the same time point or have similar options for treatment and prolonged survival or AIDS-free time [5–7]. Survivor-treatment bias should be accounted for when interpreting the results of HIV cohort studies.

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Fatal Salmonella Pulmonary Arteritis in a Patient with Eisenmenger Syndrome

Sir—Arterial infection is the most serious complication of salmonellosis [1]. Salmonella arterial infections can involve the thoracic and abdominal aortas and the peripheral arteries. Infection of the pulmonary trunk or its major branches is rare [2, 3]. The mortality rate for patients with mycotic pulmonary aneurysms (pulmonary arteritis) is reported to be >50%, and most of the causative pathogens are gram-positive bacteria, including Staphylococcus aureus and streptococci [3]. To our knowledge, Salmonella species have never been reported as a cause of pulmonary arteritis in the English-language literature.

The patient was a 61-year-old woman who had received a diagnosis of atrial septal defect with Eisenmenger syndrome. She was admitted to a regional hospital (hospital A) because of fever, severe cough, and dyspnea. The patient was treated for asthma with secondary infection. Intravenous cefazolin (1 g q8h) was administered. On the eighth day of hospitalization at hospital A, the patient developed hemoptysis and severe chest pain and was transferred to our hospital.

Physical examination revealed a body temperature of 37°C, blood pressure of 120/70 mm Hg, a regular pulse rate of 90 beats/min, and a respiratory rate of 18 breaths/min. There was a grade III/VI systolic murmur heard along the left sternal border and apex. Chest palpation revealed a right parasternal heave. Except for clubbing fingers and cyanosis, the remainder of the findings of the examination were unremarkable. Laboratory data revealed a WBC count of 17.45 × 10⁹ cells/µL (with 92.8% neutrophils), a hemoglobin level of 16.4 g/dL, and a platelet count of 154 × 10⁹ platelets/µL. Blood chemistry examination revealed a urea nitrogen value of 9.3 mg/dL, a creatinine level of 0.7 mg/dL, an albumin level of 3.2 g/dL, and a C-reactive protein level of 10.37 mg/dL. Chest radiographs revealed cardiomegaly and a bulging pulmonary conus. The bilateral pulmonary arteries were also dilated (figure 1). A hazy, ill-defined shadow extending upward from the pulmonary conus was noted. Chest CT with contrast enhancement revealed markedly dilated pulmonary trunk and bilateral central pulmonary arteries (figure 1). An abnormal consolidation around the left pulmonary artery near the hilar area was demonstrated. The consolidation consisted of inhomogeneous masses with irregular peripheral enhancement, which was consistent with mural thrombus and peri-arterial inflammation. There was an irregular tract extending from the pulmonary artery into the consolidation mass, suggesting extravasation of the infected pulmonary artery. Echocardiography demonstrated a secundum atrial septal defect of 22 mm in diameter and no evidence of infective endocarditis.

The patient developed fever, repeated hemoptysis, and hypotension during the initial 3 days of hospitalization. The infected pulmonary arteritis (pulmonary mycotic pseudoaneurysm) was considered to have an impending rupture with a hemorrhage that was probably life-threatening. Surgical resection was necessary but risky because of pulmonary hypertension with Eisenmenger syndrome. The patient was considered to be a candidate for combined heart-lung transplantation.

Intravenous ceftriaxone (1 g q12h) was administered on the first day of hospitalization, because 2 sets of blood cultures from hospital A were positive for Salmonella serogroup D (nontyphoid). The isolate was susceptible to ampicillin, ceftriaxone, and ofloxacin but resistant to trimethoprim-sulfamethoxazole, as determined by the disk diffusion method. The patient was afebrile and in stable clinical condition (subsidence of chest pain and absence of hemoptysis) on the fourth day of hospitalization. Unfortunately, the patient developed a sudden onset of intractable pulmonary hemorrhage and died on the seventh day of hospitalization, despite immediate and vigorous resuscitative efforts.

Pulmonary hypertension with Eisen...
MENGER SYNDROME LEADS TO EXPOSURE OF THE PULMONARY VASCULATURE TO SYSTEMIC ARTERIAL PRESSURE AND RESULTS IN ANEURISMAL DILATION. THE RESPONSIBLE MICROORGANISM HAS AN INCLINATION TOWARD DISEASED VASCULAR WALLS, AND MURAL THROMBI WOULD PROVIDE AN IDEAL ENVIRONMENT FOR THE SEEDING AND GROWTH OF THE MICROORGANISMS. THE CLINICAL COURSE AND MANAGEMENT OF INFECTED PULMONARY ARTERITIS IS NOT WELL DOCUMENTED BECAUSE OF THE RARITY OF CASES. FROM EXPERIENCE WITH THE MANAGEMENT OF AORTIC ANEURYSMS INFECTED WITH SALMONELLA SPECIES [4–6], SURVIVAL RATES APPEAR TO IMPROVE AMONG PATIENTS WHO RECEIVE COMBINED MEDICAL TREATMENT AND SURGICAL INTERVENTION. IN THE COLLECTIVE CASE STUDY BY HSU ET AL. [4], NONE OF THE 20 PATIENTS WHO HAD COMBINED MEDICAL AND SURGICAL TREATMENT DIED. HOWEVER, 2 OF THE 4 PATIENTS TREATED WITH ANTIBIOTICS ALONE DIED.

COMPLEX CONGENITAL HEART DISEASE WITH INDOLENT SALMONELLA INFECTION EXPOSED OUR PATIENT TO A LIFE-THREATENING SITUATION. EARLY SURGICAL INTERVENTIONS, INCLUDING RESECTION OF THE INFECTED LEFT PULMONARY ARTERY AND WIDE DEBRIDEMENT OF ADJUVANT TISSUE, FOLLOWED BY COMBINED HEART-LUNG OR LUNG TRANSPLANTATION [7], WERE CRUCIAL IN OUR CASE BECAUSE OF THE IMPENDING RUPTURE WITH PROBABLE LIFE-THREATENING HEMORRHAGE. UNFORTUNATELY, THE PATIENT HAD INTRACTABLE PULMONARY HEMORRHAGE AND DIED, DESPITE ADMINISTRATION OF APPROPRIATE ANTIBIOTIC TREATMENT 1 WEEK BEFORE DEATH.

IN CONCLUSION, SALMONELLA SPECIES SHOULD BE INCLUDED IN THE LIST OF PATHOGENS THAT CAN CAUSE PULMONARY ARTERITIS. AGGRESSIVE ANTIBIOTIC TREATMENT AND INTENSIVE SURGICAL INTERVENTION ARE ESSENTIAL TO AVOID DEATH DUE TO RUPTURE OF THE ANEURYSM.

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