SURGICAL EXCISION AND REPAIR OF CALCIFICATIONS OF THE TENDO ACHILLIS

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Soft tissue calcification is the process by which these tissues become "hardened as the result of precipitates or larger deposits of insoluble salts of calcium".[1] These calcifications may be differentiated from secondary ossification centers as they lack trabecular pattern organization.

ETIOLOGY

Black and Kanat[2] provided an excellent overview of soft tissue calcifications dividing the various calcific entities into three distinct pathologic processes - metastatic calcification, calcinosis, and dystrophic calcification. They described metastatic calcification as that which occurs in normal tissues when the calcium-phosphorus levels are elevated. Hyperparathyroidism, neoplasms, milk-alkali syndrome, hypervitaminosis D, and tumoral calcinosis were all classified as metastatic forms of calcification.

Calcinosis was described as calcification found in subcutaneous tissues, skin, and connective tissues with overall normal metabolism. Calcinosis disorders include calcinosis universalis, calcinosis circumscripta, scleroderma, dermatomyositis, and systemic lupus erythematosus.

Dystrophic calcification was described as that occurring in the presence of normal metabolism in damaged or devitalized tissues. Ehlers-Danlos syndrome, pseudoxanthoma elasticum, arteriosclerosis obliterans, venous calcifications, crystal deposition disorders, and calcifications resulting from neurological disorders are all considered dystrophic calcifications. Fiamengo et al.[3] concluded that calcifications of the tendo Achillis were dystrophic forms of calcification as the salt deposits resulted from either degeneration of connective tissue due to trauma or as a result of a primary crystalline deposition disease.

Lagergren and Lindholm conducted an angiographic and microangiographic study of the vascular distribution of the tendo Achillis and concluded that an area of decreased vascularity exists from approximately 2-6 cm proximal to the distal tendinous insertion. A direct correlation was reported between hypovascularity and tendon calcification. Physical, chemical or thermal trauma can result in the degeneration and devitalization of the connective tissue in this area. Physical trauma to the tendo Achillis occurs routinely by repeated microtrauma from shoegear, and is frequently the area traumatized when one is kicked or struck from behind. In other instances, the trauma may come in chemical form via steroid or other injections, or in thermal form via local heat or topical steroidal preparations. When soft tissues are damaged or devitalized in this manner, the metabolic rate is lowered. When the vascularity to the area is poor, this process occurs faster. This results in decreased levels of tissue oxygen and increased levels of tissue carbon dioxide causing an increase in tissue pH. In the presence of this elevated pH or alkaline state, calcium and phosphorous salts precipitate out of solution. In this manner, dystrophic calcification

of the tendo Achillis can occur secondary to many forms of trauma.

A tendo Achillis calcification may also be the result of a primary crystalline deposition disease. These diseases include gout, pseudogout, calcific periarthritis, and osteoarthritis. Generally, these diagnoses are made after surgical excision of the calcific mass and pathologic examination. Gout demonstrates pathognomonic monosodium urate crystals; pseudogout presents calcium pyrophosphate crystals and both calcific periarthritis and osteoarthritis are associated with calcium hydroxyapatite crystals.

CLINICAL PRESENTATION

Calcifications of the tendo Achillis are reportedly a rare condition. Ghormley reported a case in 1938 and reviewed the twenty-one previously published cases. These calcifications were described in association with both painful and painless Achilles tendons. Fiamengo et al.[3] performed a radiographic evaluation of clinically symptomatic and asymptomatic heels and found a tendo Achillis calcification to be present in 3.13 percent of asymptomatic heels and in 25 percent of symptomatic heels. Frequently, these calcifications are associated with a retrocalcaneal spur, a posterior calcaneal step, or a prominent posterosuperior calcaneal prominence. These deformities most likely contribute to the pathologic mechanism causing traumatic dystrophic tendo Achillis calcification.

