Brief Communication
Communication abrégée

Osteochondritis dissecans of the talar dome treated with an osteochondral autograft

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The prevalence of osteochondritis dissecans of the talar dome is often underestimated.[1,2] A variety of treatments, both conservative and surgical, have been used for these osteochondral lesions. Surgical intervention is indicated when the fracture has resulted in unstable fragments or loose bodies. Removal of these, drilling of the subchondral bone and abrading the surface of the lesion are the standard procedures.[3–8] Still, these techniques are unsatisfactory for larger defects, and such lesions may be managed by open reduction and internal fixation.[3,5] However, when the osteochondral fragment contains a minimal amount of subchondral bone, fixation may not be possible. In these circumstances, osteochondral autografting is an option.[9] The use of this procedure at the talar dome was prompted by the results that were achieved by many authors employing a similar approach (using either autografts or allografts) to treat osteochondritis dissecans of the knee.[9–14] We report on a patient who had osteochondritis dissecans of the medial talar dome treated with an osteochondral autograft from the knee.

Case report

A 35-year-old man presented with a fracture of his right distal fibula after an inversion injury. He was treated with immobilization for 6 weeks. Radiographs showed excellent fracture alignment with evidence of satisfactory healing. Two months later, he began to experience anterior right ankle pain on walking distances. He also experienced pain and stiffness after prolonged periods of sitting and on getting out of bed in the mornings. Examination revealed a full range of motion (with pain on dorsiflexion) and no swelling or tenderness of the right ankle. There was no locking or catching. Radiography (Fig. 1) revealed an area of lucency in the right medial talar dome. Magnetic resonance imaging (MRI) (Fig. 2) showed an osteochondral fracture ($11 \times 6 \times 11$ mm) of the superomedial talar dome with adjacent marrow edema. There was no deformity of the articular surface or any loose fragments. The patient was managed conservatively with restriction of activities.

Six months later the symptoms persisted along with the new finding of tenderness over the anterior aspect of the ankle. A computed tomography arthrogram (Fig. 3) showed a sclerotic lesion with cystic change in the medial talar dome without any flattening of the articular cartilage. No contrast was seen between the lesion and the underlying bone. Subsequent arthroscopy revealed a large flap of carti-

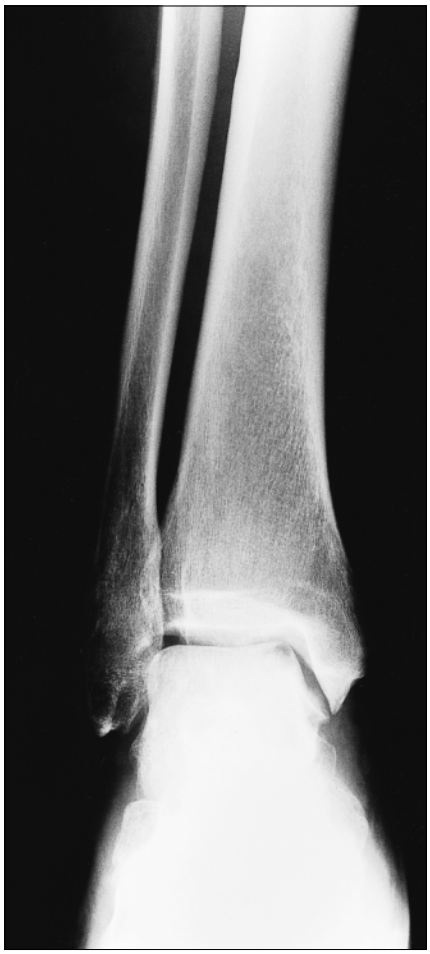

FIG. 1. Plain film of the right ankle shows an area of lucency in the talar dome.

