Tissue on the Transferred Coracoid Graft After Latarjet Procedure

Histological and Morphological Findings

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Background: Anterior shoulder instability is a debilitating condition that can require stabilization via a Latarjet procedure.

Purpose: The aim of this study was to characterize the histological composition of the articular-sided surface of the coracoid bone graft after Latarjet procedure. Specific features of cells isolated from the coracoid and graft tissues were assessed.

Study Design: Case series; Level of evidence, 4.

Methods: Tissue samples were harvested from 9 consecutive patients undergoing arthroscopic debridement and screw removal after arthroscopic or open Latarjet procedure. Tissues were processed histologically. In 2 patients, the samples were analyzed to assess specific cellular properties.

Results: Safranin O staining indicated that glenoid tissues contained variable amounts of glycosaminoglycan (GAG) and round chondrocytic cells mainly organized in clusters. Graft tissues contained less GAG and were more cellular but were not organized in clusters and had variable morphological features. An association appeared to exist between the cartilage quality of glenoid tissues and that of the graft tissues. Cells isolated from glenoid and graft tissues exhibited similar proliferation capacity.

Conclusion: The results of our analysis show that cells located at the articular-sided surface of transferred coracoid grafts demonstrate fibrocartilaginous properties and may have the capacity for chondral proliferation. Further studies are needed to confirm this observation and future application.

Keywords: shoulder; instability; Latarjet; cartilage; osteoarthritis

Anterior shoulder instability and recurrent dislocations are debilitating conditions in the young patient population, often occurring during sporting activities. These conditions greatly affect the quality of life of such patients, which is well described in the literature.[17,22,27]

Humeral head translation and impingement against the anterior glenoid rim at the time of dislocation cause various lesions that further impair glenohumeral stability: These lesions include soft tissue injuries such as labral, ligamentous, and capsular tears.[8] Osteochondral defects involving the anterior glenoid (ie, bony Bankart) and the posterolateral humeral head (Hill-Sachs) may also occur. Bony Bankart defects reduce the static support of the humeral head. Hill-Sachs defects may engage with the anterior glenoid rim in abduction and external rotation and thus trigger glenohumeral dislocation. Repetitive dislocations may also cause isolated cartilage injuries[16] that can foster degenerative changes, referred to as *instability arthropathy*, in long-term follow-up.[13]

The surgical treatment of anterior shoulder instability varies between isolated reconstruction of the aforementioned soft tissue injuries, such as the Bankart repair, and bony procedures addressing the osteochondral defects.[7,28]

As reported by Ahmed et al,[1] the recurrence rates after isolated soft tissue reconstruction with an arthroscopic Bankart repair can be highly variable, between 4.2% and 19%. Recurrence rates were correlated with the extent of osteochondral defects at the humerus and the glenoid, which are not addressed by this technique. In 1954, Latarjet[19] described the coracoid bone transfer to the glenoid as

The American Journal of Sports Medicine
2019;47(3):704–712
DOI: 10.1177/0363546518819825

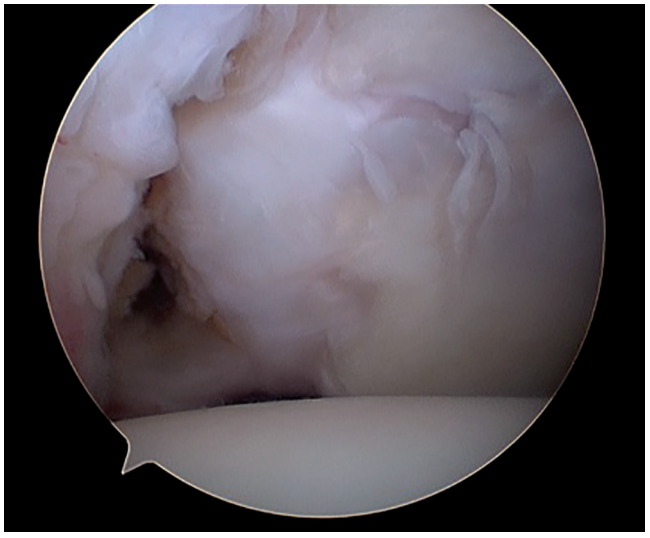

Figure 1. Arthroscopic image of a left shoulder from the anterolateral view. This intraoperative image of the coracoid graft shows macroscopic similarities to the glenoid cartilage tissue.

an alternative to isolated soft tissue procedures. Patte et al[23] later described the "triple effect," incorporating muscular, capsular, and bony factors to improve shoulder stability and reduce recurrent instability. Several open and arthroscopic techniques have since been described.[6,12,26] Walch et al[26] and Young et al[27] demonstrated that the Latarjet procedure is a safe and reliable technique with good to excellent clinical results, low recurrence rates, and high return to sport after the procedure.

