Soft Tissue Pathology of the Ankle

Benjamin D. Cullen, DPM, Glenn M. Weinraub, DPM*

KEYWORDS

• Soft • Tissue • Pathology • Ankle

Derangements of the soft tissues within the ankle joint can be secondary to a wide variety of pathophysiology. They typically involve synovial or fibrocartilaginous tissue and are chronic in nature.[1] Patients commonly present with persistent pain, swelling, and limitations on function. Left untreated, many of these conditions can progress to permanent joint degeneration. Fortunately, once diagnosed, they often respond well to current treatment options, with arthroscopic debridement playing a large role.

Suspicion for the presence of ankle soft tissue pathology is important to identifying it, as these disorders frequently have insidious onset and nonspecific symptoms. Evaluation should be guided by a detailed history and physical examination, followed by clinical, laboratory, and imaging studies as indicated. Although most symptoms are typically in the anterior compartment, the posterior ankle should also be examined, as pain sometimes may not be present with range of motion but will be elicited with palpation.

The etiology of ankle soft tissue disorders can be classified as traumatic injury, rheumatic disease, or congenital lesions. The pathophysiology, diagnosis, and management of these will be the focus of this article.

TRAUMATIC

Approximately 1 million acute ankle injuries occur annually in the United States, with the vast majority of these being diagnosed as lateral ankle sprains.[2] Depending on the severity of the trauma and the response of the tissue, sequelae can range from minimal and temporary inflammation to prolonged disability. As many as 15% to 20% of injuries can result in chronic symptoms.[3] For patients who have an extended recovery, common soft-tissue pathology includes nonspecific synovitis and secondary impingement lesions.

The authors have nothing to disclose.

Department of Orthopedic Surgery, Kaiser Hayward/Fremont, PMS-36, Rancho-Ohlone Building, 39400 Paseo Padre Parkway, Fremont, CA 94538, USA
* Corresponding author.
E-mail address: Glenn.m.Weinraub@kp.org

Clin Podiatr Med Surg 28 (2011) 469–480
doi:10.1016/j.cpm.2011.04.003 **podiatric.theclinics.com**
0891-8422/11/$ – see front matter

Synovitis

The interior of the ankle capsule is lined by a synovial membrane, a tissue that functions to provide cushioning and lubrication for the joint. Often during an inversion sprain to the lateral collateral ligaments, some damage will occur to the synovium as well. This injury will result in irritation and inflammation, causing pain and swelling of the membrane. Symptoms can be local or generalized, but are typically limited to the anterolateral aspect of the joint.[4] Many times the sensation is vague, and discomfort only occurs with increases in activities.

On examination, patients with simple nonspecific synovitis will usually have minimal objective swelling and full range of motion. Radiographs will be negative, and unless there is a systemic component (eg, rheumatoid arthritis), laboratory work-up should be nonrevealing as well. Depending on the extent of tissue involved, there may be an altered signal intensity on magnetic resonance imaging (MRI), but often this can be equivocal. Of particular diagnostic value is injection of intra-articular anesthetic, which should provide significant relief. If not, then isolated synovitis is highly unlikely.

Initial treatment is conservative, with immobilization, physical therapy, nonsteroidal anti-inflammatory medication, and potentially corticosteroid injection, reserving surgery for resistant cases. If symptoms are refractory to these measures, arthroscopic debridement is the next step. Fortunately, nonspecific synovitis typically responds very well to this modality, and recovery time is minimal (**Figs. 1** and **2**). Radical excision is not necessary, as successful outcomes can be achieved with focus aimed specifically at pathologic tissue. Ferkel and colleagues[5] performed limited synovectomies on 31 patients, with 26 reporting good or excellent results. A postoperative regimen of physical therapy may be considered to limit swelling and enhance return of function, and overall the prognosis is good for these patients.