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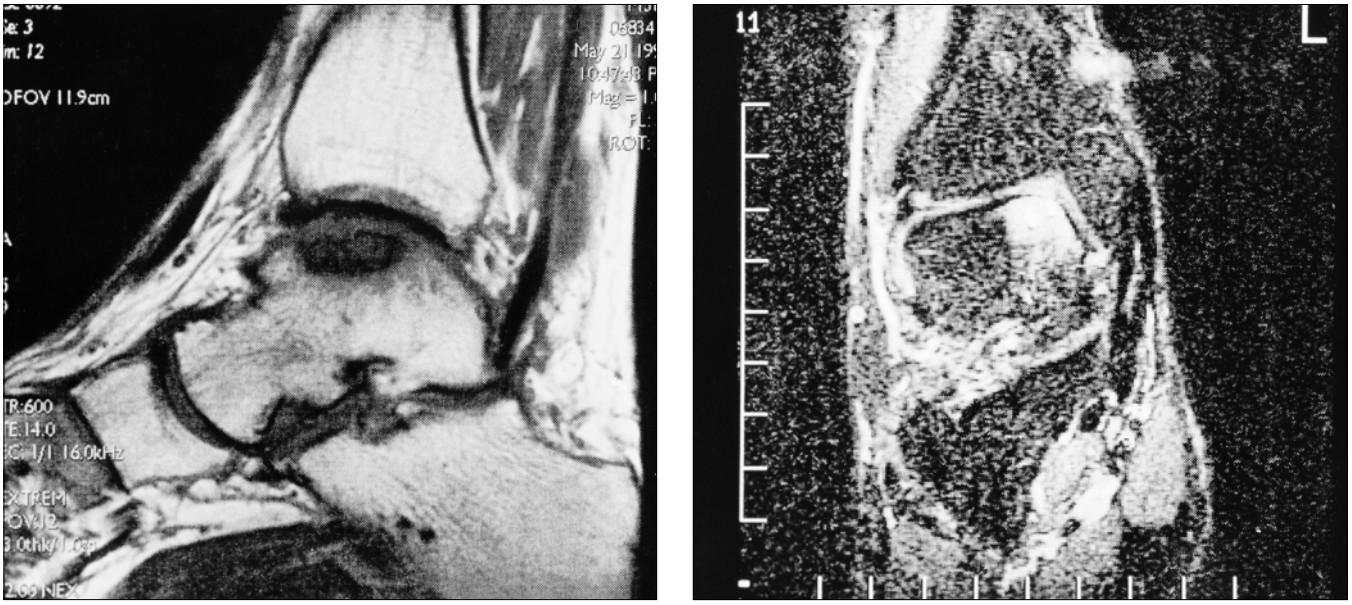

FIG. 2. Magnetic resonance imaging (MRI) demonstrates an osteochondral fracture of the superomedial talar dome with adjacent marrow edema.

lage that was loosely attached to the underlying bone. These findings were consistent with a stage II lesion (Table I). Because there was insufficient subchondral bone to attempt fixation, the defect was only debrided.

Despite temporary relief, 8 months later the patient continued to have pain, so we elected to repair the defect with an osteochondral autograft. The procedure used an anteromedial approach to the ankle. Though not in keeping with the MRI findings, the defect was sized at only 9 mm in diameter, and a 13-mm deep recipient socket of the same size was created. This was press-fitted with a 10-mm donor plug (15 mm in depth) from the ipsilateral lateral femoral condyle of the knee. This resulted in a smooth, flush articular surface. The donor site was then filled with the bone plug taken from the ankle.

