Characterization of Initial Microfracture Defects in Human Condyles

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Microfracture (MFX) is a cartilage repair technique that depends on cell migration from marrow-rich trabecular bone cavities into the cartilage lesion. This study tested the hypothesis that MFX awls with distinct geometry generate different hole shapes and variable bone marrow access in condyles with Grade III to IV lesions. Lateral and medial condyles from total knee arthroplasty (N = 24 male and female patients, 66 ± 9 years) were systematically microfractured ex vivo to 2 and 4 mm deep and the bone holes analyzed by micro-computed tomography. Subchondral bone in lesional condyles showed different degrees of sclerosis up to 2 mm deep (“porous,” sclerotic, extremely dense). MFX holes ranged from 1.1 to 2.0 mm in diameter, and retained the awl shape with evidence of slight bone elastic rebound and bone compaction lining the holes that were increased by wider awl diameter and deeper MFX. Marrow access was significantly diminished by sclerosis for all three awls, with an average marrow access varying from 70% (nonlesional bone) to 40% (extremely dense bone). This study revealed that subchondral bone sclerosis can reach a critical limit beyond which MFX creates bone compaction and fissures instead of marrow access.

Microfracture (MFX) is used to resurface focal Outerbridge Grade III to IV articular cartilage lesions1–8 and served as a gold standard in three randomized controlled clinical trials for cartilage repair.2,3,9 Patient-reported outcomes improve for 66 to 88% of all patients for 1 to 2 years postoperatively,4,6,7,10 after which clinical benefit steadily declines with a documented 25% failure rate at 5 years postoperatively.11,12 Although MFX can elicit hyaline-like cartilage repair,2 most defects are resurfaced with a fibrous or fibrocartilage tissue,2,3,13,14 or no tissue at all4,15,16 for reasons that remain unclear. Similar repair tissues are obtained by MFX of acute cartilage defects in large animal models.17–20 Cell-based therapies also elicit a wide variety of repair tissues, with similar 5-year failure rates and declining benefit with time (notably for chronic lesions)11,12; therefore, MFX remains an important therapeutic option. Improvements to the MFX technique are needed, but basic information on the initial MFX defect created in human condyles is lacking. MFX acute defect characterization could lead to insights that improve the standard of care.

In current guidelines for MFX, all degenerated cartilage and the calcified cartilage layer are debrided with a curette.
then pick holes are generated by tapping or hammering the awl with a mallet to a target depth of 3 to 4 mm, and 3 to 4 mm spacing to prevent holes from collapsing together. Successful MFX technique is guided by the observation of bleeding and fat droplets emanating from the holes, which indicates that communication channels have been created between the trabecular bone and the cartilage lesion. However, MFX holes generated in osteoarthritic (OA) lesions sometimes fail to bleed. Awl shape could potentially alter the biological reaction to MFX, and a variety of shapes is available. OA is known to generate subchondral bone sclerosis, which could also change the performance of a given awl. The extent to which MFX holes communicate with the trabecular bone cavities—where blood vessels targeted for rupture, and the marrow stromal cells required to carry out the repair response are located—is currently unknown.

The purpose of this study was to determine whether MFX to clinically recommended depths (2 and 4 mm) reliably attains the trabecular bone marrow cavities. MFX holes were created ex vivo in debrided lesional human condyles using a thin, a beveled, and a wide diameter awl. The awl tips and Jamshidi needle inside (Chondral pick 40-degree large diameter and Universal Endofemoral Aimer Handle, Refs. 72201886 and 72201732; Smith & Nephew, Andover, MA; Sontec Centennial, CO), but the awl used in different studies is most often not reported. OA knees were recruited with a history of steroid therapy and a history of prior steroid therapy, and females were recruited with a history of steroid therapy. MFX in non-OA knees, it is well-acknowledged that isolated OA is known to generate subchondral bone sclerosis, and medial femoral condyle (MFC) surgical waste from total knee arthroplasty and relevant patient parameters (steroid treatment oral, or intra-articular current, or up to 41 years prior to arthroplasty; prior trauma; medications; pain score; smoking including current smoker N = 3, or previously smoked for 20 ± 9 years as a young adult or recently quit N = 12; body mass index [BMI]; and age) were obtained from N = 25 consented patients (15 female and 10 male). Condyles from the first subject were used to standardize the method. Five male and five female subjects were recruited with no history of prior steroid therapy, and five males and nine females were recruited with a history of steroid therapy (N = 24 patients, 66 ± 9 years, BMI: 30.0 ± 4.4). Lesions were graded using the Outerbridge scoring system, where 0 is no lesion and IV is exposed bone. Grade IV lesions varied from approximately 0.5 cm² to the entire condyle.

