The multivariate analysis demonstrated that the variables influencing the quality of life (IBD-Q) were the disease activity (CDAI) (p = 0.0258), the chronic fatigue (FACT-F) (p = 0.0061) and sleep disturbances (PSQI) (p = 0.0440), for CD; whereas for UC the only variable that correlated with IBD-Q was the disease activity (Mayo score) (p = 0.0129).

Conclusion: FM is a common disorder especially in patients with other concomitant chronic diseases. This study reported a prevalence of FM of 8.7% in IBD patients without any significant differences between CD and UC. Moreover, the comorbidity of FM in IBD can have a considerable impact on quality of life and on measures of disease severity, with worst values in all PROs measurements.

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PREDICTION OF SPONTANEOUS IMPROVEMENT IN PATIENT REPORTED OUTCOME SCORES IN OSTEOARTHRITIS USING MARKERS OF JOINT TISSUE TURNOVER

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Background: Osteoarthritis (OA) is a chronic disease characterized by pain and disability. There is no modifying treatment approved for OA today. This may be attributed to the difficulty generating a robust response based on patient-reported outcomes (PROs) linked to the drug mode of action. There is a need in drug development to test and validate biomarkers that objectively relate to PROs and/or even predict changes in PROs. Biomarkers of cartilage and bone turnover are associated with structural and symptomatic progression. In addition, recent findings suggest that a subset of OA patients have elevated serum levels of C-reactive protein metabolites (CRPM), which is predictive of radiographic progression.

Objectives: This explorative study aimed to investigate the association between PROs and markers of joint tissue formation and degradation in patients with either high or low levels of CRPM. In particular, whether levels could predict spontaneous improvement in PROs.

Methods: 146 knee OA patients, 62% women, from the NYU cohort were included. Mean (SD) age, 62.5 (10.1); BMI, 26.6 (3.6); 32% NSAID users; and 67.6% w. radiographic OA (KL2-3). PROs were recorded at baseline (BL) and 2 years (FU), and the current investigation was: WOMAC pain, stiffness, and function. The mean (SD) for WOMAC pain, stiffness, and function were 35.4 (22.9), 40.8 (25.7), and 41.7 (28.3) mm on a 100 mm scale. Twenty-one healthy individuals were included as a reference. Eight serum biomarkers of type I, II, III, and IV collagen degradation (C1M, C2M, C3M, C4M) and formation (PRO-C1, PRO-C2, PRO-C3, and PRO-C4) as well as the inflammatory biomarker CRP were assessed at baseline. LN-transformed data was adjusted for race, sex, BMI, and NSAID use when comparing OA to controls and in the predictive model. Marked symptomatic (S) OA was defined as ≥40mm in either of the WOMAC scores at BL and improvement as 20mm decrease in any of the scores from BL to FU.

Results: There was no difference in mean marker levels between controls and OA patients. Only C2M correlated with the WOMAC scores at baseline in the ALL population (p <0.001). This correlation was maintained in both the high and low CRPM groups. A high correlation was observed between the PROs and PRO-C4, C1M and C3M, but only in the high CRPM group. Next, we investigated whether the markers could predict symptomatic improvement in patients with marked SOA. A combination of C4M, Age and BMI was predictive of pain improvement in the ALL population (Table 1). Interestingly the predictors were different in the low vs. high CRPM group; PRO-C2, PRO-C3, PRO-C4 and Sex predicted a 20mm decrease in WOMAC pain in the low group, while C2M alone predicted an improvement in the high CRPM group. Moreover, C2M predicted an improvement in stiffness in the CRPM high, but not in the low CRPM group. C1M and C3M predicted a 20mm decrease in function only in the high CRPM group.

Conclusion: Levels of the joint tissue markers weew subtle compared to controls. However, the markers, together with sex and BMI, could predict symptomatic improvement. This may provide novel insight into the link between tissue turnover and PROs.


Table. Prediction of symptomatic regressive OA in patients with symptomatic knee OA. Multiple variate logistic regression including In-transformed biomarkers and baseline clinical characteristics (age, gender, race, BMI and NSAID use). Not adjusted for multiplicity.

