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ORIGINAL STUDY

Clinical and MRI outcome of an osteochondral scaffold plug for the treatment of cartilage lesions in the knee

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Conflicting clinical outcomes have been reported recently with the use of an osteochondral scaffold plugs for cartilage repair in the knee. In this study, twenty patients were consecutively treated for their cartilage lesions with the synthetic plug technique. These patients were prospectively clinically evaluated with a mean follow-up of 34.15 months. Magnetic resonance imaging (MRI) was used for morphologic analysis of the cartilage repair. The patients included in this study showed a significant gradual clinical improvement after the osteochondral scaffold plug. However, this clinical improvement was not confirmed by the MRI findings of this cohort study. Subchondral bone changes were seen in all patients on MRI and deficient filling of the defect was noticed in in 30.7% of the cases at 24 months of follow-up. There was no evidence found to support osteoconductive bone ingrowth. Therefore, the use of this type of osteochondral scaffold plug in osteochondral repair is questionable.
Level of evidence : IV.

Keywords : knee ; cartilage ; osteochondral ; scaffolds.

INTRODUCTION

The treatment of osteochondral lesions has become a major interest to orthopaedic surgeons because most lesions do not heal spontaneously and may predispose the joint to the subsequent development of secondary osteoarthritis (*21*). This poor repair capacity of articular cartilage has led to the development of various surgical techniques (*31*).

Frequently used treatment options are debridement, microfracture, osteochondral auto- or allografts, or cell- based techniques such as autologous chondrocyte implantation (ACI) (*37,38*). The most popular and most used is the microfracture technique including abrading the tidemark and creating small holes perpendicular to the subchondral bone plate to allow bleeding into the defect (*33*). Microfracture usually results in a fibrous-fibrohyaline unstructured repair tissue. This tissue lacks the biomechanical and viscoelastic features of hyaline cartilage. The potential short-term improvement in symptoms is usually followed by repair tissue failure and potentially by gradual deterioration to osteoarthritis and return of symptoms (*26*).

Follow-up studies on osteochondral autologous transplantation (OATS procedure), also known as

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mosaicplasty, demonstrated failure of integration of the transplanted cartilage and adjacent cartilage, with signs of degeneration of the transplanted hyaline cartilage (*35,37*).

Since 1987, autologous chondrocytes have been implanted in chondral lesions of the human knee (*5*). To this end, cartilage is harvested from a non weight-bearing area of the knee joint, digested, and the isolated chondrocytes are propagated in vitro in the monolayer culture condition. This procedure, proposed by Brittberg et al in 1994 and called ACI, has gained scientific and clinical support for use in the repair of focal articular cartilage lesions (*5,27*). However, during in vitro propagation of the chondrocytes, dedifferentiation of the cells can occur, and afterwards these fibroblast-like chondrocytes show different biosynthetic properties than the original cartilage cells in the knee joint (*3*).

Recently, there has been an increasing interest and awareness of importance of the subchondral bone for its role in cartilage repair. One should carefully consider the subchondral bone in the treatment of articular surface damage, in the evaluation of the results over time and in the determination of the patients' prognosis. In fact, the conditions of articular cartilage and its supporting bone are tightly coupled and should be viewed as a connected osteochondral unit (*15*).

The ultimate aim of the treatment is the restoration of normal knee function by regenerating hyaline cartilage in the defect and complete integration of the regenerated cartilage with the surrounding cartilage and underlying bone. The treatment should restore the physiological properties of the entire osteochondral unit. The use of bioabsorbable scaffolds for repair of chondral and osteochondral defects has recently been explored in laboratory and preclinical investigations (*1*). Such matrix scaffolds, implanted alone or in combination with cells, allow immediate filling of the defect and support local migration of chondrogenic and osteogenic cells that synthesize new ground substance (*11*).