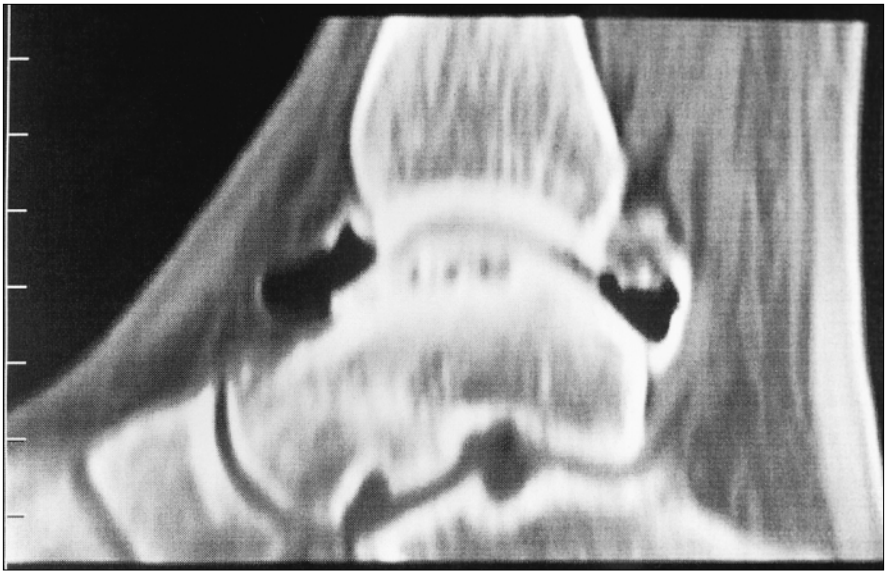

FIG. 3. A computed tomography arthrogram shows a sclerotic lesion with cystic change in the medial talar dome. There is no flattening of the articular cartilage.

After 6 weeks the patient's ankle pain had completely resolved although he still had some discomfort at the knee. At 3 months he had only mild posterolateral ankle pain on walking distances. No effusions of either the knee or the ankle were noted, and both joints had a full range of motion. Radiologic investigations demonstrated a mild irregularity of articular cartilage (approximately 2 mm) of the talar dome with no significant bone marrow edema (Fig. 4). At 12 months the patient had no complaints of pain or swelling at either joint. He was able to walk distances and jog on a treadmill without symptoms. On examination, both the knee and ankle had a full range of motion; there were no effusions or areas of tenderness. A plain radiograph (Fig. 5) now showed a defect in the medial talar dome, and MRI (Fig. 6) revealed that the depression of the articular cartilage had not progressed.

DISCUSSION

Osteochondritis dissecans may refer to an osteochondral fracture in previously healthy or ischemic bone that has resulted in non-union.[3,8,15] The ankle is the second most common joint affected by osteo-

chondritis dissecans after the knee.[16] In the ankle, the anterolateral and posteromedial aspects of the talar dome are the most frequently involved areas.[5,6,8,17] These fractures occur more frequently in males,[3,5] typically in the second, third and fourth decades of life.[3,18,19]

The etiology of osteochondritis dissecans was once controversial, but most investigators now consider trauma to be the main causative factor in osteochondral lesions of the lateral talar dome.[5,6,15] In contrast, 80% of medial fractures are not accompanied by a clear history of trauma.[6,15] The lesion in our patient was atypical in this sense. The mechanism of injury for an anterolateral fracture is inversion in a dorsiflexed position whereas for a posteromedial lesion it is inversion in plantar flexion.[5] The usual clinical presentation of such fractures is one of an inversion injury[5] followed by pain with activity, stiffness, swelling, restricted range of motion, locking, crepitus, weakness, instability and occasionally a palpable loose body.[6,16] There may be tenderness of the anterolateral or anteromedial aspect of the tibiotalar joint, depending on the site of the lesion.[16]

Standard radiography is the best imaging technique[5,8] and may be followed by tomography, CT or MRI. CT may be used in preoperative planning to accurately size and position the defect.[20] MRI is invaluable in correctly staging osteochondritis dissecans and is particularly useful in distinguishing between stable and unstable lesions.[21,22] Finally, bone scanning may be used as a screening technique for occult lesions.[23]

The most widely accepted classification system for osteochondral lesions of the talus is that of Berndt and Harty.[5] Dipaolo and associates[22] added to this scheme by correlating radiographic staging with MRI and arthoscopic findings (Table I). Furthermore, Loomer and associates[24] described a modification to Berndt and Harty's system. Their type 5 lesion constituted a radiolucent defect found on CT (subchondral cyst formation with surrounding sclerosis). They postulated that the progression of types 1, 2, and 3 osteochondral lesions would produce such a defect.[24] Interestingly, on CT, our patient appeared to have such a lesion. However, subsequent arthroscopy demonstrated that the lesion was more consistent with stage II.

