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Osteochondral Lesion of the Talus

Is There a Critical Defect Size for Poor Outcome?

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Background: Identifying factors associated with favorable or unfavorable outcomes would provide patients with accurate expectations of the arthroscopic marrow stimulation techniques.

Purpose: To investigate the prognostic significance and optimal measures of defect size in osteochondral lesion of the talus as treated with arthroscopy.

Hypothesis: A critical, or threshold, defect size may exist at which clinical outcomes become poor in the treatment of osteochondral lesion of the talus.

Study Design: Cohort study; Level of evidence, 3.

Methods: In sum, 120 ankles underwent arthroscopic marrow stimulation treatment for osteochondral lesion of the talus and were evaluated for prognostic factors. Clinical failure was defined as patients' having osteochondral transplantation or an American Orthopaedic Foot and Ankle Society (AOFAS) Ankle-Hindfoot Scale score less than 80. Linear regression analysis and the Kaplan-Meier method were used to identify optimal cutoff values of defect size.

Results: Eight ankles (6.7%) required osteochondral transplantation, and 22 ankles (18.4%) were considered failures because of AOFAS scores less than 80, which indicated fair or poor results. Linear regression analysis showed a high prognostic significance of defect area and suggested a cutoff defect size of 150 mm² for the optimum identification of poor clinical outcomes ($P < .001$). Only 10 of 95 ankles (10.5%) with a defect area <150 mm² showed clinical failure, whereas in patients with an area ≥ 150 mm², the clinical failure rate was significantly higher (80%, 20/25). There was no association between outcome and the patient's age, duration of symptoms, trauma, associated lesions, and location of lesions ($P > .05$).

Conclusion: Initial defect size is an important and easily obtainable prognostic factor in osteochondral lesions of the talus and so may serve as a basis for preoperative surgical decisions. A cutoff point exists regarding the risk of clinical failure at a defect area of approximately 150 mm² as calculated from magnetic resonance imaging.

Keywords: ankle; osteochondral lesion; defect size; prognostic factor

Osteochondral lesion of the talus (OLT) is a common cause of chronic ankle pain and disability. Although recent studies have shown that focal chondral and osteochondral defects progress to osteoarthritis, analyses of the clinical features of predictive values—such as age, sex, duration of symptoms, location, and size of the cartilage defect—have been inconsistent and unsatisfactory.^{26,29,31} Although

many patients with persistent symptoms elect to have arthroscopic surgery (with generally excellent results), the arthroscopic approach has less predictable results among patients with large osteochondral lesions.^{6,10,11} Current treatment techniques for OLTs are based solely on long-term retrospective clinical studies and extrapolated data from other human joints or animal investigations.^{2,3,17}

Identifying factors associated with favorable and unfavorable outcomes would provide patients with accurate expectations of the arthroscopic marrow stimulation techniques. Across several series, predictors of the worse possible outcomes of arthroscopic treatment include the presence and severity of associated lesions, as well as the size of the cartilage defect.^{4,6,8,10,11,13} However, these variables have traditionally been assessed with plain radiographs or during

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arthroscopic surgery. Plain radiographs are insensitive and provide only unidimensional measures of defect size, and arthroscopic examination occurs after the decision to undertake surgery has been made; thus, neither approach is suitable for preoperative prognostic assessment. The majority of patients undergoing arthroscopic surgery undergo preoperative magnetic resonance imaging (MRI), which provides excellent visualization of the cartilage and soft tissue. To date, no studies have been conducted on the association between defect size on MRI and patient outcomes of arthroscopic surgery; that is, the critical, or threshold, defect size at which clinical outcomes of arthroscopic treatment become poor has not been clearly identified.

The aim of this study was (1) to elucidate the prognostic significance of defect size using a large series of patients treated by arthroscopic marrow stimulation techniques and (2) to identify the cutoff point for defect size at which risk factors for poor outcomes can be identified with maximum sensitivity and specificity.

MATERIALS AND METHODS

This retrospective analysis included 117 patients (120 ankles) with diagnosed OLT who received arthroscopic treatment from January 2001 to June 2006. Their average age at operation was 34.6 years (range, 13-66 years). There were 80 men and 37 women. The mean duration of symptoms was 27.7 months (range, 6-120 months) and the average follow-up was 44.5 months (range, 12-81 months).

