Case Report

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Deep Crater in Heavily Calcified Aortic Valve Leaflet

A "Smoking Gun" for Embolic Stroke

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Abstract

The association of severe calcific aortic stenosis with clinically significant stroke has not been well established. This case vividly describes the relationship with clinical and pathological (gross and microscopic) findings in a 62-year-old man with a severely calcified bicuspid aortic valve. Eleven months prior to aortic valve surgery, the patient had stigmata of cerebral embolic events in the absence of any other embolic source. During the aortic valve replacement surgery for aortic stenosis, he was found to have a large atheroma on the aortic valve cusp with a crater containing friable debris in its center. These findings support the potential for embolic stroke in patients with severe calcific aortic stenosis. We recommend that the aortic valve be considered as an embolic source in patients with an otherwise cryptogenic cerebrovascular accident.

Key Words

Calcified aortic valve • Cerebrovascular events • Bicuspid aortic valve • Aortic valve replacement

Introduction

Aortic valve sclerosis is estimated to affect 21 to 26% of adults over the age of 65 [1]. Aortic sclerosis is associated with increased morbidity and mortality, particularly from myocardial infarction [1]. However, embolization from calcific aortic stenosis is an uncommon complication. We report a man who developed multiple cerebral vascular events due to embolization from a severely calcified bicuspid aortic valve.

Case Presentation

A 62-year-old man presented to the emergency department with acute onset of global amnesia, which lasted 72 hours. During this time, he was unable to recall events of the morning of the admission and was not oriented to time or place. He had no other cognitive or motor deficits. There was no antecedent history of fever, chills, night sweats, arthralgia, or weight loss. There was no history of atrial fibrillation, rheumatic fever, endocarditis, or drug abuse. He had a history of colon cancer and was cancer-free since 2001 (after a colectomy and adjuvant chemotherapy). His family history was significant for cardiac disease including a congenital abnormality requiring heart transplantation, aortic aneurysm, and sudden death in three relatives. Cardiac examination revealed a 2/6 systolic murmur at the right upper sternal border with regular rate and rhythm. There was no carotid bruit nor any clinical stigmata of endocarditis, malignancy, nor connective tissue disease. Neurological examination did not reveal any focal abnormalities.

MRI of the brain showed chronic lacunar infarcts bilaterally adjacent to the thalamus. Carotid Doppler

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ultrasonography and computerized tomography scan (CT) angiography of the intracranial and extracranial vessels revealed no evidence of cerebrovascular disease. Transesophageal echocardiography (TEE) showed moderate aortic stenosis, and a subsequent CT scan showed a 4.9-cm dilation of the proximal ascending aorta. Inpatient 24-hour electrocardiogram (ECG) telemetry was normal. Lipid panel revealed an Low-density lipoprotein (LDL) level of 123 mg/dL. The patient was discharged home with pravastatin

