CHAPTER **29**

OSTEOCHONDRAL LESIONS OF THE ANKLE

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Osteochondral lesions of the ankle have been described by multiple authors in the past. Early descriptions of this pathology date back to the mid 1800s. Although these lesions are not an everyday occurrence, they are common enough to warrant a high place on the list of differential diagnosis for patients presenting with acute and especially with chronic ankle pain.

The etiology of these lesions has been addressed by different authors, leading to several terms addressing identical pathology. Terms previously used to describe these lesions include osteochondritis dissecans, transchondral fractures, and osteochondral lesions of the talus. The primary etiology thought to be important in the development of these lesions is trauma. The mechanism of injury is also matched to certain locations of pathology

LOCATION

The two most common locations of the osteochondral lesion of the talus are posterior medial and anterior lateral, with the posterior medial dome lesion being the most common. Posterior medial lesions of the talar dome have been attributed to plantarflexion inversion injuries. These lesions are typically located in the central or posterior one-third of the medial talar dome and are significantly deeper than their lateral counterparts. The medial lesions have been described as being cup-shaped as opposed to the wafer-shaped lateral lesions.

Lateral lesions are almost always attributed to trauma (90% to 98%) and are more often displaced when compared to the medial lesions. Lateral lesions occur with dorsiflexion inversion or dorsiflexion eversion trauma.

PROGNOSIS

Although no specific reference has been made in the literature, it has been the author's experience that the predominant presenting painful lesion is the medial osteochondral lesion of the talus. Outcome for the patient with an osteochondral lesion of the talus seems to be associated with several factors including the size of the lesion (depth and surface area), proximity/involvement of the shoulder of the talus and the AP location. Lesions which are larger in size cause more change in the loading of the surface of the joint and seem to be more symptomatic as a group. Similarly the same lesions are also deeper and at least potentially affect the stability of more of the surrounding joint surface.

Location also seems to be a critical factor in the symptomatology of these lesions. Lesions which are centrally located along the medial talar dome and lesions which actually involve the medial shoulder of the talus seem to have the worst prognosis in the author's experience.

EVALUATION

Evaluation of the suspected osteochondral lesion begins with a careful history including duration of symptoms, any specific activities associated with reproduction of symptoms and particularly any history of trauma. With regard to activities associated with symptoms, climbing and descending stairs is of particular interest, as well as the patient's ability to squat and stand on his or her toes. These activities will cause maximum travel of the trochlear surface and possibly expose areas which are less often loaded in day-to-day activities.

Clinical examination should include range of motion evaluation in all directions (dorsiflexion, plantarflexion, inversion and eversion), clinical stress testing (anterior drawer and talar tilt) and direct palpation of all joint margins and landmarks.

Plain-film radiographs should be used as an initial screening tool despite their low yield. Follow-up studies may include bone scans, CT or MRI evaluation. An MRI usually supplies the most information following plain films and is only slightly less sensitive to cortical breaks. MRI also offers the advantage of visualizing the change in signal intensity of the subchondral bone and marrow associated with lower grade injuries. CT scan does offer higher resolution of the subchondral bone plate and in many facilities a finer slice thickness is available than is available with MRI.

CLASSIFICATION

Berndt and Harty proposed a classification system for osteochondral fractures based on plain film x-ray findings in 1959. This system grades articular damage ranging from a Stage 1 or contusion of the surface up to a completely displaced osteochondral fragment. More recently Ferkel and Sgaglion established a system based on CT scans. This system is similar to the Berndt-Harty system, but Stage 1 is a subchondral cyst which does not penetrate the cartilage while Stage 2 consists of the subchondral cyst which has penetrated into the joint and can be in various stages of degeneration. Stages 3 and 4 appear very similar to Berndt-Harty 3 and 4 of plain films. A system grading MRI was proposed by Anderson, Crichton and Grattan-Smith in 1989.(Fig. 1) This correlates fairly well with the CT grading system, with the exception that a Stage 2 is a fragment with incomplete detachment. Stages 3 and 4 correlate well with Berndt-Harty and Ferkel Sgaglion.(Fig. 2)

