Calcific Tendinopathy of the Rotator Cuff: Pathogenesis, Diagnosis, and Management

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Abstract

Calcific tendinopathy, or calcifying tendinitis, is a disease characterized by multifocal, cell-mediated calcification of living tissue. After spontaneous disappearance of the calcific deposits or, less frequently, surgical removal, the tendon reconstitutes itself. Attention to the clinical presentation and the radiologic, morphologic, and gross characteristics of the calcium deposit will facilitate differentiation between the formative phase and the resorptive phase, which is of paramount importance in the management of this disease. Should conservative treatment fail, surgical removal may be indicated during the formative phase, but only under exceptional circumstances during the resorptive phase. Aspiration and lavage of the deposit should be performed only during the latter phase.

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Calcific tendinopathy, or calcifying tendinitis, of the rotator cuff, is a common disorder of unknown etiology in which multifocal, cell-mediated calcification of a living tendon is usually followed by spontaneous phagocytic resorption.[1] After resorption or surgical removal of the deposit, the tendon reconstitutes itself. During the deposition of calcium, the patient may be free of pain or may suffer only a mild to moderate degree of discomfort. The disease becomes acutely painful only when the calcium undergoes resorption.

Calcifying tendinitis must be distinguished from degenerative or dystrophic calcifications, which occur at the insertion into bone but not in the midsubstance of the tendon. Radiologic signs of degenerative processes are extremely rare in calcific tendinopathies.

Pathogenesis

The etiology of calcifying tendinitis is still a matter of controversy. Circumscribed tissue hypoxia and localized pressure have been invoked as causative factors. Two fundamentally different processes leading to formation of calcium deposits in the cuff have been proposed: degenerative calcification and reactive calcification.

Degenerative Calcification

Codman[2] proposed that degeneration of the tendon fibers precedes calcification. The fibers become necrotic, and dystrophic calcification follows. Degeneration of the fibers of the rotator cuff tendons is usually attributed to a wear-and-tear effect as well as to aging. Obviously, these two causes are interrelated. The glenohumeral joint is not only a universal joint but is also probably the most used joint in the body, and studies performed in Sweden indicate that the stress and strain induced by work involving the arm can lead to supraspinatus tendinitis.[3] However, there is no evidence that even a worker engaged in heavy manual labor will necessarily develop calcifying tendinitis in time, and Olsson[4] has shown that the cuff tendons from the dominant arm show no more evidence of degeneration than those from the contralateral arm.

Aging is considered to be the foremost cause of degeneration in cuff tendons. Brewer[5] believes that with aging there is a general diminution in the vascularity of the supraspinatus tendon along with fiber changes. The most conspicuous age-related changes are seen in the fascicles, the well-delineated bundles of collagen that constitute the distinctive architecture of the

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tendon.[4] Beginning at the end of the fourth or during the fifth decade, most of the fascicles undergo thinning and fibrillation, both of which are defined as degenerative processes. The thinned fascicles show irregular cellular arrangement, and the fragmented fibers are often hypocellular. The volume of the connective tissue that carries the blood vessels between the fascicles may appear increased when compared with the volume of the fascicles.

Inasmuch as calcifying tendinitis seldom affects persons before the fourth decade, it can be argued that primary degeneration of tendon fibers is responsible for the subsequent deposition of calcium. Codman[2] proposed the degenerative nature of calcifying tendinitis, and many investigators have supported this concept. According to McLaughlin,[6] the earliest lesion is focal hyalinization of fibers, which eventually become fibrillated and detach from the surrounding normal tendon. Continued motion of the tendon grinds the detached, curled-up fibers into a wenlike substance consisting of necrotic debris, which becomes calcified.

Mohr and Bilger[7] believe that the process of calcification starts with necrosis of the tenocytes, with concomitant intracellular accumulation of calcium, often in form of microspheroliths, or psammomas. We have never observed psammomas during the early phases of formation but have noted them regularly during the phase of resorption. Our electron-microscopic examinations confirm that the electron-dense material is intracellular. It is unfortunate that Mohr and Bilger failed to distinguish between calcifications at the insertion and intratendinous calcifications, nor did they describe morphologic features characteristic of either formation or resorption.

In general, supporters of the theory of degenerative calcification fail to take into consideration the typical age distribution of affected persons, the course of the disease, and the morphologic aspects of calcific tendinopathy. The incidence of calcification increases with age in cases of degenerative calcification, whereas it peaks during the fifth decade in cases of calcifying tendinitis. Moreover, degenerative diseases never exhibit a potential for self-healing. Furthermore, the histologic and ultrastructural features of degenerative calcification and calcifying tendinosis are quite different.

Reactive Calcification

We concur with other investigators that the process of calcification is actively mediated by cells in a viable environment,[8-11] and there cannot be the slightest doubt that formation of the calcium deposit must precede its resorption. Consequently, we propose that the evolution of the disease can be divided into three distinct stages: precalcific, calcific, and postcalcific (Fig. 1).

