Multidetector Computed Tomography Angiography to Detect the Cause of Multiple Brain Infarctions

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Background: Multidetector computed tomography angiography (MDCTA) is useful to inspect cardiovascular pathologic changes with minimal invasiveness. Here we evaluated the usefulness of MDCTA to determine the cause of acute multiple brain infarction (AMBI). *Methods:* AMBI was defined as multiple recent infarcts demonstrated on diffusion-weighted imaging. A new infarction within 2 weeks from the last was also considered an AMBI. *Results:* Between January 2012 and December 2013, 967 patients were diagnosed with acute brain infarction and 138 (14.3%) with AMBI. Among them, 57 (39 men and 18 women; age, 38-93 years) were examined by MDCTA using the dual-phase method. All images were diagnostic, even if patients found it difficult to hold their breath. Fifteen patients (26.3%) were diagnosed with patent foramen ovale (PFO). Two had complications of atrial fibrillation (AF), necessitating anticoagulant therapy (ACT). Four had both PFO and severe aortic atherosclerotic plaque formation, necessitating single antiplatelet therapy (APT) and/or ACT. Fifteen patients (26.3%) developed complicated arterial plaques around the aortic arch and were administered single or dual APT and/or ACT, except 1 patient with a history of multiple cerebral bleeding. Nine patients had pre-existing AF. Furthermore, ACT was initiated for 2 other patients with thrombus or circulatory stasis in the left atrial appendage despite normal electrocardiographic findings. Two other patients were diagnosed with advanced cancer, which was considered Trousseau syndrome. The cause of AMBI was determined in 36 (63.2%) patients. *Conclusions:* MDCTA is a useful and less invasive method to identify the cause of embolic infarction. **Key Words:** Multidetector computed tomography angiography—acute multiple brain infarctions—patent foramen ovale—left atrial appendage thrombus—complicated aortic plaque.

Introduction

Acute multiple brain infarction (AMBI) is considered an embolic infarction; therefore, it is important to identify the embolic source to prevent recurrence. The origin of AMBI is considered to be caused by cardiogenic, atherothrombotic, or other factors. An AMBI of cardiogenic origin may be because of atrial fibrillation (AF), cardiomyopathy, endocarditis, cardiac myxoma, mitral valve prolapse, or patent foramen ovale (PFO), among other conditions, including Trousseau syndrome, pulmonary arteriovenous fistula, severe atherosclerotic plaque around the aortic arch, and angiitis. Because of the vast number of possible causes of AMBI, it is too difficult to investigate each in an efficient manner. Therefore, patients diagnosed with AMBI with no apparent medical history and normal waveforms and sinus rhythm on electrocardiography are often prescribed antiplatelet drugs to

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prevent recurrence without determining the true cause of the infarction.

In recent years, the use of multidetector computed tomography (MDCT) to evaluate cardiovascular and pathologic changes has rapidly increased because of its minimal invasiveness. Multidetector computed tomography angiography (MDCTA) is useful to evaluate the thickness and dissemination of aortic and carotid arterial plaques.[1,2] Moreover, it can visualize small recesses in ulcerative atheromatous plaque formations, which are known to cause brain infarction. Furthermore, previous studies have shown that MDCTA is useful to identify PFO or thrombus in the left auricular appendage (LAA).[3-5] Revel et al[3] performed both nongated MDCTA and transesophageal echocardiography (TEE) for 105 consecutive stroke patients on the same day. PFO was detected with 98% specificity and 55% sensitivity. High-grade shunts through PFO can be diagnosed with 91% sensitivity and 98% specificity. Kim et al[5] conducted a retrospective but detailed comparison of electrocardiographically gated MDCT and TEE for the diagnosis of PFO in 152 consecutive stroke patients. PFO was detected in 26 patients by TEE. A left-to-right contrast agent jet during MDCT was noted in 21 patients, 19 of whom were diagnosed with PFO by TEE. In that study, the sensitivity of MDCT was 73.1%, specificity was 98.4%, positive predictive value was 90.5%, and negative predictive value was 94.7%.