**Ex Vivo Microfracture**

Awls were purchased with three distinct geometries: a beveled tip (Chondral pick 40-degree large diameter and Universal Endofemoral Aimer Handle, Refs. 72201886 and 72201732; Smith & Nephew), thin and sharp (45 degree awl, Ref. 8203; Linvatec), and wide diameter (20 degree awl, Ref. AR-1761; Arthrex). Awls were premarked by a permanent marker at 2 and 4 mm from the tip (Fig. 1a). Condyles were kept at 4°C in humid gauze, and within 24 to 36 hours postsurgery, warmed to 37°C for 1 hour (to simulate in vivo bone material properties), biopsied with a Jamshidi needle (11 gauge; Cardinal Health/Cardinal Health/CareFusion, San Diego, open circle in Fig. 1b), and debrided inside a 20 × 10 mm² rectangle by curettage with a flat blade (Fine Science Tools, Vancouver, BC, Canada) to remove calcified cartilage (Fig. 1cd). MFX holes were generated systematically in the debrided area with the same pattern: 2- or 4-mm deep in two rows and 2- to 3-mm spacing (see Fig. 1d).

**Micro-Computed Tomography**

Post-MFX, condyles were immediately fixed in 10 volumes of 10% normal buffered formalin for at least 3 days, then microcomputed tomography (micro-CT) scanned at a 15 µm/pixel resolution (Skyscan 1172; Skyscan, Aartselaar, Belgium), at 80 kV, aluminum filter, rotation step = 0.4, frame averaging 3, 180 degrees and reconstructed with beam hardening = 30, smoothing = 2, and ring artifact correction = 4. Agarose impregnated with calcium phosphate and solidified with the awl tips and Jamshidi needle inside (N = 3) were scanned under the same configurations to generate phantoms to measure actual tool diameter by micro-CT (Fig. 1e). Image stacks were reconstructed using NRecon software (v.1.6.2.0, Skyscan), and each MFX hole was repositioned with DataViewer (v.1.4.3, SkyScan 2009) in a coronal view (for hole depth and area from the awl tip, Fig. 1f), and transverse view to measure top hole cross-sectional area (Fig. 1g) and exported to CTan for thresholding. The top of the hole was defined as the first slice where the hole was completely surrounded by bone, and the base of the hole defined as the final slice with visible bone or marrow damage. Hole cross-sectional area was obtained by tracing the bone hole perimeter with a polygon tool (excluding recessed adjacent marrow cavities), then a reverse threshold (70 to 0, minimum to maximum) was used to capture the soft tissue area inside the hole. Soft tissue area was converted to diameter \(D = 2 \times \sqrt{\frac{\text{cross sectional area}}{3.14}}\) Subchondral volume 800.9