Precalcific Stage

In the precalcific stage, the site of predilection for calcification undergoes fibrocartilaginous transformation. This metaplasia of tenocytes into chondrocytes is accompanied by metachromasia, indicative of the elaboration of proteoglycan.

Calcific Stage

The calcific stage is subdivided into the formative phase, the resting phase, and the resorptive phase.[11] Our "formative phase" seems to be identical to the "early phase of increment" of Lippmann,[12] and his "late phase of increment" is analogous to our "resorptive phase."

During the formative phase, calcium crystals are deposited primarily in matrix vesicles, which coalesce to form large foci of calcifi-

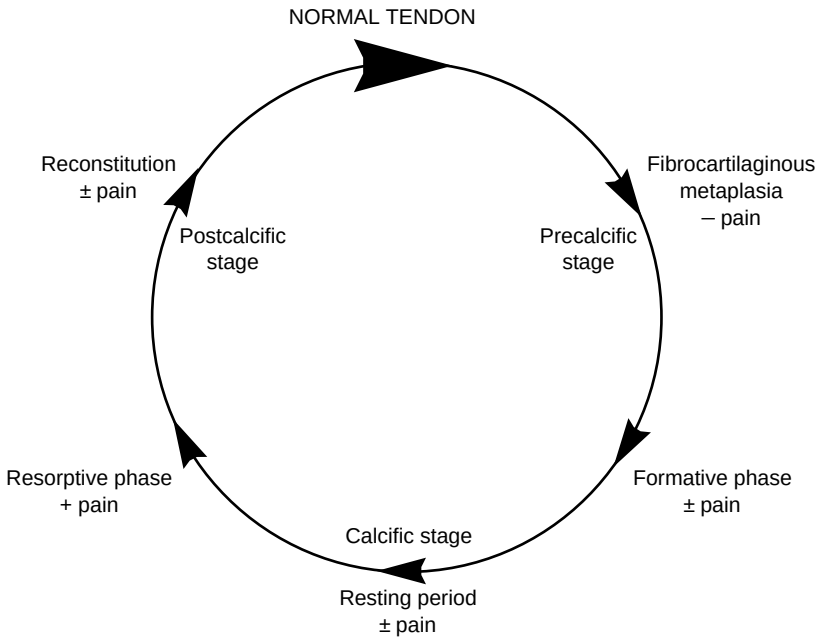

Fig. 1 Schematic representation of the progressive stages of calcifying tendinitis.

cation.[10] If the patient undergoes surgery during this stage, the deposit appears chalklike and must be scooped out. The fibrocartilaginous septa between the foci of calcification are generally devoid of vascular channels. They do not consistently stain positively for type II collagen, which is known to be a component of fibrocartilage. These fibrocartilaginous septa are gradually eroded by the enlarging deposits.

During the resting phase, fibrocollagenous tissue borders the foci of calcification. The presence of this tissue indicates that deposition of calcium at that site is terminated.

During the resorptive phase, after a variable period of inactivity of the disease process, spontaneous resorption of calcium is heralded by the appearance of thin-walled vascular channels at the periphery of the deposit. Soon thereafter, the deposit is surrounded by macrophages and multinucleated giant cells that phagocytose and remove the calcium. If an operation is performed during this stage, the calcific deposit contains a thick, creamy or toothpastelike material that is often under pressure.

Postcalcific Stage

Simultaneously with the resorption of calcium, granulation tissue containing young fibroblasts and new vascular channels begins to remodel the space occupied by calcium. These sites stain positively for type III collagen. As the scar matures, fibroblasts and collagen eventually align along the longitudinal axis of the tendon. During this remodeling process, type III collagen is replaced by type I collagen.

Although the pathogenesis of the calcifying process can be reasonably constructed from morphologic studies, it is difficult to establish what triggers the fibrocartilaginous transformation in the first place. Codman[2] suggested tissue hypoxia as the primary etiologic factor. This still remains an attractive hypothesis because of the peculiarity of the blood supply of the tendon and the mechanics of the shoulder. We have found an increased frequency of HLA-A1 in patients with calcifying tendinitis, indicating that they may be genetically susceptible to the condition.[13] Factors that trigger the onset of resorption also remain unknown.

Pathoanatomy

The calcium deposits are usually not in contact with the bone insertion; rather, they are at least 1.5 to 2.0 cm away from it. Only in isolated reports has the presence of calcific deposits in subchondral bone been described. It is important to note that not all foci of calcification in a given patient are in the same phase of evolution. In general, however, one phase predominates. The morphologic aspect of an individual deposit can vary from fibrocollagenous tissue to foreign body–like granulomatous tissue.

Precalcific Stage

We believe the disease starts with fibrocartilaginous metaplasia of tendinous tissue. The fibrocartilaginous areas are generally avascular. The intercellular substance is metachromatic, and glycosaminoglycan-rich pericellular halos around rounded cells are prominent.[14] Surprisingly, Archer et al[14] found that monoclonal collagen staining did not reveal the presence of type II collagen. In our studies performed with the use of type II collagen monoclonal antibodies, we could occasionally document its presence (Fig. 2, A). The different outcomes may be due to differences in tissue preparation, source of monoclonal antibodies, and/or staining technique.

