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 appreciate the interest and comments from Nagai et al regarding our article, "Takotsubo-Like Myocardial Dysfunction in Ischemic Stroke: A Hospital-Based Registry and Systematic Literature Review."[1]

The pathomechanism of Takotsubo syndrome (TS) is poorly understood. Among several possible mechanisms, cardiovascular system changes caused by insular cortex damage and subsequent sympathetic overactivity have been proposed to mediate stroke-related TS.[2]

In our study, stroke preceded Takotsubo-like myocardial dysfunction in 11 patients and was assumed to precede Takotsubo-like myocardial dysfunction in 10 patients. Of 21 patients in total, 11 patients (52.4%) had insular involvement, of which 10 (90.9%) showing right-sided lateralization. In addition, right-sided propensity[3] and insular involvement[4] might also be related to stroke attributed to TS with a cardioembolic source. Although this group in our study consisted of only 2 patients: 1 with a left insular lesion and 1 with multiple territory lesions, these findings correspond to our results demonstrating that infarctions were more found in the right anterior circulation with specific dominant regions of the insular cortex. With regard to lesion location, lesions in whole insula were most prevalent in 8 patients (63.6%), followed by posterior involvement in 2 patients (18.2%) and anterior involvement in 1 patient (0.09%).

Through systematic review, 27 patients with clear chronological sequence of stroke before TS or unclear onset between stroke and TS were found. Among them, insular cortex involvement (14 [51.9%]; right, 5; left, 7; and both, 2) was increased although no distinct laterality of insular lesion was observed. In addition, our study and the systematic review showed that TS or Takotsubo-like myocardial dysfunction developed in patients with ischemic lesions in different areas, including the medulla, thalamus, internal capsule, putamen, cerebellum, and frontal and parietal lobes. The insular cortex has close connections with the limbic/paralimbic systems, thalamus, and hypothalamus, and the frontal, temporal, and parietal lobes.[2,5] Specifically, the insular cortex is known to be reciprocally connected with subcortical autonomic core centers, including the lateral hypothalamic area, nucleus tractus solitarius, and nucleus parabrachialis.[2,5] Therefore, dysfunction or imbalance of insular cortex circuitry, rather than damage to the insular cortex itself, would be more important for pathophysiology of TS or Takotsubo-like myocardial dysfunction in ischemic stroke.[2] Because of several limitations of the systematic review and of our study, well-designed studies are needed to support this hypothesis and further investigate the specific role of insular cortex damage according to laterality and location.

Disclosures

None.

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