<table>
<thead>
<tr>
<th>Regressive vs. stable OA</th>
<th>ALL</th>
<th>Low CRPM</th>
<th>High CRPM</th>
</tr>
</thead>
<tbody>
<tr>
<td>WOwMAC pain n [%]3 Predictors</td>
<td>61 (41.0) C4M, Age, BMI</td>
<td>35 (44.3) PRO-C2, PRO-C3, PRO-C4, Sex</td>
<td>26 (50.0) C2M</td>
</tr>
<tr>
<td>AUC [95% CI]</td>
<td>0.73 [0.60 – 0.84]</td>
<td>0.73 [0.62 – 0.83]</td>
<td>0.79 [0.58 – 0.92]</td>
</tr>
<tr>
<td>P</td>
<td>0.0042</td>
<td>0.056</td>
<td>0.024</td>
</tr>
<tr>
<td>WOMAC stiffness n [%]3 Predictors</td>
<td>57 (47.4) C2M</td>
<td>33 (48.5) None</td>
<td>24 (45.8) C2M</td>
</tr>
<tr>
<td>AUC [95% CI]</td>
<td>0.67 [0.53 – 0.79]</td>
<td>0.67 [0.53 – 0.79]</td>
<td>0.85 [0.64 – 0.96]</td>
</tr>
<tr>
<td>P</td>
<td>0.020</td>
<td>0.085 [0.64 – 0.96]</td>
<td>0.0007</td>
</tr>
<tr>
<td>WOMAC function n [%]3 Predictors</td>
<td>61 (27.8) None</td>
<td>35 (22.7) None</td>
<td>26 (34.6) C1M, C3M</td>
</tr>
<tr>
<td>AUC [95% CI]</td>
<td>0.83 [0.63 – 0.95]</td>
<td>0.83 [0.63 – 0.95]</td>
<td>0.0074</td>
</tr>
</tbody>
</table>

3Regression was defined as ≥10 or 20 mm or more decrease in VAS pain, WOMAC pain, stiffness or function, and 30 mm or 60 mm or more decrease in WOMAC total, over two years. 2Rate of regression in percentage in each of the sub-populations.
**Objectives:** Our purpose was to determine the prevalence of ADHD symptoms in patients with FM and to assess the relationship with disease impact.

**Methods:** Consecutive patients, older than 18 years, with diagnosis of FM (ACR 2016 criteria) without known cognitive impairment, seen at the Rheumatology Unit between April 2018 and December 2019, were included. At inclusion visit the collected data included Revised Fibromyalgia Impact Questionnaire (FIQ-R) and Health Assessment Questionnaire, Argentine version (HAQ-A). During the Neurology visit, the following tests were performed: Montreal Cognitive Assessment (MoCA) test for evaluating the presence of cognitive impairment, Conners Continuous Performance Test II (CPT II) for the assessment of ADHD, and Wender-Utah Rating Scale (WURS) to retrospectively assess childhood ADHD symptoms. Univariate analysis was performed using t-tests for normally distributed continuous variables, and Wilcoxon rank sum test for non-normally distributed continuous variables. A chi-square or Fisher test was used when appropriate for categorical variables. Predictors that were found to be related to ADHD (p ≤ 0.20) were then entered into a multivariable logistic regression model.

**Results:** 60 patients with FM and 71 matched controls without FM or known cognitive impairment were included. FM patients' characteristics are shown in Table 1. 61.7% (n=37) of the patients with FM tested positive for adult ADHD. In 48.6% (18/37) of them, the diagnosis had been missed in childhood. Participants with both FM and a positive adult ADHD screening test did not score significantly higher on the FIQ-R (52.3, SD=16.1 vs. 47.9, SD=12.3; p=0.2693) and HAQ-A (0.693, SD=0.455 vs. 0.521, SD=0.428; p=0.1523) compared with patients without ADHD. Retrospectively assessed childhood ADHD was significantly associated with adult persistence (OR 55.1, CI=3.6 to 842.6, p=0.004).

**Conclusion:** The co-occurrence of adult ADHD in FM was highly prevalent. In nearly half of the patients the diagnosis had been overlooked during childhood and it was associated with adult persistence. The prevalence of cognitive impairment, and childhood and adult ADHD was higher in patients with FM compared with the control group. ADHD was not associated with the FM impact. Evaluation of ADHD symptoms in patients with FM is important for recognition and treatment of this comorbidity.

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**Disclosure of Interests:** None declared

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