Calcific Stage

Formative Phase

Under the light microscope, the calcific deposits appear multifocal, separated by fibrocollagenous tissue or fibrocartilage (Fig. 2, B). The latter consists of easily distinguishable chondrocytes (described by Archer et al[14] as chondrocytelike cells) within a matrix showing various degrees of metachromasia. The appearance of chondrocytes within the tendon substance near calcification has been documented by many authors since 1912. The ultrastructure of these chondrocytes shows that the cells often have a fair amount of cytoplasm containing a well-developed endoplasmic reticulum, a moderate number of mitochondria, one or more vacuoles, and numerous cell processes.[10] The margin of the nucleus is indented. The cells are surrounded by a distinct band of pericellular matrix with or without an intervening lacuna.

The first evidence of calcium deposition is the presence of loosely granular material that stains positive with the von Kossa method (Fig. 2, C) and coalesces to form clumps. On transmission electron microscopy, aggregates of rounded structures containing crystalline material are found in a matrix of amorphous debris or irregularly fragmented collagen fibers. Irregularly rectangular crystals are sometimes found within membrane-bound structures resembling matrix vesicles, or calcifying globules. Infrequently, crystalline densities seem to be embedded between collagen fibers. High-resolution transmission electron microscopy has revealed that the crystals are much larger than the classic apatite crystals and have a different configuration.[11]

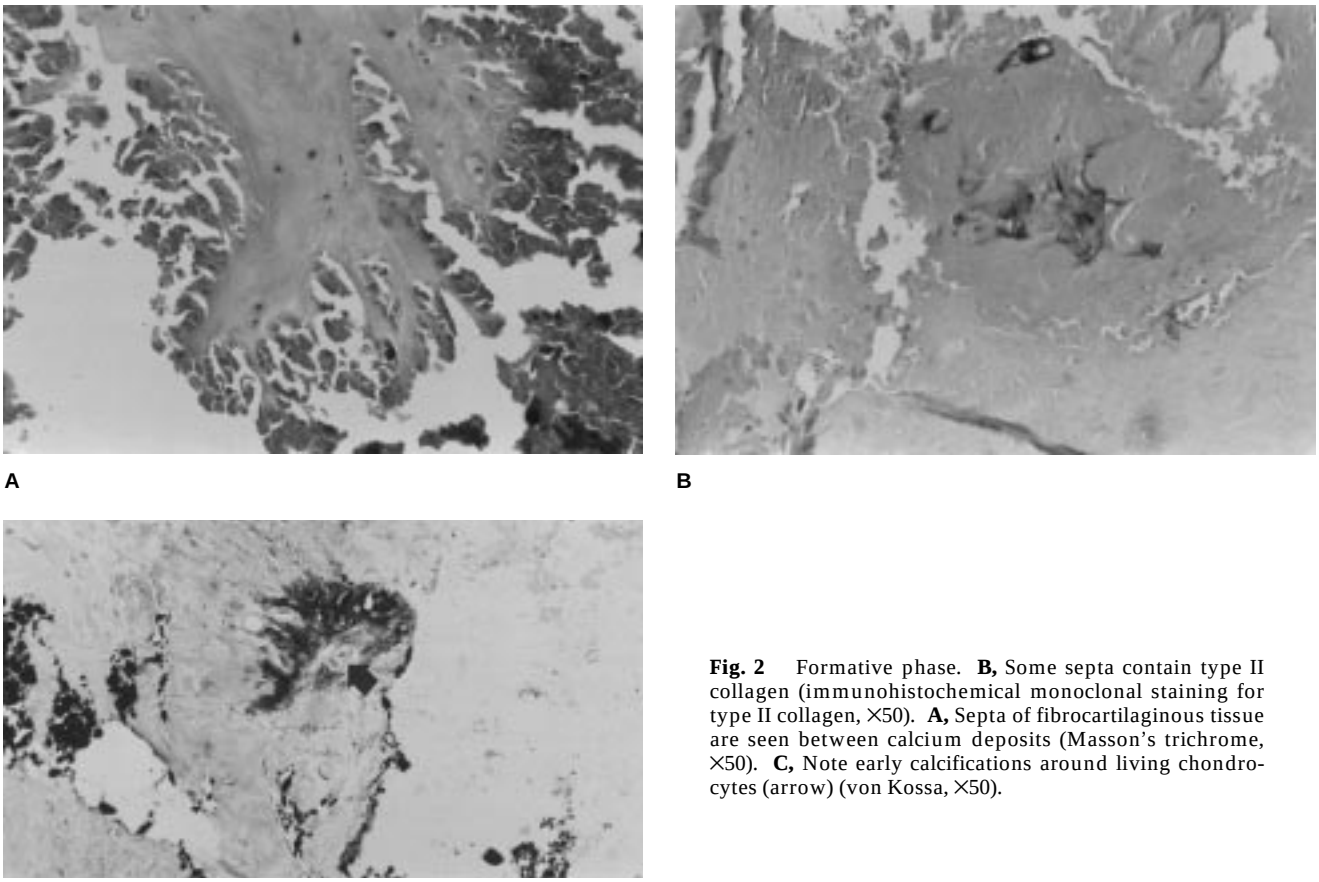

Fig. 2 Formative phase. **B,** Some septa contain type II collagen (immunohistochemical monoclonal staining for type II collagen, ×50). **A,** Septa of fibrocartilaginous tissue are seen between calcium deposits (Masson's trichrome, ×50). **C,** Note early calcifications around living chondrocytes (arrow) (von Kossa, ×50).