Impingement

Conditions that cause painful restriction of movement in the ankle joint due to tissue overgrowth are termed impingement syndromes (**Fig. 3**). In those cases where the limitation of motion is due to soft tissue hypertrophy, several distinct phenomena can be the cause. While the presence of impinging abnormal fibrous tissue in various forms and locations within the ankle joint has been thoroughly reported in the literature, the pathophysiology and certain characteristics of these lesions are subject to debate. However, the general consensus is that antecedent trauma of varying severity

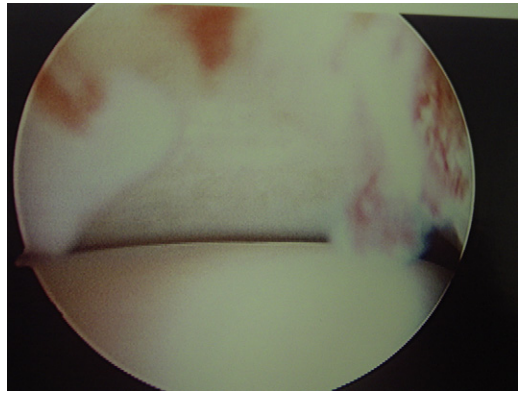

Fig. 1. Nonspecific synovitis to the anterior joint margin.

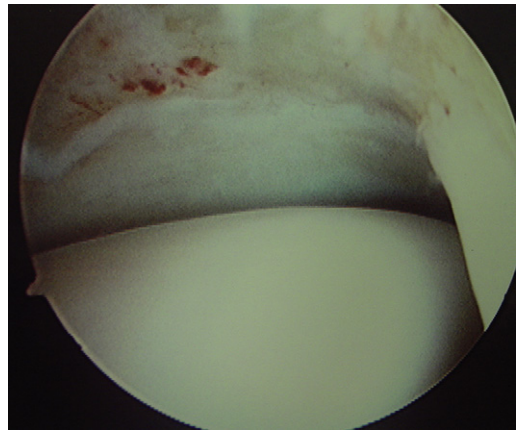

Fig. 2. The synovitis has been resected with a synovial shaver.

is typically associated with their development, usually in the form of a lateral ligament sprain.[6]

Wolin lesion

A mass of hyalinized connective tissue arising from the anteroinferior portion of the ankle joint was first described by Wolin and colleagues[7] in 1950. In their study of 9 patients with chronic anterolateral ankle joint pain and swelling following inversion injuries, a dense mass of white, fibrocartilaginous tissue was noted in the interval between the talus and the fibula upon arthrotomy. Wolin suggested the lesion developed as a result of incomplete resorption of post-traumatic tissue, wherein shear

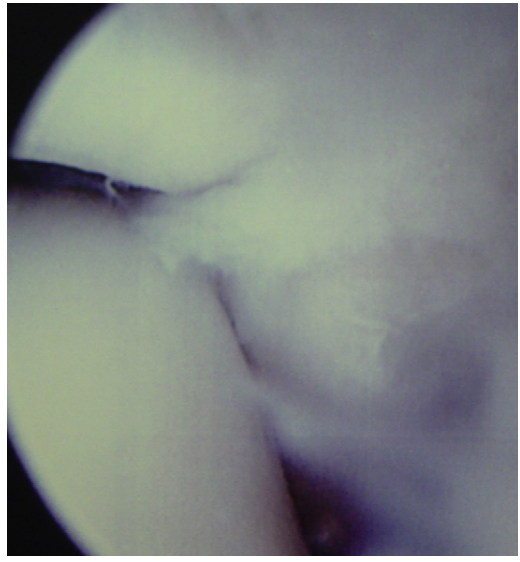

Fig. 3. Intraoperative view of anterolateral soft tissue impingement. The joint is a negative-pressure environment; thus one can see how this soft tissue would be impinged between the joint surfaces with normal dorsiflexion of the ankle.

forces between the talus and the fibula molded the scar tissue under pressure into an organized mass. As the presentation of the lesion was similar to that of a meniscus, he labeled it a meniscoid.

Controversy exists in terms of the nomenclature, as different authors have labeled similarly described pathology alternatively as plica syndrome,[8] fibrous bands,[9] or synovial impingement lesions.[10] It is likely that each of these exists on a continuum, with the organized meniscoid lesion being the well-differentiated end product. The nature of the scar tissue in question is also unclear, as traumatic synovitis was postulated by Wolin, whereas other authors endorsed a tear of the anterior talofibular ligament.[11]

Clinically, these lesions present as chronic post-traumatic pain and swelling, vaguely localized to the area of the anterolateral ankle joint. Although many patients complain of sensations of instability, objective findings are usually negative. Tenderness should be maximal along the anterolateral joint line or with compression of the fibula on the talus; however, these lesions can be intermittently asymptomatic. A potential finding is clicking or popping with ankle range of motion, which may or may not be painful.

