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# Combination of microfracture and periostal-flap for the treatment of focal full thickness articular cartilage lesions of the shoulder: a prospective study

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**Abstract** Focal full-thickness articular cartilage lesions of the shoulder are less common than those of the lower extremity but are often symptomatic and may progress to degenerative osteoarthritis. This prospective study evaluated our clinical results for cartilage repair in five patients with chondral defects localized at the humeral head using a combination of microfracture and periostal flap, all by deltoidopectoral approach. Mean follow-up was 25.8 months (range 24–31) and consisted of a clinical examination, Constant score examination, radiography, and magnetic resonance imaging; three patients underwent a second-look arthroscopy an average of 8 months following cartilage repair. We found the Constant score significantly improved over the preoperative level, from 43.4% to 81.8%. Pain was reduced significantly to 18.6 points. Radiography and magnetic resonance imaging showed progression of the osteoarthritis in two patients. Second-look arthroscopy revealed a significantly reduced cartilage lesion. This is the first report of a combination of microfracture and a periostal flap for repair of focal full-thickness cartilage lesions at the shoulder. Short-term follow-up clinical results were satisfactory. It is essential to address the underlying pathology. Results must be reconfirmed in a long-term study.

**Keywords** Shoulder · Chondral lesion · Microfracture · Periostal flap

## Introduction

Focal full-thickness cartilage lesions of the shoulder are less common than those of the lower extremity. Nevertheless these defects are often symptomatic and lack the tendency to heal. In the long-term these lesions may progress to degenerative osteoarthritis. Due to the specific structure and function of hyaline cartilage [7, 8, 28, 29] surgical intervention at the shoulder joint was limited in the past in most cases to an arthroscopic lavage, débridement, and abrasion of the chondral lesion [19, 20, 32]. Today's aim is to restore hyaline cartilage in the long-term through a practical and minimally invasive approach, preferably arthroscopic, and associated with minimal morbidity not only perioperatively but over an extended period of time [27, 34].

Due to improved surgical techniques promising clinical results have been achieved in knee surgery in recent years [10, 22, 23, 31, 34, 37]. One popular technique is the subchondral bone plate microfracture technique first introduced by Steadmann et al. [36] in 1997. Released marrow elements form a surgically induced reparative granulation clot that differentiates and remodels under protected loading and continuous passive motion into a fibrocartilage or hyalinelike repair tissue [34, 36, 37].

Another technique, the periostal transplantation for the treatment of deep cartilage defects of the patella was introduced by Lorentzon et al. [21] in 1998. Biopsy samples show a hyalinelike regeneration of articular cartilage with satisfactory clinical results. Periostal flaps are also used to implant in vitro cultured autologous chondrocytes at the site of a cartilage defect [6, 13, 24, 31].

In our study we used the periostal flap to cover the microfractured area and to protect cartilage formation. The aim of this prospective investigation was to evaluate the clinical outcome of the described operative cartilage repair technique at the glenohumeral joint with a follow-up of at least 2 years.

## Material and method

In this prospective study all five patients presented with a focal full-thickness articular cartilage defect grade IV according to Outerbridge [30] located at the humeral head with an average size of 311 mm$^2$ (range 225–400). The defects were all circumscribed with visually intact surrounding articular cartilage. The glenoid was not affected in any patient. All patients were treated with a combination of microfracture and a periostal flap for cartilage resurfacing. The operations were performed between July 1999 and August 2000. The patients' mean age was 32 years (16–56). Their demographic characteristics are summarized in Table 1.

### Study design

The preoperative evaluation consisted of a history, clinical examination, Constant score (CS), standardized radiography, and magnetic resonance imaging (MRI). Diagnostic arthroscopy was carried out to access and classify the defect; cartilage repair was then performed by an arthrotomy via deltopectoral approach and the underlying pathology was treated (see below). Second-look arthroscopy was carried out in three patients at a mean of 8 months (range 7–10) after cartilage repair (Table 2). All five patients were available for clinical follow-up, performed at an average of 25.8 months (24–31) after cartilage repair. This consisted of a history, clinical examination, CS (Table 3), standardized radiography, and MRI.