All the patients had localized defects on the talar dome, with symptoms of ankle pain or functional limitations despite a minimum of 6 months of nonsurgical management. Only primary cases with no previous surgical treatment for OLT were included. We excluded patients with diffuse arthritic changes, as well as those with associated ankle fractures. In sum, 64 ankles had microfractures and 56 had abrasion arthroplasties.

We used the American Orthopaedic Foot and Ankle Society (AOFAS) Ankle-Hindfoot Scale score¹⁸ to evaluate postoperative outcomes. Clinical results at final follow-up were graded according to the criteria of Saxena and Eakin²⁷ (excellent, 90-100; good, 80-89; fair, 70-79; and poor, <70). Clinical failure was defined as patients' having fair or poor outcomes (AOFAS score <80) or osteochondral transplantation. Success was defined as patients' having excellent or good outcomes (AOFAS score ≥80) or not requiring osteochondral transplantation.

At preoperative and final follow-up examinations, we obtained anteroposterior and lateral weightbearing radiographs to assess the ankle for degenerative arthritis. On the Ankle Arthritis Rating Scale,³² stage 0 is defined as no radiographic changes; stage 1, the presence of osteophytes without joint space narrowing; stage 2, joint space narrowing; and stage 3, deformation of the joint space. We considered stages 2 and 3 as diffuse arthritic changes. We performed MRI—including CE 3D-FSPGR images (contrast-enhanced, fat-suppressed, 3-dimensional fast spoiled gradient-recalled acquisition in the steady state)—to measure defect size and to evaluate any associated

injuries and ligament damage before the operation. This MRI technique was recently reported to have a high sensitivity and specificity in diagnosing soft tissue impingement and syndesmosis injury in the ankle joint.^{12,14,15,20} To avoid potential bias, an independent observer, uninvolved in the care of the patients and blinded to the intention of this study, evaluated the MRI films. We classified the injuries according to the criteria of Anderson et al¹ as follows: stage 1, subchondral talar compression; stage 2, incomplete separation of the fragment; stage 2a, formation of a subchondral cyst; stage 3, unattached, undisplaced fragment; and stage 4, displaced fragment. The location of the lesion was described as medial or lateral on coronal images and anterior, center, or posterior on sagittal images.

The following parameters, which reflect defect size, were defined and determined for each patient: coronal length (horizontal extension measured from the coronal image), sagittal length (horizontal extension measured from the sagittal image), depth (vertical extension measured from the sagittal image), and area (as calculated by the ellipse formula, from coronal and sagittal length: $A = ab\pi = \text{coronal length} \times \text{sagittal length} \times 0.79$). Before the MRI films of the patients were evaluated, a subgroup ($n = 37$) was randomly taken to compare size measurements (length, depth, and area) based on MRI films with those determined by arthroscopic examination. A good correlation ($r = .69, P < .001$) was found by linear regression analysis, with a systematic deviation of -5.1 mm for measurements determined by MRI.

The principal dependent variable was the AOFAS score at follow-up. This variable was essentially normally distributed, thus justifying the use of parametric statistics. Bivariate associations between preoperative and follow-up variables were assessed with the paired sample *t* test (for clinical results) and Pearson correlation coefficients (for continuous predictors and outcomes). We used stepwise multivariate linear regression to assess the associations between MRI variables and clinical outcome. The estimated probabilities of clinical failure were calculated using the Kaplan-Meier method.¹⁶ Clinical success time was measured from arthroscopic treatment to last follow-up evaluation or osteochondral transplantation. Failure (AOFAS score <80 or osteochondral transplantation) was the endpoint. The Cox proportional hazards regression model⁷ was used to assess significance probabilities of prognostic variables, including general characteristics. Differences were considered significant at $P < .05$. These calculations were performed by a statistician using SPSS 12.0.1 (SPSS Inc, Chicago, Illinois).

