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Review

Role of echocardiography in patients with stroke

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ABSTRACT

Investigation of potential embolic source is an important diagnostic step in treating patients with ischemic stroke and transient ischemic attack. Cardiogenic embolism has been estimated to be the causative factor in 15–30% of all cases of ischemic stroke. Cardioembolic strokes are generally severe and recurrence and mortality rate high. Various cardiac disorders including atrial fibrillation, ventricular thrombus, valvular heart disease, cardiac tumors, and structural heart defects can cause cardioembolic stroke. Although the aortic arch is not a cardiac structure, it is usually considered under source of cardiac embolism (cardioaortic source) and is reviewed in this article. Echocardiography (both transthoracic and transesophageal) is a widely used and versatile technique that can provide comprehensive information of thromboembolic risk in patients with stroke. This article reviews potential cardiac sources of stroke and discusses the role of echocardiography in clinical practice.

Contents

Introduction

Over 795,000 people in the USA experience new or recurrent strokes every year; 610,000 are first and 185,000 recurrent [1]. The overall prevalence of stroke is 3.0% in Americans [1], and that of silent cerebral infarction is higher, estimated to range from 6% to 28% [2–4]. Stroke is the third leading cause of death in the USA and the direct and indirect cost of stroke was $34.0 billion annually

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[1]. Cardioembolic stroke accounts for 15–30% of ischemic strokes and is more disabling than nonembolic-mechanism stroke [5–7], due to occlusion of larger intracranial arteries and larger ischemic brain volume. The clinical features most commonly associated with cardioembolic stroke are abrupt onset of maximal deficit, seizures, and accompanying hemorrhagic transformation [8]. These features, however, are unreliable to determine whether strokes are due to cardioembolic cause or not. The diagnosis of cardioembolic stroke frequently relies on the identification of a potential cardiac source of embolism. For this reason, echocardiography [both transthoracic (TTE) and transesophageal (TEE)] serve as cornerstones in the evaluation, diagnosis, and treatment of these patients.

Cardiac source of stroke

Intracardiac thrombus

Left atrial thrombus

A thrombus located in the left atrium (LA) or, more likely, the LA appendage (LAA) is the most prevalent source of cardioembolic events and is typically associated with atrial fibrillation (AF) (Table 1). Thromboembolic risk is similar between paroxysmal and persistent AF, but is strongly determined by associated cardiovascular risk factors [9]. The severity and duration of these risk factors influence the extent of LA enlargement and consequently the likelihood of AF-associated thrombus formation [10]. Risk stratification for stroke can be accomplished with clinical prediction instruments such as $CHADS_2$ and CHA_2DS_2-VASc scores. Currently, oral anticoagulation therapy is the most effective available prophylactic approach in patients with AF at high risk of thromboembolic events. However, this treatment increases the risk of major bleeding resulting in considerable discontinuance rates and lower quality of life. Newer surgical or percutaneous interventions (WATCHMAN, LARIAT, and Amplatzer devices) have been developed for stroke prevention in patients with AF [11]. These devices are typically used in AF patients who have high risks for thromboembolic events and cannot tolerate prolonged anticoagulation therapy. Although these interventions have shown feasibility, their long-term superiority to medical management remains a matter of debate.

The basis of imaging in AF patients centers on identifying the underlying cardiac causes of AF such as valvular heart disease, ventricular geometry, and function. Once an associated etiology of AF has been identified or ruled out, attention turns to details of LA anatomy and extent of LA enlargement. LA enlargement is related to thromboembolic risk, maintenance of sinus rhythm, and prognosis [12]. LA size can be expressed as either the anterior-posterior LA diameter or LA area measured according to the American Society of Echocardiography guidelines on chamber quantification [13]. The superiority of LA volume and LA volume indexed by body size as a more accurate measurement were shown in previous studies [12].

Table 1
Etiologies of cardioembolic stroke.

