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Early clinical and structural results after autologous chondrocyte transplantation at the glenohumeral joint

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Background: The purpose of the study was to report early functional and radiographic results of a small series of patients who underwent autologous chondrocyte transplantation—collagen membrane seeding (ACT-Cs) for focal chondral defects of the shoulder.

Methods: The outcome of 4 consecutive male patients (mean age, 29.3 ± 6.2 years; range, 21-36 years) who underwent ACT-Cs for treatment of large symptomatic glenohumeral cartilage defects was retrospectively evaluated with clinical and radiographic measures at a mean of 41.3 ± 24.9 months (range, 11-71 months) after surgery. The evaluation included a visual analog scale for pain, the Constant score, the American Shoulder and Elbow Surgeons shoulder index, the Rowe score, and a satisfaction scale. Magnetic resonance imaging evaluation was performed according to the Magnetic Resonance Observation of Cartilage Repair Tissue scoring system.

Results: There were 3 humeral full-thickness cartilage defects (each 6.0 cm^2) and 1 glenoid full-thickness cartilage defect (2.0 cm^2). The mean postoperative visual analog scale score (0.3 of 10), the mean unweighted Constant score (83.3 ± 9.9), and the mean American Shoulder and Elbow Surgeons index (95.3 ± 8.1) were representative of satisfactory shoulder function. The Magnetic Resonance Observation of Cartilage Repair Tissue score was indicative of satisfactory defect coverage with signs of fibrocartilaginous repair tissue.

Conclusions: Autologous chondrocyte transplantation at the glenohumeral joint is a remote option for young adults with symptomatic, isolated, large-diameter cartilage lesions. Potential complications as a result of the open approach and 2-step procedure have to be considered carefully. Long-term data, larger patient populations, and randomized studies are required to determine the potential for chondrocyte transplantation techniques to be standard procedure for treatment of symptomatic, large-diameter, full-thickness cartilage defects in the glenohumeral joint.

Level of evidence: Level IV, Case Series, Treatment Study.

Keywords: Articular cartilage; collagen matrix; autologous chondrocyte transplantation; glenohumeral joint; cartilage defect

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Focal chondral defects of the shoulder are rarely reported in the young and active patient.[6] These defects may occur as the result of a number of mechanisms. Injury to the articular surfaces has been associated with trauma involving high-impact forces and repetitive shear and torsional loads.[10] Chondral defects are also thought to be the result of progressive cartilage damage stemming from repetitive mechanical irritation occurring within the glenohumeral joint. Several etiologic factors for these defects have been proposed, including loose joint bodies, microinstability, and instability of the long head of the biceps.[22] Iatrogenic chondral wear resulting from malpositioned suture anchors or screws has also been reported.

Management of symptomatic focal chondral lesions in the young patient can be challenging because the risk for osteoarthritis (OA) is high and data regarding treatment options are limited.[1,11,15,16,24,28] Conversely, treatment guidelines for the management of chondral defects around the knee have been well established.[18,29] Specifically, management of articular defects of the knee with standardized autologous chondrocyte transplantation has resulted in significant improvements in clinical and radiologic outcome measures.[26,30] Considering the positive results seen with autologous chondrocyte transplantation procedures in the treatment of articular pathology at the knee, autologous chondrocyte transplantation−collagen membrane seeding (ACT-Cs) may be a viable treatment option for patients with chondral defects of the shoulder.

The purpose of the study was to evaluate the early functional and radiographic results of a small series of patients undergoing ACT-Cs for focal chondral defects of the humeral head.[3] Our hypothesis was that treatment of large-diameter, symptomatic chondral defects of the humeral head with ACT-Cs results in a satisfactory clinical outcome and adequate defect coverage on radiographic analysis.

Materials and methods

Four consecutive male patients underwent ACT-Cs for treatment of large symptomatic glenohumeral cartilage defects. Each patient's case is described later.

Criteria for ACT-Cs

Indications for ACT-Cs included young and active patients (aged <40 years) with a symptomatic, large-sized, full-thickness cartilage lesion without relevant subchondral bone edema (groups 1 and 2 according to Niemeyer et al[20]). During diagnostic arthroscopy, the size and shape of the chondral defect were confirmed and comparisons to preoperative magnetic resonance (MR) film measurements were made. Before cartilage biopsy, indications for autologous chondrocyte transplantation were confirmed by palpating the borders of the defect to ensure that adequate stability was present.