Nonetheless, the Latarjet procedure only reconstructs the bone and does not address cartilage loss at the level of the glenoid. Allain et al[2] stated that a lateral positioning of the graft could cause glenohumeral osteoarthritis. Still, with appropriate graft position flush to the glenoid surface, the technique does not seem to harm the humeral cartilage, even though a direct contact between the bony surface graft and the humeral head must be postulated. However, the transferred coracoid graft is covered by periosteal tissue whose cells may have the potential to differentiate into fibrocartilage, thus protecting the humeral head surface from attrition. This speculation is based on one case report[25] in a patient after the Bristow procedure for recurrent shoulder instability. Therefore, the aim of the current study was to histologically characterize the composition of the articular-sided surface of the coracoid bone graft after an arthroscopic Latarjet procedure (Figure 1). Additionally, we assessed specific features of cells isolated from the coracoid and graft tissues.

METHODS

Sample Collection

This study was approved by the institutional review board (IRB Approval: PIC 51/2017_FJD, Fundacion Jimenez Diaz, Universidad Autonoma de Madrid, Spain), and informed consent was obtained from all patients included in the study. Data were collected prospectively and analyzed retrospectively in a multicenter study. The Latarjet procedure was performed with an all-arthroscopic technique, as previously described by Lafosse and Boyle.[17] During this procedure, the coracoid graft is placed intra-articularly, flush with the glenoid subchondral bone, without additional capsular and/or labrum repair. The number of arthroscopic Latarjet procedures performed in our center is up to 60 per year. Tissue samples (without bone tissue) were harvested from 9 consecutive patients undergoing arthroscopic debridement and screw removal after an arthroscopic Latarjet procedure. In all patients, the indication for screw removal was anterior and/or posterior shoulder discomfort or pain due to screw prominence.[18]

The mean age of the patient group was 32 years (range, 21-44 years). The mean time after the arthroscopic Latarjet procedure was 26 months (range, 5-61 months). The mean number of dislocations before Latarjet was 5.5. The descriptive data and instability scores before the Latarjet procedure are listed in Table 1. In all patients, a biopsy on the articular side from the coracoid graft between the screws and another biopsy from the glenoid cartilage were performed; tissue was obtained with an arthroscopic grasper (sample size of approximately 5 mm) after shaving and debridement. In 1 patient (patient 5), a control biopsy specimen was taken from the native coracoid to permit direct comparison with the coracoid graft sample. Tissues from patients 1 through 7 were processed histologically as described below.

In 2 patients (patient 8 and patient 9), the 2 samples (glenoid and graft) were not characterized histologically (as described for the tissues from patients 1 through 7)

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One or more of the authors has declared the following potential conflict of interest or source of funding: C.H. has received education payments from Arthrex, consulting fees from Stryker, and hospitality payments from Stryker and Depuy Mitek. L.L. is a consultant for and received royalties from Depuy Mitek. AOSSM checks author disclosures against the Open Payments Database (OPD). AOSSM has not conducted an independent investigation on the OPD and disclaims any liability or responsibility relating thereto.

TABLE 1
Descriptive Data for the 9 Patients and the Time Between Latarjet Procedure and Arthroscopic Screw Removal

	Age at Last Surgery (Arthroscopic Screw Removal), y	No. of Dislocations Before Latarjet	No. of Subluxations Before Latarjet	Main Sport Before the Beginning of Problems	Walch-Duplay Score Before Latarjet	Rowe Score Before Latarjet	Time Between Arthroscopic Latarjet and Screw Removal, mo
Patient 1	34	5	15	Swimming	35	30	59
Patient 2	44	5	0	—[a]	55	35	61
Patient 3	29	6	4	Boxing	60	35	15
Patient 4	21	3	0	Ski	40	70	10
Patient 5	30	6	10	Ski	55	50	29
Patient 6	28	1	30	Motor cross	40	50	27
Patient 7	30	5	0	Swimming	40	50	5
Patient 8	38	15	15	Tennis	-5	25	24
Patient 9	34	4	0	—	—		6
Mean	32						26

[a]Dashes indicate that data were not available.

but were analyzed to assess specific cellular properties as described below.

Histological and Immunohistochemical Analyses

Native tissues (glenoid, graft, and native coracoid) were decalcified for 12 hours with a 7% EDTA 30% sucrose solution (Sigma-Aldrich). Tissues were then fixed in 4% formalin for 24 hours at 4°C, embedded in paraffin, cross-sectioned (5 mm thick), and stained with Safranin O for sulphated glycosaminoglycans.[5] Safranin O sections of glenoid and graft tissues were used to grade cartilage quality. In brief, 4 central sections per tissue, covering a total area of 450 μm, were analyzed. Only one person (A.B.) evaluated the slides. Two criteria were considered for the scoring (ie, intensity of staining and cell morphological features), each with equal weight, with a possible minimum collective score of 0 and a maximum of 6. Criterion 1, intensity of cell staining, was scored as 0, no stain; 1, weak stain; 2, moderate, even stain; and 3, even, dark stain. Criterion 2, cell morphological features, was scored as 0, condensed, necrotic, pyknotic bodies; 1, spindle or fibrous; 2, mixed spindle-fibrous with rounded chondrogenic morphologic features; 3, all round, chondrogenic.

