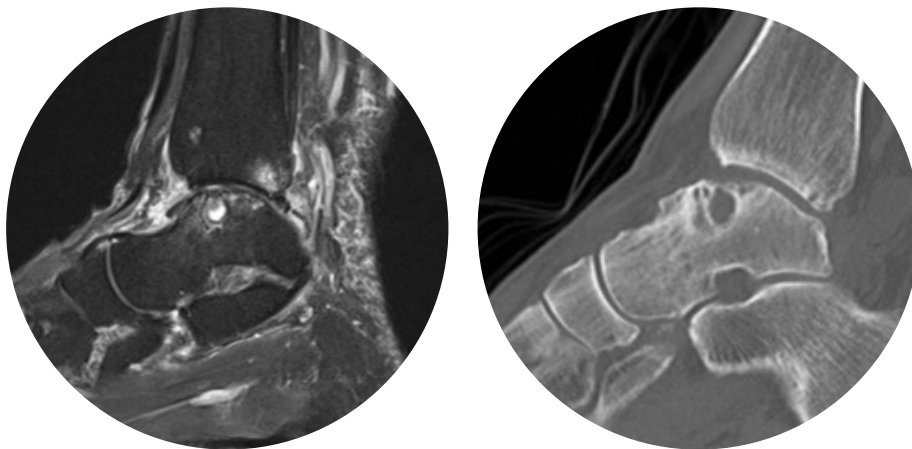


CASE STUDY

Talar Chondral Lesion Repair with CartiMax® FA Viable Cartilage Allograft

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Pre-operative sagittal-view MRI and CT scans confirming chondral lesion.

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INTRODUCTION & BACKGROUND

The ankle is the third most frequently affected joint by osteochondral lesions, comprising 4% of all reported cases.¹ Osteochondral lesions isolated to the talus are usually caused by ankle sprains, where the talus impacts the distal tibial plafond. Microfractures or damage to the chondral surface and subchondral talar bone result from this impact.

Upon weight-bearing, the increased pressure in these locations, in conjunction with the infiltration of synovial fluids, may lead to expansion of the defect site; osteonecrosis, cyst formation, and pain may be induced.² At this point, the lesions can become symptomatic.

Larger defects often fail conservative treatments such as NSAIDs, modified weight-bearing, immobilization, and physical therapy. When conservative treatment fails, surgical intervention is considered.

Varying methods of surgical intervention exist, including excision and curettage coupled with drilling or microfracture, autogenous bone graft, and chondral transplantation. Success rates with these methods have been reported anywhere between 41% to 93% depending on the technique and method used to repair the defect.^{2,3,4,5}



CartiMax® FA Viable Cartilage Allograft

The purpose of this case report is to describe a unique method to treat chondral lesions using a subchondral plug and CartiMax® FA Viable Cartilage Allograft.

TECHNOLOGY OVERVIEW

CartiMax® FA is a viable allograft containing chondrocytes and chondrogenic growth factors with putty-like handling properties, intended for treatment of confined cartilage lesions in the foot and ankle.

CartiMax® FA is composed of two components: Viable Cartilage Fibers (VCF) and Cartilage Allograft Matrix (CAM).

The VCF, containing cryopreserved native adherent chondrocytes, is combined with CAM, containing cartilage extracellular

matrix, endogenous growth factors (IGF-1, TGF- β , FGF-2), and other proteins to create a moldable, cohesive, biologic scaffold capable of repairing focal defects in articular cartilage.^{8,9,10}

CartiMax® FA has been proven to:⁷

- » Safely support cartilage repair and graft remodeling
- » Fill contained defects up to 5 cm² and remain in its intended location
- » Robustly integrate to edges of native tissue (cartilage edges and subchondral bone)

Characterization testing of CartiMax® FA verifies the presence of functioning viable chondrocytes and chondrogenic growth factors while affording maximum handling flexibility with its putty-like consistency.⁶ Multiple studies also demonstrate that chondrocytes migrate and proliferate from the cartilage fibers and secrete components found in hyaline cartilage.^{4,7,8} It has the potential to circumvent the morbidity and technical difficulties of currently available restorative procedures such as arthroscopy with marrow stimulation via microfracture or autologous chondrocyte implantation.^{10,11} Usage of CartiMax® FA does not require perpendicular access to the chondral

lesion, which eliminates the necessity of the medial malleolar or fibular osteotomy often required with other methods of cartilage repair.^{12,13} As a result, fewer procedural steps are required to apply CartiMax® FA, as it is moldable and can conform to the defect without the need for templating, suturing, or the application of fibrin glue. Due to the cryopreservation process of CartiMax® FA, there is an increased shelf-life with chondrocyte viability up to two-years post-cryopreservation,