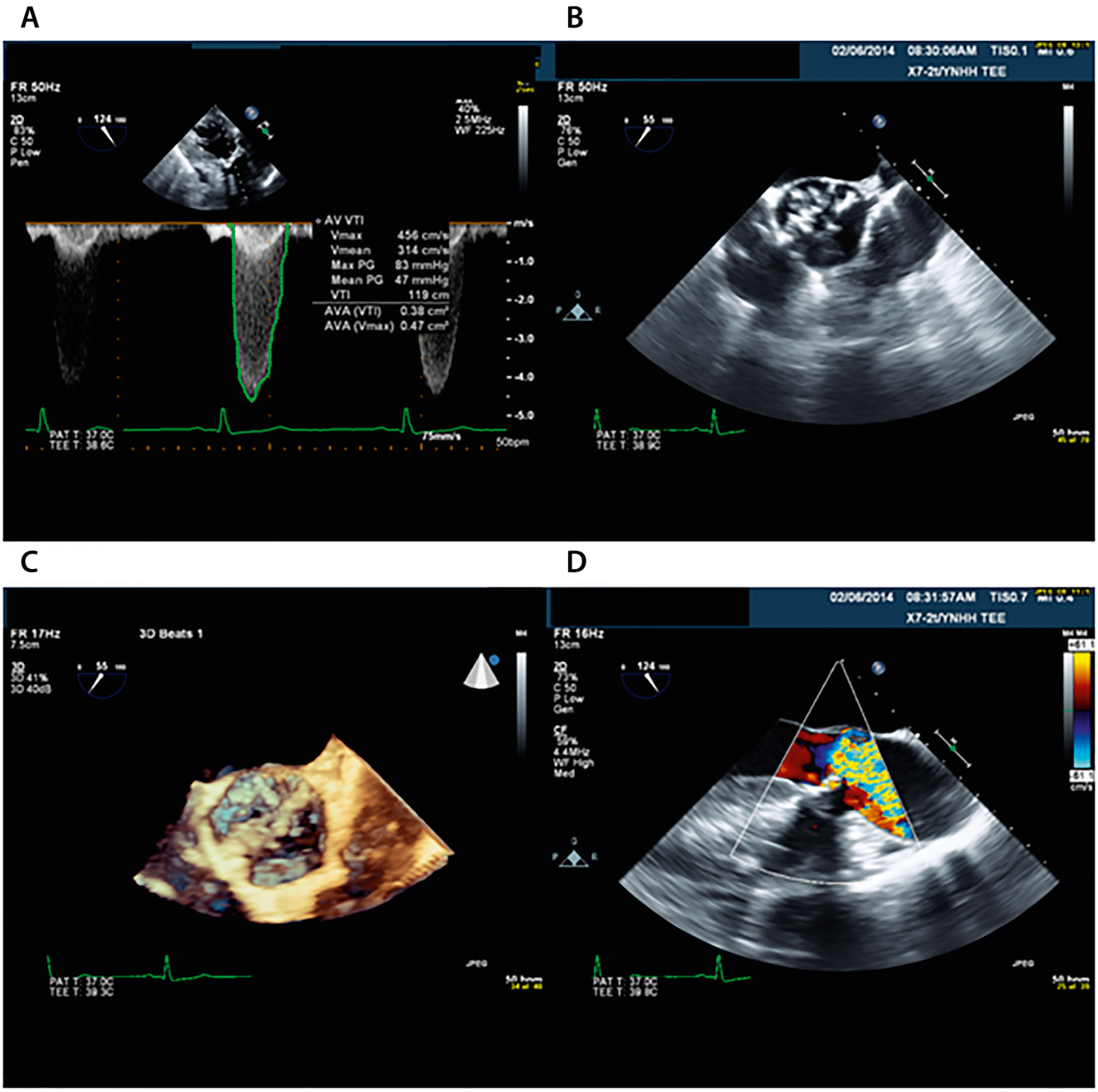

Figure 1. *Panel A*. Continuous wave Doppler profile from deep transgastric view shows an aortic valve area of 0.47 cm^2, mean velocity of 314 cm/s, and mean pressure gradient of 47 mm Hg. Trans-esophageal echocardiography (TEE) images of the aortic valve (*Panel B*) from short-axis view and (*Panel C*) from 3D reconstruction of aortic valve. Arrows point to heavy valvular calcification. *Panel D*. Color Doppler profile from long axis view shows highly turbulent flow behind aortic stenosis.

(40 mg/d) and aspirin (325 mg/d). An episode of dysarthria lasting 1 hour occurred 5 months later and prompted further evaluation. TEE showed a progression of aortic stenosis to a valve area of 0.8 cm^2.

One year after the original admission, the patient underwent aortic valve and ascending aortic replacement surgery. Intra-operative TEE showed an aortic valve area of 0.47 cm^2, a mean gradient of 47 mm Hg, and a peak gradient of 83 mm Hg (Figure 1A). Three-dimensional reconstruction imaging revealed a bicuspid valve with heavily calcified leaflets with nodular surfaces (Figure 1B and C). Color Doppler showed highly turbulent flow through left ventricular outflow tract and valve (Figure 1D).

Intra-operatively, the aorta was opened obliquely after establishing cardiopulmonary bypass, applying the cross clamp and administering cardioplegia. The aortic valve was found to be bicuspid with left-right fusion and displayed a minimal slit-like opening due to heavy calcification. In the noncoronary leaflet, a 1-cm ulcerated atheroma with a crater-shaped configuration containing reddish friable debris at its center was observed in situ (Figure 2A). The valve was excised (Figure 2B), and the annulus was debrided of exceptionally severe calcification extending down onto the mitral valve.