High risk	Minor or uncertain risk
LA thrombus (atrial fibrillation)	Atrial septal issues
LV thrombus	Patent foramen ovale
Myocardial infarction	Atrial septal aneurysm
Dilated cardiomyopathy	Valvular issues
Cardiac tumors	Mitral annular calcification
Myxoma	Calcific aortic stenosis
Papillary fibroelastoma	Mitral valve prolapse
Valvular issues	Valvular strands/Lambl's excrescence
Endocarditis	
Prosthetic valve	
Complex aortic atheroma	
Intracardiac device associated thrombus (left sided)	

LA, left atrium; LV, left ventricle.

Table 2
Comparison of the role of TTE and TEE for the patients with stroke.

TTE	TEE
Left ventricular thrombus	Left atrial appendage thrombus
Left ventricular function	Small vegetation (particularly associated with prosthetic valves)
Large vegetation	Abscess
Large tumors	Small tumors
Patent foramen ovale	Small patent foramen ovale and morphology
Atrial septal aneurysm	Atrial septal aneurysm morphology
LVAD associated thrombus	Spontaneous echo contrast
	Valvular strands
	Aortic arch atheroma
	LVAD associated thrombus

LVAD, left ventricular assist device; TEE, transesophageal echocardiography; TTE, transthoracic echocardiography.

Although LA thrombus can be identified by TTE, the sensitivity of TTE is low. Because of LA location immediately adjacent to the esophagus, TEE is considered to be the gold-standard technique to detect LAA thrombi, with values of sensitivity and specificity approaching 99% (Table 2). Fig. 1 illustrates the capability of TEE for providing a clear and diagnostic image of LAA thrombus. More specifically, TEE enables the assessment of LAA function and physiology with Doppler examination (low LAA emptying velocities and spontaneous echo contrast). Our previous study demonstrated the difference of LAA morphology and function among AF, atrial flutter, and fibrillation-flutter [14]. Both AF and fibrillation-flutter had lower LAA emptying velocities (17 ± 10 cm/s and 18 ± 8 cm/s, respectively) compared to atrial flutter (42 ± 18 cm/s). The development of real-time three-dimensional echocardiography (RT3DE) has enhanced our ability to interrogate the LAA, providing perspective relative to LAA anatomy as well as to discriminate between real and artifactual mass within the cavity. Furthermore, a recent study demonstrated that RT3DE is accurate in determining LA volume compared with two-dimensional TTE [15]. In the Stroke Prevention in Atrial Fibrillation (SPAF) III [16] and other trials, LAA thrombi, dense spontaneous echo contrast, LAA peak flow velocities $\leq$20 cm/s, and complex aortic plaques were independently associated with increased thromboembolic risk in AF patients. On the other

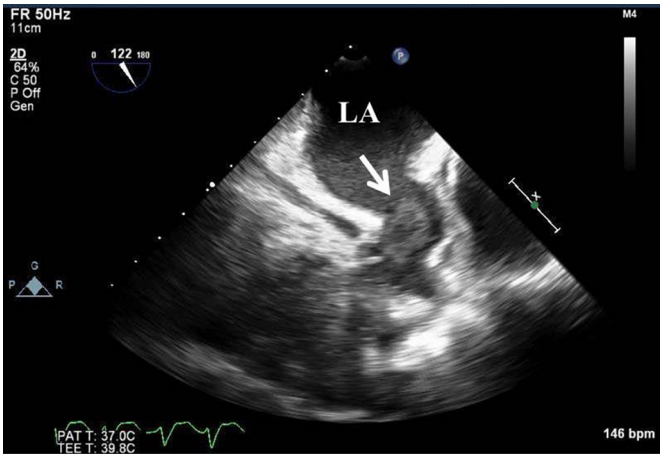

Fig. 1. Two-dimensional transesophageal echocardiography showing a left atrial appendage thrombus (arrow). LA, left atrium.