The TruFit synthetic implant (TruFit CB, Smith & Nephew, Andover, Massachusetts) is one example of commercially available and licensed synthetic resorbable biphasic implants made of a patented composite hydrophilic polymer composed of polylactide coglycolide, calcium sulfate, and polyglycolide fibers. The TruFit plug is licensed for the treatment of chondral and osteochondral defects in Europe (off-label in USA) (*20*). The scaffold consists of two "phases". The bone phase contains calcium sulfate for stimulation of bone formation. Cartilage regeneration is instigated by the integration of cells and growth factors derived from the bone marrow that infiltrates the plug. Synthetic scaffolds such as the TruFit plug offer a number of potential benefits over traditional treatment options (*37*). The combination of marrow stimulation together with structural support can offer a benefit over microfracture. In the latter technique, bone marrow stem cells migrate in the fibrin network of a blood clot, but this "fibrin clot" is not mechanically stable enough to withstand tangential forces (*11*). The structural support property of a scaffold plug should prevent this problem. There is no donor-site morbidity as seen in the OATS procedure, and it requires only a single procedure instead of two-staged procedures for ACI.

However, conflicting clinical outcomes have been reported recently with the use of the TruFit plugs for cartilage repair in the knee (*8,11,32*). This study presents our experience with this type of osteochondral scaffold plug for cartilage repair in the knee. It was hypothesized that all patients treated with this osteochondral plug technique for a cartilage lesion in the knee would improve clinically and that all plugs would fill the osteochondral defects completely with tissue with a signal similar to cartilage and subchondral bone on magnetic resonance imaging (MRI).

Study population

Patients with 1 focal cartilage defect involving the femoral condyle, patella, or trochlea that could be treated preferably with 1 plug or a maximum of 2 plugs and with clinical symptoms (1 of the following symptoms : pain, swelling, locking, and "giving away") were eligible for treatment. There was no initial trial of nonoperative management. Smokers and Workers' Compensation cases were also included. We excluded cartilage defects with a size greater than 2 cm^2. Therefore, an MRI scan or

arthro-computed tomography scan was performed preoperatively to confirm our clinical suspicion of a cartilage defect. On the basis of the defect size observed on these investigations, we considered the patients eligible for treatment with the plug device and they were included in this study. Other exclusion criteria were age under 16 years or over 55 years, body mass index greater than 32, and the presence of multiple focal cartilage defects, untreated tibiofemoral or patellofemoral malalignment or instability, diffuse osteoarthritis or bipolar "kissing" lesions, major meniscal deficiency, and other general medical conditions such as diabetes or rheumatoid arthritis. Clinical experimentation was approved by the hospital's ethics committee, and informed consent to participate in the study and to comply with the postoperative regimen was obtained from all patients. The patients included in this pilot study were treated between October 2008 and November 2010.

In total, 20 patients (8 male and 12 female patients) were treated consecutively. The mean follow-up time was 34.15 months (range, 24 to 48 months). The right side-to-left side ratio was 10:10. The lesions were focal in all cases. Of the cartilage defects, 8 were located on the medial femoral condyle, 4 on the lateral femoral condyle, 5 on the patella, and 3 on the trochlea. All lesions were International Cartilage Repair Society grade III or IV *(19)* and had a mean size of 0.83 cm^2 (range, 0.38 to 1.58 cm^2). The cause of injury was traumatic in 7 cases, focal nontraumatic (focal degenerative lesions) in 9 cases, and osteochondritis dissecans in 4 cases. The mean age of the patients was 31.65 years (range, 17 to 53 years). The mean duration of symptoms before surgery was 26.30 months (range, 2 to 122 months). Previous surgery in 10 of the patients included 4 partial meniscectomies, 4 anterior cruciate ligament reconstructions, 1 case of meniscal suture, and 3 cartilage repair procedures, such as autologous chondrocyte implantation (n = 1) or microfracturing (n = 2) of chondral lesions. Associated procedures were performed in 5 patients : 2 Fulkerson osteotomies, 1 lateral release, 1 suturing of a medial meniscus tear, and 1 lateral allogeneic meniscal transplantation *(36)*.

