

Calcium Deposits in the Hand and Wrist

Valdet Nikci, MD

Christopher Doumas, MD

Abstract

Calcium, or calcific, deposition disease in the form of acute calcific peri-arthritis of the hand and wrist is an uncommon entity that may be confused with more common crystalline or inflammatory arthropathies as well as infection. It is important for the clinician to be aware of this disease process and to include it in the differential diagnosis of patients presenting with acutely painful, focal inflammation of the hand or wrist. Nonsurgical management is often sufficient; however, considering the self-limited nature of the disease, accurate diagnosis is essential to avoid unnecessary antibiotic or surgical treatment.

Etiology

Calcific deposition disease has been variously described in the literature as calcific peri-arthritis, calcific tendinitis, calcareous tendinitis, peri-arthritis calcarea, peritendinitis calcarea, and hydroxyapatite rheumatism.¹⁻³ Typically, acute presentation of pain is limited to one joint or to the area of one joint and is frequently associated with radiographic findings of calcifications at the area of tenderness. Calcium deposition is usually idiopathic, but it has been associated with several systemic conditions such as scleroderma and renal failure.

The condition most commonly affects the shoulder; the hand and wrist are affected in only about 2% of patients.⁴ The most common location within the hand and wrist is the flexor carpi ulnaris (FCU) tendon insertion; the flexors and extensors of the fingers are also common sites (Figure 1). Multiple different locations have been described, however, including the interphalangeal (IP) and metacarpophalangeal

(MCP) joints of all five digits, as well as the dorsal side of the wrist.⁵⁻¹⁰ In studies by Carroll et al,⁷ 37 of 100 patients had a calcium deposit at the insertion of the FCU on the pisiform. Yelton and Dickey² described 73 of 107 patients with symptoms about the wrist, including the FCU insertion site, although not all of the exact locations of the symptoms were described in detail. In a study by Kim and Park,¹¹ 12 of 30 patients had a calcium deposit at the FCU insertion; one patient had a calcium deposit in the extensor carpi ulnaris tendon insertion. The other 17 patients had deposits distributed throughout the MCP and IP joints of all five digits.

Women are more commonly affected than men, with several series having only female patients.^{5,12} The female:male ratio in the literature ranges from 2:1 to 5:1.^{5,11} We are aware of only one study with >10 patients in which most of the participants were male.² The average reported age of presentation is 45 years, with a wide distribution.^{7,11,13} Few reports cite children with this condition, with the youngest child aged 3

From the Department of Orthopaedic Surgery, Rutgers–Robert Wood Johnson Medical School, Somerset, NJ.

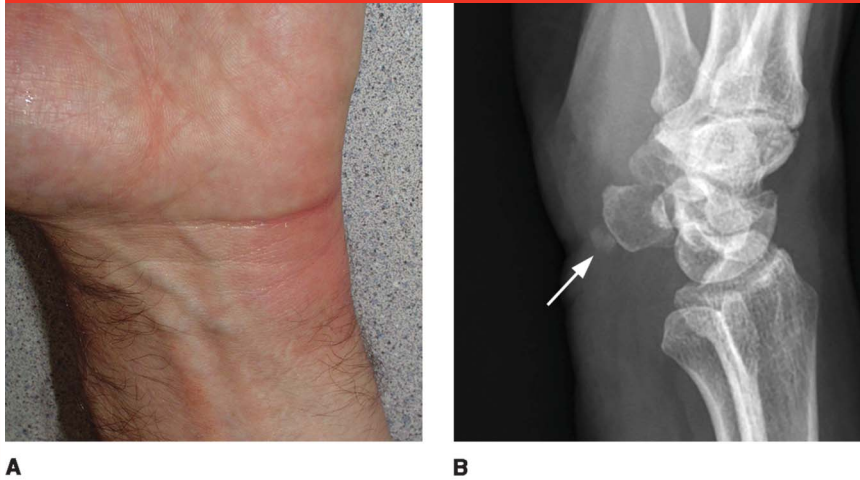
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Figure 1



A, Clinical photograph demonstrating erythema at the site of calcification of the flexor carpi ulnaris tendon. **B**, Oblique radiograph of the wrist demonstrating a 5-mm calcium deposit anterior to the pisiform.

years.¹⁴⁻¹⁶ Children tend to present in a similar manner as adults; thus, treatment algorithms are similar.

