Role of Electrocardiography in the Diagnosis and Management of Takotsubo Cardiomyopathy

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Since Sato et al first recognized the concept of reversible left ventricular apical wall motion abnormalities without coronary artery disease, and originally proposed the term “takotsubo cardiomyopathy” in 1990, this disorder has become accepted worldwide as a distinct clinical entity.1

Common abnormalities on the initial electrocardiogram (ECG) in takotsubo cardiomyopathy are ST-segment elevation and subsequent negative T wave.2 These ECG findings are similar to those of acute myocardial infarction. The differentiation of the 2 disorders on ECG is clinically important for the selection of appropriate treatment. The absence of reciprocal ST-segment elevation in the inferior leads or the absence of abnormal Q wave identifies takotsubo cardiomyopathy with a high sensitivity and specificity.3,4 The combination of the presence of ST-segment depression in lead aVR and the absence of ST-segment elevation in lead V1 is more useful for identifying takotsubo cardiomyopathy.5 Thus, the diagnostic value of ECG in takotsubo cardiomyopathy is well established,2–5 but its prognostic value remains to be investigated.

Previous studies have demonstrated acute complications of takotsubo cardiomyopathy, such as congestive heart failure, cardiogenic shock, left ventricular free wall rupture, septal perforation, apical thrombosis or life-threatening ventricular arrhythmias (Figure).6–8 A case series of small numbers reported that persistent ST-segment elevation might be associated with cardiac rupture.9,10 Indeed, in this issue of the Journal, persistent ST-segment elevation is found in case 2 of a blow-out rupture.11 QT interval prolongation is also commonly found during the early stage, and its relation to torsades de pointes has been recently recognized.12

In this issue of the Journal, Shimizu et al11 explore the J wave...
and fragmented QRS (fQRS) in patients with takotsubo cardiomyopathy, and evaluated their effects on clinical course and prognosis. J wave and/or fQRS were found in 9 (29%) of 31 patients during the very early stage. Subsequently, the J wave disappeared and fQRS diminished within 24h. These ECG findings were associated with severe myocardial damage assessed by myocardial scintigraphy or creatine kinase-MB. The J wave, especially, indicated cardiac death and/or ventricular tachyarrhythmias. In addition, the authors also point out that either a J wave or fQRS on the initial ECG might predict the clinical course and prognosis in patients with takotsubo cardiomyopathy.

The J wave plays a critical role in the pathogenesis of ventricular fibillation in patients with Brugada syndrome or early repolarization syndrome. More recently, this concept has been expanded to other structural heart diseases. Some of the fatal arrhythmias in the setting of acute myocardial ischemia may share a J wave-related electrophysiologic process. The fQRS represents distortion of signal conduction and the depolarization process within the ventricles, which is related to myocardial scar, fibrosis or ischemia. The fQRS is thought to be associated with ventricular tachyarrhythmias. Shimizu et al suggest that J wave and fQRS might be useful for detecting severe myocardial damage rather than arrhythmias. This finding might explain why the causes of cardiac death in the 2 patients with J wave and/or fQRS were low output syndrome and blow-out type cardiac rupture. It is clinically important for the selection and management of high-risk patients that the ECG can detect ongoing or subsequent severe myocardial damage at the time of admission. It is a matter of great interest whether early initiation of intensive medical therapy or intraaortic balloon pumping can improve the clinical outcome in these patients.

The current study adds to new insights to the important clinical issue of takotsubo cardiomyopathy. The J wave and fQRS are promising ECG markers of severe myocardial damage, but several problems have not been resolved. First, in this study, the J wave was observed in 5 (16%) of 31 patients. Its specificity for predicting cardiac death and/or ventricular tachyarrhythmias is high, but sensitivity is unsatisfactory. The fQRS was observed only in the no-lethal group, not the lethal group. Second, the J wave and fQRS appeared transiently only during the very early stage, and they disappeared or diminished within 24h. Although the time elapsed from onset to recording the ECG is not shown, these ECG findings appear to be time-dependent. There is a high possibility that the J wave and fQRS are not found any longer in patients with takotsubo cardiomyopathy if they are admitted beyond 24h after onset even if they have severe myocardial damage. In addition, patients with a J wave and/or fQRS had a higher incidence of ST-segment elevation and lower incidence of negative T wave than those without. It is unclear whether this finding is based in part on more severe ongoing myocardial damage or an earlier stage in patients with J wave and/or fQRS.

Despite these limitations, this study provides potentially important information that needs to be validated prospectively in a large number of patients. Further studies should be performed to clarify the prognostic value of ECG beyond initial diagnosis in takotsubo cardiomyopathy.

Disclosures

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References