Surgical Procedure

A mini-arthrotomy in a tourniquet-controlled bloodless surgical field was performed to allow access to the defect. The lesion was measured after the bottom of the cartilage defect was freshened, and the edges of the defect were trimmed back to stable walls of healthy cartilage. As described by Melton *et al* *(25)* the decision as whether to use 1 plug or multiple plugs is made at this stage, based on the characteristics of the lesion. Then, a single cylindrical hole (or multiple holes for multiple plugs) of 8 to 12 mm in depth is drilled through a drill sleeve into the defect. The drill hole size will be matched to the size of the defect and the planned implant diameter. A plug prepared to the same depth is introduced into the defect under direct vision. The implant then needs to be "tamped" down with a punch until the surface of the implant is continuous with the surrounding articular cartilage. This tamping is inherent to the technique and in accordance with the device manual. In our experience, we did not observe that this tamping caused any damage to the plugs. If more than 1 plug is required, a bridge of 1 to 2 mm should be left if possible. Finally, the implant is probed to ensure that the implant is stable and that the edges of the implant are congruent with the surrounding chondral surfaces *(25)*.

In 17 of the 20 patients, only 1 plug was inserted. In 7 patients a plug of 9 mm was used, in 2 patients a plug of 7 mm was used, and in 8 patients a plug of 11 mm was inserted. In the remaining 3 patients, 2 plugs were implanted. In 1 patient 2 plugs of 9 mm were used, in 1 patient a plug of 9 mm and a plug of 7 mm were used, and finally, in 1 patient a plug of 9 mm and a plug of 11 mm were implanted.

The postoperative regimen was as follows. The patients were non-weight bearing for 4 weeks. Achieving a normal gait pattern was advised at 10 weeks postoperatively. Maximum active flexion did not exceed 90° for the first 4 weeks of rehabilitation. Full range of motion was allowed 8 weeks postoperatively. Isometric quadriceps training, straight length raising, and hamstring isometrics were advised after the first 2 weeks. Return to low-impact sports was allowed 12 months after surgery *(17)*.

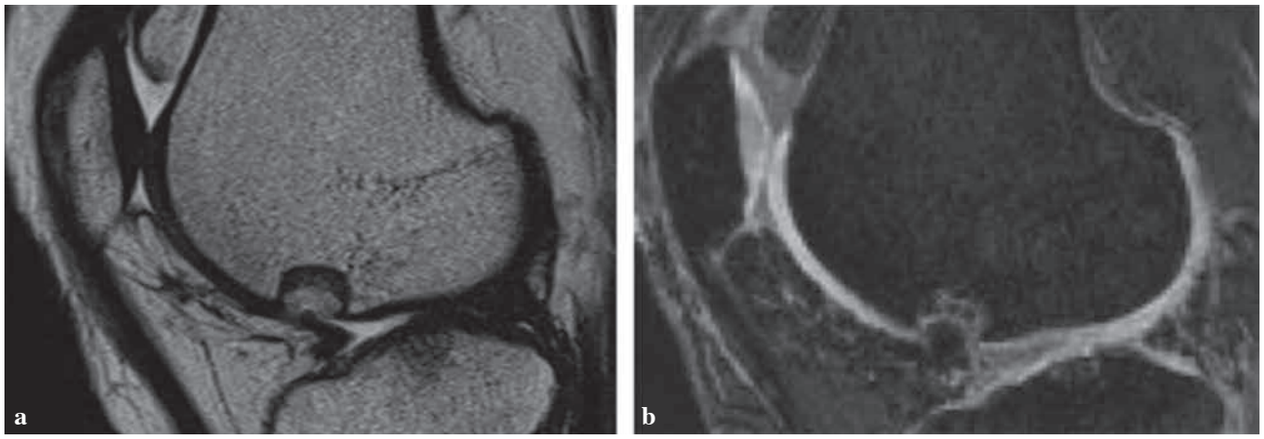

Fig. 1. — Sagittal proton-density- and T2-weighted turbo spin echo (TSE) image (a) and Sagittal FLASH-3D spoiled gradient echo MR image with water excitation (b) on 3.0 T MR unit. Two years after surgery, a partial filling at the depth of the osteochondral defect is observed with a remaining discontinuity of the articular surface.

