Letter to the Editor

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Letter by Doğanay et al Regarding Article, “Preexisting Heart Disease Underlies Newly Diagnosed Atrial Fibrillation After Acute Ischemic Stroke”

To the Editor:

We read with great interest and excitement the recently published work of Rizos et al about newly diagnosed atrial fibrillation (AF) after acute ischemic stroke and relationship between preexisting heart disease. The authors stated that the stroke patients with newly diagnosed AF and with known AF share common cardiovascular risk factors, have similar echocardiographic findings, and suffer equally severe strokes. They also concluded that preexisting heart disease is the major cause of AF that is first diagnosed after stroke. In our letter, we aimed to emphasize another possible mechanism between acute stroke and AF, the contribution of epicardial fat thickness (EFT), and cardiac autonomic nervous system function.

In the literature, Acet et al examined 197 consecutive patients about measurement of echocardiographic EFT. Seventy-one patients had paroxysmal nonvalvular AF, 63 patients had persistent/permanent nonvalvular AF, and 63 patients had sinus rhythm. They also calculate neutrophil/lymphocyte ratio by using patient blood samples. The authors concluded that EFT and neutrophil/lymphocyte ratio are highly associated with types of nonvalvular AF independent of traditional risk factors. EFT measured by echocardiography and neutrophil/lymphocyte ratio seems to be related to the duration and severity of AF.

Previously published study by Carnevali et al concluded that epicardial fat deficiency in mice leads to an imbalance of the autonomic neural modulation of cardiac function in the sympathetic direction and to a potentially proarrhythmic remodeling of electric and structural properties of the heart. Additionally, Balcıoglu et al stated that sympathovagal imbalance, detected by heart rate variability and turbulence parameters, is associated with EFT. As sympathovagal imbalance is a predictor of arrhythmic events, epicardial fat may play an important arrhythmogenic role.

Heart rate variability mirrors cardiac autonomic nervous system function that influenced with ischemic events. Lurje et al demonstrated that heart rate variability is depressed after acute myocardial infarction. They concluded that in patients also on chronic treatment with β-blockers, an increase of heart rate variability was seen during the first weeks post myocardial infarction.

As a result, we think that the impairment of cardiac autonomic nervous system function and EFT may be helpful in explaining the results of the article by Rizos et al. We hope that the above mentioned items would add to the value of the well-written article of Rizos et al regarding the newly diagnosed AF after acute ischemic stroke and relationship between preexisting heart disease.

Disclosures

None.

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