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CHANGE IN TIBIAL CARTILAGE VOLUME IS ASSOCIATED WITH CHANGE IN SYMPTOMS IN SUBJECTS WITH KNEE OSTEOARTHRITIS. Wluka, A.E., Wolfe, R., Stuckey, S. CICUTTINI, F.M. Department of Epidemiology and Preventive Medicine, Monash University, MRI Unit, Alfred Hospital.

Background: The relationship of pain and structural damage in osteoarthritis (OA) is poorly understood. No consistent relationship between change in radiological OA and change in symptoms of knee OA has been demonstrated.

Aim: To determine the relationship between change in symptoms of knee OA and tibial cartilage volume over a 2 year period.

Method: Prospective cohort study examining subjects with symptomatic early (mild to moderate) knee OA, recruited by advertising from the community, general practitioners, rheumatologists and orthopedic surgeons. At baseline and 2 years later, participants had MRI scans of their knee and completed questionnaires to quantify symptoms of knee OA (WOMAC: pain, stiffness, function) and general health status (SF-36). Tibial cartilage volume was determined using sagittal T1-weighted fat suppressed images.

Results: 126 subjects entered the study. Complete data was available for 117 subjects (93%). Over 2 years, reduction in tibial cartilage volume was associated with an increase in pain scores (r=0.27, p=0.003), and to a lesser extent with worsening of stiffness (r=-0.17, p=0.07), deterioration in function (r=-0.19, p=0.003) and improved mental health (r=-0.08, p=0.39).

Conclusion: Increased loss of tibial cartilage in knee OA is associated with worsening of symptoms in knee OA. This suggests that the structural change of loss of tibial cartilage contributes to symptoms in knee OA and provides support for a role to prevent knee cartilage loss in the management of OA.


Aims: End-stage radiographic OA in a lower limb joint may predispose to progression in other lower limb joints. Therefore we examined the association between radiological progression of Knee OA and OA progression of the hands, hips and lumbar spine in a population-based cohort.

Methods: 914 Knee x-rays were read for osteophytes (OS) and joint space narrowing (JSN). Knee OA was defined as a grade 1+ OS or JSN in any compartment at baseline. Progression was defined as a new grade 1+ feature or an increase at Yr10 from a grade 1+ at baseline. OA progression status was available for hand, hip and lumbar spine. The association of OA progression at different anatomical sites was analysed using OR ±95% CI in univariate and logistic regression models using STATA.

Results: Mean age ± SD at baseline was 54.2 ± 6.0 years and BMI 25.46 ± 4.3 kg/m2. 89 women had progression of Knee OA based on OS and 51 on JSN. Lumbar Spine progression increased 2.3-fold the risk of Knee OA progression. Hip OA progression increased 2 fold the risk of Knee OA JSN but not OS progression. The increased risks remained after adjusting for age and BMI.

Conclusions: This is the first population-based report of the association of radiographic Knee OA progression and OA progression in the lumbar spine and hip. This may have implications for the future development of DMOAD’s.

ASSOCIATION OF THE C282Y AND H63D MUTATIONS IN THE HFE GENE WITH OSTEOARTHRITIS IN THE INDEX AND MIDDLE FINGER MCP, HIP AND ANKLE/FOOT JOINTS. CARROLL GJ. ArthroCare Pty Ltd and Royal Perth Hospital.

Background and Aims: A characteristic arthropathy involving the index and middle finger MCP joints (MCP2, 3), the hips and ankles (HH target joints) is recognized in up to 70% of patients with Hereditary Haemochromatosis (HH). The aim of this study was to test the hypothesis that heterozygosity for the C282Y or H63D mutations in the HFE gene is associated with OA in the HH target joints.

Methods: Patients with clinical signs of OA in either the MCP2, 3, elbow, hip, ankle, intertarsal (IT) or tarsometatarsal (T/M) joints were evaluated. Patients with PIP or DIP OA, but no involvement of the former joints served as controls. Plain radiographs were performed. Patients were considered to have OA if they met grade 2 Kellgren and Lawrence criteria.

Results:

<table>
<thead>
<tr>
<th>Region</th>
<th>N</th>
<th>C282Y</th>
<th>H63D</th>
<th>% Heterozygous for either mutation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Finger IPs</td>
<td>40</td>
<td>2</td>
<td>3</td>
<td>13</td>
</tr>
<tr>
<td>Index or Middle finger MCPs</td>
<td>28</td>
<td>7</td>
<td>11</td>
<td>64</td>
</tr>
<tr>
<td>Elbows</td>
<td>6</td>
<td>3</td>
<td>1</td>
<td>67</td>
</tr>
<tr>
<td>Hips</td>
<td>18</td>
<td>3</td>
<td>9</td>
<td>67</td>
</tr>
<tr>
<td>Ankles, inter-tarsal or T/M joints</td>
<td>15</td>
<td>3</td>
<td>8</td>
<td>73</td>
</tr>
</tbody>
</table>

Conclusions and Discussion: These results show an association between the two common HFE mutations and OA in the HH target joints. It is possible that the HFE mutations are simply passenger mutations and that the observed OA is due to tight gene linkage. In that event there will be a need to identify the linked gene(s) before mechanistic inferences can be made. However if C282Y and H63D are not passenger mutations, disordered HFE gene function may be important in OA pathogenesis.