The management of osteochondritis dissecans is guided by the stage of defect.

Table I

Staging of Osteochondral Lesions[22]

Stage	Arthroscopic findings	MRI findings	Radiologic findings
I	Softening and irregularity of cartilage, no fragments present	Thickening of cartilage, low signal changes	Compression of subchondral bone, no fragments present
II	Cartilage breached. Definable fragment — not displaced	Cartilage breached. Low signal rim adjacent to fragment (fibrous attachment)	Definable fragment — attached
III	Cartilage breached. Definable fragment — displaceable but attached to some overlying cartilage	Cartilage breached. High signal changes adjacent to fragment (interposing fluid)	Definable fragment (nondisplaced) — without attachment
IV	Loose body	Loose body	Displaced fragment

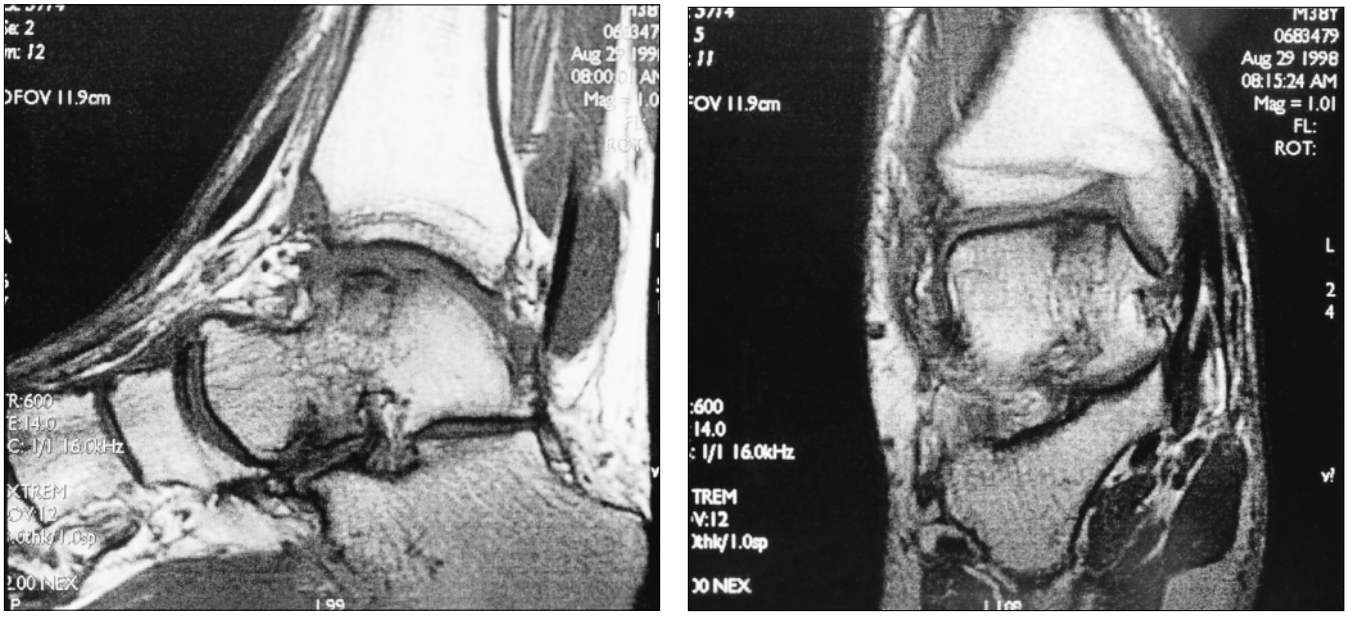

FIG. 4. MRI demonstrates a mild articular irregularity (approximately 2 mm) of the talar dome but no significant bone marrow edema.

