Chlamydia pneumoniae in Coronary Artery Disease

To the Editor:

In a recent issue of Circulation, Anderson et al.1 reported the 6-month results of the ACADEMIC trial. In this interesting study, the authors intended to answer the question whether Chlamydia pneumoniae plays a critical role in coronary artery disease, in order to justify antibiotic therapy.

Beyond the final results of this work, the methodological approach used deserves comment. First, the investigators based the sample size and the clinical event rates estimates on prior data from a small study that used the same compound.2 For this purpose, the authors did not include unstable or subacute cases, but stable coronary patients. Similarly, the British study selected postmyocardial infarction patients in the quiescent phase, thus making the present study statistically underpowered, as stated in the editorial comment by Dr Grayston.3

Second, the authors analyzed some particular and common markers of inflammation, because infection increases its plasmatic levels. These markers decreased at 6 months but interestingly not at 3 months. It is hard to explain why these markers were practically neutral at 90 days, when 39 patients experienced new clinical infections over the 3-month treatment period. Furthermore, the authors compared their study with our ROXIS trial, stating that we randomized patients with a poor characterization. At the present time, the only study conducted in uniformly unstable patients is ROXIS, in which we clearly defined the entry criteria to select a standard population with acute coronary events, as shown in the original article.4 In the final report of the study,5 we showed that the beneficial effect was sustained until 90 days. Beyond this point, the P value is no longer statistically significant. The potential reasons were presented in March 1998 (at the meeting of the American College of Cardiology) and published elsewhere.5

Finally, the authors speculate in their conclusion that the ACADEMIC trial tested C pneumoniae as an additional risk factor. The authors should clarify how a marker of chronic infection may be regarded as a risk factor, in light of the sample size, the clinical event rates estimated, the serology cutoff point at 1:16, and the immunological basis of this and other infective hypotheses.6

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