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Atrioventricular block after reperfusion: A reflection on early beta-blocker therapy for acute myocardial infarction

To the Editor,

Early coronary reperfusion achieved by primary percutaneous coronary intervention (pPCI) significantly reduces the occurrence of complete atrioventricular block (CAVB) in acute myocardial infarction (AMI) patients. The reported incidence of high-degree AVB was 7% and 1% in patients with right coronary artery (RCA) and left anterior descending artery (LAD) culprit lesion, respectively (1). We report a case of late-onset CAVB after successful pPCI, highlighting the potential risk of early beta-blocker therapy in ST segment elevation myocardial infarction (STEMI) patients.

A 43-year-old man with hypertension had chest pain lasting for 3 h. Electrocardiography revealed Q wave and ST segment elevation in leads V1 through V5 and occasional ventricular premature beats. Troponin T level was 47.3 ng/L. Coronary angiography showed total occlusion at the proximal LAD and severe stenosis in the mid-of RCA. Thrombus aspiration and stent implantation was successfully performed in LAD. Post-stent angiography revealed TIMI grade 3 blood flow of LAD with no septal branch occlusion. Drug regimen included aspirin, clopidogrel, tirofiban, perindopril, and atorvastatin. The use of beta-blocker was deferred, as large areas of infarction might put the patient at risk of heart failure and cardiogenic shock. Twenty hours after the PCI, the patient had a syncope attack. Electrocardiography revealed CAVB with no escape rhythm, which was followed by ventricular fibrillation. With external cardiac compression 60 s later, normal atrioventricular conduction was restored. No ST segment deviation was detected on electrocardiography. Such CAVB repeatedly occurred without electrolyte disturbance. Emergency coronary angiography showed that the lesion in RCA was not aggravated and also confirmed the patency of the LAD stent. With a transvenous temporary pacemaker, the patient was pacing dependent. Nine hours later, normal atrioventricular conduction was restored. Two days later, additional stenting was performed for the RCA lesion. Cardiac magnetic resonance performed 7 days later demonstrated a near transmural infarction of the septum, with a hypodense core signifying microvascular obstruction (MVO) in this region. The patient was discharged without beta-blocker considering the risk of bradycardia. At follow-up, repeated Holter monitoring showed no conduction defects and left ventricular ejection fraction was 60%; bisoprolol 2.5 mg qd was then added to his drug regimen.

Even after successful pPCI, patients are still at risk of problems such as reperfusion injury. Our patient's late-onset CAVB may be related to MVO (2), which is a type of myocardial reperfusion injury. Current clinical guidelines recommend the initiation of oral beta-blockers within 24 h in STEMI patients with no contraindications (3). However, it also cautions against early use in patients with risk factors for hemodynamic instability. However, data from an observational study showed that beta-blocker use after the first 24 h of hospitalization was associated with a 56% decreased risk of in-hospital mortality compared with early oral administration. While hemodynamically stable STEMI patients were favorable to receive early beta-block therapy, early oral beta-blocker users still experienced an increase in short-term mortality, despite reductions in the rate of cardiogenic shock (4). Severe bradyarrhythmias such as CVAB may explain the excess in mortality. Further reflection on early beta-blocker therapy in secondary prevention after AMI is therefore necessary.

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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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