

Potential Prognostic Impact of Frequent Premature Ventricular Complexes

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We have read the clinical research article by Y.S. Chen et al. with great interest, wherein the authors have described about atrial and ventricular response to treatment of premature ventricular complexes (PVC).¹ Chen et al. suggested that PVC therapy effectively reduced ventricular arrhythmic burden. This may prevent left atrium dilation and impaired cardiac function. We agree with the authors regarding the important roles of PVC treatment. However, we have some reservations about the potential detrimental effect from atrial arrhythmia. Simultaneously, we would like to discuss some additional prognostic factors, guiding us whether the PVC therapy is indicated or not.

In this study, there was a statistically significant difference in atrial burden between observation and treatment group, and atrial burden in observation group is higher than that in the treatment group (Table 1). Previous studies have reported that increased atrial burden was associated with decrease in left ventricle ejection fraction.^{2,3} Moreover, atrial burden was also associated with the risk of atrial fibrillation (AF) and left atrium dilatation.⁴ Treatment of PVCs may prevent some cardiomyopathies and left ventricle dysfunction. However, the changes of left atrium and left ventricle ejection fraction can also be caused by underlying frequent atrial burden in patients without treatment.

For assessment of left atrium enlargement, it may

be unreliable and inaccurate to measure the linear dimension of left atrium, because the left atrium is not only three-dimensional structure with symmetric shape. The enlargement of the left atrium may not occur in a uniform manner. One-dimensional assessment is not sensitive to evaluate the change of left atrial size. Measurement of left atrial volume by two-dimensional or three-dimensional echocardiography can evaluate the change of left atrial size more accurately, and the result are more quantitatively reproducible.⁵

PVC induced cardiomyopathy is a reversible condition in which left ventricular dysfunction is mainly induced by frequent PVCs. However, it does not represent that all patients with frequent PVCs should develop left ventricular dysfunction. Therefore, some studies have reported that several confounding factors were associated with cardiac function impairment, including wide QRS duration, epicardial PVCs, presence of retrograde atrial activation and interpolated PVCs. However, the prognostic significance in some asymptomatic patients might be very limited because asymptomatic patients with frequent PVCs were usually underrepresented in these previous studies.⁶ This asymptomatic population still have the risk of undesired sudden death and fatal arrhythmias. Asymptomatic presentation was reported as one poor prognostic factor because it may delay the diagnosis and lower the treatment response.⁷ It is worth noting that there was also a statistically significant dif-

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Table 1. Differences in baseline characteristics of the 2 study groups

Characteristics	Observation (n = 40)	Treatment (n = 85)	p value
Asymptomatic (%)	19 (47.5)	24 (28.2)	< 0.01
Atrial burden (%)	0.04[0, 0.29]	0.01 [0, 0.07]	0.03

ference between observation and treatment in the aspect of asymptomatic patients in this study. In the observation group, the proportion of asymptomatic patients was significantly higher (Table 1).

The treatment for asymptomatic frequent PVCs remains controversial. Nevertheless, management of asymptomatic frequent PVCs largely depends on the existence of potential structural heart diseases or not. Potential underlying structural heart disease is an important prognostic marker, which indicates that further investigation and treatment of PVCs. Several characteristics of PVCs that prompt potential structural and electrical heart disease can also predict the poor prognosis, including PVC broader duration, PVC origin, PVC complexities, PVC morphologies, PVC frequencies and exercise related PVCs.⁸ In this study, the authors have presented most of the above PVC characteristics, except the relationship between exercise and PVC burden. Increasing PVC frequency with exercise should alert to some potential underlying electrical, ischemic or structural cardiac changes. On the other hand, reducing PVC burden or PVC resolution with exercise are usually idiopathic and benign. These benign PVCs may be originated from infundibular origin region, such as right ventricle and left ventricle outflow tract.⁹ Thus, the presence of exercise-related PVCs may not be clarified by 24 Holter monitor only, and it may be necessary to carry out exercise stress test (treadmill).

In addition to exercise responses, PVC substrates can be originated from scar formation in patients with either ischemic or non-ischemic cardiomyopathy. Echocardiography may have some limitation for diagnosing some hereditary, infiltrative disease and detecting the scar tissue formation. Cardiac magnetic resonance (CMR) may provide more accurate and additional information in this diagnostic conundrum. Clinically, some patients are not required to have abnormal left ventricular function as long as scarring was detected by CMR. CMR can help to detect some concealed cardiac abnormalities.¹⁰ It can also help us to decide the treatment strategies for some patients with frequent PVC burden.⁸

In summary, more atrial burden induced more left atrial remodeling in the observation group that may be partially contributed to poor prognosis. Left atrial volume is more accurate and reproducible for the assess-

ment of left atrium size, instead of linear diameter measurement. Moreover, exercise stress test and CMR may help us to exclude some potential structural, electrical and ischemic heart disease accurately, especially for some asymptomatic individuals without therapeutic intervention.

CONFLICT OF INTEREST

All the authors declare no conflict of interest.

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