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T-Wave Abnormalities as ECG Signature of Myocardial Edema in NST-Elevation Acute Coronary Syndromes

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Introduction: Persistent T-wave abnormalities (TWAs) are often seen during the acute phase of non ST-segment elevation acute coronary syndromes (NSTE-ACS), but their pathophysiological significance has not been established.

Hypothesis: We hypothesized that persistent TWAs in NSTE-ACS correspond to the presence of myocardial edema by T2 cardiac magnetic resonance (CMR).

Methods: In 82 prospectively-enrolled patients with NSTE-ACS, 12-lead electrocardiography (ECG) and CMR with T2-weighted imaging (T2W) and late gadolinium enhancement were acquired before invasive coronary angiography. TWAs were defined as presence of inverted or biphasic T-wave in ≥ 2 leads on the ECG acquired closest in time to CMR examination. Myocardial edema by CMR was defined as ≥ 2 T2-positive left ventricular segments.

Results: Patients were studied at a median 24 (IQ 17 – 50) hours after admission. Of 79 patients with adequate T2W-CMR, 36 (46%) showed TWAs on ECG. The prevalence of myocardial edema was higher in those with vs. without TWAs (32/36 [89%] vs. 20/43 [47%], $p<0.001$). TIMI risk score, hemodynamics, ejection fraction, major cardiovascular risk factors, time-to-CMR, and troponin plasma concentrations, were similar in the 2 groups. By univariable logistic regression analysis, wall motion score index, and myocardial edema at CMR were the only variables significantly associated with TWA presence. By multivariable logistic regression analysis (adjusted for age and sex) edema at CMR was the only independent predictor of T-wave abnormalities (OR=14.6; 95% CI 2.9-73.2; $p<0.001$). TWA yielded 89% positive predictive value, with a specificity of 85%, and a sensitivity of 61% to predict edema at CMR.

Conclusions: This is the first demonstration that standard 12-lead ECG T-wave abnormalities seen during the acute phase of NSTE-ACS are related to the presence of myocardial edema. Presence of TWAs is a simple, highly specific, and moderately sensitive marker of myocardial edema. The electrophysiological mechanisms and treatment implications of this underlying pathophysiology warrant further investigation.