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Prevalence of cartilage lesions and early osteoarthritis in patients with patellar dislocation

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Abstract

Objectives Assessment of cartilage lesions and osteoarthritis (OA) of the patellofemoral joint in patients following lateral patellar dislocation using magnetic resonance imaging (MRI).

Methods MR images of 129 knees (mean age 26 years, range 11–56) grouped as acute (A), recurrent (B), and chronic (C) dislocators were analysed regarding the prevalence and severity of patellofemoral cartilage lesions. Grades of OA were assessed using modified WORMS.

Results In groups A, B, and C the prevalence of cartilage lesions was 71%, 82%, and 97%, respectively. Most lesions were located on the central patella in groups A and B

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D. Khandker Department of Radiology, Apollo Hospitals Dhaka, Bushundhara R/A, Dhaka-1229, Bangladesh (central 69% and 78%; medial 56% and 47%; lateral 31% and 42%), whereas group C revealed all regions affected (73%, 61%, and 67%). Of group A, 14% had mild OA and 64% of group B. Group C showed mild OA in 62% and moderate OA in 18%. Cartilage defect size and prevalence of OA was correlated with number of dislocations (r=0.41 and r=0.59; P<0.001).

Conclusions Cartilage lesions and early OA are common after patellar dislocation and appear to increase with the frequency of dislocation. Both conditions should be considered when interpreting MRI in such patients, because of implications for treatment.

Key Points

- Cartilage lesions are very common after patellar dislocation.
- The severity of cartilage lesions increases with number of dislocations.
- Osteoarthritis is common after recurrent patellar dislocation, even in young patients.
- Detecting cartilage lesions is important after patellar dislocation.

Keywords Patellar dislocation \cdot Cartilage \cdot Osteoarthritis \cdot Knee \cdot MRI

Introduction

Lateral patellar dislocation (LPD) primarily occurs in young and active persons [8, 33]. Dislocation of the patella damages surrounding soft tissue structures and leads to chondral and osteochondral defects. They result from sliding of the posterior patellar surface over the lateral trochlear and subsequent contusion against the lateral femoral condyle [5, 7]. Damage to articular surfaces can result in severe chronic pain, limited mobility and reduced quality of life, especially with chronic patellofemoral instability [14].

Progression of joint destruction can be prevented or slowed by adequate treatment. Magnetic resonance imaging (MRI) has shown to be a reliable diagnostic tool for quantitative assessment of cartilage damage [4]. Dedicated MR analysis of cartilage damage, therefore, can provide crucial information for selecting the best treatment. Therapeutic options include conservative measures and different arthroscopic and surgical interventions [1].

The prevalence of cartilage damage following LPD reported in the literature varies widely and appears to depend on how many episodes of dislocation a patient has experienced and the diagnostic procedure used. Studies using MRI and arthroscopy identified cartilage defects after dislocation in 40-96% of cases [7, 9, 24, 29, 37]. Cartilage defects appear to become more severe over time [23], and an association of OA with LPD has been suspected [16] (Fig. 2).

While osteochondral fracture of the inferomedial patella has long been known to be a typical concomitant injury of acute LPD, little attention has been paid to cartilage lesions of the remaining patellar surface and of the trochlea, the effects of recurrent dislocation on the patellofemoral articular surface and the occurrence of osteoarthritis (OA).

The aim of this study was to assess the prevalence, severity and localisation of cartilage damage and osteoarthritis in patients with acute, recurrent, and chronic LPD, correlating the occurrence of patellofemoral joint defects with the frequency of dislocation episodes.

Materials and methods

Study population

After approval by the institutional review board, we searched the clinical database for patients with patellar dislocation who underwent an MRI knee-examination at one of three sites of a university medical centre from July 2000 through June 2011. Patients included had a convincing history of patellar dislocation and/or typical signs at MRI (e.g. oedema of the inferomedial patella and lateral condyle, tear of the medial patellofemoral ligament). Patients who had a prior knee intervention with metal implantation close to the patellofemoral joint were excluded. Mean interval between the last episode of patellar dislocation and MRI was 21 days (range, 1–93 days).