Clinical outcome measures

Preoperative data were taken from the patient files (clinical history, visual analog scale [VAS]). Postoperative outcome was assessed by use of a VAS for pain (with 0 representing no pain and 10 representing maximal imaginable pain), the unweighted Constant score,[7] the American Shoulder and Elbow Surgeons (ASES) index,[23] the Rowe score,[25] and an overall 4-part satisfaction scale (1, very satisfied; 2, satisfied; 3, partially satisfied; and 4, not satisfied). For this retrospective study, all patients signed a consent form for enrollment in the study before follow-up testing (clinical examination and magnetic resonance imaging [MRI]).

Diagnostic imaging

Plain radiographs were obtained in 3 planes (true anteroposterior, Y-view, and axillary) preoperatively in all patients to exclude OA and additional osseous pathologies. MRI was performed preoperatively and postoperatively with a 1.5-T MR scanner (Siemens Avanto; Siemens Medical Solutions, Erlangen, Germany). Intra-articular gadolinium was used during the acquisition of the preoperative MRI scan to improve visualization and permit evaluation of defect size and location. A standard shoulder protocol consisting of a fat-suppressed PDw TSE (proton density weighted turbo spin echo) sequence in the transverse and coronal planes, a coronal T1-weighted spin echo (SE) sequence, and a sagittal T2-weighted TSE sequence was acquired in all patients.[6]

A single experienced radiologist (K.W.) specializing in musculoskeletal radiology and blinded to the clinical information evaluated all MRI scans. The progression of OA and soft-tissue changes were assessed and the Magnetic Resonance Observation of Cartilage Repair Tissue score was documented as previously described.[13,14] This score uses 9 different variables to describe morphology and signal intensity of the repair tissue compared with the adjacent native cartilage: degree of defect repair and filling, integration to border zone, surface, structure and signal intensity of the repair tissue, subchondral lamina and bone, adhesions, and effusion.

Patients

Patient 1 was a 31-year-old right-handed semiprofessional ice hockey player who underwent a body check during play. He immediately stopped playing because of pain and loss of motion in his left shoulder. Within a few days after the initial trauma, he presented to our clinic with complaints of persistent pain during sports activity (VAS score, 6-7 of 10), limited external rotation, and a locking phenomenon of his left shoulder. MR arthrography showed a grade 4 chondral lesion according to the Outerbridge classification scheme[21] on the anterosuperior portion of the humeral head with intact subchondral bone.

Patient 2 was a 21-year-old right-handed recreational handball player with a physically demanding overhead profession. Atraumatic right shoulder pain developed in this patient. He reported progressive crepitations and pain during overhead activities that started when he was aged 14 years. He presented to our clinic after an unsuccessful course of nonoperative treatment with complaints of right shoulder pain (VAS scores, 4 of 10 with low activities and 7 of 10 with overhead activities). Clinical examination showed full active and passive range of motion (ROM) with audible and

painful crepitations, general joint laxity without clinical signs of joint instability, and pain in the abduction and external rotation (ABER) position. MR arthrography showed a large grade IV chondral lesion according to the Outerbridge classification scheme[21] at the posterior portion of the articular glenoid.

Patient 3 was a 29-year-old right-handed recreational soccer player and whose job involved heavy manual labor with a history of recurrent dislocations of his left shoulder. After 5 dislocations, he underwent open shoulder stabilization with 2 titanium anchors at the age of 22 years. Postoperatively, he had increasing pain (VAS score, 6 of 10 during activities of daily life) and crepitations develop during work. Of note, he denied any episodes of instability in the postoperative period. Clinically, he presented with full active and passive ROM, posterior shoulder pain in the ABER position, and significant crepitations. There was no evidence of glenohumeral instability on physical examination. Plain radiographs showed a proud suture anchor position with no signs of early OA. On MR arthrography, the anchor position was confirmed and a large central humeral cartilage defect, grade IV according to Outerbridge classification scheme,[21] was identified.