Resting Phase

During the formative phase, inflammation and vessels are notably absent. Other foci are surrounded by tendinous tissue without evidence of inflammation. These areas seem to correspond to the resting phase.

Resorptive Phase

Other foci show the presence of young mesenchymal cells, epithelioid cells, leukocytes, lymphocytes, and occasionally giant cells. The presence of these cells is indicative of resorptive activity. Indeed, the marked cellular reaction around calcific deposits, often called a calcium granuloma, is considered to constitute a characteristic lesion of calcifying tendinitis. The granulomatous appearance is imparted by the presence of multinucleated giant cells (Fig. 3, A) and macrophages. Archer et al[14] interpreted the presence of the latter two cell types as a resorption phenomenon. The cellular reaction is often accompanied by capillaries or thin-walled vascular channels around the deposits (Fig. 3, B). Phagocytosed material within macrophages or multinucleated giant cells can be easily discerned.

Ultrastructural examination of these cells shows electron-dense crystalline particles in cytoplasmic vacuoles, but the crystals are somewhat different in appearance from those in the extracellular deposits.[10] Some of the intracellular accumulations have a rounded aspect and are known as microspheroliths, or psammomas (Fig. 3, C).

Postcalcific Stage

Small areas representing the process of repair can be found in the general vicinity of calcification, showing considerable variation in appearance. Granulation tissue with young fibroblasts and newly formed capillaries (Fig. 4) contrasts with well-formed scars with vascular channels and maturing fibroblasts that are in the process of alignment with the long axis of the tendon fibers. Using monoclonal antibodies against type III collagen,

