Original Contribution

Nongated Cardiac Computed Tomographic Angiograms for Detection of Embolic Sources in Acute Ischemic Stroke

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Background and Purpose—We assessed the feasibility of obtaining diagnostic quality images of the heart and thoracic aorta by extending the *z* axis coverage of a non–ECG-gated computed tomographic angiogram performed in the primary evaluation of acute stroke without increasing the contrast dose.

Methods—Twenty consecutive patients with acute ischemic stroke within the 4.5 hours of symptom onset were prospectively recruited. We increased the longitudinal coverage to the domes of the diaphragm to include the heart. Contrast administration (Omnipaque 350) remained unchanged (injected at 3–4 mL/s; total 60–80 mL, triggered by bolus tracking). Images of the heart and aorta, reconstructed at 5 mm slice thickness in 3 orthogonal planes, were read by a radiologist and cardiologist, findings conveyed to the treating neurologist, and correlated with the transthoracic or transesophageal echocardiogram performed within the next 24 hours.

Results—Of 20 patients studied, 3 (15%) had abnormal findings: a left ventricular thrombus, a Stanford type A aortic dissection, and a thrombus of the left atrial appendage. Both thrombi were confirmed by transesophageal echocardiography, and anticoagulation was started urgently the following day. None of the patients developed contrast-induced nephropathy on follow-up. The radiation dose was slightly increased from a mean of 4.26 mSV (range, 3.88–4.70 mSV) to 5.17 (range, 3.95 to 6.25 mSV).

Conclusions—Including the heart and ascending aorta in a routine non–ECG-gated computed tomographic angiogram enhances an existing imaging modality, with no increased incidence of contrast-induced nephropathy and minimal increase in radiation dose. This may help in the detection of high-risk cardiac and aortic sources of embolism in acute stroke patients. **(*Stroke*. 2017;48:00-00. DOI: 10.1161/STROKEAHA.117.016903.)**

Key Words: aorta ■ computed tomography angiography ■ embolism ■ heart ■ stroke

Early determination of the etiologic factors of ischemic stroke is essential for secondary prevention because the risk of recurrence is highly dependent on the underlying cause.[1] Major identified attributable sources of ischemic stroke are intracranial atheroma, extracranial atheroma (including aortic arch), nonatheromatous aortic conditions, cardioembolic sources, and microvascular disease, as well as dissections in the younger age group.[2,3] Cardiogenic emboli have been estimated to be the causative factor in 20% to 40% of all stroke cases.[4] Therefore, identification of a cardiac source of embolism in stroke is paramount and time-sensitive.[5] Moreover, in a small proportion of cases, there can be concomitant presence of abnormalities in the thoracic aorta that can strongly influence the management plan, for example, an aortic dissection or a labile aneurysm may be a relative contraindication for endovascular treatment.

Currently, in most institutions, there is no evaluation of the heart apart from an ECG in the immediate treatment period when a patient is admitted for acute ischemic stroke (AIS), and aortic evaluation with computed tomography angiogram (CTA) is limited to the top of the aortic arch. In selected patients, a 2-dimensional echocardiogram may be performed, usually only at a later date. ECG-gated computed tomographic (CT) scans or CT scans timed to improve temporal resolution and minimize imaging artifacts caused by motion of the heart may be used to diagnose morphological pathologies but at the

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expense of an increased contrast and radiation load.[6] Present day multidetector CT systems with superior temporal and spatial resolution capabilities are also rapid enough to render non–ECG-gated images with reduced cardiac motion artifacts that allow for better assessment of the cardiac structures. We use this advantage to screen the heart and aorta for relevant pathologies without causing unacceptable delays in the highly time-sensitive AIS management.

We hypothesize that an opportunistic nongated CTA of the heart can be done concurrently with the initial CTA of the brain vasculature during emergent AIS evaluation and treatment to identify high-risk cardiac sources of embolism, such as thrombi, cardiac tumors, and valvular vegetations. We postulate that the functionality of the prethrombolysis or thrombectomy CTA would be expanded using the same contrast load and with minimal increase in time. This will ultimately allow initiation of secondary prophylaxis and treatment judiciously with early risk stratification, thereby reducing the rate of recurrent stroke after the initial event.