Patient 4 was a 36-year-old right-handed recreational handball player. After a sudden rotational movement of the left shoulder, he began having persisting pain (VAS score, 7 of 10 during sports activity) and painful crepitations. He presented to our clinic with full active and passive ROM and pain in the ABER position. He did not show signs of glenohumeral instability on physical examination. MR arthrography showed a large bifocal lesion with a grade IV chondral defect of the central humerus according to the Outerbridge classification scheme[21] and a smaller chondral lesion of the anterior glenoid of grade III to IV with several loose bodies in the joint (Table I).

Surgical technique

All patients underwent ACT-Cs of the glenohumeral joint. The senior author (A.B.I.) performed each operation. The procedure was performed with the patient in the beach-chair position under general anesthesia. Before ACT-Cs, a diagnostic arthroscopy was performed to rule out concomitant intra-articular lesions. After palpation of the cartilage defect, debridement was performed and the defect was measured with a probe. In the first 2 patients, a cartilage biopsy specimen was taken from macroscopic healthy cartilage at the border of the defect. In the following 2 patients, biopsy was performed at the anterosuperior humeral head close to the cartilage-bone transition zone. The biopsy specimen was sent for cell proliferation (Metreon, Freiburg, Germany; Stryker, Duisburg, Germany) to facilitate the second stage of ACT-Cs. After 3 to 6 weeks of cellular proliferation, the second operation was performed through a standard deltopectoral approach. After detachment of the subscapularis tendon from the lesser tuberosity and incision of the capsule, the defect was visualized (Fig. 1, *A*). Debridement of the lesion was performed to its base with a ringed curette without opening the subchondral bone plate. The periphery of the defect was then abraded perpendicular to its base until stable 90° walls were achieved. Aluminum foil was used to trace the shape of the defect allowing type I/III collagen—based membrane (Chondro-Gide; Geistlich Biomaterials, Wolhusen, Switzerland) to be sized accordingly. The membrane underwent cell seeding intraoperatively with a minimum density of 10^6 cells/cm^2. After a 10-minute incubation period at room temperature, the chondrocytes adhered to the porous layer and the seeded membrane was transferred into the defect. The seeded porous layer was placed on the bone surface to facilitate cell adherence (Fig. 1, *B*). Circumferential No. 6-0 absorbable sutures placed adjacent to the articular cartilage and fibrin glue (Tissuecol; Baxter, Deerfield, IL, USA) were used to achieve fixation. Stability of the graft was confirmed intraoperatively during passive motion of the shoulder. The capsule was closed and the subscapularis tendon was fixed to the lesser tuberosity with 3 transosseous sutures (No. 5 Vicryl with atraumatic needle [Ethicon, Somerville, NJ, USA] for the first patient and No. 2 FiberWire sutures [Arthrex, Naples, FL, USA] for the other 3 patients).

From the first postoperative day, passive mobilization of the shoulder was performed by a physiotherapist. Flexion and abduction were limited to 90° for 6 weeks. Because of fixation of the subscapularis, external rotation and active ROM were restricted in a stepwise fashion for 6 weeks. A shoulder brace was used for 4 to 6 weeks postoperatively; the position of immobilization depended on the location of the defect. The patient was encouraged to perform a scheduled physiotherapy program focused on passive mobilization (physiotherapy for 3 days a week plus daily self-exercises). Active exercise was permitted at the seventh week, and return to sport was allowed after 6 months.

Results

Clinical outcome

Outcome data were collected at a mean of 41.3 ± 24.9 months (range, 11-71 months). One patient (patient 2) required a second procedure because of a fall with external rotation trauma 8 months postoperatively. A post-traumatic MRI scan was obtained, and a lesion of the reinserted subscapularis tendon was suspected. Diagnostic arthroscopy showed an intact subscapularis tendon with no signs of rupture or weakness under dynamic testing. The chondral defect of the glenoid was completely filled with stable but soft repair tissue. No other pathologies were seen during revision arthroscopy. At final follow-up, he was still satisfied with his shoulder function and he solely described a loss of strength in overhead activities (daily life, work).

Patients 1, 3, and 4 reported no pain at follow-up (VAS score, 0 of 10), whereas patient 2 reported slight pain during overhead work (VAS score, 1 of 10). Accordingly, patients 1, 3, and 4 were very satisfied with surgery, whereas patient 2 was satisfied. All patients had good to excellent scores for clinical outcome measures (Table II). A subgroup analysis of the Constant score indicated lower scores for strength and activity of daily life for patient 2. Across all patients, pain and ROM did not differ substantially. The mean Rowe score was 91.3 ± 7.3 points (range, 75-100 points). There was no limitation in passive and active ROM in comparison to the contralateral side.

