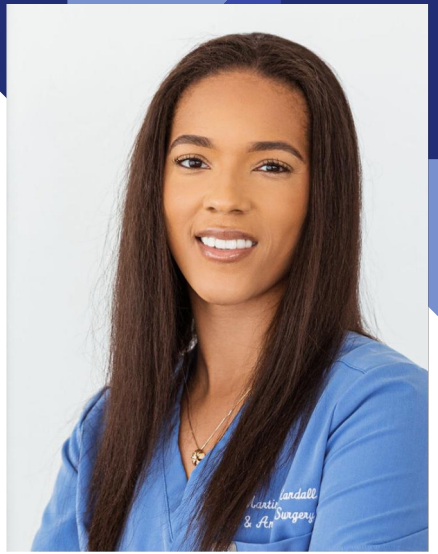
Dr. Martina Randall



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Osteochondral Lesions of the Talus

I have no conflicts with this presentation

Biology

Cartilage is:

Chondrocytes embedded in ECM

ECM→ Collagen fibers, hyaluronic acid, proteoglycans and water

Cartilage is supported by the subchondral bone plate

Biology

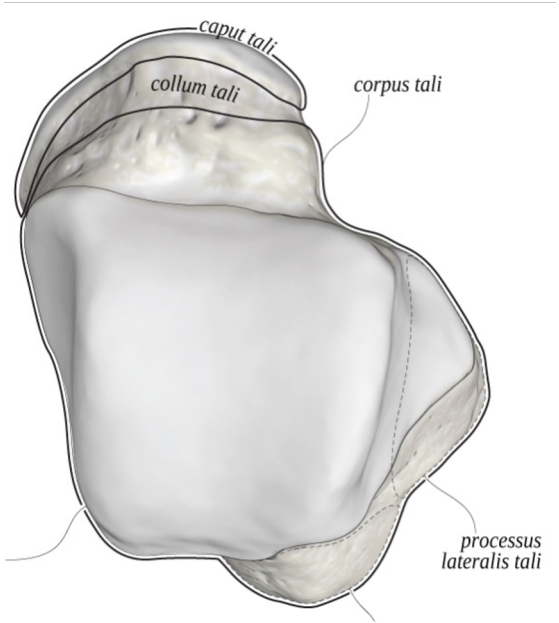
Cartilage is:

Nourished by articular fluid

No nerve or lymph tissue

Avascular; no chondrocyte migration to injuries
Slow metabolism

Anatomy



Talus

60% articular- Limited vascular access. High risk of posttraumatic osteonecrosis and overall limited healing potential for osteochondral injuries of the talus.

Talus Osteochondral Lesions

- Osteochondritis Dissecans (OCD)
- Transchondral Fracture
- Osteochondral Defects

Etiology

Most- Repetitive Microtrauma

10% genetic

Hermanson E and Ferkel RD. FAI 2009

Ankle Instability

Ankle Sprain

-The **most common musculoskeletal injury** (27,000 per day)

Up to 50% may result in cartilage injury

Saxena et al. AJSM 2007

Risk Factors for Osteochondral Lesions and Osteophytes in Chronic Lateral Ankle Instability

A Case Series of 1169 Patients

Wang et al. OJSM 2020

Chronic instability- Increased risk of Osteochondral lesions- 40%

Ankle Instability

Cartilage strains increase 21-27%

Bischoff, J Biotech 2010

Ankle Cartilage

Average Talus thickness- **1.2mm**
(Hip 1.6mm / Knee 2.2mm)

The most congruent joint
Highest compressive modulus
-Simon WH et al, JBJS 1974

Ankle Cartilage is thin
Thin=Less Elastic
More susceptible to cartilage & subchondral injury

↑ Load per Area
-5x body weight
-Force= 2,900N

Osteochondral lesion locations

Historically

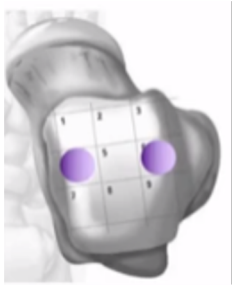
Posterior medial/ Anterior lateral

Barnes CJ and Ferkel RD. Foot Ankle Clin 2003

More Recently

Central lesions (medially and laterally)

Raian SM, et al. Foot Ankle Int 2007



Mechanism

Dorsiflexion and Inversion - lateral more shallow lesions

Plantarflexed and inverted - deeper medial lesions associated with an axial load

Osteochondral Lesions of the Talus

Cartilage +\- Bone Deficit

Reactive bone edema

Clinical presentation

Treatment

Focal Defect

Reactive Edema

Osteochondral Lesions of the Talus OLT

Evolution

1888 (Konig) 1st mentions - Osteochondritis dissecans

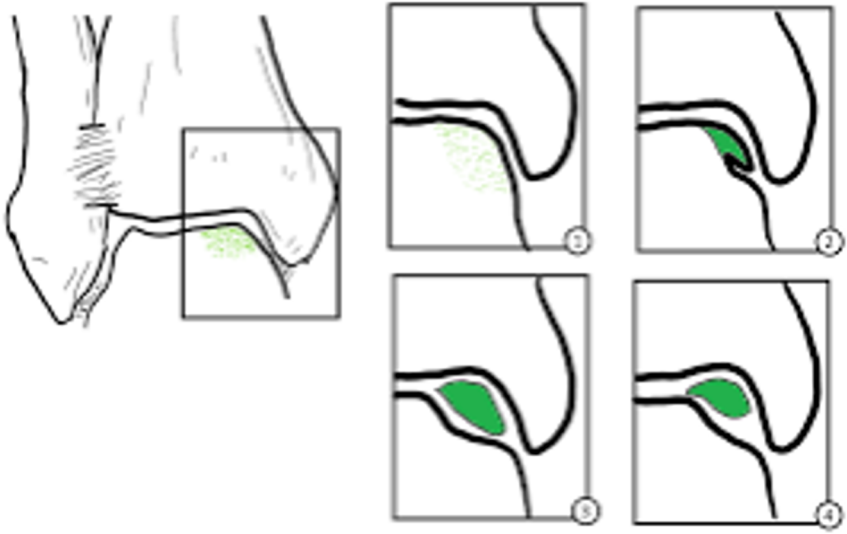
1922 (Kappis)- Osteochondral lesion of the ankle