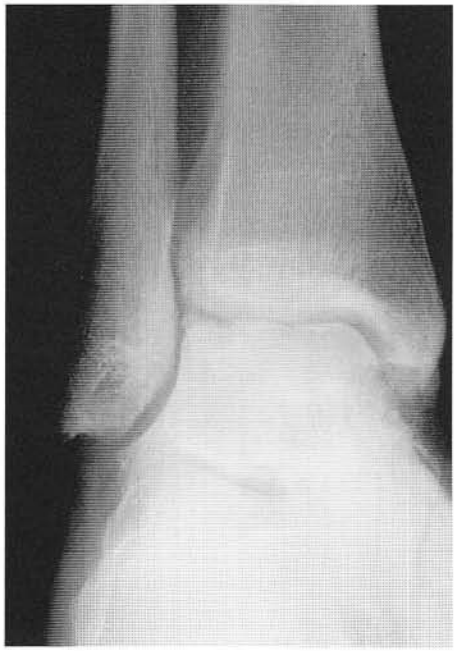

Figure 1A. Berndt-Hardy 4 plain-film radiograph.

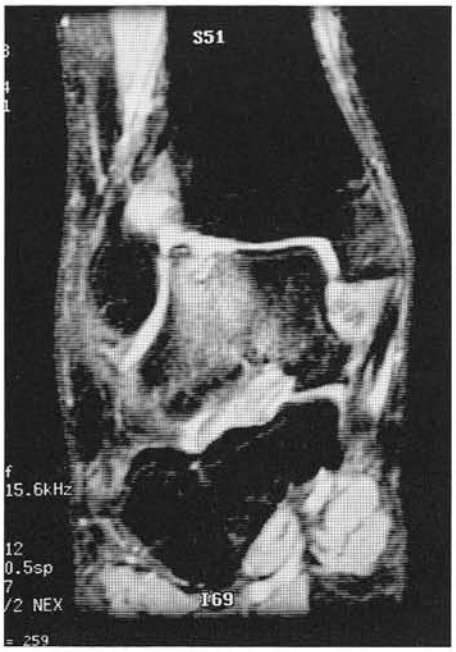

Figure 1B. Corresponding MRI, coronal view.

TREATMENT

Treatment of osteochondral lesions of the talus has traditionally ranged from benign neglect to debridement via open ankle arthrotomy, all with varying degrees of success. Diagnosis of the lesion is one of the first major obstacles, with acute lesions rarely being detected. More commonly these patients present with an average of 4 to 20 months duration of symptoms prior to appropriate diagnosis and treatment.

Conservative care with immobilization has rarely shown good results for these lesions, however in younger lesions (<6 months) this may still occasionally be effective. Later in the course of the pathology however this is rarely helpful. Diagnostic/therapeutic injections can be quite helpful for definitively identifying the involved joint as the origin of symptoms and

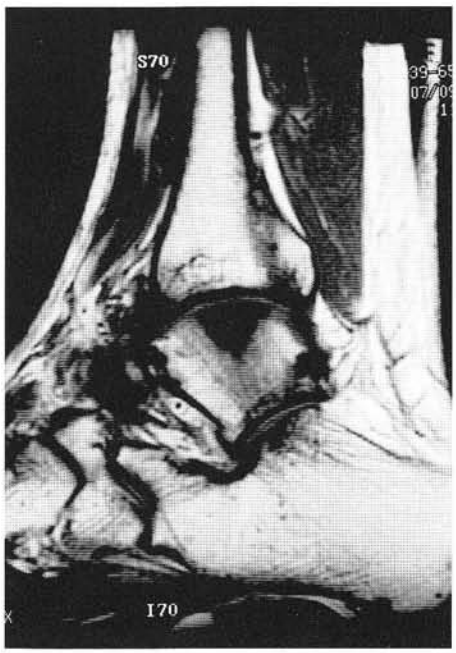

Figure 1C. Lateral view.

