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Rapidly Changing Tachyarrhythmia in Acute Stroke

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A B S T R A C T

Introduction: we report a 56-year-olds female with supraventricular arrhythmia due acute ischemic stroke without structural heart disease.

Case Description: A patient presented with sudden onset of lethargy, right hemiplegia, and global aphasia. There was previous history of stroke 1 year ago presented with left hemiplegia that recovered completely during 10 days. There was no history of comorbid illness. The brain CT revealed extensive hypodensity in left temporoparietal region suggestive of infarct without midline shift. General examination revealed hypotension and bradycardia that treated with dopamine that gradually recovered during 5 days thus infusion of dopamine discontinued, and muscular power in paretic limbs and aphasia was recovered. In 6th day of admission electrocardiographic monitoring of patient showed a rapidly changing tachyarrhythmia including sinus tachycardia, atrial fibrillation, and atrial flutter that quickly interchanged to another, without hemodynamic instability and alteration in mental status. Laboratory tests and TEE study were normal. During 48 hour arrhythmia relived spontaneously.

Discussion: Stroke can cause any type of cardiac arrhythmias that may not be constant.

1. Introduction

Approximately 75% to 92% of patients with intracranial bleeding or ischemic stroke develop new ECG abnormalities (Kevin A.B. et al., 2008). These may include, cardiac arrhythmias (CA), such as ventricular ectopic beats (VEB) or supraventricular ectopic beats (SVEB); ventricular arrhythmias (VA), especially ventricular tachycardia (VT); atrial flutter/fibrillation (AF); and repolarization abnormalities (QT interval prolongation, ST segment changes, large upright or inverted T-waves, and septal U waves) (Ornella D. et al., 2002). The Modern neuroimaging data, including positron emission tomography and functional magnetic resonance imaging, have revealed that a network consisting of the insular cortex, anterior cingulate gyrus, and amygdale play a crucial role in the regulation of central autonomic nervous system (Michiaki N. et al., 2010; Furio C. et al., 2004; Fred R. et al., 2008; Kevin AB et al., 2008). These data strongly indicate that the brain has a major influence on cardiac structure and function and that this is likely mediated through alterations in patterning of sympathovagal relationships (Fang L. et al., 2006).

2. Case Report

We are a 56-year-old female with sudden onset of lethargy, hemiplegia of right upper and lower limbs, deviation of corner of mouth to the left side and global aphasia within last 8 hours. There was previous history of stroke 1 year ago, presented with hemiplegia of left upper and lower limbs and aphasia that recovered completely during 10 days. There was no history of Diabetes, Congestive heart failure, High blood pressure, Ischemic heart disease (coronary artery disease) or other comorbid illnesses. General examination revealed

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a pulse rate of 35-40/ min and blood pressure of 90/50 mmHg, that was treated with infusion of dopamine (10-20μ/kg/min). The central nervous system examination showed lethargia, right upper motor neuron facial nerve palsy, and muscle power of upper and lower limbs were1/5, with right extensor plantar response. Examination of other systems was normal. Daily laboratory tests revealed normal levels of serum sodium, potassium, Calcium, magnesium, urea and creatinin. The initial serum troponin T and creatine phosphokinase levels were normal. The brain CT revealed extensive hypodensity in left temporoparietal region, suggestive of infarct without midline shift (Figure 1). Doppler sonography of carotid and vertebrobasilar artery revealed a floating thrombosis in right brachiocephalic artery after the origin of common carotid artery without remarkable stenosis. Gradually bradycardia and hypotension recovered during 5 days, thus infusion of dopamine was discontinued, and muscular power in paretic limbs and aphasia were recovered.

In 6th day of admission electrocardiographic monitoring of patient showed a rapidly changing tachyarrhythmia including sinus tachycardia, atrial fibrillation, and atrial flutter that quickly interchanged to another without hemodynamic instability and alteration in mental status (Figure 2). Laboratory tests rechecked, and levels of serum sodium, potassium, Calcium, magnesium, urea, creatinin, thyroid function tests and troponin I and creatine phosphokinase were normal. Transesophagial echocardiography (TEE) revealed normal LV and RV systolic and diastolic function, no LA and RA clot, with no regional wall motion abnormality (RWMA). During 48h without any specific antiarrhythmic treatment,

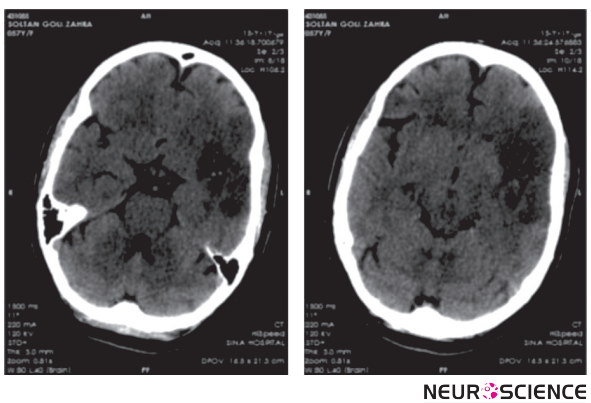

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Figure 1. Brain CT showed extensive hypodensity in left temporoparietal region suggestive of infarct. No midline shift and mass effect.

