Case Report

Cardiac Arrhythmias and Acute Cerebrovascular Events: A Case of QT Prolongation and Torsades de Pointes Early After Right Insular Stroke

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Stroke involving some areas of the cerebral hemisphere, such as insula, amygdala, and lateral hypothalamus, may cause changes in autonomic control of cardiac function. A 58-year-old woman presented to the emergency department for acute onset of left facial-brachial-crural hemiparesis and dysarthria. A brain CT scan showed subacute ischemic lesion with hemorrhagic infarction in right insular-rolandic cortex. Over the next few days ECG showed severe bradycardia with elongation of QTc, significative pauses (5 seconds), runs of nonsustained ventricular tachycardia and torsades de pointes. Drug induced and other several possible causes of elongation of QT and bradycardia such as hypokalemia, a history of heart failure, and structural heart disease were ruled out. The case confirms that insular cortex plays a major role in stroke-induced cardiovascular changes.

Key Words: Ischemic stroke—right insular stroke—cardiac arrhythmias—cardiac monitoring, QTc prolongation—torsades de pointes

Case Report

A 58-year-old woman presented to the emergency department for acute onset of left facial-brachial-crural hemiparesis and dysarthria. Clinical history was positive for arterial hypertension, rheumatoid arthritis, and previous minor stroke. Her medications immediately before hospital admission included losartan 50 mg, and methotrexate.7,5 mg every week. Last year she underwent cardiological investigations for atypical chest pain. Serial troponin I was negative. An echocardiogram showed moderate hypertrophy of left ventricle with good systolic function and normal valves and an Holter ECG did not show arrhythmias. ECG at admission showed new onset of atrial fibrillation (AF) with fast ventricular response. Brain computed tomography at admission showed no focal lesions; a brain computed tomography scan was again performed after 48 hours and showed subacute ischemic lesion with hemorrhagic infarction in right insular-rolandic cortex. Three days after the acute cerebrovascular accident, ECG continuous monitoring showed brady-tachy syndrome characterized by alternate AF with high ventricular response and sinus bradycardia with significant asymptomatic pauses (>2 seconds) and long QT (Fig 1). On day 4, patient presented torsades de pointes triggered by long QT with spontaneous regression (Fig 2). The day after, ECG showed severe sinus bradycardia (HR 30 bpm) with further prolongation of QTc (570 milliseconds), runs of nonsustained ventricular tachycardia and significant pauses (5 seconds). For this reason, a temporary pacemaker was first implanted and then, on day 6 after ischemic stroke, a definitive pacemaker was positioned. After definitive implantation of pacemaker, ventricular tachycardia was no longer detected at ECG continuous monitoring.

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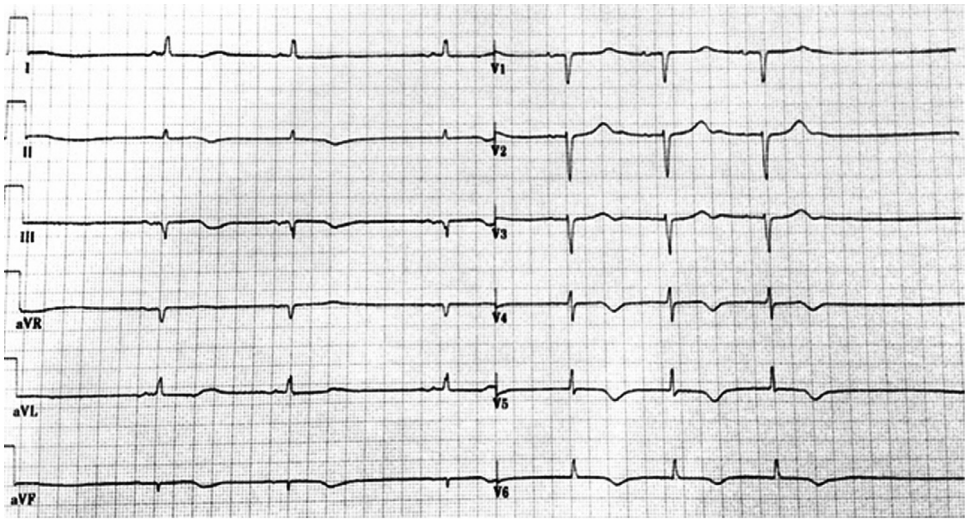

Figure 1. *Sinus bradycardia with significant asymptomatic pauses (>2 seconds) and QT elongation.*