The diagnosis is mostly based on history and physical examination, but imaging can be helpful in ruling out other pathology. Radiographs may show evidence of a previous inversion injury.

Computed tomography (CT) with contrast or MRI may reveal the presence of some abnormal soft tissue, but the potential for limited additional diagnostic value must be weighed against the cost of performing the test. As with other intra-articular soft tissue pathology, injection of anesthetic can be both therapeutic and diagnostic.

A trial of conservative therapy is not unwarranted with this condition; however, if a true organized meniscoid lesion is present, this is unlikely to be successful in eliminating symptoms. Fortunately, in all reported cases wherein an isolated mass of scar tissue was excised from the anterolateral gutter, patients have experienced significant improvement postoperatively (**Fig. 4**).

Bassett lesion

A lesion that is close in proximity to the meniscoid but a distinct clinical entity is the pathologic accessory anterior inferior tibiofibular ligament (AITFL). Bassett and colleagues[12] were the first to report an accessory fascicle of the AITFL as the cause of ligamentous impingement in the anterior ankle joint. An anatomic variant, the accessory fascicle, has been identified in anywhere from 21% to 92% of patients

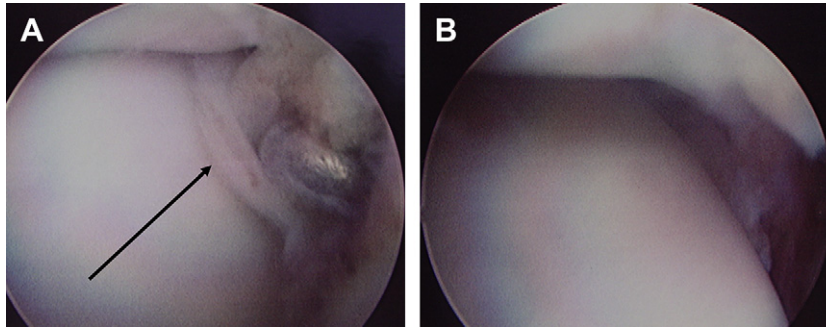

Fig. 4. (*A*) The arrow is pointing to a Wolin lesion or fibrotic scar tissue within the anterolateral ankle joint. (*B*) The subsequent picture shows the joint with the lesion debrided.

(depending on criteria) as a band oriented in parallel to the main ligament but separated from it by a fibrofatty septum.[13] The presence of this structure can therefore be considered a normal finding.

This accessory ligament can become pathologic following an inversion ankle injury that involves damage to the AITFL. If subsequent anterolateral hyperlaxity develops that results in anterior extrusion of the talar dome with dorsiflexion, the inferior fascicle of the AITFL will contact the talus with increased pressure and friction. This can often be reflected by the presence of an abraded area of the cartilage of the talus observed during arthroscopy. Bassett' lesion is thus a problem of abnormal positioning of normal anatomy (**Figs. 5** and **6**).

Much like Wolin lesion, the diagnosis of Bassett lesion should be considered in patients who have chronic ankle pain in the anterolateral region of the ankle after an inversion injury and have a stable ankle and normal plain radiographs. The main clinical difference between the 2 conditions will be the location of point tenderness, where in the case of Bassett lesion should be the anterolateral aspect of the talar dome and in the AITFL. Also, an audible popping and aggravation of pain with dorsiflexion and eversion have been reported to be more common with Bassett lesion than with other impingement lesions.[12]

Conservative management may be futile for this condition, as Akseki and colleagues[14] demonstrated that a regimen of physical therapy, nonsteroidal anti-inflammatory drugs (NSAIDs), and bracing for 3 months failed in all 21 patients they studied. In comparison, resection of the thickened and pathologic ligament was successful in relieving pain in these same patients (as well as those in Bassett's study), without causing any additional instability of the joint. A point to consider is that patients with less than 2 years of ankle pain before surgery for anterior ankle impingement showed significantly better scores in pain, swelling, ability to work, and engagement in sports postoperatively,[15] so prompt diagnosis and excision are key to better outcomes.

