based on semiquantitative MRI assessment and to determine their risk for progression over 48 months.

Methods: The FNIH was designed as a case-control study with knees showing either 1) radiographic and pain progression (i.e., “composite” cases), 2) radiographic progression only (“JSL”), 3) pain progression only, and 4) neither radiographic nor pain progression. MRI of both knees was performed on 3 T systems at the four OAI clinical sites. Two musculoskeletal radiologists read the baseline MRIs according to the MOAKS scoring system. Knees were stratified into subchondral bone, meniscus/cartilage and inflammatory phenotype. A secondary, less stringent definition for inflammatory and meniscus/cartilage phenotype was used for sensitivity analyses. The relation of each phenotype to risk of being in the JSL or composite case group compared to those not having that phenotype was determined using conditional logistic regression. Only KL 2 and 3 and those without root tears were included.

Results: 485 knees were included. 362 (75%) did not have any phenotype, while 95 (20%) had the bone phenotype, 22 (5%) the cartilage/meniscus phenotype and 19 (4%) the inflammatory phenotype. The bone phenotype was associated with a higher risk of the JSL and composite outcome (OR 1.81 [95% CI 1.4, 2.85] and 1.65; 95% CI [1.04, 2.61]) while the inflammatory (OR 0.96 [95% CI 0.38, 2.42] and 1.25; 95% CI [0.48, 3.25]) and the meniscus/cartilage phenotypes were not (OR 1.30 95% CI [0.55, 3.07] and 0.99; 95% CI [0.40, 2.49]).

In sensitivity analyses, the bone phenotype and having two phenotypes (vs. none) were both associated with increased risk of experiencing the composite outcome (bone: OR 1.65; 95% CI 1.04, 2.61; 2; phenotypes: OR 1.87; 95% CI 1.11, 3.16).

Conclusion: The bone phenotype was associated with increased risk of having both radiographic and pain progression together, or radiographic progression alone, whereas the inflammatory phenotype or meniscus/cartilage phenotype each individually were not associated with either outcome. Phenotypic stratification appears to provide insights into risk for structural or composite structure plus pain progression, and therefore may be useful to consider when selecting patients for inclusion in clinical trials.

References:

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How Many OA Patients Will Be Overtreated If Treated with DMOADs? Analysis of Osteoarthritis Initiative Data

I. Shirinsky1, V. Shirinsky2. 1Federal State Budgetary Scientific Institution Research Institute of Fundamental and Clinical Immunology, Novosibirsk, Russian Federation

Background: Significant research effort is currently put on discovering drugs able to slow structural progression of osteoarthritis (OA). In many patients OA shows little or no increase in pain or function deterioration over time. Thus, giving disease-modifying OA drugs (DMOAD) for long periods to this subset of patients can be considered as overtreatment. There is a lack of studies directly evaluating how many patients with diagnosed OA experience no impact of the disease during long-term follow up and thus probably do not need any disease modification.

Objectives: To assess proportions of patients with diagnosed symptomatic knee OA or frequent knee pain that does not result in sustained pain and limitation of function during long-term follow up.

Methods: For the current study we used 8-year longitudinal data obtained from the Osteoarthritis Initiative (OAI) progression (n= 1390) and incidence (n= 3284) subcohorts, which are publicly available at https://oa.ihi.nih.gov. For the analyses we included knees having frequent knee symptoms in the past 12 months before the baseline for at least one month. Thus the analyzed group comprised patients fulfilling the definition of symptomatic knee OA (frequent symptoms + Kellgren-Lawrence (KL) grade ≥ II) and people who might have early OA (frequent symptoms + KL grade < II) in accordance with the proposed draft classification criteria [1].

The proportion of knees experiencing no impact of OA at the 8 years follow up was assessed.

No impact of OA was defined if a knee fulfilled all the following criteria: 1. WOMAC pain score within normative values; 2. WOMAC disability score within normative values. 3. Absence of joint replacement.

We used previously reported reference population age and gender adjusted values for WOMAC pain knee and WOMAC knee function [2]. It is unlikely that any OA treatment is capable to improve WOMAC pain and function measures above these reference ranges in a given person [2].

Results: We included 3092 knees from 2147 participants in the analysis. The mean age of participants was 61.16 years, the mean BMI was 28.59. Almost half of symptomatic knees with baseline KL grades 0-1 were not impacted by the disease at 8 year follow up. Every fifth knee with symptomatic knee OA (KL grades ≥ II) had no impact of the disease at the end of follow up. Every third knee with symptomatic KL grade II OA did not develop pain or disability outside the reference range. The percentage of symptom-free knees at year 8 declined progressively with higher KL grades (Table).

Table. Percentage of knees with no impact of OA on year 8 follow up depending on baseline KL grade.

<table>
<thead>
<tr>
<th>Baseline KL grade</th>
<th>Total n of knees</th>
<th>Percentage of knees with no impact of OA</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>894</td>
<td>47 %</td>
</tr>
<tr>
<td>I</td>
<td>469</td>
<td>43.1 %</td>
</tr>
<tr>
<td>II</td>
<td>923</td>
<td>31.31 %</td>
</tr>
<tr>
<td>III</td>
<td>609</td>
<td>17.7 %</td>
</tr>
<tr>
<td>IV</td>
<td>157</td>
<td>7.1 %</td>
</tr>
</tbody>
</table>

Conclusion: If given DMOADs, a substantial proportion of OA patients would be overtreated, especially those with early OA.

References:

Disclosure of Interests: None declared

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