NSAIDs, with generally

favorable outcomes [27]. Colchicine has also been reported as an adjunctive therapy in cases of TS associated with COVID-19 infection; Tan et al. described rapid pain reduction and functional improvement following treatment with ibuprofen and colchicine, with complete symptom resolution within six weeks [13].

Local Injections and Interventional Therapies

For patients who fail to respond adequately to oral therapy, local injections of corticosteroids combined with local anesthetics are increasingly recognized as effective and safe second-line treatments. These injections may be administered with or without imaging guidance, although ultrasound guidance improves accuracy and safety.

Yıldız et al. conducted a single-center retrospective study involving 26 patients with TS refractory to at least three weeks of NSAID therapy. Patients received intra-articular injections of triamcinolone hexacetonide and prilocaine hydrochloride at the affected joints, resulting in a ≥70% reduction in pain in 75% of patients at follow-up [19]. Recently, it has been suggested that other corticosteroid formulations, including betamethasone and triamcinolone, may be used effectively for treatment of TS, based on their established efficacy in osteoarthritis [19].

Ultrasound-guided injection techniques have demonstrated particularly promising results. Kamel et al. reported that intra-articular injection of triamcinolone hexacetonide combined with lidocaine provided clinically significant, long-lasting symptom relief and was well tolerated [30]. Do et al. described an 80% reduction in pain severity one week after ultrasound-guided injection of triamcinolone and lidocaine into the affected costochondral joint [6].

Intercostal nerve blocks and local lidocaine injections have also been described as therapeutic options in selected cases [7].

Surgical Treatment

Surgical intervention is reserved for exceptional cases that do not respond to prolonged conservative and interventional management. The evidence supporting surgical treatment in TS is limited [19]. Gologorsky et al. reported complete symptom resolution following wide resection of hypertrophied costal cartilage in a patient with persistent TS unresponsive to medical therapy [17]. Kumar et al. described a patient who underwent surgical excision of the sixth and seventh costal cartilages after failure of NSAID therapy, followed by treatment with

tumor necrosis factor inhibitors (adalimumab), resulting in complete recovery over nine months [12]. While these reports suggest potential benefit in carefully selected patients, surgery remains a last-resort option and should not be performed routinely.

Alternative Therapies

Adjunctive non-pharmacological treatments have been described, though evidence remains limited. Jaya et al. reported successful pain resolution following acupuncture and cupping therapy in a patient with TS refractory to conventional treatment [42]. Manual mobilization of the costochondral joints combined with taping and breathing exercises has also been associated with pain reduction and functional improvement, although the quality of evidence is low and limited by methodological constraints [20].

Addressing perpetuating factors is a critical part of management. Treatment of underlying conditions associated with repetitive mechanical stress, such as chronic coughing, sneezing, or vomiting, is essential [14]. Patients should be advised to avoid strenuous physical activity, heavy lifting, or repetitive upper-body movements for at least 1–2 weeks during recovery, as premature exertion may impair symptom resolution and increase the risk of recurrence [3,7,38].

3.7. Prognosis

Tietze syndrome is generally regarded as a benign and self-limiting condition with a favorable prognosis [2,3]. In most patients, complete symptom resolution occurs within 1–2 weeks, particularly when appropriate conservative treatment and activity modification are implemented [3].

Nevertheless, a subset of patients may experience a prolonged or recurrent course. Chronic or relapsing symptoms have been reported, especially in the presence of persistent mechanical or inflammatory triggers, such as those described in the pathophysiology of the disease. Patients should therefore be informed about the potential for symptom recurrence and advised on strategies to avoid risk factors [2,38].

Despite the absence of standardized follow-up guidelines, clinical reassessment is recommended approximately 4–6 weeks after the initial presentation. This serves to confirm symptom resolution, monitor for recurrence, and ensure that the clinical course remains

consistent with Tietze syndrome rather than an alternative diagnosis requiring further investigation [23,38].

4. Conclusions:

Tietze's syndrome is a benign, self-limiting condition for which low-cost, symptomatic treatment is usually sufficient. However, establishing an accurate and timely diagnosis remains challenging. The absence of clearly defined diagnostic criteria, together with limited knowledge regarding its etiology and true epidemiological prevalence, often leads to diagnostic uncertainty, unnecessary investigations, and increased healthcare costs [11,23]. In this context, Tietze's syndrome is often applied as a non-specific or residual diagnosis in cases of painful parasternal swelling rather than as a clearly defined clinical entity [35]. Accurate recognition of Tietze's syndrome provides an optimal foundation for effective pain management, avoids prolonged and costly workups for alternative conditions, and, importantly, does not compromise the detection of possible malignancy. A careful history and physical examination remain central to diagnosis [7], with appropriate follow-up ensuring that the clinical course is consistent with TS and not suggestive of another pathology [23]. Although rare, Tietze's syndrome should be considered in the differential diagnosis of localized chest pain to reduce diagnostic delays and unnecessary interventions. Increasing awareness of this underdiagnosed condition among clinicians is essential to minimize patients' physical and psychological distress, time loss, and economic burden. Effective collaboration between general practitioners, rheumatology and radiology specialists is often critical in establishing an accurate and timely diagnosis [3]. Further well-documented case reports and clinical observations are needed to improve understanding of this syndrome and to refine diagnostic approaches in the future.

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Methodology: AC,KDW, BC,NT,
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