RHEUMATIC

Whereas post-traumatic lesions typically present as localized pain to the anterior aspect of the ankle joint, soft tissue disorders that have inflammatory (or unknown)

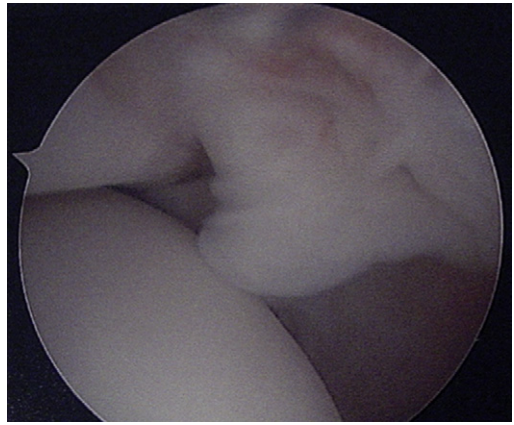

Fig. 5. A thickened inferior fascicle of the anterior inferior tibiofibular ligament that is impinging upon the lateral talar surface; this is subsequently resected.

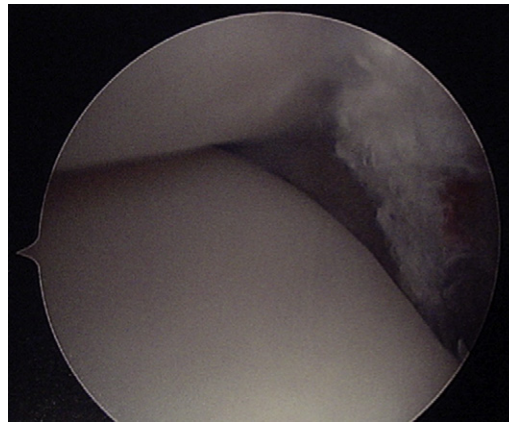

Fig. 6. The lesion in Fig. 5 following resection.

etiology can be symptomatic at virtually any anatomic site in this region. The diagnosis of these conditions is often more difficult, as onset is typically insidious with nonspecific clinical findings. Advanced imaging plays an enhanced role in investigation of these disorders, and frequently the process will only be definitively identified through histologic confirmation. Treatment is also less straightforward, as these lesions have a tendency for recurrence.

Pigmented Villonodular Synovitis

Pigmented villonodular synovitis (PVNS) is a relatively uncommon disorder characterized by proliferation of synovium, resulting in villous or nodular changes to synovial-lined joints, bursae, and tendon sheaths. The incidence has been reported as occurring in 1.8 patients per 1 million population annually, typically between the 2nd and 5th decades of life.[16] Both diffuse and local forms have been described based on the extent of tissue involvement, and Jaffe is credited with recognizing them as different presentations of the same entity.[17]

The presence of lipid and hemosiderin-laden foam cells and multinucleated giant cells has been routinely described on histologic analysis of lesions of PVNS, but the pathogenesis is controversial. Various mechanisms have been proposed, including localized lipid derangement, repeated nontraumatic inflammation, and a benign neoplastic process, but no definitive causality has been confirmed.

Due to its rarity and the nonspecific nature of symptoms, PVNS is often discovered incidentally or as a diagnosis of exclusion. It should be included in the differential diagnosis when monoarticular inflammation with or without a palpable mass presents in young patients. Persistent and generalized swelling, aching, and pain aggravated by activity are the symptoms typically expressed in the literature. Myers and Masi[16] reported that 53% of the 166 cases they studied had a history of trauma, but many case studies describe insidious onset without any inciting event.

Depending on the form and stage of PVNS, radiographs may reveal anything from subtle increases in soft tissue density to frank erosions of periarticular bone and subchondral cysts similar to that found in degenerative joint disease. A distinguishing characteristic between the 2 conditions is that PVNS is not associated with osteophyte formation.[18] MRI is very useful in diagnosis, as it reveals the presence of

hemosiderin deposits, lipids, and inflammatory tissue, as well as the degree of invasion of cartilage or bone. The clinician should expect a low-signal intensity in the area of lesions on both T1- and T2-weighted images.[19] Aspiration of fluid that features brownish discoloration (due to hemosiderin) further reinforces the diagnosis.[20]

Conservative management is not indicated with a diagnosis of PVNS, as delay in definitive treatment could result in significant cartilage and bony destruction. Nodular lesions confined to a local area within the joint may respond well to simple excision, and have a lower reported incidence of recurrence.[21] With the diffuse form, complete synovectomy has been advocated as the treatment of choice, and some authors promote the concomitant use of hydrogen peroxide irrigation.[22] Long-term follow-up is essential in these patients, and the return of pathologic tissue following excision is usually treated with radiation therapy, often with good results.[23]

