

ORIGINAL ARTICLE

Patellar chondropathy prevalence at anterior cruciate ligament reconstruction: Analysis of 250 cases

A. Oksman^{a,*}, V. Dmytruk^b, J. Proust^b, C. Mabit^b, J.-L. Charissoux^b, J.-P. Arnaud^b

^a Sarrus Teinturiers Private Hospital, 49, allée Charles-de-Fitte, 31300 Toulouse, France ^b Dupuytren University Hospital Center, Limoges, France

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KEYWORDS Summary Introduction. – Anterior knee instability caused by anterior cruciate ligament (ACL) deficiency Chondral damage: results in meniscal as well as chondral femorotibial and/or femoropatellar damages over a more Patella; or less long duration delay. This study's objectives were, in chronically deficient ACL patients, Laxity; to assess onset delay for developping chondral patella lesions and also analyse these lesions Anterior cruciate characteristics in relation to laxity duration. ligament; Hypothesis. - Chondral patellar lesions in ACL deficient knees get worse with time. ACL deficient knee Material and methods. - We reviewed 250 charts of patients who had undergone arthroscopically assisted surgery for knee anterior laxity. The arthroscopic procedures were conducted between January 1995 and January 2005. Chondral damages were evaluated at surgery according both to International Cartilage Repair Society (ICRS) and Bauer and Jackson classifications. The data were analyzed using the Kruskal-Wallis test and the Fisher exact test. Results. - Of the 250 analysed charts, 72 patients (28.8%) were found to present chondral patella lesions. The majority of these lesions were superficial and involved the lateral facet area. We observed a statistically significant ICRS worsening grade in relation to laxity duration. Discussion. – Few publications in the literature report patellar involvement in anterior laxity of the knee. However, our results are comparable to those of the rare series found. The pathomechanics of these lesions has not yet been precisely identified and requires further biomechanical studies.

* Corresponding author.

E-mail address: aoks@voila.fr (A. Oksman).

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Conclusion. – Patellar damage is frequent with anterior laxity (28.8% in our series) and duration is correlated with statistically significant aggravation of these lesions. Currently, the assessment of these patellar lesions is considered less important than meniscal and femorotibial lesions, even though the natural history of ACL disruption seems to be evolving toward degeneration of all the compartments of the knee, including the femoropatellar compartment. Level of evidence: level IV. Diagnostic study.

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Introduction

The natural history of anterior cruciate ligament (ACL) disruptions is characterized by the onset of meniscal and femorotibial lesions following a well-known evolutive process, but also by a process of patellar osteochondral lesions that are taken less seriously than these other lesions. The objective of this retrospective study was to evaluate the onset of patellar chondropathies in patients presenting ACL disruption and to study the severity of these lesions in relation to the duration of laxity.

Material and methods

We conducted a retrospective study of a continuous series of 250 files (all included) of patients who had undergone arthroscopic surgery performed by a single surgeon for anterior laxity of the knee between January 1995 and January 2005. All patients presented anterior laxity caused by disruption of the ACL with a positive Lachmann and Pivot shift test. None of them presented any other ligament damage. A standardized descriptive report on each lesion was made for every arthroscopic intervention (arthroscopic information, arthroscopic images, surgical reports). The International Cartilage Repair Society (ICRS) classification [1] (Fig. 1) and the Bauer and Jackson classification [2] (Fig. 2) were used to detail the type and topography of these lesions. We also analyzed any evidence of degenerative damage in the other knee compartments. The statistical analysis was based on the Kruskal-Wallis test and the Fisher exact test.

Results

Of the 250 patients presenting anterior laxity, 72 presented patellar chondral involvement (28.8%). These patients (57 males, 15 females) had a mean age of 32 years (range, 16–46 years). The circumstances of injury onset were for the most part pivot-contact sports (football, 27%; basketball, 13%; handball, 15%; skiing, 13%; other sports, 23%; traffic accident, 9%). The time between initial injury and surgery was a mean seven years (range, three months to 28 years), corresponding to chronic anterior laxity.

Location

The lateral side was involved in 41 patients (56.9%) (Fig. 3). The lateral facet only was involved in 13 patients (1%), the medial facet only in three patients (4.1%), the median border only in 20 patients (27.7%), the median border and

lateral facet in 22 patients (30.5%), the median border and medial facet in eight patients (11.1%) and finally, the entire patellar surface in six patients (8.3%). As for lesion extension, a mean 31% of the patellar surface was injured.