Clinical Presentation

The most common clinical manifestations of the disease include an acute and rapid onset of localized erythema, swelling, warmth, and severe tenderness. The pain is usually localized to a single area of the hand or wrist in which the patient demonstrates severe point tenderness. Typically, range of motion around the affected site is notably limited secondary to pain^{1,5,6,17} (Figure 2, A). Most patients are afebrile; however, a mild fever has been reported in some patients.^{5,15} Lymphangitis has been described in some patients with pain localized to the wrist; typically there is no associated lymphadenopathy.¹⁸

Many patients seek emergency care because of intense pain. In a series of 100 patients, Carroll et al⁷ reported that 26 patients presented to the emergency department for immediate care; the average length of time for patients seeking treatment is 3.5 days. In a series of 10 patients, 7

presented to the emergency department for acute onset of night pain.⁵ Most patients do not describe any history of trauma; however, several studies have shown that up to one third of patients report a recent traumatic event or a history of repetitive use of the hand.⁷ Patients frequently have a gradual reduction in symptoms over the first 4 to 7 days, with complete resolution of symptoms usually occurring within 3 to 4 weeks.¹⁷

Laboratory Values

The white blood cell count and erythrocyte sedimentation rate may occasionally be elevated; however, these values are typically normal.^{2,5,8} Other basic laboratory values, such as serum calcium, phosphorus, glucose, alkaline phosphatase, urea, and uric acid, are also usually within normal limits.^{5,8,17}

Negative culture results are generally found following the aspiration of calcium deposits, in conjunction with negative results for crystals of gout or pseudogout.^{6,7,19} Gross description of the aspirate is best described as chalky

white or pasty white fluid^{6,8,20} that has the consistency of toothpaste; chemical analysis reveals calcium hydroxyapatite crystals.^{5,7,8,12,17,21} These deposits do not show birefringence when viewed under polarized light.

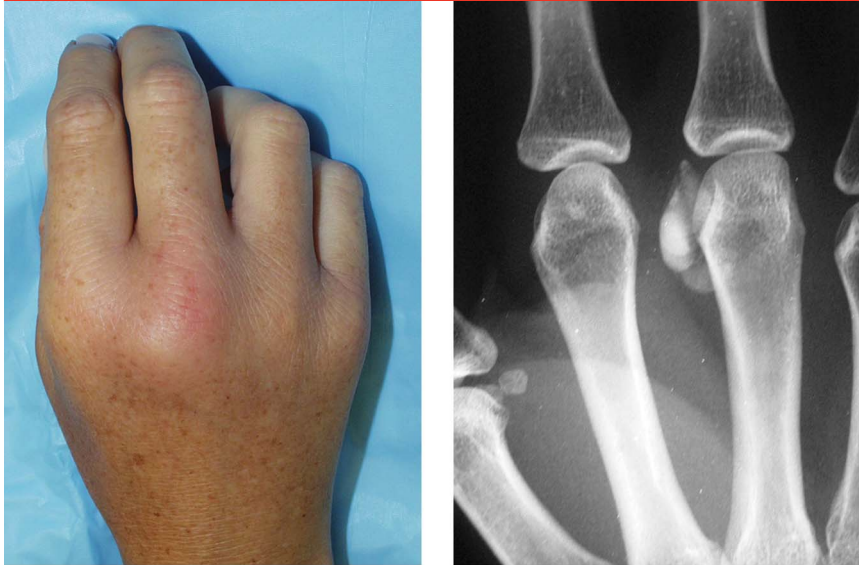
Imaging

To determine a proper diagnosis, plain radiographs are used to detect calcific periarthritis.⁶ The calcium deposits are typically dense, homogeneous, and amorphous without any cortical or trabecular pattern; these findings distinguish them from heterotopic ossification. The deposits are usually round or oval in shape and range in size from 2 to 10 mm^{1,3,5} (Figure 2, B). Dramatic changes in their appearance commonly occur over time.^{1,6,13}

Generally, orthogonal views of the joint are used to sufficiently identify the calcium deposits; however, if the initial radiographs do not identify any pathology, oblique views may be used at the physician's discretion. Deposits in the carpal bones tend to be more difficult to identify than deposits in the digits.⁸ Carroll et al⁷ reported that obtaining radiographs in the proper planes is critical to the diagnosis and should be specifically ordered depending on the point of maximal tenderness. In their series, only 73 of 100 radiographs were correctly interpreted. Of the radiographs that were incorrectly interpreted, 20 indicated there was no abnormality, 5 were misdiagnosed as chip fractures or secondary ossicles, and 2 had comments regarding the calcium deposits but provided no impression or diagnosis.

