Calcifying Tendinitis of the Shoulder: Advances in Imaging and Management

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Introduction
Calcifying tendinitis of the shoulder is a common, acute or chronic, painful disorder characterized by calcifications in rotator cuff tendons. A natural cycle exists during which the tendon repairs itself. In chronic calcific tendonitis, however, this cycle is blocked at one of the healing stages. Because chronic presentation with exacerbations is usual, initial treatment should be conservative, including rest, physical therapy, nonsteroidal anti-inflammatory drugs, and, in later stages, subacromial infiltration with corticosteroids. Surgery is recommended when conservative treatment fails. This article discusses advances in imaging and medical, physical, and surgical management, as well as current evidence for the treatment of calcifying tendonitis of the shoulder.

Past
As early as 1892, Duplay [1] recognized the subacromial–subdeltoid bursa as a source of painful shoulders. He described the condition as scapulohumeral periarthritis, later also called Duplay’s disease. Painter [2] first mentioned calcifications in 1907. Since then, various nomenclature for the same condition has been used, including calcific tendonitis or tendinitis, calcareous bursitis, calcifying tendinopathy, and others. In Europe, the term tendinosis is used, whereas in North America tendonitis or tendinitis are more common terms. Codman [3] mentioned the intratendinous origin of calcifications in his classic textbook: “The deposits do not arise in the bursa itself, but in the tendons beneath it.” In 1912, Wrede [4] described the disease, including pathologic changes in the tendon: “The cells resemble more and more chondrocytes, meanwhile the fiber arrangement of the tendon is lost.”

Calcifications in the shoulder most commonly occur in the supraspinatus tendon (51%–90%) and least commonly in the subscapularis tendon (3%). The supraspinatus tendon is 2 to 3 cm long and traverses the subacromial compartment, which is rigidly limited by the coracoacromial arch above and the humeral head below. Codman [3] noted that diseases in the supraspinatus tendon tend to occur in a specific area of the tendon, or “about half an inch proximal to the insertion.” He called this area the critical portion and later the critical zone. Microangiographic and histologic examination studies by Rothman and Parke [5] showed that the critical zone is markedly undervascularized. The articular side of the supraspinatus tendon especially underfilled, regardless of the position of the arm [6]. These critical zones also exist in the infraspinatus and subscapularis tendons, as well as in the supraspinatus tendon.

Present
The etiology of calcifying tendinitis remains largely unknown. The condition may be related to hypovascular-ity-induced fibrosis and necrosis within the tendon with subsequent degeneration [7]. The disorder has four stages [8,9]. The first stage, or precalcific stage, involves fibrocartilaginous metaplasia within the tendon. This is usually one centimeter medial to the insertion of the supraspinatus.
tendon. In the second stage, or the formative phase, calcific deposits form in the fibrocartilaginous matrix. In the third stage, or the resorptive phase, deposits disappear by cell-mediated resorption. The final stage involves healing and rotator cuff repair [8]. There is a natural cycle in which the tendon repairs itself. In chronic calcifying tendonitis, however, this cycle is blocked at any one stage.

The incidence of calcifying tendonitis in the healthy population is 2.7% and 6.8% in those with shoulder pain [8,9]. About 50% of patients with cuff calcifications have shoulder pain [9,10]. The predominant age is 30 to 50 years, with women being two times more affected than men. In 10% to 25% of patients, the condition is present bilaterally, and the relation exists with hip calcifications. It is frequently seen in people whose occupation requires prolonged use of the arms in internal rotation and slight abduction, such as typists or assembly workers, although this is certainly not a prerequisite for calcifying tendonitis. When the arm is in internal rotation and slight abduction, rotator cuff muscles are held in constant synergistic contraction and the critical zone is in its most vulnerable ischemic state. This mechanical explanation of cuff tendinitis differs from the biological explanation based on the natural tendon degeneration, occurring in various locations in the body (eg, rotator cuff tendinitis, Achilles tendinitis, plantar fasciitis, and lateral epicondylitis).

Future?
Hypovascularity may not cause degeneration, but it may be the reason for the self-healing capacity of the human body to fail in these specific tendon sites. Moreover, Sengar et al. [11] reported an increased frequency of human leukocyte antigen serotype class A1 in patients with calcifying tendinitis, suggesting a possible genetic susceptibility to this condition. Gärtner [12] did not find this relationship in patients with calcifying tendinopathy.

At tissue level, an inflammatory process is visible. An influx of inflammatory cells occurs, particularly macrophages, leucocytes, and mast cells. These cells produce cytokines (inflammatory active products), cause altered cellular activity, and lead to the extracellular deposition of calcium hydroxyapatite crystals. Murine models have clarified these processes. The responsible mutation affects the protein product of human homolog of the murine progressive ankylosis gene (ANKH), resulting in diminished production of extracellular inorganic pyrophosphate, an important inhibitor of nucleation and the growth of apatite crystals [13]. On a more clinical level, Peach et al. [14•] published a study on the association among cuff tear arthropathy, ANKH, and the tissue nonspecific alkaline phosphatase (TNAP) gene. Cuff tear arthropathy was associated with variants in ANKH and TNAP that alter extracellular inorganic pyrophosphate concentrations, causing calcium crystal deposition. Cases of cuff tear arthropathy involved more variant genotypes than in controls (ANKH, 45% and 20%, respectively; TNAP, 32% and 9%, respectively). These results support the theory that genetic variants predispose patients to primary crystal deposition that, when combined with a massive rotator cuff tear, leads to arthritis development. Because there is no entirely clear relation among ANKH and TNAP genes and crystal deposition, this mutation will only be part of the story. Zhang et al. [15], in their basic research project on heritable chondrocalcinosis, noted that distinct ANKH mutations associated with heritable chondrocalcinosis may promote disease by divergent effects on extracellular inorganic pyrophosphate and chondrocyte hypertrophy, which may mediate differences in the clinical phenotypes and severity of the disease.

Imaging
Calcific deposits in the rotator cuff can be specifically localized in rotator cuff tendons by anteroposterior radiographs of the shoulder in internal and external rotation and axillary lateral radiograph. Different stages are characterized by appearance. The most widely used classification, the French Arthroscopic Society anteroposterior view, defined four types of deposits [16]. Type-A calcifications are sharply delineated, dense, and homogenous (Fig. 1). Type B are sharply delineated and dense in appearance, with multiple fragments (Fig. 2). Type C are heterogeneous in appearance, with a fluffy deposit. Type D are dystrophic calcifications at the tendon insertion. The last two types are associated with the resorptive stage, whereas type A and B seem blocked before they can reach the resorptive stage and are therefore associated with chronic calcifying tendinitis. Calcific deposits in the active or resorptive phases, especially, are barely visible on radiographs.