Sections from glenoid and graft tissues were processed for immunohistochemistry using an antibody against matrix metalloproteinase 13 (MMP-13; Abcam) or Aggrecan cryptical epitope-DIPEN (MD Bioscience) as previously described.[21]

Statistical Analyses

Statistical evaluation was performed by use of SPSS software version 7.5 (SPSS, Sigma Stat). Values are presented as mean ± SD. Differences between histological grading were assessed by 2-tailed Mann-Whitney test. Correlations between histological gradings (glenoid vs graft) and between histological gradings and any selected parameter were assessed by use of 2-tailed Pearson tests. *P* values less than .05 were considered to indicate statistically significant differences or correlations.

Cell Culture

Glenoid and graft samples were digested with 0.15% type I collagenase, and resulting cells were cultured for 2 passages in complete medium (Dulbecco's modified Eagle medium, 10 mM HEPES, 1 mM sodium pyruvate, 100 U/mL penicillin, 100 mg/mL streptomycin, 0.29 mg/mL glutamate, all from Invitrogen) supplemented with 10% fetal bovine serum, 5 ng/mL fibroblast growth factor 2, and 1 ng/mL transforming growth factor β1 (TGF-β1) as previously described.[4] Expanded cells were then cultured for 2 weeks under conditions promoting chondrogenic or osteogenic differentiation as previously described.[5] In brief, chondrogenic differentiation was induced by culturing cells in pellets through use of a chondrogenic medium (ChM+: Dulbecco's modified Eagle medium, 10 mM HEPES, 1 mM sodium pyruvate, 100 U/mL penicillin, 100 mg/mL streptomycin, 0.29 mg/mL glutamate, 1.25 mg/mL human serum albumin, insulin-transferrin-selenium [Invitrogen], 10^{-7} M dexamethasone, and 0.1 mM ascorbic acid 2 phosphate [Sigma-Aldrich] containing 10 ng/mL TGF-β1 [R&D Systems]). Osteogenic differentiation was induced in monolayer culture in the presence of osteogenic medium (OM+: alpha-minimum essential medium, 10 mM HEPES, 1 mM sodium pyruvate, 100 U/mL penicillin, 100 mg/mL streptomycin, 0.29 mg/mL glutamate, 10 mM β-glycerophosphate, 10 nM dexamethasone, and 0.1 mM L-ascorbic acid-2-phosphate). Control cultures were performed in the aforementioned media without the chondrogenic factor TGF-β1 (ChM–) or the osteogenic factors β-glycerophosphate and dexamethasone (OM–). The extent of chondrogenic and osteogenic differentiation was assessed histologically and by reverse transcription polymerase chain reaction (RT-PCR) as described below.

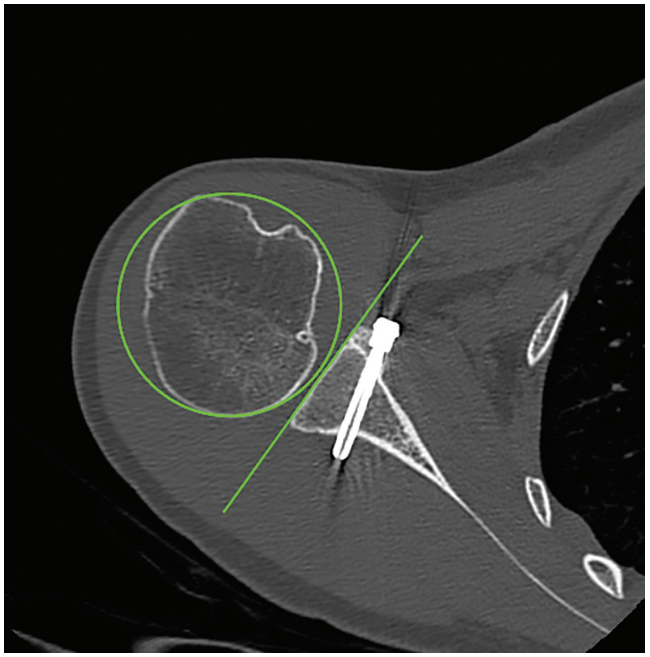

Figure 2. Computed tomography scan of a right shoulder in the axial plane. The axial graft position was analyzed via the method described and validated by Casabianca et al[11] after defining a line passing at the glenoid and a circle centered on the humeral head.