Discussion

Stroke involving both cerebral hemispheres may cause changes in autonomic control of cardiac function.[1] However, some areas of the cerebral hemisphere seem to be mostly involved. In particular, the insular cortex within the middle cerebral artery territory is the most important cortical area involved in the control of both parasympathetic and sympathetic cardiovascular activity. Because middle cerebral artery occlusion is the most frequent cause of ischemic stroke, insular cortex is frequently involved in ischemic injury. Experimental animal studies demonstrated a crucial role of the insula in stroke-induced cardiovascular and autonomic disturbances such as changes in arterial pressure, heart rate, respiration, and adrenaline secretion.[8-9] Moreover, human studies demonstrated that stroke in the region of insula, especially the right one, lead to decreased heart rate variability and to increased incidence of sudden death.[2-10] These findings altogether demonstrate that right insular cortex plays a major role in the autonomic control of cardiac function and suggest that a stroke localized in this area may cause a change in sympathovagal balance in favor of increased sympathetic tone.[6-13] As a consequence of the increased sympathetic tone,

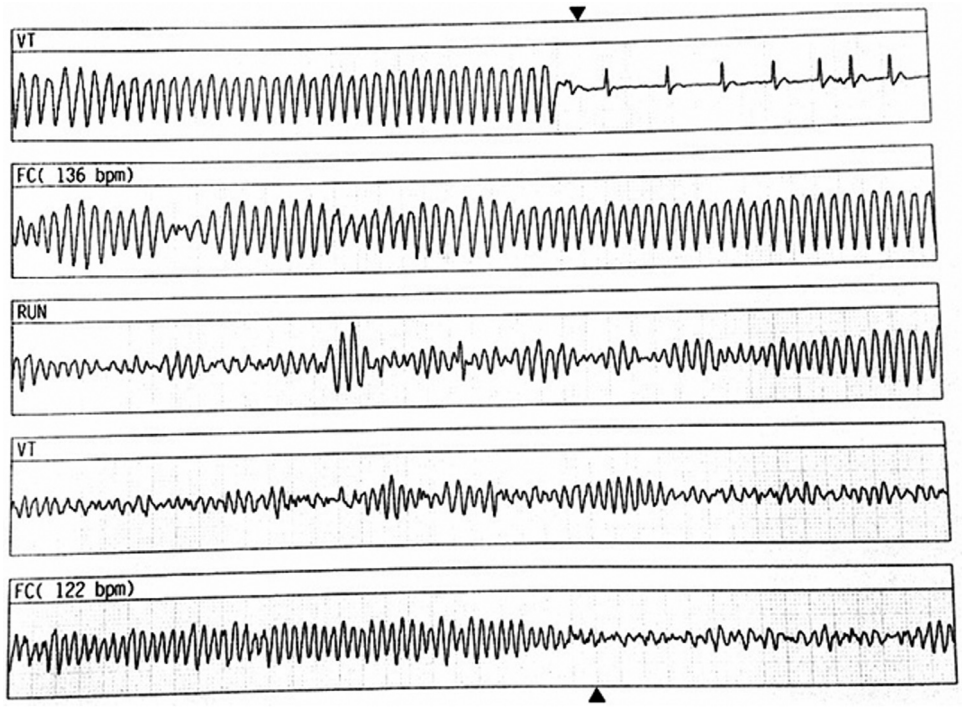

Figure 2. *Torsades de pointes presented on day 4.*

acute severe hypertension, cerebrogenic pulmonary edema caused by transient increase in pulmonary intravascular pressure and cardiac arrhythmias may occur. Cardiac arrhythmias include bradycardia, supraventricular tachycardia, atrial flutter, AF, ectopic ventricular beats, multifocal ventricular tachycardia, torsade de pointes, and ventricular fibrillation.[5-7] Furthermore, some patients seem to be more vulnerable than others for the development of ventricular arrhythmia after stroke. In particular, a number of studies have identified possible risk factors such as older age, stroke severity with larger size of brain lesion, and reduced heart rate.[14,15] Also, females have been shown to be more vulnerable than males in developing both hypokalemia and QT interval prolongation after acute stroke.[16] In our case, after ruling out several possible causes of QT prolongation and bradycardia such as drug therapy, hypothyroidism, congenital long QT syndrome and electrolyte alterations, a neurogenic cause of arrhythmia due to insular involvement was hypothesized.[5-17] This case confirms that insular cortex plays a major role in stroke-induced cardiovascular changes. Moreover, the increased risk of arrhythmias following an acute stroke in patient with newly developed ECG changes strengthens the concept that cardiac monitoring even in Neurology Department and the collaboration between Neurologist and Cardiologist is the key role to receive optimal cardiology therapy which may in turn improve quality of life and short-/long-term survival. Antiadrenergic agents that limit the effects of sympathetic response could probably be useful for the prevention or treatment of these arrhythmias. One the other hand, beta blockers should be skeptically administrated in patients with bradycardia or heart block. Finally, we believe that further studies are needed to better identified patients who carry higher risk of developing malignant arrhythmia after acute stroke and that can therefore benefit of more intensive cardiac monitoring.

Conflicts of Interest

All authors of the article confirm that there are no conflicts of interest.

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