Radiographic outcome

Preoperative radiography did not exhibit signs of OA in any patient. On the preoperative MRI scans, the 3 patients without previous surgery showed an intact rotator cuff

Table I Overview of defect properties, duration of symptoms, additional surgical interventions, and follow-up time points

Patient No.	Defect	Location defect	ICRS classification[5]	Size of defect (cm^2)	Preoperative duration of symptoms (mo)	Additional surgery	Time points of postoperative MRI (mo)	Time points of postoperative clinical follow—up (mo)
1	Unipolar	Humerus anterior-superior	4a	6.0	10	Loose body extraction	30, 71	30, 71
2	Unipolar	Glenoid posterior	4b	2.0	70		8, 12, 47	47
3	Unipolar	Humerus posterior-central	4a	6.0	72	Anchor extraction Tenodesis of long head of biceps	36	36
4	Bipolar	Humerus central Glenoid anterior	3b 3a	6.0 1.1	3	Loose body extraction Microfracture of anterior glenoid	3, 11	11

ICRS, International Cartilage Repair Society.

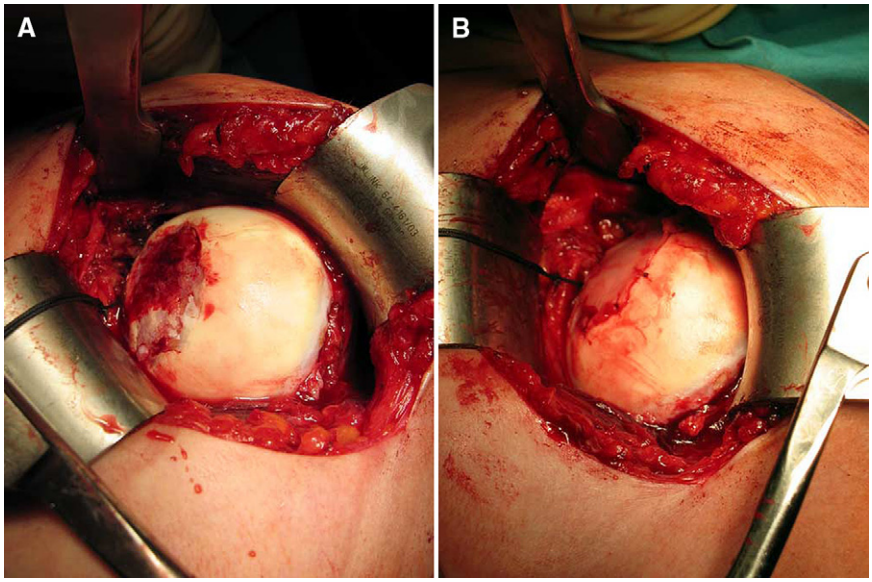

Figure 1 (**A**) Intraoperative view of chondral defect at anterosuperior aspect of left humeral head before debridement and graft implantation. (**B**) Intraoperative view after ACT-Cs for treatment of humeral cartilage defect in patient 1.

without pathologic changes. Patient 3 showed a thickened but intact subscapularis tendon after open shoulder stabilization. He had no atrophy or fatty infiltration.

General evaluation of postoperative MRI scans showed mild signs of OA in patient 3 (dislocated anchor). The other 3 patients exhibited no signs of OA. The rotator cuff was intact in all patients. Patient 2 showed continuity of the subscapularis; however, the superior half of the muscle was found to have grade II fatty infiltration according to Fuchs et al.[9]

Evaluation of the repaired tissue according to the Magnetic Resonance Observation of Cartilage Repair Tissue score showed a reduction in signal intensity and subchondral bone edema over time (Figs. 2 and 3). In all patients but patient 3 a complete integration to the border zone was documented. An adjacent cartilage defect to the region covered with ACT-Cs developed in patient 3 (Table III).