Methods

We performed a single-center, prospective open pilot study. A time period of 6 months for evaluation of the multidetector CT protocol was chosen. The study commenced on June 1, 2014, and ended on November 31, 2014. Consecutive acute stroke patients were recruited from the Emergency Department of National University Hospital, Singapore. Inclusion criteria were adults ≥21 years, admitted with AIS and suitable for intravenous thrombolysis or endovascular thrombectomy for which a CTA was performed. Patients within 4.5 hours of onset of stroke without contraindication for intravenous tPA (tissue-type plasminogen activator) were selected for thrombolysis, whereas patients with a National Institutes of Health Stroke Scale score of >10 within 6 hours of stroke onset were deemed as potential thrombectomy patients pending a confirmed large vessel occlusion on the CTA. Exclusion criteria were any contraindications to CTA, such as allergy to contrast, known renal impairment—serum creatinine >176 μmol/L or estimated glomerular filtration rate <30 mL min^{-1} 1.73 m^{-2} and the inability to provide informed consent. Patients were followed up for 3 months from the point of recruitment.

Patient demographics and National Institutes of Health Stroke Scale score was recorded at admission. Risk factors such as a history of atrial fibrillation or newly diagnosed on admitting ECG, a history of diabetes mellitus or raised blood glucose (admission blood glucose≥130 mg/dL) or HbA1c >6.5% on admission, present smoking history, a history of hypertension, and a history of hyperlipidemia were elicited from each patient. The etiologic work-up of each AIS included a 1× multidetector CT examination of the heart in addition to the usual institutional protocol of aortic arch, extra-, and intracranial vessels CTA imaging. All recruited patients also underwent a transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE) of the heart within 24 hours (flowchart see Figure I in the online-only Data Supplement),

The study protocol was approved by the National Healthcare Group Domain Specific Review Board (DSRB 2013/00913). Individual informed consent for each patient was obtained.

Imaging Protocols

In our institutional protocol, using a 64-slice CT scanner (Philips Brilliance) in the emergency department, all AIS patients who are planned for thrombolysis or thrombectomy will undergo a CTA of the brain and carotid vessels and arch of the aorta. The study patients underwent the same protocol, which consisted of the following parameters: 40 detectors, individual detector width of 0.625 mm, tube voltage of 120 kV, tube current of 300 mA, pitch of 0.2, and half-rotation reconstruction. The patient was placed in the supine, head-first position, and 70 mL of contrast (Omnipaque 300) followed by a chaser of 60 mL saline solution was injected at 4 mL/s for 60 to 80 mL into the right cubital vein with an 18-gauge intravenous catheter. The contrast load was identical to the standard dose for acute stroke CTA imaging.

A bolus-tracking method was used with an attenuation threshold of 150 HU in the ascending aorta above the baseline attenuation. The *z* axis, or cranial-to-caudal direction, coverage in our preexisting CTA protocol in patients with AIS is from the aortic arch to the vertex. For the study, this field was extended via a non–ECG-gated or non–ECG-synchronized acquisition from the domes of the diaphragm (including the heart) to the intracranial arteries (≈50 cm). This was performed with the following parameters: feet-to-head direction, section thickness of 0.8 mm, pitch of 1.0, tube voltage of 120 kV, amperage of 300 mA per section, and reconstruction filter B. The duration of the scan was not increased by >5 s (mean, 2 s; range, 2–4 s). The radiation dose was slightly increased from a mean of 4.26 mSV (range, 3.88–4.70 mSV) to 5.17 (range, 3.95–6.25 mSV) measured by a phantom device, which was unlikely to be clinically significant.[7,8]

