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Authors reply

Dear Editor,

We would like to thank our colleagues who read our study^[1] with such attention. The axis of the lead aVR is opposed to the left ventricular (LV) apical region, which provides unique information about global ischemia of this region.^[2] In our study, T-wave positivity at the lead aVR was shown to be closely related to LV thrombus formation after acute anterior myocardial infarction.^[1] Although Cetin et al. proposed that T-wave positivity in the lead aVR might reflect the presence of T-wave negativity at the inferolateral leads, only 1 patient in our study (Fig. 1b) demonstrated T-wave negativity at the inferolateral leads, which was found coincidentally. Consistent with previous studies,^[3,4] we also reported that the lead aVR might provide indirect information about reciprocal ischemic changes in the LV apical region. However, no association between T-wave positivity at the lead aVR and T-wave negativity at the inferolateral leads as a reciprocal change was found in our study. In addition, fragmented QRS (fQRS) on a surface electrocardiogram (ECG) reflects delayed ventricular depolarization time, most likely due to ventricular myocardial scarring, and has been shown to be a marker of adverse cardiovascular outcomes in several cardiovascular diseases.^[5,6] Although there are studies showing a significant association between fQRS and reperfusion failure and adverse in-hospital and long-term outcomes,^[7] none of our patients demonstrated a fQRS on the 48-hour ECG, though this might be due to ECG assessment without magnification. Long-term follow-up data is lacking in our study because of the cross-sectional design; fQRS might develop over time due to LV scarring and remodeling.

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