**Methods**

**Subject Recruitment**

This study was designed in conformity with the World Medical Association Declaration of Helsinki and approved by Institutional Review Boards. Lateral femoral condyle (LFC) and medial femoral condyle (MFC) surgical waste from total knee arthroplasty and relevant patient parameters (steroid treatment oral, or intra-articular current, or up to 41 years prior to arthroplasty; prior trauma; medications; pain score; smoking including current smoker N = 3, or previously smoked for 20 ± 9 years as a young adult or recently quit N = 12; body mass index [BMI]; and age) were obtained from N = 25 consented patients (15 female and 10 male). Condyles from the first subject were used to standardize the method. Five male and five female subjects were recruited with no history of prior steroid therapy, and five males and nine females were recruited with a history of steroid therapy (N = 24 patients, 66 ± 9 years, BMI: 30.0 ± 4.4). Lesions were graded using the Outerbridge scoring system, where 0 is no lesion and IV is exposed bone. Grade IV lesions varied from approximately 0.5 cm² to the entire condyle.

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bone properties were evaluated at three depths in a 2 x 2 x 1 mm³ volume of interest (VOI), adapted only at the surface to the bone

Fig. 1 Methodology for generating and analyzing microfracture holes in femoral condyles, with three awls of distinct geometry. (a) Smith & Nephew, Andover, MA (S, beveled tip); Linvatec, Streetsville, ON, Canada (L, sharp and thin); Arthrex, London, ON, Canada (A, wide); and a Jamshidi 11-gauge biopsy needle (b). (c) Femoral condyles were systematically microfractured to 2 and 4 mm deep in a rectangular debrided area according to the pattern shown in (d). Agarose phantoms (e) were used to obtain the actual tool shape through micro-CT scan and three-dimensional analysis. Hole depth and diameter at specific levels from the hole bottom were obtained using the method in (f). Top hole diameter was measured parallel to the debrided surface instead of perpendicular to the hole axis (g). Bone properties were measured in a 2 x 2 x 1 Agarose 1 mm³ volume of interest (VOI) at the three zones of the top, 2 and 4 mm deep (h). The % marrow access was measured by thresholding the bone hole perimeter (i, j) to obtain total surface of the hole perimeter (TS) and surface intersection (iS) (k) from which % marrow access was calculated.

Histology
Osteochondral biopsies were fixed in formalin, embedded in methyl methacrylate, and 5-µm-thick serial sections stained with Goldner trichrome, Safranin O-fast green, or von Kossa-Toluidine blue. Calcified cartilage thickness was measured in Safranin O-stained sections in 11 vertical line measures along each biopsy width and the average used for statistics.

Statistical Analyses
The General Linear Model (GLM, Statistica v6.2; Statsoft, Tulsa, OK) was used to test the hypothesis that MFX awl, steroid therapy, smoking, lesion grade, gender, and depth are predictors of the dependent variables (BMD, BVF, trabecular thickness, bone hole depth, and bone hole diameter, calcified cartilage thickness, % MA). Univariate differences were analyzed using the Tukey honestly significant difference (HSD) post-hoc test. Sample number per analysis is given in each figure. A p value less than 0.05 was considered significant.

Results
Lesional Bone Plates Show Abnormal Structure and Variable Sclerosis up to 2-mm Deep
Lateral and medial condyles had a mean Outerbridge lesion grade of 1.5 versus 3.5, respectively (p < 0.0001, N = 24).
Subchondral bone density was highest at the debrided bone surface, and higher lesion severity was associated with an increase in bone volume fraction, bone mineral density, and average trabecular thickness up to 2 mm, but not 4 mm deep ($p < 0.005$, Fig. 2a–c). Condyles with more severe cartilage lesions were found to have three grades of bone plate density (0 to 1 mm deep): porous, sclerotic, and extremely dense, irrespective of gender (Fig. 2d).

Fig. 2 Subchondral bone properties of debrided lateral femoral condyle (LFC) and medial femoral condyle (MFC) with different grade lesions. The bone volume fraction (bone volume/total volume, %) (a), bone mineral density (BMD) (b), and average trabecular thickness (c) were increased with depth and in Grade III and IV lesions, which presented with three classes of bone density in the top 1 mm of the bone plate (d). Bone properties were influenced by smoking (e) and not by prior steroid therapy (f). Data are shown as mean and uncertainty as ± standard error (box) and 95% confidence intervals (whiskers).

