

CASE REPORT

Cardiac Complications of Acute Stroke

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Introduction

Cardiovascular abnormalities are common after a stroke.¹ Disorders of the central nervous system cause a wide array of cardiovascular system dysfunction ranging from electrocardiogram (ECG) changes and transient myocardial dysfunction to sudden cardiac death. Because the majority of stroke patients have risk factors that predispose them to have pre-existing cardiac abnormalities,² it is important to distinguish whether cardiopulmonary abnormalities cause the stroke, are caused by the stroke, or unrelated to the stroke.

Case Report

A 39-year-old, African-American male presented to the emergency department with expressive aphasia. A computed tomography scan of the brain was negative. A magnetic resonance image confirmed the presence of

an acute peripheral infarct involving the posterior left frontal lobe inferiorly without other acute intracranial abnormalities. Further evaluation revealed a positive troponin T with a peak of 1.75 ng/dl (normal range = 0.00-0.06 ng/dl) indicating possible heart injury.

An electrocardiogram (Figure 1) showed the presence of peaked waves in lead V3 to V5 with 2 mm ST segment elevation in lead V3. The ECG findings suggested acute myocardial infarction. Emergent coronary angiography was negative for coronary artery disease. The ECG findings and the positive troponin were deemed to be due to the acute ischemic stroke. The patient was started on aspirin with resolution of his neurological symptoms and improvement of the ECG abnormalities in two days (Figure 2).

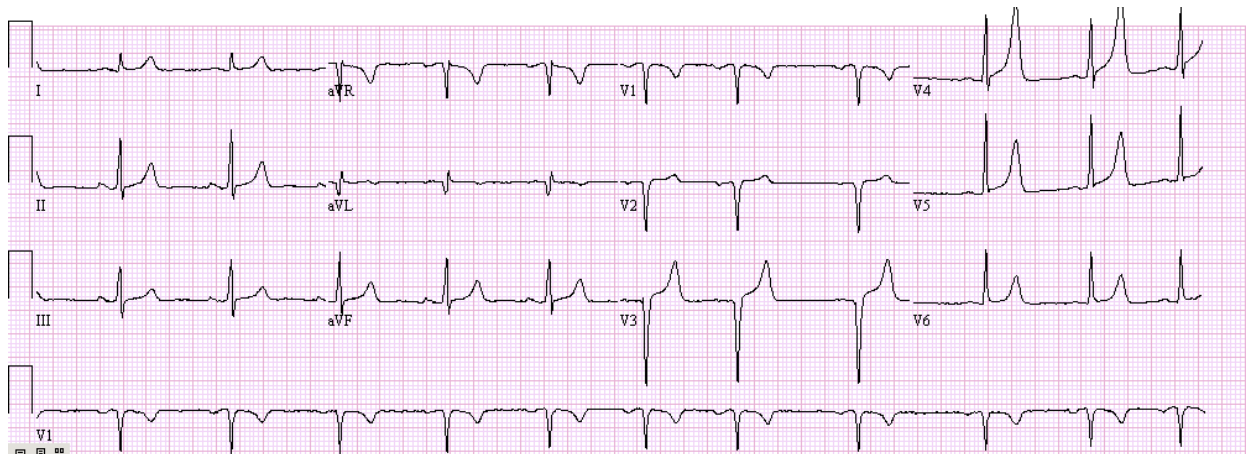


Figure 1. ECG showed peaked T wave in leads V3-V5.

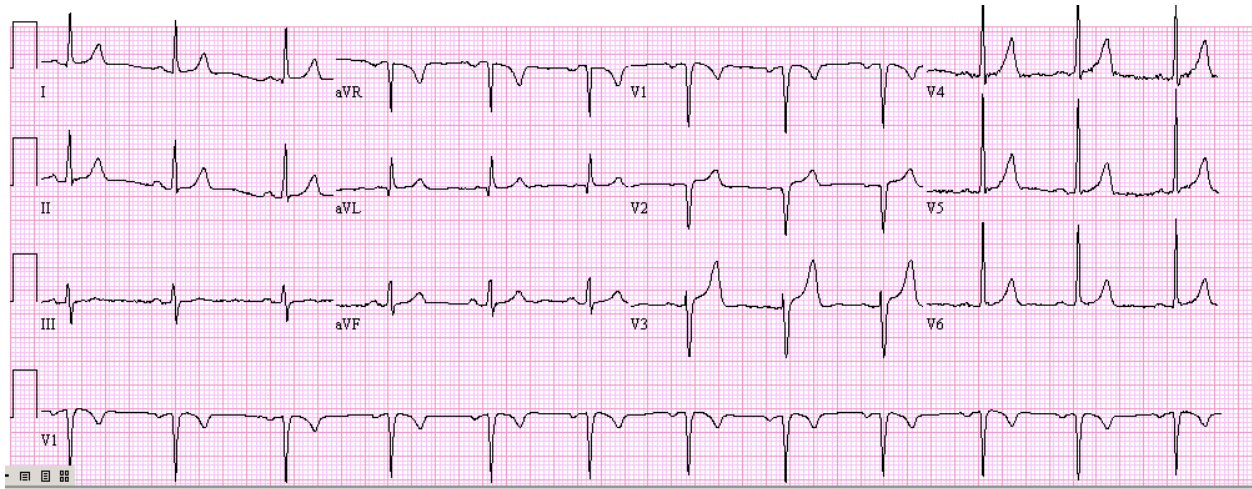


Figure 2. The follow-up ECG was normal.

