Original Research

Hallux Rigidus: The Subchondral Bone Complex – It's Role in the Long-Term Success of Surgical Efforts

Michael Coughlin, MD, Richard Ferkel, MD, Sam Adams, MD, Bob Baravarian, DPM, Kerry Zang, DPM, Daniel Schulman, DPM, Richard Silverstein, DPM, Shah Askari, DPM, Eric Naasz, DPM, Calvin Rushing, DPM, and Brian Benson, DPM.
Investigation performed at North Valley Surgical Center, Scottsdale, Arizona, United States

---

Abstract:
Advanced hallux rigidus associated with subchondral insufficiency or following microfracture from articular thinning and/or cartilaginous defects have been shown to have inferior clinical outcomes after reparative techniques such as bone marrow stimulation or even metatarsal resurfacing implant arthroplasty procedures. Autologous osteochondral transplantation has been viewed as an alternative for treating these lesions, but donor-site morbidity and cost has limited its application. Early clinical outcomes following treatment of subchondral insufficiency and focal articular defects with intra-osseous stabilizing platform technology has been encouraging, and these outcomes have been achieved at a lower cost and without donor-site morbidity.

Definition:
Subchondral insufficiency fractures (SIF) are a type of stress fracture which occurs below the cartilage on the weight bearing surface of a bone. SIF occur when normal physiological forces are repeatedly applied to an area of bone compromised by non-tumorous disease, resulting in fracture.

Introduction:
Hallux rigidus is a general term which can be used to describe several conditions involving the first metatarsal-phalangeal joint complex resulting in articular joint disease deterioration with cartilage thinning or even the complete loss of cartilage, proliferative bone formation involving both the head of the first metatarsal and base of the proximal phalanx. Pain and reduced motion are classic hallmarks of hallux rigidus. Degenerative arthritis (DJD) is a debilitating disease of the musculoskeletal system that presents with progressive degeneration of both articular cartilage and subchondral osseous tissues, causing pain and adversely limits functional capabilities. It is estimated that degenerative joint disease affects close to 30 million people in the United States alone. (1, 2) A comprehensive clinical evaluation of the entire first metatarsal-phalangeal joint complex, including imaging in conjunction with an in depth history and physical examination, can assist a surgeon to formulate an appropriate clinical plan.

Perceived Problem:
Controlling the post-operative progression of degenerative osteoarthritis, and the pain associated with this progression, has been an ongoing challenge for musculoskeletal surgeons. To date, it has been reported

that bone marrow stimulation procedures, as a primary treatment strategy, have shown a reparative effect for subchondral insufficiency and osteochondral lesions either with or without the presence of small cysts. With the lack of healthy

subchondral bone and the paucity of native stem cells, the healing response after bone marrow stimulation for patients is poor. [3]

As foot and ankle surgeons we routinely perform realignment osteotomies of the first ray to repair hallux valgus deformities. During the procedure, inspection of the articular cartilage may indicate the presence of an area of dimpling of the articular surface, or the presence of full thickness articular erosion. Heretofore, our belief was that the realignment of the first ray, including re-establishment of congruity of the metatarsal-phalangeal joint, would be sufficient to provide a satisfactory surgical result; any articular defect was felt to be insignificant to the overall success of the realignment procedure. While these procedures were performed meticulously, and the osteotomies healed without incident, many patients continued to report intra-articular pain.

Following these patients' long term, have led us to understand that these lesions involving the articular cartilage and associated subchondral region can cause substantial symptoms, such as pain, swelling, joint stiffness, and reduction of physical activity. This may result in quality-of-life issues and progressive articular joint deterioration.

With long term follow-up, progressive articular degenerative changes of the first metatarsal-phalangeal joint became evident. This occurred while the immediate post-operative radiographs showed excellent surgical alignment without any evidence of degenerative disease.

Could it be that subchondral instability and/or microfracture syndrome was present yet undetected?

Degenerative joint disease in hallux rigidus:
The hallmark of hallux rigidus is degenerative arthritis presenting with pain, loss of joint motion, disability and is a progressive degenerative joint disorder. [4] The treatment of choice for this condition for years has been a cheilectomy/bunionectomy with or without microfracture with the hope of negating the need for joint stabilization, fusion, or joint replacement procedures. These patients clearly have articular defects with subchondral disease and instability and long-term results have been disappointing, with the need for subsequent surgery. [5]

Causation:
Multiple factors may play a role in the development of hallux rigidus. These include but are not limited to trauma, degenerative and inflammatory arthritis, genetics, faulty biomechanics, etc.

Characteristics of Degenerative Osteoarthritis in Hallux Rigidus:
The first metatarsal-phalangeal complex undergoes structural changes of cartilage thinning articular joint destruction, osteophyte formation, synovial and capsular inflammation, and insufficiency of subchondral osseous structures. [6] It is the failure of the subchondral complex that is oftentimes undetected and leads to procedure failure. Subchondral bone sclerosis, together with progressive cartilage degradation, is widely considered as a hallmark of osteoarthritis (OA).
The subchondral bone is hypo-mineralized, due to abnormal bone remodeling and histopathological changes in the subchondral bone have also been detected, including microdamage, bone marrow edema and bone cysts formation.
[7,8])

Risk Factors for Subchondral Bone Dysfunction:
OA is a progressive degenerative joint disease with different etiologies, and multifaceted risk factors have been suggested for its onset of OA, which include genetic predisposition, gender, aging, obesity, physical activity, previous joint injury, joint malalignment, and abnormal joint shape. [9,10]

Recognizing the Total Problem:
This discussion recognizes the two important and distinct components of the joint: the articular cartilage and the subchondral bone, and their role in the success or failure of surgical efforts.[11]

Subchondral bone can be separated into two distinct anatomic entities: the sub- chondral bone plate and the subchondral trabecular bone.