Fig. 3 Representative histology of osteochondral biopsies collected from condyles with different classes of subchondral bone: LFC (no cartilage lesion, approximately normal porosity bone), and MFC porous bone, extremely dense sclerotic bone, and eburnated bone (as indicated). Nondecalcified plastic sections were stained with SafraninO/fast green-stain (A1) or Goldner trichrome (B1–F1) and adjacent sections were stained with von Kossa-Toluidine blue (A2–F2). Pink-stained bone edges in B1, C1, E1, F1 show nonmineralized bone osteoid while the subchondral pink-stained tissue in D1 is subchondral callus. Note the GAG loss typical of OA cartilage (B2, C2).
Previous episodic steroid administration had no influence on bone density; however, chronic smokers and previous chronic smokers had significantly less sclerosis compared with subjects who never smoked (p = 0.038, ►Fig. 2e, f). The calcified cartilage layer was 0.22 ± 0.11 µm thick in Grade I to III lesions, and variably worn away in Grade IV lesions (0.11 ± 0.10 µm thick, p = 0.0032). Sclerosis was thus due to bone accumulation and not due to the thickening of the calcified cartilage layer. The bone plate structure in lesional condyles was clearly abnormal (►Fig. 3).

MFX Hole Dimensions Are Influenced by Awl Shape, Perforation Depth, and Sclerosis

MFX holes retained the shape of each tool, including a triangular-shaped tip of the Smith & Nephew awl (►Figs. 1a and 4f). Grade IV lesions with porous bone were easily perforated and had open communication between the MFX hole and adjacent cell-laden trabecular cavities (►Fig. 4a–c). Shiny eburnated bone was difficult to perforate, especially with the larger diameter Arthrex awl, and in these condyles, 2-mm MFX holes were fully surrounded by bone (►Fig. 4d–f). Bone compaction

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**Fig. 4** Microfracture (MFX)-induced marrow access and bone compaction as revealed by 3D-microCT. Panels show the macroscopic appearance (a, d), coronal micro-CT “Top view,” in the middle of a 4-mm-deep MFX hole (b, e), and 3D cylindrical micro-CT image of the MFX hole, where the threshold encompasses the hole and adjacent marrow cavities (c, f). (Panel F shows the MFX hole created by the Smith & Nephew awl). Bone compaction flanked the MFX holes in a coronal view about 1 mm below the bone surface (g), and along the sidewalls of the holes in a transverse view of two example MFX holes (h, i). The white arrow in (g) and blue arrows in (h, i) show the strain placed on the bone during MFX. A, Arthrex; L, Linvatec; S & N, Smith & Nephew; 4 = 4-mm-deep hole; 2 = 2-mm-deep hole.
around MFX holes could be seen by micro-CT up to 2 mm deep in sclerotic bone (Fig. 4g–i). The Jamshidi needle and Arthrex awl induced fissures or cracks in several condyles with eburnated bone (unpublished data, Jun Sun, MD and Hongmei Chen, PhD), revealing that extremely dense bone can fail under strain induced by perforation with a tool more than 2 mm in diameter.

Holes generated in MFCs with an approximately 2-mm target depth were slightly deeper according to micro-CT measures (Fig. 5a). Holes generated to approximately 4 mm attained the target depth in condyles with porous and sclerotic bone plates but failed to reach 4 mm when the bone plate density was higher than 80% BVF (p = 0.0015 effect of bone density, Fig. 5b). These data showed that hole depth was a controlled variable among the three awls in this study and provide quantitative evidence that subchondral bone resists compaction by MFX. The hole diameter at debrided bone surface was significantly increased by deeper MFX with a wider awl (p < 0.0001, Fig. 5c). Deep MFX with the Arthrex awl generated a 2.0-mm diameter hole while shallow MFX with the Linvatec awl made a 1.1-mm diameter hole. Inside the hole, 1.5-mm from the tip, the hole diameter was consistently smaller than the agarose phantom tool shape for all three awls (Fig. 5d). These data provide novel quantitative evidence that trabecular bone resists compaction by MFX and undergoes elastic rebound (Fig. 5d). The difference in theoretical and measured hole diameter is consistent with the notion that “hidden” fracture damage is created systematically around the periphery of each MFX hole. By contrast, the Jamshidi bone biopsy needle, a tool which removes bone, showed no compaction with a very similar diameter in bone and the agarose tool phantom (dotted gray arrow, Fig. 5d).