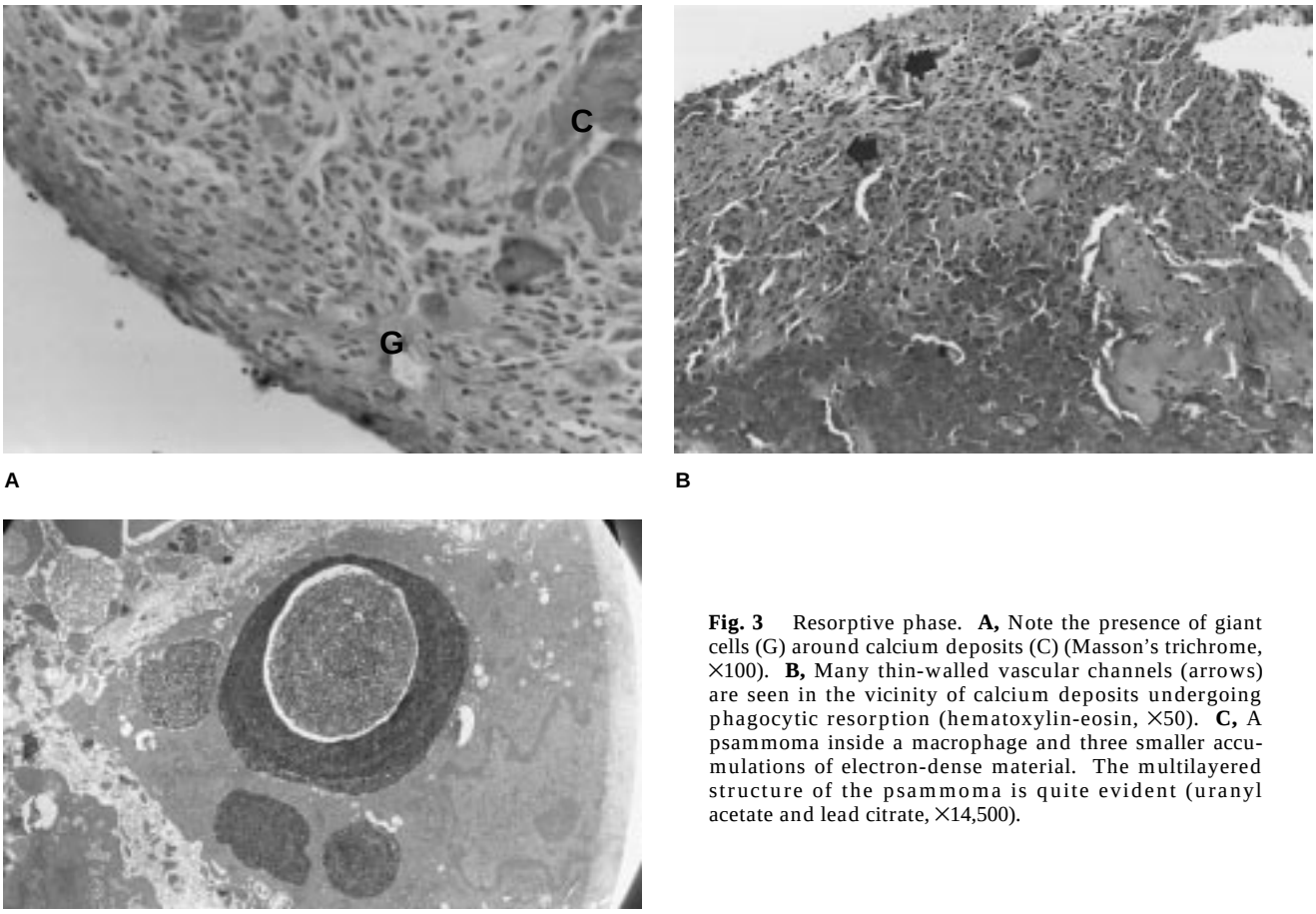

Fig. 3 Resorptive phase. **A,** Note the presence of giant cells (G) around calcium deposits (C) (Masson's trichrome, ×100). **B,** Many thin-walled vascular channels (arrows) are seen in the vicinity of calcium deposits undergoing phagocytic resorption (hematoxylin-eosin, ×50). **C,** A psammoma inside a macrophage and three smaller accumulations of electron-dense material. The multilayered structure of the psammoma is quite evident (uranyl acetate and lead citrate, ×14,500).

we were able to confirm collagen neoformation, which was most pronounced around vascular channels.

The subacromial bursa is rarely the site of a reaction. Should the size of the calcific deposit provoke a subacromial impingement, a localized bursal reaction may be present.

Radiologic Evaluation

Calcium deposits in calcifying tendinitis are most often localized in the supraspinatus tendon. Radiographs must be obtained whenever calcification of the cuff is suspected. Radiographic evaluation is also important during follow-up examinations because it permits assessment of changes in density and extent of calcification.

Initial radiographs should include anteroposterior views with the shoulder in the neutral position and in internal and external rotation. Deposits in the supraspinatus are readily visible on films obtained in neutral rotation, whereas deposits in the infraspinatus and teres minor are best seen on internal-rotation films. Calcifications in the subscapularis occur only in rare instances; a radiograph obtained with external rotation will show them well. Axillary views are rarely indicated. Scapular views, however, will help to determine whether a calcification is causing impingement.

Calcium deposits are often barely visible on radiographs, particularly in the acute or resorptive phase. We suspect that computed tomography may show them. Magnetic resonance imaging may be indicated in rare circumstances. On T1-weighted images, calcifications appear as areas of decreased signal intensity. T2-weighted images frequently show a perifocal band of increased signal intensity compatible with edema.

We have not found bursography to be of great value. Arthrograms show a distinct delineation between

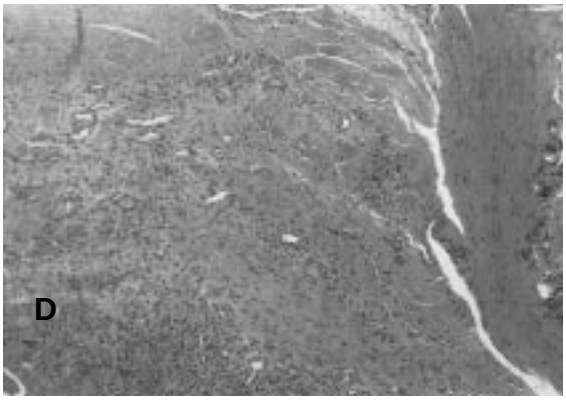

Fig. 4 Postcalcific stage. In the immediate vicinity of the calcific deposit (D) there is still evidence of phagocytic resorption. Farther away, fibroblasts elaborate new collagen (hematoxylin-eosin, ×25).

deposit and joint cavity. We believe they are indicated only in exceptional instances, as when a tear is suspected.

Radiographs not only allow confirmation of the absence or presence of calcium deposits, but also permit assessment of their extent, delineation, and density. DePalma and Kruper[15] described two radiographic types. Type I has a fluffy, fleecy appearance, with a poorly defined periphery. It is usually encountered in patients with acute pain. An overlying crescentic streak indicates rupture of the deposit into the bursa, which occurs only in this type. Type II is characterized by discrete, homogeneous deposits with uniform density and a well-defined periphery. This type is seen in subacute and chronic cases. DePalma and Kruper reported that in 52% of their patients, the calcification was seen as a single lesion.

Our observations confirm those of DePalma and Kruper.[15] During the formative phase, when pain is chronic or even absent, the deposit is dense, well defined, and homogeneous (Fig. 5, A). During the resorptive phase, which is characterized by acute pain, the deposit is fluffy, cloudlike, ill defined, and irregular in density (Fig. 5, B). Rupture of the calcific deposit into the bursa can occur only during the resorptive phase, because of the toothpastelike or creamy consistency. Radiographs show a crescentic radiodensity overlying the deposit (Fig. 5, C). In longitudinal studies, a change from a dense, well-delineated deposit into a fluffy, ill-defined deposit can be observed, but the contrary is never seen.