The clinical presentation in the symptomatic patient is very similar to that of the patient suffering from Haglund's disease. The patient may complain of pain, tenderness, erythema, and edema. Typically, the patient experiences a dull aching pain with localized tenderness near the insertion due to Achilles tendinitis and peritendinitis often combined with retrocalcaneal bursitis. Pain is aggravated by passive and active ankle range of motion, and a decrease in the range of passive ankle dorsiflexion (i.e., ankle equinus) may be noted on clinical examination. Occasionally a palpable "hardness" or "thickening" in the area of the Achilles tendon just proximal to its insertion into the calcaneus may be noted, especially when compared to the contralateral side. Patient ages range from the early twenties to the seventies, and younger patients are usually involved in athletics (particularly distance running, basketball, and tennis). Although the condition is probably equally prevalent in men and women, women are generally more symptomatic and are more frequently seen with this condition. On rare occasions, a distal tear of the Achilles tendon through areas of calcification may initiate the patient's symptom complex.[3]

Radiographically, a standard lateral view will demonstrate the calcification. Soft tissue radiographic attenuation or xerography, although unnecessary, may be utilized again in the lateral projection for excellent delineation of the calcific mass. Calcifications occur most commonly in the area just proximal to the tendo Achillis insertion and will lack a cortex, a medulla, or trabecular pattern. Care must be taken to avoid misdiagnosis of arterial or venous calcification in the tarsal canal as a tendo Achillis calcification. These calcifications occur along their respective vessels and are anterior to the Achilles tendon.

CONSERVATIVE TREATMENT

Modes of nonsurgical treatment have included oral non-steroidal anti-inflammatory drugs (NSAIDs), local steroidal injections (although these should be limited due to their contribution to the pathologic process and potential side effects), heel lifts in the shoes to eliminate the traumatic effect of the heel counter on the symptomatic area, orthoses to help control biomechanical influences, and immobilization. Birdsell et al.[3] demonstrated a significantly lowered metabolic rate in tendon when immobilized. Stretching exercises of the posterior crural musculature may be helpful if muscular equinus has been found to be a contributing factor.

SURGICAL CONSIDERATIONS

Several anatomic and functional considerations must be addressed when surgical excision of a calcific mass from the tendo Achillis is contemplated. First, if a retrocalcaneal exostosis and/or a retrocalcaneal spur is present, as is usually the case, the surgical plan generally must be adjusted to alleviate their pathologic influence. Secondly, the amount of tendo Achillis dissection must be kept to a minimum. Finally, the incisional

approach must allow adequate exposure and yet avoid critical anatomical structures.

It has been the authors' experience that when a Haglund's deformity and/or tendo Achillis spur are present along with a tendo Achillis calcification that they must also be excised. As stated earlier, the retrocalcaneal spur, posterior calcaneal step, and posterosuperior calcaneal prominence are believed to contribute to the traumatic mechanism of dystrophic calcification by altering the biomechanical attitude of the Achilles tendon. Surgical exposure, therefore, must allow resection of these associated deformities.

The amount of tendo Achillis dissection must be kept to a minimum to decrease the postoperative convalescence time. Many anatomic misnomers are found in relationship to the insertion of the tendo Achillis. Sarrafian reported the tendon's insertion as occurring over the distal one-half of the calcaneus. This is in direct contradiction to the classical anatomic descriptions of its insertion into the middle one-third of the calcaneus. The authors tend to agree with Sarrafian and have found that the tendons' fibers "fan out" over the distal one-half of the calcaneus from medial to lateral. We have found many insertional fibers along the medial and lateral sides of the calcaneus. Generally, exposure to the retrocalcaneal area can be achieved with only minimal reflection and dissection of tendo Achillis fibers. When the calcific mass is excised from the tendo Achillis further weakening of the tendinous structure will occur. This can be a potential problem in older patients where tendinous healing can be quite slow. There is a direct graphic relationship between age and recovery time following tenotomies of the tendo Achillis. Much the same can be expected following surgical reflection of the tendon. Therefore, when large amounts of the tendon are reflected at the time of surgery, especially in older patients, the expected convalescence period will be longer.

Finally, the surgical approach must allow adequate visualization of all structures to be surgically addressed. Several incisional approaches have been utilized. A direct posterior incision allows adequate visualization of the tendon but may prevent sufficient visualization of associated calcaneal prominences. Further, the posterior scar is more likely to present postoperative sequelae from direct shoe pressure. A lateral or curvilinear incision allows much better visualization of the calcaneal landmarks along with adequate tendinous visualization. Care must be taken to avoid the sural nerve with all laterally based incisions.

