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Case Report

Internal Carotid Artery Occlusion and Supraventricular Tachycardia Leading to MCA/ACA Ischemic Stroke: A Case Report

Roper J* and Sokoloski P
Department of Emergency Medicine, Penn State Health Milton S. Hershey Medical Center, USA

***Corresponding author:** Roper J, Department of Emergency Medicine, Penn State Health Milton S. Hershey Medical Center, USA

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Abstract

Background: Stroke is defined by the American Stroke Association as brain, spinal cord and retinal cell death attributable to ischemia, based on neuropathological, neuroimaging, and/or clinical evidence of permanent injury. In the United States, stroke kills 140,000 people annually and costs an estimated $34 billion per year. Cerebrovascular accidents are a leading cause of serious long-term disability, resulting in reduced mobility in more than 50% of stroke survivors 65 years old and over.

Case Presentation: We present the case of a 54 year old woman who presented to an outside emergency room with symptoms of stroke while also in supraventricular tachycardia. Thrombolysis and control of the patient's heart rate resulted in notable improvement in the patient's stroke score and mobility.

Conclusions: In the setting of large vessel stenosis, any state causing decreased blood flow can result in hypoperfusion and ischemia. This case shows tachyarrhythmia was an important co-factor in the exacerbation of this patient's stroke symptoms. This patient also had several untreated risk factors for stroke and would have likely benefited from preventative measures, including anti-hypertensive and lipid-lowering medications, and cessation of smoking.

Keywords: Internal carotid artery occlusion; Supraventricular tachycardia; Ischemic stroke

Introduction

Stroke is a leading cause of death and disability in the United States, affecting an estimated 800,000 people per year. The vast majority of strokes are ischemic in nature (approximately 87%) compared to hemorrhagic (13%) [1]. Causes of brain ischemia include thrombosis, embolism or systemic hypoperfusion, with thrombosis estimated as the cause of one-third of all ischemic strokes [1,2]. Thrombosis can occur in large or small cerebral vessels. Common locations for large-vessel occlusions are cerebral vessel branch points, namely the origin of the internal carotid artery (ICA). An estimated 23% of ischemic strokes originate from carotid atherosclerosis or stenosis. The carotid arteries are the primary conduit for the anterior circulation, perfusing 80% of the brain, including the optic nerve, retina, frontoparietal and anterior-temporal lobes. The ICA supplies blood to a large vascular territory (anterior cerebral artery, middle cerebral artery, anterior choroidal artery) and its occlusion can range from asymptomatic to catastrophic [1,3]. Severe stenosis or occlusion of the ICA usually results in a decreased cerebral perfusion pressure (CPP), leading to autoregulatory compensatory mechanisms and collateral vessel recruitment. The ACA and posterior communicating arteries (PCA) allow the contralateral or unaffected ICA to perfuse the affected hemisphere. When autoregulation or the collateral circulation is affected, hypoperfusion and ischemia can result [4].

In the following case report, we discuss the clinical presentation of a patient with symptoms of a large vessel occlusion ischemic stroke, possibly unmasked by supraventricular tachycardia causing decreased cerebral perfusion in the setting of internal carotid artery occlusion.

Case Presentation

A 54 year old, Caucasian female with past medical history of anxiety, depression and active smoking presented to an outside emergency department with sudden onset of anxiety, left sided face, arm and leg weakness, left facial droop, slurred speech and tachycardia. EKG showed the patient to be in supraventricular tachycardia (SVT). A non-contrast head CT was unremarkable and the patient was within the window for IV TPA, which was given. CT angiogram showed complete occlusion of right internal carotid artery (ICA). CT perfusion showed abnormal perfusion in the right MCA and ACA territories with penumbra and delayed perfusion in the right hemisphere secondary to right ICA stenosis. SVT was refractory to adenosine and two bolus doses of Cardizem but responded to a Cardizem infusion. The patient's stroke symptoms were noted to improve with rate control and resolution of her SVT. Patient was then transferred to a tertiary care stroke center for possible neurovascular intervention. On arrival to the tertiary center, the patient's facial droop and weakness improved (NIHSS decreased to 3) and she was in a normal sinus rhythm. Neurosurgery was consulted, however with patient already improving following TPA and otherwise patent intracranial vessels with intact collateral circulation on imaging, she was not deemed a candidate for intervention.