A total of 129 knees (72 left, 57 right) of 125 patients (68 women, 59 men) were included (Table 1). The mean age was 26 years (range, 11–56 years). The number of patellar dislocations for each patient was determined from the

medical records or retrospective personal questioning. Based on the number of dislocations, patients were assigned to the following groups: first-time dislocators (group A) with one dislocation; recurrent dislocators with two to nine dislocations (group B); and chronic dislocators with ten or more dislocations or chronically subluxated patella (group C).

Information on prior surgery for patellofemoral instability was available for 33 subjects of groups B and C. Nineteen patients (57.6%) had repair of the medial patellofemoral ligament, ten patients (30.3%) had tibial tuberosity advancement and five patients had lateral release (15.2%). None of the patients had prior trocheloplasty or derotational osteotomy.

MRI

MRI was performed on one of six MRI systems, using either dedicated knee coil or an extremity coil with clinical routine standard protocols. MRI systems, sequence specifications and coil combinations are described in Table 2. Since some sequence specifications were adjusted over the 11 years of the study period, the last-used parameters are described. All patients were imaged in the supine position with the leg in full extension.

MRI evaluation

MR images were evaluated separately on a PACS workstation (RadiForce R22; Eizo Nanao Corporation, Hakui, Japan). Images were assessed for cartilage defects and OA by two radiologists in consensus (reader 1 had 8 years' experience in musculoskeletal imaging and reader 2 had 3 years' experience). Retrospective reading was done in chronological order of MRI examinations. The readers were blinded to number of LPD and other patient data.

MRI evaluation of cartilage defects

Cartilage defects were evaluated in three anatomic subregions of the patellofemoral joint as previously published [37]: the central dome, medial and lateral facets of patella; the central, medial and lateral aspects of trochlea. As in other studies [27, 34, 37], the Outerbridge classification [25] was used to assess the depth of cartilage defects; briefly: grade 0—normal cartilage; grade I—smooth cartilage surface but focal signal heterogeneity; grade II—partial-thickness defect not deeper than 50%; grade III—defect of>50% of cartilage thickness; grade IV—full-thickness defect and osteochondral defect (Fig. 1). In all subregions, the lesion with the highest grade was documented.

Morphologically, cartilage defects were categorised using arthroscopic terminology [22, 23]: category I—fissure; II fibrillation; III—erosion. This classification was supplemented by three types of cartilage defects typically found

| | | Total | First-time dislocators (group A) | Recurrent dislocators (group B) | Chronic dislocators (group C) |
|------------|--------------|--------------|----------------------------------|------------------------------------|----------------------------------|
| Number (%) | | 129 (100%) | 51 (39.5%) | 44 (34.1%) | 34 (26.4%) |
| Sex | Men (%) | 59 (45.7%) | 34 (66.7%) | 15 (34.1%) | 10 (29.4%) |
| | Women (%) | 70 (54.3%) | 17 (33.3%) | 29 (65.9%) | 24 (70.6%) |
| Age | Mean (range) | 25.9 (11–56) | 22.4 (13–48) | 26.1 (11–56) | 31.1 (12–50) |

 Table 1
 Patient characteristics. Descriptive data on case numbers, sex and age distribution in the three groups based on the number of prior patellar dislocations

after LPD: IV—lamination; V—dislocated cartilage fragment; VI—osteochondral defect. Additionally, the largest extent of the cartilage defect was measured on axial and sagittal images and the area was calculated (mm²).

Inter- and intraobserver reliability with regard to Outerbridge classification of cartilage defects was evaluated by analysis of 25 randomly selected subjects. Weighted Cohen's Kappa statistics showed good intraobserver (κ =0.89) and interobserver agreement (κ =0.84).

MRI evaluation of osteoarthritis

The severity of OA of the patellofemoral joint was evaluated using a modified WORMS classification [26]. Briefly, WORMS is a semi-quantitative scoring method for evaluation of the knee in osteoarthritis based on MRI findings using 14 different features, including meniscal, ligamentous, cartilage and bony lesions. These features are graded by severity and summed to a total score. Here we used a reduced version of WORMS since only the patellofemoral joint was investigated. The patella and trochlea were evaluated for articular cartilage integrity, subarticular bone marrow abnormality, subarticular cysts, subarticular bone attrition and marginal osteophytes (Fig. 2). In the full version, a total score of 332 can be assigned versus 88 in our version. Scores were summarised in three grades: grade 1 (mild)—scores 1–29; grade 2 (moderate)—scores 30–58; grade 3 (severe)—scores 59–88. The intraclass correlation coefficient (ICC) showed an excellent intra- and interobserver reliability using the modified WORMS classification (intraobserver reliability 0.98); interobserver reliability 0.98).