The bicuspid valve was sampled in cross-section, and the calcified atheroma with friable debris was seen histologically (Figure 3A and B).

After replacing the bicuspid valve with a 23-Mosaic valve (Medtronic, Inc., Minneapolis, Minnesota, USA), the ascending aortic aneurysm was resected and replaced by a 24-mm Hemashield graft (Maquet, Inc., Rastatt, Germany), and a deep hemi-arch replacement was performed.

Overall, the patient tolerated the operation well and was discharged in good condition on postoperative day 5. He resumed his home medications including pravastatin (40 mg/d) and aspirin (325 mg/d) and has since been entirely free of cerebrovascular symptoms.

Discussion

This case is presented to emphasize that aortic valve atheroma, in addition to those of the carotid arteries and the aortic arch, can be a source of embolic debris to the brain—with stroke as a consequence. Spontaneous calcific embolization from the aortic valve to coronary, retinal and cerebral vessels was described by Holley et al. [2]. Forty-five instances of calcific embolization were noted in 28 patients in this histologic study [2].

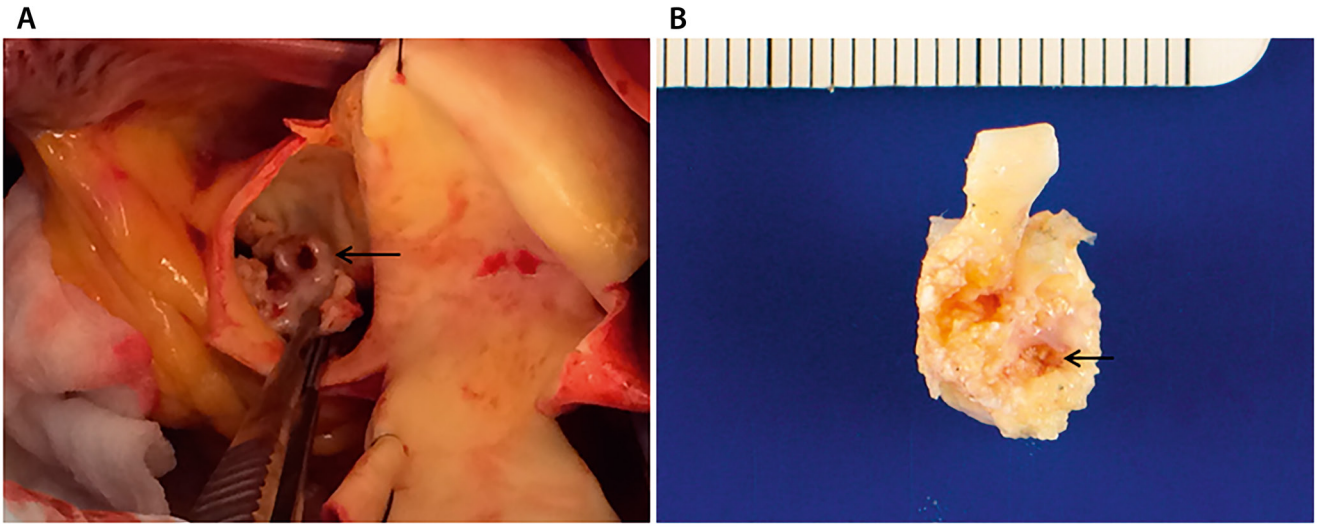

Figure 2. *Panel A.* Intra-operative photograph of the aortic root after incisions exposing extensive aortic valve calcification and 1 cm crater-shaped atheroma on luminal surface in situ (see arrow). The aortic valve is bicuspid with left-right fusion and there remained a minimal slit-like opening between the two immobile leaflets. *Panel B.* A segment of the aortic valve gross specimen sectioned to show severe calcification of the valve and crater-shaped surfaces (see arrow). Scale: one small bar is 1 mm.