Most authors agree that radiographic evidence of degenerative joint disease is usually lacking in patients with calcific tendinopathies. This is true of patients in the fourth and fifth decades of life, when calcifying tendinitis peaks. It is not surprising, however, that acromioclavicular osteophytes were observed in three of our patients in the seventh decade.

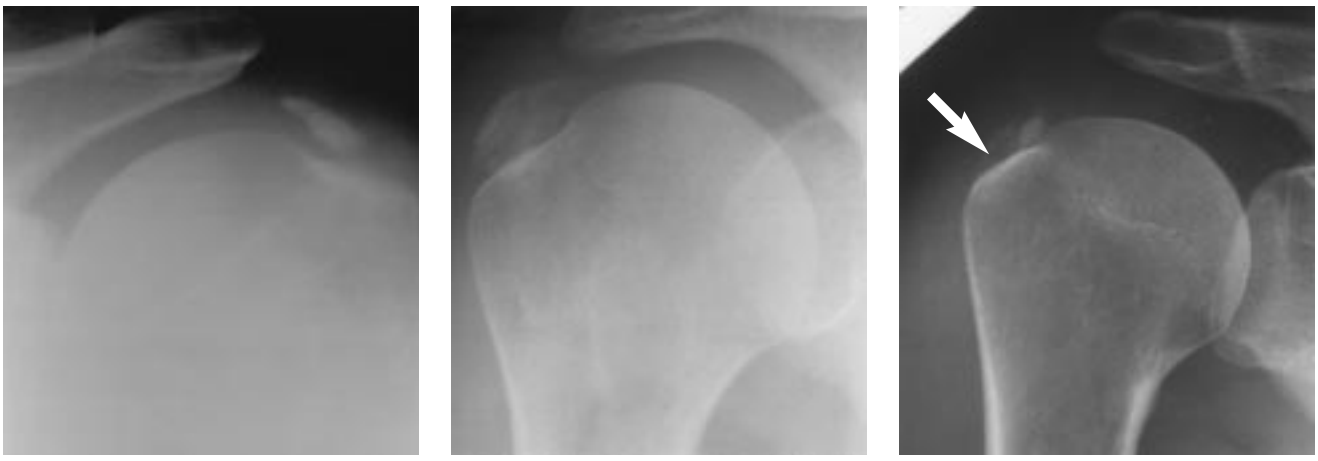

Fig. 5 **A,** In the formative phase, the deposit is dense, well circumscribed, and homogeneous. In the resorptive phase, the deposit is fluffy and ill defined **(B),** and the calcium that has ruptured into the subacromial bursa is seen as a crescentic shadow (arrow) overlying the intratendinous deposit **(C)**.

Calcifications seen in arthropathies have a quite different appearance. They are stippled and overlie the bone insertion and are always accompanied by degenerative osseous or articular changes. These calcium deposits must be clearly distinguished from reactive intratendinous calcifications.

In a study of 217 patients, Hartig and Huth[16] found sonography more sensitive than radiography in detecting calcium deposits. The deposit was visualized sonographically (as well as histologically) in 100% of cases but was depicted radiographically in only 90%. In addition, sonography permits more exact localization of the deposit without subjecting the patient to radiation.

Management

Distinguishing between the formative phase and the resorptive phase is important for proper management. During the formative phase, the pain is chronic or even absent. On radiographs, the deposit appears as a well-delineated, dense, and homogeneous calcification with a chalklike consistency. Histologic examination shows calcification around living chondrocytes. During the resorptive phase, the pain is acute; the deposit has a fluffy, ill-defined radiographic appearance; the consistency is creamy or toothpastelike; and the histologic features are compatible with phagocytic resorption.

Conservative Measures

The patient is instructed to do a daily program of exercises to avoid loss of mobility of the glenohumeral joint and to keep the arm in abduction as much as possible. The latter can be achieved by placing the arm on the backrest of a chair or, when lying down, by putting a pillow in the axilla. Application of moist heat is suggested when the symptoms are subacute. Although ultrasound is occasionally used in our physiotherapy department and some patients have commented on its beneficial effect, we have not seen any evidence that this accelerates the disappearance of calcium.

During the formative phase, when the symptoms are chronic, intrabursal injections of corticosteroids are appropriate only in the presence of an impingement syndrome. Needling of dense, homogeneous deposits has never been attempted by our group, nor has lavage been successful, presumably because of the chalklike consistency of the calcification.

During the resorptive phase, when the symptoms are acute or subacute and when radiographs indicate ongoing resorption, we attempt lavage of the deposit with the use of two large-bore needles and 2% lidocaine. The site of lavage is determined radiologically and clinically. In the outflow, liquid calcium particles can be recognized easily. Even when the lavage is negative, the multiple perforations of the site of deposition will decrease the intratendinous pressure and thus the pain. In a few instances, the lavage must be repeated.