Analytical Methods

Proliferation Rate. Proliferation rate was calculated as the ratio of $\log_2(N_f/N_0)$ to T, where N_0 and N_f are the numbers of cells at the beginning and end of the expansion phase, respectively; $\log_2(N_f/N_0)$ is the number of cell doublings; and T is the time required for the expansion.

Histological Analyses. Pellets and native tissues were fixed in 4% formalin for 24 hours at 4°C, embedded in paraffin, cross-sectioned (5 mm thick), and stained with Safranin O.[5] Osteogenically cultured layers were fixed for 10 minutes in 4% formalin and stained with Alizarin Red 2%.[21]

RT-PCR Analyses. Total RNA was extracted from pellets and osteogenically cultured monolayers by use of TRIzol (Life Technologies), and cDNA was generated as previously described.[4] The PCR master mix was based on AmpliTaq Gold DNA polymerase (Perkin Elmer/Applied Biosystems). TaqMan Gene Expression or On-Demand assays (Life Technologies) were used on an ABI 7900 Fast Real-Time PCR System (Life Technologies) for 40 cycles to measure gene expression of sex-determining region Y-box 9 protein (Sox9) (Hs00165814_m1), type II collagen (Col II) (Hs00264051_m1), runt related transcription factor 2 (Runx2) (Hs00231692_m1), and osteocalcin (OC) (Hs01587814_g1) using glyceraldehyde-3-phosphate dehydrogenase (Hs99999905_m1) as the housekeeping gene.

Analysis of Coracoid Graft Position

During the arthroscopic procedure, the senior author (L.L.) subjectively labeled the macroscopic graft position as medial, flush, or lateral.

Preoperatively, computed tomography (CT) scanning was performed in all 9 patients to evaluate the graft position, any associated osteolysis, and screw position and prominence. A CT scan analysis with 3-dimensional reconstruction was also performed. The axial graft position was analyzed by a single observer (D.L.H.) using the method described and validated by Casabianca et al[11]: The graft position was measured in the axial view after the observer defined a line passing at the glenoid (anterior and posterior subchondral glenoid bone) and a circle centered at the humeral head (Figure 2). The graft position was then classified as described by Casabianca et al[11] into 1 of 5 categories: lateral, congruent, flush, medial, or too medial.

RESULTS

Histological Characterization of Native Tissues

Safranin O staining indicated that glenoid tissues contained variable amounts of glycosaminoglycan (GAG) and round chondrocytic cells mainly organized in clusters (Figure 3, A, C, E, G, I, K, and M). Graft tissues contained less GAG and were more cellular; cells in these tissues were not organized in clusters and exhibited a variable shape (ranging from round to elongated/fibroblastic) (Figure 3, B, D, F, H, J, L, and N). The histological score based on Safranin O staining confirmed the better quality of the cartilage in the glenoid compared with the graft tissues (Table 2). Interestingly, a significant correlation was noted between the cartilage quality of glenoid and that of the graft tissues (Figure 4). In contrast, no statistically significant correlations were observed between the cartilage quality of the tissues and the time after the primary Latarjet procedure ($r = 0.333$ and $P = .233$ for glenoid; $r = -0.022$ and $P = .481$ for graft) or the donor age ($r = 0.041$ and $P = .465$ for glenoid; $r = -0.308$ and $P = .251$ for graft). Coracoid tissue from patient 5 contained a loose extracellular matrix and a few fibroblastic-like cells localized mainly at the edges (Figure 3O). MMP-13 and DIPEN were expressed in scattered cells in glenoid but not graft tissues (Figure 3, P-S).

Proliferation and Differentiation Capacity of Cells Isolated From Glenoid and Graft Tissues

Cells isolated from glenoid and graft tissues from patient 8 and patient 9 exhibited similar proliferation capacities (Figure 5, A and B). After the expansion phase, all cells cultured in 3-dimensional pellets were capable of forming compact spheroidal tissues. GAG deposition, however, occurred only in pellets cultured from cells of patient 8 in ChM+ and was more pronounced in the tissues formed