1959 (Berndt and Harty)- Xray Classification OCD

1990 (Ferkel)- CT Classification OCD

1999 (Hepple)- MRI Classification)OCD

Berndt and Harty



Stage 1: Compression border

Stage 2: Incomplete Detachment of fragment

Stage 3: Complete detachment, no displaceme

Stage 4: Displaced fragment/loose body

Ferkel (CT)

Stage 1: Cystic lesion

Heppel (MRI)

Stage 5: Subchondral bone cyst formation

Symptoms

History- often active individuals, history of significant ankle trauma within 1 year of symptom onset

The majority of patients are 20 to 40 years old, M>W

Presentation- nonspecific ankle pain, swelling, mechanical symptoms of clicking and locking

+/- instability *

Pain with loading; maximum dorsiflexion

Work Up

Xray

Ct/MRI

Diagnostic injection

(+/- Fluoro/US)

Treatment

Conservative/Symptoms management:

Protective WB or NWB

Activity Modification

Gradual restart to impact

Gradual RTS

Reimaging

Injection(PRP, steroid)

Physical Therapy

Asymptomatic or minimally symptomatic OLT did not progress over time when treated nonoperatively

Klammer G et al. FAI 2015

Success rate 20-50% by conservative treatment for symptomatic OLT

Zengerink M, Van Dijk NC, et al. KSSTA 2010

Treatment

Cartilage Repair

Microfracture

Denovo

Biocartilage

Cartilage Replacement

Autograft

Allograft

Cartilage Regeneration

ACI - Autologous cartilage implantation

MACI- Matrix induced

Treatment

Goal of Surgery

1. Restore articular surface
2. Seal the subchondral bone
3. Improve symptoms
4. Decreased adjacent cartilage wear

Surgery Indications

Acute- Displaced / Unstable lesion

Symptomatic Stage 3 and 4

Persistent pain in stage 1 and 2
(failed conservative treatment)

Arthroscopic Surgery

Minimally Invasive

Excellent Diagnostic modality

MOST of the ankle can be visualized

No concern for malleolar non unions

Open Surgery

Arthrotomy

Malleolar osteotomy

24% of the talar dome could not be accessed perpendicular arthroscopically in without an osteotomy.

Talar dome access for osteochondral lesions

Dawson Muir [1], Charles L Saltzman, Yuki Tochigi, Ned Amendola

Microfracture

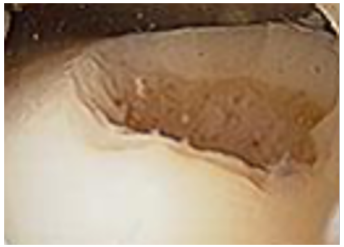
Marrow Stimulation (2-4mm depth)

Unstable cartilage is removed

Create a stable “cup”

Preserve the subchondral plate

Results in the production of fibrocartilage (different mechanical characteristics)



Infiltration of mesenchymal cells

Awl

Marrow holes

fibrin clot forms within the defect →inflammatory response →release of cytokines and growth factors→ fibrocartilage

Microfracture Outcomes

FAI FOOT & ANKLE INTERNATIONAL

Tol JL et al, Foot and Ankle Int. 2000

Average lesion size= 7mm

Meta-analysis 32 studies; 1966-1998

Success of Osteochondral lesion surgery

-Excision, curettage, drilling: 85% (good to excellent outcomes)

-Excision, curettage: 78%

-Excision only: 38%

Microfracture Outcomes

Short Term

Saxena and Eakin, AJSM 2007

26 athletic patient (14 “elite, 6 footballers)

AOFAS score 54.6 → 94.4

96% return to sport at mean 15.1 weeks

Zengerink et al, KSSTA 2010

85% success in short-term

Midterm Outcomes of Bone Marrow Stimulation for Primary Osteochondral Lesions of the Talus

Toale J, Shimozono Y, Kennedy JG et al.

OJSM 2019

15 Studies

AOFAS 89.9 at 6 years

MRI outcomes

-48% complete filling, 76% surface damage

-Reoperation 6%

Microfracture Outcomes

Long Term

Ferkel et al, AJSM 2008

35% deterioration in outcome scores at 5 years

Choi SW et al, AJSM 2020

165 ankles, 6.7 year F/U

13.3% repeat surgery due to symptoms

Van Bergen et al. JBJS 2013

50 patient, 12 year F/U

78% good-excellent Ogilvie-Harris score

AOFAS avg 88 pts

12% repeat surgery

33%: 1 grade increase in OA on xray

Plat et al. KSSRA 2016

82 patients, 10 years F/U / AOFAS 85.5

57% symptomatic / 33%: 1 grade increased OA on xray

Variables for Success

Historically Size < 1.5 cm

Recent literature shows BMS success in lesions < 1 cm, 100 sq cm

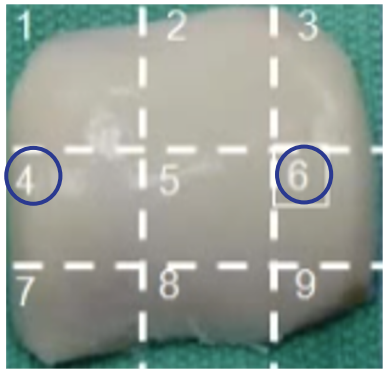
Patient age / Patient biology

Location (contained vs uncontained)