Synovial Chondromatosis

A rare disorder featuring multiple cartilaginous nodules originating within the synovium, primary synovial chondromatosis is a benign process, typically monoarticular, with symptomatic presentation reported most often between the 3rd and 5th decades of life (**Figs. 7** and **8**).[24] The exact pathophysiology is unclear, but leading theories suggest metaplasia of synovium into cartilaginous tissue versus a primary benign neoplasm. This is in contrast to secondary synovial chondromatosis, where arthritic conditions are the source of the pathologic tissue.

In 1977, Milgram classified synovial chondromatosis into 3 distinct phases.[25] Phase 1 features purely intrasynovial involvement. Active synovitis and nodule formation is present, but no loose bodies can be identified. Phase 2 is considered a transitional period. Nodular synovitis and now loose bodies are present in the joint, but these are primarily still cartilaginous. They may present similar to rice bodies seen in other inflammatory arthritides. In phase 3, the synovitis is quiescent, and the loose bodies are mostly calcified.

There was minimal difference in the duration of symptoms for patients in each of the 3 phases in Milgram's paper, and so the rate of progression from stage to stage likely varies considerably. In comparison, the quality of symptoms for each group was

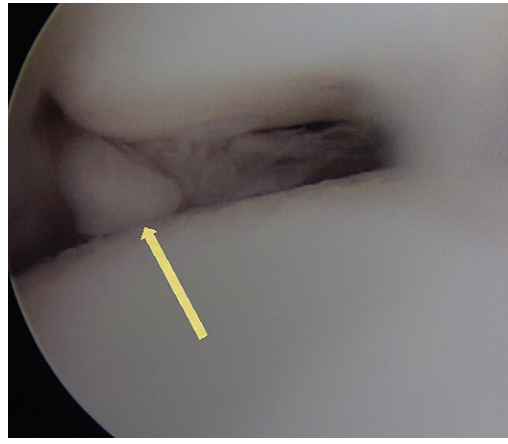

Fig. 7. Arthroscopic view of typical cartilaginous lesion seen with synovial chondromatosis. While this view is of 1 isolated lesion, it should be noted that a multitude of these loose bodies can be encountered within the joint.

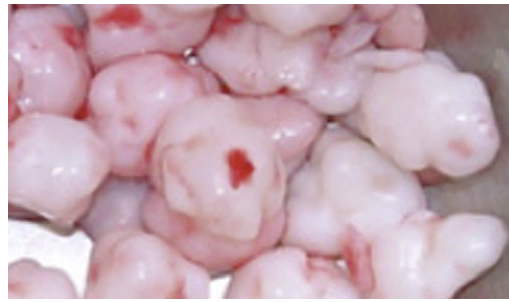

Fig. 8. Multiple loose bodies removed from a single joint with synovial chondromatosis.

significantly dissimilar. Patients with purely intrasynovial disease reported minimal pain, clicking, or locking; rather chronic swelling was their chief complaint. Those patients with phase 3 disease largely related pain and loss of motion. Patients in the transitional phase had a combination of symptoms from the other 2 phases.

Symptoms in all patients typically develop insidiously over a period of months to years.[26] Clinical findings at any stage may include crepitus, locking, pain, or limitation on range of motion, focal swelling, or palpable nodules. Plain films have greatest diagnostic value in phase 3 and late phase 2 disease when lesions have begun to calcify, with the presence of multiple intra-articular bodies of similar size and shape being very suggestive of the diagnosis.[27] CT and MRI can be very helpful in identifying and localizing lesions, although appearance will vary dramatically depending on the amount of calcification and synovial proliferation. Histologic examination confirms the diagnosis, and allows distinction between primary and secondary processes.

No formal recommendations for treatment exist, but the general consensus is that indications for surgery correlate with the phase of the disease. In phase 3, simple removal of the loose bodies has been shown to provide symptomatic relief with minimal complications.[28] However, when synovitis is present, failure to excise this pathologic tissue is associated with increased rates of recurrence, and so partial synovectomy is necessary.