The subchondral bone plate lies directly beneath the calcified cartilage and contains the thin cortical lamella. The bone plate is porous and contains channels which allows access to the articular cartilage by arteries, veins, and nerves from the subchondral trabecular bone.

Arising from the subchondral bone plate are the supporting trabeculae, which comprises subchondral trabecular bone, together with deeper bone structure. Subchondral trabecular bone exerts important shock-absorbing and supportive functions in normal joints and may also be important for cartilage nutrient supply and metabolism. [12] Relative to the subchondral bone plate, subchondral trabecular bone is more porous and metabolically active, containing blood vessels, sensory nerves, and bone marrow. [13] Subchondral bone is a dynamic structure and adapts to the mechanical forces imposed across the joint. Mechanical stress also modifies the contour and shape of subchondral bone by means of bone modeling and remodeling.

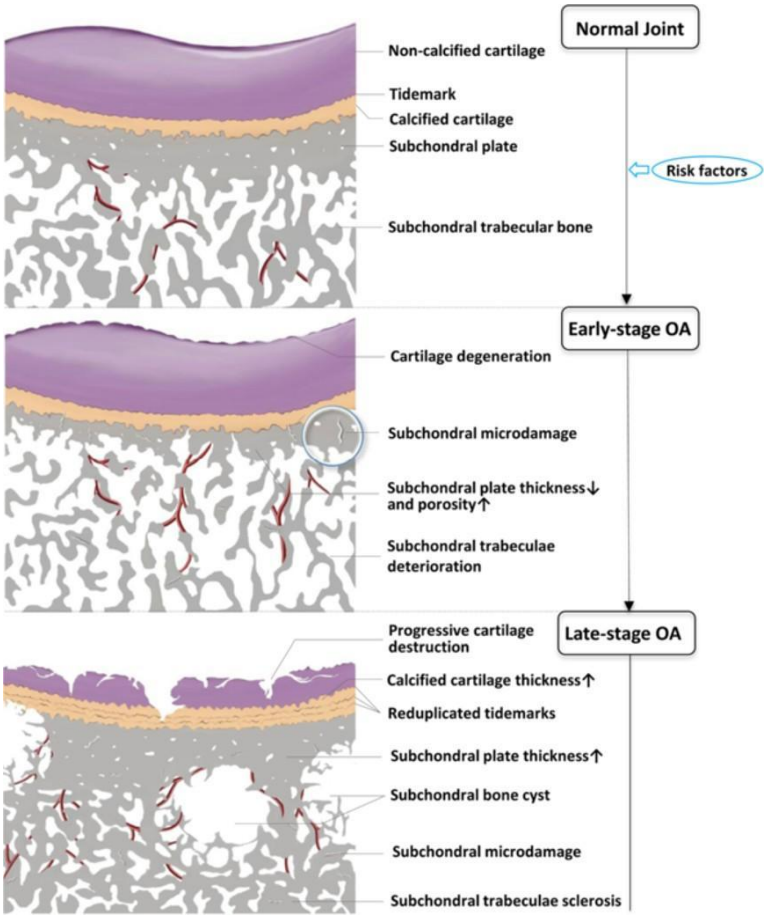
Functional Interaction between articular cartilage and subchondral bone:

The articular cartilage covers the subchondral bone and helps to maintain the normal chemical balance and function within the joint. It contains both superficial non-calcified and deep calcified cartilage which are separated by the tidemark. The tidemark provides a transitional structure between the soft and hard cartilage. [14] This functional unit forms the osteochondral junction. The osteochondral junction is complex and consists of a deeper layer of
non-calcified cartilage, the tidemark, calcified cartilage, the cement line, and subchondral bone. [15] Any alteration of any one of the components will alter the functions of any of the other parts. All the components work in concert with each other. Subchondral bone provides a supportive platform for the overlying articular cartilage and distributes the load in an appropriate manner. When there is degenerative and articular joint destructive disease present there is a significant increase in the load that is transmitted through the articular cartilage to the subchondral osseous structures. [16,17]

Microdamage of Articular Cartilage and Subchondral Bone in Osteoarthritis[18]

(*Subchondral bone in osteoarthritis: insight into risk factors and microstructural changes

Guangyi Li1,2, Jimin Yin1, Junjie Gao2, Tak S Cheng2, Nathan J Pavlos2, Changqing Zhang1* and Ming H Zheng2*0



Sequela of Subchondral Insufficiency Syndrome:
When functional alignment and joint motion become out of synch and dysfunctional, the pathologic process of bone loss develops as the unstable articular surfaces track unevenly. This functional abnormality leads to joint space narrowing, degeneration and thinning of the articular cartilage. This leads to a progression of the biomechanical imbalances and the destructive changes progress.

Could it be that subchondral instability and/or microfracture syndrome was present yet undetected?

It was almost 20 years ago that Dr. Coughlin researched and authored the following classification for hallux

rigidus. Coughlin Classification of Hallux Rigidus

*Grade 0 - DF of 40-60° (20% loss of normal motion), normal radiographic results, and no pain
*Grade 1 - DF of 30-40°, dorsal osteophytes, and minimal to no other joint changes
*Grade 2 - DF of 10-30°, mild flattening of the MTP joint, mild-to-moderate joint narrowing or sclerosis, and dorsal, lateral, or medial osteophytes
*Grade 3 - DF of less than 10°, often less than 10° PF, severe radiographic changes with hypertrophied cysts or erosions or with irregular sesamoids, constant moderate to severe pain, and pain at the extremes of the ROM
*Grade 4 - Stiff joint, radiographs showing loose bodies or osteochondritis (OCD), and pain throughout the entire ROM
(19)

Is it time to revisit this important study? Will we find that subchondral osseous insufficiency is present in Grade 0 or Grade 1? What occurs first, degenerative changes of the articular cartilage or subchondral insufficiency or do they occur simultaneously? At this point do we really know?