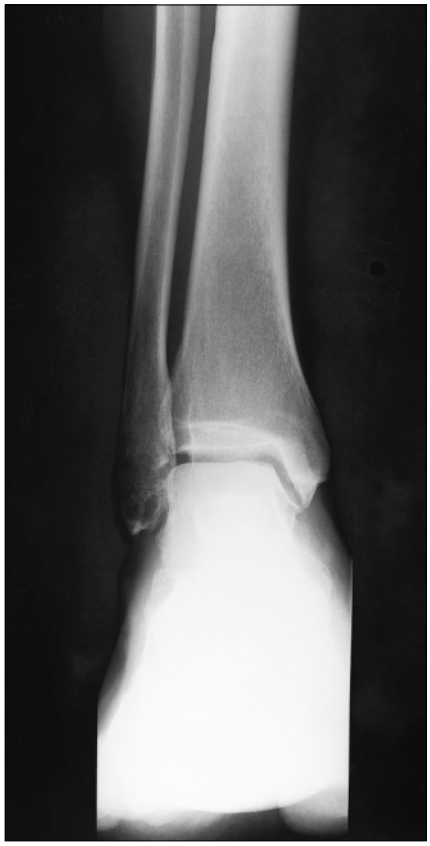

FIG. 5. Plain radiograph 12 months postoperatively shows a defect in the medial talar dome.

With stage I lesions, partial weight bearing with restriction of activities and limitation of motion is usually adequate to permit healing.[3,6,8] Patients with stage II lesions are better treated with cast immobilization for 6 weeks.[8] Stage III osteochondral lesions may be considered for surgical management. Although medial lesions may first undergo a trial of conservative therapy, lateral stage III lesions should be operated on immediately as they fare poorly with nonsurgical interventions.[6] Stage IV lesions (loose bodies) are always managed surgically.[3,6–8]

The many operative techniques used to treat osteochondritis dissecans of the talar dome all share a few essential elements: removal of the necrotic fragment or loose body, drilling of the subchondral bone and abrading the crater left by the sequestrum.[3–8] The site of the defect and the extent of the procedure determine whether an arthroscopic approach is possible.[25–28] The long-term results of both arthroscopy and arthrotomy are similar.[28] Although larger unstable defects may be managed with bone pegs, [29–31] countersunk cancellous screws or Herbert screws,[5] these methods are ineffective in securing fragments with insufficient subchondral bone. Therefore, osteochondral autografting is a welcome additional surgical technique that addresses this subgroup of lesions.

Osteochondral autografts and allografts have been used to treat osteochondritis dissecans of many joints. Many[9–14] have demonstrated good to excellent results after treating osteochondral lesions of the knee with such grafts. Johnson and Warner[32] also obtained an excellent outcome after treating an osteochondral lesion of the humeral head with a matched osteochondral allograft.

In the ankle, Hangody and associates[9] were the first to report on the results of autogenous osteochondral grafting to treat osteochondritis dissecans of the talar dome. In their preliminary report, 11 patients with osteochondral lesions 10 mm or larger were managed surgically using the mosaicplasty system of osteochondral autograft transplantation. The donor site was either the medial or lateral femoral condyle of the ipsilateral knee. An open technique was used in all the cases, with arthotomies of both the knee and the ankle. The mean follow-up was 16 months (range from 12 to 28 months). All the patients returned to full activities and