Fig. 5 Micro-CT measures of subchondral bone hole dimensions and marrow access generated by three different microfracture (MFX) awls in OA medial femoral condyles (a–d) and lateral vs. medial femoral condyles (e–g). MFX hole target depth was attained in 2-mm MFX (a) but attenuated for a 4-mm-deep MFX (b) in extremely dense bone plate (> 80% bone volume fraction). Hole diameter was significantly influenced by awl shape (p < 0.0001, c and d) and depth of perforation (p < 0.0001, c). % Marrow access increased with depth from the bone surface in the perforations (p < 0.0001, e, f), was higher in LFC versus MFC (p < 0.0001, e vs. f), and decreased by sclerosis (p < 0.0001, g) with no effect of awl (p = 0.36) except in extremely dense bone. For Panel D, agarose phantom tool diameter measured at 1.5 mm from the bottom of the jamshidi hole (f): 2.4 ± 0.02 mm, and at 1.5 and 2.5 mm from the MFX hole tip, respectively (N = 3, mean ± S.D, mm): Arthrex 1.64 ± 0.02 and 2.01 ± 0.03, Linvatec 1.01 ± 0.07 and 1.41 ± 0.08, and S & N 1.34 ± 0.08 and 1.68 ± 0.01. All data are shown as the mean and uncertainty as ± 95% confidence intervals.
Marrow Access is Influenced by Hole Depth, Lesion Grade, and Awl Shape

In LFCs with “normal” bone density, all three awls produced 55 to 73% MA with increasing depth from the debrided surface ($p < 0.0001$, $N = 5$, -Fig. 5e), where 0% MA is an MFX hole completely surrounded by bone and 100% MA is a hole edge completely surrounded by soft cellular marrow tissue. In MFCs, all three awls generated only 30 to 59% MA ($p < 0.0001$ vs. LFC, -Fig. 5f). Advancing bone sclerosis led to a similar decline in % MA for all three awls, except the Linvatec awl which produced higher % MA than the Smith & Nephew beveled awl ($p = 0.016$, $N = 9$, -Fig. 5g).

To summarize, 4-mm-deep MFX holes in porous bone (40% BVF at the bone plate, 20% BVF at 2 mm deep) routinely communicate with trabecular marrow cavities (-Fig. 4h, i). Loss of trabecular communication is wholly detrimental because a strong cell influx is required to produce new extracellular matrix, and the resulting volume of repair tissue predicts clinical benefit. As one of the goals of MFX is to generate physical channels that connect the cellular bone marrow to the debrided cartilage lesion, our data demonstrate that failure of MFX to penetrate the sclerotic subchondral bone plate should be regarded as a technical error (-Fig. 6d). MFX-induced compaction of sclerotic bone could potentially explain why some MFX holes fail to bleed into the joint.

Erosion of the articular cartilage layer removes elastic damping during load-induced compression and has a reciprocal effect on the subchondral bone balance that has yet to be fully understood. Some researchers believe that bone stiffening could precede and lead to cartilage loss, while others have found no evidence of this in the tibial plateau during a 2-year study. Our data showing different degrees of subchondral sclerosis in lesional condyles are consistent with a recent report that female OA condyles can be sclerotic, or else porotic with bone marrow cell inflammatory gene expression (Opest and Freeman, unpublished data, 2012). Attenuation of sclerosis in condyles of smokers, or prior chronic smokers, is consistent with dysvascular bone loss, and previous studies showing smokers have higher hip fracture risk.