SCB health / cystic formation

Raikin et al FAI 2007

Ant-Med



Post Lat

Zone 4- 53%

Zone 6- 26%

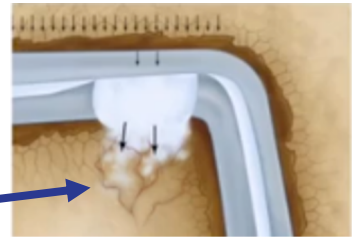
Bone Marrow Edema
Subchondral Bone Cyst

Cartilage damage/thinning/loosening increases focal stress to the underlying SCB

Osseous resorption and cyst formation

Walker WT, Fortier LA et al. AJVR 2020

Cysts develop by repetitive increased loading to the SCB



BMS alters the microarchitecture of SCB, which may degenerate over time.

Degradation of SCB influences outcomes

Bone Marrow Edema
Subchondral Bone Cyst

Function:

Support

Elasticity for shock absorption during joint loading

*Cartilage only absorbs 1% of shock

Change in the Treatment paradigm

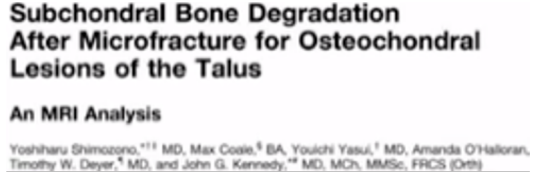
Subchondral cancellous bone supports the cortical bone-like subchondral plate

Madry et al 2010

Subchondral bone is a dynamic and responds to increased demands or loads by increasing its density and mineralization

Bone Marrow Edema
Subchondral Bone Cyst

Microfracture/ BMS techniques result in abnormal SCB in 100% of cases



Subchondral Bone Degradation After Microfracture for Osteochondral Lesions of the Talus

An MRI Analysis

Yoshiharu Shimozono,*†‡ MD, Max Coole,§ BA, Youichi Yasui,† MD, Amanda O'Halloran, Timothy W. Deyer,* MD, and John G. Kennedy,*¶ MD, MCh, MMSc, FRCS (Orth)

The Subchondral Bone Is Affected by Bone Marrow Stimulation: A Systematic Review of Preclinical Animal Studies

SAGE

Dexter Seow[1,2,*], Youichi Yasui[1,2,*], Ian D. Hutchinson[1,4], Eoghan T. Hurley[1,2], Yoshiharu Shimozono[1,2,3], and John G. Kennedy[1]

SCB was not restored after microfracture
SCB quality associated with outcomes at midterm (4-5 years)

Change in the Treatment paradigm

Improving Bone Marrow Stimulation

Decreased awl size (1mm) improved articular repair, and decreased SCB damage

BMAC - Improved outcomes, and decreased SCB resorption

Improving Bone Marrow Stimulation

Improving Biology

Biocartilage: Allograft cartilage extracellular matrix
Type II collagen
Proteoglycans
Cartilaginous growth factors

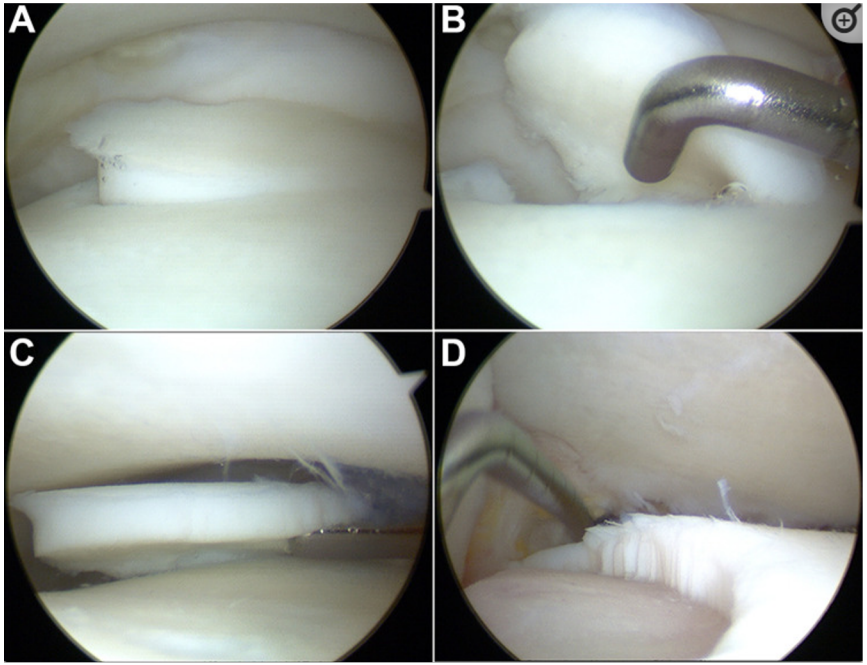
Shimozono Y, Kennedy JG et al

Microfracture & BMAC vs Biocartilage & BMAC (48 total 24/24)

