EPIEDEMOLOGICAL AND CLINICAL DIFFERENCES BETWEEN ANTI-MDAS PHENOTYPES: DATA FROM A LARGE COHORT (MEDRA5) STUDY

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Background: Idiopathic inflammatory myopathies are a heterogenous group of systemic autoimmune diseases. Several phenotypes have been linked to specific autoantibodies. Clinically amyopathic dermatomyositis with rapidly progressive interstitial lung disease, the most severe form of ILD, is associated with the anti-MDAS antibodies. However not all the patients with dermatomyositis and anti-MDAS positive antibodies develop this severe condition.

Objective: We aim to define different phenotypes from a large cohort of patients diagnosed with dermatomyositis who were positive to anti-MDAS antibodies.

Methods: We retrospectively analyzed the clinical and immunological data of 90 anti-MDAS patients (50 female, 55.6%, mean (SD) age at diagnosis 47 (15.4) yrs.) with dermatomyositis recruited from a multicenter register in Spain (MEDRAS) including 30 hospitals. All the patients fulfill de International Myositis Classification Criteria (IMCC). EULAR/ACR for dermatomyositis (score >90%). Anti-MDAS were detected by means of commercial immunoblot (EUROMMUN®). The chi-square test was used to assess the relationships between qualitative variables. The Kruskal-Wallis test was used to compared medians between groups.

Results: Sixty-six patients (73.3%) were diagnosed with clinically amyopathic dermatomyositis. Three different phenotypes linked with the anti-MDAS antibody were identified: (1) Hospital Vall de Hebron, Rheumatology, Barcelona, Spain; (2) Hospital Universitario Fundación Jiménez Díaz, Rheumatology, Madrid, Spain; (3) Hospital General de Granollers, Rheumatology, Granollers, Spain; (4) Hospital La Princesa, Rheumatology, Madrid, Spain; (5) Hospital de Bellvitge, Internal Medicine; Hospital de Llobregat, Barcelona; (6) Hospital Universitario Central de Asturias, Internal Medicine, Oviedo, Spain; (7) Hospital Universitario La Paz, Internal Medicine, Madrid, Spain; (8) Hospital Universitaria Mutua de Terrassa, Internal Medicine, Terrassa, Spain; (9) Hospital Universitario Basurto, Rheumatology, Bilbao, Spain; (10) Hospital Miguel Servet, Internal Medicine, Zaragoza, Spain; (11) Hospital Vall de Hebron, Internal Medicine, Barcelona, Spain.

Conclusion: Different phenotypes of patients positive to anti-MDAS were identified. The presence or not of ILD, or the different type (rapidly progressive or not) of ILD were the main feature that allow to differentiate these phenotypes, which are relevant in clinical practice.

REFERENCES:

Acknowledgements: This work was supported by the Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq) 303.379/2018-9, Fundação de Amparo à Pesquisa do Estado de São Paulo (FAPESP) 2019/11776-6, Facul- dade de Medicina da USP/SP to SKS.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.3723

CO PRESCRIPTION OF ANTI-ACID THERAPY REDUCES THE BIO AVAILABILITY OF MYCOPHENOLATE MOFETIL IN SYSTEMIC SCLEROSIS PATIENTS

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Background: Mycophenolate mofetil (MMF) is an effective treatment option for systemic sclerosis (SSC). However, many patients require co-administration of proton pump inhibitors (PPI) or H2 receptor blockers (HRB) because of significant gastrointestinal manifestations in SSC. Co-treatment with PPI or HRB have shown to be associated with reduced drug exposure in patients with SSC. We evaluated the drug concentration of MMF over 12 hours of exposure and assessed the impact of ranitidine and PPI in twenty patients with SSC.

Objectives: To assess the effect of esomeprazole or ranitidine on the bioavailability of MMF in SSC patients who are on a stable dose of MMF.

Methods: Twenty SSC patients, who were on a stable dose of MMF (1.5-3 g) for the past 3 months were selected for the study after obtaining informed written consent. All patients were given either MMF (without PPI or HRB), MMF + esomeprazole, MMF + ranitidine for one month each. At the end of each month, EDTA plasma samples were collected at various time points including 0, 1/2, 1, 1½, 2, 2½, 3, 4, 5, 6, 8 and 12 hours following drug administration to determine the 12-hour area under curve (AUC) of myco- phenolic acid (MPA) levels. Estimation of MPA levels was carried out using reverse phase high performance liquid chromatography (HPLC). Total gastrointestinal score was calculated at the end of each month using UCLA Scherderma Clinical Trial Consortium GIT 2.0 scoring. To compare the mean AUC, linear mixed effect model was fit by considering treatment as the fixed effect and subject as the random effect. MMF was set as the reference treatment for the other three treatments and these were analysed together using Linear mixed effect model.

