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"Takotsubo"-shaped cardiomyopathy manifesting as perioperative T-wave inversion: a reply

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In reply: We thank Chang, Ogawa, and Hanaoka for their interest in our article. These authors question whether the T-wave inversion found in our study is really caused by myocardial ischemia. There is no gold standard for the diagnosis of perioperative myocardial ischemia. A variety of perioperative factors, such as fiuid and electrolyte disturbances, drugs, and metabolic disorders can cause T-wave inversion mimicking ischemia. in our study, we considered postoperative T-wave changes to be compatible with ischemia when these T-wave inversions in multiple precordial leads correlated with segmental wall motion abnormalities on the echocardiogram. In particular, T-wave inversions preceded by a transient ST segment elevation strongly suggest myocardial stunning. However, we are aware that the evidence of myocardial ischemia was insufficient, as the authors pointed out, due to the discrepancy between the ECG changes and the hypokinetic area in some cases. Besides, our study lacked evaluation by coronary angiography.

The reversible asynergy of the apical wall motion associated with giant negative T-waves during the course of recovery found in our study resembled the clinical features of takotsubo cardiomyopathy, although the akinetic area of the left ventricular apex is more extensive in the latter compared to findings in our patients. Furthermore, the hyperkinesis in the basal segments that is characteristic of takotsubo cardiomyopathy was not seen on the echocardiograms in our patients, including those with ECG abnormalities initiated by ST-segment elevation. The balloon-like asynergy of the apex with basal hypercontraction, the typical morphology of "takotsubo"-shaped cardiomyopathy, must be confirmed by left ventriculography, and we cannot say anything definite regarding whether or not any patients with such as apical wall motion abnormality were included in our cohort. According to a multicenter retrospective study of patients with transient left ventricular apical ballooning [1], the results of coronary angiograms, even those done during the acute phase with ST elevation, showed on definite evidence of myocardial ischemia in any of the patients. Therefore, it is unlikely that takotsubo cardiomyopathy represents the results of a postischemic event, although the possibility of microvascular spasm still remains [2].

Prominent T-wave inversion may refiect excessive sympathetic stimulation. Myocardial ischemia results almost exclusively from an imbalance between myocardial oxygen demand and myocardial oxygen supply, and it can be triggered by both physical and mental stress. Changes in autonomic nervous system activity cause changes in coronary vascular tone through changes in oxygen demand. Takotsubo cardiomyopathy is also triggered by emotional and physical stress, such as noncardiac surgery, resulting in severe myocardial dysfunction, with clinical manifestations similar to those of catecholamine cardiomyopathy [1]. However, whether the T-wave inversion induced by sympathetic stimulation is associated with transient myocardial ischemia or microscopic injury to the myocytes is uncertain.

Although takotsubo cardiomyopathy and the transient left ventricular asynergy shown in our study have several common clinical features, further study is needed to clarify whether there is a common underlying pathophysiology in the two conditions.

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