The use of ultrasonography to identify calcium deposits is also described.²² The utility of this method for the hand has not been well studied. It is possible that ultrasonography may detect subclinical calcium deposits, but it is largely operator

Figure 2

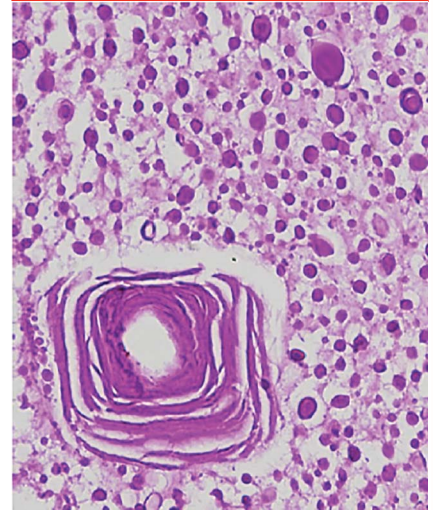


A

B

Clinical photograph (A) and AP radiograph (B) demonstrating a calcium deposit adjacent to the metacarpal head.

Figure 3



Histology demonstrating subtle (upper right) and conspicuous (low center) psammoma-like concentrically layered particles indicating deposition of hydroxyapatite (hematoxylin and eosin stain, original magnification $\times 400$).

dependent. Smaller deposits may be difficult to see radiographically; however, CT or MRI is usually not required. Should advanced imaging be obtained, Chung et al²³ reported that MRI does not show bone destruction or bone edema; however, it is useful for detecting soft-tissue inflammation and edema within the surrounding soft tissues. Anderson et al²⁴ reported that calcifications may appear as signal voids on MRI. Moyer et al⁸ used bone scans to identify increased uptake in the involved areas.

Histology

Calcium hydroxyapatite is the most common form of calcium found in human bone and it is thought to be the usual pathologic deposit that occurs in acute calcific periarthritis; however, other forms of calcium are described, such as calcium octaphosphate.³

When histology is available, it demonstrates fibrous or fibroadipose tissue with inflammatory cells and calcium deposits surrounded by giant

cells and/or fibrocollagenous tissue.^{5,8} Gravanis and Gaffney²⁵ reported that hydroxyapatite is deposited into psammoma-like bodies that incite a significant inflammatory reaction, involving mostly neutrophils. Lehmer and Ragsdale²⁰ described psammoma-like particles in an amorphous basophilic background. Many of their specimens are associated with a histiocytic infiltrate; however, only two demonstrated significant neutrophilic infiltrate (Figure 3).

Pathogenesis

Theories have been developed as to why these calcifications are formed, with some of the data coming from examination of the calcific deposits occurring in the shoulder.^{26,27} Uthoff²⁷ described multiple phases of calcium deposition that are characterized as precalcific, formative, resorptive, and healing. Initially, metaplasia of the tendon or pericapsular tissue occurs, creating fibrocartilaginous tissue. This may be

caused by vascular or mechanical factors that create a site of either poor blood supply and/or decreased oxygen tension; this triggers the metaplasia, which then has a propensity for mineralization via calcium deposition. The deposition is mediated by chondrocytes. A rest period then follows during which the calcific deposits are finalized, and lastly, there is a resorptive phase. Both the deposition and resorption phases are cellularly mediated. Over time, phagocytes infiltrate and accumulate around the calcium deposits and facilitate the resorption process. It is typically this resorptive phase that causes the considerable pain and the usual symptoms that patients experience. Resolution of symptoms is typically associated with complete or nearly complete disappearance of the calcium deposits.^{26,27}

Another prevailing theory is that this particular form of deposition is caused by dystrophic calcification, or calcification that occurs within degenerating or necrotic tissues, as opposed to viable tissue.¹⁷ Local

Figure 4

AP (A) and oblique (B) radiographs of the hand demonstrating a well-circumscribed, homogenous calcific 10-mm mass (black arrow) with surrounding amorphous and disorganized calcifications (white arrows) involving the soft tissues of the metacarpophalangeal joints. AP (C) and oblique (D) radiographs obtained 4 days later demonstrate resolution of the 10-mm calcific mass and some reduction in the amount of amorphous calcifications.

tissue hypoxia or necrosis within the tendon or soft tissue may lead to metaplasia, fibrocartilage formation, and subsequent deposition of calcifications.^{1,7,12} Poor blood supply or decreased oxygen tension are common to the theories, and some authors believe that vascular or mechanical factors, such as trauma, may cause this; however, only about one third of reported cases are associated with trauma.

