Introduction: Echocardiography is often the sole imaging technique used for evaluation of cardiac sources of embolism in patients with recent systemic embolic event (e.g., stroke). However, up to 40% of ischemic strokes have no identifiable cause [1], and importantly, up to 30% of these patients may have recurrent strokes [2]. Contrast-enhanced MRI has shown improved diagnostic accuracy compared to echocardiography for the identification of LV thrombi [3]. In addition, evaluation for other sources of embolism, including full evaluation of the thoracic aorta, can be performed with MRA. We evaluated the potential additive value of the MRI and MRA techniques for assessment of cardioembolic sources in a cohort of patients with recent systemic embolism and cardiac dysfunction.

Methods: We prospectively enrolled 66 patients (49 men, mean age 64 ± 14 years) with recent systemic embolic event and evidence of left ventricular (LV) dysfunction (EF < 40% or regional wall motion abnormalities) on transthoracic echocardiography (TTE) performed for evaluation of cardiac source of embolism; signed informed consent was obtained. All patients also underwent imaging using an ultrasound contrast agent (Definity, Lantheus Medical Imaging) and focused imaging of the LV apex with a high frequency transducer. Routine transesophageal echocardiography (TEE) was also performed in a subset of patients (n=23). All patients underwent MRI examinations within 1 week of the TTE study on a 1.5T system (Avanto, Siemens Healthcare) using a multichannel phased array body coil with ECG gating. Cine imaging using steady-state free precession (TR 2.6ms/TE 1.3ms/flip angle 68°), and late contrast-enhanced T1-weighted images using a magnetization-prepared spoiled GRE (TR 2-RR intervals/TE 1.3ms/flip angle 40°) with inversion time set to null normal myocardial signal intensity were performed in standard long and short axis views of the heart. In addition, interpolated fat-saturated 3D GRE imaging of the thoracic aorta was performed immediately after 0.15 mmol/kg Gd contrast agent administration. MRI and MRA were performed preferentially with breath holding, but in patients unable to suspend respiration, sequence parameters were adjusted with use of parallel imaging techniques to decrease acquisition time to less than 5 seconds (MRA) or multiple (2 – 3) acquisitions were averaged.

Echocardiography studies were read by an experienced cardiac radiologist for potential sources of cardiovascular embolism. MRI and MRA studies were read by an experienced cardiologist for potential sources of cardiovascular embolism.

Results:
Baseline characteristics of the patient cohort are shown in Table 1. Sixty-three patients (95%) had a cerebrovascular event, with the remaining 3 patients diagnosed with retinal artery occlusion, peripheral artery occlusion and splenic infarct. Combining all modalities, a potential cardiovascular source of embolism was identified in 31 patients (47% of cohort), 5 with more than one potential source. The most common etiology was significant atheromatous plaque in the arch and descending aorta (n=23), followed by LV thrombus (Table 2). Routine clinical TTE detected a potential source of embolism in 8 patients. The addition of contrast TTE identified 1 additional patient with a potential source of embolism (LV thrombus). The addition of TEE identified an additional 9 patients with a potential source of embolism. Finally, the addition of MRI/MRA identified an additional 13 patients with a potential source of embolism not identified by TTE or TEE. Underlying potential etiology for source of embolism in the additional 13 patients identified only by MRI/MRA included 9 with significant arch and descending aorta atheroma, 3 with LV thrombus and 1 with LA thrombus. Of these 13 additional patients, 5 underwent a clinical TEE that was non-diagnostic for cardiac source of embolism etiology. Of note, there were 8 patients in whom a potential diagnostic etiology was only made with TTE or TEE and not by MRI/MRA. These included 3 with LV thrombus, 2 with patent foramen ovale (PFO), 1 with mitral valve mass and 2 with significant aortic atheroma. Potential reasons for missed diagnosis on MRI/MRA may include small, mobile structures with limited visualization with current MR techniques, particularly in patients unable to comply with breath-holding (mitral valve mass and PFO) and interval changes in anatomy (thrombus, atheroma) between the echocardiographic and MRI/MRA studies related to ongoing treatment.

Conclusion:
This study evaluated the diagnostic utility of cardiovascular MR imaging in the assessment for cardiovascular sources of systemic embolism in patients with LV dysfunction. In this cohort of patients, echocardiography diagnosed 18 patients with a potential cardiovascular source of embolism, including 8 patients (12% of cohort) with a potential source that was undetected by MRI/MRA. However, MRI/MRA identified an additional 13 patients (20% of cohort) with a potential cardiovascular source of embolism that was undetected by echocardiography, including 5 patients who underwent clinical TEE in addition to TTE. MRI/MRA provides a valuable adjunctive diagnostic imaging method for evaluation of patients with a potential cardiovascular source of embolism, particularly in patients with a negative echocardiography study or who are unable to undergo clinical TEE. Future analyses will address interobserver variability and reader confidence for the diagnosis of cardiovascular sources of embolism determined by the different imaging techniques.

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