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Cardiovascular Sources of Embolic Stroke: I

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Cardiovascular Sources of Embolic Stroke: I

Craig M. Brodsky, MD

I. INTRODUCTION

Cerebrovascular accidents can result from both ischemic and hemorrhagic infarcts. Ischemic events account for 85% of all strokes. Atherosclerotic cerebrovascular disease is typically considered to be the cause of ischemic strokes; however, 14% to 30% of these events may be the result of cardiogenic emboli¹ (**Figure 1**). Cardiac sources of emboli include thrombi from the left atrium, left atrial appendage, or left ventricle; tumors; and valvular abnormalities including thrombosis, vegetations, and excrescences. Additionally, passage of thrombotic or other material from the venous system through an atrial septal defect or patent foramen ovale (PFO) can lead to systemic embolization. Finally, atherosclerotic debris on the thoracic aorta can embolize to the brain.

In almost all cases of cardiovascular embolism to the central nervous system, a neurologist is already involved with the care of the patient, and s/he requests a cardiology evaluation to help determine the etiology of the stroke. This review details the evaluation of a patient with known embolic stroke.

This is the first half of a 2-part review focusing on the various etiologies of embolic stroke. The first part will discuss embolic strokes caused by atrial fibrillation, atrial flutter, left ventricular thrombosis, myxoma, and aortic atherosclerosis. The second part will discuss embolic strokes caused by PFO, valvular excrescences, endocarditis, cardiac catheterization, and mitral valve prolapse (*see "Cardiovascular Sources of Embolic Stroke: II" in Hospital Physician Cardiology Board Review Manual, Volume 8, Part 5*). Diagnostic and treatment options for these disorders will be discussed. Where applicable, evidence for the current standard of care will be presented. The case presentations highlight various important aspects of the care of patients with embolic stroke.

II. ETIOLOGY OF EMBOLIC STROKE

In the absence of an embolic event, intravascular thrombosis is typically asymptomatic. In more than 80%

of patients, the cerebroembolic event is the first clinical manifestation of the thrombus. Peripheral embolization may occur in other patients. A small subset of patients with large thrombi can present with significant valvular obstruction. Because the thrombi are typically asymptomatic, an awareness of the clinical conditions associated with cerebral emboli may improve early detection of the thrombus and may lead to the initiation of measures that prevent the embolic syndrome.

ATRIAL FIBRILLATION

Atrial fibrillation, the most common cause of cardiogenic embolism, is associated with a 5-fold increase in the risk of stroke.² The chaotic rhythm in atrial fibrillation results in stasis in the atrium and atrial appendage. The atrial appendage is a multilobar structure with a reservoir-like shape; 80% of hearts have 2 or more lobes.³ As a result of this configuration, conditions that promote stasis or dilatation of the left atrium render the patient particularly prone to the development of thrombus (**Figure 2**). Rheumatic mitral valvular disease, especially mitral stenosis, is a condition with a particularly high incidence of thrombus in the left atrial appendage. Once formed, the thrombus may dislodge and embolize to the cerebral or peripheral vasculature.

Although considered a vestigial structure, the left atrial appendage normally contracts along with the rest of the atrium. During normal sinus rhythm, pulsed-wave Doppler interrogation of the appendage typically reveals a systolic velocity of 60 cm/sec \pm 14 cm/sec during atrial systole.⁴ In individuals with atrial fibrillation, the peak flow velocity of the appendage is dramatically reduced. Reduced contractility and dilation of the left atrial appendage are associated with the formation of thrombi.^{5,6} In those whose peak contraction velocity is less than 20 cm/sec, the risk of left atrial appendage thrombi increases from 5% to 17% and the risk of ischemic stroke increases by 2.6 times.⁷ In a group of 54 patients with nonrheumatic atrial fibrillation, those with systemic embolism had a lower peak contraction velocity than those without a history of embolism (25 \pm 19 cm/sec versus 39 \pm 23 cm/sec; $P < 0.05$).⁸ Spontaneous echocardiographic contrast can be noted in areas of reduced flow. This finding, often present in