Based on studies of calcific tendinitis of the shoulder, Yosipovitch and Yosipovitch⁵ believed that the calcium deposits form weeks or months before an acute rupture; this action then incites an inflammatory response once the calcium substance is released into the tenosynovial space. This action is in accordance

with the observation that an acute inflammatory reaction may be elicited by the injection of crystalline materials into synovial spaces.¹⁰ Yelton and Dickey² described several patients with asymptomatic calcium deposits, although they believed asymptomatic deposits are rare in the hand. In one patient, symptomatic peri-arthritis developed after a traumatic event. This acute episode is self-limited, and eventually macrophages resorb the calcific areas. Usually the deposits undergo resorption within a few weeks.^{8,12}

Management

The management of calcific peri-arthritis should be nonsurgical. Reports

have shown that the condition is self-limited and is best treated with nonsteroidal anti-inflammatory medications, with activity modification or splinting during the acute period (Figure 4). Lidocaine injections with and without steroids have been shown to provide rapid pain relief.⁸

Yosipovitch and Yosipovitch⁵ injected 7 of 10 patients with a combination of steroid and lidocaine; all 7 reported immediate relief of pain. Cooper¹⁹ and Greene and Louis¹⁷ stressed the importance of accurate placement of the needle. Attempting aspiration and injection concurrently may help to disperse the calcification, thus speeding the process of dissolution. The degree of pain relief experienced may be due, in part, to the mechanical action of the needle itself.⁸ Carroll et al⁷ injected patients with procaine, with and without steroid, and reported no difference between the two groups; relief of acute pain was seen within hours, with patients reporting complete resolution of pain in 6 days on average. For their patients, surgical excision resulted in pain relief within 24 hours, whereas patients receiving no treatment had resolution of pain within 20 days. Thus, the authors posited that the indications for surgical intervention are limited. In a series of 107 patients, Yelton and Dickey² did not perform surgery on any of their patients; most patients who received injections of procaine reported excellent results. Surgery is typically reserved for patients with recurrent or persistent lesions that do not resolve with nonsurgical management.

Complications

The most common problem encountered with calcium deposition of the hand and wrist is that it has a high rate of misdiagnosis.^{1,8} This leads to a delay in diagnosis as well as risks associated with other treatments that

are initiated. Misdiagnosis may be largely due to the rarity of the condition as well as its initial presentation being similar to other, more serious conditions, such as infection. However, because the correct diagnosis is often made by hand surgeons, especially in the acute period,⁸ it is possible that the high rate of misdiagnosis is mostly the result of a lack of familiarity with the disease process. Given the frequency with which the condition is misdiagnosed, there have been many reports of more extensive or invasive treatment modalities. These situations can be avoided if the clinician is aware of the disease process and includes it in the differential diagnosis.

In the study by Carroll et al,⁷ only 46% of patients were correctly diagnosed at the time of initial presentation. Even with high rates of misdiagnosis, most patients have excellent outcomes once appropriate treatment is started. In a small case series, two of three patients were initially misdiagnosed, with one patient undergoing a wrist aspiration and receiving antibiotics; however, all three patients underwent treatment consisting of splinting and anti-inflammatory medications and experienced complete resolution of symptoms within 7 to 10 days of the initial onset of pain.¹ Lehmer and Ragsdale²⁰ evaluated 12 histologic specimens taken from the operating room, with only one correct diagnosis at the time of surgery; the most common diagnoses were mass, lesion, or cyst.

Recurrence is rare when the disease is not associated with systemic conditions. Most series reported no recurrence at 1 year follow-up.^{1,5,6} When there is a recurrence, the predilection for recurrence is at the FCU insertion onto the pisiform. Phalen²⁸ reported performing surgical excision in two of seven patients, with recurrent symptoms at the FCU insertion. Kim and Park¹¹ reported 4

of 30 patients who had recurrent symptoms within 1 year after initial presentation. All four patients had initial and recurrent calcium deposits at the FCU insertion on the pisiform, and all four received steroid injections regardless of the initial treatment. One of the four patients underwent surgical excision because of persistent symptoms.

