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CASE REPORT

Stroke secondary to calcific bicuspid aortic valve: Case report and literature review

Nitin Mahajan (MD, MPH)*, Vipin Khetarpal (MD), Luis Afonso (MD)

Department of Medicine, Detroit Medical Center, Wayne State University School of Medicine, Detroit, MI, USA

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KEYWORDS
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Summary
Background: Aortic valve calcification is common in rheumatic endocarditis, elderly patients, and congenital bicuspid aortic valve. It is not a recognized risk factor for stroke. We report a case of non-calcific embolization from calcified bicuspid aortic valve.
Methods: A 52-year-old male with bicuspid aortic valve presented with aphasia and right arm weakness of less than 3 h duration. CT head revealed hypodensity in the left middle cerebral artery (MCA) distribution and laboratory testing showed factor V leiden heterozygosity. The patient improved after intra-arterial tissue plasminogen activator (t-PA), but developed recurrence of right sided hemiparesis and silent myocardial infarction (MI). Cerebral angiography revealed clot in the left MCA. The patient received t-PA followed by transcatheter clot retrieval and was started on anticoagulation.
Conclusions: Embolization from calcific bicuspid aortic valves can lead to stroke and MI. Conservative management with anticoagulation for treatment of associated poststagnation thrombosis or aortic valve replacement as treatment is debatable. This patient was successfully managed with anticoagulation.

Introduction

Aortic valve calcification is common in rheumatic endocarditis, elderly patients, and congenital bicuspid aortic valve. It is not recognized as a risk factor for stroke [1]. We report a case of non-calcific embolization from calcified bicuspid aortic valve in a middle-aged patient.

Case report

A 52-year-old right-handed Caucasian male with a history of bicuspid aortic valve presented with

* Corresponding author at: Department of Internal Medicine, Wayne State University, University Health Center-5C, 4201 St Antoine, Detroit, MI-48201, USA. Tel.: +1 3472776497; fax: +1 3139666400.
E-mail address: nitin9696@yahoo.com (N. Mahajan).

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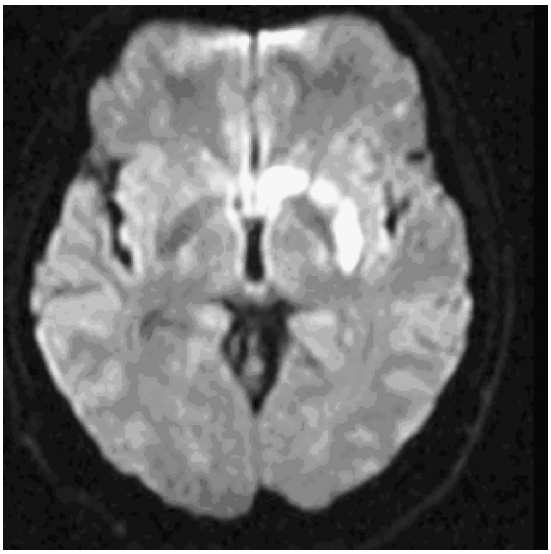

Figure 1 Magnetic resonance image shows infarction in the territory of left middle cerebral artery.

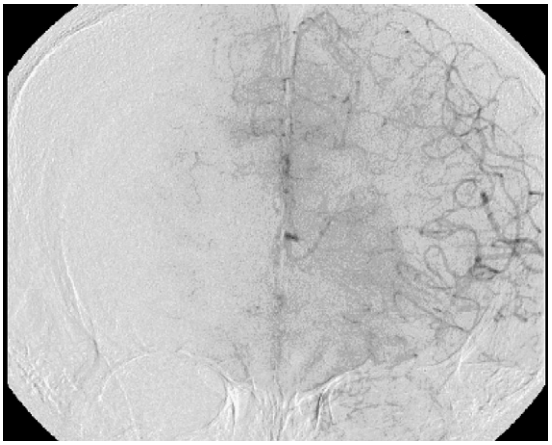

Figure 2 Angiogram reveals restricted perfusion in the distribution of left middle cerebral artery.

sudden headache, aphasia, and right arm weakness of less than 3 h duration. He had a history of 8-pack-year smoking but no other cardiovascular risk factors. On examination, vital signs were stable. A grade III/VI ejection systolic murmur was audible over the left sternal border. There were no peripheral stigmata of infective endocarditis. Neurological examination revealed pronator drift with weakness (power-3/5) in the right arm. Head CT was remarkable for a hypodensity in the left middle cerebral artery distribution. The following were normal: blood count, prothrombin time, erythrocyte sedimentation rate, antinuclear antibody, protein C, protein S, antithrombin III, plasminogen, with absence of lupus anticoagulant, anticardiolopin antibody, and activated protein C resistance. He received intra-arterial tissue plasminogen activator (t-PA) and neurologic deficits improved. The patient had dramatic improvement, but developed a recurrent episode of right sided hemiparesis and aphasia on the 10th day of hospitalization. Brain magnetic resonance imaging revealed restricted diffusion and infarction in the distribution of lenticulate striate branches and cortical branches of left middle cerebral artery (Fig. 1). On cerebral angiography, recurrence of embolic clot in the left middle cerebral artery with involvement of anterior temporal branch was reported (Fig. 2). He underwent infusion of intra-arterial t-PA with incomplete recanalization and transcatheter retrieval of residual clot. He also had a silent myocardial infarction with peak troponin I of 7.2. Electrocardiogram showed ST depression in inferior leads. The patient remained free of chest pain and was managed conservatively. Transesophageal and transthoracic echocardiography revealed a calcified bicuspid aortic valve with moderate aortic stenosis (Fig. 3) in the absence of any apparent clot or vegetation and preserved left ventricular ejection fraction. Regional wall motion was normal. The peak aortic velocity was 3.8 m/s, with a mean gradient of 32 mmHg. Non-invasive carotid studies were normal. Blood cultures were negative. Hypercoagulability work up revealed heterozygosity for Factor V leiden. The patient remained in normal sinus rhythm through the entire course of his hospital stay, and the ST depression in inferior leads recovered within 24 h. The clot formation and cerebral embolization were attributed to calcified bicuspid aortic valve in the presence of underlying predisposition to hypercoagulabil-