Although we prescribe nonsteroidal anti-inflammatory drugs for 1 week, we have no proof of their beneficial action, nor could we find a relevant publication to support their use. The symptoms usually decrease after 1 week, at which point the patient is referred to the physiotherapy department. Patients are assessed clinically and radiographically every 4 weeks. We have never used ultrasound during this phase, nor do we recommend radiotherapy.

Extracorporeal Shock-Wave Therapy

Extracorporeal shock-wave therapy, which is now commonly employed for lithotripsy in urology, has recently been used for treating calcific deposits. Rompe et al[17] reported on a series of 40 patients who received 1,500 impulses to the shoulder area under regional anesthesia during a single therapy session. Fifteen patients had no improvement, but in 25 a partial or complete disappearance of the calcific deposit was observed. A similar experience was reported by Loew et al.[18] Of 20 patients with "chronic, symptomatic calcifying tendinitis," 14 experienced symptomatic improvement at the time of follow-up 12 weeks after the procedure. Local hematomas developed in 14 patients after this therapy. Thirty percent of the patients had an improvement of the Constant-Murley score; in 7, the deposit had disappeared completely.

This technique is still under investigation. Longer follow-up studies, a larger patient population, and reports from other centers are needed before it can be recommended.

Surgical Indications

Should conservative therapy fail during the formative phase, surgery may become necessary. During the resorptive phase, when natural mechanisms usually succeed in removing the deposit, surgery is very rarely indicated. De Sèze and Welfling[19] have stated that during the hyperalgic phase, the disease usually heals with the use of only supportive measures.

Gschwend et al[20] formulated three indications for surgery: (1) progression of symptoms, (2) constant pain interfering with activities of daily living, and (3) absence of improvement of symptoms after conservative therapy. Surgery,

whether performed arthroscopically or as an open procedure, should be done on an outpatient basis.

Arthroscopy

Ark et al[21] cited a number of advantages of arthroscopic surgery in treating calcifying tendinitis. These include a shorter rehabilitation time, the possibility of a better functional result, and a better cosmetic appearance than after open surgery.

The recommended arthroscopic technique is as follows: The patient is placed in a beach-chair position for surgery under general endotracheal anesthesia. Interscalene regional anesthesia is also instituted for postoperative pain relief. Posterior, anterolateral, and, if necessary, posterolateral portals are used.

Initially, the glenohumeral joint is explored through the posterior portal with a 4.5-mm 30-degree tilt arthroscope. A vascular injection pattern can sometimes be seen on the articular surface of the rotator cuff tendons, indicating an inflammatory response to the calcific deposit; this should be marked with a suture.

The scope is then introduced into the subacromial space. A working cannula is placed through the anterolateral portal, and the surface of the rotator cuff is palpated. The acromion is then inspected, as well as the coracoacromial ligament and the undersurface of the acromioclavicular joint. The rotator cuff is palpated for any hardening indicative of a calcific deposit. Needling can then be performed; during the resorptive stage, an 18-gauge spinal needle will usually fill with the calcific material when withdrawn from the tendon. Depending on the consistency of the deposit, the calcium might be extruded as a hard paste, or small flakes will be seen. The latter are present when the deposits are sharply demarcated radiographically, which is an ideal indication for arthroscopic surgery.

Once the deposit has been identified, we prefer to make a longitudinal incision in line with the direction of the fibers, avoiding deep penetrating cuts. In general, the use of a hook will best facilitate removal of the calcific material. Large curettes, knives, or tissue cutters should not be used, because of the risk of creating a rotator cuff defect.

Careful irrigation of the subacromial space is then performed, as the calcific debris can act as an irritating agent in the subacromial bursa. Subacromial decompression is performed only if there is an associated lesion, such as an obvious acromial beak or signs of subacromial impingement. There is no compelling published evidence that routine acromioplasty improves the surgical result. Once the subacromial space has been drained, a suction drain is inserted into the subacromial space.

The drain is removed 24 hours postoperatively, and range-of-motion exercises are begun, starting with pendulum exercises, followed by active assisted exercises after the third day, and progressing to active exercises as tolerated. An arm sling is usually not necessary except for patient comfort at night.

Open Procedures

It should be stressed that surgical removal is the exception and that it is indicated only when conservative measures have failed and symptoms interfere with work or activities of daily living.

Calcium deposits are removed under general anesthesia. The patient is in a supine position, and a sandbag is placed under the affected shoulder. We make sure that the side of the patient to be operated on is as close to the edge of the table as possible. The arm is draped free to permit full mobilization during surgery.

We use the skin incision recommended by Neer,[22] going from the acromion to the coracoid process. The deltoid fibers are bluntly separated. The deltoid muscle is not detached from the acromion. The bursa is then opened, the edges are retracted, and the bursal wall is inspected. The narrowness of the interval between the rotator cuff and the ligament is then tested, usually using the little finger, the introduction of which is made easier by longitudinal traction of the arm. While the finger is in place, the arm is rotated and lifted in a position between flexion and abduction. The undersurface of the acromion is also palpated. If the space between the ligament and the rotator cuff is tight, it is usually necessary to proceed with an anterior acromioplasty, although this is definitely the exception. External and internal rotation of the arm will permit inspection of the entire rotator cuff. If a bursal reaction is present, it is usually limited to a hyperemic reaction around the calcific deposit.