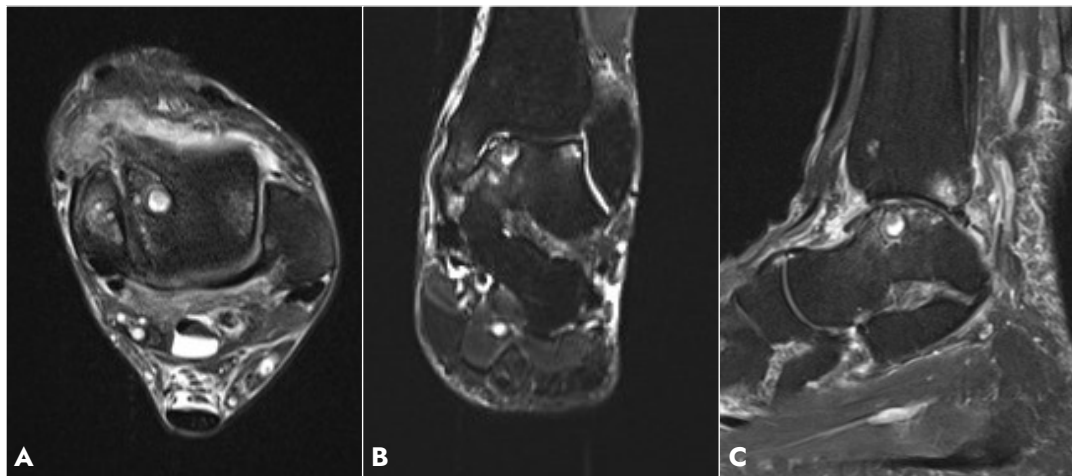


Figure 1. Diagnostic axial (A), coronal (B), and sagittal (C) STIR MR images showing a hyperintense focal lesion of the medial talar dome.

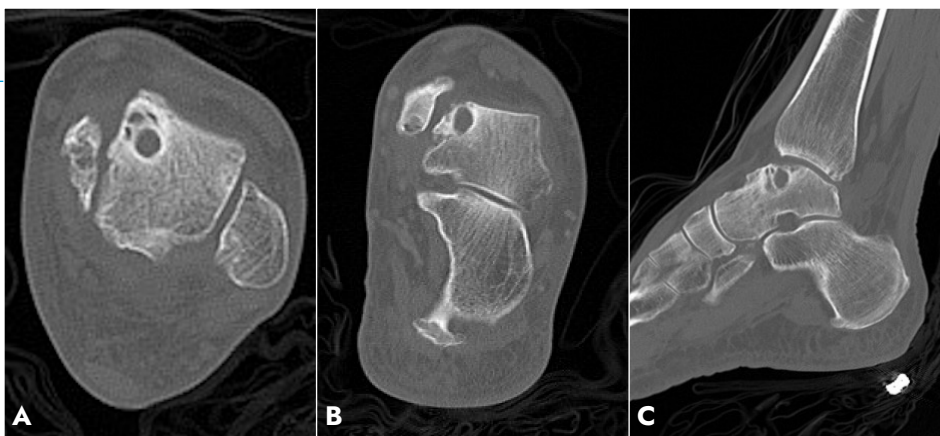


Figure 2. Diagnostic axial (A), coronal (B), and sagittal (C) CT images showing a focal osteochondral lesion in the medial talar dome.

compared to fresh osteochondral grafts that may maintain viable cells for limited periods of time.¹⁴

METHODS

A 53-year-old female with a history of hypertension and elevated BMI presented with moderate pain to the left ankle upon ambulation and passive range of motion, with peak pain indicated during dorsiflexion. Preliminary radiographic review indicated excessive osteoarthritis of the left ankle joint with associated joint space narrowing, and potential cystic changes of the medial talar dome.

An MRI was obtained after the patient failed to respond to conservative treatments inclusive of injections, oral NSAIDs and steroid medications, and modified weight-bearing with an ankle brace and walking boot.

The resulting MRI revealed severe osteoarthritis of the tibiotalar joint with multiple subcortical cysts which were most pronounced at the medial aspect of the talar dome (**Figure 1**). The largest cyst measured 0.9cm, with no subchondral collapse or loose intra-articular body appreciated. Small tibiotalar and subtalar joint effusions with no acute bony abnormality, marrow edema, or AVN, were also noted.