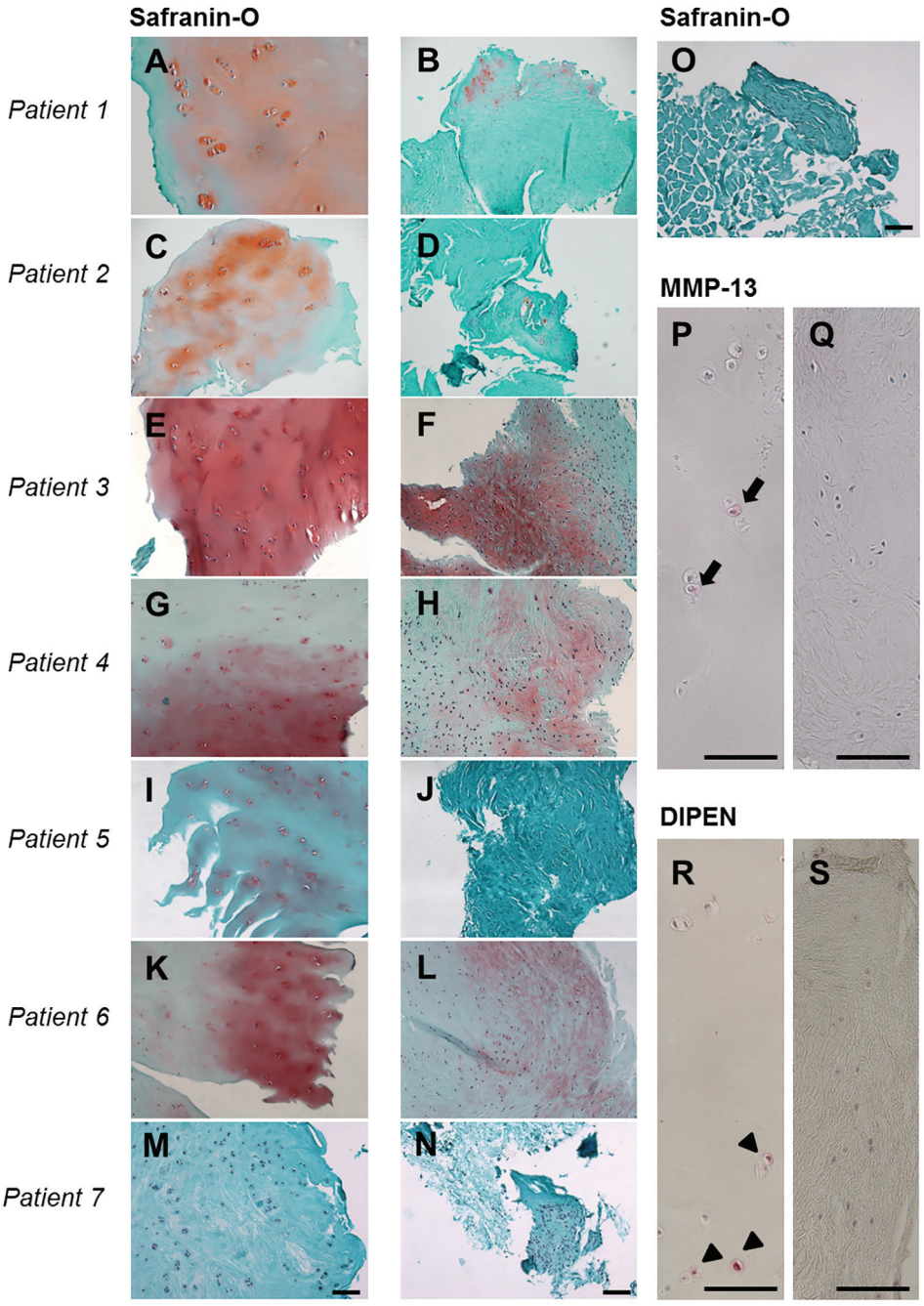

Figure 3. Histological and immunohistochemical characterization of native tissues. Safranin O staining of (A, C, E, G, I, K, and M) glenoid tissues and (B, D, F, H, J, L, and N) graft tissues from patients 1 through 7. (O) Safranin O staining of the coracoid from patient 5. Bars = 100 μm. (P, Q) Matrix metalloproteinase 13 (MMP-13) and (P, R) DIPEN staining of glenoid and (Q, S) graft tissues from patient 2. Scale bars = 100 μm. Arrows and triangles indicate MMP-13-positive and DIPEN-positive cells, respectively.

by glenoid cells. In particular, as shown in Figure 5C for patient 8, pellets generated with glenoid cells were uniformly and intensely stained with Safranin O and contained mainly round-shaped cells, whereas pellets generated with graft cells contained only scattered areas positive for Safranin O and fibroblastic cells (*II* vs *IV* in Figure 5C). Glenoid and graft cells from patient 9, in contrast, formed tissues with no cartilaginous appearance (Figure 5D) even when cultured in ChM+. RT-PCR results generally confirmed the histological data. Glenoid cells and, to a lesser extent, graft cells from patient 8 upregulated Sox9 (2.2- and 1.5-fold, respectively) and Col II (11.4- and 1.8-fold, respectively) in ChM+ (Figure 5E). In contrast, these chondrogenic genes remained expressed

TABLE 2
Histological Grading[a]