Statistical analysis

Descriptive statistics of cohort and prevalence of cartilage defects and OA were calculated. The chi-squared test was

Table 2 MRI systems and specifications. Technical data of MRI systems with details on field strength, coils, and sequences

| Туре | Avanto | Symphony Vision | SignaExcite | SignaExcite | Intera | Panorama |
|--------------------------------------|--|---|---|---|--|---|
| Manufacturer | Siemens Medical Syst | tems | GE Healthcare | | Philips Medical Sys | tems |
| | Erlangen, Germany | | Milwaukee, WI, US | А | Best, The Netherlan | ds |
| Fieldstrength | 1.5 T | 1.5 T | 1.5 T | 3.0 T | 1.5 T | 1.0 T |
| Coil | 8 Ch. Knee-Coil | CP extremity coil | QuadKnee Coil | QuadKnee Coil | ST Knee Coil or Sy | n.Flex-M Knee-Coil |
| Sequences Axial orientation | T2w GRE (TR/TE 1,290/27 ms; FA 30°), FOV 180 mm, matrix 256 × 167, ST 3 mm, SS 3.3 mm | T2w SE (TR/TE 3,310/32 ms; FA 150°), FOV 180 mm, matrix 384 × 384, ST 3 mm, SS 3.6 mm | PDw SE (TR/TE 2,800/40 ms, FA 90°), FOV 160 mm, matrix 320 × 224, ST 4 mm, SS 5 mm | T2w SE (TR/TE 2,400/30 ms, FA 90°), FOV 180 mm, matrix 320 × 224, ST 4 mm, SS 5 mm | PDw SE (TR/TE 2,170/35 ms, FA 90°), FOV 180 mm, matrix 256 × 205, ST 4 mm, SS 4 5 mm | T2w SE (TR/TE 3,000/30 ms; FA 90°), FOV 180 mm, matrix 320 × 252, ST 3 mm, SS 4 mm |
| Sequences Sagittal orientation | PDw+T2w SE (TR/ TE 3,630/100 ms; FA 150°), FOV 160 mm, matrix 256 × 256, ST 4 mm, SS 4.8 mm | PDw+T2w SE (TR/ TE 3,580/30 ms; FA 150°), FOV 160 mm, matrix 256 × 256, ST 3 mm, SS 3.6 mm | PDw SE (TR/TE 2,820/40 ms, FA 90°),FOV 160 mm, matrix 320 × 224, ST 3 mm, SS 4 mm | T2w SE (TR/TE 4,000/95 ms, FA 90°), FOV 160 mm, matrix 384 × 224, ST 3 mm, SS 4 mm | T1w SE (TR/TE 580/15 ms, FA 90°), FOV 160 mm, matrix 256 × 205, ST 3 mm, SS 3.5 mm | T2w SE (TR/TE 3,000/30 ms; FA 90°), FOV 160 mm, matrix 320 × 252, ST 3 mm, SS 4 mm |

*T1w*T1-weighted, *T2w*T2-weighted, *PDw*proton density-weighted, *GRE* gradient echo sequence, *SE* spin echo sequence, *TR* repetition time, *TE* echo time, *FA* flip angle, *FOV* field of view, *ST* slice thickness, *SS* slice spacing

Fig. 1 Classification of cartilage lesions. Patients after lateral patellar dislocation; MRI: axial, T2-weighted, fat saturated TSE sequences (a, b, d) and axial, PD-weighted, fat saturated FSE sequence (c). a Grade I: intact cartilage surface, altered signal intensity of cartilage layer in patellar dome and lateral facet. b Grade II: local cartilage lesion. < 50% of cartilage thickness, in patellar dome. c Grade III: erosion of cartilage surface, > 50% of cartilage thickness, in patellar dome. d Grade IV: full surface defect of patellar cartilage in central dome



used to compare prevalence between groups and anatomical regions. Linear regression analysis using ANOVA was done to assess associations between number of LPD and cartilage defect areas, as well as WORMS. Adjustments for age and sex were made by multivariate analysis. Statistical analysis, calculations and drawings of boxplot diagrams were done with SPSS version 15.0 (SPSS, Chicago, IL).

