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Case Report

Severe Calcific Aortic Stenosis as a Rare Etiology of Cerebral Vascular Accidents: Case Report

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Abstract

Cerebral vascular accidents and transient ischemic attacks can have many causes. They are often attributed to factors such as uncontrolled hypertension, paroxysmal atrial fibrillation, hypercoagulable states, atherosclerosis of the carotid or cerebral arteries, or heart defects such as a patent foramen ovale or atrial septal defect. However, when these factors are excluded, the etiology of a stroke can remain perplexing. This case report suggests that embolisms from calcific aortic stenosis may serve as a cause for cerebral vascular accidents.

Keywords: *Calcific Aortic Stenosis; Embolism; Transient Ischemic Attack; Stroke; Cerebral Vascular Accident*

Abbreviations

CVAs: Cerebral Vascular Accidents; CT: Computed Tomography; TEE: Transesophageal Echocardiogram

Introduction

The common symptoms of calcific aortic stenosis are well established. Most are precipitated by exertion and result in dyspnea, presyncope, syncope and angina. The sequelae of calcific aortic stenosis are also well established and include arrhythmias and left ventricular hypertrophy that may progress to heart failure. However, a rarer, yet very serious consequence of calcific aortic stenosis can be cerebral vascular accidents (CVAs). Here we present a case of severe calcific aortic stenosis causing recurrent CVAs.

Case Report

A 61-year-old Caucasian male presented with new onset generalized weakness, slurred speech and difficulty maintaining balance during ambulation. He had a history of well-controlled type I diabetes mellitus, congenital bicuspid aortic valve with stenosis and calcification, and a 4.9 cm ascending thoracic aortic aneurysm. Physical exam was positive only for a grade III/VI harsh late peaking systolic murmur. Vitals were stable and lab abnormalities were significant for an elevated BUN/Cr at 28.6 mg/dL. This was the third time in five months the patient had presented with stroke symptoms to this hospital. A head Computed Tomography (CT) without contrast showed no signs of acute intracranial hemorrhage, but it did show a subacute infarction of the left frontal lobe in the territory of the Middle Cerebral Artery. Additionally, it demonstrated numerous previous infarctions throughout the cerebrum including the left lateral temporal lobe, left occipital lobe, lateral right frontal lobe, right temporal lobe and right parietal lobe. Magnetic Resonance Imaging confirmed CT results of the new left frontal lobe infarction and previous infarctions (Figure 1).

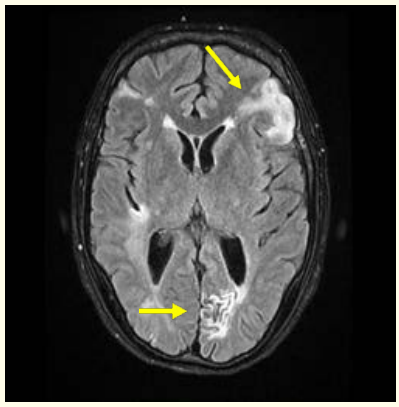

Figure 1: *MRI showing recent infarction of the left frontal lobe and a chronic infarction of the left occipital lobe.*

The patient's symptoms gradually resolved and causes for the patient's recurrent CVAs were investigated. Paroxysmal Atrial fibrillation had never been diagnosed in this patient; nonetheless, the patient was taking 5mg of Eliquis twice daily during the three presentations to this hospital. There was no history of hypertension. Carotid Doppler ultrasound showed no carotid stenosis bilaterally. Furthermore, the patient did not have any other atherosclerotic vascular diseases. Coagulation studies of prothrombin time and partial thromboplastin time were normal. Hypercoagulable causes of stroke were also excluded. A transthoracic echocardiogram with bubble study showed no patent foramen ovale or atrial septal defect. A transesophageal echocardiogram (TEE) confirmed severe calcification of the aortic valve, moderate aortic regurgitation, and a congenital bicuspid aortic valve with fusion of the right and left leaflets resulting in severe aortic stenosis. Mean aortic systolic gradient was recorded at 50.72 mm Hg, peak systolic gradient was 84.09 mm Hg, peak velocity was 4.6 m/sec and aortic valve area was 0.9 cm2 (Figure 2). No aortic valve vegetations were noted. Additionally, an ascending thoracic aortic aneurysm measuring 4.9 cm was present. The aneurysm was confirmed by a CT thorax with contrast which also showed the calcific aortic valve (Figure 3a and 3b).

