

THE ASSOCIATION OF CHRONIC COUGH WITH THE RISK OF MYOCARDIAL INFARCTION: THE FRAMINGHAM HEART STUDY

To the Editor:

We wish to comment on the interesting article by Haider et al (1) that reported an association between chronic cough and the subsequent development of myocardial infarction in the Framingham cohort. Their well-controlled, prospective study adds to a body of evidence associating risk of heart disease with asthma (2,3), chronic bronchitis (4), and rapid decline in pulmonary function (5). The authors hypothesized that chronic pulmonary infections, including those resulting from *Chlamydia pneumoniae*, could be involved and called for prospective serologic studies of infections as predictors of risk of coronary artery disease. If infections are indeed an underlying cause of these associations, questions arise regarding the most appropriate serologic markers to study (6).

We have enrolled approximately 300 patients into a prospective study of *C. pneumoniae* serology as a predictor of further cardiac interventions after an initial percutaneous transluminal coronary angioplasty (PTCA) (7). Serologic results were also obtained on other patients with PTCA who had previous coronary interventions. One- to 2-year post-PTCA follow-up on the prospective cohort is now complete, and the serologic results will be analyzed soon. In a preliminary analysis on a subgroup including patients with prior procedures, we found significant associations between serology and disease severity (7). Coronary artery disease severity was categorized according to the number of vessels and frequency of intervention: one PTCA on a single vessel versus sequential PTCAs on the same vessel versus interventions on

two or more vessels. We tested three serologic markers (*C. pneumoniae*-specific IgG and IgA, and chlamydial immune complexes), and all three were positively and significantly associated with disease severity. Remarkably, serum IgA antibodies were detected only in patients within the most severe disease category. Because serum IgA antibodies are mucosal in origin and imply chronic infection, this finding supports the hypothesis that persistent *C. pneumoniae* lung infection is a risk factor for multivessel coronary atherosclerosis. It remains to be seen whether similar associations will be found in the prospective cohort study.

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SEVERE HYPONATREMIA RESULTING FROM INSUFFICIENT CONVERSION OF ANGIOTENSIN I TO ANGIOTENSIN II

To the Editor:

Acquired selective hypoaldosteronism is characterized by aldosterone deficiency without an associated reduction in the other hormones secreted by the adrenal cortex (1). It is most often attributed to a reduction in renin secretion and is observed mainly in patients with chronic renal disease associated with diabetes mellitus. We report a patient with selective normoreninemic hypoaldosteronism and severe hyponatremia resulting from insufficient conversion of angiotensin I to angiotensin II that was corrected completely by mineralocorticoid therapy.

A 64-year-old man was admitted in May 1998. He had never been hospitalized, had no past medical history, and was taking no medications. On admission he was pale and slightly confused, with a systolic blood pressure of 60 mm Hg. His pulse was 100 beats per minute and his body temperature was 35°C. He complained of upper abdominal pain, and a computed tomographic scan demonstrated an intra-abdominal hemorrhage. (The patient had fallen down because of dizziness and hit his abdomen, and emergent surgery confirmed that the hemorrhage was the result of traumatic injury of the transverse mesocolon.)

Laboratory data on admission, including serum sodium, creatinine, blood urea nitrogen, glucose, and electrolyte levels, and urinalysis were within normal limits, except for mild leukocytosis and a low hematocrit