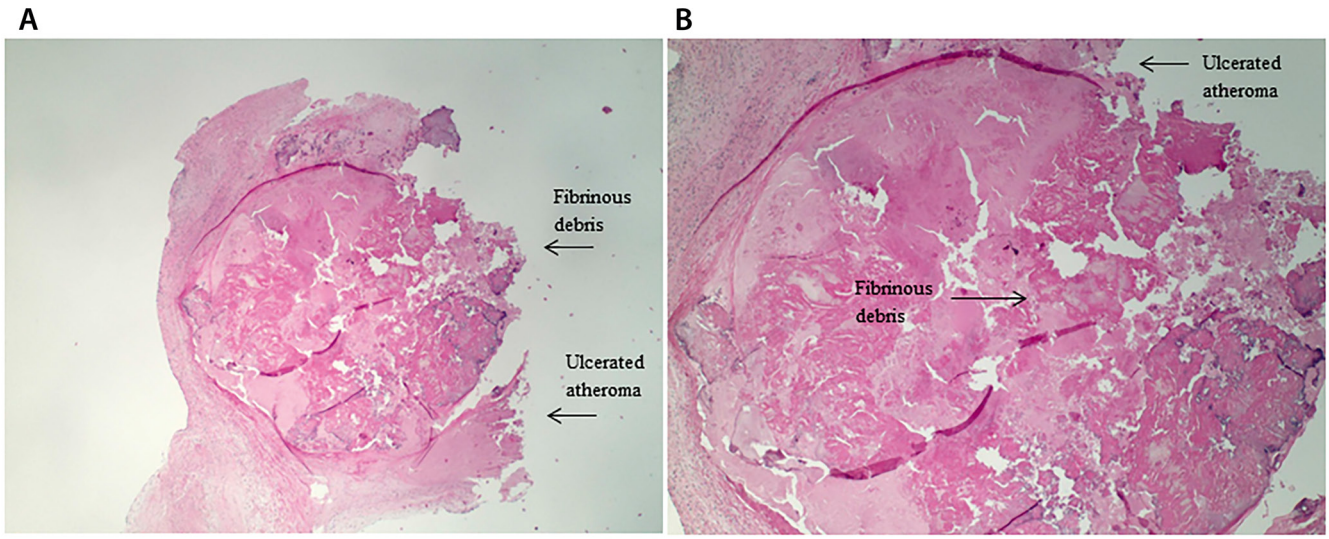

Figure 3. *Panel A*. Histologic section of the aortic valve at 40× magnification showing extensive myxomatous degeneration and calcification of the valve with atheromatous plaque on the luminal side. ***Panel B***. Histological section at 400× magnification showing the unstable atheromatous plaque.

In our case, the deep crater in the heavily calcified aortic valve leaflet constituted a "smoking gun" embolic source. During surgery, we were immediately impressed by the discrete crater, as well as its depth and its fresh thrombotic debris. It is the only apparent source for this patient's multiple neurologic events. No other potential source was identified on detailed pre-operative investigations.

The association between calcified aortic leaflet and stroke has been questioned based on cohort studies. In a study comprising 815 patients with aortic valve calcification, with or without stenosis, and 562 control subjects, the investigators did not find a significant increase in stroke risk in patients with calcified aortic valve [3]. In the Strong Heart Study, following 2723 patients, Kizer et al. [4] failed to find a significant risk elevation for stroke in patients with aortic valve sclerosis compared to those without. On the other hand, necropsy in 165 patients with calcified aortic stenosis revealed systemic embolization of calcific emboli to coronary arteries, cerebral, retinal and renal arteries [2]. A few reports have associated symptomatic stroke with calcified aortic valve in past. In four case reports, the aortic valves were bicuspid [5-8]. We present this case because of the striking direct visual evidence from the operating room followed by gross pathology and microscopic pathology.

This case presents striking visuals, including in situ gross and microscopic pathology images, vividly demonstrating the likely origin of cerebral embolic material in a deep ulcer crater in a severely calcified and stenotic aortic valve. These images not only support the pathophysiological link between cerebral embolism and calcified aortic valve, but also raise a clinically pertinent alarm, as calcified aortic valve is not an adequately recognized as a risk factor for stroke. It is debatable whether anticoagulation is an appropriate or sufficient management for these patients, rather than aortic valve replacement. Our case combined with the others suggests that the identification of a calcified bicuspid valve as an embolic source probably exceeds what medical treatment can offer [5-8]. We suggest that the threshold to consider surgical management should be low in patients with symptomatic cerebrovascular accident (CVA) events and calcified aortic valve with absence of other likely embolic sources.

Conflict of Interest

The authors have no conflicts of interest relevant to this publication.

Comment on this Article or Ask a Question

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