Diagnostic Yield of Echocardiography in Transient Ischemic Attack

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Background: Echocardiography is often performed to identify a cardiac source of embolism (CSE) causing transient ischemic attack (TIA). However, the diagnostic yield of echocardiography in TIA remains uncertain, and its role in routine evaluation of TIA is controversial. *Methods:* Patients with acute TIA were prospectively enrolled at 4 stroke centers. A CSE was defined using the Causative Classification of Stroke system; patent foramen ovale was considered a relevant CSE only if the patient underwent closure or was placed on anticoagulation. Patients with a known CSE at time of admission were excluded from analysis of the yield of echocardiography. *Results:* A total of 869 patients were enrolled at stroke centers, and 129 had a known CSE at presentation. Of the 740 remaining patients, 603 (81%) underwent echocardiography. A potential CSE was identified in 60 (10%) of these patients. The most common CSEs noted on echocardiography were complex aortic arch atherosclerosis and patent foramen ovale. History of coronary artery disease ($P < .001$), lack of prior stroke or TIA ($P = .007$), and presence of acute infarction on magnetic resonance imaging (MRI) ($P < .001$) were predictors of CSE on echocardiography. The yield of echocardiography was 29% in patients with both history of coronary artery disease and acute infarction on MRI, 14% with one of these features, and 5% with neither of these features ($P < .0001$). A CSE identified by echocardiography prompted initiation of anticoagulation in 15 of the 603 (2.5%) subjects. *Conclusions:* Echocardiography demonstrates a relevant CSE in a significant portion of patients with TIA. However, changes in antithrombotic therapy resulting from echocardiography are infrequent. **Key Words:** Echocardiography—cardioembolism—transient ischemic attack (TIA)—cardiomyopathy—aortic atheroma—patent foramen ovale (PFO).

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Introduction

Patients with transient ischemic attack (TIA) face a high short-term risk of stroke. In most population-based studies, this risk exceeds 10% within the first 3 months.[1] Prevention of stroke in these patients requires identification of the underlying TIA mechanism, as optimal prevention strategies vary based on the cause of TIA. Although echocardiography is considered a standard part of the evaluation of patients with stroke to identify high-risk cardioembolic sources, its use in TIA patients is more variable. This likely reflects both uncertainty about the yield of echocardiography in TIA and also challenges in expeditiously and efficiently obtaining echocardiography.

Compared to stroke, diagnostic certainty is often much lower with TIA, exemplified by the poor inter-rater agreement for TIA diagnosis even among vascular neurologists.[2] This raises important concerns about whether patients undergoing evaluation for suspected TIA are likely to have abnormalities on echocardiography with sufficient frequency to justify routine testing. Additionally, patients with TIA are increasingly likely to be evaluated using a rapid Emergency Department (ED) or observation unit triage protocol, in which urgent brain and vascular imaging is obtained, and those felt to be low risk are discharged. Inclusion of echocardiography in such protocols is often limited by its lack of availability in these settings, particularly outside of routine business hours.

The aims of the present study were to evaluate the yield of echocardiography for identifying a relevant cardiac source of embolism (CSE) in patients with suspected TIA, to determine how often changes in management occurred based on echocardiogram findings, and to identify predictive factors for the subset of TIA patients most likely to benefit from this testing.

Methods

Prospectively collected observational cohort data from 4 centers were pooled for this study (Table 1). These centers all enrolled patients with acute TIA at presentation. TIA was defined by the neurologist evaluating the patient using the traditional "time-based" definition (acute focal symptoms lasting <24 hours presumed due to a cerebrovascular cause). Patients underwent evaluation and treatment consistent with routine local clinical practice. A standardized case report form was completed collecting data on the clinical features of TIA, medical history, examination findings, results of diagnostic testing, and treatment at baseline and hospital discharge. Echocardiography was performed by experienced operators and interpreted by a local cardiologist at each institution according to standard clinical protocols. Evidence of acute infarction on MRI (magnetic resonance imaging) diffusion-weighted imaging, when performed, was determined by the radiologist and/or vascular neurologist at each site. ABCD^2 score was dichotomized into low (0-3) or moderate-to-high (4-7) risk categories. Interval clinical events and therapeutic interventions were determined at hospital discharge and 90-day follow-up. The 90-day follow-up visit was performed by a vascular neurologist in a clinic or, if the patient was unable to attend, by telephone interview. Informed consent was obtained from patients and each institutional protocol was approved by the local institutional review board at each site. A subset of patients included in this study has been described previously.[3-8]

Table 1. *Cohort characteristics*

Center location	Number of patients	Dates enrolled
US—PA	167	2002-2007
US—CA	234	2010-2013
Canada	232	2008-2010
Ireland	236	2005-2006

Abbreviations: CA, California; PA, Pennsylvania; US, United States.

Echocardiography reports were reviewed by the enrolling investigator at each site and scored for predefined potential CSE based on the Causative Classification of Stroke system.[9] Patients were specifically scored for presence of left atrial or ventricular thrombus, ejection fraction less than 35%, global hypokinesis, apical akinesis, infective or nonbacterial thrombotic endocarditis, papillary fibroelastoma, left atrial myxoma, isolated left atrial spontaneous echo contrast, complex aortic arch atheroma (defined as plaque with ≥4 mm protrusion, or involving a mobile or ulcerated component),[10] or any other cardioembolic source noted on echocardiography and deemed clinically significant by the reviewing investigator. Given uncertainty about the significance of patent foramen ovale (PFO) in unselected patients with TIA, PFO (with or without atrial septal aneurysm) was only scored as a potential CSE if its identification resulted in the initiation of anticoagulation or PFO closure. Transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE) results were scored separately.

Groups of patients were compared with *t*-test, chi-square test, or Fisher's exact test as appropriate. Factors associated with CSE were examined using multivariable logistic regression including variables significant at $P < .10$ in univariate analysis. All tests were two sided. An association was considered significant at $P < .05$. Patients with known CSE at time of admission or CSE identified by other methodology (e.g., telemetry demonstrating atrial fibrillation) were excluded from analysis of yield of echocardiography, but patients with multiple identified stroke mechanisms of which one was a potential CSE were included. Statistical analyses were performed using JMP (Version 9, SAS Institute Inc, Cary, NC).