**Table 1** Patient characteristics

| Patient no. | Sex | Age (years) | Dominant side/OP | Defect size (mm$^2$) | Outerbridge grade |
|---|---|---|---|---|---|
| 1 | F | 56 | R/L | 15×15=225 | 4 |
| 2 | M | 31 | R/R | 20×20=400 | 4 |
| 3 | F | 33 | R/R | 15×25=375 | 4 |
| 4 | M | 24 | R/R | 15×22=330 | 4 |
| 5 | M | 16 | R/R | 15×15=225 | 4 |

**Table 2** Second-look arthroscopy

| Patient no. | Second-look arthroscopy | Outerbridge grade |
|---|---|---|
| 1 | Yes, 7 months | 3 |
| 2 | Yes, 10 months | 1 |
| 3 | Yes, 7 months | 2 |
| 4 | No | – |
| 5 | No | – |

### Patients

Patient 1 was a 56-year-old female physical education teacher who presented at our outpatient clinic in March 2000 with a painful left nondominant shoulder. She had no past medical history of trauma. A sudden pain appeared while assisting a pupil in a sports exercise, but there was no defined trauma. The reason for the central humeral cartilage defect is unknown.

Patient 2 was a 31-year-old male student and high-performance swimmer who presented at our outpatient clinic in January 1999 describing recurrent dorsal subluxations of his right dominant shoulder. He had no past medical history of acute trauma. Examination revealed a dorsal instability and a hyperlaxity type III according to Gerber [11], and arthroscopy showed a central humeral cartilage defect caused by the recurrent posterior dislocations.

Patient 3 was a 33-year-old woman who presented at our outpatient clinic in July 1999 with a first traumatic anterior inferior luxation and an open Bankart repair of her left non-dominant shoulder having been performed in January 1999. She complained of persistent pain and restricted range of motion. Examination revealed a glenohumeral crepitation with an elevation of 140°, internal rotation of 60°, and external rotation of 30°. Arthroscopy showed a focal full-thickness cartilage lesion at the center of the humeral head caused by the eyelet of a titanium anchor.

Patient 4 was a 24-year old male handball-player who presented at our outpatient clinic in September 2000 with arthroscopic Bankart repair by titanium anchor technique of his dominant right shoulder having been performed in February 2000 in domo. Revision surgery was necessary because of persistent pain, which showed a focal humeral full-thickness cartilage defect caused by the eyelet of a titanium anchor.

Patient 5 was a 16-year-old male professional soccer player who presented at our outpatient clinic in July 2000 after primary anterior inferior luxation of his dominant right shoulder in 1993 and two arthroscopic stabilizations in 1993 and 1997. Physical examination revealed a persistent anterior inferior instability and a hyperlaxity and arthroscopy revealed a Bankart/superior labrum anterior and posterior II lesion and a focal humeral full-thickness cartilage defect caused by recurrent instability.

An overview of all patients is presented in Table 4.

**Table 3** Constant score preoperatively and at follow-up (*ADL* activities of daily life, *ROM* range of motion)

| Patient no. | Preoperative CS | | | | Length of follow-up (months) | Follow-up CS | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Pain | ADL | ROM | Total (%) | | Pain | ADL | ROM | Strength (kg) | Total (%) |
| 1 | 5 | 11 | 30 | 35 | 24 | 10 | 16 | 30 | 1.5 | 59 |
| 2 | 0 | 18 | 40 | 58 | 31 | 13 | 19 | 38 | 8.7 | 88 |
| 3 | 5 | 6 | 26 | 28 | 26 | 15 | 20 | 38 | 3.9 | 81 |
| 4 | 5 | 18 | 40 | 47 | 24 | 15 | 18 | 36 | 8.6 | 87 |
| 5 | 10 | 15 | 40 | 49 | 24 | 15 | 20 | 38 | 10.0 | 94 |
| Mean | 5 | 13.6 | 35.2 | 43.4 | 25.8 | 13.6 | 18.6 | 36 | 6.5 | 81.8 |

