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ScienceDirect

Journal of Electrocardiology 47 (2014) 684–689

JOURNAL OF Electrocardiology

www.jecgonline.com

Review

Electrocardiographic findings of takotsubo cardiomyopathy as compared with those of anterior acute myocardial infarction☆

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Abstract Takotsubo cardiomyopathy (TC) is a recently recognized novel cardiac syndrome characterized by transient left ventricular dysfunction without obstructive coronary disease, electrocardiographic (ECG) changes (ST-segment elevation and/or negative T wave) or elevated cardiac enzymes. Because the clinical features and ECG findings of TC mimic those of anterior acute myocardial infarction (AMI) with occlusion of the left anterior descending coronary artery, differential diagnosis has an important role in selecting the most appropriate treatment strategy. Especially in the acute phase, differential diagnosis is essential for deciding whether reperfusion therapy is required. Although it has been suggested that ECG does not allow reliable differentiation between TC and anterior AMI, several ECG criteria distinguishing TC from anterior AMI have been proposed. In this review, we discuss ECG findings of TC, especially in the acute phase, compare them with those of anterior AMI, and identify ECG features that may facilitate early recognition of this disease.

Keywords: Electrocardiogram; Takotsubo cardiomyopathy; Myocardial infarction

Introduction

Takotsubo cardiomyopathy (TC) is a recently recognized novel cardiac syndrome defined as follows: (1) transient hypokinesis, akinesis, or dyskinesis of the left ventricular mid segments with or without apical involvement; the regional wall motion abnormalities extend beyond a single epicardial vascular distribution; a stressful trigger is often, but not always present; (2) absence of obstructive coronary artery disease or angiographic evidence of acute plaque rupture; (3) new electrocardiographic (ECG) abnormalities (either ST-segment elevation and/or T wave inversion) or modest elevation in cardiac troponin; and (4) absence of pheochromocytoma and myocarditis [1–5]. Because the clinical features and ECG findings of TC mimic those of anterior acute myocardial infarction (AMI) with occlusion of the left anterior descending (LAD) coronary artery, differential diagnosis has an important role in selecting the most appropriate treatment strategy. Especially in the acute phase, differential diagnosis is essential for deciding whether reperfusion therapy is required. Fibrinolytic therapy in patients with TC is associated with an increased risk of bleeding, which is particularly problematic because most cases occur in postmenopausal elderly women who are prone to bleed [6,7]. In contrast to patients with anterior AMI, those with TC have complete resolution of left ventricular dysfunction within several days to weeks, accompanied by a generally good prognosis [1–5]. Early (i.e., before angiography), accurate, and noninvasive differentiation of TC from anterior AMI is thus a major clinical issue with important prognostic and therapeutic implications. The 12-lead ECG is the simplest, most widely available, initial clinical diagnostic test. To date, ECG characteristics of TC remain to be well defined. In this review article, we discuss ECG findings of TC, especially in the acute phase when ST-segment elevation is observed, compare them with ECG findings in anterior AMI, and identify ECG features that may facilitate early recognition of this disease.

Common ECG abnormalities: ST-segment elevation and negative T waves

Although recent review articles and other reports propose that ECG findings in TC are heterogeneous, the most common abnormality on initial ECG is ST-segment elevation or negative T waves [1–5]. However, the frequencies of ST-segment elevation (11%–100%) and of

☆ There are no relationships with industry and financial disclosure.

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http://dx.doi.org/10.1016/j.jelectrocard.2014.03.004
0022-0736/

negative T waves (17%–100%) have varied considerably in previous studies assessing ECG findings in TC [2,3,8]. The results of previous studies must be interpreted with caution because the elapsed time from symptom onset to recording ECG widely varied among studies and was not specified in some articles [2,8]. Mitsuma et al. [9] and Kurisu et al. [10] examined detailed ECG changes in TC and showed that TC was characterized by 4 ECG phases: phase 1, initial ST-segment elevation immediately after symptom onset; phase 2, initial T-wave inversion after resolution of ST-segment elevation from days 1 to 3; phase 3, transient improvement in T-wave inversion in the subacute period; and phase 4, a second deeper T-wave inversion persisting for several months. Given the time-dependency of ECG findings in TC, the reported heterogeneity of ECG findings may have in part resulted from the wide variability in time from symptom onset to ECG recording. Moreover, the perception of symptoms is subjective, and the timing of symptom onset is unclear in some patients or difficult to decide in others whose symptoms wax and wane. In patients with TC, time from symptom onset to presentation might be able to be estimated from ECG findings: ST-segment elevation indicates the acute phase (phase 1 as mentioned above), and negative T waves after resolution of ST-segment indicate the subacute phase (phase 2 as mentioned above) of TC. ECG changes in TC may reflect the pathologic nature of the myocardium and suggest the clinical phase [9].