Clinical evaluation

All 20 patients were clinically prospectively evaluated with use of the Knee injury and Osteoarthritis Outcome Score (KOOS), the Tegner activity scale and the visual analogue scale (VAS) for pain preoperatively and at 6, 12, 18, 24, 36 and 48 months of follow-up (*2,4,14,28-30,34*).

MRI technique

All MRI examinations at 6, 12 and 24 months of follow-up were performed on a 1.5-T or a 3-T MR unit (either a Magnetom Avanto, a Magnetom Symphony Tim or Magnetom Trio, Siemens Medical Solutions, Erlangen, Germany). Of the 20 patients, 19 had consented to follow the postoperative MRI evaluation protocol. The imaging parameters of the sequences were the same as published previously (*10-13*) (Fig. 1).

MOCART system

For the description of the repair tissue, we used the MOCART system previously published by Marlovits *et al* (*23,24*). This morphological MRI classification system was applied to the MRI images taken at 6, 12 and 24 months of follow-up. All MR images were evaluated by one independent reviewer who was blinded to the time following surgery. The MOCART scores were expressed as a percentage of the maximum score (*10-13*).

Statistical analysis

All data are expressed in terms of means and standard deviations. The Wilcoxon test was used to analyse statistical differences between between baseline and various follow-up measurements. For all tests $P < 0.05$ was considered significant. Statistical analysis was performed using SPSS statistics 21 (SPSS Inc, Chicago, IL).

RESULTS

During the follow-up period the VAS scores for pain indicated by the patients improved significantly (Fig. 2). The differences between the preoperative and postoperative values (12, 18, 24 and 36 months) were statistically significant ($P < .05$).

According to the Tegner activity scale, no significant improvement became apparent during the postoperative period ($P > .05$), except for the time point 6 months after the operation ($P < .05$) indicating that the patients were significantly worse according to the Tegner score at 6 months of follow-up.

The mean total KOOS scores improved statistically significantly when pre- and postoperative values (12, 18, 24 and 36 months) were compared

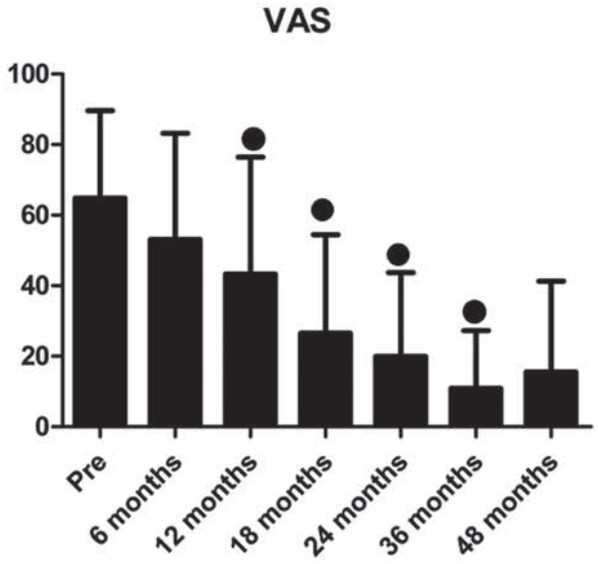

Fig. 2. — Mean values and standard deviations of the Visual Analogue Scale (VAS) for pain : preoperative (Pre) (N = 20) and postoperative : 6 (N = 20), 12 (N = 19), 18 (N = 15), 24 months (N = 14), 36 months (N = 7) and 48 months (N = 4). Values are expressed in millimetres. The black dots indicate statistically significant differences ($P < .05$) between the preoperative and postoperative values.