All components of the articular, sub-articular/transitional and subchondral structures are functionally interdependent. Any alteration in structure of any component will alter the functions of any other component and all components work in concert with each other. [19]
When degenerative and articular joint destructive disease is present there is significant increase in load that is transmitted through the articular cartilage to the subchondral osseous structures. Subchondral Insufficiency Syndrome can result in articular cartilage loss, progressive pain, and deformity. [20]

Could it be that Subchondral Insufficiency and/or Subarticular Microfracture was present yet undetected?

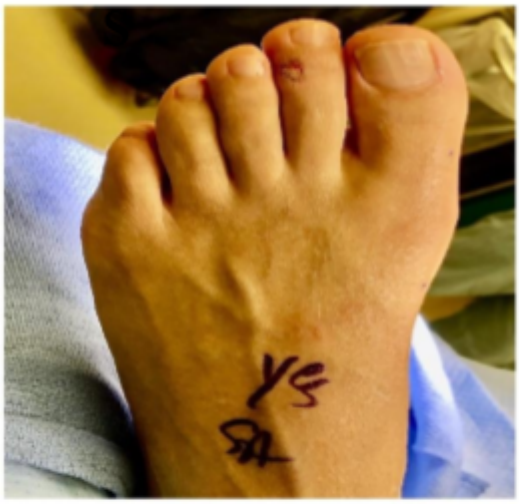
Could this be the answer why surgeons have sub-optimal results when the surgical procedures were accomplished flawlessly and with excellent alignment and all anatomic structures looked great?
Patient history, subjective reporting and objective evaluations are all clinical risk factors for subchondral insufficiency. History:
1. Hypertrophy of great toe joint
2. Progressive loss of motion, first metatarsophalangeal joint
3. Achiness and progressive pain in great toe joint
4. Trauma great toe joint
5. Repetitive stress activities
6. Activity level diminishes

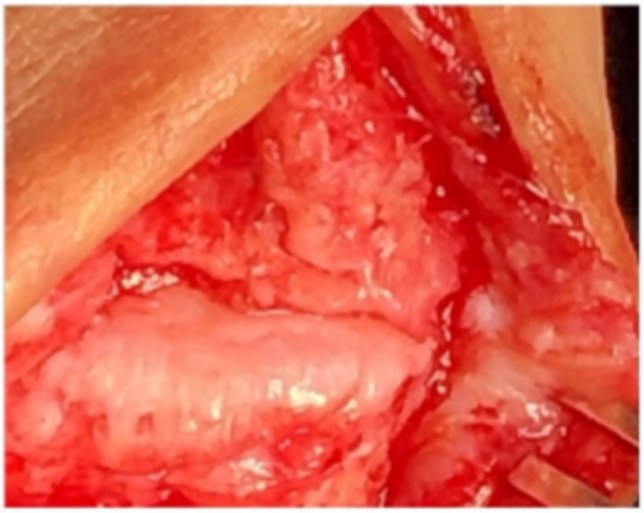
Subjective: Typical patient complaints:
1. Loss of motion of the big toe joint, progressive in nature
2. Localized pain in the great toe joint
3. Painful ambulation
4. Bone spurs – osteophyte formation
5. Burning and tingling pain with pressure on dorsal digital nerve as it passes over the bony prominences
6. Limitations of shoe gear

Objective:
1. Hypertrophic bone – dorsal and dorsal lateral aspect 1st MPJ
2. Pain on palpation dorsal and dorsal lateral aspect 1st MPJ
3. Limitation of motion 1st MPJ
4. Crepitus oftentimes present
5. A positive grind test may be present – pain produced with dorsiflexion and rotation of base of proximal phalanx on the head of the 1st MPJ
6. A positive Tinel's with percussion of the dorsal digital nerve over the hypertrophic bone
7. Compensatory functional gait abnormality
8. Structural abnormality of the 1st ray
9. Biomechanical imbalances

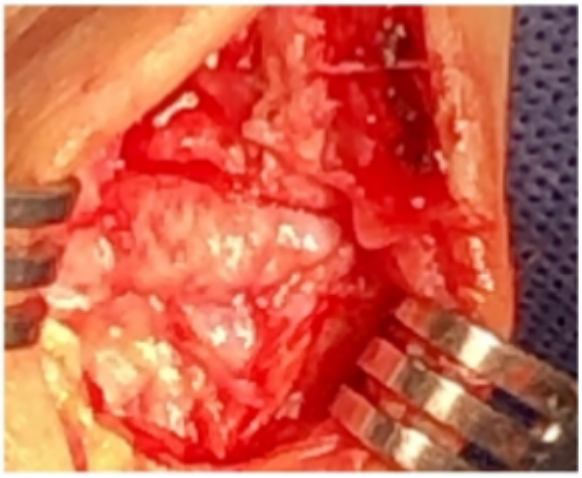
Case Presentation #1:

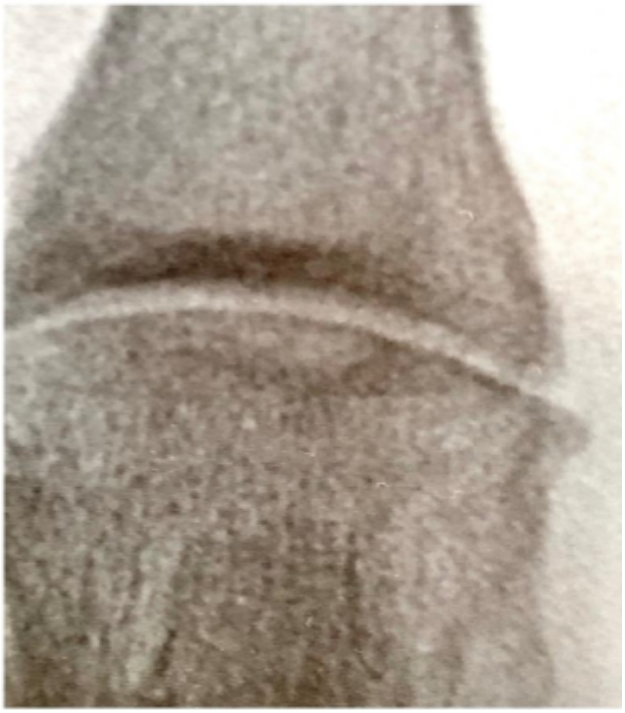
HALLUX
RIDIGU



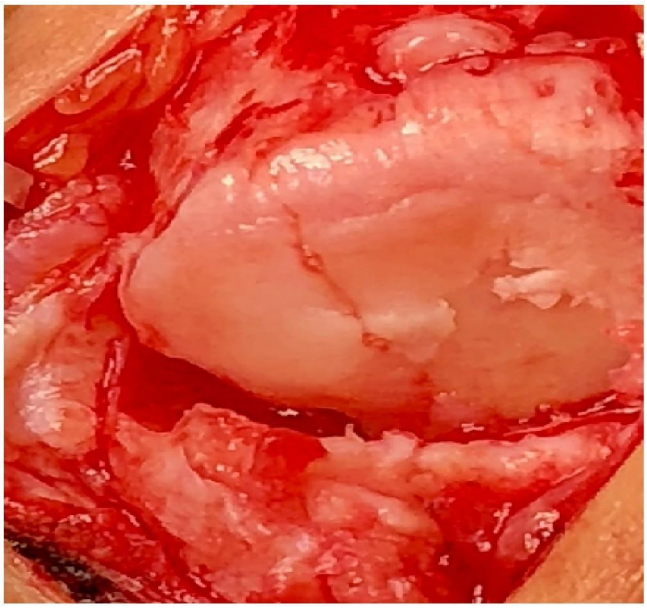


Severe Articular Joint Disease
Articular Loss
Subchondral Insufficiency
Microfracture