**Table 4** Previous surgery, cause of chondral lesion with underlying pathology and surgery at index operation

| Patient no. | Previous surgery | Cause of chondral lesion, underlying pathology | Surgery at index operation |
|---|---|---|---|
| 1 | – | Idiopathic | Cartilage repair |
| 2 | – | Hyperlaxity with posterior instability type III (Gerber) | Posterior capsule shift (Neer), cartilage repair |
| 3 | 1× open Bankart repair | Chondral damage (caused by titanium anchor) | Anchor removal, cartilage repair |
| 4 | 1× arthroscopic Bankart repair | Chondral damage (caused by titanium anchor) | Anchor removal, cartilage repair |
| 5 | 2× arthroscopic Bankart repair | Recurrent anterior inferior instability | Capsular shift, labral augmentation (Harryman), cartilage repair |

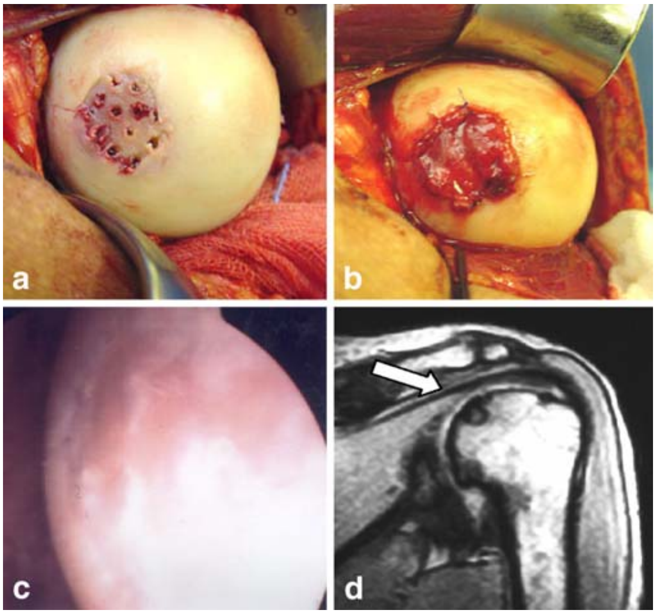


**Fig. 1** Patient 1. **a** Microfractured humeral chondral lesion. **b** Periostal flap sutured on top of the microfractured area. **c** Second-look arthroscopy with grade 3° chondral defect. **d** Follow-up MRI with central osteochondral lesion

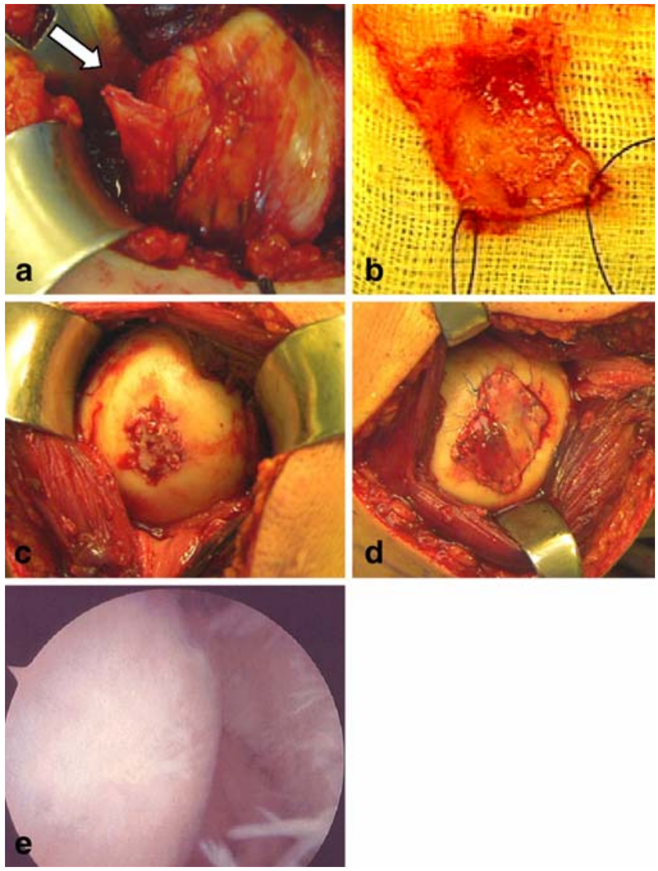


**Fig. 2** Patient 2. **a** Harvesting of the periostal flap. **b** Periostal flap. **c** Microfractured humeral chondral lesion. **d** Periostal flap sutured on top of the microfractured area. **e** Second look arthroscopy with grade 1 chondral defect

