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Cerebral Hypoperfusion as a Cause of Ischemic Stroke in a 46 Year-Old Female with Stanford Type A Acute Aortic Dissection: A Case Report- Katherine Rose A. Acosta - MD HB Heart and Vascular Institute

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Abstract

We report a case of a 46 year-old hypertensive female smoker with sudden, severe retrosternal chest pain associated with vague, intermittent epigastric pain as the initial presentation of Stanford type A acute aortic dissection (AAD), who developed decrease in sensorium and verbal output and focal extremity weakness few hours into optimal systolic blood pressure (SBP) and heart rate (HR) control as a manifestation of ischemic stroke probably secondary to cerebral hypoperfusion. Approximately 6% of elderly male patients with type A AAD have been reported to have concomitant ischemic stroke due to extension of dissection to the cervicocerebral arteries or emboli from the site of dissection. Few studies state the lack of clear evidence of cerebral hypoperfusion as a cause of ischemic stroke in patients with type A AAD. One study involving type B AAD proposed that a higher mean arterial pressure (MAP) on presentation and greater decline in MAP, as what happened to our patient, are associated risk factors. Cranial MRI/MRA of our patient revealed recent infarcts in the watershed distribution with normal carotid and intracranial vessels, without involvement of the arch vessel. She was successfully managed with beta- blockers and other antihypertensive medications maintaining SBP between 120-140 mmHg following the stroke and HR of 60 bpm. Her neurologic and cardiac status improved after two weeks. While strict BP and HR control is the cornerstone in the medical management of AAD, caution should be observed in lowering the MAP to prevent debilitating sequelae, such as ischemic stroke due to cerebral hypoperfusion. Aortic thoracic dissection (AD) is a serious cardiovascular disease. According to the Stanford classification; type A involves the ascending aorta and type B the descending distal to the left subclavian artery. Neurological complications secondary to AD are devastating. Ischaemic stroke and hypoxic encephalopathy are early-recognised complications of type A as the arch vessels can be involved AD. Although, late ischaemic stroke had been reported in 1.4–5% of patients with type B dissection, early stroke is very unusual as it cannot be simply explained by AD anatomical pathogenesis. We report two patients who presented with type B AD complicated by early ischaemic strokes. Work-up revealed significant cardiomyopathies in both patients but with left ventricle thrombus in one. In both patients the strokes were felt to be of cardioembolic origin. The dominance of neurologic symptom in the early stage of AD may make its early diagnosis difficult. Besides chest pain and widened mediastinum in chest x-ray, variable neurologic symptoms including left hemiparesis with asymmetric pulse and hypotension may suggest underlying AD. Stanford type A acute aortic dissection with ischemic stroke is a rare yet highly morbid presentation of sudden onset neurological symptoms. We present a case of a 57-year-old African American male brought to the emergency department with a witnessed syncopal episode, abdominal pain, right sided gaze preference, and left side weakness. Upon initiation of the hospital's "Code Stroke" response protocol, a computed tomography angiogram of the neck incidentally identified Stanford type A aortic dissection with extension into and occlusion of the brachiocephalic artery, occlusion of the right common carotid artery and right internal carotid

artery, and dissection flap propagation into the origin of the left common carotid artery. Postoperative magnetic resonance imaging of the brain and the entire spine demonstrated multifocal cerebral infarcts, as well as T10 - L1 spinal infarct. Despite provision of multidisciplinary intensive care unit level of care, the patient failed to demonstrate clinically significant neurological recovery and was transitioned to comfort care after 3 weeks of hospitalization. This case highlights the importance of considering acute aortic dissection as a potential etiology of acute ischemic stroke through the use of computed tomography angiography, as this diagnosis carries profoundly different implications for the consideration of thrombolytic agents and other emergent treatment modalities.