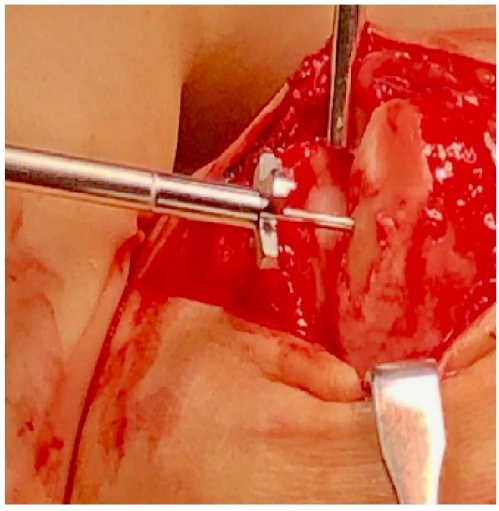


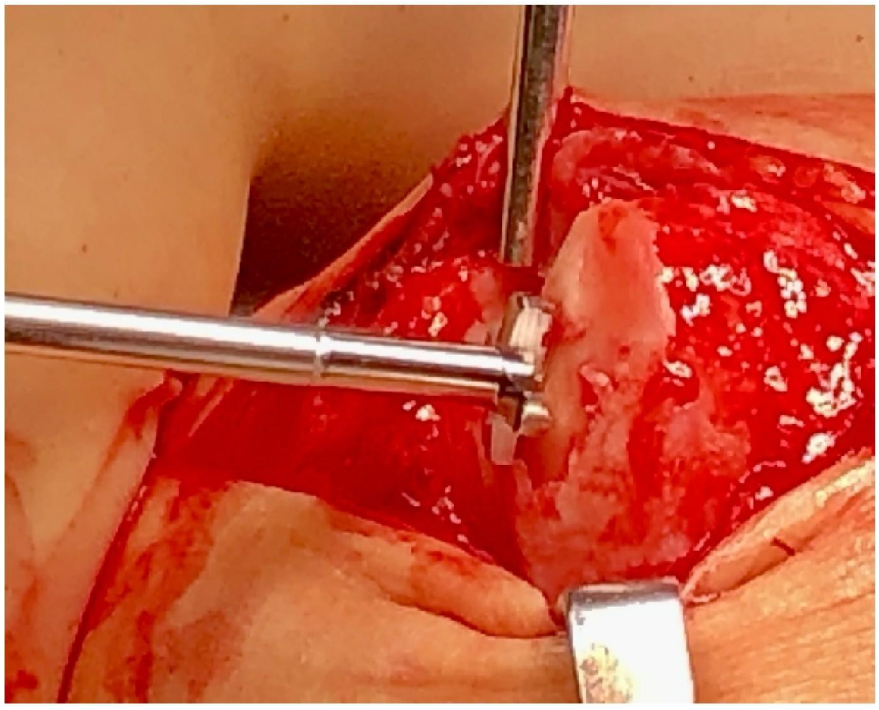


Hypertrophic Bone With Degenerative Erosive Articular Joint Disease

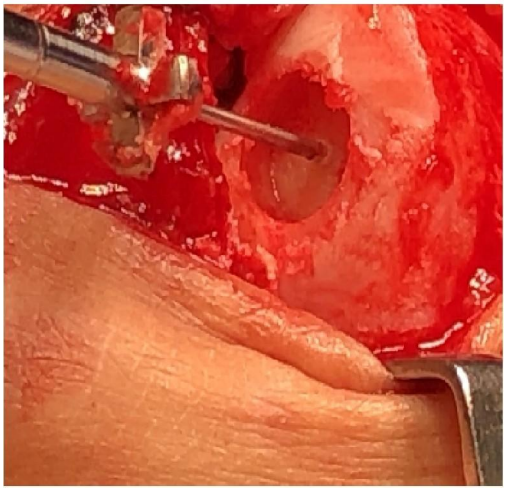


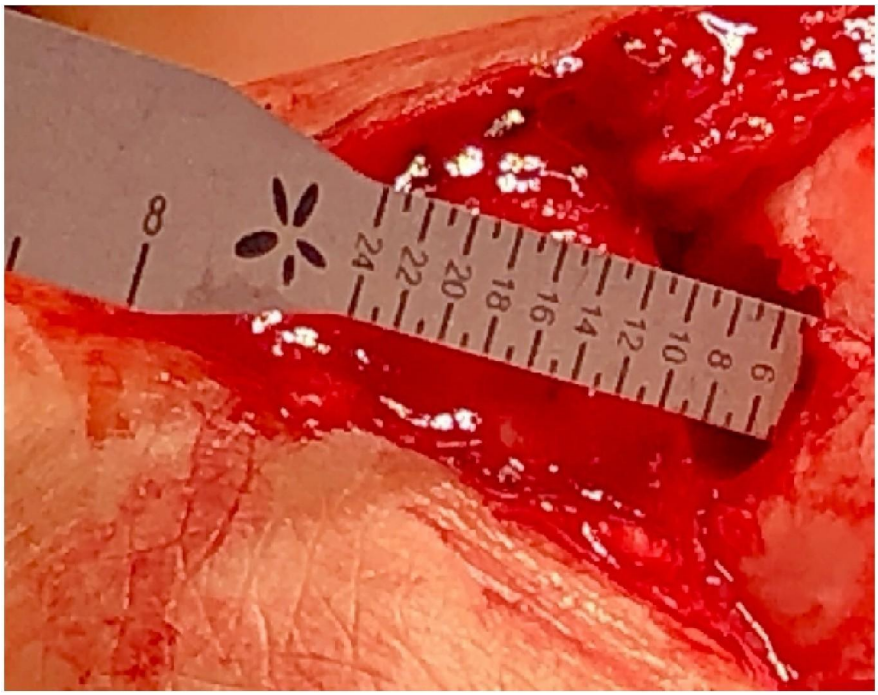
Guide Pin with Countersink



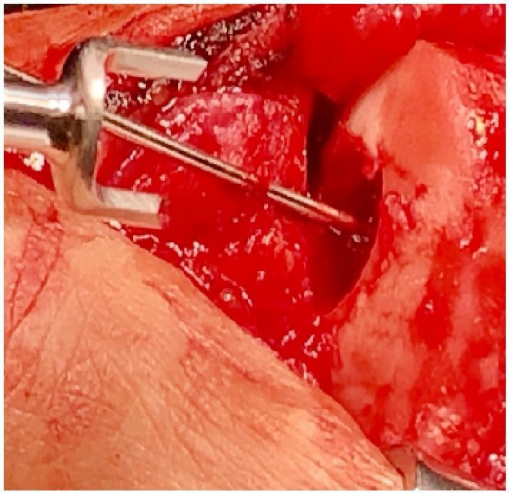


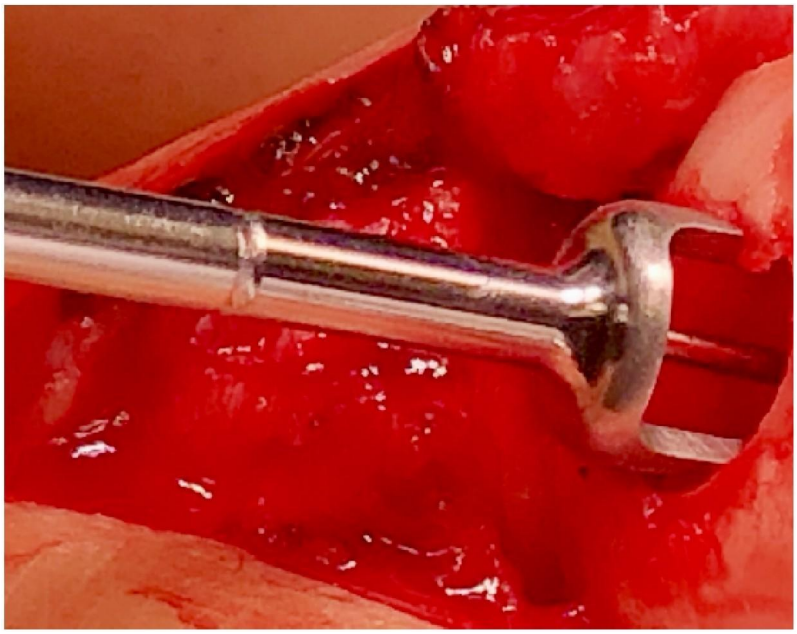
Debridement of Diseased articular cartilage-depth 5mm