Shields et al²⁹ reported one recurrence in a patient with idiopathic acute calcific periarthritis of the MCP joint of the thumb at the insertion of the flexor pollicis brevis. The patient initially sustained a displaced avulsion fracture of the radial collateral ligament of the thumb; subsequent development of acute calcific periarthritis occurred >1 year later. The patient was treated with local anesthetic, nonsteroidal anti-inflammatory drugs, and immobilization, with good results, but experienced several recurrences over the course of a year. All management throughout was nonsurgical.

Few reports have noted patients with recurrences of calcium deposits at any of the MCP or IP joints of the other digits.³⁰ It is not known if this observation is due to a specific risk factor for calcium deposits at the FCU insertion or whether recurrence is simply related to the higher number of deposits seen at this site. Recurrence is common when calcium deposits develop within tendons in other areas of the body;^{11,30} therefore, it is likely that peritendinous pathology has some predilection for recurrence compared with that of periarticular deposition. Although local recurrence is rare, several studies have shown that recurrences of calcium deposition can occur in different areas of the body; this is suggestive of an underlying systemic disorder.^{13,21} Most patients do not experience recurrence; therefore, it is likely that most calcium deposits are isolated and unrelated to systemic disease.

Other Calcific Deposits

It is important to delineate acute calcific periarthritis from other non-inflammatory forms of calcium deposition. Tumoral calcinosis is a condition with similar presenting symptoms. It is not commonly seen in the hand; however, when it occurs, the presenting features may be similar to those described for acute calcific periarthritis.^{31,32} African-Americans are most commonly affected. Patients typically have pain and swelling about a single area of the hand. One of the features that distinguishes tumoral calcinosis from calcific periarthritis is the duration of symptoms. Tumoral calcinosis tends to last longer and is refractory to non-surgical management. Many patients have symptoms for a longer period of time before seeking treatment than do patients with calcific deposition disease, and they usually report no history of trauma. Because these deposits often do not resolve with medical therapy, they are typically treated with surgical excision, which is usually curative.

On gross examination, the chalk-like material described earlier is not usually seen. Instead, examination reveals a dense mass of calcification surrounded by fibrocartilage, usually larger than such areas found in calcific periarthritis. The histopathology of both conditions is similar in that there is calcific material formed into psammoma-like bodies; however, tumoral calcinosis has characteristic fibrohistiocytic nodules, cavity formations, and hyaline stroma that usually are not seen in calcific periarthritis.³¹

Calcium pyrophosphate dihydrate (CPPD) deposition disease, or pseudogout, has presenting features similar to those of acute calcific periarthritis. CPPD deposition has been extensively studied and reviewed in the literature; therefore, we

Figure 5

AP radiograph of the wrist demonstrating extensive calcification.

will mention it only briefly here. The calcium deposits are usually distinguished radiographically because they tend to be more linear and less rounded or oval shaped.³ The typically associated chondrocalcinosis and degenerative changes seen in the bones and joints of CPPD deposition disease are generally not seen in calcific peri-arthritis.¹

Associated Conditions

A review of each associated syndrome is beyond the scope of this article; therefore, the following information discusses the systemic conditions that are associated with calcific peri-arthritis of the hand and wrist.

Scleroderma

Patients with scleroderma frequently have subcutaneous and periarticular calcifications; however, the incidence of symptomatic peri-arthritis is not necessarily increased. When the calcifications do occur, they are more likely to occur in patients with CREST (calcinosis, Raynaud phenomenon,

esophageal dysmotility, sclerodactyly, and telangiectasia) syndrome rather than in patients with diffuse systemic sclerosis, due to the increased incidence of calcinosis in patients with CREST.³³ The overall incidence of calcinosis of the hand in patients with scleroderma is reported to range from 12% to 40%.^{22,34,35} Some studies suggest that the longer the patient has the disease, the more likely it is that he or she has calcium deposition; however, it is not clear whether this deposition results in an increased incidence of symptomatic calcific peri-arthritis.