Discussion

Symptomatic large-diameter cartilage defects of the shoulder in young and active patients require careful

Table II Detailed functional outcome of all patients

	Age (y)	Follow-up (mo)	Constant score[8]					ASES index[30]
			Pain	ADL	ROM	Strength	Sum	
Patient 1	31	71	15	18	36	15	84	98.0
Patient 2	21	47	14	9	36	10	69	83.3
Patient 3	29	36	15	20	38	16	89	100.0
Patient 4	36	11	15	19	38	19	91	100.0
Mean	29.25	41.25	14.75	16.50	31.25	15.00	83.25	95.33

ADL, activities of daily living.

consideration of the limited available treatments. Whereas procedures for cartilage repair have been successfully introduced and established for chondral and osteochondral lesions in the knee, techniques for similar lesions in the shoulder are less developed.

In knee surgery, therapy options are directed by the type and size of the defect. Transplantation of autologous osteochondral cylinders is a widely used technique for smaller osteochondral defects, whereas microfracture is recommended for full-thickness cartilage defects with intact subchondral bone smaller than 2 to 3 cm^2. For larger lesions or locations in the knee with increased shear forces, the chondrocyte transplantation technique has been advocated.[19] Application of this algorithm to the pathologies in our presented cases would indicate the chondrocyte transplantation technique for the management of these focal chondral lesions.

Literature regarding autologous chondrocyte transplantation in the shoulder is limited to a single case report. Romeo et al[24] reported satisfactory results at a 1-year follow-up of a young athlete with a unipolar humeral full-thickness cartilage defect of the humerus who underwent autologous chondrocyte transplantation. Considering the lack of evidence for treatment in this population, the criteria used in knee surgery were adapted to direct the treatment of the patients in our case series. In addition, care was taken in ruling out the presence of subchondral bone edema preoperatively because of high failure rates reported with chondrocyte transplantation and edema.[20] During the index procedure, excessive debridement and bone marrow—stimulating techniques were avoided because inferior results for chondrocyte transplantation techniques combined with bone marrow-stimulating techniques have been reported.[17]

At present, the discussion about the effect of chondrocyte transplantation techniques compared with microfracture in the knee remains controversial.[31] However, current studies seem to favor our approach, considering the advantages of chondrocyte transplantation techniques.[27] Matrix-supported transplantation techniques are preferred over the previously described autologous chondrocyte transplantation with a periosteal flap in the current literature regarding cartilage repair.[2,4] There is no donor-site morbidity, and the rate of graft hypertrophy is theorized to be lower.[12] Furthermore, a stable collagen matrix is more feasible to be brought into the defect and does not require excessive suturing, making it a valuable option for treatment of glenoid defects where fixation is technically demanding. In this study, ACT-Cs was preferred because it provided collagen matrix support, and as a result of the exact sizing of the matrix before cell seeding, loss of cultured chondrocytes was avoided.[24,30]

Alternate procedures for addressing chondral defects in the shoulder range from simple debridement over arthroscopic interpositional graft implantation to replacement with a prosthesis.[1] In the young and active patient, joint-preserving interventions should be favored over prosthetic replacement. Single arthroscopic debridement of a symptomatic osteochondral lesion of the shoulder is a technically simple procedure, but clinical data are limited. Cameron et al[5] studied the outcome of this intervention in a middle-aged cohort (mean age, 49.5 years). After a minimum follow-up of 2 years, they found satisfactory results with significant pain relief in patients with lesions smaller than 2 cm^2.

Few studies have examined the effects of microfracture for full-thickness glenohumeral cartilage lesions. Millett et al[16] performed microfracture in 24 patients (mean age, 43 years) with a follow-up of 47 months with comparable cartilage defects. Microfracture was combined with arthroscopic stabilization in 6 patients. In 6 patients, an arthroscopic shoulder stabilization was added, and 10 patients also underwent subacromial decompression. At follow-up, the authors reported a VAS score of 1.6 of 10 for pain and an ASES score of 80.0 points.

