Commentary

In this issue of *Journal of Neurosciences and Rural Practice*, Eşref Akıl and colleagues report the autonomic changes occurring in a scenario of acute cerebrovascular attack.[1] They studied the heart rate variability (HRV) parameters and catecholamine levels in 60 stroke patients and 31 healthy controls. The patient groups had relatively equal number of patients with right middle cerebral artery (MCA) and left MCA territory involvement. The authors have reported a significant increase in catecholamine levels in stroke patients compared to controls, especially in patients with right MCA territory lesions. There was a statistically significant increase in sympathetic tone when measuring HRV in patients with right MCA territory lesions when compared to controls. But there was no statistically significant difference in the HRV parameters among the patient groups. Furthermore, the authors have found a significant correlation between the catecholamine levels and the HRV parameters.[1] The current study brings out the subclinical autonomic dysfunction occurring in the mileu of damage to the central autonomic network (CAN), in particular the insular cortex.

The CAN is spread throughout the neuraxis and regulates the cardiac autonomic function. The important sites which integrate the cardiac autonomic activity are the hypothalamus, amygdala and insular cortex.[2] There are evidences for cortical asymmetry in regulation of autonomic functions.[3] The stimulation of right insular cortex leads to an increase in sympathetic tone, whereas an increase in parasympathetic tone was noted with left insular stimulations.[4]

The insular cortex is one territory which is supplied by the MCA and its occlusions leading to ischemic stroke is the leading cause of cerebrovascular attacks.[3] Thus, a stroke patient with MCA territory involvement will invariably have a damaged insular cortex. Autonomic disturbances pertaining to increased sympathetic tone are noted during the acute phase of the cerebrovascular attack.[5] The same picture was seen in the current study too. This is more so in patients with lesions of right insular cortex. These patients have increased incidence of cardiac arrhythmias following stroke. There are studies reporting increased incidence of supraventricular tachycardia in patients with right hemisphere infarction.[6] An increase in sympathetic or a decrease in parasympathetic tone in these patients might be an important cause for the arrhythmias.[7] The increased sympathetic activity after stroke significantly influences the prognosis of these patients. The cerebrovascular attacks involving the insular cortex lead to significant autonomic dysfunction and to increased incidence of sudden deaths.[3] This makes it necessary for the monitoring of cardiac autonomic activity in patients with stroke. These parameters can also be used as an adjunct to predict the outcome and prognosis in stroke.

Though there is literature available in the area of autonomic dysfunction and stroke, the uniqueness of this article is that the authors have correlated the various HRV parameters with the catecholamine levels. The article stands out for its stratified comparison of the location of the lesion and the HRV parameters. The increase in sympathetic tone observed in patients with right MCA territory involvement in the current study would have been due to the damage of the right insular cortex. The main limitation of this work is that the involvement of the insular cortex was not investigated, which would have given a greater comprehension of the neurobiology. While additional work will be required to attain a more complete understanding of the pathophysiology of autonomic dysfunction in stroke, the findings of the present study take us a step forward in understanding the subclinical autonomic dysfunction occurring after cerebrovascular attacks. This study is further evidence that monitoring of HRV parameters would help in understanding the autonomic imbalance or dysfunction prevailing in this patient population.

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