Patients with scleroderma typically present with polyarticular deposits with polyarthritis. These deposits are not limited to the hand and wrist; however, when treated nonsurgically, patients with scleroderma are more likely to have incomplete resolution or recurrence, especially at previously symptomatic sites. Surgical excision of calcium deposits in patients with scleroderma is rarely reported. Melone et al³⁶ described a series of 70 patients who underwent 272 procedures in the hand. Only 12 of those surgeries were for the excision of calcium deposits. The authors described isolated deposits that were symptomatic with more diffuse calcinosis; some areas were more painful and more prone to ulceration. They recommended removing isolated, well-defined deposits; however, performing an extensive débridement of all areas of diffuse calcium deposition was not advised because of the risk of potential damage to the vascularity of the digits.

Hemodialysis

Patients with chronic renal failure, and especially those who undergo hemodialysis, are at an increased risk for calcium deposition disease. As in patients with scleroderma, these

patients are at a particularly higher risk for the development of multiple sites of calcification, in addition to having a propensity for the deposits to be somewhat larger than in the idiopathic form of the condition.¹⁰ The reported prevalence in this patient population is between 0.5% and 3%, with the hand and wrist accounting for a small portion of that number.³⁷ It is important to make a proper diagnosis in patients who undergo hemodialysis because an incorrect diagnosis of infection, for example, may result in patients being removed from the transplant list.³⁸

Patients on hemodialysis often present with similar reports of acute pain, erythema, swelling, and difficulty with range of motion, while remaining afebrile. Inflammatory markers are also typically normal. Some patients may present with an increased serum phosphorus level that may play a role in the deposition of calcium phosphate salts. Radiographic findings are also similar to those seen in patients without systemic conditions. The nature of the deposits themselves are somewhat varied. They can be smaller, with toothpaste-like consistency, and yet grow rapidly and over time develop thick fibrous capsules akin to tumoral calcinosis.³⁸ It is not clear whether patients with renal failure can develop both calcific peri-arthritis and tumoral calcinosis or whether these conditions represent a spectrum of the same condition. However, given the systemic effects of chronic renal failure, it is possible that the same disease process leads to varying presentations of calcium deposits.

The preferred treatment of these patients is surgical excision; however, complete removal of the deposit is often impossible secondary to the extensive infiltration. As such, recurrence is usually common after incomplete excision (Figure 5).

Inherited Conditions

Inherited conditions that present with calcific periarthritis have been reported.^{39,40} Hypophosphatemia, a heritable disorder, is known to present with acute calcific periarthritis of several different areas of the body, including the hand and wrist. These patients fit the same symptomatology that appears in patients with systemic conditions; the patients tend to have multiple rather than isolated sites of involvement that are usually recalcitrant to the treatments used for idiopathic disease.

Caspi et al³⁹ reported on three siblings with low levels of serum alkaline phosphatase; the diagnosis was suggestive of hypophosphatasia⁴⁰ but was not confirmed. The most severely affected patient had multiple episodes of polyarticular acute periarthritis lasting about 1 week, with frequent recurrences that increased over time. Radiographs demonstrated periarticular calcifications seen as lumps near various joints that occasionally preceded the attacks. This information lends support to the theory that the calcifications may develop before the onset of acute inflammation.

Guañabens et al⁴⁰ reported on a series of three sisters with manifestations of hypophosphatemia characterized by acute calcific periarthritis. The patients had acute and recurrent cases of calcific periarthritis in the shoulders, elbows, wrists, and hips. Their symptoms were typically refractory to the usual anti-inflammatory medications. Subsequent testing led to the discovery of a single gene mutation that caused the hypophosphatasia.

Summary

Calcium deposition disease of the hand and wrist is an uncommon entity that usually resolves spontaneously over a period of 1 to 2 weeks, or more quickly with injection of

local anesthetic. Given the high rate of misdiagnosis, it is important for the physician to be aware of this disease process to optimize treatment and avoid unnecessary interventions. Although the pathogenesis is not fully understood, the clinical presentation and the radiographic findings are fairly similar among patients and should lead the clinician to the correct diagnosis. Calcium deposits associated with systemic conditions such as scleroderma or chronic renal failure are more likely to be widespread and less likely to resolve spontaneously.

References

Evidence-based Medicine: Levels of evidence are described in the table of contents. In this article, references 22 and 35 are level III studies. References 1, 2, 4-8, 10-12, 18-20, 27, 31, 33, 34, 36, 37, 39, and 40 are level IV studies. References 3, 9, 13-17, 21, 23-26, 28-30, 32, and 38 are level V expert opinion.

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