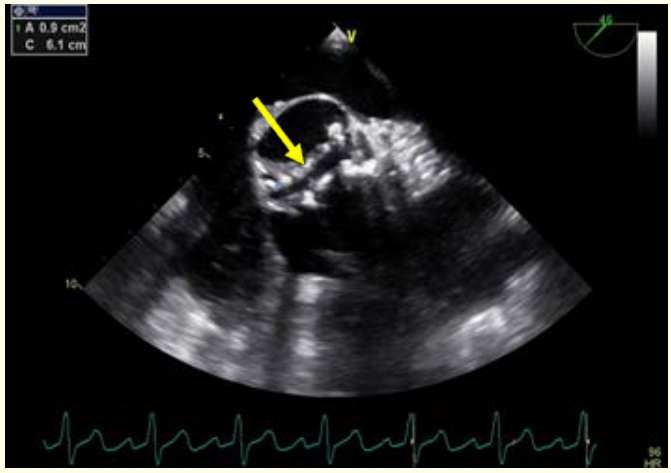

Figure 2: *TEE showing severe stenosis and calcification of the bicuspid aortic valve.*

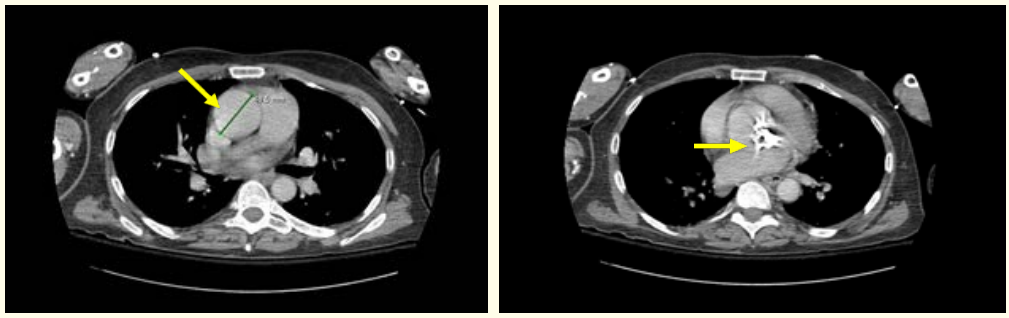

Figure 3A: *CT thorax with contrast showing an ascending thoracic aortic aneurysm.*

Figure 3B: *CT thorax with contrast showing calcific aortic stenosis.*

The patient successfully underwent open heart surgery replacing his aortic valve with a prosthetic heart valve and a tube graft replacing his ascending thoracic aortic aneurysm. The surgical and pathology report excluded atherosclerosis of the ascending thoracic aortic aneurysm (Figure 4a) as well as Lambl's excrescences and a papillary fibroelastoma. Heavy calcification was noted of the aortic valve extending all the way through the left ventricle and onto the anterior leaflet of the mitral valve (Figures 4b, 4c). Embolisms from the patient's severely calcific aortic stenosis were hypothesized as the etiology of his recurrent CVAs.

