RESULTS: 152 patients were included, (80 adalimumab-treated and 72 etanercept-treated). No statistically significant difference between mean IL-17A, IL-17AF, IL-17F and IL-10 levels at baseline and 3 months follow-up were observed (Figure 1). For IL-17A, those patients classified as good responders demonstrated an increase in mean serum levels of IL-17A from 1.06 pg/ml at baseline to 1.23pg/ml at 3 months. This came close to significance (p=0.07). Further analysis was carried out by drug group and also a subgroup analysis by drug group linked to responder status. No statistically significant results were obtained. Adjusting for gender, baseline DMARD use and DAS-28 scores did not alter findings.

<table>
<thead>
<tr>
<th>IL-17 Correlation</th>
<th>Mean IL-17A Baseline</th>
<th>Mean IL-17AF Baseline</th>
<th>Mean IL-17F Baseline</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Baseline</strong></td>
<td>1.16</td>
<td>1.21</td>
<td>1.97</td>
</tr>
<tr>
<td><strong>3 month</strong></td>
<td>1.06</td>
<td>1.19</td>
<td>1.28</td>
</tr>
</tbody>
</table>

Figure 1. Mean interleukin levels at pre-treatment and 3 months

CONCLUSION: There was a lack of statistically significant data to suggest a correlation between pre-treatment and 3 month IL-17F, IL-17AF and IL-10 concentrations. However, an increase in IL-17A serum levels between baseline and 3 months may be associated with a good EULAR response status by 6 months. Larger sample sizes are required to confirm this.

REFERENCES

Disclosure of Interests: None declared

AB0077 SERUM IL17 CORRELATED WITH INFLAMMATORY CHANGES BUT NOT WITH BONY CHANGES IN BOTH HAND OSTEOARTHRITIS AND RHEUMATOID ARTHRITIS
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Background: previous studies showed that serum IL17 is correlated with disease activity in both osteoarthritis and rheumatoid arthritis which may have therapeutic reflections (1,2). Taking in consideration that OA has different pathogenesis and presentations in comparison with RA; it is not known what the source of high serum level of IL17 in both diseases is?

Objectives: To evaluate the association of IL17 serum level with musculoskeletal ultrasound (MSK US) findings and clinical activity disease in RHEUMATOID arthritis

Methods: It is a randomized controlled clinical trial, which was done in the period between June 2017 and June 2018. The total number of the included subjects was 120 in 3 equal groups (RA, OA, and control). All subject was subjected to between June 2017 and June 2018. The total number of the included subjects

RESULTS: Levels of serum IL17 were significantly higher (p<0.0001) in both RA group and OA (141.37±51.09 and 151.89±23.13) group in comparison to control group (32.4±62.71).

The serum level of IL17 was correlated with disease activity parameters (DAS28 & VAS for RA and VAS for OA) both RA and OA groups. As regards ultrasonographic findings, serum IL17 levels were correlated with the presence of synovitis in both RA and OA groups. Ultrasonographic detected bony changes (erosions and osteophytes) were not correlated with serum level of IL17 in neither RA nor OA group. As regards functional assessment, serum level of IL17 was correlated with HAQ in RA group but not correlated with AUSCAN in OA group.

CONCLUSION: Serum level of IL17 was correlated with inflammatory changes detected by US and not correlated with bony changes (erosions and osteophytes). According to the results of this study, the inflammatory process is the source of increased level of IL17 in OA and RA.

REFERENCES

Disclosure of Interests: None declared

AB0078 THE THERAPEUTIC EFFECT OF GPMB IN A TRAUMATICALLY-INDUCED OSTEOARTHRITIC MODEL
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Background: Osteoarthritis is a severe joint disease that affects millions of people. At this time, the current treatment for osteoarthritis is total joint reconstruction surgery. GPNMB plays a key role in bone remodeling and bone growth. Data from our lab suggested that GPNMB is positive regulator of osteoblastogenesis and a negative regulator of osteoclastogenesis.

Objectives: The role of GPNMB in cartilage has not been investigated before. In this study we examined the therapeutic effects of GPNMB on damaged cartilage using post-traumatic osteoarthritic mouse model.

Methods: The destabilization of the medial meniscus (DMM) surgery in mice has been found to be an excellent model for studying post-traumatic osteoarthritis. We performed the DMM surgery on 21 C57BL6 mice. These mice were divided into three intra-articular injection treatment groups consisting of a control, low dose GPNMB, and high dose GPNMB. These mice were divided into three intra-articular injection treatment groups consisting of a control, low dose GPNMB, and high dose GPNMB. Moderate to severe osteoarthritis develops around six to eight weeks with this model.

Results: Here we present that damaged human cartilage has significantly higher levels of GPNMB compared to undamaged cartilage. In addition, human osteoarthritic chondrocytes treated with GPNMB showed a protective response to inflammation induced by IL1-beta. In this study, we examined whether recombinant GPNMB has an anti-inflammatory effect in a model of post-traumatic osteoarthritis. Based on studies performed in our lab, we expected cartilage degeneration to be dramatically decreased in response to the therapeutic effects of GPNMB. A protective factor against osteoarthritis progression, GPNMB-injected mice had significantly reduced cartilage damage and OAASI scores in comparison to the control group, proving GPNMB a promising therapy in lieu of total joint reconstruction. GPNMB-injected mice also had reduced expression of IL-6 and MMP13 but significantly increased expression of aggrecan in comparison to control mice.

Conclusion: Our data clearly showed that GPNMB has therapeutic anti-inflammatory effects on protecting cartilage damaged. Hence, future studies will be directed towards examining the therapeutic effects of GPNMB on larger animal models for osteoarthritis. Given the remarkable ability of GPNMB to reduce expression of key inflammatory markers, we conducted this study to reveal GPNMB therapeutic effects in traumatically induced osteoarthritis.

REFERENCES