Operative technique

All procedures were performed by the senior author with the patient in a beach chair position under interscalene block and general anesthesia. For cartilage repair the shoulder joint was exposed via standard deltopectoral approach. The cephalic vein was preserved. The subscapularis muscle was detached with the joint capsule medial of the lesser tuberosity in a U-shaped fashion, armed with holding sutures for the later refixation, and retracted medially. The humeral head with the lesion was exposed by external rotation and abduction.

In a first step the microfracture was performed according to Steadman et al. [36, 37] with curved awls and a distance of 3–4 mm between the perforations to provoke bleeding from the subchondral bone plate. As many perforations as possible were performed. The integrity of the subchondral bone plate was maintained (Figs. 1a, 2c, 3b).

The surgical technique for implanting the periostal flap was performed according to Lorentzon et al. [21] and Minas et al. [24]. The defect was débrided with all damaged cartilage and fibrocartilage removed, leaving exposed the subchondral bone with a circumferenced stable cartilage rim. The integrity of the subchondral bone plate was maintained. A 1- to 2-mm oversized periostal flap was then harvested from the proximal humeral metaphysis inferior to the greater tuberosity (Fig. 2a, b). The periostal flap was transferred to the chondral defect at the humeral head with the cambium layer facing the cartilage lesion. This was sutured to the cartilage rim with a 6-0 nonresorbable suture spaced every 2–3 mm (Figs. 1b, 2d, 3c). The graft-defect interface was sealed with fibrin glue to ensure a watertight seal.

Next we addressed the underlying or associated pathology of the cartilage defect. Patient 2 was treated with a posterior capsula