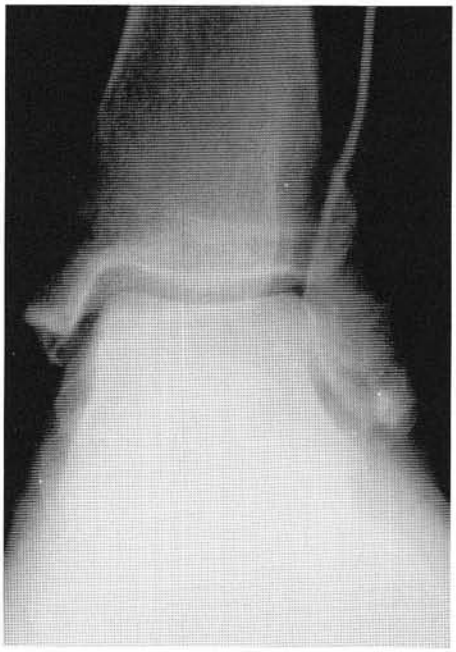

Figure 2A. Plain-film radiograph in a patient with chronic ankle pain and a history of injury.

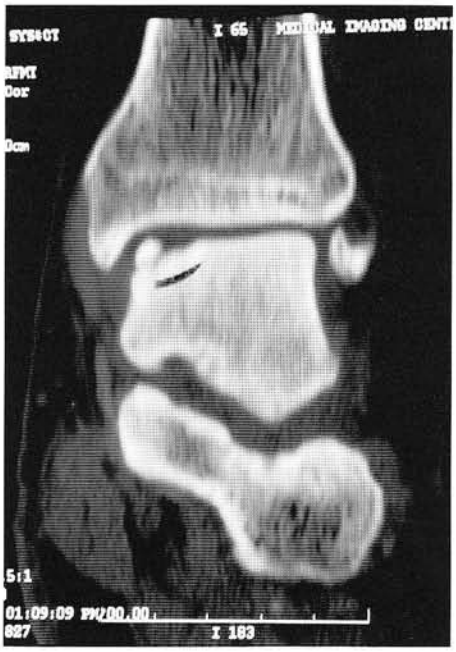

Figure 2B. Corresponding CT scan demonstrating a typical posterior medial osteochondral lesion of the talar dome.

also, with steroid added to the injection, for helping reduce inflammation of the joint lining.

Once the joint itself has been identified clinically as the point of origin of symptoms, plain-film x-rays and special imaging studies should be considered as they can be quite helpful for further diagnosis and mapping of potential osteochondral lesions.

The author now feels that arthroscopic debridement for these lesions is the treatment of choice. With currently available instrumentation and improved distraction devices, the ankle can be completely evaluated in most cases with minimal difficulty. Arthroscopic debridement offers the advantage of less tissue disruption and postoperative edema compared to open ankle arthrotomy with or without malleolar osteotomy. Also, since lesions can be addressed without osteotomy, immediate postoperative range of motion can be a reality when desired.

Arthroscopic debridement of the joint is performed utilizing the small joint (2.9mm) arthroscope and shaver (3.5mm full radius) to initially clear any hypertrophic synovium. Once adequate visualization of the anterior pouch has been attained, the non-invasive ankle distractor is applied and the joint distracted. The arthroscope is then advanced posteriorly and the remainder of the joint explored. The extent of the lesion is then mapped using a probe to determine the margins of the periphery. After the expanse has been defined, debridement is then accomplished using curettes and shaver as necessary. After completion of debridement to the floor of the lesion, the base is fenestrated with either a K-wire or a set of hand awls (45 and 90 degree are the most common sizes). After completion of the debridement, joint lavage is performed with a minimum of 1 additional liter of solution. Closure is then performed.

After closure, a modified Jones compression dressing is applied. The patient is maintained immobilized for 72 hours prior to the first dressing change. At the first dressing change the patient is placed into a CAM walker so that active gentle range of motion dorsiflexion/plantarflexion exercises may be begun. The patient is instructed to perform these at least 5 times daily, for a period of 20 minutes per session. Non-weightbearing is maintained for 6 weeks, then is progressed from partial to full-weightbearing over the next three weeks. At 9 weeks postoperative, the patient is allowed to return to regular shoes with guarded activities initially.

In the author's experience, the majority of patients who have undergone this treatment have experienced significant improvement or complete relief of symptoms. It is anticipated that further experience in the treatment of this condition and advances in cartilage resurfacing and repair will continue to improve the outcome for these patients who once would have been presumed to have a poor outlook for future activities.

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