Is it only Inflammation or Infection as well?

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To the Editor,

We would like to congratulate the authors for the publication of their article in this Journal (Arq Bras Cardiol 2009; 92 (6): 439-45)[1].

Our study group in coronariopathies believes that the acute coronary syndrome (ACS) is a systemic inflammatory condition. However, we are not convinced that the etiology of the inflammatory process is infectious[12-5-4].

We hypothesize that the alterations in the inflammatory markers of ACS occur due to the underlying inflammatory process and that the activation of the immunological system can result in a transient increase in serum antibody titers.

Moreover, regarding the diagnosis of an infectious entity, we believe in the importance of the association of the serology and the clinical picture, considering that the isolated use of the serological method can yield false-positive results, including the cases where there is laboratory error. Therefore, we believe that the probability of a high antibody titer level is higher than the actual risk of having an infection.

Finally, we would like the authors' opinions on these observations.

Key words

Chlamydophila pneumoniae; mycoplasma pneumoniae; coronary disease; inflammation, infection.

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Answer to letter to the editor

In response to the Letter to the Editor on the article published at Arq Bras Cardiol 2009; 92 (6): 436-45, "Prevalence of *Chlamydia pneumoniae* and *Mycoplasma pneumoniae* in different forms of Coronary Disease", we justify:

"It seems unlikely that the activation of the immune system without the specific participation of the infectious agent would be able to activate memory cells, which would lead to the increase in anti-Mp and anti-Cp antibodies. In other words, our hypothesis is that the infection is the starting point of the activation of the inflammatory process. We did not think of primary infection by

Cp and Mp, but rather of chronic infection, which might explain the lack of clinical data on these infections. Regarding the methodology used to evaluate the agents, we agree that there are many limitations. That is the reason why we chose the immunofluorescence method, which has the advantage of being more specific than the ELISA technique, to mention just

one example, and therefore, has a lower chance of yielding biological false-positive results.

In summary, what we demonstrated in our study was a clear elevation in the Ac level (more evident in the group with unstable myocardial ischemic syndrome and ST-segment elevation), which would be only justified by the presence of specific infectious agents at the lesion site. On the other hand, such observation does not in any way decrease the significance of the inflammatory process itself, certainly very important in triggering the acute ischemic event."