Letter to the Editor

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

To the Editor:

We read the interesting clinical report by Jung et al,[1] who investigated clinical and radiological characteristics of ischemic stroke patients with Takotsubo cardiomyopathy (TC). Jung et al[1] found that ischemic lesions were mainly located in the right insular cortex (IC), which was the most frequently overlapped. However, there were little descriptions in the pathophysiology for the lesion laterality in the IC.

One possible mechanism underlying TC was increased sympathetic nervous system activity.[2] Elevated plasma norepinephrine concentrations have been observed during acute stroke. Large hemispheric ischemia caused by embolism in the middle cerebral artery might be associated with higher sympathetic nervous system activity because of subcortical autonomic network disruption.[2]

On the contrary, several studies have shown that the cardiovascular system is regulated by a central cortical autonomic network consisting of the IC, anterior cingulate gyrus, and amygdala.[3] Because the IC is located in the region of the middle cerebral arteries, its structure tends to be exposed to a higher risk of stroke.[3]

In an earlier study,[4] the right hemispheric inactivation induced an increase of high frequency of heart rate and the left inactivation induced an increase of low frequency of heart rate. These results suggest that left hemisphere is predominantly associated with parasympathetic autonomic tone, whereas right hemisphere is involved in sympathetic nervous system activity. Oppenheimer et al[5] reported that the degree of bradycardia was significantly greater on stimulation of the left posterior than the left anterior IC, whereas tachycardia was significantly greater on stimulation of the right anterior than the right posterior IC.[5] Thus, the possibility could be pointed out that the ischemia with left IC involvement or right posterior IC was related with increased sympathetic nervous system activity caused by relatively increased right anterior IC activation, which was suggested to be a pivotal pathophysiology of TC.

Until now, there have been few reports assessing the IC stroke and TC in the stand points of hemispheric laterality and regional specificity. The data presented in the article by Jung et al[1] would make much more importance if the exact mechanism underlying that relationship in relation with TC were provided.

Disclosures

None.

Michiaki Nagai, MD
Keigo Dote, MD
Masaya Kato, MD
Department of Cardiology
Hiroshima City Asa Hospital
Japan

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(*Stroke*. 2017;48:e74. DOI: 10.1161/STROKEAHA.116.015926.)

Stroke
Journal of the American Heart Association

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Stroke. 2017;48:e74; originally published online January 9, 2017;
doi: 10.1161/STROKEAHA.116.015926
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

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