Introduction
The underlying cause of OA has alluded clinicians for decades. Some believe that mechanical stress on joints is the underlying cause of osteoarthritis [2], while other evidence has implicated obesity [3], genetics [4,5], and changes in sex hormones in later years among postmenopausal old women [6,7] as underlying causes. To that end, the etiology of OA remains unclear and appears to be multi-factorial. High prevalence’s of OA have been reported in chronic ACL-deficient soccer players at 12 to 14 years post-injury [8,9]. Yet, chronic ACL-deficiency as a potential contributor of OA among non-athletes has not been well studied. Full thickness cartilage defects in the knee frequently continue to degenerate over time, and defect larger than 9 mm² often lead to degenerative arthritis [1]. As such, this study sought to analyze the defect characteristics from a large cohort of patients treated for articular cartilage defects of the medial femoral condyle.

Objectives

- Investigate and compare the characteristics of medial femoral condyle cartilage defects in chronic/acute ACL-deficient and non-ACL-deficient knees.
- Study design was a retrospective analyses of 566 knees that underwent cartilage repair for cartilage defects in the weight-bearing portion of the medial and/or lateral femoral condyles.
- Inclusion Criteria: Degenerative or traumatic cartilage defects of the medial femoral condyle with/without medial meniscus tear in chronic or acute ACL-deficient knees.
- Exclusion Criteria: Knees without cartilage defects of the medial femoral condyle.
- ACL Deficiency: 1) Complete ACL tear or 2) partial/intact ACL with laxity to intra-operatively tested anterior drawer sign.

Patients and Methods

- 465 knees total were included and patients were stratified by knee pathology (Table I).
- Chronic-degenerative and acute-traumatic cartilage defects were stratified into separate sub-groups (Table II).
- Chronic-degenerative sub-groups were the primary focus.

Table I Patient Groups

<table>
<thead>
<tr>
<th>Group</th>
<th>Knee Pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>Isolated Cartilage Defect</td>
</tr>
<tr>
<td>Group B</td>
<td>ACL-Deficiency + Defect</td>
</tr>
<tr>
<td>Group C</td>
<td>Medical Meniscus Tear + Defect</td>
</tr>
<tr>
<td>Group D</td>
<td>ACL-Deficiency + Medial Meniscus Tear + Defect</td>
</tr>
</tbody>
</table>

Table II Cartilage Defect Sub-Groups and Patient Characteristics

<table>
<thead>
<tr>
<th>Sub-Group</th>
<th>Patients N</th>
<th>Age* (mean)</th>
<th>BMI* (kg/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A-Chronic</td>
<td>N=135</td>
<td>48.1 (24.70)</td>
<td>33.1 (20.44-48)</td>
</tr>
<tr>
<td>Group A-Acute/Traumatic</td>
<td>N=19</td>
<td>42.0 (20.46)</td>
<td>34.0 (21.3-46)</td>
</tr>
<tr>
<td>Group B-Chronic</td>
<td>N=63</td>
<td>41.5 (23.67)</td>
<td>31.7 (20.44-48)</td>
</tr>
<tr>
<td>Group B-Acute/Traumatic</td>
<td>N=19</td>
<td>44.3 (19.71)</td>
<td>28.0 (21.48-46)</td>
</tr>
<tr>
<td>Group C-Chronic</td>
<td>N=156</td>
<td>54.3 (27.48)</td>
<td>33.4 (21.3-54.6)</td>
</tr>
<tr>
<td>Group C-Acute/Traumatic</td>
<td>N=19</td>
<td>55.5 (31.67)</td>
<td>32.2 (29.0-41.6)</td>
</tr>
<tr>
<td>Group D-Chronic</td>
<td>N=35</td>
<td>49.1 (24.63)</td>
<td>34.0 (20.57-48)</td>
</tr>
<tr>
<td>Group D-Acute/Traumatic</td>
<td>N=13</td>
<td>43.3 (18.92)</td>
<td>31.0 (22.3-48)</td>
</tr>
</tbody>
</table>

Observations and Assessments
- Duration of symptoms prior to cartilage repair surgery
- Concomitant medial meniscal tears and/or ACL-deficiency
- Defect size
- Defect location

Cartilage Defect Mapping System

A number-modified version of the International Cartilage Repair Society (ICRS) articular cartilage mapping system was used to map defect location on the medial femoral condyle.

Results

Prevalence of ACL-Deficiency
- Overall prevalence of ACL-deficiency among the original series of 566 knees (before exclusions) treated for femoral articular cartilage defects was 27.3% (n=157).

Defect Distribution (Central Condyle Only)
- A greater percentage of medial condyle defects was observed more laterally (C2/3) and C3 in both chronic ACL-deficient groups versus chronic non-ACL-deficient groups.

Figure 3. Defect Distributions (Chronic groups A and B)

Figure 4. Defect Distributions (Chronic groups C and D)

Figure 5. Scatterplot Diagram

Figure 6. Scatterplot Diagram

Discussion

- We could not definitively determine whether defects of the chronic ACL-deficient groups occurred after ACL injury (as degenerative defects) or at the time of ACL injury (as traumatic defects). However, defects of both degenerative ACL-deficient groups (B & D) were larger than those of the respective acute-traumatic groups.
- Therefore one of two inferences can be made: The defects that were treated in chronic ACL-deficient knees either 1) occurred at the time of ACL injury as traumatic defects and enlarged over time or 2) subsequently detected after ACL injury as degenerative defects from altered knee biomechanics.
- Porat et al reported radiographic evidence of advanced OA in 41% of 219 male soccer players at a mean of 14 years post-ACL injury [8]. Lohmander et al reported symptomatic radiographic evidence of OA in 42% of 67 female soccer players at a mean of 12 years post-ACL injury [9].

Conclusion

Chronic ACL-deficiency should not go overlooked in athletes and non-athletes alike when clinically detected, and we hope that this study will inspire further investigation by others.

References