els were still higher than normal (19 U/mL; Figure 1). A ^{13}C-urea breath test (BreathMat, FinniganMat) was performed because of her worsening dyspepsia and was found to be positive. Endoscopy of the upper gastrointestinal tract with multiple biopsies of the gastric mucosa showed superficial antral gastritis and *Helicobacter pylori* infection. Treatment with clarithromycin (500 mg twice daily), metronidazole (250 mg twice daily), and omeprazole (20 mg per day) was administered for 7 days. Three months later, another breath test confirmed successful eradication of the infection, and the patient reported complete disappearance of dyspeptic symptoms and a gradual resolution of the neurologic symptoms. Her migraines significantly improved, and the Raynaud's phenomenon disappeared. Interestingly, aPL antibodies levels became normal (IgG 9 U/mL, IgM 7 U/mL) in May 2000, and remained so in October 2000.

Although we cannot definitely assert that *H. pylori* eradication was responsible for the improvement, the temporal association of the disappearance of aPLS after *H. pylori* eradication is suggestive. *H. pylori* could have acted as an aspecific promoter of aPLS, through the chronic activation of proinflammatory cells (4) and the chronic systemic release of proinflammatory cytokines (5–7). Alternatively, cross-mimicry mechanisms between *H. pylori* and some cell surface expressed phospholipids could be the base for an autoimmune *H. pylori*–induced reaction. We therefore suggest that *H. pylori* may be one of the infectious agents possibly involved in aPLS, although larger studies are needed to confirm this.

Valerio Cicconi, MD
Emilia Carloni, MD
Francesco Franceschi, MD
Roberto Nocente, MD
Nicolo Gentiloni Silveri, MD
Raffaele Manna, MD
Serenella Servidei, MD
Anna Rita Bentivoglio, MD
Antonio Gasbarrini, MD
Giovanni Gasbarrini, MD
Departments of Internal Medicine and Neurology
Catholic University of Rome
Rome, Italy

1. Nahass GT. Antiphospholipid antibodies and the antiphospholipid antibody syndrome. *J Am Acad Dermatol*. 1997;36:149–168.
2. Wilson WA, Gharavi AE, Koike T, et al. International consensus statement on preliminary classification criteria for definite antiphospholipid syndrome: report of an international workshop. *Arthritis Rheum*. 1999;42:1309–1311.
3. Capel P. Antiphospholipid antibodies, lupus anticoagulant and thrombosis. *Rev Med Brux*. 1997;18:108–110.
4. Ren Z, Pang G, Lee R, et al. Circulating T cell response to *Helicobacter pylori* infection in chronic gastritis. *Helicobacter*. 2000;5:135–141.
5. Isomoto H, Mizuta Y, Miyazaki M, et al. Implication of NF-kappaB in *Helicobacter pylori*-associated gastritis. *Am J Gastroenterol*. 2000;95:2768–2776.
6. Bodger K, Crabtree JE. *Helicobacter pylori* and gastric inflammation. *Br Med Bull*. 1998;54:139–150.
7. Yamaoka Y, Kodama T, Kita M, et al. Relation between clinical presentation, *Helicobacter pylori* density, interleukin 1β and 8 production, and cagA status. *Gut*. 1999;45:804–811.

GEMELLA MORBILLORUM BACTEREMIA ASSOCIATED WITH ADENOCARCINOMA OF THE CECUM

To the Editor:

The association between bacteremia and gastrointestinal neoplasms is well known. Since the first association between *Streptococcus bovis* bacteremia and colonic neoplasms was reported in 1977 (1), other streptococcal species and bacteria associated with underlying colonic malignancy include *S. sanguis*, *S. salivarius*, *S. equinus*, *S. milleri*, *S. agalactiae*, group G streptococci, and *Clostridium septicum* (2–7). We report an association between *Gemella morbillorum* and colon cancer.