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The patient was admitted to the neurosciences intensive care unit for post-TPA care and further stroke work up. Cerebrovascular duplex showed occlusion of the right bulb and internal carotid artery and minimal calcific plaque with possible thrombosis in the right bulb and ICA. A follow up brain MRI 24 hours later showed an acute infarct in the watershed area of the right hemisphere and no intracranial bleeding. Echocardiogram showed mild left ventricular hypertrophy, grade 1diastolic dysfunction, mildly dilated left atrium and mildly calcified aortic valve with mild aortic stenosis and moderate aortic regurgitation. Holter monitoring showed mostly normal sinus rhythm with occasional sinus tachycardia and rare premature ventricular and atrial complexes. Work up revealed the patient had hyperlipidemia, type 2 diabetes and hypertension. Patient was started on Plavix, a statin and Losartan.

The patient had an unremarkable hospital course and did not require physical therapy. She was discharged home in stable condition with primary care and Neurology follow-ups. At Neurology clinic follow up one month later, the patient reported ability to complete activities of daily living, cessation of smoking and being started on Metformin by her primary care physician. On exam, she had persistent left sided facial asymmetry and some left hand weakness but was otherwise neurologically intact.

Discussion/Conclusion

Cerebrovascular accidents are the fifth leading cause of death in the U.S. and over 20% have been associated with carotid artery stenosis or atherosclerosis [1,5]. Any state which decreases blood flow through stenotic lesion(s) increases the risk of hypoperfusion and ischemia, including thrombus, embolus to the area resulting in further restriction of blood flow, and systemic hypoperfusion [4]. In the above case, our patient presented with supraventricular tachycardia, which via decreased cardiac output could have caused a cerebral hypoperfusion state, namely in the vascular territory corresponding to her right internal carotid artery, where significant stenosis was already present. This is supported by the patient's weakness reportedly improving with rate control and termination of her SVT. The patient's ultrasound showed not only the degree of her carotid artery stenosis but also a possible thrombus in the area of stenosis. An acute thrombus could have also caused her acute ischemic stroke, with co-existing SVT an additional co-factor.

Cardiac arrhythmias following stroke are well reported in the literature and are thought to be due to brain ischemia causing disruption in the central autonomic system of the heart. Arrhythmias associated with stroke include tachyarrhythmias (ventricular, supraventricular) and bradyarrhythmias. Studies of acute stroke patients have shown arrhythmia incidence peaks at 24-48 hours after hospital admission and require intervention 10-30% of the time [6-8]. It is unlikely the patient in our case suffered an arrhythmia secondary to her cerebrovascular accident, considering she presented within hours of symptom onset.

The patient in our case also had risk factors unknown to her that increased her chances of stroke, including hypertension, hyperlipidemia and diabetes. Her lifestyle consisted of active, heavy cigarette smoking and a poor diet. Such factors were integral to the development of atherosclerosis and her flow-limiting proximal ICA lesion. Our patient would have benefited from preventative measures such as routine lipid screening, lipid lowering agents, an active lifestyle and not smoking.

In conclusion, the above patient suffered a large territory ischemic stroke due to her underlying, severe internal carotid artery stenosis, which acutely worsened in the setting of supraventricular tachycardia and possibly an acute thrombus. TPA in combination with arrhythmia control resulted in marked improvement in the patient's neurological deficits. This case also highlights the importance of preventative medicine in treatment of stroke risk factors. Our patient had several untreated risk factors such as diabetes, hypertension and hyperlipidemia, in addition to heavy smoking, all of which likely contributed to the pathogenesis of her ICA stenosis. This patient's acute stroke presentation shows how a patient high risk for cerebrovascular accident with underlying ICA stenosis experienced a watershed infarct, with supraventricular tachycardia an important co-factor in the acute stroke presentation.

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