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Loeffler's endocarditis associated with a massive left ventricular thrombus

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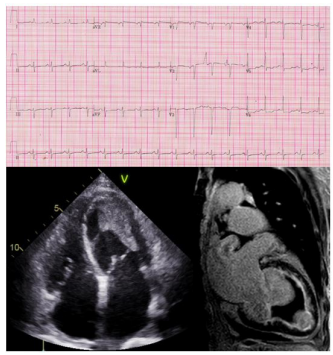
Loeffler endocarditis is rare restrictive cardiomyopathy caused by eosinophlic cell infiltrations of heart tissue with subsequent tissue damage from degranulation, consequentially leading to fibrosis. It is present in 40~50% of patient of hypereosinophilic syndrome. Loeffler endocarditis is a disease with significant mortality and morbidity. Often diagnosed at late stages, therapy Methods are limited once fibrosis occurs, usually requiring heart failure medications or surgical intervention. We experienced a case of Loeffler endocarditis with a massive left ventricular(LV) thrombosis. A 58-year-old woman with worsening chest discomfort and dyspnea was transferred to our hospital. Electrocardiogram showed sinus tachycardia and left atrial enlargement and laboratory test showed hypereosinophilia(1.86×10³/㎣). Transthoracic echocardiogram(TTE) revealed huge echogeneic mass obliterating cavity of LV(75.5mm×29.5mm), decreased LV systolic function(ejection fraction=46%) and restrictive physiology of LV filling pattern. Cardiac magnetic resonance imaging(CMR) showed diffuse subendocardial enhancement of LV with a massive intraventricular thrombus. To suppressing eosinophil-based cardiac inflammation and preventing thromboembolic event, she was administered systemic corticosteroid and anticoagulant(Warfarin). And we prescribed medications for relieving heart failure including angiotensin II receptor blocker, ß-blocker, loop diuretics and spironolactone. After anti-inflammatory therapy and supportive care, her symptoms improved significantly and discharged admission day 15. Loeffler endocarditis is a fatal disease requiring proper diagnosis and treatment. Patient with hypereosinophilia should be monitored by TTE or CMR because of the development of thrombosis and fibrotic, restrictive cardiomyopathy.



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Prior beta blocker uses against ventricular arrythmias following acute myocardial infarction

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Background/Aims: Ventricular arrhythmia (VA) remains the major cause of mortality and sudden cardiac death in patients suffering from acute myocardial infarction (AMI). Early use of beta blockers (BB) in the setting of AMI reduces mortality and the incidence of VA However, the best timing for initiation of BB is still not well established. Also, preventive role of BB against VA following AMI is controversial. The goal of this study is to evaluate the effect of prior beta blocker uses against VA following AMI in the porcine model. **Methods:** Twenty pigs were randomly divided into two groups based on the beta blocker medication (BB, N=10; Control, N=10). Oral beta blockers (bisoprolol) was administered for one week before experiment. After implanting a loop recorder, AMI was induced by occlusion of the middle left anterior descending artery (LAD). Each swine was closely observed for one hour for development of ventricular arrhythmia, after which the swine was carried back to the breeding house and monitored until recovery. One week later, animals were euthanized and the loop recorder were analyzed **Results:** The incidence of VA in the acute AMI phase was not different between the two groups (6 VAs in the BB vs. 6 VAs in the control, P=1.000). However, the incidence of premature ventricular complex (PVC) was lower in the BB group (0.9%) than in the control group (19.4%) (p=0.036). More pigs experienced arrhythmic death in the control group than in the BB group in the delayed AMI phase (2 death in the BB vs. 5 deaths in the control, P=0.236) **Conclusions:** Our data showed that BB did not prevent VA in the acute AMI phase. However, it could reduce the risk of arrhythmic death in the delayed AMI phase restoring electrical stability, which might be associated with an attenuation of PVC. These results suggest that prior use of beta blockers might help the patients at high AMI risk.