Surgical Technique

The arthroscopic procedure could be performed in a standardized manner for every case. After removal of the cartilage cap and as followed by excision of the lesion, a microfracture was performed at areas where the subchondral bone was intact. Multiple perforations perpendicular to the joint surface were made by a microfracture awl (Linvatec, Largo, Florida), as described by Steadman et al.³⁰ For areas with losses of subchondral bone, abrasion arthroplasty was performed by removing loose chondral or osteochondral

fragments with a ring-shaped or curved curette and by trimming damaged cartilage with a power shaver until a stable, smooth articular surface was created. For associated lesions, the presence of ankle instability, subchondral cysts, impingement syndrome and distal tibial plafond lesions were checked and treatment modalities noted. After the operation, we recommended tolerable weightbearing for the patients without associated lesions. For the 26 patients with lateral ligament reconstruction, postoperative short leg walking cast immobilization with partial weightbearing for 4 weeks was followed by joint motion and muscle-strengthening exercise for 8 weeks.²³⁻²⁵ Sports or high-impact activities were limited for at least 3 months.

RESULTS

Preoperative MRI Findings

All patients had at least some degree of cartilage defect in at least 1 compartment. The most commonly affected surface was the medial talar dome, for which 80 cases (66.7%) had cartilage signal abnormalities, followed by the lateral talar dome, where 35 cases (29.1%) had such abnormalities. Only 5 cases (4.2%) had bilateral lesions. In regard to the talar dome in an anteroposterior plane, medial lesions involved the posterior dome in 42.1% of cases (35/83) and the center in 40.9% (34/83). However, lateral lesions predominantly involved the anterior dome (64.8%, 24/37). We analyzed clinical outcomes relative to the lesion location. Although no significant difference could be determined among the 6 zones, the anterior lesions had better results than the posterior lesions. This difference was statistically significant ($P = .029$). The MRI staging system by Anderson et al¹ showed 27 cases with stage 1 (22.5%), 31 cases with stage 2 (25.8%), 35 cases with stage 2a (29.2%), 26 cases with stage 3 (21.6%), and 1 case with stage 4 (0.9%). There was no statistically significant difference among these 5 groups with regard to clinical outcome ($P > .05$).

Outcomes at Follow-up

The mean preoperative AOFAS score was 62.95 ± 13.92 , which increased to 85.09 ± 8.45 postoperatively. The pain subscore (total 40 points) improved from 23.2 ± 9.91 to 32.1 ± 6.16 , showing an increase of 8.9 ± 9.58 points ($P < .001$). No differences were noted between microfracture and abrasion arthroplasty groups ($P > .05$). At the average 35.6-month follow-up, the overall clinical failure rate was 25% (30/120). Eight ankles (6.7%) required osteochondral transplantation and 22 (18.4%) were considered failures because of AOFAS scores less than 80. In 8 cases of osteochondral autograft transplantation, the AOFAS score was 53.6 ± 11.01 preoperatively, which increased to 85.2 ± 5.06 postoperatively ($P = .043$).

Associations Between Defect Size and Outcome

The mean length of lesions was 10.47 ± 2.57 mm in the coronal images, as compared with 12.33 ± 3.14 mm in the sagittal images. The mean depth was 5.77 ± 2.45 mm, and

TABLE 1
Comparison of Defect Size and Clinical Results
Between Clinical Success and Failure Groups^a

	Success (n = 90)	Failure (n = 30)	Total (n = 120)
Defect size			
Coronal length, mm	9.83 ± 2.09	12.40 ± 2.93	10.47 ± 2.57
Sagittal length, mm	11.40 ± 2.45	15.10 ± 3.38	12.33 ± 3.14
Depth, mm	5.35 ± 2.40	7.03 ± 2.20	5.77 ± 2.45
Area, mm ²	90.15 ± 33.45	176.46 ± 56.30	111.73 ± 54.93
AOFAS score			
Preoperative	63.31 ± 13.94	61.90 ± 14.05	62.95 ± 13.92
Postoperative	88.70 ± 5.61	74.26 ± 5.88	85.09 ± 8.45

^aResults of the paired *t* test were all significant ($P < .05$) except preoperative AOFAS score. AOFAS, American Orthopaedic Foot and Ankle Society.