Results: All patients were females with mean age of 45 years. Addition of either ranitidine or esomeprazole significantly reduced the mean AUC and C max of the MMF over 12-hour time period. On the other hand, PPI or HRB helped in reduction of the total GI score at the end of 1 month. Details of pharmacokinetics are depicted in the table 1

REFERENCES:


Acknowledgements: List of contributors of MEDRAS group: Aguilar-Jacaré (Internal Medicine, Hospital Costa del Sol, Marbella), Carrión-Barberá I (Rheumatology, Hospital del Mar, Barcelona), Cobo-Ibáñez T (Rheumatology, Hospital Infanta Sofía, San Sebastián de los Reyes), de Escalante-Yangüela B (Internal Medicine, Hospital Clínico Lozano Blesa, Zaragoza), Fonseca-Aizpuru EM (Internal Medicine, Hospital de Cádiz, Jerez de la Frontera), González-Cubilló L (Intensive Medicine, Hospital Universitario de Cruces, Barakaldo), González-Gay MA (Rheumatology, Hospital Marqués de Valdecilla, Santander), Prieto-González S (Internal Medicine, Hospital Clínico, Barcelona), Ruiz-Román A (Rheumatology, Hospital Universitario Virgen del Rocío, Sevilla), Calero-Paniagua I (Internal Medicine, Hospital Virgen de la Luz, Cuenca), Callejas-Rubio JL (Internal Medicine, Hospital Clínico San Cecilio, Granada), Gil-Vila A (Internal Medicine, Hospital Vall d’Hebron, Barcelona), de Miguel-Campo B (Internal Medicine, Hospital Doce de Octubre, Madrid), García-Sevilla R (Pneumology, Hospital General Universitario de Alicante, Alicante), Iriarte-Fuster A (Internal Medicine, Hospital de Bellvitge, Hospital de Llobregat), Jovani-Casano V (Rheumatology, Hospital General Universitario de Alicante, Alicante), Lozano-Rivas N (Rheumatology, Hospital Virgen de la Arrixaca, Murcia), Martín-Gascon M (Internal Medicine, Hospital Morales Meseguer, Murcia), Martínez-González O (Rheumatology, Hospital Universitario de Salamanca, Salamanca), Monteagudo-Jiménez M (Internal Medicine, Hospital Parc Taulí, Sabadell), Mora-Ortega GM (Rheumatology, Hospital Universitario Infanta Sofia, San Sebastián de los Reyes), Moral-Moral Pedro (Internal Medicine, Hospital Universitari I Politecnic La Fe, Valencia), Pérez-Dé Pedro I (Internna Medicine, Hospital Regional Universi- tario de Málaga, Málaga), Picazo-Talaver A (Rheumatology, Hospital del Sureste, Madrid), Rubio-Rivas M (Internal Medicine, Hospital de Bellvitge, Hospital de Llobregat)

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.3837
**IMPLICATION IN SYSTEMIC SCLEROSIS**

**MACROVASCULAR DYSFUNCTION AND ITS CLINICAL IMPLICATION IN SYSTEMIC SCLEROSIS**

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**Background:** Even though microvascular dysfunction has been implicated in pathogenesis of scleroderma (SSc), there is minimal evidence to suggest presence of macrovascular dysfunction. The clinical implication of macrovascular dysfunction in SSc is unknown. Moreover, data on the correlation between dysfunction in small and large blood vessel is inconclusive. [1-2]

**Objectives:** To study the correlation between macrovascular dysfunction as assessed by percent change in flow mediated vasodilation (FMD) of brachial artery and microvascular dysfunction as assessed by nail fold capillaroscopy (NFC) findings in SSC. To assess the clinical impact of macrovascular dysfunction.

**Methods:** This cross-sectional comparative study enrolled patients with SSc and age and gender-matched healthy controls. FMD change was calculated using standard USG probe of 5 to 6 MHz in right brachial diameter from the average of 3 consecutive end diastolic frames. NFC was performed using portable nail fold capillaroscopy microscope at 800X magnification. Clinical features of SSc were characterized by an autoimmune disorder with vasculopathy that leads to an excess of collagen and other extracellular matrix proteins deposition. This process results in progressive fibrotic and vascular damage of skin and visceral organs.