Results

Cartilage defects

Cartilage defects of different grades and morphology were identified in 79.8% of cases (103/129 cases) (Table 3). The central patellar dome was the most common site of cartilage defects, accounting for 75% of instances (P=0.026). Of patients with a central dome defect, 51.9% had an additional medial defect (40/77) and 48.1% an additional lateral defect (37/77). An isolated defect of the lateral or medial compartment in an otherwise intact patella was present in 10.6% (11/103) and 4.9% (5/103) of cases, respectively. A total of 21.4% (22/103) had defects of all patellar subregions.

Trochlear cartilage defects were less common than patellar defects (32.0% trochlea vs 80.2% patella, P < 0.001) and were most common in lateral cartilage, which were seen in one third

of the cases (P < 0.001). Only 4.8% of patients had trochlear cartilage defects without concomitant patellar lesion.

In group A, approximately two-thirds of patients had cartilage defects. Cartilage defects of the central dome were primarily grades II and III (Table 4). These defects were primarily due to erosion (31.6%) or fibrillation (26.3%). Defects of the medial facet were exclusively due to osteochondral defects. The most common trochlear lesions were Outerbridge II defects of the lateral aspect (37.5% of cases).

In group B, 79.5% of patients had cartilage defects, most commonly involving central and medial patellar regions; however, one-third of patients had concomitant defects of the lateral facet (Table 4). Most central dome lesions were Outerbridge II lesions, predominantly with fibrillation and erosion (52.9% and 35.3%, respectively). In the medial region were Outerbridge IV lesions typically due to osteo-chondral defects (85.7%), along with Outerbridge II and III lesions. The lateral trochlea predominantly showed erosive Outerbridge II and III lesions.

Patients of group C had highest prevalence of cartilage defects (93.3%, P=0.009). As in the other two groups, cartilage defects were mostly found in the central dome, slightly less commonly in the medial and lateral facets (Table 4). Erosive cartilage lesions were most common in all patellar subregions with partially severe lesions in medial facet and otherwise predominantly moderate lesions

Fig. 2 Signs of osteoarthritis after LPD. Patients after recurrent (a, b) and chronic (c, d) lateral patellar dislocation; MRI: axial, T2-weighted, fat saturated TSE sequences (a, b, d) and PD-weighted, fat saturated FSE sequence (c). a Erosive changes of patellar and trochlear cartilage, cyst-like lesion in patella (arrow). b Erosive changes in patellar and trochlear cartilage, focal bone marrow lesion in patella (arrow). c Erosive changes in patellar and trochlear cartilage, small osteophyte at lateral trochlea (arrow). d Nearly complete erosive destruction of patellar and trochlear cartilage, bone erosion, bone marrow lesion and osteophytes



(Outerbridge II or III). Erosive lesions were also common in all three trochlear subregions compared with groups A and B (Tables 3, 4 and 5).

The total area of cartilage defects was largest in group C (Fig. 3). Regression analysis of the total defect area and the number of LPDs yielded a regression coefficient B of

27.889 (standard error, 6.022; r=0.413; P<0.001). This corresponds to a 27.9 mm² increase in the total defect area per dislocation. With age and sex as cofactors, we calculated regression coefficients of B=20.184 (standard error, 5.804; r=0.552; P<0.001) and B=30.645 (standard error, 6.638; r=0.423; P<0.001), respectively.

Table 3 Prevalence and distribution of cartilage defects. Distribution of cartilage lesions in the six anatomic subregions of the patella and trochlea in the total study population and in the three groups

distinguished according to number of patellar dislocations (group A1 dislocation, group B2–9 dislocations, group $C \ge 10$ dislocations)