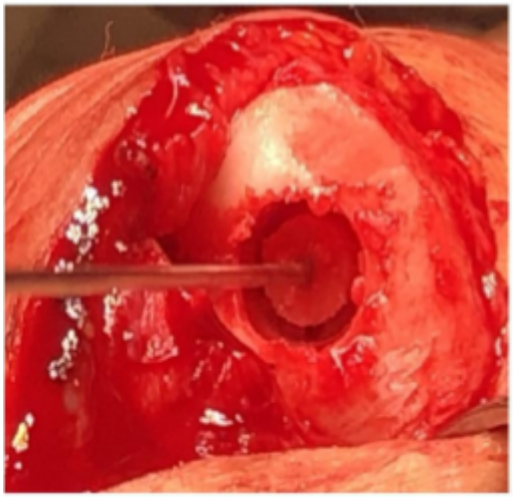


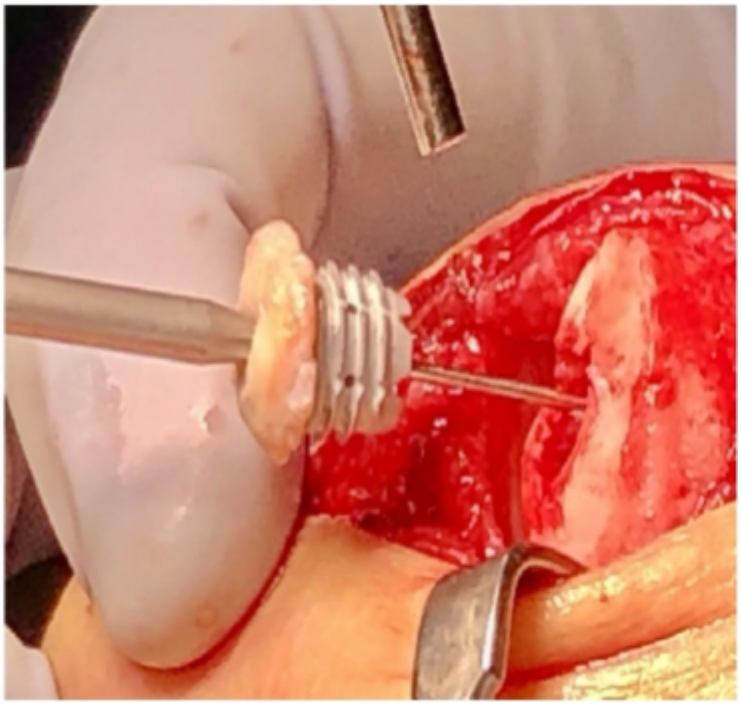
Creating Platform for S-Core



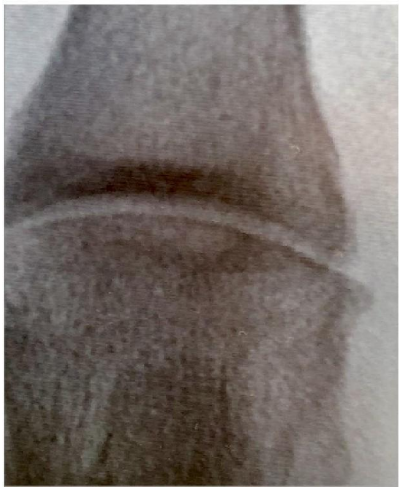


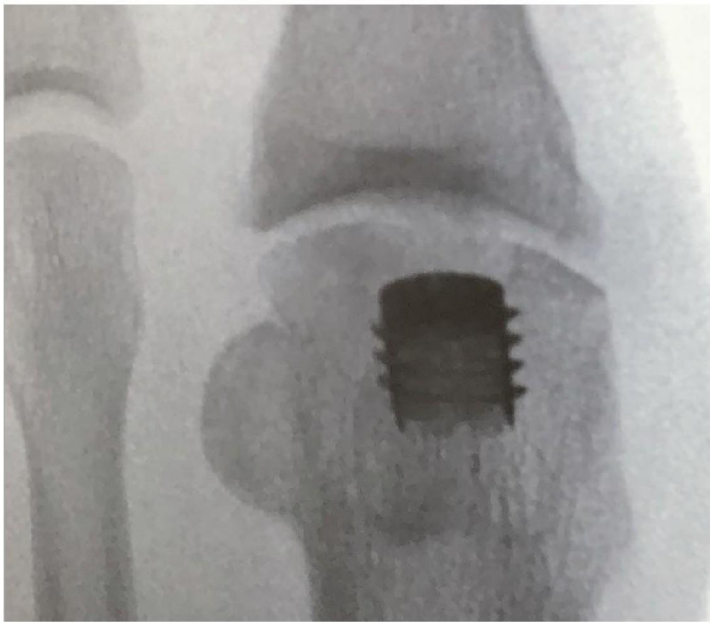
Ready for S-Core



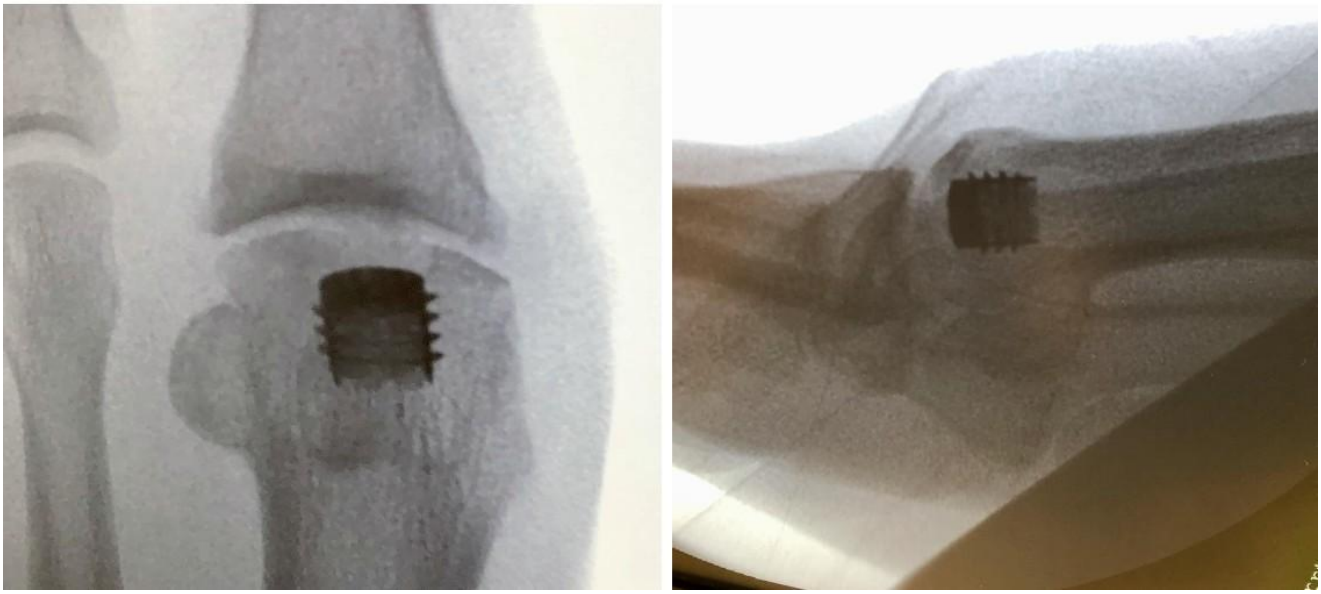


Articular Defect Repaired
& Subchondral Insufficient
Microfractures Stabilized



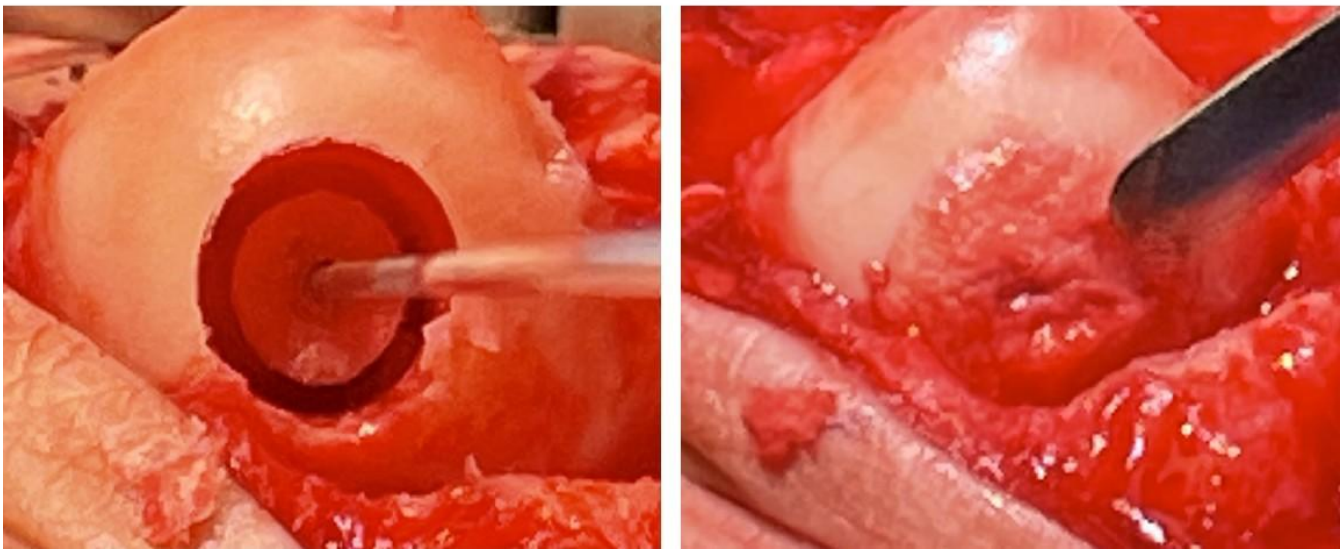


Excellent Alignment 1st MPF & Implant Placement



Case #2:

Insufficiency Secured with Implant + Graft



Conclusions:

Subchondral insufficiency fractures of the foot and ankle were considered a rare etiologic factor in chronically progressive foot and ankle pain. These microfractures were considered a rare complication after foot and ankle surgery and were idiopathic. [23] These subchondral insufficiency deficits, when identified, were treated with
disease-modifying antirheumatic drugs (DMARDs), anti-TNF, analgesics, immobilization, physical therapy, and multiple invasive procedures from micro-drilling to joint replacement/fusion procedures and more. Definitive diagnosis, if established, was often delayed resulting in undesirable outcomes.
Also, insufficiency fractures have been attributed in patients taking DMARDs and further evaluation was not forthcoming. [24] Another significant feature associated with SIFs is that x-ray evaluation oftentimes did not detect microfracture, despite MRI findings confirming insufficiency fractures and dissolution of continuity of the subchondral osseous structures. From this we can appreciate the importance of investigating patients with MRI, which is superior at identifying early changes associated with fracture, such as bone marrow oedema, which allows complete evaluation of the problems present, should enable the establishment of a complete treatment plan. An incomplete or a delay in diagnosis reduces the potential for a positive and a better prognosis.