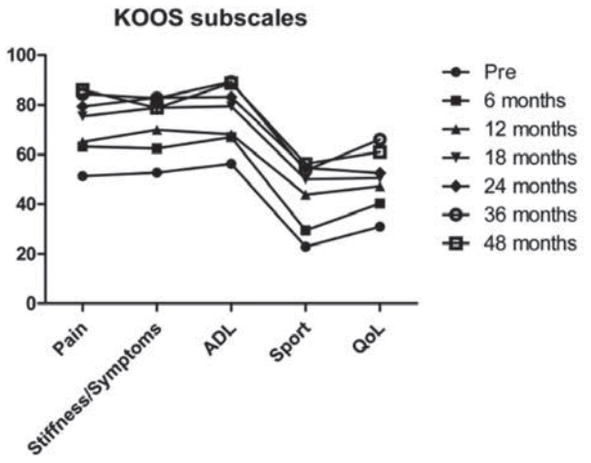

Fig. 3. — Mean values of the KOOS subscales (Pain, Symptoms/Stiffness, ADL (activities of daily living), Sport and Quality of Life (QoL)) : preoperative (Pre) (N = 38) and postoperative : 6 (N = 20), 12 (N = 19), 18 (N = 15), 24 months (N = 14), 36 months (N = 7) and 48 months (N = 4).

($P < .05$). In general, all KOOS subdomains improved statiscally significantly in the postoperative period, starting at 12 months of follow-up (Fig. 3).

The exact mean values and standard deviations of all clinical scores and *P* values of the comparison between preoperative and postoperative scores are given in table I.

No infections occurred in the postoperative period.

Of the 20 patients, 6 (30.0%) showed persistent symptoms or even more symptoms after the implantation of the synthetic plug. The symptoms did not improve over time. These patients were not treated for their cartilage lesions previously and were considered as clinical failures and therefore eligible for revision surgery. As published previously, the indications for revision surgery were persistent clinical symptoms or even more clinical symptoms after the insertion of a plug for a minimal period of 9 months (*11*). During revision surgery, the remnants of the plug were carefully removed. The remaining osteochondral defect was filled with autologous bone grafts harvested from the iliac crest. This approach leaves us the possibility to perform another cartilage repair procedure in the future if the clinical status of the patients would deteriorate.

Two patients underwent revision surgery at 9 months after the initial procedure. One patient underwent revision at 12 months of follow-up. The last patient had already undergone a mobilization under anesthesia because of a limited range of motion at 3 months of follow-up due to arthrofibrosis. Another two patients underwent revision surgery at 16 months of follow-up and 1 patient at 20 months of follow-up. All 6 patients experienced immediate and persistent relief of symptoms after the revision procedure.

Twenty-four month longitudinal follow-up of the repair tissue with the MOCART system :

During the 24-month follow-up period, it was shown that the MOCART scores statistically, significantly decreased over time (Fig. 4). In other words, a significant tendency of deterioration of the repair tissue was observed on MRI during the 24 months postoperative period.

Table I. — Mean values and standard deviations of total KOOS, individual KOOS subdomains, VAS for pain scores and Tegner activity levels : preoperative (Pre) (N = 20) and 6 (N = 20), 12 (N = 19), 18 (N = 15), 24 months (N = 14), 36 months (N = 7) and 48 months (N = 4). *P* values of the comparison between preoperative scores and scores at 6, 12, 18, 24, 36 and 48 months of follow-up are also given. The VAS for pain scores are expressed in mm

