Microorganisms and valve tissue/ Demonstration of *Chlamydophila pneumoniae, Mycoplasma pneumoniae, Cytomegalovirus,* and Epstein-Barr virus in atherosclerotic coronary arteries, nonrheumatic calcific aortic and rheumatic stenotic mitral valves by polymerase chain reaction

Mikroorganizmalar ve kapak dokusu / Aterosklerotik koroner arterler, romatizmal olmayan kalsifik aort ve romatizmal stenotik mitral kapaklarda Chlamydophila pneumoniae, Mycoplasma pneumoniae, Cytomegalovirus ve Epstein-Barr virüsünün polimeraz zincir reaksiyonu ile gösterilmesi

Dear Editor,

We read with great interest the article by Bayram et al. (1) regarding the presence of bacterial and viral pathogens in coronary and valve specimens determined by means of polymerase chain reaction method. The authors sought to evaluate the presence of two bacterial (*C. pneumoniae* and *M. pneumoniae*) and two viral (CMV and EBV) pathogens in heart valves extracted during surgery. They found out that *C. pneumoniae, M. pneumoniae,* and CMV were detected in patients with stenotic aortic and mitral valves and in patients with coronary atherosclerosis with similar frequencies. The most striking findings for us are the relatively high frequency of *C. pneumoniae, M. pneumoniae* and CMV in mitral valve tissue (21%, 7%, and 12% respectively). The underlying mechanism and the progression of chronic rheumatic valve disease are remained to be a dilemma in regard to pathophysiologic insight. At least but not last, we know that the chronic phase of rheumatic valve involvement is strongly associated with ongoing release of serum inflammatory mediators, which correlate with the severity of valve involvement, valve scarring and subsequent valve calcification (2, 3). However, the most intriguing question so far, remained to be as an unknown triggering factor for progression and ongoing inflammation. Now according to Bayram et al. (1) we can speculate that one of the triggering mechanisms for valve scarring and ongoing inflammation might be a coexistence of some bacteria or viral microorganism? Alternatively, is there a proactive role of those organisms in terms of fibrotic tissue progression? Although the study by Bayram et al. (1) is too preliminary and needs to be clarified with respect to a cause–effect relationship, the results excite us for shedding more information about what presence of in micro word of valve.

We thank authors for whipping us for a little more questioning the etiology of rheumatic valve.

Vedat Davutoğlu, Süleyman Ercan, Muhammed Oylumlu[1]
Department of Cardiology, Faculty of Medicine, Gaziantep University, Gaziantep
[1]Clinic of Cardiology, Şehitkamil State Hospital, Gaziantep-*Turkey*

References

1. Bayram A, Erdoğan MB, Ekşi F, Yamak B. Demonstration of Chlamydophila pneumoniae, Mycoplasma pneumoniae, Cytomegalovirus, and Epstein-Barr virus in atherosclerotic coronary arteries, nonrheumatic calcific aortic and rheumatic stenotic mitral valves by polymerase chain reaction. Anadolu Kardiyol Derg 2011; 11: 237-43.
2. Sarı I, Davutoğlu V. Association of chronic subclinical inflammation with severity and progression of rheumatic valve disease. Int J Cardiol 2008; 124: 263.
3. Davutoğlu V, Çelik A, Aksoy M. Contribution of selected serum inflammatory mediators to the progression of chronic rheumatic valve disease, subsequent valve calcification and NYHA functional class. J Heart Valve Dis 2005; 14: 251-6.

Address for Correspondence/Yazışma Adresi: Dr. Vedat Davutoğlu
Department of Cardiology, Faculty of Medicine, Gaziantep University, 27310, Gaziantep-*Turkey*
Phone: + 90 342 360 60 60/76290 Fax: + 90 342 360 39 28
E-mail: davutoglu@gantep.edu.tr
Available Online Date/Çevrimiçi Yayın Tarihi: 11.08.2011

doi:10.5152/akd.2011.147

Author's Reply

Dear Editor,

We agree with the information that authors of the letter have shared with us. We are thankful for their interest in our study (1). Additionally, we want to answer their questions about the potential role of microorganisms in the pathogenesis of atherosclerosis.

The hypothesis that several bacterial and viral agents may induce the progression of atherosclerosis has been extensively studied for two decades. Several studies have suggested an association between infectious agents and atherosclerosis (2, 3).