| Total (n=129) | | | First-time dislocators (group A) $(n=51)$ | Recurrent dislocators (group B) $(n=44)$ | Chronic dislocators (group C) ($n=34$) | P value |
|------------------|---------|-------------|---|--|--|---------|
| Cartilage defect | | 105 (81.4%) | 36 (70.6%) | 36 (81.8%) | 33 (97.1%) | 0.009 |
| Patella | medial | 57 (54.3%) | 20 (55.6%) | 17 (47.2%) | 20 (60.6%) | 0.134 |
| | central | 77 (73.3%) | 25 (69.4%) | 28 (77.8%) | 24 (72.7%) | 0.112 |
| | lateral | 48 (45.7%) | 11 (30.6%) | 15 (41.7%) | 22 (66.7%) | < 0.001 |
| P value | | 0.026 | 0.067 | 0.086 | 0.834 | |
| Trochlea | medial | 6 (5.7%) | 0 (0.0%) | 0 (0.0%) | 6 (18.2%) | < 0.001 |
| | central | 15 (14.3%) | 1 (2.8%) | 1 (2.8%) | 13 (39.4%) | < 0.001 |
| | lateral | 36 (34.3%) | 7 (19.4%) | 10 (27.8%) | 19 (57.6%) | < 0.001 |
| P value | | < 0.001 | 0.005 | < 0.001 | 0.035 | |

| population and in the | three grou | ps distingui | ished accord | ing to numl | ber of patel | llar disloca | tions (grou | <i>p</i> A1 disloc | ation, grou | p B2-9 dis | locations, | group C ≥ | 10 disloca | ations) | | fame m |
|-----------------------|------------|--------------|--------------|-------------|-----------------|--------------|-------------|--------------------|-------------|---------------|------------|-----------|------------|-------------|-----------|----------|
| | Total | | | | First-time | ; dislocato | rs group B) | | Recurrent | t dislocators | (group B) | - | Chronic d | lislocators | (group C) | |
| | (n=105) | | | | (<i>n</i> =36) | | | | (n=36) | | | | (n=33) | | | |
| Grade | I | II | III | IV | I | Π | III | IV | I | II | III | IV | I | II | III | N |
| Medial patella (%) | 6 (5.7) | 11 (10.5) | 12 (11.4) | 28 (26.7) | 2 (5.6) | 1 (2.8) | 1 (2.8) | 16 (44.4) | 0 | 5 (13.9) | 5 (13.9) | 7 (19.4) | 4 (12.1) | 5 (15.2) | 6 (18.2) | 5 (15.2) |
| Central patella (%) | 5 (4.8) | 31 (29.5) | 31 (29.5) | 10 (9.5) | 2 (5.6) | 8 (22.2) | 11 (30.6) | 4 (11.1) | 2 (5.6) | 17 (47.2) | 6 (16.7) | 3 (8.3) | 1 (3.0) | 6 (18.2) | 14 (42.4) | 3 (9.1) |
| Lateral patella (%) | 16 (15.2) | 14 (13.3) | 14 (13.3) | 4 (3.8) | 7 (19.4) | 3 (8.3) | 1 (2.8) | 0 | 6 (16.7) | 4 (11.1) | 4 (11.1) | 1 (2.8) | 3 (9.1) | 7 (21.2) | 9 (27.3) | 3 (9.1) |
| Medial trochlea (%) | 1(0.9) | 2 (1.9) | 3 (2.9) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 (3.0) | 2 (6.1) | 3 (9.1) | 0 |
| Central trochlea (%) | 1 (0.9) | 7 (0.7) | 6 (5.7) | 1 (0.9) | 0 | 0 | 0 | 1 (2.8) | 0 | 1 (2.8) | 0 | 0 | 1 (3.0) | 6 (18.2) | 6 (18.2) | 0 |
| Lateral trochlea (%) | 4 (3.8) | 15 (14.3) | 11 (10.5) | 6 (5.7) | 2 (5.6) | 3 (8.3) | 0 | 2 (5.6) | 1 (2.8) | 4 (11.1) | 3(8.3) | 2 (5.6) | 1 (3.0) | 8 (24.2) | 8 (24.2) | 2 (6.1) |
| | | | | | | | | | | | | | | | | |

Osteoarthritis

Overall, we identified patellofemoral OA of variable severity in about 50% of the study population (Table 6). Nearly all patients with degenerative damage had low-grade disease; however, four patients in group C (3.1% of total) had moderate OA. High-grade OA (WORMS>58) of patellofemoral joint was not detected in this study population consisting of predominantly young patients.