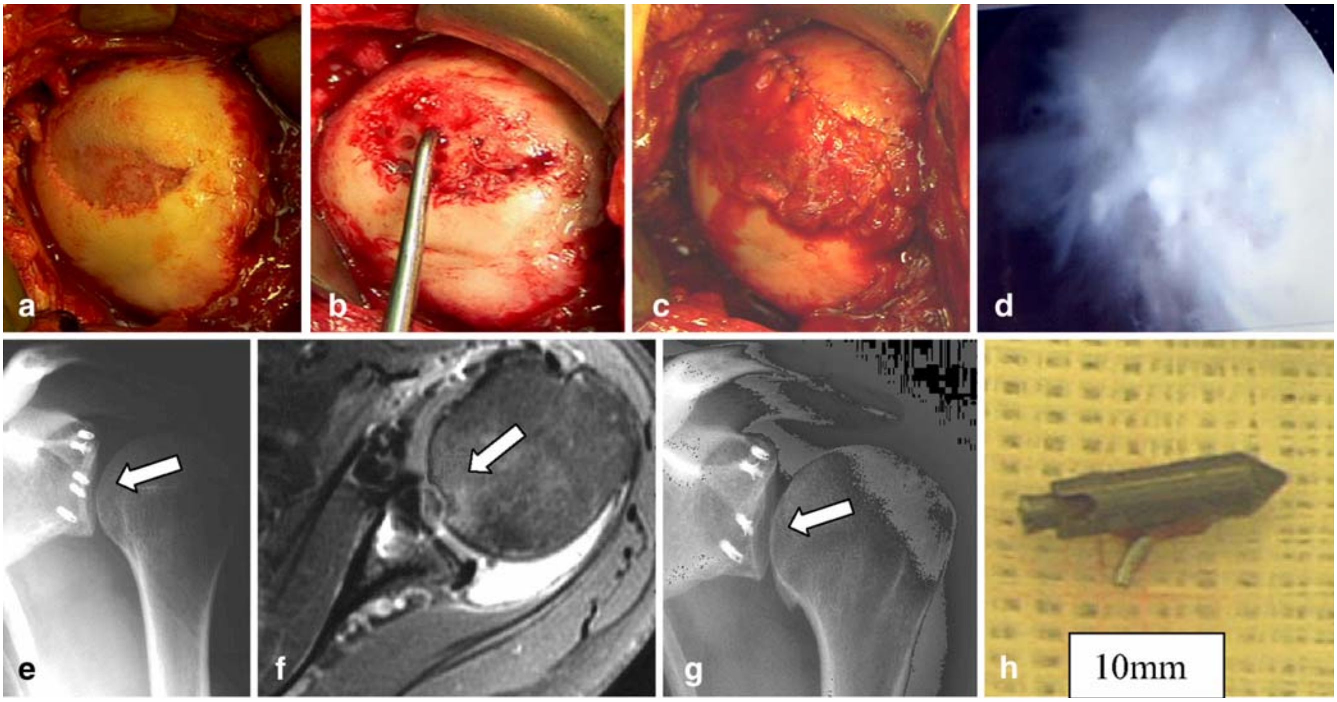


**Fig. 3** Patient 3. **a** Chondral lesion (375 mm$^2$). **b** Microfractured humeral chondral lesion. **c** Periostal flap sutured on top of the microfractured area. **d** Second look arthroscopy with grade 2 chondral defect. **e** Preoperative radiography; titanium anchor with corresponding osteochondral humeral damage. **f** Preoperative MRI; osteochondral humeral damage. **g** Follow-up radiography; central osteochondral lesion. **h** Removed titanium anchor

shift according to Neer [26]. In patients 3 and 4 we removed the inserted titanium anchor which caused the cartilage damage (Fig. 3h). Patient 5 was stabilized with a capsular shift and labral augmentation according to Harryman et al. [17] (for overview see Table 4).

For closure we sutured the rotator interval and the subscapularis muscle with the capsule at its original insertion, and closure of the subcutaneous layer and skin was performed. A sling was used to immobilize and protect the repaired cartilage defect.

### Rehabilitation modus

Postoperatively the patients were immobilized in a sling for 48 h for initial protection of the periostal flap. Thereafter the sling was replaced by an abduction pillon for 3 weeks. Passive assisted range of motion started the on the 3rd postoperative day (30° abduction, 30° flexion, 60° IRO, 0° ARO), as recommended by Alfredson et al. [2], who reported good clinical results with early passive postoperative motion following periostal transplantation at the knee. At 4–6 weeks postoperatively the patients were allowed passively and actively to increase the range of motion to at least 90° of flexion and abduction. Beyond this time the shoulder was moved freely without limitations. After reaching a free range of motion a special muscle strengthening program was recommended.

### Radiological evaluation

The evaluation for osteoarthritic changes consisted of anteroposterior, outlet, and axillary views. The radiological classification was performed according to the typical radiological changes such as narrowing of the glenohumeral joint space, subchondral sclerosis or cystic changes, humeral osteophytes, and an irregularity or a deformation of the glenohumeral joint [9, 14]. Osteoarthritis due to a glenohumeral instability was classified according to Samilson [33].

### MRI evaluation

The evaluation of the shoulder was performed in paracoronal, transaxial, and parasagittal planes in a T1-weighted gradient echo sequence (TE 900, TR 24, SE 4.0) and gradient echo stir sequence (TE 1380, TR 16, SE 4.0). Nakanishi et al. [25] reported that cartilage damage is visible as a cartilage irregularity in native MRI and gradient echo sequence, but it is important to know that there is a great physiological variety of the thickness of the humeral cartilage [18].

### Statistics

For statistical evaluation we used Student's *t* test. The significance level was set at $P$=0.05.

