Correspondence

Incidental Live-Saving Polymerase Chain Reaction in a Case of Prosthetic Valve Dual-Pathogen Endocarditis

To the Editor—An otherwise healthy 44-year-old woman (a resident of a mountainous region of Switzerland) presented to the emergency department with acute onset of fever, myalgia, and disorientation. On physical examination, she was in shock without an evident infectious source. Laboratory analyses revealed leukocytosis (WBC count, $27{,}000 \times 10^9$ cells/L) and an elevated C-reactive protein level (260 mg/L). The patient had a history of congenital left ventricular inflow and outflow tract obstruction that was surgically corrected at the age of 15 years. In 1986 and 1996, she had infectious endocarditis of the aortic valve due to *Staphylococcus aureus;* the aortic valve was replaced by a homograft in 1990.

Transesophageal echocardiography showed vegetations on the aortic valve and possibly also on the mitral valve, with severe aortic and moderate mitral regurgitation. Therapy with vancomycin, gentamicin, and rifampicin was initiated. Blood cultures grew methicillin-sensitive *S. aureus,* supporting a diagnosis of *S. aureus* prosthetic valve endocarditis, and treatment was changed to flucloxacillin and rifampicin. The clinical course was complicated by acute renal failure, severe thrombopenia, and multiple septic emboli (in the brain, skin, retina, and spleen). After 4 weeks of antibiotic therapy, the aortic and mitral valves were replaced by mechanical prostheses. Cultures of the aortic valve were sterile, but broad-range eubacterial PCR had results positive for *Coxiella burnetii.* Serologic results confirmed chronic Q-fever (phase 1 IgG titer, 1:6400; phase 1 IgA titer, 1:200; phase 2 IgG titer, 1:12,800; and phase 2 IgA titer, 1:400); analysis of stored serum samples from the patient revealed that the infection had been acquired in the previous 12 months. Thus, the final diagnosis was chronic Q-fever prosthetic valve endocarditis complicated by acute *S. aureus* prosthetic valve endocarditis. The postoperative course was uncomplicated. *S. aureus* prosthetic valve endocarditis was treated for a total of 6 weeks with flucloxacillin and rifampicin. For Q-fever endocarditis, therapy with doxycycline and hydroxychloroquine was introduced and will be continued until the phase 1 IgG titer is <1:400, with a minimum treatment duration of 18 months. At the most recent follow-up visit, 10 months after the initial presentation, the patient was asymptomatic and titers of anti-*Coxiella* antibodies were decreasing (phase 1 IgG titer, 1:3200; phase 1 IgA titer, 1:50; phase 2 IgG titer, 1:6400; phase 2 IgA titer, 1:100).

This case is remarkable for the simultaneous implication of two different bacterial species in the pathogenesis of infective endocarditis, with one of the pathogens being discovered incidentally. Reports in the literature on dual-pathogen endocarditis are scarce; 2 cases with concomitant streptococcal and Q-fever endocarditis have been previously described in a large, prospective French study [1]. Our patient had underlying cardiac abnormalities predisposing her to *C. burnetii* endocarditis and resided in an area of Switzerland with a seroprevalence of *Coxiella* species infection up to 30% [2]. Nevertheless, *C. burnetii* infection was not suspected, because all clinical manifestations were compatible with staphylococcal prosthetic valve endocarditis. The diagnosis of Q-fever endocarditis was thus a lucky strike, which is disconcerting given the poor prognosis of unrecognized chronic *Coxiella* infection. In view of our experience, we believe that *C. burnetii* serologic analysis or PCR testing of heart valve tissue are worth performing in all patients with infective endocarditis who have a preexisting valvulopathy and live in a region in which Q-fever is endemic, regardless of whether another etiological agent has been identified or not.

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In Patients with Type 2 Diabetes Mellitus, Are Glycosylated Hemoglobin Levels Higher for Those with *Helicobacter pylori* Infection Than Those without Infection?

