A Mexican Standoff: Mitral stenosis, mitral balloon valvuloplasty, Tp-e interval, Tp-e/QT and Tp-e/QTc ratios and sympathetic activity

To the Editor,

I have read with great interest the study entitled, "Evaluation of Tp-e Interval, Tp-e/QT Ratio and Tp-e/QTc Ratio in Patients with Mitral Valve Stenosis Before and After Balloon Valvuloplasty" (1). The authors demonstrated that Tp-e interval and Tp-e/QT and Tp-e/QTc ratios were prolonged in patients with mitral stenosis compared with healthy subjects and these parameters were favorably affected by percutaneous mitral balloon valvuloplasty (PMBV) even 24 h after the procedure. In addition, they affirmed that the link between ventricular repolarization abnormality, mitral stenosis, and effects of PMBV was sympathetic activation. Improvement seen in these arrhythmic markers in such short time and limited number of patients is striking and may be debatable. Similar to the present study, Özdemir et al. (2) showed that heart rate variability indices improved 1 day after PBMV. However, in another study, Ashino et al. (3) showed a reduction in sympathetic activity measured by muscle sympathetic activity in patients with mitral stenosis at 1 week after PBMV. Similarly, Yuasa et al. (4) demonstrated that muscle sympathetic nerve activity attenuated and cardiopulmonary baroreflex sensitivity improved 1 week after PBMV in patients with mitral stenosis. In the studies in which sympathetic activity is evaluated 1 week after PMBV, significant changes in electrocardiographic parameters just 1 day after PMBV seem to be too early. I believe that it would be more accurate and valuable if a sympathetic activity marker is measured and analyzed to determine significant correlations with electro- and echocardiographic parameters before and after the procedure.

In conclusion, this study can be a source of inspiration for further research in patients with aortic stenosis treated with transcatheter aortic valve implantation and hypertrophic cardiomyopathy treated with septal ablation because of similar mechanisms.

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Author`s Reply

To the Editor,

First, we would like to thank you for your interest in our article entitled, "Evaluation of Tp-e Interval, Tp-e/QT Ratio and Tp-e/QTc Ratio in Patients with Mitral Valve Stenosis Before and After Balloon Valvuloplasty" (1).

It has been demonstrated that sympathetic nervous system activation increases the Tp-e interval, and this increase immediately develops after stimulation (2). Previous studies have shown that sympathetic activity in patients with mitral stenosis (MS) reduced by balloon valvuloplasty after 1 week (3, 4). Moreover, it has been shown that the effects of reduction in sympathetic activity after balloon valvuloplasty occur much earlier. Özdemir et al. (5) showed significant improvement in heart rate variability parameters 1 day after balloon valvuloplasty in their study. The investigators attributed these results to reduced sympathetic activity and increased parasympathetic activity after balloon valvuloplasty. Similarly, our study showed that there was a significant reduction in Tp-e interval 1 day after balloon valvuloplasty in patients with severe MS. The reduction in sympathetic activity after balloon valvuloplasty in such a short time, such as 1 day, and the important parameters of ventricular repolarization are the distinctive results of our study .

As noted, our study could be more valuable if the sympathetic biomarker level was observed and correlated with the other parameters. Only those who had a sinus rhythm and did not have any additional cardiovascular disease were included in the study. Therefore, the number of patients in our study was relatively low. However, the analyses were performed after power analysis of the group sample sizes.

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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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