Before the actual study was initiated, we experimented with different section thickness, including 1 mm thickness, 3 mm thickness, minimal intensity projections, and maximum intensity projections. Subsequently, we determined that scans of 1 mm thickness were superior for structural determination, although minimal intensity projections were preferable for thrombi detection (Figure 1). Eventually, we determined that all scans were to be 0.5-mm-thick slices to obtain the best resolution. The cardiac CTA images were subsequently read by the cardiologist and radiologist independently to identify any potential high-risk embolic sources such as intracardiac thrombi, atrial appendage thrombus, cardiac tumors, or valvular vegetations. We also screened for aneurysms of the interatrial septum which was predefined as a bulge of the septum secundum that was >10 mm in 1 of the 2 atria.[9]

All patients underwent echocardiography within 24 hours of the CTA performed with a Philips IE33 (Philips Healthcare) scanner, which included 2.5- and 3.5-MHz transthoracic probes. For TTE, apical 4-, 2-, and 3-chamber views and short-axis basal, midventricular, and apical views were obtained. If the treating physician deemed it to be necessary, the patient would undergo a TEE with a 5-MHz transesophageal probe. For TEE, first cardiac structures were examined at 0°, 45°, and 120° at the midesophageal and transgastric views. Then, the transducer was gradually withdrawn so that short-axis views of the descending aorta and aortic arch could be obtained. Aortic arch atheroma was evaluated as per previously established criteria.[10]

Results

Sixty-two consecutive patients admitted to our stroke unit were eligible for the study, of whom 20 provided informed consent and were prospectively recruited. The recruited patients were representative of the population studied (Table I in the online-only Data Supplement). All 20 patients underwent intravenous tPA, and 3 underwent mechanical thrombectomy.

The mean age was 63 years, and median National Institutes of Health Stroke Scale score at admission was 14 (range, 3–17). Four (20%) patients had atrial fibrillation at presentation, 7 (35%) had diabetes mellitus, 14 (70%) had hypertension, and 6 (30%) had hyperlipidemia.

Of the 20 patients, 1 had an internal carotid artery occlusion with middle cerebral artery territory infarct, 16 had M1 occlusions with middle cerebral artery infarcts, and 3 had no occlusion seen. The demographic data and risk factors for these patients are summarized in Table 1. All patients had sufficient level of consciousness and mental capacity to provide informed consent. On follow-up CT at 24 hours, 3 (15%) patients had intracranial bleeding, of whom 1 had