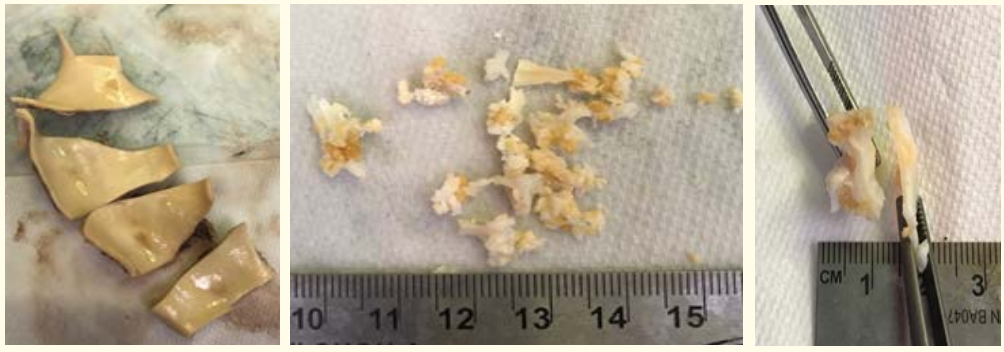

Figure 4A: *The ascending thoracic aortic aneurysm without atherosclerotic changes.*

Figure 4B: *Remnants of the aortic valve leaflets with severe calcifications.*

Figure 4C: *Segment of the thickened and calcified aortic valve compared to a normal segment of aortic valve.*

Discussion

Territorial brain lesions seen in this patient suggest an embolic source of stroke [1]. Previous case reports and literature reviews support the idea of calcific aortic valves producing emboli to the brain resulting in stroke [2-4]. Additionally, a 1963 study by the Mayo clinic followed 165 patients with calcific aortic stenosis over 21 years. At autopsy, they noted 45 instances of calcific emboli to various systemic organs in 31 patients [5]. Furthermore, recent research by Rodriguez., *et al.* looked at 2,665 subjects who were given both brain MRIs and echocardiograms. There was a statistically significant association between the presence of covert brain infarctions and either aortic or mitral valve calcification. People who had left sided valve calcification had a 33% greater risk for silent strokes after adjustment of potential confounders. Also, the degree of calcification directly correlated with risk of infarction [6]. It also appears that people with calcific aortic stenosis who experience stroke are prone to repeat strokes. The American Journal of Neuroradiology published a 2014 literature review

that found 43% of patients who had a CVA from a documented calcified embolism experienced at least 1 recurrent stroke [7]. Despite this evidence, calcific aortic stenosis itself is not generally considered to be a consensus risk factor for stroke in the medical community [8].

Factors that contribute to increased turbulent blood flow in the setting of calcification need to also be considered. Severe aortic stenosis, aortic regurgitation and an ascending thoracic aortic aneurysm, all of which were seen in our patient, are all factors that can contribute to non-laminar blood flow [9]. Concomitant severe calcifications may act as a nidus for thrombus formation and subsequent emboli during severe turbulent flow. Additionally, this type of turbulent blood flow may be the mechanism causing disruption of these calcifications and their subsequent release into systemic circulation. Further research is warranted to explain the exact mechanism.

Another factor that likely contributed to this case was the patient's dehydration. The three times the patient presented at this hospital for stroke symptoms, he had a BUN/Cr of 28.6 mg/dL, 31.1 mg/dL, and 30.7 mg/dL. Previous research demonstrates that a reduced circulating blood volume, in the setting of embolisms to the brain, results in impaired washout of the embolism [10]. This important consideration has not been considered in previous research of calcific aortic stenosis and its relationship to stroke.

Conclusion

Further research is needed to demonstrate that severe calcific aortic stenosis may cause CVAs. This will make it more widely known among clinicians and help them consider this etiology when they are faced with a perplexing stroke patient. Treatment recommendations and guidelines are also needed. Clinicians who have patients with calcific aortic stenosis should remember to educate their patients on the importance of maintaining adequate hydration. This topic is important because aortic stenosis with calcification presents later in adulthood and is progressive [11]. Thus, with an aging population in places such as the United States, the incidence of strokes related to calcific aortic stenosis may begin to increase.

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