To the Editor—In recent years, a significant association has been reported between cardiovascular diseases, diabetes, and dyslipidemia and *Helicobacter pylori*

infection [1–6]. However, there are conflicting reports [2], and lack of data from Latin American should prompt local studies for establishing the relationship between *H. pylori* and such chronic diseases. We hypothesized that among Peruvian patients with type 2 diabetes mellitus, *H. pylori* infection may be associated with reduced metabolic control of disease. Therefore, we evaluated the relationship between glycosylated hemoglobin (HbA_{1c}) levels and *H. pylori* infection in a cohort of patients with type 2 diabetes mellitus from Peru.

We enrolled 75 patients (40 men and 35 women; mean age ± SD, 52.8 ± 6.6 years) affected by type 2 diabetes mellitus with a mean (±SD) diabetic duration of 8.2 ± 4.3 years, all of whom had similar geographical origin and socioeconomic status. Body mass index; cholesterol, triglyceride, and HbA_{1c} levels; and diabetic complications were evaluated. *H. pylori* infection was demonstrated through use of the urea breath test and biopsy of gastric tissue.

Most evaluated patients had a certain degree of gastritis (89.3% moderated superficial chronic gastritis), and 65.3% were infected with *H. pylori*. Although we did not find a statistically significantly higher incidence of *H. pylori* infection among patients with type 2 diabetes mellitus who disease was poorly controlled (HbA_{1c}, >7.0%) than among those with appropriate disease control (HbA_{1c}, <7.0%)—23 (71.9%) of 32 patients versus 26 (60.5%) of 43 patients (OR, 1.67; CI 95%, 0.56–5.03; $\chi^2_{Yates} = 0.61$; $P = .434$)—the levels of HbA_{1c} were significantly increased among infected patients, compared with uninfected patients (mean ± SD HbA_{1c}, 7.63% ± 0.23% versus 7.25% ± 0.49%; Mann-Whitney *U*, 442.0; Wilcoxon *W*, 793.0; $Z = -2.171$; $P = .03$) (figure 1). We found no statistically significant difference for body mass index or cholesterol or triglyceride levels between those with or without *H. pylori* infection.

Previous studies have demonstrated that there is a high prevalence of *H. pylori* infection among diabetic patients and that it is correlated with dyspeptic symptoms, as we see in our study [1]. Such findings support the hypothesis that permanent eradication of *H. pylori* would improve metabolic control of diabetes and may reduce the development of diabetes complications [3–5]. More importantly, diabetic subjects who have complications of cardiovascular autonomic neuropathy and dyspepsia are at high risk of *H. pylori* infection and should be carefully investigated and considered for eradication therapy [1]. Therefore, urea breath test as follow-up screening seems to be a useful tool to use to detect patient infection and reinfection, which would optimize the ability to control glucose levels [3, 5].

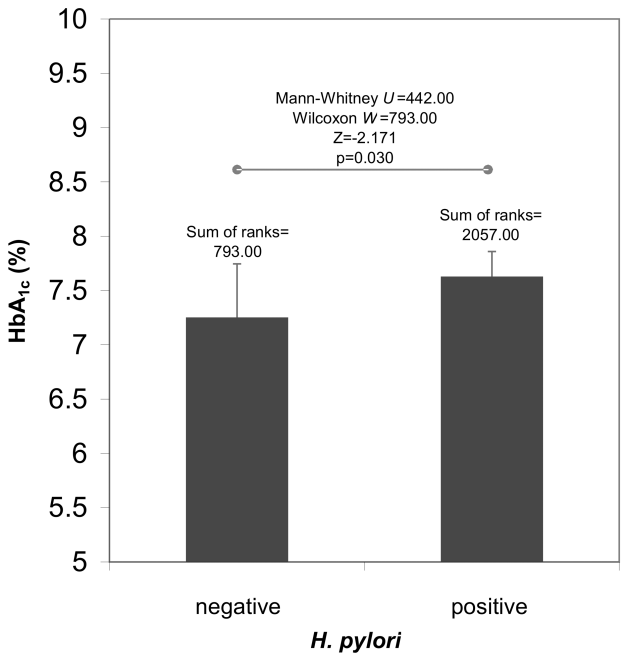

Figure 1. Comparative glycosylated hemoglobin levels among patients with type 2 diabetes mellitus, by status of *Helicobacter pylori* infection.

