LETTER TO THE EDITOR

Abnormal P-Wave Terminal Force in Lead V₁ Predicts Left Main and/or Three-Vessel Disease in Patients with Non-ST-Segment Elevation Myocardial Infarction P-Wave Abnormality and Extensive Coronary Artery Disease

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Ann Noninvasive Electrocardiol 2015;20(6):612–613

To the Editor,

Recently, Li, et al. reported that abnormal P-wave terminal force in lead V₁ (PtFV₁) on discharge electrocardiogram was associated with an increased risk of long-term adverse cardiac events in patients with non-ST-segment elevation acute coronary syndrome.¹ They theorized that abnormal PtFV₁ might be associated with irreversible myocardial damage or residual ischemia of nonrevascularized vessels. However, detailed angiographic data were not reported. The extent of coronary artery disease (CAD) has been shown to be associated with worse prognosis in acute coronary syndrome population.^{2,3} Therefore, it is of clinical interest to see if abnormal PtFV₁ is associated with the extent of CAD.

We performed a retrospective analysis of 336 patients with non-ST-segment elevation myocardial infarction (NSTEMI) to clarify the association between abnormal PtFV₁ and the extent of CAD. This study complied with the Declaration of Helsinki and was approved by the institutional review board at our institution. We excluded patients with atrial fibrillation, atrial-paced rhythm, junctional rhythm, history of coronary artery bypass grafting (CABG), and those with nonobstructive CAD, defined as <50% stenosis. Electrocardiograms on admission were reviewed by two independent physicians. Abnormal PtFV₁ was defined as terminal

negative component of P wave that meets both depth ≥ 1.0 mm and duration ≥ 40 ms. Left main disease was defined as stenosis $\geq 50\%$, and three-vessel disease was present if there was stenosis $\geq 70\%$ in all three major epicardial coronary arteries.

Among 336 patients, 104 patients had abnormal PtFV₁. No significant difference was observed in baseline characteristics between those with and without abnormal PtFV₁. Patients with abnormal PtFV₁ had a higher rate of Killip class >1 on admission (28% vs 7%, $P < 0.001$), and left main and/or three-vessel disease (LM/3VD) than those without abnormal PtFV₁ (38% vs 25%, $P = 0.015$). There was a trend toward a higher incidence of in-hospital CABG in patients with abnormal PtFV₁ (17% vs 11%, $P = 0.098$). Patients with abnormal PtFV₁ had a higher, albeit statistical insignificant, in-hospital mortality (2.9% vs 0.9%, $P = 0.17$).

Our study demonstrated that abnormal PtFV₁ was associated with a higher incidence of LM/3VD in NSTEMI population. Our finding was consistent with a previous study that demonstrated the association between abnormal PtFV₁ and three-vessel disease in patients with Q-wave acute myocardial infarction.⁴ Previous study also has shown that abnormal PtFV₁ is associated with elevated left ventricular end-diastolic pressure (LVEDP).⁵ We speculate that extensive CAD

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DOI:10.1111/anec.12290

in abnormal PtfV₁ group resulted in elevated LVEDP, as indicated by a higher rate of Killip class >1 on admission, which subsequently led to abnormal PtfV₁. The extent of CAD may explain the increased cardiac events associated with abnormal PtfV₁ in Li's study, and it is interesting to know the extent of CAD in their study, which mainly consisted of patients with unstable angina. In conclusion, our study suggested that abnormal PtfV₁ on admission have a predictive value for LM/3VD in patients with NSTEMI.

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RESPONSE OF THE AUTHOR

Response: The Electrocardiographic Changes in Mid-basal (Inverted) Takotsubo Syndrome

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Ann Noninvasive Electrocardiol 2015;20(6):613–614

Dear Editor,

I read with great interest the recently published article by the authors Duran-Cambra et al.¹ published on November 4, 2014 in the journal ahead of print regarding systematic review of the electrocardiographic (ECG) changes in takotsubo syndrome (TS). The authors have carefully described the ECG changes in the classical TS, the time course of the ECG changes, and some other changes as QRS voltage attenuation. They have also described different ECG criteria for differentiation between anterior myocardial infarction and TS. With this letter, I would like to complement and highlight some other ECG changes, which also occur and are important to recognize in TS. The localization of the underlying pathology and the left ventricular dysfunction determine

the ECG changes in TS. In mid-basal TS, also known as inverted TS, the ECG findings are the reverse of the ST elevation and the giant T-wave inversion seen in mid-apical TS. It is characterized by ST depression localized in the majority of patients with mid-basal TS in inferior leads II, III, aVF, and precordial leads V₃ through V₆. These ECG changes are followed by peak upright T waves and QT prolongation in some patients. Mid-basal TS with the above-mentioned ECG changes is more frequently triggered by physical stress factors as stroke, pheochromocytoma, and therapeutic or accidental epinephrine administration but it may even be triggered by emotional stress factors. Waller et al.² have reported on a case of 46-year-old female with mid-basal TS triggered by subarachnoid hemorrhage and

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complicated by cardiogenic shock. The ECG in that patient revealed right bundle branch block and anterior precordial lead ST depression. Cho et al.³ have reported only peak upright T wave and prolonged QT interval (QTc 467 milliseconds) in a patient with mid-basal TS triggered by pheochromocytoma. Magri et al.⁴ reported on a patient with epinephrine-triggered mid-basal TS with ST sagging in leads II, III, aVF, V₅, and V₆, and nonspecific diffuse T-wave flattening. Follow-up ECG showed tall upright T waves in leads II, III, aVF, and V₃ through V₆. The tallest T waves were seen on day 3. Cacciotti et al.⁵ have reported on emotional-induced midbasal TS showing diffuse ST segment depression and evolved with tall and pointed T waves in leads V₂–V₄. Interestingly, Byer et al.⁶ in 1947 had reported on large upright T waves and long QT intervals induced by hypertensive encephalopathy; intracranial diseases are now well-recognized trigger factors for TS.

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