These microorganisms not only bear triggering mechanisms for ongoing inflammations, they also have proactive roles in fibrotic tissue progression. They may comprise triggering factors for the initiation and/or acceleration of an ongoing inflammatory process by increased expression of adhesion molecules and inflammatory cytokines, procoagulant effects, increased receptor expression and activity, enhanced uptake of cholesterol and of modified low-density lipoprotein, increased smooth muscle cell migration and proliferation, anti-apoptotic effects, and autoimmune response to infection (4).

The question whether microorganisms have proactive role in fibrotic tissue progression is answered partly by Tang et al. (5), who detected viral DNA in the lungs of patients with idiopathic pulmonary fibrosis (IPF) in their study. Similar to the lung pathology in patients with IPF, another study supported the concept that viral infection of renal epithelial cells contributed to the pathogenesis of chronic interstitial nephritis with characteristic interstitial fibrosis (6). According to data obtained from these studies, we can conclude that chronic viral infection of epithelial cells may be a trigger for fibrogenesis in several organs, as well as in vessel wall.

Ayşen Bayram, Mustafa B. Erdoğan[1], Fahriye Ekşi, Birol Yamak[1]
Department of Microbiology and Clinical Microbiology, Faculty of Medicine, Gaziantep University
[1]Clinic of Cardiovascular Surgery, Sanko Hospital, Gaziantep-*Turkey*

References

1. Bayram A, Erdoğan MB, Ekşi F, Yamak B. Demonstration of Chlamydophila pneumoniae, Mycoplasma pneumoniae, Cytomegalovirus, and Epstein-

Barr virus in atherosclerotic coronary arteries, nonrheumatic calcific aortic and rheumatic stenotic mitral valves by polymerase chain reaction. Anadolu Kardiyol Derg. 2011; 11: 237-43. [CrossRef]

2. Kaplan M, Yavuz SS, Çınar B, Köksal V, Kut MS, Yapıcı F, et al. Detection of Chlamydia pneumoniae and Helicobacter pylori DNA in atherosclerotic plaques of carotid artery by polimerase chain reaction. Int J Infect Dis 2006; 10: 116-23. [CrossRef]
3. Ibrahim AI, Obeid MT, Jouma MJ, Moasis GA, Al-Richane WL, Kindermann I, et al. Detection of herpes simplex virus, cytomegalovirus and Epstein-Barr virus DNA in atherosclerotic plaques and in unaffected bypass grafts. J Clin Virol 2005; 32: 29-32. [CrossRef]
4. Shi Y, Tokunaga O. Herpes virus (HSV-1, EBV and CMV) infections in atherosclerotic compared with non-atherosclerotic aortic tissue. Pathol Int 2002; 52: 31-9. [CrossRef]
5. Tang YW, Johnson JE, Browning PJ, Cruz-Gervis RA, Davis A, Graham BS. Herpes virus DNA is consistently detected in lungs of patients with idiopathic pulmonary fibrosis. J Clin Microbiol 2003; 41: 2633-40. [CrossRef]
6. Becker JL, Miller F, Nuovo GJ, Josepovitz C, Schubach WH, Nord EP. Epstein-Barr virus infection of renal proximal tubule cells: possible role in chronic interstitial nephritis. J Clin Invest 1999; 104: 1673-81. [CrossRef]

Address for Correspondence/Yazışma Adresi: Dr. Ayşen Bayram
Department of Microbiology and Clinical Microbiology, Faculty of Medicine, Gaziantep University, Gaziantep-*Turkey*
Phone: +90 342 360 60 60/77533
E-mail: aysenbayram@hotmail.com
Available Online Date/Çevrimiçi Yayın Tarihi: 11.08.2011

Complete cure with medical treatment of prosthetic mitral valve endocarditis, which is initially diagnosed as mitral valve thrombus

Başlangıçta mitral kapak trombüsü tanısı konulup medikal tedavi ile tam iyileşme sağlanan yapay mitral kapak endokarditi

