Letter to the Editor

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Response by Jung et al to Letter Regarding Article, "Takotsubo-Like Myocardial Dysfunction in Ischemic Stroke: A Hospital-Based Registry and Systematic Literature Review"

In Response:

We thank El-Battrawy et al for their interest in our article entitled "Takotsubo-Like Myocardial Dysfunction in Ischemic Stroke: A Hospital-Based Registry and Systematic Literature Review."[1]

Takotsubo syndrome mostly presents with typical apical ballooning, but variants have been reported. Right ventricular (ie, biventricular) Takotsubo syndrome is gaining recognition because of the likelihood of poor outcome.[2]

In our study, right ventricular involvement was defined as any wall-motion abnormality (akinesis, hypokinesis, or dyskinesis) on transthoracic echocardiography. On the basis of the above definition, biventricular Takotsubo-like myocardial dysfunction (Bi-TMD) developed in 6 patients (26.1%), and it was included in only typical TMD group, not atypical. These patients had uniform involvement of the apical or mid- to-apical right ventricular wall. Those with Bi-TMD were older and had a higher wall-motion index score, suggestive of more severe left ventricular dysfunction, compared with the TMD group: median (interquartile range) is 77.5 (61–89) versus 72 (64.5–78) years (P=0.044) for age and is 2.13 (1.69–2.28) versus 1.50 (1.44–1.94; P=0.036) for wall-motion index score. Other echocardiographic and laboratory findings and clinical manifestations did not differ between the 2 groups. The Bi-TMD group showed a tendency toward poor clinical short-term outcome with no statistical difference: 3 (50%) versus 8 (47.1%; P=0.59) for neurological deterioration; 3 (50%) versus 6 (35.3%; P=0.538) for in-hospital mortality; and 5.5 (5–6) versus 5 (5–6; P=0.355) for modified Rankin Scale at discharge. In fact, the major cause of mortality was brain herniation (77.8%), attributed to cerebral or cerebellar edema, not cardiopulmonary dysfunction (11.1%), indicating that underlying disease plays a major role in patient outcome.[3] Additionally, 3 (50%) of the Bi-TMD group clearly had stroke onset before Bi-TMD, and the other 3 (50%) had unclear time of onset. Intraventricular thrombus formation, whether left or right sided, was not observed in the entire Bi-TMD group. Thus, Bi-TMD itself may not predispose to a higher risk of cerebral embolism. However, the clinical significance of Bi-TMD in ischemic stroke patients might still be underestimated or masked, because of the compelling effect of stroke as the underlying disease, considering that our study had a small sample size and associated limitations.

In stroke patients, medication during hospitalization was generally intended for secondary prevention according to stroke subtype based on TOAST classification (Trial of Org 10172 in Acute Stroke Treatment): large artery atherosclerotic, cardioembolic, small vessel occlusive, other cause, and undetermined cause (Table 1).[4] We preferentially selected antiplatelet agents in stroke attributed to large artery atherosclerosis or small vessel occlusion and anticoagulants in stroke attributable to cardioembolism; medications were continued after discharge. TMD or Bi-TMD was not treated with anticoagulants except in cases accompanied by left ventricular thrombus. Initiation and maintenance of standard therapies for heart failure including β-blockers, diuretics, nitroglycerin, or inotropic agents was left to the discretion of the attending cardiologist. These medications were continued until decreased ventricular function normalized. In actual practice, treatment of Takotsubo syndrome has been based on expert opinion and personal experience or case series, rather than guidelines.[5]

As noted in the letter, the pathophysiology and treatment of Takotsubo syndrome remain unclear. We agree that evidence-based treatment approaches and management strategies should be developed through further high-quality research.

Disclosures

None.

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(*Stroke*. 2017;48:e73. DOI: 10.1161/STROKEAHA.116.015998.)

Stroke
Journal of the American Heart Association

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Stroke. 2017;48:e73; originally published online December 20, 2016;
doi: 10.1161/STROKEAHA.116.015998
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Print ISSN: 0039-2499. Online ISSN: 1524-4628