Here we used MDCTA to identify the cause of AMBI because MDCTA is less invasive than TEE and evaluated the impact on stroke etiology.

Materials and Methods

Diagnosis of AMBI

From January 2012 to December 2013, 967 patients were admitted to our hospital with acute brain infarction. All patients underwent conventional magnetic resonance imaging (MRI) on a 1.5-T or 3.0-T system (GE Healthcare, Milwaukee, WI) unless MRI was contraindicated. AMBI patients were prospectively and consecutively enrolled. AMBI was defined as multiple recent infarcts demonstrated on diffusion-weighted imaging (DWI). Scattered small, but noncontiguous, lesions in the same cortical arterial territory were included. Uninterrupted lesions visible in contiguous territories were considered as a single lesion and were consequently excluded. New infarctions developed on DWI within 2 weeks from the last infarction were also considered an AMBI. For all patients with AMBI, we performed MDCTA to determine the presence of cardiac or aortic vascular disease. If the cause of AMBI was determined to be cardiovascular disease, such as AF, we also performed MDCTA to identify possible thrombi in the LAA and other causes.

MDCTA

All MDCTA examinations were performed using a Light Speed VCT XT scanner (GE Healthcare) with electrocardiographic gating. We performed dual-phase, .625-mm-thick, helical CT scans with the patient in a breath-hold position. The first phase of MDCTA displayed an angiograph of the coronary and aortic arteries and the second phase showed the contrast medium equilibrium phase. The timing of the first phase of scanning was based on the test injection method. For vascular enhancement, iopamidol (Bystage 370; Teva Pharma Japan, Inc., Nagoya, Japan) was injected using a power injector (Medrad Stellant D; Medrad, Inc., Indianola, PA) at the rate of .07 mL/kg/s for 12 seconds, followed by 30 mL of saline chaser using the same flow rate.

The scanning field in the vertical direction included the entire heart, aortic arch, and origin of the bilateral common carotid and vertebral arteries. We began second-phase scanning 120 seconds after the contrast medium was injected. To efficiently detect PFO, we acquired the entire phase of cardiac movement. An intravenous β-blocker (Corebeta, landiolol hydrochloride; Ono Pharmaceutical Co., Ltd, Osaka, Japan) was administered to decrease the heart rate 1 hour before examination of patients with a heart rate of more than 70 beats/min. A retrospective, electrocardiographically gated, volumetric data set was acquired during a single breath-hold. We found that it is also effective to produce sine and 4-dimensional images. All reconstructed data sets were transferred to an off-line 3-dimensional workstation (AZE VirtualPlace; AZE, Ltd, Tokyo, Japan) for analysis.

The first phase of MDCTA was used to evaluate PFO. The foramen ovale is formed as the result of incomplete growth of the septum secundum, with incomplete overlapping to the ostium secundum. At birth or shortly thereafter, the septum primum and septum secundum typically fuse, and inadequate fusion may result in PFO.[6] In MDCTA, PFO is depicted as a tunnel-like structure between 2 thin membranes.[5]

The dual-phase method was used to evaluate the presence of a thrombus in the LAA.[7,8] If a filling defect in the LAA was detected in the first phase, we assessed the Hounsfield units of the defected filling area within the LAA and ascending aorta (AA) in the second phase. If the LAA/AA ratio was less than .5, we considered the defect to be a thrombus, and if it was more than .5, we considered the defect as circulatory stasis without a thrombus.

The first phase of MDCTA was used to evaluate the extent of aortic arch plaque formation. A plaque thickness of more than 4 mm or the presence of a ulceration was considered to be a complicated plaque and the potential embolic source.

Results

Between January 2012 and December 2013, 138 patients were diagnosed with AMBI. Among them, potential embolic sources were identified using MDCTA in 57 patients (39 men and 18 women; age, 38-93 years; median