As nodules have been observed to absorb over time and surgery predisposes patients to joint scarring, invasive procedures may be relatively contraindicated in asymptomatic patients.[29]

However, a relative risk of 5% of cases for malignant degeneration to chondrosarcoma has been reported,[30] so histologic diagnosis is prudent in all cases, especially for episodes of recurrence.

Crystalline Deposition

Derangements in the metabolism of monosodium urate (MSU) in gout and calcium pyrophosphate dihydrate (CPPD) in pseudogout can result in crystal deposition at any of various sites, including the ankle. Whereas MSU crystals precipitate in tissues systemically throughout the body as a result of purine catabolism, CPPD crystals are thought to be a primary disorder of articular cartilage associated with the production of inorganic pyrophosphate by chondrocytes.[31] With an identical macroscopic appearance and clinical presentation, distinction between the 2 processes can only be made with microscopic analysis.

Diagnosis of a crystalline deposition disease is fairly straightforward, as the differential of an acutely red, hot, swollen and excruciatingly painful joint is limited to few entities. The significance of the correlation between prolonged crystal deposition and the development and progression of osteoarthritis is less apparent. A strong association exists between the presence of crystals and cartilage degeneration,[32] but whether crystals preferentially deposit in damaged cartilage or if the changes they produce with chronic disease mimic that of osteoarthritis is unclear. The acute flares are considered to be caused by leukocyte ingestion of crystals, thus triggering an inflammatory cascade and extensive joint synovitis. Traditionally, cartilage wear was thought to result from repeated bouts of inflammation, but studies have shown that crystals can be present in uninflamed joints,[33] and so they may induce damage biomechanically.

Management of both diseases is currently primarily pharmacologic. Acute flare-ups can be treated with colchicine, NSAIDs, and potentially injected or oral corticosteroids. Elevated serum uric acid may be controlled with indefinite use of daily medications. If these conservative measures fail to alleviate inflammation, arthroscopic lavage has been advocated as a secondary option for acute attacks (**Fig. 9**).[1] Removal of crystals in asymptomatic joints may prevent future flares, but this has not been thoroughly reported on. If gouty tophi provide discomfort or affect function, these may potentially be excised, but this should be accompanied by medical therapy to limit hyperuricemia and the recurrence of lesions.

Rheumatoid Arthritis

A thorough discussion of the pathophysiology and treatment of rheumatoid arthritis (RA) is beyond the scope of this article. This disorder of the immune system characterized by global synovial hyperplasia and inflammatory cell proliferation is mentioned here as another instance (besides diffuse PVNS) in which aggressive synovectomy may be indicated. As opposed to the majority of pathology described elsewhere in this article in which a limited surgical approach is sufficient, RA features widespread involvement of the synovium. If debridement is to be performed, most authors who advocate it suggest near total excision for maximum benefit and to limit recurrence of symptoms. Beyond providing excellent access to the inflamed tissue, an open arthrotomy will also allow any necessary tenosynovectomy to be accomplished simultaneously. Although the procedure is controversial, it is an option for patients with disease that is inadequately controlled by conservative measures.

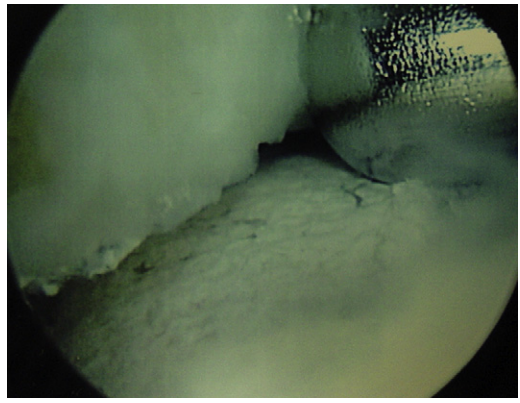

Fig. 9. Advanced crystalline deposition within the ankle joint.

CONGENITAL

Plicae are natural folds of synovial tissue that are found in the anterior, posterior, or syndesmotic recesses of the ankle joint.[34] These loose, pliable, and elastic projections normally move freely on the articular surface with joint motion. Plica syndrome refers to the painful impairment of joint function in which the only finding that helps explain the symptoms is the presence of a thickened and fibrotic plica. It is unclear what causes plicae to become symptomatic; however, overuse and trauma have been suggested etiologies.