In relation to the number of dislocations, the proportion of OA was highest in group C (76.7%, P<0.001). In group B, two-thirds of patients had low-grade OA, in group A OA rarely was detected (Fig. 4).

Regression analysis of WORMS and number of dislocations yielded B=1.556 (standard error 0.208; r=0.590; P< 0.001). With each dislocation in the previous medical history, there was an increase in the WORMS of 1.556 points. The results were similar when sex was taken into account (B=1.578; r=0.590; P<0.001). With age as a cofactor, the regression coefficient was slightly smaller (B=1.251; r=0.695; P<0.001), identifying age as an additional influence. It is noteworthy that we observed a few cases of OA in very young patients. A 16-year-old youth in group A had first signs of early OA (WORMS of 7). A 26-year-old patient with chronic LPD had marked signs of OA (WORMS of 44)

Discussion

The aim of this study was to determine the prevalence and severity of cartilage defects and osteoarthritis of the patellofemoral joint on MRI in relation to the number of LPDs. Nearly all patients had cartilage lesions and more than half of the patients had at least mild or moderate OA, which is a significant finding regarding the low mean age of the study population. Cartilage lesions in first-time and recurrent dislocators were primarily accounted for erosions and fissures of the central patellar dome and osteochondral defects of the medial facet. Chronic dislocators additionally had severe defects of the lateral patella facet and of trochlear cartilage. Overall, the prevalence of cartilage defects and OA was highest in chronic patellar dislocators. The extent of cartilage defects and severity of OA correlated moderately with the number of prior LPDs.

A normal population without a history of LPD has a markedly lower prevalence of cartilage damage or OA, even at a higher age [18]. The mechanism of LPD, with slipping out of the trochlear groove, sliding of the patellar cartilage surface over the lateral trochlear edge, and contusion of the medial patella at the lateral femoral condyle is associated with abnormal loading of the articular surface of the patella. Although we did not obtain longitudinal data in our study, we may safely assume that both first-time LPD and, above

| | Total | | | | | | First-time d | lislocators (gro | up A) | | | |
|---|------------------|------------------|------------------|---------------|--------------|-----------------|-----------------|------------------|----------------|---------|--------------|-----------------|
| | (<i>n</i> =105) | ; | ; | ; | ; | : | (<i>n</i> =36) | ; | ; | ; | ; | ; |
| Morphology ^a Medial patella (%) | I 4 (3.8) | II 1 (0.9) | III 18 (17.1) | IV 2 (1.9) | V 4 (3.8) | VI 23 (21.9) | I 1 (2.8) | II 1 (2.8) | 111 1 (2.8) | 2 0 | V 2 (5.6) | VI 14 (38.9) |
| Central patella (%) | 9 (8.6) | 19 (18.1) | 32 (30.5) | 4 (3.8) | 7 (6.7) | 3 (2.9) | 5 (13.9) | 5 (13.9) | 6 (16.7) | 1 (2.8) | 6 (16.7) | 1 (2.8) |
| Lateral patella (%) | 5 (4.7) | 3 (2.9) | 20 (19.0) | 4 (3.8) | 2 (1.9) | 0 | 1 (2.8) | 1 (2.8) | 2 (5.6) | 1 (2.8) | 0 | 0 |
| Medial trochlea (%) | 0 | 0 | 5 (4.7) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Central trochlea (%) | 3 (2.9) | 1(0.9) | 9(8.6) | 0 | 1(0.9) | 0 | 0 | 0 | 0 | 0 | 1 (2.8) | 0 |
| Lateral trochlea (%) | 1(0.9) | 1 (0.9) | 23 (21.9) | 0 | 2 (1.9) | 3 (2.9) | 0 | 0 | 2 (5.6) | 0 | 1 (2.8) | 1 (2.8) |
| | | | | | | | | | | | | |
| | Recurrent | dislocators (gro | up B) | | | | Chronic d | islocators (grou | up C) | | | |
| | (n=36) | | | | | | (n=33) | | | | | |
| Morphology ^a | Ι | П | III | IV | > | Ν | I | II | III | IV | Λ | ΙΛ |
| Medial patella (%) | 2 (5.6) | 0 | 7 (19.4) | 1 (2.8) | 1 (2.8) | 6 (16.7) | 1 (3.0) | 0 | 10 (30.3) | 1 (3.0) | 1 (3.0) | 3 (0.9) |
| Central patella (%) | 3 (8.3) | 10 (27.8) | 9 (25.0) | 2 (5.6) | 1 (2.8) | 2 (5.6) | 1 (3.0) | 4 (12.1) | 17 (51.5) | 1 (3.0) | 0 | 0 |
| Lateral patella (%) | 3 (8.3) | 0 | 4 (11.1) | 2 (5.6) | 1 (2.8) | 0 | 1(3.0) | 2 (6.1) | 14 (42.4) | 1 (3.0) | 1 (3.0) | 0 |
| Medial trochlea (%) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 5 (15.2) | 0 | 0 | 0 |
| Central trochlea (%) | 1 (2.8) | 0 | 0 | 0 | 0 | 0 | 2 (6.1) | 1 (3.0) | 9 (27.3) | 0 | 0 | 0 |
| Lateral trochlea (%) | 0 | 0 | 6 (16.7) | 0 | 0 | 2 (5.6) | 1(3.0) | 1 (3.0) | 15 (45.5) | 0 | 1(3.0) | 0 |