the mean area was 111.73 ± 54.93 mm². Lesions in patients who showed clinical failure were significantly larger in whole size measurements. In the failures, the mean length of the lesion was 12.40 ± 2.93 mm in the coronal images and 15.10 ± 3.38 mm in the sagittal images. The mean depth was 7.03 ± 2.20 mm, and the mean area was 176.46 ± 56.30 mm², which was larger than 150 mm² (Table 1). Among these 4 variables, the strongest MRI predictor of clinical outcome was area of the lesion ($r = -.843$, $P < .001$). The depth of cartilage defect had a weak association with clinical outcome ($r = -.344$, $P < .001$), whereas the length in the coronal and sagittal images had moderately strong correlations ($r = -.642$, $-.676$, respectively; $P < .001$).

In multivariate analyses, the area of cartilage defect explained 71.1% of the variability in follow-up AOFAS score. Interestingly, a cutoff point seemed to exist for prognostic influence of defect size. As illustrated in the scatter plot with a regression line (Figure 1), patients with a defect area larger than 150 mm² were scored lower than 80 points significantly more often; therefore, we dichotomized patients at the critical defect area of 150 mm² for comparative analysis of clinical outcomes. For Kaplan-Meier analysis, clinical failure (osteochondral transplantation or AOFAS score <80) was the endpoint. The group of patients with smaller defect areas showed a significantly superior clinical success rate as compared with the group with larger lesions at 48-month follow-up (94% versus 60%, $P < .001$) (Figure 2). Only 3 of 58 ankles (5.2%) with a defect area <100 mm² showed clinical failure, likewise for 7 of 37 ankles (18.9%) with an area between 100 mm² and 150 mm², whereas the clinical failure rate was significantly higher in patients with an area greater than 150 mm² (80%, 20/25) (Figure 3). Table 2 lists the characteristics of the patients based on defect area (<150 mm² versus ≥ 150 mm²).

Other Prognostic Factors

On review of medical records and arthroscopic findings, 85 cases (70.8%) involved soft tissue impingement; 35 cases (29.2%), subchondral cyst; 26 cases (22.3%), chronic lateral

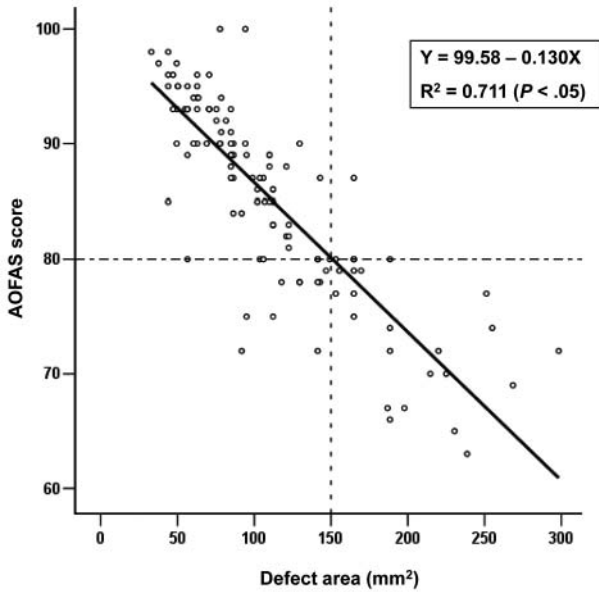


Figure 1. Correlation between defect size and clinical outcome.

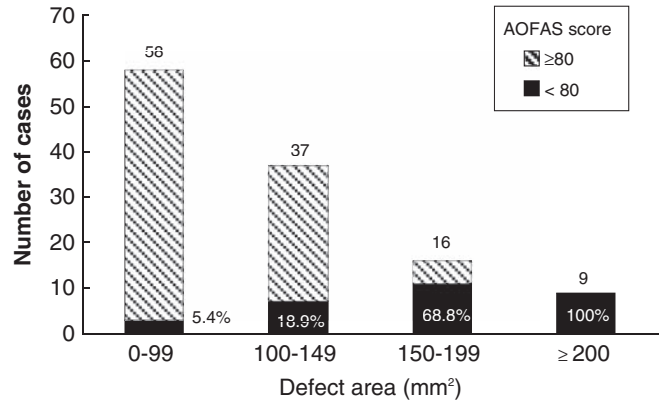


Figure 3. Distribution by defect size and clinical outcome in 120 ankles.