These preliminary findings, which need to be confirmed and extended in further studies, give evidence that *H. pylori* infection influences metabolic control among adults with type 2 diabetes mellitus; previous studies have demonstrated these findings among children and adolescents with type 1 diabetes mellitus [6]. Given the high prevalence of *H. pylori* infection that is found among these diabetic subjects and its influence on control of glucose levels, screening for such patients should be performed to aid in reducing the rate of infection and to improve the control of glucose levels.

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Comment on the Cover

To the Editor—We are pleased to see an illustration by Serefeddin Sabuncuoglu as the cover art of the 15 May 2007 issue of *Clinical Infectious Diseases* and deeply appreciate the editorial decision to highlight it as an important piece of work in the history of the struggle against infectious diseases. However, some corrections are required to improve the text that aims to explain the illustration.

First, there is a high degree of agreement among the experts that the correct name of the author of the manuscript from which the image was taken, and presumably the physician in the illustration, is Serefeddin Sabuncuoglu, who lived and practiced medicine in northern Anatolia during the 15th century [1]. His birth and death dates are almost accurately documented as 1385 and 1470, respectively, which is another point that needs clarification in the text describing the cover.

Secondly, Serefeddin Sabuncuoglu was a member of a Turkish family with no known Arabic connection. It seems that the Arab surgeon Albucasis's book *at-Tasrif*, which served as a main resource to Serefeddin Sabuncuoglu's book *Cerrahiyetü'l Haniyye*, and the Arabic script of the text led to a false impression that the author was an Arab physician. In addition, the text of Sabuncuoglu's book, which is housed in the Bibliotheque Nationale, is written in Turkish. Several articles derived from this handwritten manuscript can be searched for in the Medline database.

Finally, because of the unique illustrations, all of which are lacking in Albucasis's book, and because of numerous original additions embedded in the text, Serefeddin Sabuncuoglu's book is clearly more than a translation. The idea that medical procedures may be depicted for teaching purposes regardless of religious prohibitions is revolutionary in nature and has survived and developed over centuries, as is shown in the cover illustration.

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Viral Infections and Acute Otitis Media in Young Children

To the Editor—We read with interest the article by Chonmaitree et al. [1], which highlights the association between viral respiratory tract infections and acute otitis media (AOM) complication in young

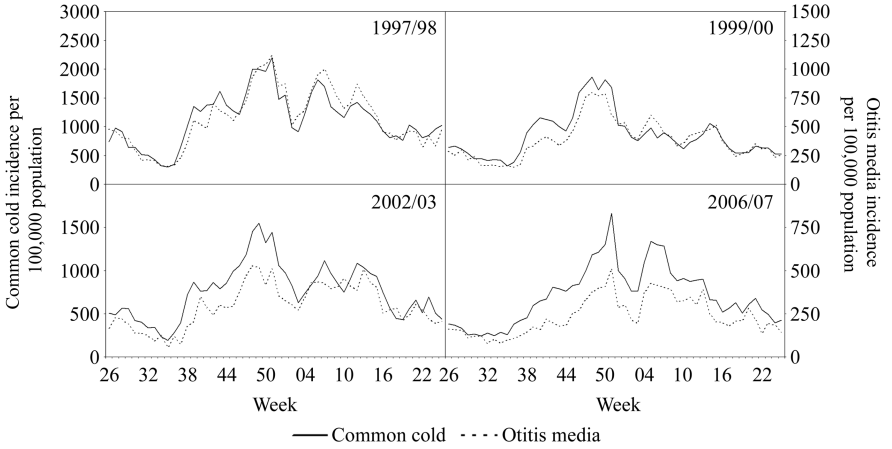

Figure 1. Clinical rate of otitis media and the common cold diagnosed in young children, aged 0–4 years, during 4 sample years. Winter weeks appear at the midpoints of the horizontal axes.