

Abstracts of current literature on epidemiology, diagnosis, and treatment

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PERSISTENT ANTIBODY RESPONSES TO BORRELIA BURGENDORFERI AFTER ACTIVE LYME DISEASE

A study was conducted to determine the antibody levels in serum samples of patients (n = 79) who had had Lyme disease (*Borrelia burgdorferi* infection) 10 to 20 years ago and who did not currently have signs or symptoms of active Lyme disease. Of the 79 patients, 40 had had early, localized, or disseminated Lyme disease 10 to 20 years ago (the early disease group), and 39 had had Lyme arthritis, which is a late manifestation of the illness (the late disease group). A 2-test approach (enzyme-linked immunosorbent assay and Western blot) was used to determine serologic status. In the follow-up evaluation 10 to 20 years later, 4 (10%) of the patients who had had early Lyme disease still had a positive IgM antibody response, and 10 (25%) of the patients currently had a positive IgG antibody response to *B. burgdorferi*. Of the 39 patients who had had Lyme arthritis, 6 (15%) of the patients still had IgM responses, and 24 (62%) still had IgG reactivity to *B. burgdorferi* at long-term follow-up. The researchers concluded that IgM and IgG antibody responses to *B. burgdorferi* may persist for 10 to 20 years; however, these responses are not indicative of active infection.

Kalish RA, McHugh G, Granquist J, et al. Persistence of immunoglobulin M or immunoglobulin G antibody responses to Borrelia burgdorferi 10–20 years after active lyme disease. Clin Infect Dis 2001;33:780–5.

HELICOBACTER PYLORI INFECTION AND THE DEVELOPMENT OF GASTRIC CANCER

A prospective, long-term study was conducted to determine the relation between *Helicobacter pylori* infection and the development of gastric cancer. Patients (n = 1526) who had active duodenal ulcers (n = 275), active gastric ulcers (n = 297), gastric hyperplastic polyps (n = 229), or nonulcer dyspepsia (n = 725) were prospectively studied. Except for 280 of the patients in the nonulcer dyspepsia group, all of the patients had *H. pylori* infection (n = 1246). *H. pylori* infection was assessed by histologic examination, serologic testing, and rapid urease tests and was defined by a positive result on any of these tests. Patients underwent endoscopy with biopsy at enrollment and then between 1 and 3 years after enrollment. The mean follow-up was 7.8 years (range, 1.0 to 10.6 years). Gastric cancers developed in 36 (2.9%) of the patients with *H. pylori* infection and in none of the patients without the infection. Gastric cancers were detected in 21 (4.7%) of the patients with nonulcer dyspepsia and *H. pylori* infection, 10 (3.4%) of the patients with active gastric ulcers and *H. pylori* infection, 5 (2.2%) of the patients with gas-

tric hyperplastic polyps and *H. pylori* infection, and none of the patients with active duodenal ulcers and *H. pylori* infection. Among the patients with *H. pylori* infection, those with severe gastric atrophy, corpus-predominant gastritis, and intestinal metaplasia were at significantly higher risk for gastric cancer. The researchers concluded that gastric cancer develops in persons infected with *H. pylori* but not in those uninfected with the organism.

Uemura N, Okamoto S, Yamamoto S, et al. Helicobacter pylori infection and the development of gastric cancer. N Engl J Med 2001; 345:784–9.

COMMUNITY-ACQUIRED METHICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS INFECTION

A retrospective cohort study involving a medical record review was conducted at a small Indian Health Service hospital located in a rural midwestern American Indian community to document the occurrence of community-acquired methicillin-resistant *Staphylococcus aureus* (MRSA) infections. The study was also conducted to evaluate risk factors for community-acquired MRSA infection compared with those for community-acquired methicillin-susceptible *S. aureus* (MSSA) infection. Included in the study were patients (N = 112) whose medical records indicated laboratory-confirmed *S. aureus* infection diagnosed in 1997. Of the patients, 62 (55%) had an MRSA infection and 50 (45%) had an MSSA infection. Forty-six (74%) of the patients with MRSA infection were classified as having community-acquired infection. Thirty-four (89%) of 38 community-acquired MRSA isolates available for evaluation via pulsed-field gel electrophoresis subtyping were clonally related and distinct from nosocomial MRSA isolates found in the region. The risk factors for community-acquired MRSA infection were not significantly different from those for community-acquired MSSA infection. The researchers concluded that community-acquired MRSA may have replaced community-acquired MSSA as the dominant strain in this community and that MRSA is circulating beyond nosocomial settings in this and possibly other US communities.

Groom AV, Wolsey DH, Naimi TS, et al. Community-acquired methicillin-resistant Staphylococcus aureus in a rural American Indian community. JAMA 2001;286:1201–5.

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