Lesion ingrowth
MF= 46.5%
Biocartilage 87.5%

Trend

Less destructive cartilage repair



10 NBA players with OLT

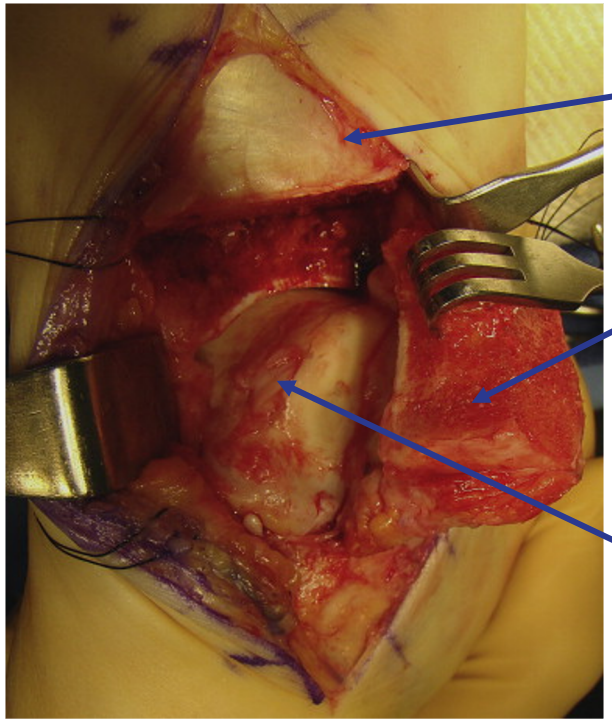
Arthroscopic debridement of an OLT without drilling or microfracture, there was a high rate of return (10/10)

Orthop J Sports Med. 2021 Jan; 9(1): 2325967120970205.
Published online 2021 Jan 6. doi: 10.1177/2325967120970205
PMCID: PMC7802091
PMID: 33457433

Athletic Performance in the National Basketball Association After Arthroscopic Debridement of Osteochondral Lesions of the Talus

Christopher Sheu, MD* and Richard D. Ferkel, MD†‡

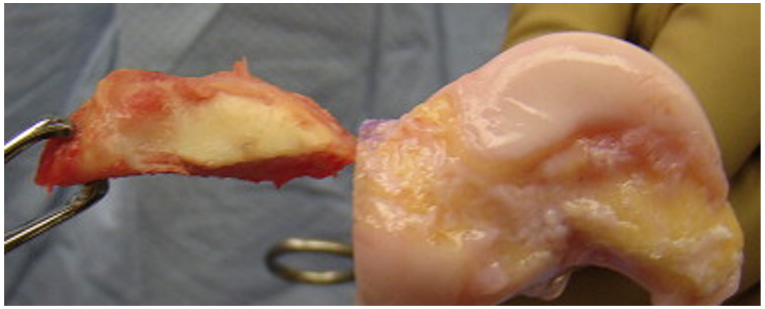
Osteochondral Allo/Autograft Transfer

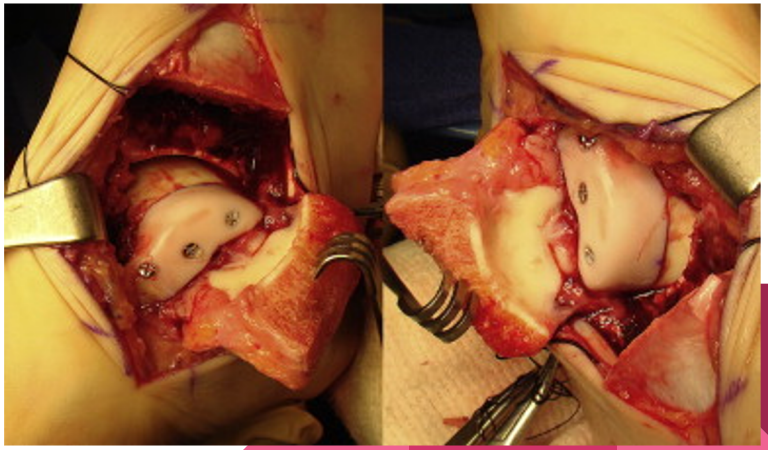


Medial Tibia

Medial Malleolus

Medial Talus





OATs Outcomes

Hangody et al. AJSM 2010

39 patients, 9.6 years F/U

92% Good to Excellent outcomes

Shimozono Y, Kennedy JG et al. KSSTA 2018

Systemic Review

11 Studies, 500 Ankles

63 months F/U

87% Good or Excellent AOFAS Scores

Failure 1%: Ankle fusion or Revision

Graft Positioning

1mm sunken - 0.4mm proud

Graft 1mm proud increase contact pressure up to 675%

Fansa AM, Kennedy JG et al. AJSM 2011

OATs Outcomes

Athlete Outcomes

Fraser, Prado and Kennedy et al. KSSTA 2016

36 athletes, mean 5.9 year F/U

AOFAS: 65.5 → 89.4

RTS: 90% professional at 12 month F/U

Nguyen A, Calder JDF, et a. AJSM 2019

38 athletes

87% returned to sports at pre injury level

OATs

Predictors of Outcomes

Previous microfracture

Uncontained

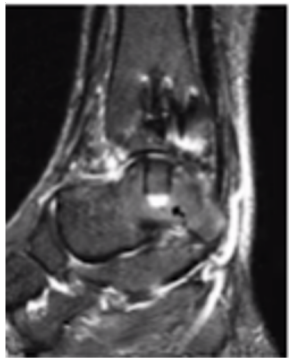
BMI

Ross AW, Kennedy JG et al. Arthroscopy 2016

22 Primary AOT vs 54 secondary AOT after failed BMS

Significant difference in FAOS Score

Primary 83.2 vs Secondary 72.4



Magnetic Resonance Imaging Evidence of Postoperative Cyst Formation Does Not Appear to Affect Clinical Outcomes After Autologous Osteochondral Transplantation of the Talus

Ian Savage-Elliott, B.A., Niall A. Smyth, M.D., Timothy W. Deyer, M.D., Christopher D. Murawski, B.S., Keir A. Ross, B.S., Charles P. Hannon, B.S., Huong T. Do, M.S., and John G. Kennedy, M.D., M.Ch., M.M.Sc., F.R.C.S.(Orth)

