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Review Article

Calcifying Tendinitis of Shoulder: A Concise Review

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ABSTRACT

Calcifying Tendinitis (CT) shoulder a self limiting disorder characterized by deposition of calcium salts in rotator cuff muscles. The main symptom being pain followed by activity restriction resolving on its own in most cases. Symptomatic patients are initially managed by NSAIDs, Physiotherapy, Corticosteroid injections. ESWT involves acoustic waves causing fragmentation of deposits with pain releif. Ultrasound guided needling barbotage have shown promising results. Arthroscopic excision remains the definitive management for patients associated with complications as cuff tear and for uncomplicated patients. In calcifying tendinitis the initial evaluation, maintenance of function and appropriate choice of treatment modalities determines the prognosis

1. Introduction

Calcifying Tendinitis (CT) Shoulder a self limiting disorder of shoulder characterized by deposition of calcium salts in rotator cuff muscles. Names synonymous are Calcific periarthritis, Calcifying Tendinitis (CT) Shoulder a self limiting disorder of shoulder characterized by deposition of calcium salts in rotator cuff muscles. Names synonymous are Calcific periarthritis[1]. The etiology still remains unclear with many proposed theories of etiopathogenesis. The presenting symptom often is pain associated with activity persisting for months with spontaneous regression in most of the cases. Some have persistent pain & edema requiring active intervention. This article focus about the overview of calcifying tendinitis of shoulder and multiple management options for symptomatic cases.

2. History and demographics

Calcifying tendinitis was first described by Duplay in 1872 as painful periarthritis of the shoulder.[2] In 1934 Codman described the calcification occuring in tendons instead of Subacromial bursa as thought earlier.[3] In 1952 Plenk coined the term Calcifying Tendinitis.[4]

In a series of 6061 asymptomatic patients Bosworth et al. reported an incidence of 2.7%.[1] Documented incidence by different authors varies from 2.7 to 22% more in women compared to men.[1,5] Bilateral incidence in about 10–20% of cases.[6] The common age group affected is between 30–50 years.[5,7] It commonly involves Supraspinatus(80%) Tendon followed by Infraspinatus rarely affecting Teres minor and Subscapularis.

2.1. Natural history of disease

The Calcifying tendinitis is hypothesized to occur in following stages[8]:

(I) Precalcific Stage
(II) ICalcific Stage
  a) Formative Phase b) Resting Phase c) Resorptive Phase.
(III) Repair Stage

The deposits of Calcium are amorphous to semisolid in texture. The deposits consist of Calcium carbonate hydroxyapatite identified by means of Spectroscopy and X-ray diffraction techniques.[9] The hydroxyapatite salts consists of two forms Type A and Type B. Chiou et al. stated that the proportion of Type B hydroxyapatite increases with decrease in Type A during the process of progressive calcification.[9] Based on USG findings deposits are classified into following morphological shapes and their correlation with clinical features are described as follows.[9]

Formative Phase associated with arc or fragmented/punctuate deposits and mild pain; Resting phase with nodular deposits associated with moderate to severe pain. Resorptive phase with cystic deposits associated with severe pain.

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2.2. Complications

The progression of natural course of untreated disease leads to following complications[10]

Adhesive Capsulitis
Rotator cuff tear
Greater Tuberosity osteolysis
Osssifying Tendinitis

2.3. Etiology and pathogenesis

2.3.1. Etio pathogenesis

Etiopathogenesis of CT remains a debatable topic with multiple theories proposed. Two broad groups of theories attempt to explain CT. A group proposing Degenerative changes or minor trauma of the tendons predisposes to Calcification which is basically Dystropic type of calcification. Sandstorm proposed that Vascular ischemia of tendons leading to tendon necrosis that promotes dystropic Calcification.[11]

Bishop and Bosworth individually came out with a different theory of repetitive trauma of tendons that in-turn leads to tendon degeneration followed by calcification.[1,12]

Mohr again emphasized the theory of tendon necrosis predisposing to intracellular calcium accumulation as micro spheroliths and Psammomas.[13]

Other group of theories describes the process as an active process mediated by chondrocytes that arise from metaplasia which inturn causes calcium deposition in the Matrix.

