The differential diagnosis of the association of pneumonia and haemorrhagic pericarditis should include viral aetiologies

Sir,

For the sake of completeness, the differential diagnosis of bacterial pneumonia with haemorrhagic pericarditis should include Chlamydia infection in immunocompetent subjects, and the association of pneumonia and staphylococcal pericarditis. Where a bacterial pathogen has not been isolated, and the atypical pneumonia screen, as is usually the case, does not extend beyond *Mycoplasma pneumoniae*, *Legionella* spp., *Chlamydia pneumoniae* and *C. psittaci*, the differential diagnosis also ought to include viral pathogens, given the association of viral pneumonia and haemorrhagic pericarditis in human rhinovirus type C infection, and in Coxsackie group B infection, respectively, and also in view of the association of haemorrhagic pleural effusion and haemorrhagic pericarditis in Coxsackie B infection. A favourable outcome after antibiotic treatment of what was perceived to be bacterial pneumonia with haemorrhagic pericarditis does not necessarily validate presumption of a bacterial aetiology, given the fact that a similar outcome can occur when antibiotics are administered during the course of either rhinovirus type C pneumonia with pericarditis or Coxsackie B pleuroperticarditis. Viral aetiology had been validated by real-time polymerase chain reaction targeting human rhinovirus in the first case, and by culture of pericardial fluid in the patient with Coxsackie B pleuroperticarditis. In both instances, bacterial coinfection had been rigorously excluded by the fact that pericardial fluid was sterile on bacterial and mycobacterial culture. Where patients with bacterial pneumonia are coinfected with viral pathogens, there is also the potential risk that the viral agent might be the one that infects the pericardium, as was the case in an instance of *Hemophilus influenzae* pneumonia where concomitant pericarditis was attributable to Coxsackie A9 cultured from the pericardial fluid. Even where polymorphs predominate in the pericardial fluid cell count this does not necessarily signify a bacterial aetiology given the fact that one example of Coxsackie pericarditis was characterized by a serosanguineous effusion with a total white cell count of 2100/mm³ including 1470/mm³ polymorphs.

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References


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Cholesterol-lowering and cancer in the prevention of cardiovascular disease

Sir,

Since Goldstein et al. have cautioned about the possible long-term effects of statins on the incidence of cancer, citing adverse results from the primary and secondary cholesterol-lowering trials PROSPER, LIPID, CARE and WOSCOPS, the worrying results of the first major primary prevention cholesterol-lowering trial using another drug should not be forgotten.

The WHO Clofibrate Trial, of which I was the initiator and principal investigator, comprised 15,745 healthy middle-aged men aged 30–59 years, who were studied for an average of 5.3 years. Comparison of the results between the hypercholesterolaemic subgroup (5331 men) that received 1.6 g Clofibrate daily with a comparable subgroup (5296 men) randomly allocated to placebo not only showed a significant reduction in non-fatal myocardial infarction (4.6 vs. 6.2 per annum \(P<0.05\)) but also a non-significant increase in cancer deaths (40 vs. 24: \(P\approx 0.06\)), mostly intestinal. Overall mortality was increased (\(P<0.05\)).

An explanation for this apparent adverse effect of treatment on the incidence of cancer has never been clear but, interestingly, it was not evident during the 7.9 years of complete post-trial follow-up. Clofibrate was an inappropriate choice to lower plasma cholesterol, but the only drug available in the 1960s; it achieved an overall reduction of total cholesterol of only 9%, although there was a larger effect on serum triglycerides.

We speculated at the time that the failure to reduce all-cause mortality, mostly due to the adverse trend in the incidence of cancer, might be either due to some hitherto unrecognized effect of Clofibrate or a consequence of reduction of raised cholesterol concentrations or, least likely, due to chance. We emphasized also the importance of a full follow-up of all drug trials following their period of administration. This is seldom done, particularly if the main end point has been reduced.

It is disconcerting that we may now have to consider the possibility of a similar effect of statins. I certainly support the authors wise cautionary notes and encourage studies to try to resolve this issue.

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References

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