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**PERIPHERAL T HELPER SUBSET PROFILING DIFFERS IN VARIOUS SUBSETS OF IDIOPATHIC INFLAMMATORY MYOSITIS**

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**Background:** There is a dearth of biomarkers in Idiopathic Inflammatory Myositis (IIM) to identify ongoing inflammation in the muscle and distinguish it from inactivity or damage.

**Objectives:** Since myositis is autoantibody mediated and tertiary lymphoid organogenesis (TLO) reported in the diseased muscles, we investigated peripheral blood T helper subset profiling as a reflection of ongoing muscle inflammation.

**Methods:** Twenty-six patients of IIM (ACR EULAR criteria) were compared with 15 healthy controls (HC) and 21 patients with sarcoidosis.

**Results:**

**Table 1. Baseline characteristics of patients with inflammatory myositis**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Demographic details (n, % or median, IQR)</th>
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<tbody>
<tr>
<td>Age</td>
<td>37±25.25 vs. 26±32</td>
</tr>
<tr>
<td>Gender(M:F)</td>
<td>5 vs. 21 vs. 12 vs. 3</td>
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<tr>
<td>Diagnosis</td>
<td>PM 3 vs. DM 15 vs. OM 4 vs. ASS 4</td>
</tr>
<tr>
<td>Disease course</td>
<td>Monocyclic 5 vs. Polycyclic 7 vs. Chronic continuous 1 vs. Undefined 13</td>
</tr>
<tr>
<td>Clinical Profile</td>
<td>Myositis 4 (15.3%) vs. ILD 5 (19.23%) vs. Rash 3 (11.53%) vs. Arthritis 6 (23%) vs. Other 16 (23.69%)</td>
</tr>
<tr>
<td>Disease duration (years)</td>
<td>1.3 ± 6.91</td>
</tr>
<tr>
<td>Disease activity</td>
<td>Active 12 [PM(1), OM(1), ASS(4), DM(5)] vs. Inactive 14 [PM(2), OM(2), ASS(0), DM(8)]</td>
</tr>
</tbody>
</table>

**Antinuclear Antibodies**

Positive Nuclear
- Speckled 9 (34.61%)
- Homogeneous 4 (15.38%)
- Nucleolar 1 (3.8%)
- Other 5 (19.23%)

Cytoplasmic 3 (11.53%)

Negative 4 (15.38%)

**Myositis Specific Antibodies**

Positive ARS 2 (7.69%)

Mi-2 3 (11.53%)

SAl-1 2 (7.69%)

NXP2 2 (7.69%)

MDA5 0

MAA 0

Ku 1 (3.8%)

dsDNA 0

U1RNP 0

Ro52 4 (15.38%)

Negative 12 (46.15%)

**Results:** All T helper subsets were higher in myositis as compared with healthy controls (Figure 1A a-d). Between various IIM subsets, polymyositis had higher Th1 and Treg cells (Figure 1B a, c) while Th17 and Th17.1 cells (c) were higher in Overlap Myositis (Figure 1B a, d) as compared with healthy controls. Patients with sarcoidosis had similar subset profiling as myositis. (Figure 5a-f)

Patients who were either arthritis or were positive for myositis specific autoantibodies had higher Th17.1 cells (Figure 3 a(iii) & b(iii)) than those negative for MSA. There was no difference in T cell profile between the various autoantibody subsets (Figure 4a-d).

There was no difference in subsets between active and inactive disease although active disease had lower Th1/Treg, Th17/Treg and Th17.1/Treg ratios.

**Conclusion:** T Helper cell subsets are distinct from HC but similar to sarcoidosis patients. However, they differ in various subsets of myositis, suggesting different pathogenic mechanisms are operative. Autoantibody positivity is associated with elevated Th17.1 population suggesting plasticity in TLO which needs to be explored further. However, T cell profiling cannot distinguish active from inactive disease limited predictive potential as a biomarker.

**Figure 1** A. Representative plot depicting all T helper subsets quantified were higher in myositis as compared with healthy controls1B: Representative plot comparing % cell subsets in various subsets of myositis with healthy controls showing that % Th1 cells (a) and Tregs (d) are highest in Polymyositis than controls while % Th17 (b) and % Th17.1 cells (c) are higher in Overlap Myositis.

**Figure 2** A. Comparisons between various phenotypic subsets suggest patients positive for MSA had higher Th17.1 cells (Figure 2A a(iii)) than those negative for MSA. Similarly, patients with arthritis had higher Th17.1 cells (Figure 2A b(iii)) than those negative for MSA. There was no difference in T cell profile between the various autoantibody subsets (Figure 2B).

**Figure 3** A. Positive plots depicting all T helper subsets quantified were higher in myositis as compared with healthy controls1B: Representative plot comparing % cell subsets in various subsets of myositis with healthy controls showing that % Th1 cells (a) and Tregs (d) are highest in Polymyositis than controls while % Th17 (b) and % Th17.1 cells (c) are higher in Overlap Myositis.

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**Disclosure of Interests:** None declared

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**ADAPTOR PROTEINS ACTIVATE FIBROBLASTS IN SYSTEMIC SCLEROSIS**

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