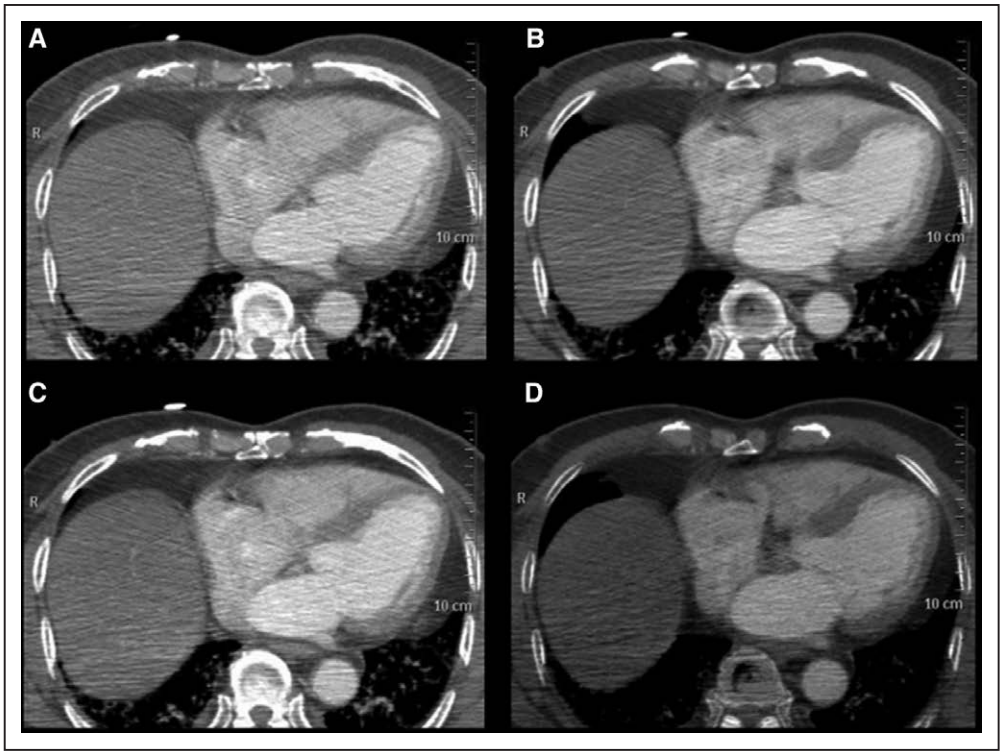

Figure 1. Various computed tomography angiogram section thickness at the level of the heart to illustrate the effects on resolution: (**A**) 1 mm thick, (**B**) 3 mm thick, (**C**) minimum intensity projection, and (**D**) maximum intensity projections.

symptomatic intracranial hemorrhage. Eight (40%) patients had excellent outcomes with modified Rankin Scale score of 0 to 1 at 3 months. None of the patients died or developed contrast nephropathy at the 3-month follow-up.

In the evaluation of the major sources of cardioembolic stroke, we identified 1 case of ventricular thrombus, which was localized at the apex of the left ventricle and confirmed on TTE (Figures 2 and 3). One patient had a localized dissection in the ascending aorta, and another had a thrombus at the atrial appendage (Figure 4). Intravenous tPA was withheld for the patient with a dissection. Both thrombi were confirmed by transesophageal echocardiography, and anticoagulation was started urgently the next day. None of these patients developed further strokes or intracranial bleeding.

Despite actively looking for minor embolic sources such as septal abnormalities (patent foramen ovale, atrial septal defect, and atrial septal aneurysm), as specified in the study protocol, none were identified. Echocardiography did not identify any further sources of cardioembolic stroke (major or minor) not diagnosed from CTA imaging.

Table 1. Baseline Characteristics (n=20)

Variables	Number
Mean age (SD)	64 (±12)
Male sex (%)	13 (65.0)
Hypertension (%)	14 (70.0)
Diabetes mellitus (%)	7 (35.0)
Dyslipidemia (%)	6 (30.0)
Smoker (%)	3 (15.0)
Atrial fibrillation (%)	4 (20.0)
Prethrombolysis systolic blood pressure (range)	151 (110–236)
Prethrombolysis NIHSS (range)	14 (3–17)
Median onset-to-treatment time (range)	160 (74–350)
Modified Rankin Scale score of 0–1 at 3 mo (%)	8 (40.0)
Median ASPECT score (range)	8 (2–10)

ASPECTS indicates Alberta Stroke Program Early CT Score; and NIHSS, National Institutes of Health Stroke Scale.

Discussion

Our study shows that nongated cardiac CTA can be readily incorporated into the acute stroke scanning protocol to effectively provide hyperacute screening for cardioembolic sources with minimal increase in time taken or radiation delivered and no increase in contrast dose. Discovering abnormalities through this expanded field of imaging may enable physicians to initiate treatment more promptly and better prognosticate the risk of recurrent stroke.

Although TTE is widely available, it is operator dependent and is often technically challenging with poor acoustic windows. It is not specific for detection of cardioembolic sources and can at times fail to detect a cardiac thrombus. TEE is considered the gold standard for the detection of potential cardiac sources of cerebral embolism but is an invasive procedure, requiring specially trained personnel and is usually performed under sedation, which may not be ideal for all AIS patients.[11–13]

To our knowledge, this is the first such study using nongated CT scans during acute stroke to examine for cardioembolic sources. High-risk sources of emboli such as a cardiac thrombus,

