Parasympathetic overactivity: A manifestation of temporal lobe epilepsy

Sir,

Epilepsy may be associated with a variety of changes in the autonomic nervous system function. Especially temporal lobe epilepsy is associated with ictal and interictal autonomic dysfunction which is predominantly of sympathetic overactivity. Seizure-related autonomic hypo and hyperactivity modifies the function of various systems such as the respiratory, GIT (gastrointestinal tract), genitourinary, and most important cardiovascular system.[1] The increase in the heart rate (HR) and blood pressure (BP) frequently precede or accompany discharges during the ictal phase, while decrease in heart rate and blood pressure are rather rare.[2] A 35-year-old female presented to medicine ICU in unconscious state and was found to be in same state by relatives. No history of fever,

seizures, consumption of any poison or drug abuse. Past hospitalization 1 year before was for unusual prolonged unconscious state without no localizing sign or discernable cause. CT Brain was normal and EEG revealed abnormal awake waves arising from left temporal lobe. Since then she was regularly on antiepileptic drug. On examination she was E1V1M1of Glasgow Coma scale of 3, pulse was 40/min regular, systolic blood pressure was 60 mmHg, respiratory rate was 14/min, pupils were bilaterally contracted, and nonreacting, fundus was normal, and no focal neurological deficit. Investigation revealed normal blood sugar and complete blood count. Electrocardiogram was suggestive of sinus bradycardia with occasional ventricular ectopics. MRI brain showed normal study. Toxicological analysis of blood and gastric lavage was normal. She was given supportive treatment in the form of ionotropic support. Later on after 10-12 hrs she recovered completely. BP was normalized and the HR increased. Her autonomic function test was normal. EIectroencephalogram this time was suggestive of abnormal waves from left temporal lobe with secondary generalization.

Temporal lobe epilepsy presents with different autonomic nervous system (ANS) manifestation in adults and children. During acute epileptic seizures, epileptic seizures may manifest themselves as ANS dysfunction only or sudden cardiac death.[3] Neuroanatomic connections between the brain and the heart provide links that allow cardiac arrhythmias to develop in response to activation of discrete areas in the brain. The biological event is initiated by the neural aberration and lead to the secretion of catecholamine that may contribute to the induction of cardiac arrhythmias or damage. During seizures, tachyarrhythmias that too sinus tachycardia, are far more common than bradyarrhythmias.[4] There is evidence from animal experiments that interictal epileptogenic discharges enhance cardiovascular sympathetic tone more than parasympathetic outflow. Tachyarrhymias and hypertension are a common phenomenon and that localized discharges in the temporal lobe and medial frontal cortex are often associated with major cardiorespiratory and cardiovascular changes similar to changes during generalized seizures.[5]

Bradycardia and asystole result from increased parasympathetic flow through the vagus nerve, which originates in the nucleus ambiguous and dorsal nucleus of the vagus in the medulla. The connections of the cerebral cortex and subcortical areas to the brainstem vagal nuclei are not well defined. However, because left cortical stimulation and right vagal stimulation affect the heart rate in a similar way, the fibers from the left cortex must cross to stimulate the right brainstem vagal nuclei.[6] Hypotension develops in the course of a prolonged seizure and neurogenic pulmonary oedema is a well described phenomenon in status epilepticus.[4] Ictal and interictal epileptogenic activity spreading from temporal lobe areas may interfere with the cardiovascular modulation in neighboring structures of central autonomic control, such as the orbitofrontal cortex, amygdala, the singular gyrus and their pathways. Time and frequency domain analysis of HR variation and post-ganglionic innervation of the heart by the means of metaiodobenzylguanide (MIGB)-SPECT was studied in TLE patients and revealed predominant parasympathetic activity in patients with TLE compared to the control subjects and significantly decreased cardiac MIGB uptake, revealing altered post-ganglionic sympathetic innervation of the study patients compared to the control.[7] Sudden unexpected death in epilepsy (SUDEP) is common and the pathology lies on cardiorespiratory dysregulation that predisposes a patient to potentially fatal cardiac arrythmia and central hypoventilation or apnea. This case implies that if any patient with no past history presents with hypotension and bradyarrythmia after ruling the other common cause one should work out the patient to search for temporal lobe epilepsy.

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