Siebold et al[28] performed open microfracture with periosteal flap coverage. At 25.8 months postoperatively, the 5 patients (mean age, 32 years) had a mean Constant score of 81.8 points, with signs of OA progression in 2 of the 5 patients. The mean size of the chondral lesion measured 3.1 cm^2, significantly smaller than the defects in our study. Furthermore, longer follow-up (>2 years) was not reported. Autologous and allogeneic transplantations have been described for osteochondral defects or osteochondritis dissecans.[11] Kircher et al[11] presented long-term follow-up data (8.8 years) of 7 patients (mean age, 45.3 years) who underwent autologous osteochondral transplantation. A mean

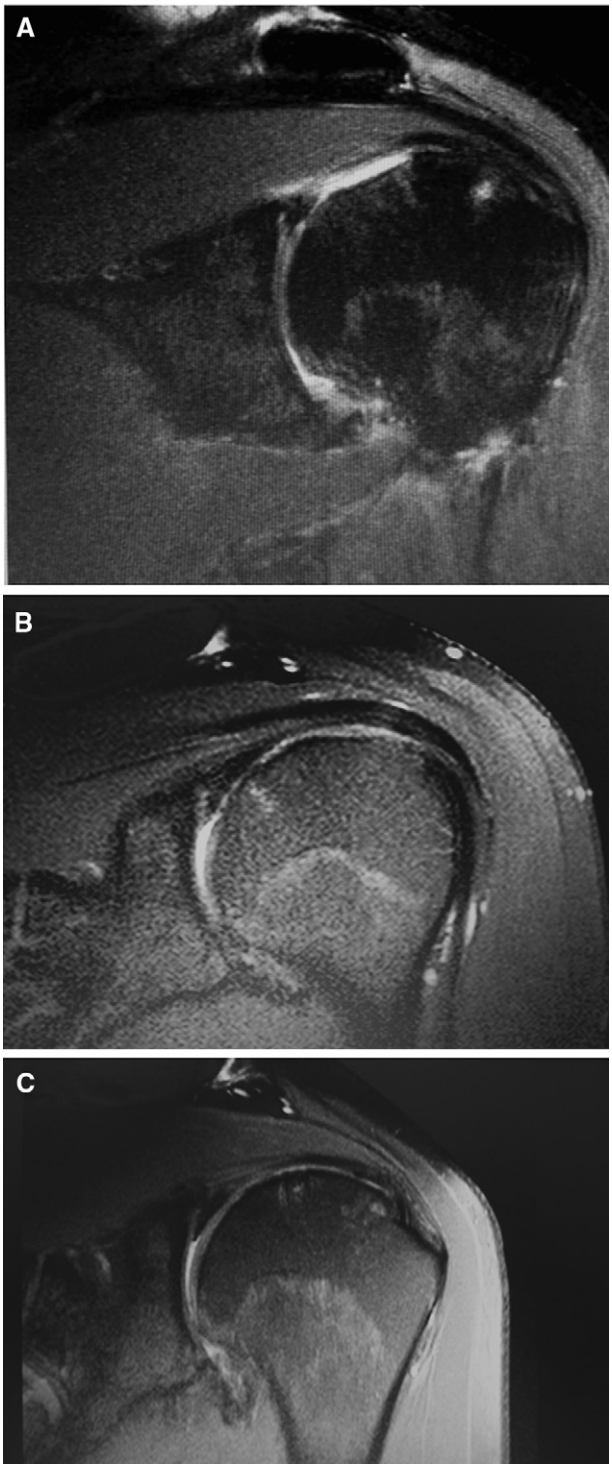

Figure 2 MRI assessment of patient 1 (fat-suppressed PDw TSE coronal sequences): preoperative humeral cartilage lesion (**A**) repair tissue 30 months postoperatively (**B**) and repair tissue with residual minimal subchondral edema 71 months postoperatively (**C**).

defect size of 1.4 cm^2 was covered with cylinders from the ipsilateral knee joint. The postoperative Constant score was 90.9 points; however, despite these satisfactory clinical results, signs of progressive OA were observed in 3 of 5 patients.