An 89-year-old woman came to the emergency room because of generalized body weakness and a nonproductive cough of 3 weeks' duration. She reported vague, lower abdominal pains, as well as significant weight loss over the past few months. She reported no hematemesis, vomiting, jaundice, melena, hematochezia, or change in stool habits. Her medications included aspirin and nonsteroidal anti-inflammatory agents. There was no family history of malignancies. On admission, her vital signs were normal except for an oral temperature of 39.4°C. Examination of her heart and lungs was unremarkable. Her abdomen was slightly distended, and no masses were palpable. A rectal examination revealed no masses and heme-negative stools.

Laboratory data included a white blood cell count of 19,100 $\times 10^3$ cells per μL with 85% neutrophils, hemoglobin of 10.4 with mean corpuscular volume of 82 fL, and normal platelets. Liver function tests were normal. Radiographs of the chest and abdomen were unremarkable. A sonogram of the liver, gallbladder, and pancreas revealed a normal-size liver with no gallstones and a normal pancreas. Results of urine cultures were negative. Blood cultures later revealed growth of *G. (Streptococcus) morbillorum*. A subsequent colonoscopy revealed a fungating mass in the cecum. Biopsy specimens showed a well-differentiated adenocarcinoma. A transesophageal echocardiogram showed a calcified mitral annulus, no vegetations, and normal left ventricular function. The patient improved greatly after a 2-week course of intravenous penicillin and gentamycin therapy, and results of repeat blood cultures were negative. She refused to undergo surgery or chemotherapy for the colon cancer.

How neoplasms of the colon predispose to bacteremia is not understood. The normal intestinal barrier may be disrupted in neoplastic tissues, permitting bacterial transloca-

tion into the blood stream (8). *Gemella morbillorum* is a commensal organism in the gastrointestinal, respiratory, and genitourinary tracts in humans. Human infections are rare, but the spectrum of disease is very similar to that caused by *S. viridans*. Isolated cases of endocarditis, septic shock, arthritis, and meningitis have been reported (9,10). Treatment consists of intravenous penicillin and gentamycin. *Gemella morbillorum* is now on the list of bacteria that should lead to a search for an underlying colonic neoplasm.

Ramon Reyes III, MD
Aberra Abay, MD
Mark Siegel, MD
Division of Gastroenterology
Beth Israel Medical Center
New York, New York

1. Klein RS, Recco RA, Catalano, MT, et al. Association of *Streptococcus* bacteremia, and underlying gastrointestinal disease. *Arch Intern Med*. 1977;297:800–802.
2. Marinella MA. *Streptococcus sanguis* bacteremia associated with cecal carcinoma: case report and review of the literature. *Am J Gastroenterol*. 1997;92:1541–1542.
3. Legier JF. *Streptococcus salivarius* meningitis and colonic carcinoma. *South Med J*. 1991;84:1058–1059.
4. Gilon D, Moses A. Carcinoma of the colon presenting as *Streptococcus equinus* bacteremia. *Am J Med*. 1989;86:135–136.
5. Rich MW, Radwany SM. *Streptococcus milleri* septicemia in a patient with colorectal carcinoma. *Eur J Clin Microbiol Infect Dis*. 1993;12:225.
6. Wiseman A, Rene P, Crelinsten GL. *Streptococcus agalactiae* endocarditis: an association with villous adenomas of the large intestine. *Ann Intern Med*. 1985;103:893–894.
7. Kornbluth AA, Danzig JB, Bernstein LH. *Clostridium septicum* infection and associated malignancy. Report of 2 cases and review of the literature. *Medicine (Baltimore)*. 1989;68:30–37.
8. Diethch EA, Berg R. Bacterial translocation from the gut: a mechanism of infection. *J Burn Care Rehab*. 1987;8:475–482.
9. Debast SB, Koot R, Mas JF. Infections caused by *Gemella morbillorum*. *Lancet*. 1993;342:560.
10. Omran Q, Wood CA. Endovascular infection and septic arthritis caused by *Gemella morbillorum*. i. 1993;16:131–134.