Grade

According to the Bauer and Jackson classification (Fig. 4), the lesions were stage 1 (linear) in ten patients (13.8%), stage 2 (stellate) in eight patients (11.1%), stage 3 (flap) in five patients (6.9%), stage 4 (crater) in one patient (1.3%), stage 5 (fibrillation) in 38 patients (52.7%), stage 6 (degrading) in ten patients (13.8%). According to the ICRS classification (Fig. 5), the lesions were grade 1 in 48 patients (66.6%), grade 2 in five patients (6.9%), grade 3 in eight patients (11.1%), and grade 4 in 11 patients (15.2%). On the whole, a majority of superficial lesions were noted (ICRS grade 1, Bauer and Jackson stages 1 and 5).

Statistical analysis

We found no correlation between the onset of damage and patient age or between lesion topography and type. On the other hand, the Kruskal-Wallis test demonstrated a statistically significant aggravation (p = 0.0016) of the ICRS grade in relation to the duration of laxity. However, there was no correlation between lesion location and laxity duration.

Associated lesions

The medial meniscus was injured in 33 patients (45.8%), the lateral meniscus in ten patients (13.8%) and both menisci in 21 patients (29.1%). The medial femoral condyle was affected in 25 patients (34.7%) and the lateral femoral condyle in seven patients (9.7%). The medial tibial plateau was injured in seven patients (9.7%) and the lateral tibial plateau in three patients (4.1%). There was a trochlear groove lesion in 14 patients (19.4%). Seven knees (9.7%) presented no associated lesion.

We found no statistically significant correlation between patellar lesions and these associated meniscal or femorotibial lesions.

Discussion

This descriptive epidemiological study did not include clinical evaluation of the results. Several classifications of chondral lesions have been proposed: the Insall classification and the Outerbridge classification [3] in 1961, the classifi-



Figure 1 ICRS classification.

cation proposed by Ficat et al. [4] in 1977, one put forward by the French Society of Arthroscopy [5] in 1994, etc. None of them has enjoyed unanimous support, except the ICRS classification (Fig. 1) (Brittberg) [1], as Smith et al. [6] as well as Robert et al. [7] underline. It classifies lesions based on their depth into five grades and allows a topographic classification. However, the assessment of the extension of the damage, measured in square millimeters, seemed highly random and we preferred using the breakdown into nine zones. In addition, it did not provide a very precise morphological assessment of these lesions; we therefore used the Bauer and Jackson classification [2] (Fig. 2), initially described to classify osteochondral lesions of the femoral condyles.

There are few publications in the literature on patellar involvement in anterior laxity of the knee. These lesions are



Figure 2 Bauer and Jackson classification.



Figure 3 Patellar lesion topography.





usually relegated to second place, well behind femorotibial and meniscal lesions. Segal et al. [8] found 33% patellar chondropathies in a series of 110 knees with anterior laxity. Mansat et al. [9] report 16.7% femoropatellar syndrome in anteromedial laxity, but they do not specify whether there was chondropathy nor the duration of laxity. In his study on the natural history of ACL damage, Dupont [10] analyzed the femoropatellar compartment lesions over time since the initial sprain: 10% were affected the first year, nearly 30% between five and 10 years, and 50% after 10 years. In a series of 129 ACL disruptions, Grégory et al. [11] found that 29% of patients had patellar chondral lesions. Our results are in agreement with these different publications, since during the review of a series of ACL repairs (Mabit et al.) [12], we found a 16.3% chondropathy rate for a mean 41-month preoperative anterior laxity duration, and in our current series, we found 28.8% at seven years of laxity progression.

The question arises of the mechanism(s) causing these patellar lesions, particularly of whether the damage is directly related to anterior laxity. Taking the femorotibial lesions with onset after ACL disruption as a reference, we assumed that patellar lesions were correlated with the duration of preoperative laxity, which was partly confirmed by our study (a statistically significant aggravation of the ICRS grade related to the duration of anterior laxity). However, these results reflect the depth of these lesions and not their onset; it cannot be concluded that their onset is directly attributable to ACL disruption. Certain seemingly recent lesions that we encountered could have occurred at the time of injury that led to ACL disruption. In other cases, the patellar damage could have occurred before the injury (many athletes suffer from femoropatellar syndrome that may correspond to preexisting patellar chondropathy).

The pathogenesis of these patellar lesions is not well understood and only biomechanical studies can identify the lesional mechanism(s) involved.

Today, the result of curative patellar lesion treatment remains uncertain [13,14]. In addition, since the lateral side is affected in 56.5% of the cases in this series and given that certain ACL repair techniques can lead to femoropatellar hyperpressure, the value of lateral patellar retinaculum release in acute tear reconstructions, as suggested by Shelbourne et al. [15], should be debated.

Conclusion

Today, the mechanisms behind the onset of meniscal and femorotibial lesions after ACL disruption are well-known. The presence of patellar chondropathy is not rare (nearly one-third of the cases in our series), but their lesional mechanisms remain much less well-known. In all cases, the increase in the duration of laxity is responsible for a statistically significant aggravation of these lesions.

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