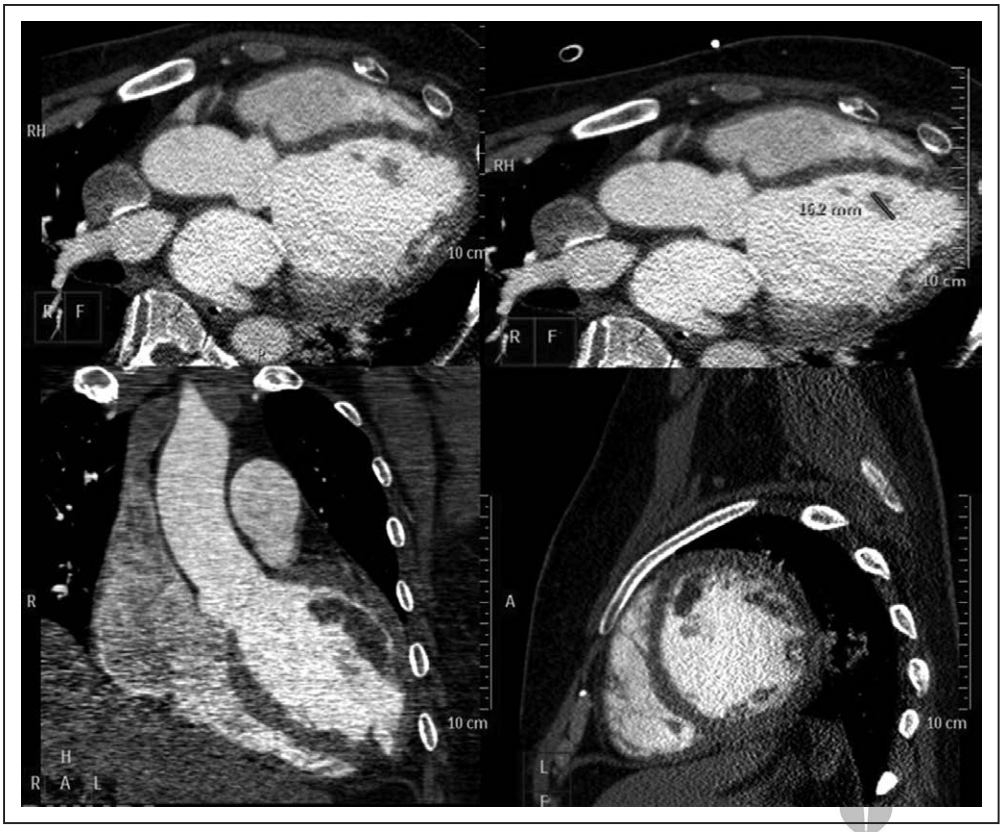

Figure 2. Computed tomography angiogram showing the left ventricular thrombus in various views.

cardiac tumor, or valvular vegetation can be identified by CTA using our protocol. We may also be able to identify paradoxical venous thromboembolism such as right-sided cardiac thrombi or vena cava clots with a right to left shunt. However, medium-risk sources such as patent foramen ovale, atrial septal defects, or atrial septal aneurysms may not be as easily diagnosed because of the static medium of imaging in CTAs. Although TEE especially excels in detection of abnormalities with medium embolic risk, management strategies and optimal therapies for this subgroup of patients remain unclear. In contrast, high-risk sources of emboli once identified generally require treatment through anticoagulation. TEE therefore does not confer additional therapeutic gains in terms of early clinical decision making.[14–17]

ECG-gated contrast material–enhanced multidetector CT has also been shown to be effective for studying left ventricular wall motion, ejection fraction,[18,19] intracardiac thrombus,[20] and patent foramen ovale.[21] However, it requires the use of β-blockers and involves 2 distinct phases of scanning with 2 doses of contrast, thus increasing radiation dose and take up significantly more time, rendering it unsuitable for AIS management.

