References


Seroepidemiology in Chlamydia pneumoniae — atherosclerosis association

See page 301, doi: 10.1053/euhj.2001.2778 for the article to which this Editorial refers

The association of Chlamydia pneumoniae with atherosclerosis has been detected in a seroepidemiological study[1]. Most patients with acute myocardial infarctions had a seroresponse against a chlamydial lipopolysaccharide epitope. In addition, the patients with chronic coronary heart disease had elevated titres of antibodies against the pathogen. The latter observation has been verified in numerous cross-sectional and even in some prospective studies[3]. However, seroepidemiology is not able to prove or disprove a causal association. The final verification of the association, suggesting a possible causal role, was to demonstrate the agent in atherosclerotic lesions[3]. This finding has been repeatedly verified by others[4].

To show the possible presence and current activity of the pathogen simply by measuring serum antibodies would offer a rapid and easy diagnostic tool when compared to the difficulty of direct demonstration of the agent from vascular tissue. Unfortunately, it has been shown repeatedly that this is not the case. It is extremely rare to avoid contamination by the agent and individual antibody responses vary enormously. Persons without measurable antibodies can show cell-mediated immunity to the agent[5] indicating that they have also been exposed to C. pneumoniae. Moreover, age and epidemiological situation can affect antibody prevalence in controls[6]. In the course of our studies, the first patient material[1] was collected during an inter-epidemic period and consisted of ‘young’ patients in order to avoid serological scars. During collection of the second material, there was an epidemic of C. pneumoniae infection, and IgG and IgA prevalences in controls were boosted by reinfections. No statistically significant differences in IgG and IgA prevalences between patients and controls was demonstrated[7] (and Leinonen, unpublished). Moreover, by 1993 it was evident that in some patients with C. pneumoniae in their atherosclerotic plaques no antibodies were demonstrable at the microimmunofluorescence test[8].

In this issue Tavendale et al. demonstrate that C. pneumoniae antibody levels cannot foretell who is going to be attacked by a cardiac event in future years[9]. The authors used a new, recently developed ELISA method. Use of these new methods is deceptively easy when compared to the classic micro-
immunofluorescence test, which has been mastered only by a few[10]. However, new tests have been developed for the diagnosis of acute infections and their applicability to chronic infections is an open question. It has been suggested that IgA and precipitated or trapped immunocomplexes possess a better predictive value than IgG antibodies[11,12]. Other than IgA, Tavendale et al. also measured immune complexes in sera with the conglutinin method. Both tests were unable to predict events in years to come[9].

In the future, e.g., circulating white blood cells, is lacking and in-house tests are variable in their results[13]. I n t h e from, e.g., circulating white blood cells, is lacking and in-house tests are variable in their results[13]. In the future it is to be hoped that we will find diagnostic markers circulating in the blood which will indicate the amount of C. pneumoniae activity in the body and, perhaps in future intervention studies, how well we have succeeded in eradicating the agent from vascular lesions. So far we only have non-specific markers of inflammation or autoimmunity, which, in combination with C. pneumoniae markers, may be better predictors of future outcome[14,15].

P. SAIKKU
Department of Medical Microbiology,
University of Oulu,
Oulu, Finland

References


Moving beyond unfractionated heparin for acute coronary syndromes: Xeno’s Paradox revisited

See page 308, doi:10.1053/euhj.2001.2779 for the article to which this Editorial refers

If we’ve learned anything at all from the numerous clinical trials performed in recent years in patients with acute coronary syndromes, it is that there are a lot of things that can improve clinical outcomes in these patients. Low molecular weight heparins, direct thrombin inhibitors, thienopyridines, GP IIb/IIIa inhibitors, invasive management strategies; all of these have been shown to improve outcomes over ‘standard’ medical management.

In this issue, a report by Antman et al.[1] presents 1-year follow-up data from the patients in TIMI 11B...