KOOS and Subdomains	Pre	6 months	12 months	18 months	24 months	36 months	48 months
KOOS total	214.2 ± 92.5	262.5 ± 135.3 (n.s.)	294.5 ± 148.2 (*P = 0.04*)	334.2 ± 118.9 (*P = 0.02*)	352.7 ± 73.4 (*P = 0.01*)	376.1 ± 89.5 (*P = 0.02*)	370.8 ± 104.5 (n.s.)
Pain	52.7 ± 16,7	63.2 ± 31.2 (n.s.)	65.1 ± 32.3 (n.s.)	75.4 ± 24.8 (*P = 0.02*)	79.4 ± 14.9 (*P = 0.01*)	84.4 ± 14.6 (*P = 0.02*)	86.0 ± 15.0 (n.s.)
Symptoms/Stiffness	56.3 ± 23,2	62.4 ± 28.9 (n.s.)	70.0 ± 24.7 (*P = 0.04*)	78.9 ± 19.9 (*P = 0.01*)	82.9 ± 11.4 (*P = 0.01*)	82.7 ± 14.6 (*P = 0.02*)	78.8 ± 14.3 (n.s.)
ADL	56.3 ± 23,2	67.1 ± 30.5 (n.s.)	68.2 ± 31.9 (n.s.)	79.4 ± 23.2 (*P = 0.02*)	83.2 ± 15.7 (*P = 0.01*)	89.3 ± 13.1 (*P = 0.02*)	88.8 ± 13.7 (n.s.)
Sport	22.9 ± 24.4	29.5 ± 30.1 (n.s.)	43.9 ± 38.2 (n.s.)	50.0 ± 34.5 (*P = 0.04*)	54.6 ± 27.6 (*P = 0.03*)	53.6 ± 32.4 (n.s.)	56.3 ± 39.0 (n.s.)
QoL	31.0 ± 18.2	40.3 ± 24.5 (n.s.)	47.3 ± 28.7 (n.s.)	50.6 ± 28.0 (*P* = 0.01)	52.5 ± 15.1 (*P* = 0.03)	66.1 ± 30.9 (*P* = 0.04)	61.0 ± 29.3 (n.s.)
	Pre	**6 months**	**12 months**	**18 months**	**24 months**	**36 months**	**48 months**
VAS for pain	64.8 ± 24.8	53.2 ± 30.0 (n.s.)	43.3 ± 33.2 (*P* = 0.04)	26.5 ± 27.9 (*P* = 0.01)	19.9 ± 23.8 (*P* = 0.01)	10.9 ± 16.5 (*P* = 0.02)	15.5 ± 16.5 (n.s.)
Tegner	3.8 ± 2.5	2.3 ± 2.1 (*P* = 0.02)	2.8 ± 2.5 (n.s.)	3.9 ± 2.2 (n.s.)	4.5 ± 2.4 (n.s.)	4.0 ± 3.6 (n.s.)	3.8 ± 3.6 (n.s.)

MRI data evaluated with the original MOCART system at 12 and 24 months of follow-up :

At 6, 12 and 24 months after procedure the MRI data were analysed according to the MOCART system (Table II). Briefly, complete filling of the defect was found in 4 cases (30.8%) at 24 months of follow-up. Bone marrow and subchondral lamina changes were observed in all patients (100.0%) at the same evaluation point. Synovitis was found in 3 patients (23.0%) two years after surgery.

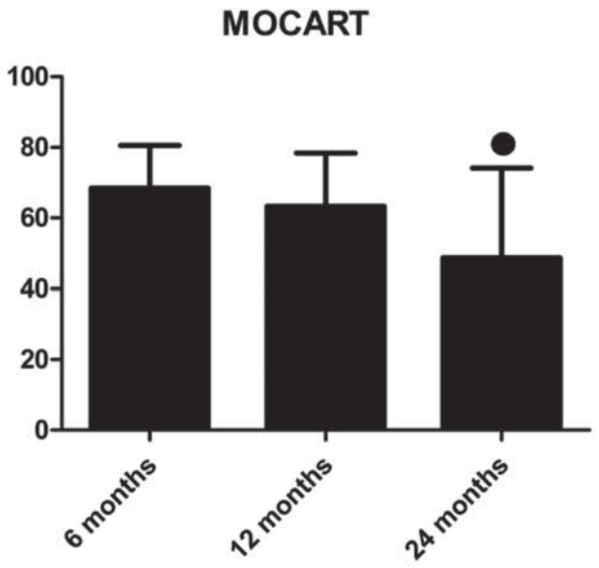

Fig. 4. — Mean values and SD of the original MOCART scores expressed in percentages : postoperative : 6 months (68.4 ± 12.1) (N = 19), 12 months (63.3 ± 15.1 ; 6 months à 12 months : n.s.) (N = 17) and 24 months (48.7 ± 25.4 ; 6 months à 24 months : P = .02) (N = 13). The black dots indicate statistically significant differences (P < .05) between the postoperative values.

