Table 1. Clinical and immunological characteristics of patients who died and patients who lived

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Dead (n=19)</th>
<th>Alive (n=66)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female sex, n (%)</td>
<td>17 (89.5)</td>
<td>55 (83.3)</td>
<td>0.594</td>
</tr>
<tr>
<td>Age at diagnosis (years), mean (SD)</td>
<td>56.9 (13.7)</td>
<td>50.4 (13.6)</td>
<td>0.076</td>
</tr>
<tr>
<td>Time since diagnosis (years), mean (SD)</td>
<td>11.6 (7.3)</td>
<td>14.2 (9.2)</td>
<td>0.215</td>
</tr>
<tr>
<td>iSSc, n (%)</td>
<td>6 (33.3)</td>
<td>35 (53.03)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>dSSc, n (%)</td>
<td>12 (66.7)</td>
<td>10 (15.1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Digital ulcers, n (%)</td>
<td>9 (47.4)</td>
<td>33 (50.0)</td>
<td>0.748</td>
</tr>
<tr>
<td>Calcification, n (%)</td>
<td>1 (5.3)</td>
<td>16 (24.2)</td>
<td>0.061</td>
</tr>
<tr>
<td>Telangiectasias, n (%)</td>
<td>15 (78.9)</td>
<td>56 (84.8)</td>
<td>0.184</td>
</tr>
<tr>
<td>ILD, n (%)</td>
<td>15 (78.9)</td>
<td>56 (84.8)</td>
<td>0.021</td>
</tr>
<tr>
<td>FEV1 at diagnosis of ILD, mean (SD)</td>
<td>78.3 (19.3)</td>
<td>78.7 (17.7)</td>
<td>0.955</td>
</tr>
<tr>
<td>FVC at diagnosis of ILD, mean (SD)</td>
<td>65.2 (13.1)</td>
<td>78.1 (20.7)</td>
<td>0.301</td>
</tr>
<tr>
<td>DLCO at diagnosis of ILD, mean (SD)</td>
<td>56.9 (17.3)</td>
<td>63.9 (16.7)</td>
<td>0.043</td>
</tr>
<tr>
<td>Pulmonary hypertension, n (%)</td>
<td>11 (57.9)</td>
<td>14 (21.9)</td>
<td>0.002</td>
</tr>
<tr>
<td>sPAP (mmHg), mean (SD)</td>
<td>49.2 (24.7)</td>
<td>30.2 (8.2)</td>
<td>0.009</td>
</tr>
<tr>
<td>Gastrintestinal involvement, n (%)</td>
<td>13 (68.4)</td>
<td>30 (45.4)</td>
<td>0.125</td>
</tr>
<tr>
<td>Cardiac involvement, n (%)</td>
<td>3 (15.8)</td>
<td>11 (16.7)</td>
<td>0.907</td>
</tr>
<tr>
<td>Muscle involvement, n (%)</td>
<td>1 (5.3)</td>
<td>2 (3.03)</td>
<td>0.851</td>
</tr>
<tr>
<td>Arthritis or arthralgia, n (%)</td>
<td>5 (26.3)</td>
<td>23 (34.8)</td>
<td>0.436</td>
</tr>
<tr>
<td>Renal crisis, n (%)</td>
<td>0</td>
<td>2 (3.03)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Cancer, n (%)</td>
<td>5 (26.3)</td>
<td>2 (3.03)</td>
<td>0.001</td>
</tr>
<tr>
<td>Positive ACA, n (%)</td>
<td>5 (26.3)</td>
<td>35 (53.03)</td>
<td>0.034</td>
</tr>
<tr>
<td>Positive ATA, n (%)</td>
<td>10 (52.6)</td>
<td>9 (13.6)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>


Conclusion: Survival at 10 years was greater than in the study cohort. The main causes of death were ILD, pulmonary hypertension and cancer. The main factors associated with mortality were proximal skin thickening, older age at diagnosis, and lower forced vital capacity.

Disclosure of Interests: None declared.

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AB0434
VITAMIN D IN SYSTEMIC SCLEROSIS PATIENTS
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Teramo, Italy.

Background: Systemic Sclerosis (SSc) is a generalized and systemic autoim-
mune disease that affects the connective tissue of the skin and internal organs,
especially kidneys, heart and lungs [1].

Objectives: Numerous data from recent literature confirm the regulatory action
of vitamin D on the immune system and, therefore, how a deficit of this micronu-
trient can lead to alterations in the immune induction, as is known to happen in
many allergic and autoimmune diseases [2]. We studied the association between
vitamin D levels and SSc, evaluating their correlation with the characteristic man-
ifestations of the pathology.

Methods: We dosed the serum levels of 25 hydroxy-vitamin D in 42 patients with
SSc (average age 64.63 +/- 7.233) and 40 healthy controls comparable for sex and
age. The diagnosis of SSc was formulated in accordance to 2013 ACR/EULAR
criteria. None of the subjects involved in the study took vitamin D products.

Results: Patients' vitamin D levels (26.22 +/- 9.82 ng/ml), although they tended to
be lower than controls (27.80 +/- 16.53 ng/ml), showed no significant decrease. In patients with pulmonary fibrosis, vitamin D levels were 23.28 +/- 12.30 lower than in patients with trophic ulcers and compared to patients without complications 26.07 +/- 9.92, although with not statistically significant values. No statistically
significant difference was found between vitamin D levels in patients with trophic
ulcers compared to controls without complications.

Conclusion: According to the studies in the literature, in our sample, vitamin D
deficiency was therefore greater in patients with SSc, especially with pulmonary
fibrosis, than in controls [3,4]. Vitamin D levels in diffused-type SSc patients were
significantly lower than those in limited-type SSc patients. Further studies are
needed to clarify the role that vitamin D deficiency plays in SSc, but lower vitamin D
levels in these patients may suggest the need to monitor blood levels of vitamin D
and supplement it appropriately.

REFERENCES:
[1] De Martinis M, Ciccarelli F, Sirufo MM, Ginaldi L. An overview of envi-
[19. PMID: 26610037.
[20] Capillaroscopy (p=0.684), disease progression on capillaroscopy (p=0.781),
capilloropic scoring (p=0.92) and CV risk score (p=0.98) were not predictive
factors of MACE.

Disclosure of Interests: None declared
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AB0436
OUTCOMES OF DOSE-REDUCTION OR DISCONTINUATION
OF TOCILIZUMAB IN PATIENTS WITH EARLY DIFFUSE
CUTANEOUS SYSTEMIC SCLEROSIS
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Background: Potential efficacy and favorable safety profiles of tocilizumab (TCZ)
have been demonstrated in patients with diffuse cutaneous systemic sclerosis
dcsScC [1, 2]. However, clinical outcomes after dose-reduction or discontinua-
tion of TCZ due to an improvement of skin thickness remain unclear.

Objectives: To investigate the clinical outcomes after dose-reduction or discon-
 tinuation of TCZ in patients with dcSSc in a real-world setting.

Methods: This is a single-center, retrospective, observational study using a data-
based of consecutive SSc patients who visited our center between April 2014 and
October 2020. For this study, we selected eligible patients from the database based
on the following criteria: patients who (i) fulfilled the ACR/EULAR classification cri-
teria, (ii) were classified as having dcSSc, (iii) had been treated with TCZ for at
least 6 months, and (iv) were follow-up >6 months after TCZ introduction. Clinical
information including demographic and clinical characteristics at TCZ introduction;
cosing, administration route, and adherence of TCZ; and serial clinical parameters