Hepatitis A Virus Seropositivity and Coronary Artery Disease

To the Editor:

We read with great interest the paper by Marek Smieja and colleagues showing that exposure to hepatitis A virus (HAV) was not associated with an excess risk of subsequent myocardial infarction, stroke, or cardiovascular death in Heart Outcomes Prevention Evaluation (HOPE) study patients. Total pathogen score based on seropositivity of 4 pathogens (Chlamydia pneumoniae, Helicobacter pylori, HAV, and cytomegalovirus) predicted a small increased hazard of cardiovascular events.

Many intracellular pathogens that induce a long-lasting antibody could contribute to atherogenesis. It is not known if persistent infection is actually required or if effects of acute temporal-limited infections as circulating antibodies per se can induce atherosclerosis. Acute infection with HAV leads to hepatitis but usually does not induce persistent liver disease as do other hepatitis viruses like hepatitis B or hepatitis C virus. HAV is believed to be eliminated from the host after the acute infection. There is no other target tissues in which this virus is known to reside or to produce disease. Once infected with HAV, anti-HAV immunoglobulin G (IgG) antibodies usually persist for the life of the host. Depending on age, 50% to 80% of people in developed countries are found to be seropositive for anti-HAV IgG antibodies. Persistent anti-HAV antibodies are commonly believed not to reflect persistent viral infection. HAV is the least studied of the viral candidates. In 2 recently published studies from the same cohort, a strong association between HAV and coronary artery disease (CAD) was reported. These findings by Zhu et al are at odds with a case-control study of Canadian patients and with the findings of Smieja et al. Marek Smieja and colleagues correctly point out that further data are needed to determine whether these results indicate true risk in certain populations or confounding by socioeconomic status or other risk factors.

We recently performed a cross-sectional study to investigate the possible association between HAV infection and angiographically proven CAD. Blood from patients undergoing coronary angiography was tested for serum IgG antibodies to HAV. Of the 218 patients, 81.7% had anti-HAV IgG antibodies. CAD (coronary artery diameter stenosis of 50% or more in at least at one epicardial vessel) prevalence was 66.3% in HAV-seropositive and 57.5% in HAV-seronegative patients (P=0.39). Despite the limitations of a cross-sectional study that exclusively shows an association and cannot establish causality, this analysis demonstrates that HAV seropositivity is not a predictor of risk for CAD. Additionally, this study supports previous data and the findings by Marek Smieja and colleagues that a total pathogen burden based on multiple infections (HAV, C pneumoniae, H pylori, cytomegalovirus, and influenza virus type A and type B) predicts CAD risk.

This finding, in addition to the results of the study by Smieja and colleagues, contributes to a growing body of data that show that HAV seropositivity is not associated with an excess risk of cardiovascular disease.

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