Venous thromboembolic events in systemic vasculitis

In a population-based study published in the *Annals of Rheumatic Disease*, Aviña-Zubieta *et al* showed that there is almost 2.5-fold increase in risk of venous thromboembolism (VTE) in patients with giant cell arteritis (GCA) compared with that in the general population. Higher incidence of VTE is apparently not unique for GCA as a similar increase in risk of deep vein thrombosis (DVT) and pulmonary embolism was recently found in a meta-analysis of studies in patients with different inflammatory rheumatic diseases. The frequency of VTE (around 8%) in patients with ANCA-associated vasculitides (eg, hypercoagulability, endothelial damage, stasis of blood, etc) and/or high-dose glucocorticoids to the development of VTE. Our data agree with the authors that the risk of VTE should not be applied to all patients with GCA or ANCA-associated vasculitides. We cannot recommend routine anticoagulation for all patients with GCA or ANCA-associated vasculitides. We agree with the authors that the risk of VTE should not be ignored in patients with GCA and other vasculitides. And we need more studies to evaluate the role of vasculitis-associated thrombogenic risk factors and to develop a strategy of screening to identify patients who really require prolonged anticoagulation.

What is a cost-effective strategy of prevention of VTE in systemic vasculitides? Using compression ultrasonound in a pilot study, we have found asymptomatic DVT in four (9.5%) of 42 patients with ANCA-associated vasculitides. Therefore, its prevalence may be higher than expected and can justify wider screening though obviously we need more data. Low-dose aspirin is frequently used in patients with GCA to prevent ischaemic events. It should be probably administered more often in patients with ANCA-associated vasculitides as in our previous study more than half of 102 patients with granulomatosis with polyangiitis had high or very high predicted 10-year risk of fatal cardiovascular diseases calculated using SCORE charts. Aspirin significantly reduces the risk of arterial thrombosis, but it is less effective in the prevention of VTE and, therefore, cannot replace anticoagulants. Aviña-Zubieta *et al* suggested anticoagulation in a high-risk GCA population. But who is in high risk? All patients? In our cohort, there were no fatal VTE events. Therefore, we cannot recommend routine anticoagulation for all patients with GCA or ANCA-associated vasculitides. We agree with the authors that the risk of VTE should not be ignored in patients with GCA and other vasculitides. And we need more studies to evaluate the role of vasculitis-associated thrombogenic risk factors and to develop a strategy of screening to identify patients who really require prolonged anticoagulation.

**Table 1** Characteristic of patients with VTE events

<table>
<thead>
<tr>
<th>VTE, n (%)</th>
<th>GPA (n=243)</th>
<th>MPA (n=45)</th>
<th>EGPA (n=69)</th>
<th>Total (n=357)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men, n</td>
<td>20 (8.2)</td>
<td>3 (6.7)</td>
<td>7 (10.1)</td>
<td>30 (8.4)</td>
</tr>
<tr>
<td>Median age, year (range)</td>
<td>51 (25–78)</td>
<td>49 (46–64)</td>
<td>53 (30–65)</td>
<td>52 (25–78)</td>
</tr>
<tr>
<td>Traditional VTE risk factors*</td>
<td>5</td>
<td>1</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>VTE events</td>
<td>14</td>
<td>2</td>
<td>5</td>
<td>21</td>
</tr>
<tr>
<td>DVT</td>
<td>12</td>
<td>2</td>
<td>5</td>
<td>19</td>
</tr>
<tr>
<td>DVT+PE</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Other vein thrombosis†</td>
<td>8</td>
<td>1</td>
<td>2</td>
<td>11</td>
</tr>
</tbody>
</table>

*Metabolic syndrome, type 2 diabetes mellitus, multiple intravenous injections, chronic venous insufficiency.
†Orbital vein (2), jugular vein (1), renal vein (1), testicular vein (1), superficial veins (6).
DVT, deep vein thrombosis; EGPA, eosinophilic granulomatosis with polyangiitis; GPA, granulomatosis with polyangiitis; MPA, microscopic polyangiitis; PE, pulmonary embolism; VTE, venous thromboembolism.
REFERENCES


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