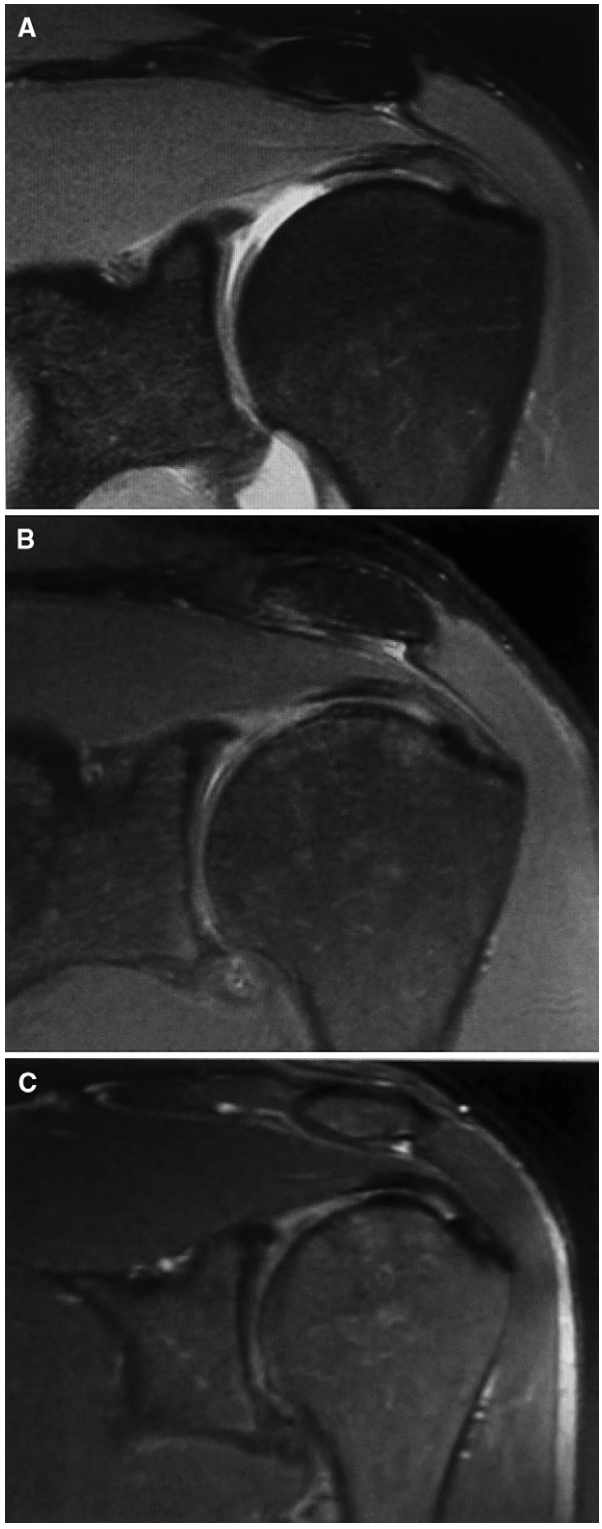

Figure 3 MRI assessment of patient 4 (fat-suppressed PDw TSE coronal sequences): preoperative humeral cartilage lesion (MR arthrography) (**A**) repair tissue with generalized hyperintensity 3 months postoperatively (**B**) and repair tissue with reduced hyperintensity 11 months postoperatively (**C**).

Table III Subgroup analysis of Magnetic Resonance Observation of Cartilage Repair Tissue score

	Patient 1	Patient 2	Patient 3	Patient 4
Follow-up (mo)	$30^1/71^2$	$12^1/47^2$	36	$3^1/11^2$
1. Degree of defect repair and filling of defect				
Complete	x	x	x	
Hypertrophy				
Incomplete				
>50% of adjacent cartilage				x
<50% of adjacent cartilage				
Subchondral bone exposed				
2. Integration to border zone				
Complete	x	x		x
Incomplete				
Demarcating border visible (split-like)				
Defect visible				
<50% of length of repair tissue				
>50% of length of repair tissue			x	
3. Surface of repair tissue				
Surface intact		x	x	
Surface damaged (fibrillations, fissures, ulcerations)				
<50% of repair tissue depth	x			x
>50% of repair tissue depth or total degeneration				
4. Structure of repair tissue				
Homogeneous	x		x	
Inhomogeneous or cleft formation		x		x
5. Signal intensity of repair tissue (dual T2 FSE)				
Isointense	x	x^2	x	
Moderately hyperintense		x^1		x^2
Markedly hyperintense				x^1
6. Subchondral lamina	Intact	Not intact	Intact	Intact
7. Subchondral bone	Not intact^1/ intact^2	Not intact	Intact	Not intact (edema)
8. Adhesions	No	No	No	No
9. Effusion	No	No	No	No
10. Note		Hypertrophic SSC tendon, moderate fatty degeneration	Adjacent cartilage defect, early OA	

FSE, Fast spin echo; *SSC*, subscapularis. Superscripted numbers 1 and 2 indicate first and second MRI, respectively.