**Results:** This study enrolled 59 SSc patients including 29 (49.2%) diffuse, 20 (33.9%) limited and 10 (16.9%) overlap SSc patients and 64 age and gender-matched healthy controls. FMD change was calculated using standard USG probe of 5 to 6 MHz in right brachial diameter from the average of 3 consecutive end diastolic frames. NFC was performed using portable nail fold capillaroscopy microscope at 800X magnification. Clinical features of SSC were compared between SSc patients with and without macrovascular dysfunction.

**Discussion:** Macrovascular dysfunction in SSC is substantial and it seems to be independent of the microvascular dysfunction. The clinical implications of macrovascular dysfunction are yet to be identified.

**REFERENCES:**


**POS0890**

**MACROVASCULAR DYSFUNCTION AND ITS CLINICAL IMPLICATION IN SYSTEMIC SCLEROSIS**

**Figure 1.** Scatter plot showing correlation between FMD percentage change and average capillary density. (r= 0.116) (P-value - 0.381)

**Table 1.** Comparison of various parameters between the SSc patients with healthy controls.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Frequency (percentage)/median (interquartile range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age in years</td>
<td>38 (27-46) 36.5 (28.25-42)</td>
</tr>
<tr>
<td>Gender</td>
<td>Male 05 (5.1%) Female 56 (95.1%)</td>
</tr>
<tr>
<td>FMD findings</td>
<td>FMD % change* 4.54 (3.13-8.82) 10.30 (8.33-13.16)</td>
</tr>
<tr>
<td>NFC findings</td>
<td>Number of capillaries* 51 (38-63) 121 (113-128)</td>
</tr>
<tr>
<td>FMD change and capillary density</td>
<td>Average capillary density* 3.19 (2.38-3.34) 7.56 (706.8)</td>
</tr>
<tr>
<td>Abnormal (%)</td>
<td>36.11 (14.03-55.26) 0</td>
</tr>
<tr>
<td>Avascular area (%)</td>
<td>50 (3125-75) 0</td>
</tr>
</tbody>
</table>

**Table 1.** Pharmacokinetics and Gl score with MMF in combination with PPI / HRB

<table>
<thead>
<tr>
<th>Parameter</th>
<th>MMF</th>
<th>MMF + R</th>
<th>MMF + E</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>AUC</td>
<td>6797 (62.73, 73.20)</td>
<td>53.04 (44.68, 61.27)</td>
<td>45.69 (41.10, 50.28)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>T- MAX</td>
<td>42.00 (33.60, 50.40)</td>
<td>46.50 (32.48, 60.52)</td>
<td>79.50 (58.99, 100.01)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>C-MAX</td>
<td>29.61 (26.74, 32.46)</td>
<td>15.14 (11.32, 18.97)</td>
<td>12.62 (10.58, 14.66)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Mean Gl score</td>
<td>0.28 (0.15,0.40)</td>
<td>0.19 (0.09,0.30)</td>
<td>0.14 (0.06,0.23)</td>
<td>0.009</td>
</tr>
<tr>
<td>Mean (95% CI)</td>
<td>29.61 (26.74, 32.46)</td>
<td>15.14 (11.32, 18.97)</td>
<td>12.62 (10.58, 14.66)</td>
<td>&lt;0.001*</td>
</tr>
</tbody>
</table>

**References:**


**POS0891**

**IMPROVED SURVIVAL IN SYSTEMIC SCLEROSIS PATIENTS DURING LAST DECADE: CURRENT FINDINGS AND COMPARISON WITH DIFFERENT PREVIOUS ITALIAN COHORTS**

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**Background:** Systemic Sclerosis (SSSc) is a chronic rheumatic disease characterized by an autoimmune disorder with vasculopathy that leads to an excess in collagen and other extracellular matrix proteins deposition. This process results in progressive fibrotic and vascular damage of skin and visceral organs.

**Table 1.** Comparison of various parameters between the SSc patients with healthy controls.

**Disclosure of Interests:** None declared

**DOI:** 10.1136/annrheumdis-2021-eular.3916

**Disclosure of Interests:** None declared

**DOI:** 10.1136/annrheumdis-2021-eular.3988

**Figure 1.** Scatter plot showing correlation between FMD percentage change and average capillary density. (r= 0.116) (P-value - 0.381)

**Disclosures:** None declared

**DOI:** 10.1136/annrheumdis-2021-eular.3988