The tendon is incised in the direction of its fibers, and the calcific mass is removed by curettage. We then proceed with a limited resection of the frayed tendon edges, which are usually sites of calcium encrustation. Sometimes more than one deposit is present, necessitating separate tendon incisions. If no calcium can be seen during inspection, small incisions are made at the site of calcifications suspected on the preoperative radiographs.

After removal of the deposit, a copious lavage is performed. The shoulder is then moved through its full range of motion, and the tendon edges are approximated if necessary. A sling is applied after

surgery. The sling must be removed at least four times a day for pendulum and gentle passive range-of-motion exercises. The sling is discontinued entirely after 3 days, and active exercises are started. We encourage patients to keep the arm in abduction as much as possible. We have never used postoperative corticosteroid injections.

Summary

For optimal treatment results, it is not sufficient to diagnose calcifying tendinitis; one must also determine the stage of the disease. Chronic calcific tendinitis and acute calcific tendinitis, rather than being separate entities, are actually two phases of the same disease. If conservative management fails, surgery may become necessary, preferably in the formative phase of the disease.

References

1. Uhthoff HK, Sarkar K: Calcifying tendinitis, in Rockwood CA Jr, Matsen FA III (eds): *The Shoulder*. Philadelphia: WB Saunders, 1990, vol 2, pp 774-790.
2. Codman EA: *The Shoulder: Rupture of the Supraspinatus Tendon and Other Lesions in or About the Subacromial Bursa.* Boston: Thomas Todd, 1934, pp 178-215.
3. Herberts P, Kadefors R, Högfors C, Sigholm G: Shoulder pain and heavy manual labor. *Clin Orthop* 1984;191: 166-178.
4. Olsson O: Degenerative changes of the shoulder joint and their connection with shoulder pain: A morphological and clinical investigation with special attention to the cuff and biceps tendon. *Acta Chir Scand Suppl* 1953;181: 5-130.
5. Brewer BJ: Aging of the rotator cuff. *Am J Sports Med* 1979;7:102-110.
6. McLaughlin HL: Lesions of the musculotendinous cuff of the shoulder: III. Observations on the pathology, course and treatment of calcific deposits. *Ann Surg* 1946;124:354-362.
7. Mohr W, Bilger S: Morphologische Grundstrukturen der kalzifizierten Tendopathie und ihre Bedeutung für die Pathogenese. *Z Rheumatol* 1990;49: 346-355.
8. Perugia L, Postacchini F: The pathology of the rotator cuff of the shoulder. *Ital J Orthop Traumatol* 1985;11:93-105.
9. Remberger K, Faust H, Keyl W: Tendinitis calcarea: Klinik, Morphologie, Pathogenese und Differential diagnose. *Pathologe* 1985;6:196-203.
10. Sarkar K, Uhthoff HK: Ultrastructural localization of calcium in calcifying tendinitis. *Arch Pathol Lab Med* 1978; 102:266-269.
11. Uhthoff HK, Sarkar K, Maynard JA: Calcifying tendinitis: A new concept of its pathogenesis. *Clin Orthop* 1976;118: 164-168.
12. Lippmann RK: Observations concerning the calcific cuff deposit. *Clin Orthop* 1961;20:49-60.
13. Sengar DPS, McKendry RJ, Uhthoff HK: Increased frequency of HLA-A1 in calcifying tendinitis. *Tissue Antigens* 1987;29:173-174.
14. Archer RS, Bayley JIL, Archer CW, Ali SY: Cell and matrix changes associated with pathological calcification of the human rotator cuff tendons. *J Anat* 1993;182:1-12.
15. DePalma AF, Kruper JS: Long-term study of shoulder joints afflicted with and treated for calcific tendinitis. *Clin Orthop* 1961;20:61-72.
16. Hartig A, Huth F: Neue Aspekte zur Morphologie und Therapie der Tendinosis calcarea der Schultergelenke. *Arthroskopie* 1995;8:117-122.
17. Rompe JD, Rumler F, Hopf C, Nafe B, Heine J: Extracorporal shock wave therapy for calcifying tendinitis of the shoulder. *Clin Orthop* 1995;321: 196-201.
18. Loew M, Jurgowski W, Mau HC, Thomsen M: Treatment of calcifying tendinitis of rotator cuff by extracorporeal shock waves: A preliminary report. *J Shoulder Elbow Surg* 1995;4: 101-106.
19. de Sèze S, Welfling J: Tendinites calcifiantes. *Rhumatologie* 1970;22:45-50.
20. Gschwend N, Scherer M, Löhr J: Die Tendinitis calcarea des Schultergelenks (T. c.). *Orthopade* 1981;10:196-205.
21. Ark JW, Flock TJ, Flatow EL, Bigliani LU: Arthroscopic treatment of calcific tendinitis of the shoulder. *Arthroscopy* 1992;8:183-188.
22. Neer CS II: Impingement lesions. *Clin Orthop* 1983;173:70-77.