Altogether, these data suggest that subchondral bone mass is perhaps more susceptible to changes due to lifestyle or disease/inflammation than cartilage lesion status. Bone

Discussion

This study is the first to report that MFX hole dimensions and consequently bone compaction are significantly influenced by awl shape and subchondral bone structure in human condyles. When bone is microfractured, lateral and hoop strains are produced that drive bone beyond its elastic limit in a radial direction. As bone mass and brittleness increases, the MFX-induced bone compaction is so great that it fills the trabecular spaces around the hole (-Fig. 4h, i). Loss of trabecular communication is wholly detrimental because a strong cell influx is required to produce new extracellular matrix, and the resulting volume of repair tissue predicts clinical benefit. As one of the goals of MFX is to generate physical channels that connect the cellular bone marrow to the debrided cartilage lesion, our data demonstrate that failure of MFX to penetrate the sclerotic subchondral bone plate should be regarded as a technical error (-Fig. 6d). MFX-induced compaction of sclerotic bone could potentially explain why some MFX holes fail to bleed into the joint.

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Fig. 6 Successful marrow stimulation requires perforation to the cellular trabecular bone marrow. Micro-CT images (a, c) and corresponding tracings (b, d) are shown for a 4-mm microfracture hole in porous bone with communication between the microfracture hole and bone marrow (light gray, a, b), and a 2-mm-deep hole in sclerotic bone with virtually no “marrow stimulation” (c, d).
marrow cells are also altered by smoking, and aging, which could also limit the repair response to MFX.5,22,26,44

Our data suggest that uniform debridement of approximately 200 µm of mineralized tissue should be sufficient to remove the calcified cartilage layer in most lesions, including Grade IV lesions. Curetage offers a tactile appreciation of the “grating” away of mineralized tissue but is time-consuming, nonuniform, and can lead to iatrogenic damage of surrounding cartilage. A motorized shaver can unintentionally debride the bone plate.45 The MFX technique would be improved by new methods that reveal preoperative subchondral bone properties, whether the debridement technique is removing mineralized tissue, and better debridement tools.

One limitation in our study was that bone properties were sampled from relatively small tissue volumes (2 × 2 × 1 mm3), which may not represent all subchondral features. In addition, holes from one awl were always generated in the center of the condyle (Linvatec), although similar %MA measures were obtained for the three awls, suggesting only a slight bias. Bone remodeling could also potentially overcome differences due to awl shape, although several studies show that bone sometimes fails to remodel or resurface between the MFX holes.2,16,19 Implants that induce bone remodeling of MFX holes46,47 could help promote marrow-derived cell influx in chronic lesions with evidence of early bone plate sclerosis.

In conclusion, this study revealed that bone sclerosis has a significant influence on the degree of “marrow access” in the acute defect, and hence the potential biological response of a debrided defect to MFX (i.e., cell migration from the bone marrow into the cartilage lesion area). As the awl is driven deeper into the subchondral bone, marrow access increases near the base of the hole and decreases at the bone plate, due to a ring of bone compaction that forms around the MFX hole. Compaction damage forms in a 0.3- to 1.0-mm ring beyond the macroscopically visible hole, with a larger radius of fracture damage for thicker awls and deeper holes. Thin tapered awl geometries such as the Linvatec tool allowed easier perforation of dense bone and produced less bone compaction, which may be more suitable for MFX therapy in OA condyles. More holes can be created with a thin versus thick awl, but the impact of awl geometry on the clinical response remains to be confirmed with in vivo studies. MFX holes generated in bone with a 50% bone volume fraction at 2 mm below the surface may not connect with the marrow, and could represent the maximum tolerable bone volume fraction for a patient to be allowed as a candidate for MFX.

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