All prognostic factors—including the patient’s age (<30 years or ≥30 years), duration of symptoms (<1 year or ≥1 year), trauma, associated lesions, and location of lesions—were entered as variables for Cox regression analysis, which did not show a significant influence on clinical outcome ($P > .05$) (Table 2).

There were no complications in the current study, including nerve injury, infection, and delayed wound healing. For patients who received osteochondral transplantation, integration of the donor plug was confirmed on follow-up radiographs. One patient complained of a “giving way” symptom of the knee 6 weeks after surgery, which resolved in 3 months. Twenty-two patients who showed clinical failure (AOFAS score <80) but refused to receive more aggressive surgery were managed with nonoperative measures, including rest, immobilization, anti-inflammatory medication, and physical therapy. Although the AOFAS score in this subset of patients improved by a mean of 14 points postoperatively (from 61.22 ± 16.12 to 75.22 ± 5.81), they did not meet our criteria for a clinical success.

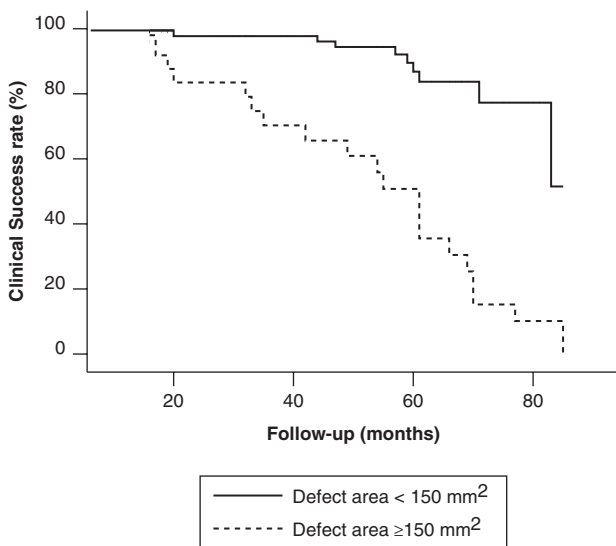


Figure 2. Kaplan-Meier survival curve for size-dependent clinical success rate. The solid line represents the size of osteochondral lesion smaller than 150 mm², whereas the broken line represents the defect size larger than 150 mm². Note that a clear cutoff point of change in clinical success rate exists at 150 mm².

ankle instability; and 18 cases (15%), chondral lesion in the tibial plafond (Table 2). Analysis of radiographic imaging studies and clinical results in regard to the presence of associated lesions showed that soft tissue impingement, chronic lateral ankle instability, and tibial plafond lesions had no substantial effect. However, based on MRI, cases with subchondral cysts (8.16 ± 3.38 mm) had deeper lesions than did those without cysts (4.81 ± 2.03 mm, $P < .001$).

DISCUSSION

Whereas clinical diagnosis, etiologic factors, lesion description, and treatment of OLT have been well delineated, the size of the talar lesion as a preoperative prognostic factor for arthroscopic marrow stimulation treatment has not been reported in the clinical literature. Although it is unknown if defect size alters clinical outcome, some biomechanical results indicate that ankle contact pressure and contact area are affected by larger dome lesions. Christensen et al⁵ suggested that the size of the lesion may alter contact stresses in the ankle and that statistically significant changes in contact characteristics occur with lesions larger than 7.5×15.0 mm. They concluded that this finding may indicate that lesion size plays a role in predicting long-term outcome in patients with OLT.