Unless pathologic, plicae are not usually associated with local reactive signs. The primary symptom of plica syndrome is pain; however, there may also be a snapping sensation within the joint as the thickened plica is rubbed by bony structures during range of motion. Sometimes a band of tissue is palpable, and if the plica becomes severely irritated, joint swelling may occur. Plicae can be differentiated from meniscoid lesions in that they are not seen in the talofibular interval.[34]

Identification of plica syndrome is primarily through exclusion of other causes for symptoms. Radiographs, CT, and MRI may be used to rule out coexisting pathology, but are often unnecessary. If the history and physical strongly suggest the presence of a symptomatic plica, and conservative measures fail to provide adequate relief, arthroscopy may be indicated for both diagnostic and therapeutic purposes. Resection is curative, and no known sequelae are associated with the removal of plicae.

SUMMARY

The spectrum of pathology that can involve the intra-articular soft tissues of the ankle joint ranges from acute and self-limited to chronic and debilitating. While some form of trauma is implicated in the development of many of these conditions, the exact pathophysiology is still largely theoretical at this time. This is especially evident when comparing patients with the same disorder, yet with dramatically different accounts of the severity of any associated injury. Confounding matters even further is the wide variation in nature and progression of symptoms for the same process in different patients. Clearly, although a single inciting episode may initiate the cascade of events, host susceptibility is also very important to how the disease develops.

Diagnosis should be pursued through an algorithmic approach. A detailed history and physical examination are usually the most critical aspects of the work-up, followed closely by a strong understanding of the potential disease processes that can occur at this anatomic location. The value of a diagnostic injection of anesthetic for determining intra-articular pathology cannot be overstated, as this is a simple and easy modality with high yield. Imaging studies such as radiograph, CT and MRI can be useful in evaluating the presence and extent of a particular condition, or ruling out other causes for symptoms. If the clinician has a strong suspicion that intra-articular soft tissue pathology is present, proceeding with arthroscopy of the joint can confirm the diagnosis while simultaneously allowing for definitive treatment.

The nature of treatment pathways for these disorders depends on the severity of symptoms and the natural course of the disease. Conservative management such as activity modification, NSAIDs, physical therapy, and corticosteroid use may be indicated with mild symptoms or a process that will not eventually result in deterioration of the joint. More urgency is associated with a condition that is disabling or that can cause permanent arthrosis. In those instances, surgical correction may have to be pursued at an earlier interval to prevent unacceptable consequences that would result from delay in action. Fortunately, most of the derangements presented here respond

very well to debridement, and overall the prognosis is very good for this category of pathology.

REFERENCES

1. Guhl JF, Boynton MD. Soft tissue pathology. In: Guhl JF, Parisien JS, Boynton MD, editors. Arthroscopy of the foot and ankle. 3rd edition. New York: Springer-Verlag; 2004. p. 99–114.
2. Wexler RK. The injured ankle. Am Fam Physician 1998;57(3):474–80.
3. Ogilvie-Harris DJ, Gilbart MK, Chorney K. Chronic pain following ankle sprains in athletes the role of arthroscopic surgery. Arthroscopy 1997;13:564–74.
4. Meislin RJ, Rose DJ, Parisien JS, et al. Arthroscopic treatment of synovial impingement of the ankle. Am J Sports Med 1993;21:186–9.
5. Ferkel RD, Karzel RP, Del Pizzo W, et al. Arthroscopic treatment of anterolateral impingement of the ankle. Am J Sports Med 1991;19(5):440–6.
6. Umans H. Ankle impingement syndromes. Semin Musculoskelet Radiol 2002;6: 133–9.
7. Wolin I, Glassman F, Sideman S, et al. Internal derangement of the talofibular component of the ankle. Surg Gynecol Obstet 1950;91:193–200.
8. Gächter A, Gerber BE. Arthroskopie des oberen Sprunggelenkes in Lokalanästhesie [arthroscopy of the ankle under local anesthesia]. Arthroskopie 1991;4: 37–41 [in German].
9. Schonholtz GJ. Arthoscopic surgery of the ankle joint. Arthoscopic surgery of the shoulder, elbow, and ankle. Springfield (IL): Charles C Thomas; 1986. p. 59–72.
10. Stone JW, Guhl JF. Meniscoid lesions of the ankle. Clin Sports Med 1991;10: 661–76.
11. Andrews JR, Drez DJ, McGinty JB. Symposium: arthroscopy of joints other than the knee. Contemp Orthop 1984;9:71–100.
12. Bassett FH III, Gates HS III, Billys JB, et al. Talar impingement by the anteroinferior tibiofibular ligament. J Bone Joint Surg Am 1990;72:55–9.
13. Nikolopoulos CE, Tsirikos AI, Sourmelis S, et al. The accessory anteroinferior tibiofibular ligament as a cause of talar impingement: a cadaveric study. Am J Sports Med 2004;32:389–95.
14. Akseki D, Pinar H, Bozkurt M, et al. The distal fascicle of the anterior inferior tibiofibular ligament as a cause of anterolateral ankle impingement. Acta Orthop Scand 1999;70(5):478–82.
15. van Dijk CN, Tol JL, Verheyen CC. A prospective study of prognostic factors concerning the outcome of arthroscopic surgery for anterior ankle impingement. Am J Sports Med 1997;26(6):737–45.
16. Myers BW, Masi AT. Pigmented villonodular synovitis and tenosynovitis: a clinical epidemiologic study of 166 cases and literature review. Medicine 1980;59:223–38.
17. Jaffe HL, Lichtenstein L, Sutro CJ. Pigmented villonodular synovitis, bursitis and tenosynovitis. Arch Pathol 1941;31:731–65.
18. Smith JH, Pugh DG. Roentgenographic aspects of articular pigmented villonodular synovitis. Am J Roentgenol Radium Ther Nucl Med 1962;87:1146–56.
19. Mandelbaum BR, Grant TT, Hartzman S, et al. The use of MRI to assist in diagnosis of pigmented villonodular synovitis of the knee joint. Clin Orthop 1988; 231:135–9.
20. Rao AS, Vigorita VJ. Pigmented villonodular synovitis (giant cell tumor of the tendon sheath and synovial membrane). A review of 81 cases. J Bone Joint Surg Am 1984;66:76–94.

