Letters to the Editors

Myocardial fibrosis in systemic sclerosis assessed by cardiac magnetic resonance is associated with vascular endothelial growth factor expression

Sir,

The hallmarks of systemic sclerosis (SSc) are endothelial dysfunction and fibrosis of the skin and the internal organs, including the heart. Primary cardiac involvement related to SSc is more related to fibrosis and its complications (1). Recurrent episodes of vasospasm with ischaemia and reperfusion cause abnormal myocardial perfusion with subsequent fibrosis (2). In SSc vascular damage and chronic tissue hypoxia promote angiogenesis with production of pro-angiogenic factors such as vascular endothelial growth factor (VEGF) (3). Endomyocardial biopsy is an invasive procedure used to diagnose myocardial fibrosis. To date, cardiac magnetic resonance (CMR) is employed in SSc to detect myocardial fibrosis showing patchy fibrosis distributed in both ventricles (4). As confirmed by a histopathological study, myocardial fibrosis can be detected through late gadolinium enhancement (LGE) on CMR with the same degree of reliability provided by myocardial biopsy (5).

In this pilot study we aimed to evaluate myocardial fibrosis in SSc using CMR with LGE and VEGF expression.

The protocol, according to Declaration of Helsinki, was approved by local Ethics Committee. Twenty-eight SSc patients [20 women, aged 40 (36-48) years] were enrolled. Exclusion criteria were coronary artery disease, congestive heart failure, left ventricular dysfunction, pulmonary hypertension, valvular abnormalities. Serum VEGF levels were determined in SSc patients by commercial ELISA kit (Human VEGF, Quantikine ELISA, R&D Systems, Minneapolis MN), with a sensitivity of 9 pg/ml and an assay range of 31.2-2000 pg/ml.

CMR imaging was performed with a 1.5-T unit (Magnetom Avanto; Siemens Healthcare GmbH, Erlangen, Germany) using an 8-channel phased-array coil and vectocardiogram triggering. LGE imaging was performed between 10 and 15 min after the second bolus injection of contrast agent using a segmented TI-weighted phase-sensitive inversion-recovery pulse. Table I shows clinical and CMR features. Serum median value of VEGF was 187 pg/ml (128-251).

LVEF (n, %) 17 (60.7)
Focal myocardial oedema (n, %) 4 (14.3)
Pericardial effusion (n, %) 5 (17.8)
ECV (%) (mean ± SD) 26.5 (25.4-29)

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Table I. Anthropometric, clinical and cardiac magnetic resonance features in systemic sclerosis patients.

<table>
<thead>
<tr>
<th>Feature</th>
<th>Value (Mean ± SD)</th>
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<tbody>
<tr>
<td>Age, years</td>
<td>40 (36-48)</td>
</tr>
<tr>
<td>Female, n and %</td>
<td>20 (71.5%)</td>
</tr>
<tr>
<td>Disease duration, years</td>
<td>8 (6-11)</td>
</tr>
<tr>
<td>LDLcSSc/dcSSc</td>
<td>10/18</td>
</tr>
<tr>
<td>nKSS</td>
<td>14 (12-20)</td>
</tr>
<tr>
<td>DAI</td>
<td>2 (1.5-3.75)</td>
</tr>
<tr>
<td>Digital ulcers history, n and %</td>
<td>18 (64.3%)</td>
</tr>
<tr>
<td>Capillaroscopic pattern</td>
<td>(early/active/late), n</td>
</tr>
<tr>
<td>VEGF (pg/ml)</td>
<td>187 (128-251)</td>
</tr>
</tbody>
</table>

LVEF shows a positive correlation with VEGF. We can suppose that in primary cardiac involvement related to SSc, microvascular damage stimulates impaired angiogenesis. The defect in the vascular repair and new blood vessels formation favours myocardial fibrosis.

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References