Discussion

Two to six percent of all stroke patients die from cardiac causes in the first three months after ischemic stroke.¹ Regulation of the normal functioning of the heart by the central nervous system (CNS) is well recognized.^{2,3} In fact, the CNS regulates the heart rate, blood pressure, vasomotor tone, and cardiac output and plays an important role in myocardial metabolism and cardiac contraction. Further, the CNS can affect the cardiovascular system by altering fluid and electrolytes balance. Cardiac systolic function also is affected after an acute CNS event.⁴

Myocardial injury following a neurological event may be due to catecholamine-mediated cardiac dysfunction.^{2,5} An increase in intracranial pressure causes an acute surge of catecholamine release both systemically and at the neuronal synapses.⁶ This surge is hypothesized to affect coronary microcirculation and alter membrane permeability. The latter is manifested by an increase of intracellular calcium that leads to a continuous stimulation of actin and myosin filaments that leads to cell death. In addition, free radicals released by the metabolism of catecholamine contribute to membrane damage and cell death.²

Coronary vasospasm caused by catecholamines has been evaluated as a possible mechanism for cardiac dysfunction, but studies have failed to confirm this hypothesis.⁷ Other proposed mechanisms include disruption of the hypothalamic pituitary axis with depletion of thyroid hormones and cortisol.²

Cardiac dysfunction after a stroke is manifested by a wide variety of arrhythmias, ECG changes, elevated cardiac biomarkers, and hemodynamic alteration that can lead to cardiogenic shock and pulmonary edema.² Arrhythmias are common after stroke occurring in 78% of patients after a hemorrhagic stroke and 51% after an ischemic stroke.⁸ Patients may present with sinus bradycardia, supraventricular tachycardias, atrial flutter, atrial fibrillation, ectopic ventricular beats, multifocal ventricular tachycardia, torsades de pointes, ventricular flutter, and ventricular fibrillation.^{2,8} Cardiac arrhythmias are due to increased sympathetic tone and decreased vagal activity that are common in stroke.²

ECG changes were seen in 80% to 92% of patients with acute stroke.^{2,9} Abnormalities included ST segment elevation and depression, T wave abnormalities, U wave, prolonged QT

interval, and pathological Q waves. New T wave abnormalities appeared in approximately 15% of patients with acute stroke. Upright prominent and flat or inverted T waves have been described.² The suggestion that these abnormalities are neutrally-induced is supported by the observation that inverted T waves may normalize if brain death occurs.¹⁰ Nonspecific ST changes were the most common ECG abnormalities, excluding arrhythmia. ST changes are generally most apparent in the precordial and lateral leads and are usually transient.¹¹

Physicians should be aware of another ECG manifestation called the J or Osborn wave. The J wave is a slow upright deflection that appears in the ECG at the end of the QRS complex or early portion of the ST segment. It generally can be observed in hypothermic patients. However, brain injury¹² and subarachnoid hemorrhage¹³ have been reported to cause J waves. It is thought that heterogeneous distribution of a transient outward current-mediated spike-and-dome morphology of the action potential across the ventricular wall underlies the manifestation of the electrocardiographic J wave. The presence of a prominent action potential notch in epicardium, but not endocardium, is shown to provide a voltage gradient that manifests as a J wave or elevated J-point in the ECG.^{14, 15}

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Cardiac enzymes may be elevated after an acute intracranial neurological event. In an analysis of 149 patients with symptoms of acute stroke, 27% had elevated serum troponin.¹⁶ Elevated troponin T is a poor prognostic sign after acute ischemic stroke. Troponin elevations correlated with the severity of neurologic injury and cardiovascular abnormalities including left ventricular dysfunction, pulmonary edema, and hypotension requiring pressors. In-hospital mortality among patients with an elevated troponin T was significantly higher than patients with a normal troponin T.

Echocardiographic wall motion abnormalities have been described in patients with stroke.² Some patients develop a transient regional left ventricular dysfunction that mimics myocardial infarction. In the absence of significant coronary artery disease this condition is known as takotsubo cardiomyopathy.

Conclusion

A wide variety of cardiac manifestations occurs in patients with stroke. The majority of stroke patients have risk factors that predispose them to have pre-existing cardiac abnormalities. It is important to distinguish the cause of the cardiopulmonary abnormalities. As seen in the illustrative case above, such abnormalities may be the complications of acute stroke.

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