DISCUSSION

In recent years, there has been an increasing interest in the subchondral bone, for its role in the etiopathogenetic processes of articular surface damage and for the careful consideration of it needed in treatment (*22*). In fact, the conditions of articular cartilage and its supporting bone are tightly coupled and should be viewed as a connected osteochondral unit. The biomechanical alterations caused by osteochondral defects affect the articular cartilage surrounding and opposing the lesion, as well as the homeostatic balance of the entire joint. As such, there is increased likelihood for clinical progression

Table II. — MRI evaluation of the repair tissue at 6 (N = 19), 12 (N = 17) and 24 months (N = 13) after the procedure in terms of number and percentage

Cartilage repair tissue grading scale (MOCART)			
Variables	6 months	12 months	24 months
1. Degree of defect repair and filling of the defect			
Complete (on a level with adjacent cartilage)	11 (57.8)	7 (41.2)	4 (30.8)
Hypertrophy (over the level of the adjacent cartilage)	1 (5.2)	0 (0.0)	1 (7.7)
Incomplete (under the level of the adjacent carilage; underfilling)			
>50% of the adjacent cartilage	6 (31.8)	8 (47.2)	4 (30.8)
<50% of the adjacent cartilage	1 (5.2)	1 (5.8)	3 (23.0)
Subchondral bone exposed (complete delamination or dislocation and/or loose body)	0 (0.0)	1 (5.8)	1 (7.7)
2. Integration to border zone			
Complete (complete integration with adjacent cartilage)	1 (5.2)	0 (0.0)	1 (7.7)
Incomplete (incomplete integration with adjacent cartilage)			
Demarcating border visible (split-like)	18 (94.8)	15 (88.4)	8 (61.5)
Defect visible			
< 50% of the length of the repair tissue	0 (0.0)	1 (5.8)	2 (15.4)
> 50% of the length of the repair tissue	0 (0.0)	1 (5.8)	2 (15.4)
3. Surface of the repair tissue			
Surface intact (lamina splendens intact)	5 (26.3)	3 (17.6)	2 (15.4)
Surface damaged (fibrillations, fissures and ulcerations)			
<50% of repair tissue depth	14 (73.7)	12 (70.8)	7 (53.8)
>50% of repair tisue depth or total degeneration	0 (0.0)	2 (11.6)	4 (30.8)
4. Structure of the repair tissue			
Homogenous	18 (94.8)	15 (88.4)	6 (46.2)
Inhomogenous or cleft formation	1 (5.2)	2 (11.6)	7 (53.8)
5. Signal intensity of the repair tissue			
T2-FSE			
Isointense	7 (36.8)	5 (29.4)	3 (23.0)
Moderately hyperintense	10 (52.7)	8 (47.1)	4 (30.8)
Markedly hyperintense	2 (10.5)	4 (23.5)	6 (46.2)
3D-GE-FS			
Isointense	5 (26.3)	4 (23.4)	0 (0.0)
Moderately hypointense	9 (47.4)	11 (64.8)	9 (69.2)
Markedly hypointense	5 (26.3)	2 (11.6)	4 (30.8)
6. Subchondral lamina			
Intact	0 (0.0)	0 (0.0)	0 (0.0)
Not intact	19 (100.0)	17 (100.0)	13 (100.0)
7. Subchondral bone			
Intact	0 (0.0)	0 (0.0)	0 (0.0)
Not intact	19 (100.0)	17 (100.0)	13 (100.0)
8. Adhesions			
No	19 (100.0)	17 (100.0)	13 (100.0)
Yes	0 (0.0)	0 (0.0)	0 (0.0)
9. Effusion			
No	8 (42.2)	9 (52.9)	10 (77.0)
Yes	11 (57.8)	8 (47.1)	3 (23.0)

to more widespread joint degeneration through mechanical disruption of joint motion, loose body formation and mechanical wear in the involved compartment. This progressive decline may lead to degenerative joint disease with earlier onset of osteoarthritis. Therefore, the treatment goal for osteochondral defects should be to restore the physiological properties of the entire osteochondral unit, aiming to achieve a more predictable repair tissue that closely resembles native articular surface and remains durable over time (*15*).