The current multidetector CT systems have improved scanning capabilities that minimize cardiac motion artifacts with greater temporal and spatial resolution. This allows better evaluation of the cardiac structures (Table 2). Furthermore, multidetector CT has been demonstrated to be effective and reproducible in the detection and quantification of aortic arch atheroma.[26,27] The major advantage of our protocol for acute stroke situations is the minimal increase in the duration of scanning by not >5 s. Although the radiation dose is slightly

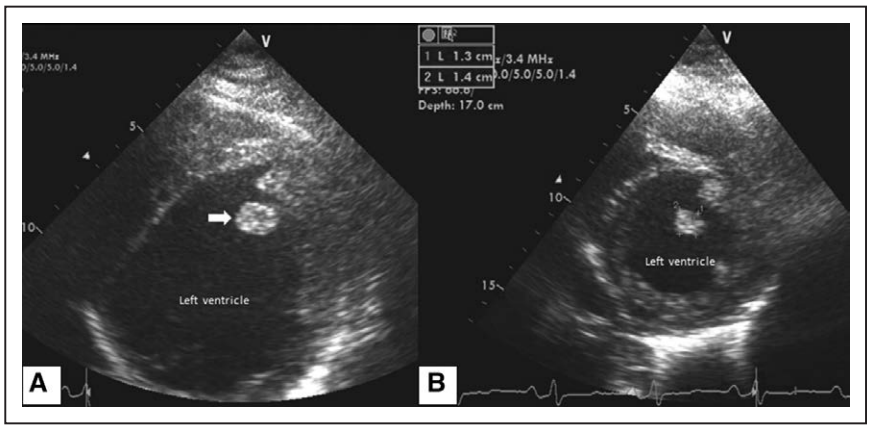

Figure 3. A, Mobile thrombus seen in the apical views. **B**, Same thrombus in the short-axis views

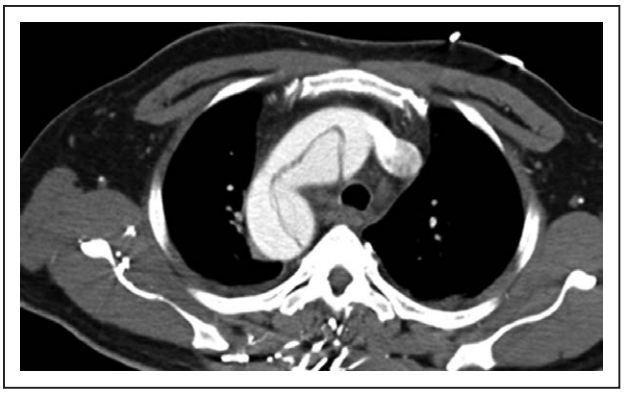

Figure 4. Computed tomography angiogram showing dissection of the ascending aorta.

increased by the equivalent of one fifth that of a normal CT brain scan, the total dose of radiation from the proposed protocol of the usual CTA of the brain, carotids, aortic arch, and the new nongated cardiac scan is together still less than a single ECG-gated cardiac CTA scan. Most importantly, there is no increased dose of contrast used, thereby eliminating any increased risk of contrast-induced nephropathy compared with the current standard CTA. We have demonstrated that at the resolution achieved by our imaging protocol, we will be able to identify high-risk embolic sources, which translates into early initiation of anticoagulation or other appropriate treatment. Although not demonstrated in our patients, contrast-enhanced CT imaging permits evaluation of wall attenuation, myocardial thinning, and focal dilatation.[22,24] This can potentially help identify left ventricular pseudoaneurysms, which may rupture with intravenous tPA and have catastrophic consequences. Ultimately, our imaging protocol improves the way AIS is evaluated and hedges AIS multimodality imaging toward CTAs.

Certain limitations should be acknowledged. The gold standard for comparison is TEE. However, not all patients received TEE, which could have resulted in some cardiac sources of embolism being overlooked, and the incidence could have been underestimated. Second, although we performed the TTE/TEE within 24 hours, there was still some delay between the CTA and the echocardiography, during which embolic sources could have changed. A further limitation was the absence of direct measurement of received patient radiation dose. In patients who were uncooperative, the CTA images may have been suboptimal. Finally, we have not used this technique in tachycardic patients and more study is required to determine whether the images are suboptimal at certain baseline heart rates.

Our study is a pilot with a small cohort to determine its feasibility. A larger prospective study of cardiac CTA for all acute stroke patients with cardiac ventricular thrombi should be performed. A follow-up echocardiography or embolic imaging with Transcranial doppler to determine the effectiveness of early anticoagulation can also be done.