A review of the literature reveals some variation in defect size at which authors choose to treat OLTs. Giannini and Vannini¹⁰ reported that arthroscopic treatment seems to be the treatment of choice for lesions smaller than 1.5 cm²

TABLE 2
Patient Characteristics and Clinical Outcome for Groups by Defect Size^a

Variable	Defect Size				Cox Regression ^b	
	<150 mm ² (n, 95)		≥150 mm ² (n, 25)		RR	P
	n	%	n	%		
Age, y					1.26	.58
<30	41	43.2	15	60		
≥30	54	56.8	10	40		
Symptom, year					1.69	.19
<1	49	51.6	12	48		
≥1	46	48.4	13	52		
Trauma	69	72.6	22	88	0.95	.93
Location					0.99	.98
Medial/lateral	62/33	65.3/34.7	21/4	84/16		
Anterior/middle/posterior	32/32/31	33.7/33.7/32.6	6/9/10	24/36/40		
Associated lesions						
Soft tissue impingement	68	71.6	17	68	1.56	.32
Ankle instability	22	23.2	4	16	1.02	.96
Subchondral cyst	27	28.4	8	32	0.49	.13
Tibial chondral lesion	17	17.9	1	4	1.23	.79
Clinical outcome, AOFAS score						
Excellent/good (≥80)	43/42	45.3/44.2	0/5	0/20		
Fair/poor (<80)	10/0	10.5/0	14/6	56/24		

^aRR, relative risk; AOFAS, American Orthopaedic Foot and Ankle Society.

^bCox proportional hazards regression analysis.

(150 mm²) and that it may also be attempted in lesions from 1.5 to 2.0 cm². They also stated that mosaicplasty and autologous chondrocyte implantation are the 2 most suitable methods for patients younger than 50 years with lesions more than 1.5 cm² or with failed previous surgery. Gobbi et al,¹¹ using the Pearson correlation analysis, showed an inverse relationship between defect size and outcome (microfracture, $r = -.92$; osteochondral transplantation, $r = -.89$). Chuckpaiwong et al⁶ reported a strong correlation between lesion size and successful outcome. They found excellent results in patients with osteochondral lesions smaller than 15 mm, regardless of location. These results are consistent with the outcomes of our study, which found significant correlations between defect size and clinical outcomes. Patients with osteochondral lesions smaller than 150 mm² had better results than did those with larger lesions.

However, in most studies, arthroscopic findings served as a basis for determining initial defect size, although it is unknown whether arthroscopic evaluation of the ankle joint can yield correct information about the size of a cartilage lesion. Schäfer et al²⁸ found that assessing the size of an osteochondral lesion based on arthroscopy resulted in over- and underestimation of the defect size. Furthermore, an arthroscopic measurement is invasive, particularly in patients for whom nonoperative therapy is warranted. Magnetic resonance imaging has been shown to accurately delineate the size and location of osteochondral lesions, and it is more sensitive than computed tomography in detecting radiographically occult lesions.^{1,14,15}

The management of OLT is complex, and options include nonsurgical treatment, marrow stimulation techniques, and tissue transplantation techniques. Small lesions are managed differently from large lesions in that an attempt is made to induce fibrocartilage formation by marrow stimulation techniques. Given that recently innovated surgical techniques, such as osteochondral transplantation and autologous chondrocyte implantation, may be used exclusively for large osteochondral defects, an accurate noninvasive method for measuring the defect size would allow surgeons to determine surgical options, and it would aid in their preoperative planning.

An interesting finding of this study is that a cutoff point exists for prognostic influence of defect size—that is, 150 mm². This phenomenon is not evident in unidimensional measures based on plain radiographs, in which the risk of clinical failure steadily increases with increasing size. We can assume that a critical defect size characterizes a cartilage loading around the lesions. Minas and Nehrer²¹ observed that the marrow technique reduces weight stress on the area of cartilage repair and redistributes weight-bearing to other, less fragile tissues. For larger defects, the weightbearing redistribution is a disadvantage because the stress increment could produce damage in some regions of the healthy cartilage, which may explain why patients with large lesions have yielded poor outcomes and why the marrow stimulation techniques were not adequate to redistribute the weight in those cases.