64.8% cyst occurrence at 6 mos

92% of pts with cysts asymptomatic

Autograft Concerns

Donor Site morbidity

Single case series of 12 pts reported 50% with knee pain

Hangody et al. AJSM 2011

Shimozono Y, Kennedy JG et al. CORR 2019

Meta-Analysis

1055 pts , mean F/U 41 mos

DSM 5%

Allo/Autograft Summary

For Large lesions >10mm, 100mm squared

Excellent outcomes in general population and athletes

Previous Microfracture = worse outcomes

Bone marrow edema

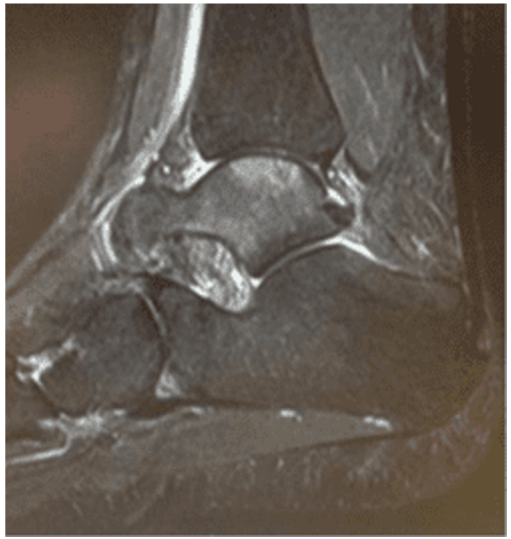
MRI Finding

Bone Marrow Lesion

T2 (fat suppressed)- fluid sensitive

Bone marrow lesions contain areas with less mineralization, increased fibrosis, necrosis, and microfractures.

Bone marrow edema



Etiology

1. Acute trauma (bone bruise)
2. AVN
3. Stress/Insufficiency fracture
4. OA

Bone marrow lesions are associated with increased pain

Bone marrow edema

BMLs are structural changes of the subchondral bone

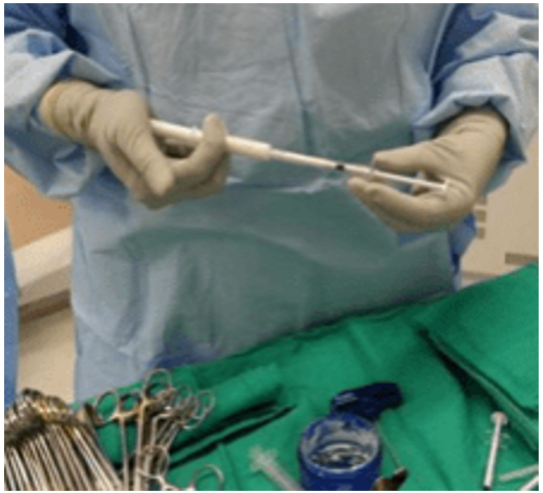
Precursor of advancing cartilage destruction, subchondral bone attrition, and acceleration of joint deterioration/collapse

Subchondroplasty

"Theory" Stabilize the subchondral bone, limit subchondral bone attrition

Injection of calcium Phosphate between the subchondral cancellous trabeculae without damaging the existing bone scaffold

Bone marrow edema



Technique

Arthroscopic assessment

Fluoroscopically guided cannula insertion

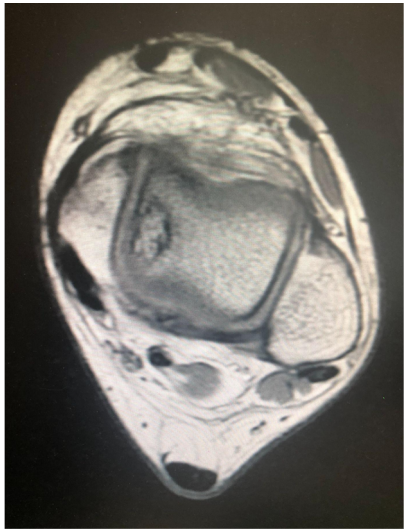
Benefits

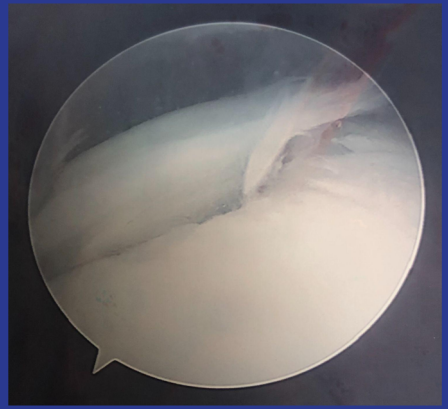
minimally-invasive

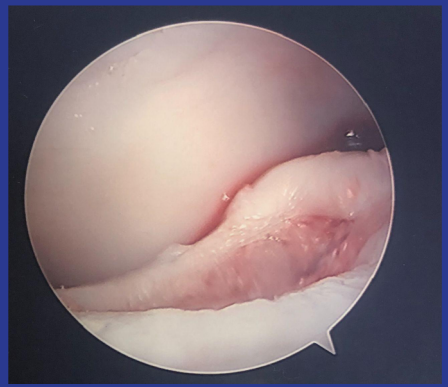
prompt return to WB (1- 2 weeks) while restoring pathologic subchondral bone