Several techniques with variable success rates haven been proposed to treat osteochondral defects (*9,31*). In a recent review, all current available evidence for the treatment of osteochondral defects with a TruFit plug was published (*37*). This review stated all groups reported an improvement in clinical outcome at 6 to 12 months of follow-up. However, these study groups were not compared to a control group, in which an improvement can be expected in the natural history after an acute trauma with an osteochondral lesion (*6*).

Longer clinical follow-up data are scarce and conflicting. Hindle *et al* indicate improvement of clinical outcome scores at 22 months (± 8.6 months) compared to preoperative status (*18*).

Joshi *et al* also describe a longer follow-up period of 24 months, with worsening of clinical outcome in almost all included patients (*20*). Carmont *et al*,who reported a case of delayed incorporation of an articular cartilage defect treated with TruFit plugs, claim that alleviation and resumption of functional activity after 24 months of continued rehabilitation can still be expected (*7*). Our study with a mean follow-up time of 34.15 months displays an overall gradual clinical improvement in time. However, a clinical failure rate of 30% was observed.

In the available literature, radiological findings show favourable MRI findings at 6-month follow-up regarding filling of the defect and plug incorporation in the adjacent cartilage (*37*). This statement was confirmed by our findings. We observed a filling of more than 50% of the osteochondral defect in 94.8% of the cases at 6 months of follow-up. However, in most of the studies these findings deteriorate in the intermediate postoperative period and improve again in a longer follow-up period (*37*). This was not seen in our study. We observed a statistically significant deterioration of the repair tissue at 24 months of follow-up as indicated by the MOCART scores. There was no evidence found to support osteoconductive bone ingrowth. We observed a high rate (100.0%) of subchondral bone abnormalities, including bone edema, sclerosis, granulation tissue and cysts instead of subchondrol bone ingrowth. Moreover, we observed a deficient filling of the defect in 30.7% of the cases at 24 months of follow-up.

In general, one can say that the majority of the available literature indicates some clinical and MRI issues with the use of this type of plugs in the intermediate follow-up period (12 to 24 months of follow-up) (*37*). This statement was confirmed with the clinical and MRI findings observed in the presented cohort study. This could be explained by the lack of subchondral bone formation. It is probably crucial for the newly formed cartilage to achieve mechanical characteristics that match those of native cartilage (*20*). As the bone formation is poor after treatment with a TruFit plug, its use in osteochondral repair is questionable. Even more, a deep lesion is made in the subchondral bone, which makes revision surgery more difficult. Future designs of synthetic biphasic scaffolds should focus further on establishing subchondral bone that has the biomechanical and structural potential to support cartilage formation (*16*). The use of this type of scaffolds for repair of osteochondral defects should be carefully and extensively explored in laboratory and preclinical investigations. Well-designed, large-scale, randomized controlled trials are needed to investigate the value of future synthetic biphasic plug before it can be implemented in clinical practice.

It must be emphasized that only a relatively small number of study objects were presented in this study, that the follow-up period was limited, that all MRI images were evaluated by only one independent observer and that no control group was used. Moreover, the MOCART scoring system used in this study does not correlate with clinical scores and gives no indication on the quality of the repair tissue. Other important drawbacks of this pilot study population are that associated procedures were performed in 5 patients (25.0%), that different lesion

locations of the knee were treated (8 were located on the medial femoral condyle, 4 on the lateral femoral condyle, 5 on the patella, and 3 on the trochlea) and that 4 patients (20.0%) underwent a previous partial meniscectomy. These drawbacks could also have their implications on the cartilage healing potential of the presented plug technique.

CONCLUSION

The osteochondral scaffold plug demonstrated a clinical improvement during a mean follow-up of 34.15 months. However, a clinical failure rate of 30% was observed. Moreover, subchondral bone changes were seen in all patients on MRI and deficient filling of the defect was noticed in in 30.7% of the cases at 24 months of follow-up. There was no evidence found to support osteoconductive bone ingrowth. Therefore, the use of this type of osteochondral scaffold plug in osteochondral repair is questionable. Well-designed, large-scale, randomized controlled trials are needed to investigate the value of future synthetic biphasic plug before it can be implemented in clinical practice.

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