	Glenoid			Graft		
	Intensity of Staining (0-3)	Cell Morphology (0-3)	Total Score (0-6)	Intensity of Staining (0-3)	Cell Morphology (0-3)	Total Score (0-6)
Patient 1	2.4	2.7	5.0	0.8	2.1	2.9
Patient 2	2.1	2.8	4.9	0.6	1.2	1.8
Patient 3	3.0	2.8	5.8	2.3	2.1	4.4
Patient 4	2.6	2.0	4.6	1.8	1.6	3.2
Patient 5	1.5	2.3	3.8	0	1.0	1.0
Patient 6	2.3	2.5	5.8	1.5	2.0	3.5
Patient 7	0.5	2.0	2.5	0	0.5	0.5
Mean ± SD	2.1 ± 0.8[b]	2.4 ± 0.4[b]	4.6 ± 1.2[b]	1.0 ± 0.8	1.5 ± 0.6	2.5 ± 0.8

[a]See the Methods for a description of histological grading.
[b]$P < .05$ from the corresponding score in graft tissues.

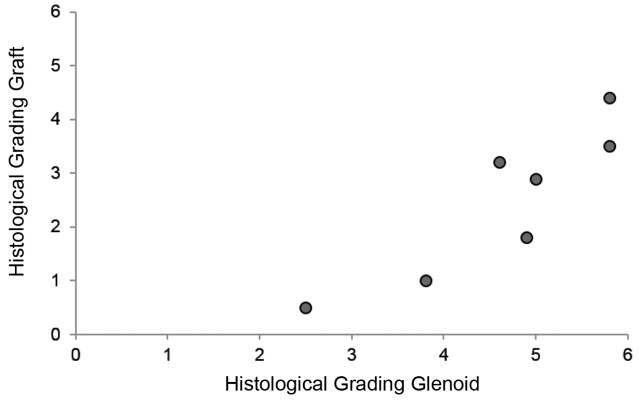

Figure 4. Correlation between the histological gradings of glenoid and graft tissues ($r = 0.892$, $P = .003$).

at baseline levels for both glenoid and graft cells from patient 9 after culturing in ChM+ (Figure 5F).

Glenoid and graft cells from patient 8 and patient 9 deposited similarly low amounts of calcified matrix in OM– as demonstrated by a weak staining for Alizarin Red. Calcium deposition was not enhanced in OM+. Similarly, the expression of the osteogenic genes Runx2 and OC also was not enhanced in the presence of osteogenic factors (Figure 5, I and J).

Radiological Assessment

The results of the CT assessment are shown in Table 3. In none of the 9 patients was non-union or massive osteolysis observed.

Intraoperative Assessment

Intraoperatively, the position of the graft was as follows: in 6 patients the graft position was considered flush, in 2 patients lateral, and in 1 patient medial.

DISCUSSION

This study provides, for the first time, a histological analysis of the coracoid graft and glenoid after a Latarjet procedure as well as a characterization of the differentiation capacity of cells isolated from these 2 tissues. In addition, these data were analyzed with intraoperative macroscopic and radiological graft position.

A case report[25] published in the Japanese literature showed interesting histological results after a Bristow procedure. Three years and 3 months after the primary procedure, a diagnostic arthroscopy that was performed because of pain syndrome showed an intra-articular position of the screw. The articular side of the coracoid graft was analyzed with a biopsy, and the histological analysis showed fibrocartilage of the tissue. However, in that study, no histological characterization of the glenoid side was performed.

We demonstrated here that glenoid and graft tissues had fibrocartilaginous appearances even if the qualities of the cartilage varied drastically among the different patients considered. We expected to see some evidence of tissue remodeling, especially in the graft tissues, and checked for the presence of the cartilage-degrading enzyme MMP-13 and its products (and DIPEN). Quite surprisingly, these markers were absent or weakly expressed in the analyzed tissues. Future characterization of tissues at an earlier time after surgery and assessment of a larger panel of degrading enzymes and their products will be required to better clarify tissue remodeling.

The histological score based on the Safranin O staining confirmed the better quality of the cartilage at the level of glenoid compared with the graft tissue (Table 2). Interestingly, a significant correlation appeared to exist among the cartilage quality of glenoid tissues and that of the graft tissues.

In patient 8, who was 1 of the 2 patients whose samples were analyzed to assess specific cellular properties, cells isolated from glenoid tissue exhibited good postexpansion chondrogenic capacity. A pronounced donor-dependent, TGF-β-mediated, cartilage-forming capacity was previously observed in chondrocytes from knee and talar