A CT scan confirmed the MRI findings, with the largest osteochondral defect located on the medial talar dome. This defect was measured to be 10x12mm with

associated subarticular fracture and loose bodies in the ankle joint measuring up to 2mm (**Figure 2**).

Surgical intervention was offered and pursued by the patient due to the unsatisfactory response to conservative treatment methods.

SURGICAL INTERVENTION

The patient was positioned on the operating table in the supine position, and a standard pneumatic thigh tourniquet was placed on the lower left extremity. After prepping and draping the extremity using standard aseptic technique, attention was directed to the anterior portion of the ankle.

Prior to inflation of the tourniquet a left ankle arthroscopy was performed using the standard anteromedial and anterolateral portals. All synovitis was debrided from the joint. The OCD lesion was visualized to the medial aspect of the talar dome and almost the entire medial shoulder. After debridement and visualization of the lesion it was determined that an arthroscopic approach would not be adequate. All arthroscopic instrumentation was removed. The extremity was then exsanguinated with an Esmarch and the tourniquet was raised at 250mmHG.

The incision from the anteromedial portal was extended in a longitudinal manner along the anterior aspect of the medial gutter of the ankle joint. Dissection was carried down to the level of the ankle

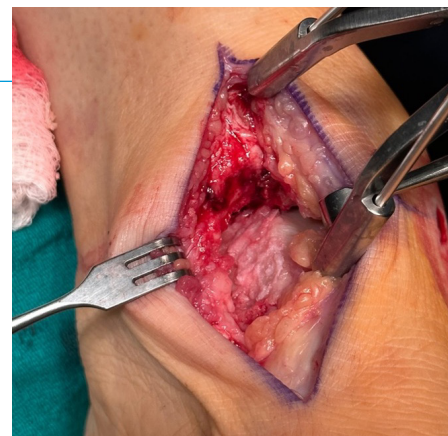


Figure 3. The osteochondral defect has been repaired with morselized subchondral graft and CartiMax®. The medial talar dome and shoulder have been remodeled back to the appropriate anatomic shape with the help of CartiMax® FA viable cartilage allograft.

joint capsule where a parallel longitudinal capsulotomy was performed in order to visualize the ankle joint. The medial talar dome and shoulder was easily observed and the osteochondral damage to the medial dome and shoulder was observed. A Hintermann retractor was used to distract the ankle joint and gain better access to the lesion. After debriding all loose cartilage around the lesion, the osteochondral defect was noted to be 2.0 x 1.0 x 1.0cm, covering most of the medial shoulder. The area was then irrigated with sterile saline.

Due to the expansiveness and location of the lesion, preparation and insertion of the **AlloAid SubChon®** engineered allograft implant was not possible. Therefore, the subchondral plug was morselized and packed within the lesion until the defect was filled to 1.5mm below the level of surrounding cartilage. After confirming the site was completely dry, CartiMax® FA viable cartilage allograft was evenly spread over the entire medial talar shoulder taking care to ensure the entire defect was covered to the level of the surrounding cartilage. This effectively remodeled the medial talar shoulder back to its normal anatomic shape (**Figure 3**).

The joint distractor was removed and anatomic layers were closed in a sequential fashion. The tourniquet was released and the patient was placed in a posterior splint.

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FOLLOW-UP

The patient was made non-weight bearing (NWB) in the posterior splint until sutures were removed at the two week follow-up appointment. She was then placed in a CAM boot and instructed to continue NWB status for another six weeks, with passive range of motion encouraged during this time. The patient states there was no pain with passive ROM post operatively, a stark contrast to her preoperative condition where minimal ankle joint ROM would illicit pain.

The patient was transitioned to partial weight bearing in the boot at eight-weeks post-op, with no increase in pain indicated. The patient was able to graduate to a supportive athletic shoe and ankle brace at twelve weeks post-op. Physical therapy was initiated at this time. To date, the patient is walking in an ankle brace with minimal to no pain to the ankle joint.

CONCLUSION

We have described a unique case of successful talar osteochondral lesion repair utilizing CartiMax® FA viable cartilage allograft. We achieved a stable and reliable repair without need for microfracture or drilling, thereby minimizing morbidity. Occupying most of the medial talar shoulder, this lesion was extensive and did not lend itself to some of the traditional and standard treatment techniques. This patient had improved pain and function at 3-months postoperatively and continues to ambulate without pain. Chondral lesion treatment is safe and effective with CartiMax® FA viable cartilage allograft.



Figure 4. Postoperative radiographs of the ankle joint demonstrating the repaired osteochondral defect and remodeled medial talar shoulder.

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