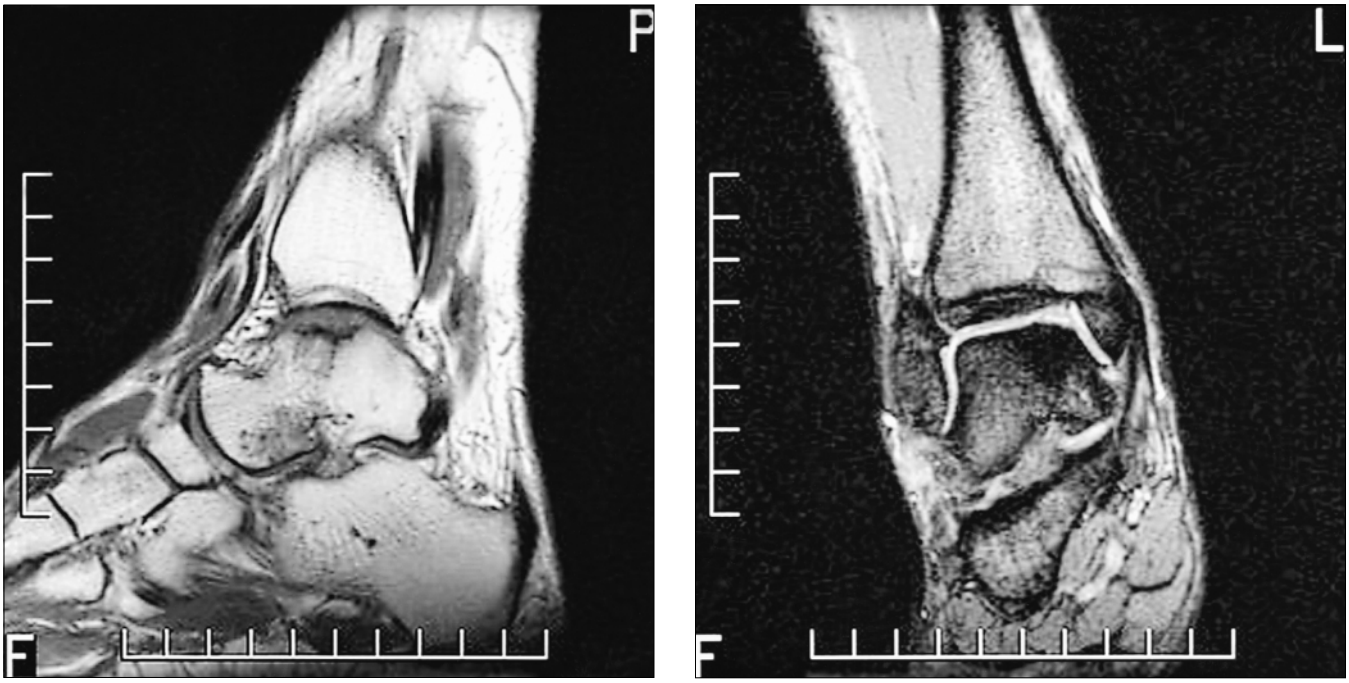

FIG. 6. MRI 12 months after grafting shows that the articular irregularity has not progressed.

achieved high marks on the Hannover ankle and Bandi knee scoring systems.[9]

We used a similar procedure to the one described by Hangody and associates. The main difference was in the size and number of grafts used. Our patient received only a single 10-mm wide graft whereas the 11 patients reported by Hangody and associates were treated with 2 to 4 3.5-mm or 4.5-mm grafts. We anticipated that the use of a single osteochondral plug would minimize fibrocartilage ingrowth, a prominent feature of the mosaicplasty technique. In addition, it was postulated that the use of a solitary 10-mm graft could produce a congruent articular surface. For these reasons, we expected our variation of the procedure to produce a similar clinical result. However, the use of a single large osteochondral autograft may have implications for the immediate postoperative period and the course of subsequent rehabilitation. In theory, one larger graft would create a more solid fit at the recipient site while at the same time generating a larger defect at the femoral condyle. This may lead to a faster reduction in symptoms at the ankle but could delay the resolution of symptoms at the knee. This was not the case with our patient.

Despite a successful clinical outcome, radiologically there is still a notable defect. The incongruity of the talar dome is most likely due to settling of the graft. In retrospect, a flush articular surface may have been better achieved by leaving the osteochondral plug approximately 2 mm proud. Whether this would have changed the patient's clinical course is unknown.

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