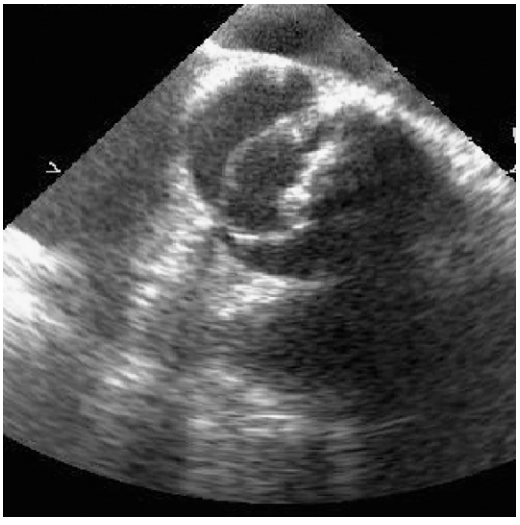

Figure 3 Image of bicuspid calcific aortic valve.

ity. He has some residual hemiparesis and mild expressive aphasia, but is otherwise doing well on anticoagulation.

Discussion

The prevalence of calcific aortic stenosis in the general population is estimated at 1—2% [2]. It is commonly seen in elderly patients and in patients with rheumatic endocarditis or congenital bicuspid aortic valve. Mitral, but not aortic valve, calcification has been well recognized as a risk factor for stroke. However, calcified aortic valves have been reported to be associated with systemic embolization to coronary, renal, retinal arteries, and the peripheral circulation [3]. The incidence of cerebral thromboembolism from calcified aortic valve is doubtless underestimated. There are only four reported cases of symptomatic stroke from calcified aortic valve [3—6]. In three cases, the aortic valves were tricuspid and in two the stroke followed a cardiac procedure (cardiac catheterization or aortic valvotomy). The embolization from calcific aortic valves varies from calcium deposits to non-calcific (thrombus) embolization. The amount of valvular calcium, the presence of mixed disease including, aortic valve stenosis with concomitant regurgitation, and bicuspid aortic valves (causing turbulence with release of adenosine diphosphate and partial thromboplastin from damaged erythrocytes) have been suggested as risk factors contributing to stroke [7,8].

Bicuspid aortic valve is the most common congenital heart abnormality. Bicuspid valves tend to become calcified with aging. Recognized complications include infective endocarditis, calcification, pseudo-aneurysm of the mitral—aortic intervalvular fibrosa, aortic stenosis and regurgitation [9]. Pleet et al. reported non-calcific embolic strokes in four patients with non-infected bicuspid aortic valves [8]. Presumably, valvular thickening increases the propensity to microthrombus formation and results in embolization of clot. Microthrombi with evidence of organization have been observed in 53% of stenotic aortic valves [8]. The stresses imposed by the blood against the physiologic obstruction (secondary to bicuspid aortic valve) leads to thrombus formation; the amount of thrombus has been correlated to the intensity of turbulence [10].

Heavy calcification of bicuspid valves may predispose to spontaneous dislodgement of calcific emboli. Magnetic resonance imaging scanning may show the extent of the ischemia, but often misses the calcific embolus. A gradient recalled echo technique is more likely to identify artifact from calcium, but is not included in many radiologists' standard cerebral magnetic resonance imaging protocols. CT angiography and spiral CT have been shown as modalities of choice to diagnose calcific embolism [11]. Besides early use of t-PA, recanalization with percutaneous angioplasty using Filtrap to capture thrombus reduces the risk of distal thromboembolism [12]. In the absence of a randomized trial comparing anticoagulation, antiplatelet therapy, and aortic valve replacement (of competent valves) treatment remains speculative.

Conclusion

Systemic thromboembolism including stroke may occur secondary to embolization from calcific bicuspid aortic valves. In a vast majority of cases, systemic thromboembolism tends to be asymptomatic. Treatment remains speculative. Whether conservative management with antiplatelet agents, anticoagulation for treatment of associated post-stagnation thrombosis, or aortic valve replacement (of competent valves) ought to be recommended remains unresolved due to the relative infrequency of this entity and the lack of trial data.

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