level >60 IU/L (OR=5.4; CI95%: 1.9-15.3) were predictive for progression to RA.

Conclusion: Our study suggests that clinical evaluation of EIA by DAS28-CRP from the time of diagnosis, as well as evaluating the presence of RA auto-antibodies, can predict progression to RA.

Disclosure of Interests: None declared

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AB0220

TENOSYNOVITIS AS THE PRESENTING FEATURE OF FLARE IN RHEUMATOID ARTHRITIS

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Background: The importance and relevance of tenosynovitis (TS) has long been recognised in rheumatoid arthritis (RA), but it is not usually considered in disease activity assessments. The significance of TS in early arthritis (EA) has also been recognised and, using ultrasound (US) it has recently been identified in disease activity assessments. The significance of TS in early arthritis (EA) has also been recognised and, using ultrasound (US) it has recently been identified as a precursor to RA1. The ongoing BIO-FLARE (Biological Factors that Limit sustained Remission in rheumatoid arthritis) observational study aims to investigate the pathogenesis of flare in RA. Patients with RA in remission stop their disease modifying anti-rheumatic drug medication (DMARDs: methotrexate, sulfasalazine and/or hydroxychloroquine) and are closely followed for 6 months, in anticipation that approximately 50% will experience a flare. We investigated whether TS occurrence was a frequent herald of flare in this cohort.

Objectives: To review the case notes of 49 patients in the BIO-FLARE study with confirmed flare to date, seeking evidence of US tenosynovitis prior to or concurrent with flare.

Methods: Patients in the study who are deemed to be in remission based on a disease activity score (DAS28-CRP) < 2.4 stop their DMARD medication and attend regularly for review over 6 months, with provision for ad-hoc appointments if symptoms return between visits. Patients are defined as having a flare if their DAS28-CRP ≥ 3.2 at any point or two consecutive DAS28-CRP ≥ 2.4. Targeted US assessment occurs at baseline only for patients that consent to an optional US assessment which occurs at baseline only for patients that consent to an optional US assessment. We performed a 6-monthly evaluation for 2 years and recorded US data at each visit. A 6-monthly evaluation for 2 years and recorded US data at each visit. A 6-monthly evaluation for 2 years and recorded US data at each visit. A 6-monthly evaluation for 2 years and recorded US data at each visit.

Results: To January 2020, 120 patients had been recruited into the study and 49 experienced a flare. Seven patients had a flare predominantly or initially characterised by TS or paratenonitis, the results of which are summarised in Table 1.

Table 1. Tenosynovitis in BIO-FLARE

<table>
<thead>
<tr>
<th>DMARD stopped</th>
<th>Time to TS, weeks</th>
<th>Tendon involved</th>
<th>Time to flare, weeks</th>
<th>Joints involved</th>
</tr>
</thead>
<tbody>
<tr>
<td>Methotrexate, sulfasalazine, hydrochloroquine</td>
<td>12</td>
<td>Extensor carpi ulnaris</td>
<td>12</td>
<td>Shoulders andPIPJs, no synovitis suitable to biopsy</td>
</tr>
<tr>
<td>Methotrexate</td>
<td>7</td>
<td>Bilateral extensor carpi ulnaris</td>
<td>7</td>
<td>Shoulders, wrists, knees, PIPJ with no accompanying synovitis</td>
</tr>
<tr>
<td>Methotrexate</td>
<td>5</td>
<td>Tibialis posterior</td>
<td>5</td>
<td>No joints flared, no synovitis but treated as a flare due to severity of TS</td>
</tr>
<tr>
<td>Methotrexate</td>
<td>8</td>
<td>Tibialis posterior – attributed to increase in patient activity</td>
<td>22</td>
<td>MCPJ, PIPJs, mid tarsal and MTPJ</td>
</tr>
<tr>
<td>Methotrexate and hydrochloroquine</td>
<td>7</td>
<td>Extensor pollicis longus</td>
<td>8</td>
<td>Polyradicular flare</td>
</tr>
<tr>
<td>Methotrexate and hydrochloroquine</td>
<td>2</td>
<td>Extensor carpi ulnaris – attributed to overuse</td>
<td>6</td>
<td>Polyradicular flare</td>
</tr>
<tr>
<td>Methotrexate</td>
<td>12</td>
<td>Extensor paratenonitis at PIPJ4 &amp; 5</td>
<td>12</td>
<td>MCPJ synovitis</td>
</tr>
<tr>
<td>Methotrexate</td>
<td>12</td>
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</tr>
</tbody>
</table>

Conclusion: Although highlighted as a precursor of RA in early arthritis1, the occurrence of TS in the context of flare – and the prodrome heralding this – has not been studied. Our findings show that TS in early flare is reminiscent of the features sometimes seen in EA or clinically suspect arthralgia2. Further data are required to determine the role of periartricular inflammatory phenomena, such as TS, as risk factors for joint synovitis. Our study did not entail formal US assessments, therefore the rate of TS in this population may be underestimated.