Conclusions

This study confirms the feasibility and value of a composite CTA protocol as described above that allows evaluation of the heart for potential cardiac sources of embolism and ascending aorta together with the mandatory imaging of the caroticovertebral circulation. Such a protocol allows maximal returns of a scan that is already part of most acute stroke protocols with minimal additional risk and resource cost to the patient.

Table 2. Lesions Visible on Nongated Cardiac Computed Tomographic Angiography

Lesions	Findings and Associations
Thrombus	Nonenhancing filling defect typically found in left atrial appendage or left ventricular apex.[20] Right-sided cardiac thrombi or vena cava thrombi can be a source of stroke with a right to left shunt.
Intrachamber tumors	Metastatic intrachamber deposits present as enhancing filling defects with possible invasion of adjacent structures.[22,23] Cardiac myxomas present as low attenuation filling defects usually in the left atrium.
Metastases to myocardium	Manifest as filling defects within the cardiac chambers, multiple masses, nodules, or diffuse infiltration. Metastases to the myocardium are more common than primary cardiac tumours. Cardiac or pericardial metastases can occur through direct invasion from adjacent structures, hematogenous or lymphatic spread.[23]
Left ventricular thinning	Usually defined as interventricular septum on axial image <10 mm.[22,24]
Left ventricular thickening	Usually defined as interventricular septum on axial image >25 mm.[22,24]
Coronary artery calcification	Calcified coronary arteries are associated with increased risk myocardial infarction and arrhythmia.[25]
Rheumatic heart disease	Calcification of the left atrial wall is associated with rheumatic heart disease. It most frequently involves the mitral valve.[19]
True ventricular aneurysm	True aneurysms have a continuous wall of thinned, scarred myocardium, which can be complicated by thrombus formation.[26,27] Most commonly located at the anterior wall of the left ventricle or cardiac apex, usually secondary to transmural myocardial infarction. Rarer causes include trauma, Chagas disease, sarcoidosis, and congenital causes.
False ventricular aneurysm	Arise 5–10 d postinfarction after myocardial rupture, usually involving the inferior wall of the left ventricle. They can be identified by the narrow ostium, discontinuity of the myocardium at the neck of the aneurysm, and absence of coronary arteries overlying the aneurysm.[26,27]

Acknowledgments

The study protocol was approved by the National Healthcare Group Domain Specific Review Board (DSRB 2013/00913). Individual patient consent was obtained for the use of materials and for publication.

Disclosures

Dr Andersson declares the following: Neuravi, Speaker, Modest (<$10 k or 5%); Medtronic, Speaker, Modest (<$10k or 5%); Amnis,

Consultant, No Compensation; Ablynx, Consultant, Modest; and Rapid Medical, Consultant, No Compensation. None of the other authors declare any conflicts of interest or competing interests.

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SUPPLEMENTAL MATERIAL:

Supplementary table I. Comparison of included and excluded patients.

Variables	Included patients (n=20)	Excluded patients (n=42)	P value
Mean age (SD)	64 (+/-12)	68 (+/-14)	0.384
Male gender (%)	13 (65.0)	25 (59.5)	0.680
Hypertension (%)	14 (70.0)	28 (66.6)	0.985
Diabetes (%)	7 (35.0)	12 (28.6)	0.612
Dyslipidemia (%)	6 (30.0)	24 (57.1)	0.047
Smoker (%)	3 (15.0)	19 (23.8)	0.430
Atrial fibrillation (%)	4 (20.0)	14 (14.3)	0.571
Pre-thrombolysis systolic blood pressure (range)	151 (110-214)	154 (100-236)	0,836
Pre-thrombolysis NIHSS (range)	14 (3-17)	18 (4-33)	0.455
Median onset-to-treatment time (range)	140 (71-250)	160 (74-350)	0,118
Modified Rankin Scale 0-1 at 3 months (%)	8 (40.0)	18 (42.8)	0.836
Median ASPECT score (range)	8 (2-10)	6 (0-10)	0.334

Supplementary Fig I.