We found no association between several prognostic factors—including the patient's age, duration of symptoms,

trauma, associated lesions, and location of lesions—and AOFAS score at follow-up. Ferkel et al⁹ reviewed the long-term results of patients with chronic symptomatic OLT treated by arthroscopic excision and drilling and found no correlation between the patient's age, sex, duration of symptoms, and location of lesions and clinical outcomes, which is consistent with our results. They also found no correlation between plain radiograph, computed tomography, or MRI grade and clinical outcomes. Our proposed hypothesis is that the majority of classification systems for OLTs are based exclusively on depth and not area. Several authors have found that cartilage defects with cysts adversely affected outcomes.^{9,19} In our study, OLTs with subchondral cysts had deeper lesions on MRI than did those without cysts, whereas the depth of lesions had a weak association with clinical outcomes. This is in agreement with the findings of Han et al,¹³ who believed that the existence of a small cyst in an OLT does not affect the postoperative prognosis.

One question that had to be primarily addressed was, how far did length, as measured from MRI, reflect the actual size of the lesion?

Mori et al²² described a close correlation between the size as determined by MRI and that as based on arthroscopic findings. A comparison of our measurements, as based on MRI and arthroscopic findings in the subgroup (n = 37), showed good correlation with a minor systematic deviation of -5.1 mm. To exclude any bias by patient selection, we compared the patient characteristics and AOFAS scores of the group under investigation with the entire group of patients. There was good concurrence between the subgroup and the entirety, in all respects. We can therefore consider the selected group to be representative. Because values determined by arthroscopy are more likely to represent the real size of the lesion, this deviation might indicate that MRI systematically underestimates the defect size.

Another bias could arise from calculating the defect areas by regarding them as ideal ellipses. Because of the convexity of the talar domes, conventional axial planes could not always clearly visualize the lesion. With the assistance of the Picture Archiving Communication System (General Electrics, Milwaukee, Wisconsin), we attempted to accurately estimate the size of the defect area by computer-assisted evaluation of axial MRI. We compared these findings with the areas determined by the ellipse equation, and linear regression showed a close correlation of values determined by either method. However, areas calculated by measured lengths were approximately 15% larger than areas evaluated by computer assistance. We therefore have 2 opposing effects: on one hand, a moderate systematic underestimation of area when using MRI for determination of lesion diameters; on the other, a systematic overestimation by direct calculation as ideal ellipses. These 2 effects would partially neutralize each other, which shows that we did not deal with the real chondral lesions but with good and easily available approximations. New technologies—such as computer-assisted area determination, regardless of image plane—could solve this problem. This might be a worthwhile prospective goal. Nevertheless, for the aim of this retrospective study, diameter determinations using MRI, as well as area calculations with equations, were

reliable methods for examining the prognostic influence of defect size. These simple methods for estimating defect size can be useful in large, multicenter studies.

These data are the first that we are aware of showing that preoperative defect size may help to predict the outcome of OLT. The validity of this measure was comparable to the other well-proven predictors: age, trauma, duration of symptoms, and location of lesions. Given that many patients undergo MRI preoperatively, prognostic information from MRI is routinely available before surgery for the majority of patients. Surgeons can use this information to help set appropriate preoperative expectations of surgical outcome in patients who elect to have arthroscopic marrow stimulation treatment. Other studies indicate that intraoperative assessment of defect size is an important predictor of outcome.^{6,28} However, arthroscopic assessment is not performed until after the surgical decision has been made; therefore, it cannot be used to help surgeons and patients decide whether to remain in the nonoperative therapy or to undertake arthroscopic surgery.

In the current study, the major postoperative complaint of the dissatisfied patient was pain, and the improvement of AOFAS score was mainly a result of the marked decrease in pain, which led to an increase in the patient's capacity to walk. The patient—who did not improve with time and finally showed clinical failure—had persistent pain, which may have limited the success of the procedure. We found that postoperative improvement of the pain subscore was critical for the long-term success of an arthroscopic marrow stimulation treatment and that the difference in the AOFAS scores was primarily attributable to a statistically significant difference in the pain subscores postoperatively.

We conclude that the preoperative measurement of initial defect size using MRI provides valuable prognostic information about clinical outcome of OLT. We suggest that preoperative MRI be used, not only for diagnostic methods, but also for prognostic purposes. The presence of such a risk factor may encourage surgeons to find new treatment strategies as well as counsel patients with larger lesions differently.

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