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References:

AB0221

ULTRASOUND ASSESSMENT OF INFLAMMATORY ARTHRALGIA: PREDICTORS FOR CHRONIC ARTHRITIS DEVELOPMENT

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Objectives: Establishing US findings that can be related to CA development in patients with inflammatory arthralgia without arthritis. Assess the link among US, clinical and biochemical parameters.

Methods: We performed a prospective longitudinal study of a cohort of patients with IA. Patients with less than one year of AI evolution and involvement of at least one small joint from hands or feet were included. Patients with arthritis, osteoarthritis, fibromyalgia and those treated with DMARDs or steroids were excluded. We made a 6-monthly evaluation for 2 years and recorded the CA development during that period. The number of painful joints (PJC) and biochemical data (CRP, ESR) were assessed at the first visit. A blind US exploration was made using a MyLabTwice (Esaote) equipment with a 5-15MHz probe for greyscale (GS) and Power Doppler (PD), examining 36 joints (radio-carpal, MCP, IPP, 2nd-5th MTP, elbows, shoulders and knees) and 14 tendon compartments (2nd, 4th and 6th wrist extensors, 3rd and 4th finger flexors and posterior tibial and fibularis tendons), giving an overall score of GS, PD (0-3) and number of erosions by rating the presence of sinovitis on each location.

We performed a descriptive analysis based on the frequencies of qualitative variables and means/SD/median (IQR) of quantitative variables, comparing the characteristics between patients with and without CA progression by Chi-Square and Mann-Whitney U tests. Also, the possible relationship of those variables and the disease progression was assessed by a univariate binary logistic regression analysis.

We designed a reduced US examination (RUE) selecting the most affected joints and those with greatest differences between groups in the statistical analysis.

Results: Of the 49 patients included, 21 (42.9%) progressed to CA. 87% were females and 71.4% non-smokers with a mean age of 44 ± 12 years. The median of PJC was 4 (1-9). RF and/or CCPA were positive in 18.4% and 34.7% had high CRP/ESR. The suggested RUE included carpi, 2nd-4th MCP, 2nd-3rd IPP, 2nd and 5th MTP, 4th and 6th wrist extensors and fibularis tendons. Scores and comparative analysis within subgroups are listed in Table 1. The RUE score was significantly greater in both GS (OR 1.4, CI 95%) and PD (OR 1.3, CI 95%) on patients that progressed to CA.
Background: Physician’s global assessment of disease activity (PhGA) is highly influential upon treatment decisions taken by rheumatologists, surpassing the impact of DAS28. [1, 2]. However, data regarding its psychometric properties are scarce.

Objectives: To evaluate the reliability and responsiveness of PhGA.

Methods: We included two consecutive visits of RA patients followed in a Tertiary Rheumatology Department. Socio-demographic (age and gender) and clinical data were collected including tender (TJ28) and swollen joint counts (SJC28), Disease activity Score (DAS28-3v-CRP, DAS28-3v-ESR, DAS28-4v-CRP, DAS28-4v-ESR), PhGA and Patient Global Assessment of disease Activity (PGA) through a Visual Analogic Scale (VAS) 0-100mm. Changes (Δ) between the two visits were calculated. Only patients without missing data were included. Correlations between ΔPhGA and change of other variables were assessed using Pearson’s correlations. Reliability was evaluated through Intraclass Correlation Coefficient (ICC) between two consecutive appointments in a subgroup of patients with stable disease activity evaluated through Standardized Response Mean (SRM). The respective intervals of confidence were obtained through bootstrapping procedures. SRM above 0.8 were considered large. Independent factors associated with ΔPhGA were identified through multivariate linear regression analysis, p<0.05 was considered statistically significant.

Results: 121 RA patients (84.3% female and 64.0±12.6 years) were included. ΔPhGA was weakly correlated with ΔCRP (r=0.23), Δ PGA (r=0.31) and Δ pain (r=0.37). Moderate to strong correlations were observed with Δ DAS28-3v-ESR (r=0.57), Δ SJC28 (r=0.56), Δ DAS28-3v-CRP (r=0.58), Δ DAS28-4v-CRP (r=0.60), Δ TJ28 (r=0.62) and Δ DAS28-4v-ESR (r=0.63). ICC between two consecutive visits was 0.7, [95%CI: 0.47-0.83] and SRM was -1.01 [95%CI: -1.26(-1.73)]. In the multivariate regression analysis, ΔSJC28 (β=-0.41; 95% CI: 0.37 to 4.96) and Δ Pain (β=0.18; 95% CI: 0.07 to 0.28) remained as independent factors associated with ΔPhGA (R2=0.49, p<0.01).

Conclusion: In this study, PhGA showed a high reliability and sensitivity to change regarding disease activity, in clinical practice. Changes in SJC had the strongest association with change in PhGA scoring, but Δ Pain was also significantly correlated (graph 1).