ELEVEN-YEAR SURVIVAL OF A PATIENT WITH BULLOUS SARCOIDOSIS AFTER BILATERAL PLEURODESIS

To the Editor:

Large bulli of the lungs can complicate end-stage sarcoidosis. That can cause respiratory failure, cor pulmonale, and eventually death (1,2). We report the case of a patient with bullous sarcoidosis who survived for 11 years after repeated pneumothoraces that were treated by bilateral pleurodesis.

A 57-year-old man had sarcoidosis since 1968. Pleurodesis (with 1500 mg tetracycline) was done on the left side in 1986 and on the right side in 1988 for several episodes of bilateral pneumothorax. He had a chronic cough and shortness of breath afterward that required home oxygen therapy at 4 L/min. He also had a smoking history of 15 pack-years.

The patient was admitted with exacerbation of shortness of breath in 1998. On physical examination he was alert; his pulse rate was 103 beats per minute, blood pressure 114/68 mm Hg, and respirations 26 breaths per minute. The chest was hyperinflated with hyperresonance to percussion and decreased breath sounds. Physical examination was otherwise unremarkable. Total leukocyte count was 5900/μL, hemoglobin 10.5 g/dL, pH 7.33, PCO_2 97 mm Hg, PO_2 82 mm Hg, total CO_2 53.4 mEq/L, oxygen saturation 95% on 4 liters of oxygen, and angiotensin-converting enzyme 10 IU/L (normal range, 9 to 67). A chest radiograph showed multiple bilateral emphysematous bulli, and a computed tomography scan of the chest showed multiple bulli, calcified hilar lymph nodes, and prominent interstitial marking. A gallium^{67} scan showed no uptake in the lungs. The patient was treated with bronchodilators, steroids, and antibiotics. He refused lung reduction surgery and bullectomy. At the time of discharge, he was receiving prednisone, theophylline, albuterol, and ipratropium bromide. He died of acute respiratory failure 1 month later, after refusing endotracheal intubation.

Bullous sarcoidosis is one of the rare causes of several types of thin-walled lung cysts, the most common being congenital bullous emphysema (3). It is important to differentiate bullous sarcoidosis from the cavitary sarcoidosis that may be seen in the early stages of the disease, characterized by central necrosis caused by conglomerate granulomas (4). Surgery to reduce lung volume is beneficial in some patients with bullous sarcoidosis (1,5). Bilateral pleurodesis prevented recurrence of pneumothorax for 11 years in our patient. Pleurodesis might be a useful option for patients with bullous sarcoidosis and recurrent pneumothoraces who are high-risk surgical candidates.

P. Dileep Kumar, MD
Donald L. Epstein, MD
Department of Medicine
Huron Hospital/
Cleveland Clinic Health System
Cleveland, Ohio

1. Pena CM, Cosgrove DM, Eng P, et al. Bullectomies for bullous sarcoidosis. *Cleveland Clinic J Med*. 1993;60:157–160.
2. Judson MA, Strange C. Bullous sarcoidosis. A report of three cases. *Chest*. 1998;114:1474–1478.
3. Godwin JD, Webb WR, Savoca CJ, et al. Multiple thin walled cystic lesions of the lung. *Am J Roentgenol*. 1980;135:593–604.
4. Lynch JP, Kazerooni EA, Gay SE. Pulmonary sarcoidosis. *Clin Chest Med*. 1997;18:755–785.
5. Teramoto S, Matsuse T, Ouchi Y. Sarcoidosis is a significant cause of bullous emphysema. *Chest*. 1999;115:175–178.

INCREASED PLASMA ADRENOMEDULLIN LEVELS IN KAWASAKI DISEASE WITH CORONARY ARTERY INVOLVEMENT

To the Editor:

Adrenomedullin is a potent vasodilating and natriuretic peptide orig-