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Fig. 3 Cartilage defect sizes following lateral patellar dislocation. Boxplots showing the size of cartilage defects (mm²) of the patellofemoral joint in first-time (n=51), recurrent (n=44), and chronic dislocators (n=34)

all, recurrent dislocation are considerable risk factors for cartilage defects of patellofemoral joint. Chondral and osteochondral lesions have been reported as complications of LPD in arthroscopic and radiological studies [2, 3, 7, 9, 13, 17, 21, 22, 24, 29-32, 35-37]. Earlier investigators identified the inferomedial patella as the main site of articular defects resulting from patellar impaction on the condyle [7, 13, 24]. During dislocation, the cartilage of the central dome of the patella is exposed to strong shear forces and pressure. This might explain the high prevalence of central and medial patellar cartilage defects [11, 22, 24]. Lateral patellar damage primarily is observed in chronic luxation and is attributable to persistence of the patella in lateral subluxation. With the patella in this position, its lateral portion glides down the lateral trochlear, resulting in abrasion of patellar cartilage. Damage to the trochlear groove in acute dislocation appears to be less severe, probably because the groove is protected by the concavity of its surface.



Fig. 4 Patellofemoral osteoarthritis following lateral patellar dislocation. Boxplots showing the WORMS scores and grades of patellofemoral osteoarthritis in first-time (n=51), recurrent (n=44), and chronic dislocators (n=34). The maximum osteoarthritis score in the modified WORMS system is 88 points

Differences in anatomy may influence the severity of cartilage damage. Most important factors are trochlear dysplasia, patellar height, and possibly also lateralisation of the force vector caused by an abnormal trochlear-groove-to-tibial-tuberosity distance. Additionally, several forms of soft tissue hyperlaxity (e.g. Ehlers-Danlos syndrome, Marfan syndrome) can predispose to LPD. These conditions may be associated with lower mechanical loading of articular cartilage. Hence, not all dislocators will develop higher-grade cartilage damage. Future studies should investigate whether there is an association between the severity of anatomic risk factors and the severity of chondral lesions and OA.

Reports of prevalence of cartilage defects after LPD vary widely. In an arthroscopy-based study, Nomura and Inoue [22] detected medial patellar defects in two-thirds of the cases and lesions of the central dome in half of the cases. Slightly lower prevalence was found in the arthroscopy-

 Table 6
 Prevalence and grades of osteoarthritis. Occurrence and severity of osteoarthritis according to the modified WORMS system.

 Distribution in the total study population and in the three groups

according to dislocations (group A1 dislocation, group B2–9 dislocations, group $C \ge 10$ dislocations)