21. Flandry F, Hughston JC. Pigmented villonodular synovitis. J Bone Joint Surg Am 1987;69:942–9.
22. Saxena A, Perez H. Pigmented villonodular synovitis about the ankle: a review of the literature and presentation in 10 athletic patients. Foot Ankle Int 2004;25: 819–26.
23. O'Sullivan B, Cummings B, Catton C, et al. Outcome following radiation treatment for high-risk pigmented villonodular synovitis. Int J Radiat Oncol Biol Phys 1995; 32:777–86.
24. Iossifidis A, Sutaria PD, Pinto T. Synovial chondromatosis of the ankle. Foot 1995; 5:44–6.
25. Milgram JW. Synovial osteochondromatosis: a histopathological study of thirty cases. J Bone Joint Surg Am 1977;59:792–801.
26. Krebbs VE. The role of hip arthroscopy in the treatment of synovial disorders and loose bodies. Clin Orthop Relat Res 2003;406:48–59.
27. Murphey MD, Vidal JA, Fanburg-Smith JC, et al. Imaging of synovial chondromatosis with radiologic–pathologic correlation. Radiographics 2007;27:1465–88.
28. Young-in Lee F, Hornicek FJ, Dick HM, et al. Synovial chondromatosis of the foot. Clin Orthop Relat Res 2004;423:186–90.
29. Yu GV, Zema RL, Johnson RW. Synovial osteochondromatosis. A case report and review of the literature. J Am Podiatr Med Assoc 2002;92:247–54.
30. Davis RI, Hamilton A, Biggart JD. Primary synovial chondromatosis: a clinicopathologic review and assessment of malignant potential. Hum Pathol 1998;29(7): 683–8.
31. Silcox DC, McCarty DJ. Elevated inorganic pyrophosphate concentration in synovial fluid in osteoarthritis and pseudogout. J Lab Clin Med 1974;83:518–31.
32. Hayes A, Harris B, Dieppe P, et al. Wear of articular cartilage: the effect of crystals. Proc Inst Mech Eng 1993;207:41–58.
33. Martinez-Sanchez A, Pascual E. Intracellular and extracellular CPPD crystals are a regular feature in synovial fluid from uninflamed joints of patients with CPPD related arthropathy. Ann Rheum Dis 2005;64:1769–72.
34. Stienstra JJ. Intra-articular soft-tissue masses of the ankle. Meniscoid lesions and transarticular fibrous bands. Clin Podiatr Med Surg 1994;11(3):371–83.