

Do Microfractures Work?

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ABSTRACT

It has been observed that injuries, which involve only a part of the joint cartilage thickness, show a partial healing ability, but some healing potential is present in the case of injuries that affect the subchondral osseous tissue. The methods of treatment for chondral defects are non-surgical: pharmacological chondroprotection (nutraceuticals containing chondroitin sulphate and glucosamine sulphate), viscosupplementation, PRP and cytokine modulation. The surgical options include palliative approaches, marrow stimulation, and cell-based regeneration techniques. The microfracturing technique has started to be used in the '80s using equine models. The basic principle of microfracturing is to stimulate cartilage repair, not regeneration. It represents the first line of therapy for cartilage defects that affect its full thickness. The advantages of microfracturing are: it is a minimally invasive procedure, technical simplicity, decreased surgical morbidity, and low costs, thus making it a common procedure. However, microfracturing is not a bulletproof technique. There is a high rate of treatment failure after 5 years. Constant decline of the outcome during the 5-year follow-up after surgery is also described. Lesion size represents a better predictor regarding outcome, compared to age. The best results are obtained in young patients, small lesions and low-demand patients. Microfractures can be augmented using BMAC (bone marrow aspirate concentrate) containing 0–0.1% progenitor cells. The matrix aids in the improvement of microfractures, and it also increases the stability of the blood clot, acting as a barrier that avoids the fibroblast invasion of the graft. There is still a place for microfractures in chondral defects. Better results can be achieved with an improved technique, an optimized rehabilitation, and adding several augmentation techniques.

Keywords: chondral defect, microfracture, knee cartilage

INTRODUCTION

In 1743 William Hunter observed that injuries that involve only a part of the joint cartilage thickness show a partial healing ability, but some healing potential is present for injuries that affect the subchondral osseous tissue.

A total of 25,124 knee arthroscopies have shown injured cartilage in 15,074 knees (60%), and around 67% have been categorized as focal damage. Approximately 5–10% of them were focal and frequently asymptomatic injuries.¹

The methods of treatment for chondral defects are nonsurgical: pharmacological chondroprotection (nutraceuticals containing chondroitin sulphate and glucosamine sulphate), viscosupplementation, PRP and cytokine modulation.

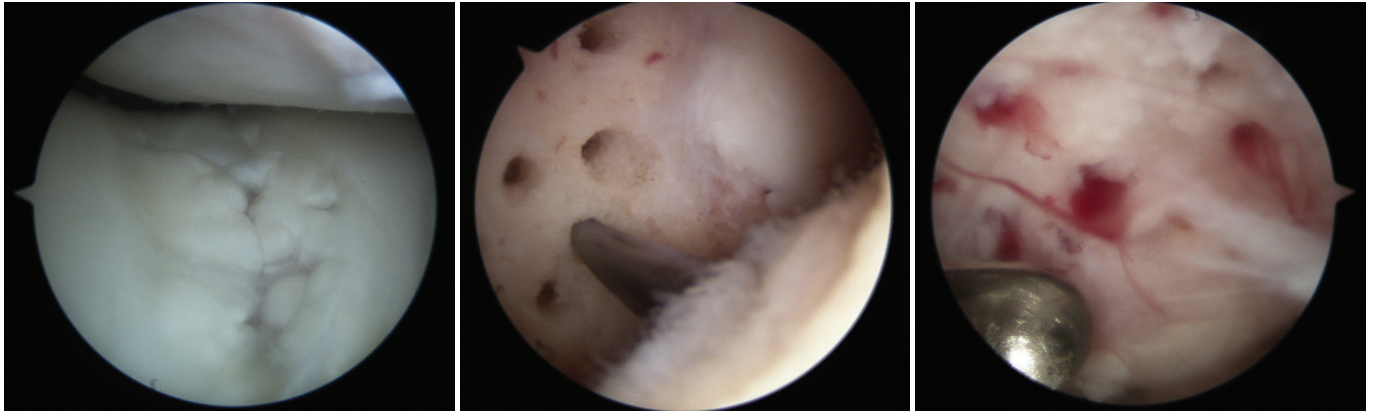


FIGURE 1. The microfracture technique for the treatment of articular cartilage lesions in the knee (from the photo archive of the 1st Department of Orthopedics and Traumatology, “Pius Brînzeu” Clinical Hospital, Timișoara, Romania). **A** – Chondral defect. **B** – Microfracture holes. **C** – Bleeding via drilling holes.