Perhaps, subchondral insufficiency syndrome really is not a rare phenomenon. Perhaps this condition was just undiagnosed because of the lack of objective evidence early in the disease process. Establishing an early definitive diagnosis will enable earlier intervention which would then result in better long-term results.

1. R. C. Lawrence, D. T. Felson, C. G. Helmick, L. M. Arnold, H. Choi, R. A. Deyo, S. Gabriel, R. Hirsch, M. C. Hochberg, G.
G. Hunder, J. M. Jordan, J. N. Katz, H. M. Kremers, F. Wolfe; National Arthritis Data Workgroup, Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. Part II. Arthritis Rheum. 58, 26–35 (2008)
2. J. A. Buckwalter, C. Saltzman, T. Brown, The impact of osteoarthritis: Implications for research. Clin. Orthop. Relat. Res. 427, S6–S15 (2004).
3. Walley KC, Gonzalez TA, Callahan R, *et al*. The role of 3D reconstruction True-Volume analysis in osteochondral lesions of the talus: a case series. *Foot Ankle Int* 2018;39:1113–9.
4. Grynpas MD, Alpert B, Katz I, Lieberman I, Pritzker KP: Subchondral bone in osteoarthritis. Calcif Tissue Int 1991, 49:20–26.
5. Goldring M, Goldring S: Articular cartilage and subchondral bone in the pathogenesis of osteoarthritis. Ann NY Acad Sci 2010, 1192:230–237.
6. Bingold AC, Collins DH. Hallux rigidus. J Bone Joint Surg (Br). 1950;32-b(2):214–22.
7. Suri S, Walsh DA: Osteochondral alterations in osteoarthritis. Bone 2012, 51:204–211.
8. Madry H, van Dijk CN, Mueller-Gerbl M: The basic science of the subchondral bone. Knee Surg Sports Traumatol Arthrosc 2010, 18:419–433.
9. Brandt KD, Dieppe P, Radin E: Etiopathogenesis of osteoarthritis. Med Clin North Am 2009, 93:1–24.
10. Shane Anderson A, Loeser RF: Why is osteoarthritis an age-related disease? Best Pract Res Clin Rheumatol 2010, 24:15–26.
11. Goldring M, Goldring S: Articular cartilage and subchondral bone in the pathogenesis of osteoarthritis. Ann NY Acad Sci 2010, 1192:230–237.
12. Milz S, Putz R: Quantitative morphology of the subchondral plate of the tibial plateau. J Anat 1994, 185:103–110.
13. Madry H, van Dijk CN, Mueller-Gerbl M: The basic science of the subchondral bone. Knee Surg

