Influence of Inflammatory Variables on Intima-Media Thickness

To the Editor:

The increase of carotid intima-media thickness (IMT) is a validated marker for atherosclerotic progression,1 and it has been related to a higher incidence of cardiovascular events.2 However, data focusing on its determinants are scarce. Lorenz et al3 reported the relationship between C-reactive protein (CRP) and the progression of carotid IMT in a population-based study.

We have studied the influence of inflammatory markers in the development of atherosclerosis (baseline and IMT increase/decrease) in Human Immunodeficiency Virus (HIV)–infected patients (n=141).4,5 We performed a standardized protocol for the acquisition of IMT (far wall IMT of common, bulb and internal portions of both carotid arteries, and both common femoral arteries). The same protocol was applied in the first (baseline)4 and in the second exams (mean follow-up of 2.5 years).3 Several inflammatory-related variables (serum CRP [Quantex hs-CRP kit, Biokit] and monocyte chemoattractant protein 1 concentration) in addition to inflammatory-related genetic polymorphisms (monocyte chemoattractant protein 1-2518G, SDF-1–3′A, CX3CR-1 249I) were determined.

We did not find any significant association between CRP concentrations and baseline, follow-up, and changes in IMT during the follow-up period (ANOVA, Table). We have also analyzed data from carotid and femoral arteries separately, and we did not find any significant relationship between CRP and IMT. Furthermore, a subset of these patients fulfilled the criteria for lipodystrophy (n=43, characterized by adipose tissue redistribution and higher IMT values).6 In those patients, CRP concentrations did not exert any significant influence on carotid IMT values.

We conclude that CRP is not a useful marker for atherosclerosis in a population with active inflammatory processes (HIV-infected people with apparently higher CRP concentrations than in the general population). CRP has been largely studied in atherosclerosis in patients infected with HIV with inflammation is influenced by a mutant monocyte chemoattractant protein 1 allele. Circulation. 2004;110:2204–2209.


Table. Characteristics of HIV-Infected Participants in the Study According to Quartiles of CRP

<table>
<thead>
<tr>
<th></th>
<th>First Quartile, n=32</th>
<th>Second Quartile, n=34</th>
<th>Third Quartile, n=37</th>
<th>Fourth Quartile, n=34</th>
<th>P Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>39.7 (7.01)</td>
<td>37.5 (8.1)</td>
<td>39.8 (6.5)</td>
<td>38.4 (7.5)</td>
<td>0.5</td>
</tr>
<tr>
<td>CRP, mg/L</td>
<td>0.55 (0.28)</td>
<td>1.54 (0.33)</td>
<td>3.25 (0.68)</td>
<td>9.96 (5.22)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Baseline IMT, mm</td>
<td>0.77 (0.18)</td>
<td>0.70 (0.16)</td>
<td>0.74 (0.20)</td>
<td>0.77 (0.17)</td>
<td>0.39</td>
</tr>
<tr>
<td>Follow-up IMT, mm</td>
<td>0.84 (0.16)</td>
<td>0.82 (0.12)</td>
<td>0.84 (0.18)</td>
<td>0.84 (0.12)</td>
<td>0.97</td>
</tr>
<tr>
<td>ΔIMT, mm</td>
<td>0.07 (0.21)</td>
<td>0.11 (0.15)</td>
<td>0.10 (0.23)</td>
<td>0.06 (0.13)</td>
<td>0.64</td>
</tr>
</tbody>
</table>

Missing values, n=4. ΔIMT=IMT follow-up–IMT baseline.


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*Stroke*. 2008;39:e16; originally published online December 6, 2007;
doi: 10.1161/STROKEAHA.107.493452

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

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World Wide Web at:
http://stroke.ahajournals.org/content/39/1/e16

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