## Results

### Clinical results

The CS improved significantly in each patient, from a preoperative mean of 43.4% to 81.8% at follow-up ($P$=0.0018; (Table 3). "Pain" reduced significantly from 5 points to 13.6 points ($P$=0.0053) and activities of daily life increased from 13.6 points to 18.6 points. There was no significant change in range of motion. Isometric strength at follow-up measured by the Isobex (Veribor, Germany) averaged 6.5 kg (1.5–10).

**Table 5** Radiological findings and classification of osteoarthritis preoperatively and at follow-up

| Patient no. | Preoperative | | Follow-up | |
|---|---|---|---|---|
| | Classification | Findings | Classification | Findings |
| 1 | 0 | 0 | 0 | 0 |
| 2 | Samilson 1 | Humeral subchondral irregularity, 3 mm caudal osteophyte | Samilson 2 | Joint space narrowing, humeral subchondral irregularity, 5 mm caudal osteophyte |
| 3 | Samilson 1 | 5 titanium anchors with corresponding humeral subchondral irregularity, 3 mm caudal osteophyte | Samilson 1 | Unchanged signs of humeral subchondral irregularity, 3 mm caudal osteophyte |
| 4 | 0 | 3 titanium anchors with corresponding humeral subchondral irregularity | Osteoarthritis 1 | Unchanged signs of humeral subchondral irregularity, 2 mm caudal osteophyte |
| 5 | 0 | 0 | 0 | 0 |

### Radiological results

Preoperatively two patients had a grade 1 glenohumeral osteoarthritis according to Samilson and Prieto [33] due to a glenohumeral instability. Three patients showed no signs of osteoarthritis. In four patients the osteochondral defects was visible as a subchondral irregularity at the humeral head. At follow-up three patients showed no radiological changes compared to the preoperative images. One patient with grade 1 osteoarthritis developed grade 2 according to Samilson and Prieto [33] with a joint space narrowing and a 5-mm caudal humeral osteophyte. Another patient with a grade 1 osteoarthrtis developed a 2-mm caudal humeral osteophyte (Fig. 3e, g). Radiological results are presented in Table 5.

### MRI results

Preoperatively the humeral cartilage defect was visible in four patients as an irregularity of the cartilage and subchondral bone plane (Fig. 3f). In one patient the cartilage defect was not visible on the preoperative MRI at all. Follow-up MRI reconfirmed the radiological findings. In all patients the area of the chondral defect was covered with a thin layer of regeneration tissue. Additionally MRI revealed an irregularity of the subchondral bone plate.

### Second-look arthroscopy

Second-look arthroscopy was performed in 3 patients and revealed a significant reduction of the repaired cartilage lesions. Patient no. 2 presented with a completely resurfaced cartilage lesion of grade 1 according to Outerbridge [30] (Fig. 2e). Patient no. 3 showed a resurfaced cartilage area of grade 2 which consisted of a soft fibrocartilage tissue (Fig. 3d). Patient no. 1 had a grade 3 cartilage lesion. In this patient the subchondral bone plate was covered with a thin regeneration tissue and islands of fibrocartilage tissue, which was an improvement over the preoperative cartilage lesion (Fig. 1c).

### Complications

There were no intraoperative or postoperative complications, especially no infection and no neurovascular complication.

## Discussion

During the past decade promising results have been reported in the operative treatment of symptomatic focal articular cartilage lesions at the lower extremity [4, 5, 6, 10, 12, 15, 22, 37]. This development raised the interest of surgeons and patients for addressing these lesions also at the shoulder joint but surgical technique was limited in most cases to an arthroscopic abrasion and débridement [3, 19, 20]. This did not solve the problem of the cartilage defect.

Therefore several advanced operative techniques have been developed in recent years. An elegant way of transferring autologous type II cartilage to the site of the chondral defect is the technique of osteochondral autograft transplantation introduced by Bobic et al. [5] and Hangody et al. [14, 15, 16]. The major disadvantage of this technique if used at the shoulder is that these plugs cannot be harvested at the shoulder itself but must be harvest from the knee joint which may lead to donor site morbidity [34, 35]. Additionally there is a significant limitation of autograft availability. Articular cartilage lesions greater than 2.5–3 $cm^2$ can also be restored by autologous chondrocyte implantation [6, 13, 24, 31]. The disadvantage of this technique is that it is still unpredictable and inconsistent for obtaining repair tissue that is at best a rather mosaic of bone, fibrous, fibrocartilaginous, and hyalinelike tissue [31, 34].