Sports Traumatol Arthrosc 2010, 18:419–433).
14. Castaneda S, Roman-Blas JA, Largo R, Herrero-Beaumont G: Subchondral bone as a key target for osteoarthritis treatment. Biochem Pharmacol 2012, 83:315–323.
15. Suri S, Walsh DA: Osteochondral alterations in osteoarthritis. Bone 2012, 51:204–211.

16. Goldring SR: Alterations in periarticular bone and cross talk between subchondral bone and articular cartilage in osteoarthritis. Ther Adv Musculoskelet Dis 2012, 4:249–258.
17. Lyons TJ, McClure SF, Stoddart RW, McClure J: The normal human chondro-osseous junctional region: evidence for contact of uncalcified cartilage with subchondral bone and marrow spaces. BMC Musculoskelet Disord 2006, 7:52.
18. Subchondral bone in osteoarthritis: insight into risk factors and microstructural changes
Guangyi Li1,2, Jimin Yin1, Junjie Gao2, Tak S Cheng2, Nathan J Pavlos2, Changqing Zhang1* and Ming H Zheng2*
19. Coughlin MJ, Shurnas PS. Hallux rigidus: demographics, etiology, and radiographic assessment. *Foot Ankle Int*. 2003 Oct. 24 (10):731-43.
20. Neogi T, Nevitt M, Niu J, Sharma L, Roemer F, Guermazi A, Lewis CE, Torner J, Javaid K, Felson D: Subchondral bone attrition may be a reflection of compartment-specific mechanical load: the MOST Study. Ann Rheum Dis 2010, 69:841–844.
21. Neogi T, Nevitt M, Niu J, Sharma L, Roemer F, Guermazi A, Lewis CE, Torner J, Javaid K, Felson D: Subchondral bone attrition may be a reflection of compartment-specific mechanical load: the MOST Study. Ann Rheum Dis 2010, 69:841–844.
22. Hu, Y., Chen, X., Wang, S. *et al.* Subchondral bone microenvironment in osteoarthritis and pain. *Bone Res* **9,** 20 (2021). https://doi.org/10.1038/s41413-021-00147-z
23. Insufficiency fractures: A rare cause of foot and ankle pain in three patients with rheumatoid arthritis. Radiol Case Rep. 2018 Jun 29;13(4):855-861. doi: 10.1016/j.radcr.2018.05.016. PMID: 30002787; PMCID: PMC6039983.
24. Fukunishi S, Fukui T, Nishio S, Imamura F, Yoshiya S. Multiple pelvic insufficiency fractures in rheumatoid patients with mutilating changes. *Orthop Rev.* 2009;1((October) 2):e23.

25.

26. Goldring SR: Alterations in periarticular bone and cross talk between subchondral bone and articular cartilage in osteoarthritis. Ther Adv Musculoskelet Dis 2012, 4:249–258.
27. Lyons TJ, McClure SF, Stoddart RW, McClure J: The normal human chondro-osseous junctional region: evidence for contact of uncalcified cartilage with subchondral bone and marrow spaces. BMC Musculoskelet Disord 2006, 7:52.
28. Subchondral bone in osteoarthritis: insight into risk factors and microstructural changes
Guangyi Li1,2, Jimin Yin1, Junjie Gao2, Tak S Cheng2, Nathan J Pavlos2, Changqing Zhang1* and Ming H Zheng2*
29. Coughlin MJ, Shurnas PS. Hallux rigidus: demographics, etiology, and radiographic assessment. *Foot Ankle Int*. 2003 Oct. 24 (10):731-43.
30. Neogi T, Nevitt M, Niu J, Sharma L, Roemer F, Guermazi A, Lewis CE, Torner J, Javaid K, Felson D: Subchondral bone attrition may be a reflection of compartment-specific mechanical load: the MOST Study. Ann Rheum Dis 2010, 69:841–844.
31. Neogi T, Nevitt M, Niu J, Sharma L, Roemer F, Guermazi A, Lewis CE, Torner J, Javaid K, Felson D: Subchondral bone attrition may be a reflection of compartment-specific mechanical load: the MOST Study. Ann Rheum Dis 2010, 69:841–844.
32. Hu, Y., Chen, X., Wang, S. *et al.* Subchondral bone microenvironment in osteoarthritis and pain. *Bone Res* **9,** 20 (2021). https://doi.org/10.1038/s41413-021-00147-z
33. Insufficiency fractures: A rare cause of foot and ankle pain in three patients with rheumatoid arthritis. Radiol Case Rep. 2018 Jun 29;13(4):855-861. doi: 10.1016/j.radcr.2018.05.016. PMID: 30002787; PMCID: PMC6039983.
34. Fukunishi S, Fukui T, Nishio S, Imamura F, Yoshiya S. Multiple pelvic insufficiency fractures in rheumatoid patients with mutilating changes. *Orthop Rev.* 2009;1((October) 2):e23.