Uhtoff identified that cartilage metaplasia of tendons predisposing f or calcification of tendons as an active cell mediated process.[14]

Benjamin also proposed that cartilage metaplasia leading to Enchondral ossification of fibrocartilage.[15]

Rui came out with new theory proposing erroneous differentiation of tendon-derived stem cells (TDSCs) leading to chondral metaplasia.[16]

Recent theories involving role of BMP-2, BMP-4 and BMP-7 in metaplasia of tendon cells leading to calcification are also proposed.[17]

In a cadaveric study[18] by Riley et al. the chemical composition of deposits found to have amorphous calcium phosphate and Hydroxyapatite predominantly unlike degenerative tendon which contains many forms of calcium salts including calcium triphosphate, pyrophosphate, carbonate and Hydroxyapatite which is predominantly 'Dystrophic calcification'. Degenerate tendons have increased Type III collagen but in CT no significant increase in these concentrations. Resorption of Calcific deposits is evaluated and involvement of Multinucleated Giant cell has been identified.[19] TRAP positive Giant cells contain Cathepsin K which confirms the Osteoclastic lineage and its involvement in Resorption.[19,20]

2.4. Clinical features

Clinical manifestations include pain in shoulder with or without restriction of movements. Symptoms commonly resolve on its own, except for some cases where they persist. Bosworth described resolution rate of 6.4% of deposits per year, with 9.3% of deposits resolving within 3 years.[1,21] Wolk and Wittenberg described resolution of calcification and symptoms in about 70% of the patients within a period of 49 months with spontaneous resolution of 82% within 8.6 years[22]. Benno et al. in study of 63 patients described association of calcifying tendinitis with renal lithiasis in 33% of individuals in comparison with 9% in control group.[23]

Pain being the major clinical symptom Neer described different causes of pain occurring Calcifying tendinitis[24] (Table 1).

The clinical features are documented by several scoring systems among which a commonly used scoring system includes Constant Murley score with Total 100 points distributed as

Subjective : Pain-15 points ; Ability to perform ADLS- 20 points
Objective : ROM- 40 points ; Muscle power- 25 points

Table 1
Causes of Pain occurring in calcifying tendinitis.

	Causes of Pain
1.	Calcium causing chemical irritation of tissues
2.	Tissue edema causing pressure
3.	Bursal thickening due to irritation causing impingement
4.	Pain caused by chronic stiffening of Glenohumeral joint

Strength is measured in 90° abducted arm with 30° flexion with extended elbow.

3. Imaging evaluation

3.1. Xray

Radiographic evaluation of shoulder is done by 2 views mainly

- Rockwood View: It is a true AP view with 30° caudal tilt focusing on subacromial space.
- Shoulder Outlet View

The commonly used radiographic classifications for CT are Gartner Hayer classification and SFA classification as in Table 2.

The locations of deposits are described based on True AP and shoulder outlet view using Quadrant technique.[6] In AP view (Fig. 1A) a perpendicular reference line drawn from Lateral border of Acromion the distance between medial border of the deposit and the reference line measured in millimeters and noted with minus if it is medial and noted with plus if it is lateral to the line.

In outlet view (Fig. 1B) 5 sectors are defined Sector 0 anterior to the Anterior border of acromion, Length of Acromion divided into 3 sectors from anterior to posterior as Sectors 1–3, Sector 4 lies posterior to the Posterior border of acromion.

The radiographic volume of the deposits determined by the product of Length (l), Breadth (b) Obtained from AP view, and depth (d) from outlet view.

Radiographic volume (V) = l*b*d

3.2. Ultrasonogram

Standard USG imaging using longitudinal and transverse views determines size, localization, echogenicity of the deposits (Fig. 2). Based on these characteristics Farin and Jaroma[27] classified them as in Table 3.

High Resolution Ultrasound (HRUS) is useful in determining the calcification, morphology and presence of associated Rotator cuff tear. HRUS with color Doppler is more descriptive about the calcification and their vascularity. Chiou et al.[28] classified the deposits based on findings on HRUS into 4 types as in Table 3.

The non arc shaped deposits described by HRUS are usually associated with Resorptive phase.[28]

Table 2
Radiologic classifications of Calcifying Tendinitis.

Gartner and Hayer[25]	Type I	Dense calcifications with well defined border
	Type II	Dense with Indefinite borders
	Type III	Transparent with indistinct border
SFA Classification (French Society of Arthroscopy)[26]	Type A	Dense, well Defined, Circumscribed
	Type B	Dense, Well Defined,Segmented
	Type C	Transparent and nonhomogenous
	Type D	Dystropic deposit at tendon origin.