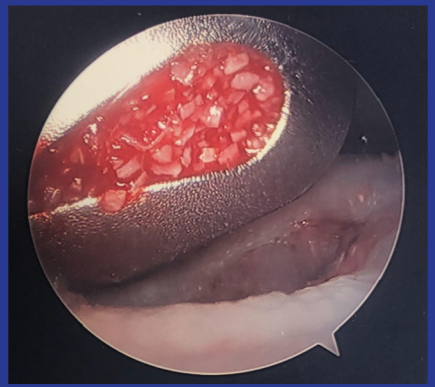
Traditional Microfracture

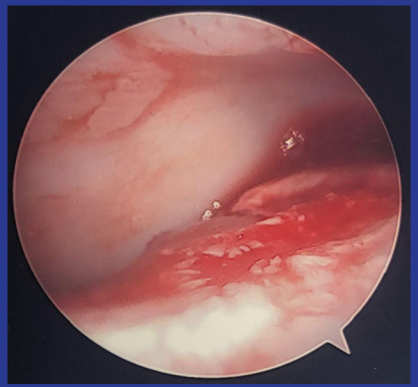
Case 1



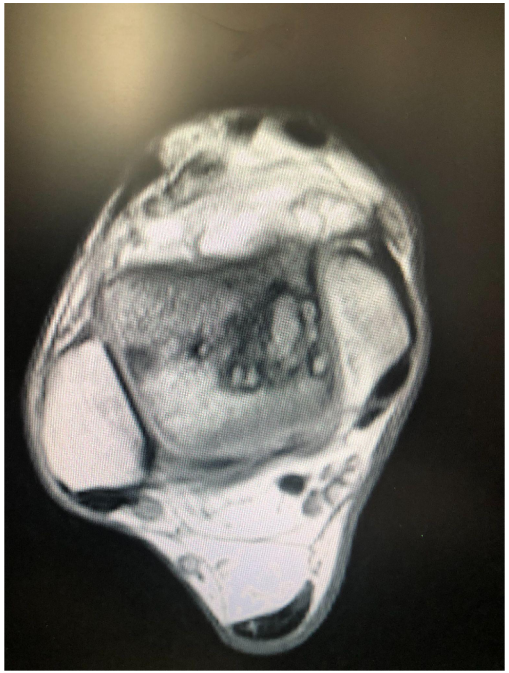


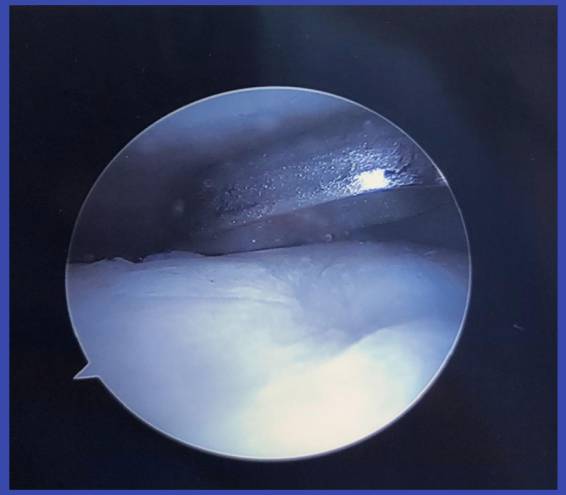


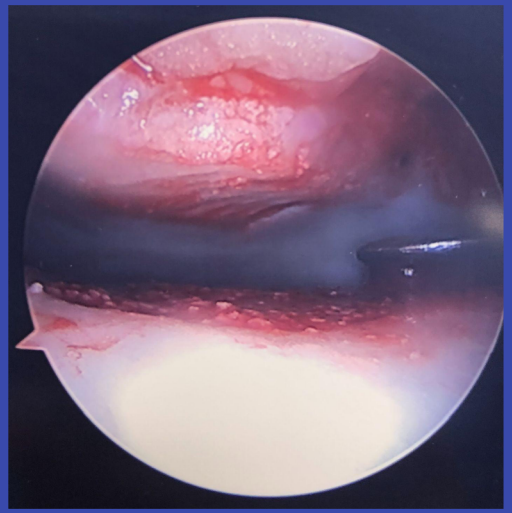


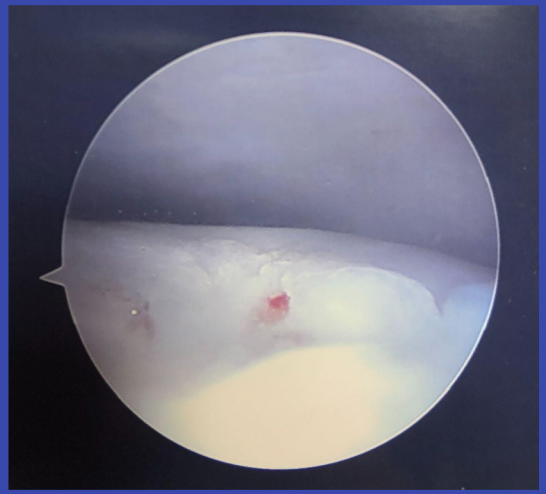


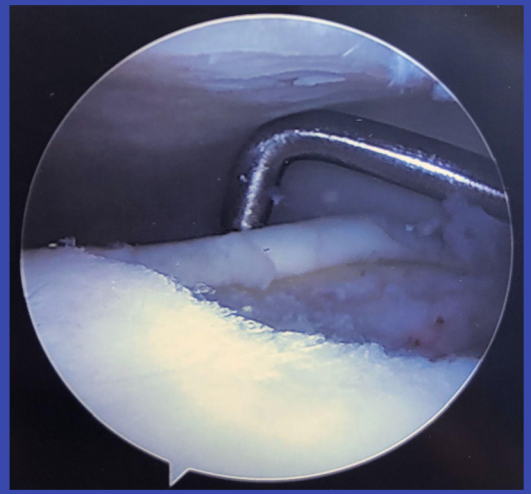
Case 2

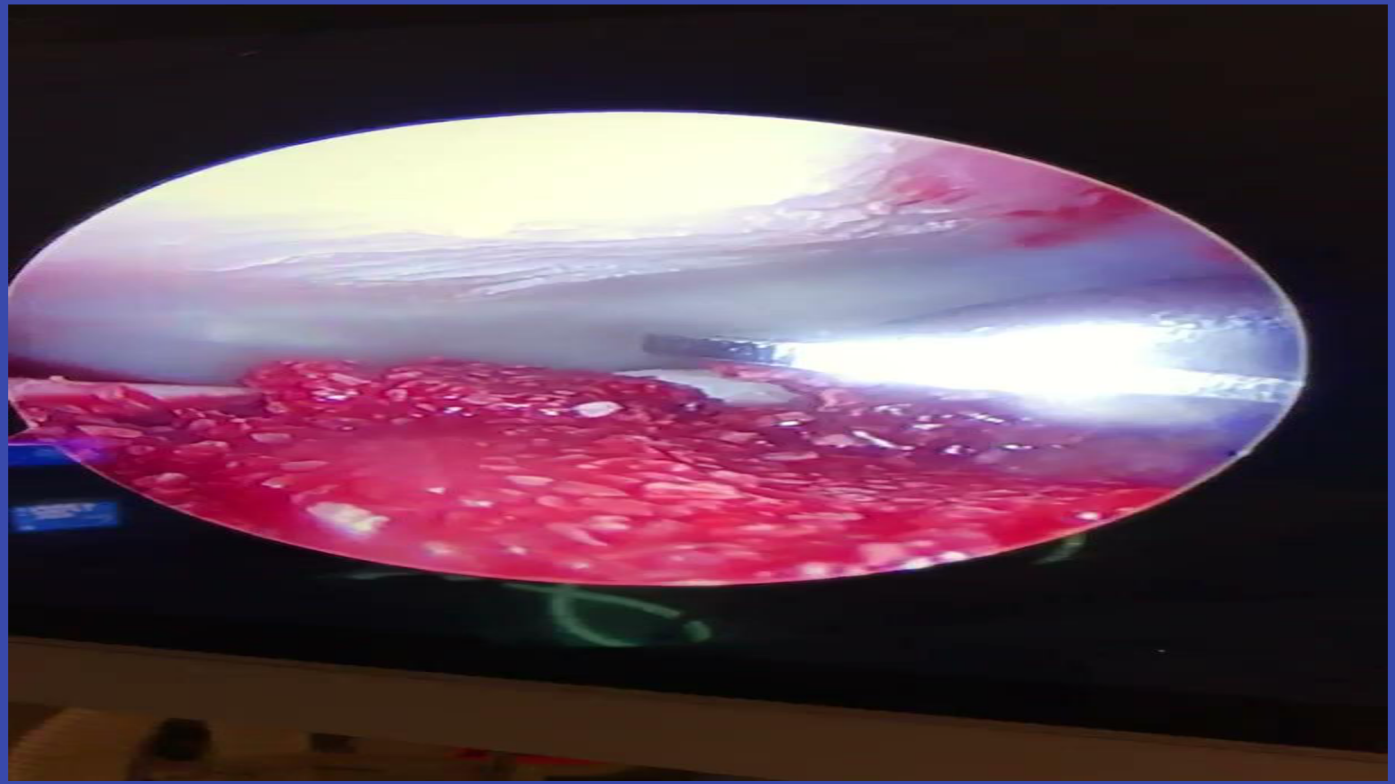


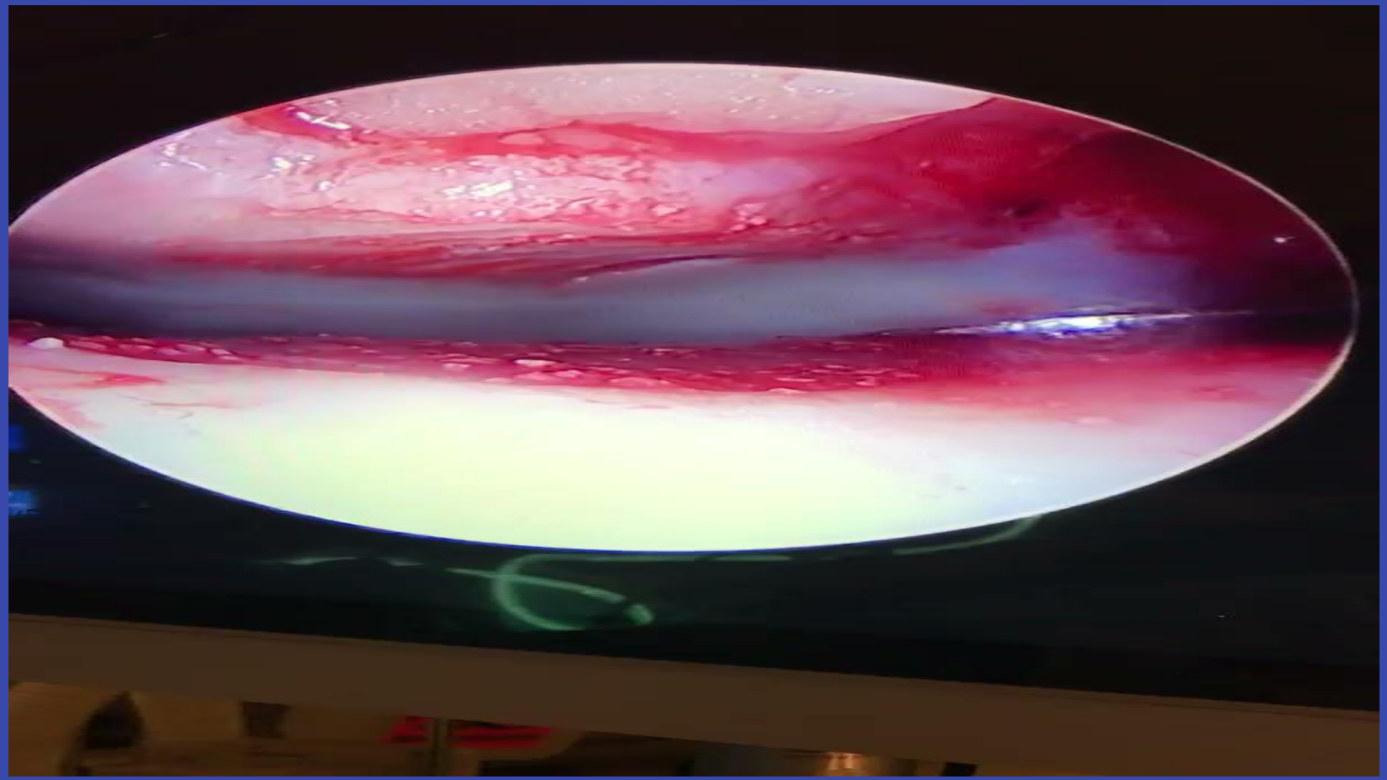




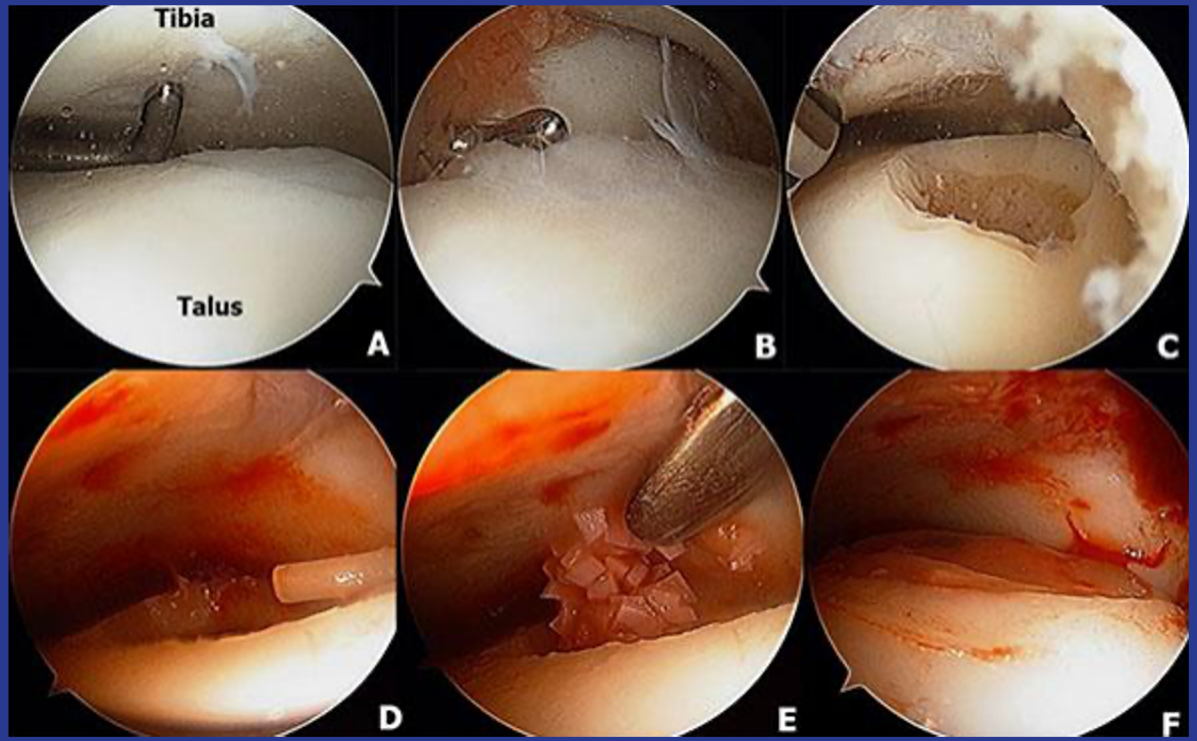








Juvenile Particulate cartilage



Post Operative Protocol

Motion at 2 weeks

Physical Therapy
-joint mobilization, lateral ankle strengthening, proprioception
