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Stenotrophomonas maltophilia pericarditis

To the Editor,

Pericarditis is defined as a pericardial inflammation caused by infectious and non-infectious etiology. It may be clinically silent or may result in severe hemodynamic collapse and mortality. Developments in the field of antibiotic therapy, cardiac surgery, hemodialysis, cancer chemotherapy, and organ transplantation, as well as the current epidemic of HIV infection and AIDS, have expanded the spectrum of agents in the etiology of pericarditis and cardiac infections (1, 2). Etiology of the disease effects the outcome. Although purulent bacterial pericarditis and tuberculous pericarditis are less common, they may cause serious morbidity and mortality (1). It is essential to establish a correct diagnosis because if left untreated, the combination of tamponade and sepsis may result in up to 100% mortality. The accurate incidence of pericarditis has not yet been reported. A few reports on the antimicrobial susceptibility of common causative microorganisms of bacterial pericardial effusions have been published. In a study conducted by Sotoudeh Anvari et al. (3) in Iran, 320 patients hospitalized with pericardial effusion at Tehran Heart Center between 2007 and 2012 were prospectively examined. Bacterial cultures were positive in 35 patients. The most common pathogens were *Staphylococcus epidermidis*, *S. auerus*, and *S. haemolyticus*, and other causative organisms were *Streptococcus spp.*, *Enterococcus faecium*, *Pseudomonas aeruginosa*, and *Acinetobacter baumannii* (3).

Stenotrophomonas maltophilia are motile, glucose non-fermentative, gram-negative aerobic bacilli. They are an infrequent cause of health care-associated infections. This paper presents a patient with underlying lung cancer who was admitted to the emergency department with complaints of dyspnea, cough, and wheezing and was diagnosed with *S. maltophilia* pericarditis.

A 78-year-old male patient who had lung cancer with no history of chemotherapy and radiotherapy was admitted to the emergency department with complaints of chest pain, cough, dyspnea, and wheezing. Pericardial friction was heard on cardiac auscultation. Echocardiography revealed pericardial effusion. The patient was transferred to the cardiology intensive care unit. Laboratory results were as follows: CRP level, 3.44 mg/dL; WBC count, 14.25 106/mL; and neutrophil count, 70.9%. Evaluation of the pericardial puncture fluid revealed total protein level of 4.9 g/dL, albumin level of 3.2 g/dL, LDH level of 267 U/L, and adenosine deaminase level of 33.6 U/L. Microscopy revealed 400 leukocytes/mm^3. An empirical treatment with ceftriaxone 2x1 g and clarithromycin 2x500 mg was started. Massive pericardial effusion and tumor were detected in the lung and thorax by computerized tomography. Pericardial puncture fluid culture yielded non-fermentative, gram-negative bacteria identified as S. maltophilia by MALDI-TOF MS (VITEK MS, Biomerieux, France). Minimum inhibitory concentration results via gradient test (E test, Biomerieux, France) were found as sensitive for minocycline, trimethoprim/sulfamethoxazole, levofloxacin, ceftazidime, and chloramphenicol. There was no evidence of acid-resistant bacilli in mycobacterial staining and culture. Administration of i.v. levofloxacin 500 mg/day was started after consultation from the infectious diseases department. Pericardial effusion was not detected in the control echocardiography performed after 12 days. During follow-up, i.v. ceftazidime 3x2 g/day was started due to persistent fever, and the patient was transferred to an infectious disease clinic. Levofloxacin and ceftazidime were continued for 28 and 10 days, respectively, while the fever was resolving. During follow-up, the CRP level was 0.61 mg/dL and WBC count was 6.37 106/mL. The patient was discharged with a good general condition and no active complaint. No relapse occurred after 6 months of follow-up.

Bacterial pericarditis is a clinical condition that may conclude in serious consequences. Our case emphasizes the importance of bacterial culture in terms of detecting rare pathogens of pericarditis in etiology and also pathogen oriented antibiotic treatment. To our knowledge, this case is the first case of S. maltophilia associated pericarditis in the literature.

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Assessment of hemodynamic changes in preterm infants with respiratory distress syndrome

To the Editor,

Although the cardiac effects of invasive and noninvasive ventilation have previously been investigated separately (1), we aimed to investigate the right and left ventricular systolic and diastolic dimensions and functions of preterm infants who were treated with surfactant and who received mechanical ventilation support due to respiratory distress syndrome (RDS).

Preterm infants with birth weight of ≤1500 g and/or born at ≤32 gestational weeks within the first 6 h of life and requiring mechanical ventilation for at least the first 24 h of life due to RDS were considered. The first echocardiographic evaluation of the infants was during invasive ventilation. The second echocardiographic evaluation was in nasal continuous positive airway pressure (NCPAP) 24 h after infants were extubated, and the positive end-expiratory pressure (PEEP) was 6 cmH_20. Forty infants were studied (22 males and 18 females); mean gestational age was 27.2±2.1 (mean±SD) weeks,and mean birth weight was 1050±270 (mean±SD) g. A significant decrease in systolic blood pressure was observed in infants with patent ductus arteriosus (PDA), but no change was observed in left ventricular sizes and functions. In addition, no significant change was observed in right ventricular functions and cardiac output (CO) and fractional shortening values. For this reason, PDA is thought to have no effect on ventricular functions.

In preterm infants, incorrect measurements may be obtained due to paradoxical septal wall movements and left ventricular distortion due to right ventricular dominance. In healthy infants, right ventricular cavity dimension at end-diastole (RVEDd) and right ventricular cavity dimension at end-systole (RVESd) decrease in the first 2 days of life, and this is similar for ventilated infants (2). We found that RVEDd, RVESd, and CO values of infants followed up in mechanical ventilators were lower than those obtained in infants after taking them to NCPAP. This difference was due to a decrease in right ventricular function, which was in the first 2 days of life, and due to a negative effect on the right ventricular function, which was caused by severe RDS (3). All infants were monitored with the same PEEP value, and improvements were determined in the hemodynamic and echocardiographic evaluations during noninvasive ventilation. This situation may be related in the recovery of lung problems rather than in the PEEP effect.

We did not find any difference in hemodynamic parameters in relation to PDA. We noticed that PDA was associated with an increase in left atrial diameter and decrease in aortic root diameter while on invasive and noninvasive ventilation. We hypothesized that PDA may not be clinically characterized in the first days after delivery as the flow through it is generally not turbulent, wherein, as no physical sign is audible, it was not statistically significant. We believe that the treatment of RDS rather than of PDA in the first days of life is better based on hemodynamic and echocardiographic findings.

Mechanical ventilation reduces the right and left ventricular preload and improves the left ventricular afterload (4). Mechanical ventilation should be used with the most optimal methods possible and the lowest mean airway pressure value for preterm infants in the presence of RDS. Preterm infants should be extubated as soon as possible and should be tried to be made with noninvasive ventilation.

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