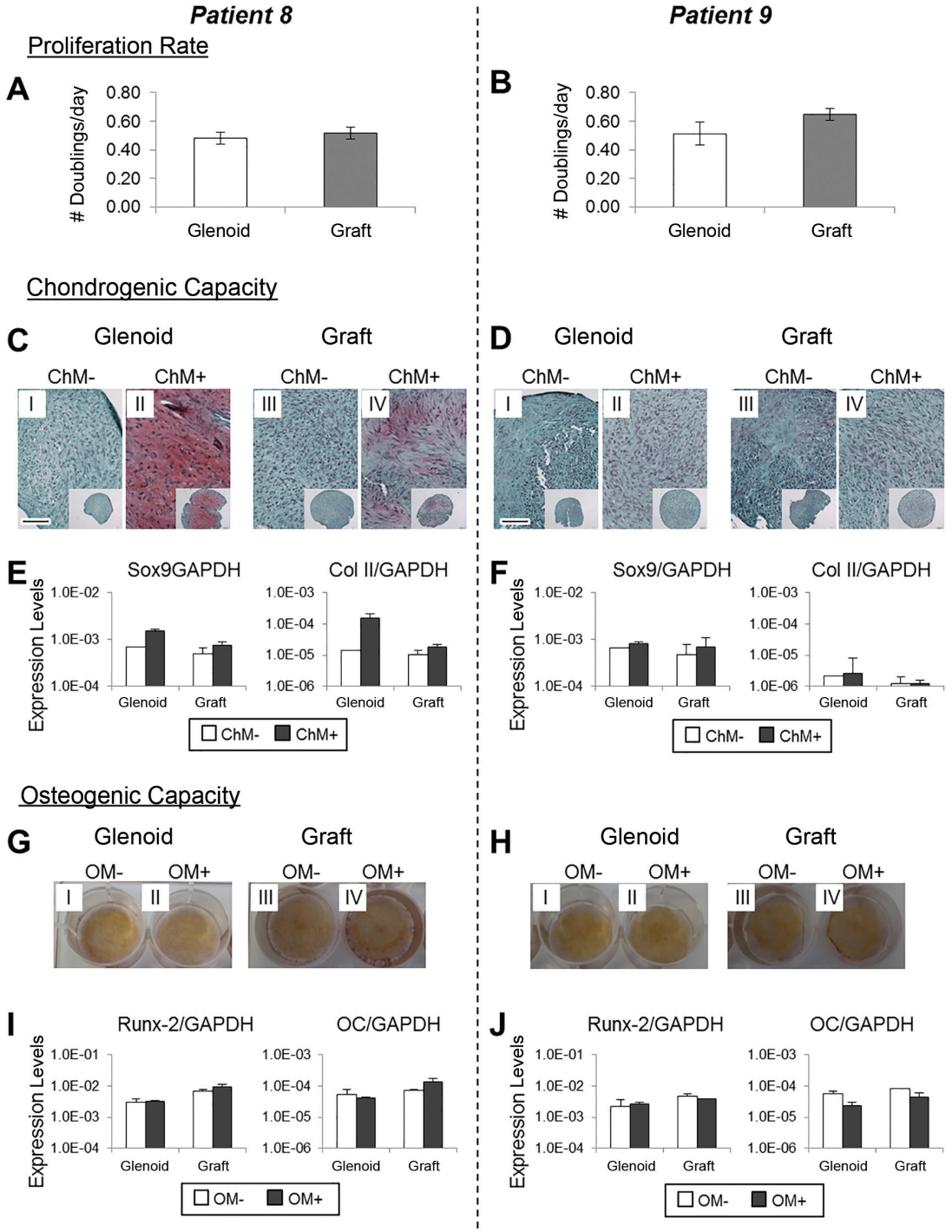

Figure 5. Expansion and differentiation capacity of cells isolated from glenoid and graft tissues. Proliferation rate of cells isolated from glenoid and graft tissues from (A) patient 8 and (B) patient 9. Data are mean ± SD of triplicate culture. (C, D) Safranin O staining of pellets generated by postexpanded glenoid cells (*I* and *II*) or graft cells (*III* and *IV*) in chondrogenic medium containing transforming growth factor β1 (TGF-β1) (ChM+) (*II* and *IV*) and chondrogenic medium not containing TGF-β1 (ChM–) (*I* and *III*). Scale bars = 100 μm. The inserts are low-magnification images of the entire pellets. (E, F) Real-time reverse transcription polymerase chain reaction (RT-PCR) analyses of the pellets carried out by use of specific primers for Sox9 or type II collagen (Col II). Values are mean ± SD of duplicate pellets. (G, H) Alizarin Red staining of layers generated by postexpanded glenoid cells (*I* and *II*) or graft cells (*III* and *IV*) in osteogenic medium containing β-glycerophosphate and dexamethasone (OM+) (*II* and *IV*) or osteogenic medium not containing β-glycerophosphate and dexamethasone (OM–) (*I* and *III*). (I, J) Real-time RT-PCR analyses of the pellets carried out by use of specific primers for Runx-2 or osteocalcin (OC). Values are mean ± SD of duplicate layers.