-jumping/dynamic activity @ 3+ months

Microfracture- WB protection
NWB x 3-6 weeks
WB in boot x 3 weeks

Medial Malleolar Osteotomy/Allograft
NWB x 6-8 weeks
WB in boot x 4 weeks

Conclusion

Treatment for osteochondral lesions of the talus continues to evolve

Nonoperative treatment is the ideal starting point
Healing properties of articular cartilage cause non-operative treatment to be limited.

Microfracture, drilling, and other bone marrow stimulating techniques yield positive results for smaller lesions.
Cell base repair, PRP and hyaluronate has shown promising results to enhance the efficacy of marrow stimulation.

Osteochondral Grafting is generally indicated for larger lesions.

The SCB is the foundation for any cartilaginous repair.

Take Home

Adequate work up

- Xray, CT, MRI, diagnostic injections

Exhaust Conservative treatment

Symptoms: Pain, Swelling, clicking/popping

Surgical Algorithm

<1cm Microfracture

>1cm allo/autograft

Adjuncts: PRP, BMAC

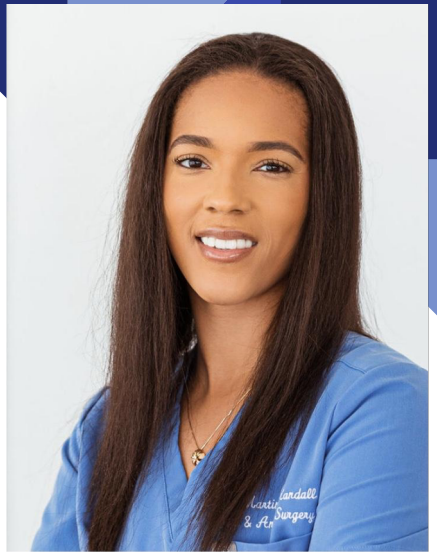
Special considerations

-Bone graft for subchondral cyst/edema

Thank You!

martina.randall@gmail.com

@doctorrandall



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