Surgical options include palliative approaches, marrow stimulation, and cell-based regeneration techniques. The Pridie drilling for osteoarthritis was introduced in 1959, and it was used as a marrow stimulation technique by subchondral drilling.²

MICROFRACTURES — OLD AND UP-TO-DATE TECHNIQUE

The microfracturing technique has started to be used in the '80s using equine models.³ Its principle is to stimulate repair, not regeneration. It represents the first line of therapy for cartilage defects that affect its full thickness. The advantages of microfractures are: it is a minimally invasive procedure, technical simplicity, decreased surgical morbidity, and low costs, thus making it a common procedure.

A correct approach to microfracturing is taking into account the patient's history, the clinical examination, the result of the MRI assessment, the available equipment (the area, size and location of the defect require an array of tools), costs, and the patient's option. When examining the subchondral edema, one can observe areas of marrow edema with abnormal perfusion, and a decreased number of progenitor cells.⁴

The microfracture method is indicated for acute, full-thickness and well-defined chondral injuries. An inevitable bad result often occurs in case of microfracture procedures when it comes to approaching large defects, pre-existing malalignment, primary or secondary instability or osteochondritis dissecans.

Microfractures have no indication in case of existing malalignment, partial thickness injuries, kissing lesions, and age over 50 years. In order to achieve good results, several aspects should be carefully considered: the lesions should

be smaller than 2 cm, the injury should be new, only contained lesions should be included, the patient should be younger than 35 years and the BMI lower than 30.

From a technical standpoint, the drilled holes should be at 3–4 mm, with a depth of 2–4 mm. New recommendations state that the surgeon should drill as many holes as possible, without creating macrofractures, and the drilling should be deep enough to create bleeding. The rough surface thus created, holds the marrow clot.⁵

HISTOLOGICAL ASPECTS

The cells in the clot, which are non-differentiated mesenchymal cells, will mature to create a fibrocartilage repair tissue with type II collagen that has lower rigidity and lower wear compared to that of the typical hyaline chondroma.⁵

Drilling and microfracturing stimulate the proliferation of a different osseous structure and necrosis compared to the one that occurs during bone-marrow stimulation for cartilage repair.⁶

The adverse effect of the microfracturing procedure is fractured and compacted bone around the drilled holes. This phenomenon creates a barrier for viable bone marrow that can hinder the repair process. Therefore, a different approach should be considered. On the other hand, drilling removes bone, thus creating a clean route for the bone marrow.⁶

In the first 2 years following the procedure, there is a 75–100% improvement rate, decreasing after 2 years to 67–86%. Functional deterioration is 47–80%. The improvement rate after 10 years is over baseline.^{7,8}

MRI performed after the microfracturing procedure shows a complete fill in 18–95% of cases, and a poor fill

is described in 17–57% of cases. A complete integration is observed in 4–8% of cases. The function correlates with the fill.^{8,9,10}

Microfracturing is not a bulletproof technique. There is a high rate of treatment failure after 5 years. Constant decline of the outcome during the 5-year follow-up after surgery is also described. Lesion size represents a better predictor regarding outcome, compared to age. The best results are obtained in young patients, small lesions and low-demand patients.^{11,12}

MICROFRACTURING FAILURE

Revision or failure at <2 years is around 2.5%, and between 2–10 years it is 2–38%.

An increased failure rate with lower repair tissue quality and a longer duration of symptoms is approximately 48% Kellgren I at 10 years.^{7,8,13}

The chance of failure increases as the incidence of the following factors is higher: age over 40 years, duration of symptoms over 12 months, lesion's size larger than 4 cm, BMI above 30, preoperative activity level as classified using the Tegner Score lower than 4, a defect fill greater than two thirds of the original injury.⁸

Subchondral hypertrophy is found in 46% of patients one year after surgery and up to 62% around 22 months after surgery. A higher BMI, an injury located in the external compartment of the knee, and the choice of surgical technique is associated with a higher prevalence of subchondral hypertrophy.

AUGMENTATION CONCEPT

The augmentation process is performed using BMAC (bone marrow aspirate concentrate) containing 0–0.1% progenitor cells. The matrix aids in the improvement of microfractures, and it also increases the stability of the blood clot, acting as a barrier that avoids the fibroblast invasion of the graft. This method extends the applicability of microfractures from small to medium-sized cartilage defects.

CONCLUSIONS

Recent results show that there is still a place for microfractures in injuries that affect the full thickness of the cartilage. Practitioners must take into account the reasons of failure, and try to minimize them as much as possible. Improving the outcomes can be achieved with a better technique, an optimized rehabilitation, and augmentation techniques. There is still room for improvement in this field of injuries, and prospective randomized trials can yield objective results.

CONFLICT OF INTEREST

The authors have nothing to declare.

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