TABLE 3
Data Regarding Graft Position on Axial Computed Tomography and Intraoperative Graft Position

Patient No.	Graft Position on Axial Plane[a]	Graft Position Intraoperatively[b]
1	1	1
2	1	1
3	3	2
4	3	2
5	3	2
6	4	2
7	5	3
8	3	2
9	3	2

[a]Scored as 1, lateral; 2, congruent; 3, flush; 4, medial; 5, too medial.
[b]Scored as 1, lateral; 2, flush; 3, medial.

cartilage tissues[4,9] (but not in chondrocytes from septal cartilage tissue[10,20]) and thus might be a characteristic of cells from articular cartilage. Even when reduced, cells from the graft of patient 8 exhibited a certain chondrogenic differentiation capacity. The origin of the chondrogenic cells in the graft remains unknown. They might be derived from the periosteal tissue covering the coracoid graft, or they might have originated from a population of cells migrating from the glenoid.

The low or absent propensity of the postexpanded graft and glenoid cells to osteodifferentiate may be explained by the absence of osteoblastic cells in the analyzed native tissues. Characterization of the properties of cells from larger amounts of glenoid and graft tissues is required to substantiate the aforementioned findings.

In patients 8 and 9, the graft position was flush. Patient 8 was potentially able to form cartilage. We were not able to correlate graft position with chondrogenic capacity since the graft position was the same in both patients. It is interesting to observe that 24 months after the primary procedure, the chondrogenic capacity was intact.

Recently, Auffarth et al[3] reported a morphological and histological analysis after glenoid reconstruction of the glenoid with iliac bone graft. Those investigators performed tissue biopsies in 2 patients, and the examination revealed hyaline-like cartilage covering the articular side of the graft. In a recent study by Hintermann et al,[14] osteochondral lesions of the talus were treated with a free vascularized bone graft in 14 patients who had ankle surgery. Malleolar screw removal was then performed with concomitant arthroscopic debridement and tissue biopsy in 3 patients. The histological analysis was consistent with fibrocartilage. Hintermann et al[14] concluded that a vascularized autograft has the potential to restore the articular surface and provide a stable foundation resulting in fibrocartilage without collapse or resorption of the graft, which may occur if an avascular graft is used. Shimogaki et al[24] performed a histological analysis of the articular cartilage in rabbits after rotational acetabular osteotomy for hip dysplasia. The investigators showed that in rabbits undergoing osteotomies, primary cloning of chondrocytes and metaplasia of the cartilage in the fibrous tissue were seen. In an experimental study on rabbits, Hiranuma et al[15] showed similar results with metaplastic changes to fibrocartilaginous tissue after Chiari pelvic osteotomy.

Our results showed that some patients retain the capacity to produce cartilage tissue but do not always do so. The reason for this finding remains unclear, although it could be due to methodological limitations. The sample size is a limiting factor. Although the biopsy was performed intraoperatively at the same level in all patients, we cannot exclude the possibility of a sampling error. Furthermore, the time between the 2 procedures varied and could have affected our results. The population characteristics showed a broad age spectrum, but no association was found between histological tissue quality and age. Another limitation is that the differentiation capacity was assessed in only 2 patients. The collected biopsy samples were small (<5 mm in width for the majority of patients). We could not use the same samples for the histological characterization and assessment of cellular properties, because the latter assessment requires a certain amount of native tissues to obtain a sufficient number of cells. We thus had to perform the experiments related to the cell properties only in separated tissue samples (ie, tissues from patient 8 and patient 9).

Analysis of the CT scan could not confirm a correlation between the graft position and the quality of the coracoid graft tissue, as hypothesized before the study. Further limitations of our study are related to the low number of patient tissues available for histological analysis, which precluded finding a statistically significant pattern. Furthermore, the study population varied with respect to the average time after Latarjet procedure and screw removal with concomitant biopsy.

In our study, the relation between CT scan and intraoperative graft position can be considered good (Table 3). To our knowledge, this has been analyzed for the first time in the literature.

The natural history for anterior shoulder instability shows variable rates of arthropathy. Gordins et al[13] found a 34% rate of moderate to severe arthropathy at 33 to 35 years after Bristow-Latarjet procedure, reporting that younger age at surgery and primary dislocation were protective against arthropathy. Gordins et al[13] stated that moderate to severe arthropathy increased with 1 percentage point per year after the primary dislocation. It has been speculated that the lateral graft position could be related to earlier glenohumeral osteoarthritis.[2] Intraoperative assessment of the graft position is reliable. Hypothetically, before the coracoid graft fixation is performed during the Latarjet procedure, a chondrogenic matrix could be added on the articular side of the graft, which could stimulate the cells and form repair tissue. As a next step to evaluate this hypothesis, further analysis that assesses chondrogenic matrix stimulation should be conducted via in vitro studies.

CONCLUSION

The results of our analysis show that cells located at the articular-sided surface of transferred coracoid grafts

show fibrocartilaginous properties and may have the capacity for chondral proliferation. Further studies are needed to confirm this observation and inform future application.

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