Dear Editor,

A 59-year-old male patient was admitted to hospital with a complaint of fatigue and anorexia. He underwent mitral valve replacement operation nine months before the presentation. After the operation, patient attended control examinations irregularly. On admission; his blood pressure, heart rate and body temperature were within the normal range. Physical examination was also normal except prosthetic valve sound on auscultation. Electrocardiogram showed normal sinus rhythm and first laboratory findings were as following: INR: 1.64, prothrombin time: 21.8 second, sedimentation rate: 83 mm/h, hemoglobin: 13.9 gr/dl, hematocrit: 41.8%, platelets: 278000/mm^3, white blood cells: 11200/ mm^3 with 82% of granulocytes. Transthoracic echocardiography revealed thrombus at the edge of prosthetic valve. Transesophageal echocardiography (TEE) displayed multiple and mobile with a maximum of 1.4x0.4 cm sized thrombus at sutured site of prosthetic valve (Fig. 1). During first 3 days, patient was managed with warfarin and unfractionated heparin. Despite 3-day heparin infusion, control TEE did not show any regression in thrombus size. After that, 50 mg of alteplase was infused with a 4 mg/h dosage. TEE revealed mild regression in the thrombus size after thrombolytic therapy (Fig. 2). However, 24 hours after alteplase infusion, prominent fever, malaise and deterioration of consciousness were observed. Infective endocarditis was thought as possible diagnosis and eight tubes of blood culture was taken. Then, methicillin resistant *Staphylococcus aureus* was isolated in the four specimens as causative microorganism although first two specimens that had been taken during initial evaluation were clear. After six-weeks of antibiotics treatment, control TEE was free of the thrombus and/or vegetation (Fig. 3) and patient was discharged from hospital with a complete cure of prosthetic valve endocarditis (PVE).

PVE is associated with a high mortality rate despite diagnostic and therapeutic improvements. It's incidence is increasing and reaches 20-30% of all infective endocarditis episodes. PVE is a common indication for surgery (1, 2). Complete cure with medical therapy was reported up to 20% of selected cases (2, 3). TEE is a standard method for diagnosis of PVE. However, differentiation of thrombus and vegetation in the prosthetic valves could be difficult in the atypical presentation as our case (4). In such cases, final diagnosis usually is made according to clinical picture (5). Suspicion of endocarditis in such cases could prevent overlooked diagnosis of endocarditis. In the progression of our case, we thought that, initial thrombolytic therapy elicit the clinical signs of endocarditis. Thrombolytics could clear the surface of vegetation from covering thrombus and direct exposing of vegetation surface can lead to development of fever and other signs of endocarditis. Thrombolytic therapy may also enhance the effect of antibiotics via cleaning of thrombus coat, and by the way, antibiotics could penetrate

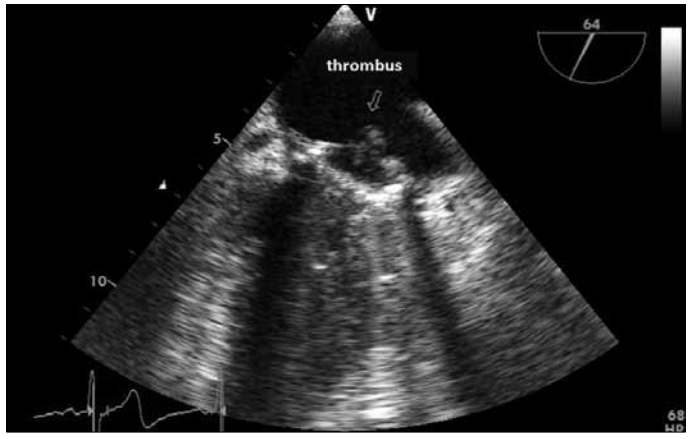

Figure 1. TEE images of mobile vegetation before thrombolytic treatment
TEE - transesophageal echocardiography

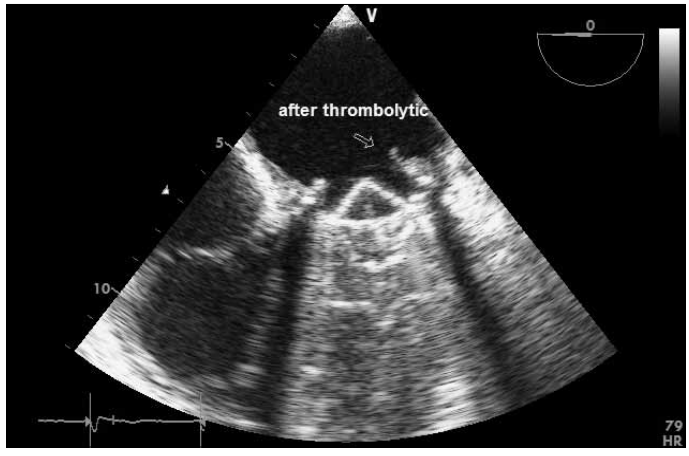

Figure 2. TEE images after thrombolytic treatment
TEE - transesophageal echocardiography