ECG features of TC as compared with those of anterior AMI

The differential diagnosis of TC and anterior AMI in the acute phase is essential for deciding whether reperfusion therapy is required. Although previous studies have suggested that ECG does not allow reliable differentiation between TC and anterior AMI [1,3,8], several ECG criteria for distinguishing TC from anterior AMI have been proposed [11–15]. The criteria proposed by 4 articles, including our study, are shown in Table 1 [11–14]. The number of subjects was small in the studies by Ogura et al. [11] and Bybee et al. [12] and was considerably larger in our study [13] and the study by Tamura et al. [14] ECG criteria shown in Table 1 allowed TC to be distinguished from anterior AMI with high sensitivities (67%–100%) and specificities (69%–96%), probably because the subjects of most studies [12–14] were limited to patients who were admitted within 6 h from symptom onset. On the basis of these 4 articles [11–14] and previous reports, we summarize ECG findings in the acute phase of TC as compared with those of anterior AMI.

Less ST-segment elevation and the absence of abnormal Q waves

In both TC and anterior AMI, ST-segment elevation is observed mainly in the precordial leads, but the magnitude of such elevation is usually less in TC than in anterior AMI [1,3,8,11–13,15]. ECG criteria proposed by Bybee et al. [12] appear to have incorporated this finding. Some [3,11,13] but not all [8,12] studies have shown that the absence of abnormal Q waves, an ECG marker of irreversible myocardial necrosis, is more common in TC than in anterior AMI. The magnitude of the increase in myocardial biomarkers is smaller in patients with TC than in those with anterior AMI [1–3,8,10,11,13]. Scintigraphic imaging and cardiac magnetic resonance imaging have failed to document myocardial necrosis in patients with TC [1,2]. These findings suggest that TC is associated with less myocardial damage/necrosis as compared with anterior AMI.

The frequencies of no abnormal Q waves in TC (6%–85%) have widely varied in previous studies [1,3,8,11,13]. Nevertheless, in TC, Q-wave regression and R-wave reappearance are often observed, suggesting electrical stunning, even if abnormal Q waves occur in the acute phase. After the resolution of initial ST-segment elevation, negative T waves progressively develop in association with QT prolongation in both TC and anterior AMI [10,16], and such ECG changes are especially prominent in TC [16]. In patients with reperfused anterior AMI, the development of negative T waves in the acute phase has been attributed to viable but sympathetically denervated myocardium [17], because sympathetic denervation delays repolarization. These ECG findings in association with minor elevations of myocardial biomarkers relative to the degree of severe left ventricular dysfunction in the acute phase suggest that TC might be associated with a greater mass of viable, but sympathetically denervated myocardium (i.e., underlying electrophysiologic mechanisms might differ between TC and anterior AMI).

ST-segment deviation in inferior leads

ST-segment depression of ≥ 1.0 mm (0.1 mV) in inferior leads has been shown to be suggestive of the LAD coronary artery occlusion proximal to the first septal branch [18]. When the LAD coronary artery is proximally occluded, ST-segment depression in inferior leads can be caused by reciprocal changes associated with transmural ischemia in the high anterobasal region. In contrast, when mid or distal portion of the LAD coronary artery is occluded, ST-segment deviation in inferior leads is not influenced. Furthermore, when mid or distal portion of the LAD coronary artery that wraps around the apex and supplies a large portion of inferior wall is occluded, ST-segment elevation in inferior leads is thought to be often observed [19].

I) No ST-segment depression in inferior leads
Several small studies reported that the absence of reciprocal ST-segment depression in inferior leads can facilitate the distinction of TC from anterior AMI [11–13,15], because basal anterior myocardial dysfunction is absent in TC, and reciprocal ST-segment depression consequently does not occur in inferior leads. However, reciprocal ST-segment depression in inferior leads also does not occur in anterior AMI caused by mid or distal LAD coronary artery occlusion, as mentioned above. Specificity for the prediction of TC therefore decreases when this ECG finding is used [13,15]. Inoue et al. [15] demonstrated that no reciprocal change in inferior leads could differentiate TC from anterior AMI associated with a proximal occlusion of the LAD coronary artery, but