useful for assessing disease activity\(^2\), the relation between MRI findings and RPILD has not been investigated.

**Objectives:** In this study, we assessed whether WB-MRI findings are related to RPILD in patients with DM.

**Methods:** This retrospective study comprised 33 patients with DM who underwent WB-MRI in our hospital before the initiation of treatment for myositis. Muscular and myofascial signal abnormalities were scored on 42 muscular groups. The ratio of myofascial score to muscular score (myofascial/muscle ratio) was calculated and used to evaluate myofascia-dominant inflammation. RPILD was defined as the acute onset and rapid worsening of dyspnea, hypoxemia and radiographic ILD/fibrosis within 1 month.

**Results:** Of 33 patients, 16 were CADM, and 24 had ILD, including 8 patients with RPILD. All patients including CADM showed abnormal signal intensity in muscle and myofascia (scores median: 15 and 21, respectively). Muscle scores positively correlated with serum level of creatine kinase (r=0.672, p<0.001). The patients with RPILD showed a significantly higher myofascia/muscle ratio compared with that in the non-RPILD patients (2.167 vs 1.000; p=0.018). Logistic regression analysis identified myofascia/muscle ratio >1.53 (odds ratio: 141.90, p=0.043) and older age (odds ratio: 1.15, p=0.041) as independent risk factors for RPILD.

**Conclusion:** We newly defined myofascia-dominant inflammation using WB-MRI as a predictor to develop RPILD in patients with dermatomyositis.

**REFERENCES:**


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**OP0214 Disturbed PD-L1 upregulation is characteristic of anergic B cells in SLE**

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**Background:** Prior studies on systemic lupus erythematosus (SLE) B cells reported an altered responsiveness to TLR9 stimulation, such as proliferation, cytokine production as well as indications for impaired T-B cell interaction [1]. The role of co-inhibitory and co-stimulatory (check-point) molecules in this setting has not been delineated in detail so far.

**Objectives:** Assess the expression of co-inhibitory (PD-1, PD-L1 and PD-L2) and co-stimulatory molecules (CD86 and CD40) by SLE B cells after in vitro stimulation and their potential contribution for B cell hyporesponsiveness in SLE.

**Methods:** PBMCs from 10 SLE patients and 10 healthy donors (HD) were stimulated with IL-2/IL-10, anti-CD40L (anti-CD40L) and CD86 (CD86) alone or in combination. Expression of PD-1, PD-L1, PD-L2 as well as CD86, CD40 on CD19+ or CD20+ B cell subsets after 48h stimulation was analyzed by FACS. CD71 was employed to measure proliferation of CD19+CD20+ B cells after 48 h stimulation.

**Results:** SLE B cells exhibited a substantially decreased upregulation of PD-L1 (p = 0.006) and CD86 (p = 0.018, Fig. 1) associated with significantly reduced B cell proliferation (p = 0.0039) after 48 h stimulation with CpG alone and in combination compared with HD. While TLR9 engagement in SLE B cells appeared to be abnormal, activation of CD40 resulted into a consistent upregulation of both, inhibitory and stimulatory molecules. PD-L1 was positively correlated with B cell proliferation (p = 0.003). Notably, the expression of PD-L1 and CD86 correlated inversely with Siglec-1, as surrogate marker for interferon signature (p < 0.001 and p = 0.0021 respectively). PD-L1 expression correlated inversely with cSLE-DAl (p = 0.0087).

**Disclosure of Interests:**


**Disclosure of Interests:** Ana-Luisa Stefanski: None declared, Annika Wiedemann: None declared, Karin Reiter: None declared, Andrea Lino: None declared, Thomas Dömer Grant/research support from: Eli Lilly, Janssen, Roche, UCB Pharma, Consultant for: Eli Lilly, Janssen, Roche, UCB Pharma, Speakers bureau: Eli Lilly, Janssen

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FRACURE liaison service: an opportunity for rheumatologists to focus on secondary fracture prevention

**OP0215**

**THE RISK OF FIRST AND SUBSEQUENT FRACTURES IN PATIENTS WITH RHEUMATOID ARTHRITIS IN THE UK**

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**Background:** Osteoporosis is a well-known complication of rheumatoid arthritis (RA) (1). NICE advises that health providers do not routinely assess fracture risk in people with RA aged under 50 years unless they have other major risk factors. However, studies suggest that young people with RA are at increased fracture risk even before age 50 years (2-4).

**Objectives:** To measure the incidence and risk of first and subsequent fracture in adults with RA aged under 50 in the UK.

**Methods:** A retrospective observational cohort study with matched control using data from Clinical Practice Research Datalink (CPRD) of adults >18 years with diagnosis of RA recorded from 1987 to 2016. Patients were followed from index date to the first fracture and subsequent fracture. Cases were adults with a new diagnosis of RA with at least one day of follow-up during the period. These cases were matched to 3 controls by age, sex, GP surgery and calendar year. The date of RA diagnosis date is the index date for cases and their matched controls. CPRD data has a high level of data validity in reporting of fractures but subsequent fractures may be recorded more than once. In this analysis when subsequent fractures were of the same body part the fracture had to occur at least 6 months after the previous fracture to be classified as a “subsequent fracture”. “First fracture” is the first fracture occurring after the index. “Subsequent fracture” is the fracture occurring after the first fracture. “Pre-index fracture” is a fracture that occurs before the index date.

**Results:** The characteristics of the study population are shown in Table 1.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Case (n=11,120)</th>
<th>Control (n=33,364)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age group 0-9</td>
<td>1.542 (13.87)</td>
<td>1.522 (13.95)</td>
</tr>
<tr>
<td>40-49</td>
<td>3.430 (30.85)</td>
<td>3.328 (30.40)</td>
</tr>
<tr>
<td>Gender (female)</td>
<td>6.148 (55.29)</td>
<td>6.019 (55.16)</td>
</tr>
<tr>
<td>Glucocorticoid prescription</td>
<td>8.232 (74.03)</td>
<td>8.207 (73.96)</td>
</tr>
<tr>
<td>Pre-index fracture</td>
<td>930 (7.46)</td>
<td>931 (7.11)</td>
</tr>
<tr>
<td>Index fracture</td>
<td>1,648 (14.82)</td>
<td>1,651 (14.27)</td>
</tr>
<tr>
<td>First fracture</td>
<td>954 (8.57)</td>
<td>955 (8.57)</td>
</tr>
<tr>
<td>Subsequent fracture</td>
<td>197 (1.77)</td>
<td>197 (1.77)</td>
</tr>
</tbody>
</table>

The incidence rate of first fracture for cases was 9.02 per 1000-person years (95% CI 8.46-9.61), for controls 6.85 per 1000-person years (95% CI 6.55-7.15). The incidence rate for subsequent fracture was 1.32 (95% CI 1.22o-1.426; p<0.00). The incidence rate of subsequent fracture was 1.78 (95% CI 1.55-2.02) per 1000-person years for cases and 1.17 (1.05-1.29) per 1000-person years for controls. The IRR for subsequent fracture was 1.53 (95% CI 1.283-1.833; p=0.00). A Cox’s proportional hazards model was used to estimate the hazard (or risk) of first and subsequent fracture for cases and controls stratified by age. Those with a pre-index fracture and with a glucocorticoid prescription prior to the index date had significantly higher hazard ratios for first and subsequent fracture. Women had a higher hazard ratio for first fracture.

**Conclusion:** This study shows that the incidence of fracture in patients with RA aged under 50 is significantly higher than in matched controls. This is true for the first fracture and for the subsequent fracture and is significantly higher for women in those under 50 for first fractures.

**REFERENCES:**