Although these studies aid in understanding various treatments for chondral defects of the shoulder, the heterogeneous nature of the patients included and frequency of concomitant procedures do not permit direct comparison to our case series.[5,11,16,28] In this regard, the disadvantages of osteochondral transplantation require discussion. Transplantation may result in limited coverage because of the size of the cylinders. In addition, donor-site morbidity of the knee may be a concern. Siebold et al[28] reported on a combination of microfracture and periosteal flap for the treatment of focal full-thickness chondral defects. In a comparison of clinical outcomes, slightly superior results in larger chondral defects were reported in our case series. MRI was not performed, thereby limiting information regarding defect filling over time.[28]

In our study, 1 patient showed signs of progressive OA and a cartilage defect adjacent to the repair tissue. On the basis of our experience in the knee joint, the mechanical irritation (dislocated anchor) may have damaged more cartilage than macroscopically visible, leading to inadequate repair of the ''whole'' defect, and the previously impaired adjacent cartilage may have degenerated over time. This highlights the importance of high-resolution MR arthrography and careful intraoperative palpation to avoid underestimating the expansion of the defect. In our experience, this procedure should not be considered in patients with early-stage OA.

There was 1 complication because of partial subscapularis insufficiency after external rotation trauma sustained during a fall 8 months postoperatively. No direct connection of this complication to the cartilage repair itself

has been identified, and the risk of subscapularis insufficiency without trauma was believed to be low in a young patient. Careful detachment and stable reinsertion of the subscapularis should be performed until minimally invasive techniques for chondrocyte transplantation are available. In addition, active postoperative rehabilitation has to be reduced in the first few weeks to allow subscapularis tendon healing. Although there is no standardized rehabilitation protocol for cartilage repair of the shoulder, principles that guide the activity progression at the knee joint (reduced shear forces, high-frequency passive mobilization/continuous passive motion, partial/minimal weight bearing) should be adopted for the shoulder to assist in decision making postoperatively.[8] In addition, the costs of a 2-step procedure including the incubation of the chondrocytes might be taken into consideration and checked preoperatively with the patient's insurance company. Furthermore, the technique used is not available in the United States.

We believe that the most important finding of this study was that satisfactory clinical results were observed in patients who underwent ACT-Cs for focal chondral defects of the shoulder. In addition, there appears to be structural healing as evidenced by MRI of the repair tissue, as well as integration over time, which supports our hypothesis. To our knowledge, this is the first homogeneous case series of young, active patients with large-diameter, full-thickness cartilage defects that underwent a standardized ACT-Cs treatment with a clinical and radiographic evaluation in a standardized manner in the shoulder.

This case series has several important limitations. First, it includes only a small number of patients because of the rareness of this pathology in young patients. Therefore, blinding of the radiologist to the preoperative and postoperative films of the 4 patients was not practicable. Exclusion of patients with a concomitant pathology, such as shoulder instability, further reduces the number of individuals. The presented group is not completely homogeneous in matters of defect location and etiology; however, compared with existing literature, efforts were made to account for numerous bias factors (eg, age and additional procedures). Because the pathology is rare, larger and more homogeneous cohorts might be difficult to investigate. Multicenter studies should be considered for further investigation.

The second limitation is that this is a retrospective analysis without a standardized preoperative evaluation. Third, there is no control group, which makes it difficult to compare our results with those of other methods. Fourth, with regard to OA progression, the follow-up is far too short, and routine radiographic control for a standardized evaluation of OA progression was absent because of ethical considerations. In addition, for ethical reasons, MR arthrography could not have been performed postoperatively in asymptomatic patients because of the potential risk of infection. Despite these limitations, we believe this case series highlights the potential for biologic treatment of symptomatic, large-diameter cartilage defects in young, active patients.

Conclusion

This study reports satisfactory clinical outcomes for a small case series of patients 3.5 years after autologous chondrocyte transplantation for focal chondral defects of the shoulder. Potential complications as a result of the open approach and 2-step procedure have to be considered carefully. Long-term data, larger patient populations, and randomized studies are required to determine the potential for chondrocyte transplantation techniques to become standard procedure for treatment of symptomatic, large-diameter, full-thickness cartilage defects in the glenohumeral joint. We conclude that autologous chondrocyte transplantation at the glenohumeral joint is a remote option for young, active adults with symptomatic, isolated, large-diameter cartilage lesions.

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