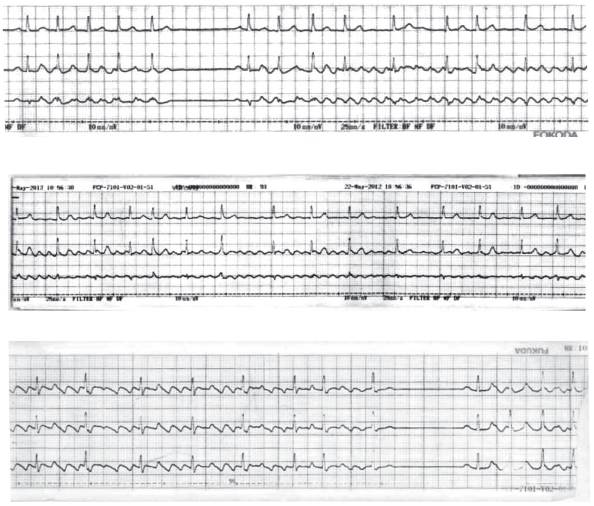

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Figure 2. Electrocardiogram showed a rapidly changing tachyarrhythmia including sinus tachycardia, atrial fibrillation, and atrial flutter that quickly interchanged to another.

arrhythmias relieved and normal sinus rhythm established. 5 days later, patient was discharged without any arrhythmia in electrocardiogram.

3. Discussion

We described a case of ischemic stroke with involvement of left temporoparietal region that reveals rapidly changing tachyarrhythmia. Cardiac abnormalities in patients with acute stroke were first reported in 4 patients in 1947 (Stephen O. et al., 1992). In humans, stroke in both hemispheres has been shown to produce changes in autonomic mechanisms, which leads to myocardial necrosis, arrhythmias, and even sudden death through related mechanisms. However, the localization of stroke may have differential effects (Sadberk L.T. et al., 1999). Lane et al have shown that right hemisphere infarction is associated with a greater number of supraventricular tachycardia, and they speculated that a decrease in cardiac parasympathetic activity in right sided infarction may cause the probable reciprocal rise in the sympathetic tone (Lane R.D. et al., 1992). It has been shown in humans that, lesions ablating part or all of the left anterior insula and its efferent connections and ablation of inhibitory circuminsular efferent's to the right insular cortex are of consequence with regard to determining cardiovascular outcomes after neurological damage (Stephen O. et al., 2006). There is evidence supporting long-term activation of the autonomic nervous system after stroke with increased levels of norepinephrine and pathologic nighttime blood pressure increases, a combination that represents an independent risk for future

cardiovascular and cerebrovascular events (Fred R. et al., 2008). Serious arrhythmic tachycardia (ventricular or supraventricular >130 beats/min) was more frequent than bradycardic arrhythmia (sinus-node dysfunction, bradyarrhythmia, or atrioventricular block °II and °III).12 Atrial fibrillation is the most common arrhythmia reported, occurring with a frequency of 9% (Giuseppe M. et al., 2008).

Although the exact mechanism for arrhythmia during stroke has not yet to be known, we propose some probable causes of tachyarrhythmia in this patient.

In the present case, there are two interesting findings that distinguish it from other patients, which have been reported so far. The first note was beginning time of arrhythmia based on literature, the common time period for occurrence of arrhythmia is in the first 48 hours, but in our case, arrhythmia occurred 6 days after admission.

The previous studies have analyzed patients with first over ischemic stroke, but our patient had history of right side stroke in one year ago, which could be the cause of this difference and late occurrence of tachyarrhythmia. Also other probable cause of late tachyarrhythmia may be presence of floating thrombus in right brachiocephalic artery that could be cause recurrent microemboli to right carotid artery which cause right hemispheric ischemia that indistinguishable in brain CT studies. This kind of microemboli can cause transient right insular ischemia with parasympathetic blocked, which increase brain sympathic tone and produce tachyarrhythmia in our patient.

Second, type of arrhythmia in this patient differed from similar cases reported previously. The most common reported type is atrial fibrillation (AF), but interestingly we detected a variable form of tachyarrhythmia which quickly interchanged to another. This variability in the type of arrhythmia may be due to the local stimulatory effects of existing floating thrombosis in great vessels (brachiocephalic artery) on the endothelium, or its dynamic retrograde effects on the left and consequently right heart.

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