SURGICAL TECHNIQUE

Surgical approaches for the correction of Achilles tenocalcinosis and associated posterior calcaneal exostoses vary depending upon the primary site of pathology, medial to lateral, and whether or not the patient can be operated on in the prone position. If the patient must be kept supine, a single 4-8 cm linear or smooth curvilinear lateral incision is utilized. Preferably, the patient can be placed in the prone position and a 4-8 cm curvilinear incision can be utilized. The incision begins superomedially along the medial aspect of the tendon, sweeps laterally in transverse fashion across the tendon, and ends inferolaterally at the level of the insertion of the tendo Achillis. A midthigh tourniquet may be used if desired but is not necessary.

The incision is carried deep through subcutaneous tissue taking care to avoid the sural nerve. If the sural nerve is encountered it is gently retracted with a penrose drain. The subcutaneous tissue is bluntly freed from the underlying deep fascia. The deep fascia, calcaneal periosteum, and peritenon of the tendo Achillis are then incised along the lateral aspect of the tendon's insertion. At this point, the calcific mass can usually be palpated within the tendinous architecture. A sagittal plane, longitudinal midline incision can then be utilized to split the tendon. This produces equal medial and lateral halves of the tendon that remain attached to the calcaneus distally. The halves are reflected side-to-side allowing extraction of the intratendinous calcification. Deepening the tenotomy allows excision of the inflamed retrocalcaneal bursa, release of any paratendinous fibrosis, and exposure of the retrocalcaneal exostoses.

If adequate visualization cannot be obtained through the posterior longitudinal tendinous incision, the tendon may be retracted medially revealing any associated retrocalcaneal spur or exostosis. The osseous pathology can be resected utilizing power or hand instrumentation. In the rare event that satisfactory visualization has not

been obtained, then the tendo Achillis is carefully transected in transverse fashion until such visualization is achieved. This stepwise approach to tendinous reflection is utilized, as care must be paid to preserve as much of the tendon's integrity as possible.

Any tendinous defects can then be reapproximated utilizing an absorbable or nonabsorbable suture. If the tendon has been reflected it can be reapproximated to the lateral calcaneal periosteum utilizing either absorbable (e.g., 2-0 Dexon) or nonabsorbable suture (2-0 Polydek or Ethibond). Remaining periosteum is closed using an absorbable suture (e.g., 2-0 Dexon). If desired, a closed suction drain may be inserted. Deep fascia and peritenon followed by subcutaneous tissue are closed utilizing absorbable suture (3-0 and 4-0 Dexon respectively). Skin is closed with either an absorbable 6-0 Dexon or nonabsorbable 5-0 Prolene suture. Steri-strips, saline moistened sponges, and a dry sterile dressing are applied followed by a below-knee Jones' compression dressing.

The wound is examined on the third postoperative day and placed in a dry sterile dressing. A synthetic cast is applied and the patient instructed to remain non-weightbearing.

POSTOPERATIVE CARE

Postoperative care varies markedly depending on the amount of tendo Achillis that was reflected and on the patient's age. The patient is generally kept non-weightbearing for two to eight weeks in a below-knee cast. In cases of extreme tendinous reflection, an above-knee cast may be utilized. The cast may be bivalved prior to final removal to allow gentle range of motion exercises of the ankle. The cast is kept in place with ace bandages whenever the patient is not performing exercises. The patient is kept non-weightbearing until the tendon is felt to be adequately healed. Upon cast removal, ace bandages or tubigrip may be utilized for several months to decrease postoperative edema and to minimize excessive scar formation. Triceps surae strengthening exercises are initiated after cast removal.

DISCUSSION

The authors have found tendo Achillis calcifications to be more common than generally reported in the literature. When present we have found these calcifications to be associated with Haglund's deformities, retrocalcaneal spurs, and posterior calcaneal steps. When conservative treatment fails, surgical treatment directed at the correction of the calcification and all associated deformities usually provides the most satisfactory result.

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