| | | Total $(n=129)$ | First-time dislocators $(group A)$ $(n=51)$ | Recurrent dislocators $(group B)$ $(n=44)$ | Chronic dislocators (group C) $(n=34)$ | P values |
|--------------------|--------------|-----------------|---|--|--|----------|
| | | (# 12)) | (group 11) (n - 51) | (group b) (n H) | (group c) (n - 51) | |
| Osteoarthritis (%) | | 62 (48.1%) | 7 (13.7%) | 28 (63.6%) | 27 (76.7%) | < 0.001 |
| WORMS | mean | 5.3 | 0.3 | 3.56 | 15.06 | < 0.001 |
| | range | 0-46 | 0-4 | 0-14 | 0-46 | |
| OA grade | mild (%) | 56 (43.4%) | 7 (13.7%) | 28 (63.6%) | 21 (61.8%) | 0.002 |
| | moderate (%) | 6 | 0 | 0 | 6 (17.6%) | < 0.001 |
| | severe | 0 | 0 | 0 | 0 | |
| | | < 0.001 | < 0.001 | < 0.001 | < 0.001 | |

based study of Virolainen et al. [36]. The arthroscopic findings in both studies were obtained at time of arthroplasty or resection of the osteochondral fragment [24], indicating mainly patients with severe trauma and probably severe damage were included. Our population may include more patients with less severe trauma, resulting in lower rates of severe osteochondral lesions.

Several longitudinal studies have shown that cartilage defects progress with further dislocations [22, 23]. The high prevalence of cartilage lesions of 96% that Nomura and Inoue [23] found in an arthroscopic study of chronic LPD correlates well with the rate in our study. Moreover, the prevalence of lesions of the central patellar dome (77%) is slightly higher than the prevalence reported here (66%), confirming the frequent occurrence of damage at this site, in particular in chronic dislocators. In a follow-up investigation, Nomuraand Inoue [23] found a marked increase in cartilage defects. To our knowledge, this is the first MRI study analysing the prevalence of OA in patients with LPD. Our results show that there is a correlation between OA and the number of LPD and that even adolescents may develop mild or moderate OA. In contrast, in young, healthy adults OA appears unlikely [19]. Although, the significance of small cartilage defects with respect to clinical symptoms and progression of OA is still unclear, there is strong evidence that abnormal loading of cartilage can lead to chondropathy and degenerative disease over time [10, 12] Ding et al. [6] showed that the prevalence and severity of knee cartilage defects are significantly associated with tibiofemoral osteophytes, knee cartilage volume, suggesting an important role of cartilage defects in early OA. An experimental model in rabbits confirms an association between induced cartilage defects and development of OA [15]. The high prevalence of OA in the group of chronic dislocators might thus be attributable to cartilage lesions occurring during LPD.

Demonstration of cartilage damage after LPD is pivotal for therapeutic decision-making. The choice between conservative and surgical management crucially depends on the morphology, size, and depth of cartilage defects. However, a consistent international therapeutic approach is still lacking [20]. Smaller and more superficial cartilage defects are treated conservatively. Extensive and deeper defects can be managed by a variety of arthroscopic and open surgical options [28]. Therefore, precise documentation of cartilage condition in the radiologist's report is central for treatment planning.

Our study has some limitations. We analysed MRI findings without arthroscopic correlation, primarily because only patients with osteochondral or significant chondral defects underwent arthroscopy and also because, due to the retrospective study setting, there was no consistent documentation of cartilage damage by surgeons at the three participating hospitals. However, published studies have shown excellent agreement of MRI grading of cartilage damage with arthroscopy as the "gold standard", while suggesting that MRI may have limitations in identifying low-grade cartilage damage [37]. Moreover, our study patients were examined on different MR systems using routine clinical pulse sequences. Since the isotropic threedimensional pulse sequence was not acquired in all patients, only the standard pulse sequences were included in the analysis in order to use identical images in all cases. This might have resulted in a lower sensitivity for the detection of low-grade cartilage lesions. Moreover, the analysis did not include prior therapeutic procedures in recurrent and chronic dislocators, which might also have influenced our results. Moreover, the grouping of the patients was done in such a way to separate patients with recurrent dislocations (less than ten) from those with a chronically luxated patella. It must be noted that there is no clinical consensus regarding the number of previous episodes that defines a recurrent or chronic dislocator.

In conclusion, cartilage lesions are very common after LPD, and their severity correlates with the number of previous dislocations. Moreover, many patients with chronic patellar subluxation have degenerative changes of the patellofemoral joint, which is a significant outcome considering the young age of these individuals. We conclude that it is important to put emphasis on cartilage lesions in MRI reports and allow for a more dedicated management strategy to prevent subsequent damage to the joint.

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