In 1997 the senior author started to use the microfracture technique according to Steadman [36, 37] for large symptomatic traumatic cartilage lesions at the humeral head. According to Steadman et al. [36, 37], the indications include both focal and degenerative grade 4 articular cartilage lesions. Contraindications are subchondral bone loss, significant compartment malalignment, and noncompliance with postoperative rehabilitation protocols. In knee surgery an optimal outcome has been noted in younger patients with smaller lesions and a well defined history of trauma [1].

A disadvantage of this technique is that the released marrow elements including mesenchymal stem cells, growth factors, and other healing proteins create only a fibrous cartilage or hyalinelike cartilage at the defect site. It is unclear whether the tissue will respond optimally to compression and shear loads [34], and whether it will be durable over time.

In our study an autologous periostal flap which was harvested from the area inferior to the greater tuberosity was sutured on top of the microfractured area. The tissue flap allows a localized complete coverage of the microfractured area and protects the cartilage formation that is induced by the microfracture. Furthermore the periosteum has a healing potential of its own and can serve as a matrix onto which other cells and/or growth factors can be adhered. According to O'Driscoll and Fiztsimmons [29], it contains pluripotential mesenchymal stem cells with the potential to form either cartilage or bone. Furthermore many growth factors that regulate chondrocytes and cartilage development are synthesized by the cambium layer of the periosteum in conditions conducive to chondrogenesis. These include transforming growth factor $\beta_1$, insulin-like growth factor 1, growth and differentiation factor 5, bone morphogenetic protein 2, integrins, and the receptors for these molecules [29].

Clinically all of our patients were pain-free at follow-up with activities of daily life. The overall CS improved significantly over preoperative values. One patient, a professional soccer player, returned to play in the first German soccer league. Another patient, a high performance swimmer, returned to recreational swimming. One patient stopped playing handball to prevent further damage to his nondominant shoulder. The other two patients did not perform a regular physical training prior to the surgery. A comparison of the preoperative radiographs to those at follow-up revealed a progression of the osteoarthritis in two cases. This finding raises the question of whether a progression of the osteoarthritis can be slowed by this kind of procedure. Nevertheless these two patients were pain free with activities of daily life and satisfied with the result of the operation.

Follow-up MRI revealed that the area of the chondral defect was covered with a thin layer of regeneration cartilage tissue in all patients, but there were still signs of an edema in the underlying subchondral bone plate. Arthroscopic second-look visualization and probing of the resurfaced defects documented that the chondral damage was reduced significantly in these patients from a grade 4 localized chondral defect to a grade 1, a grade 2, and a grade 3 chondral lesion covered with fibrocartilaginous tissue (Figs. 1, 2, 3). Corresponding to the knee joint, it is essential to correct the underlying or associated pathology of the cartilage defect. It is remarkable that two patients presented with a grade 4 cartilage defect at the humeral head caused by a titanium suture anchor following glenohumeral stabilization.

This study has several limitations. One must bear in mind that the clinical outcome may depend not only on the effect of microfracture and periostal flap surgery but also on the result of surgery for the underlying pathology. No histology of the regeneration tissue was taken at the time of second-look arthroscopy to verify the kind of repair tissue that filled in the defect. Finally, only five patients were included in the study.

Nevertheless the described operative technique leads to an improvement for repair of localized cartilage defects at the shoulder joint. At short-term follow-up the clinical and radiological results were satisfactory. It is important to perform second-look arthroscopy of the repaired chondral lesion to perform a histology of the regeneration tissue and to inspect the repaired area. Furthermore it is essential to address the underlying pathology of the shoulder. Long-term studies are necessary to confirm the clinical result of the described technique. It must be determined whether the development of an degenerative osteoarthritis can be slowed by this kind of procedure.

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