Thrombi in the heart have been estimated to be the causative factor in 20%–40% of all stroke cases [1]. Therefore, identification of a cardiac source of thromboembolism in stroke patients is important for proper therapeutic management. The LAA is an important anatomic location of thrombus formation and subsequent cardioembolic events, especially related to arrhythmias, such as atrial fibrillation [2].

The diagnostic performance of cardiac computed tomographic (CT) for detection of LAA thrombi in stroke patients is promising [2]. We present a case, who suffered from severe complications of LAA thrombi, and was demonstrated clearly by CT scans.

CASE REPORT

A 51-year-old male suffered from progressive dyspnea for two days, and called at our ER department for help. To review his past history, he was a victim of type 2 DM, together with hypertensive cardiovascular disease (HCVD) for many years. He ever got three episodes of strokes in the past four years, and received regular medicine control (including anticoagulation therapy). About one year ago before this admission, he got acute PAOD and aortic dissection (Stanford type B), and then received a surgical thrombectomy successively on his right lower limb at that admission at our hospital, and regular imaging follow-up.

At this admission, his chief complaint was progressive dyspnea in the recent two days. At ER, abnormal cardiac enzymes (CK= 576, CK-MB= 61, Troponin I= 0.65) and poor renal and liver functions were obtained (BUN/Cr. = 114/ 4.6, GOT = 1121) at the same time. To rule out his underlying problem of unknown chest pain, he received MDCT scan (triple-rule-out) [3], which demonstrated near total occlusion of thoracic aorta combined with thrombi formation in the left atrium and ventricle (Fig. 1). No significant stenosis of bilateral coronary arteries or pulmonary embolism is demonstrated. Comparison with his prior CT scans (Fig. 2-4) were made, progressive thrombi formation in the descending aorta, and even newly-developed thrombi in the LAA are identified at his latest CT scan, and very
Figure 1. 64-MDCT of heart and whole body scans with contrast-medium enhancement. 

**a.** On ECG-gated cardiac CT scan, multifocal thrombi (red arrows) in the left atrial appendage, thoracic aorta, and left ventricle are identified on axial plane, reformatted vertical long axis and three-chamber views. 

**b.** On whole body contrast-enhanced scan of sagittal view, thrombus formations (red arrows) in the descending aorta reveal long and continuous appearance. Near total occlusion of ascending aorta is demonstrated at the L2 level of lumbar spine.

Figure 2. 64-MDCT of bilateral lower extremities scans with contrast-medium enhancement. 

**a.** Volume rendering image reveals occlusion of the right common iliac artery, and proximal portion of right external iliac artery. 

**b.** Maximal intense projection (MIP) image demonstrated filling defects in the right common iliac artery, and irregular filling in the proximal portion of right external iliac artery, indicated of acute PAOD.
Severe complications of left atrial appendage thrombi

**Figure 3.** 64-MDCT of heart and whole body scans with contrast-medium enhancement.  
**a.** CT scan of Jun-2009: the intimal flap (black arrows) and partial thrombus formation in the false lumen were identified.  
**b.** CT scan of Apr-2010: progressive change of thrombi formation (red arrows) in the descending aorta.

**Figure 4.** Comparison of CT scans between Jun-2009 **a, b.** and Apr-2010 **c, d:** newly-developed thrombi in the left ventricle and left atrial appendage were identified.
high D-Dimer value (DDI=20236) was also shown. Near total occlusion of thoracic aorta was impressed. Under the circumstances, he was sent to our ICU waiting for emergent surgery, and subsequently, he underwent emergent exploratory laparotomy with thrombectomy of thoracic-abdominal aorta, fenestration of dissecting flap, and femoral-femoral bypass with Polytetrafluoroethylene (PTFE) graft.

Through his overall history (Table 1), it seemed thrombus formation attacks on him intermittently (from brain stroke, then PAOD, and then thrombosis in aorta). During his hospital days, malperfusion of visceral organs was serious, maybe due to stenosis of supplying arteries, and the condition got worse day after day. He also suffered from profound metabolic acidosis, hyperkalemia, acute renal failure, gastrointestinal bleeding, hypoglycemia, and hypothermia, which was related to his multiple organs malperfusion. Because of dropping life condition, he died of multiple organ failure three days after his surgery. Based on all of his clinical history, medical images, and reported literatures [2,4], we believe LAA thrombi could result in a vicious circle of thrombus formation in the body, so that multiple organ failure of this case might be due to subsequent thrombi formation after surgery.

DISCUSSION

LAA thrombus is common in patients with atrial fibrillation or with a recent embolic event [4]. As for our presented case, he also has history of atrial fibrillation and recurrent strokes. The patient was exposed to a high risk of intracardiac thrombus formation, especially in the LAA. Although aortic dissection may be not related to LAA thrombi, comparisons with the prior CT scans of the patient were made, partial thrombus formation in the false lumen of aorta was noted in the last-year CT scan, but progressive thrombosis in the descending aorta (including true and false lumens) combined with newly-developed thrombi in the left ventricle and atrium, which is critical to him, were identified in the latest CT scan. In addition, exploratory laparotomy with thrombectomy of abdominal aorta cleared the aortic thrombi, but malperfusion of multiple organs still occurred post surgery. LAA obliteration was not done during surgery, and LAA thrombi indicate a vicious circle of thrombus formation in next days [2, 4].

Transesophageal echocardiography (TEE) is a semi-invasive and diagnostic standard examination that is widely available and portable for detection of intracardiac thrombi [4]. Although conventional transthoracic echocardiography (TTE) shows LAA in the specific mode, imaging quality of TEE is much better than that of TTE, so that it is not routine to use TTE for LAA evaluation [5]. At present, cardiac CT is a noninvasive, fast and sensitive modality to detect thrombi in the LAA, which has the advantage of being able to show images of uniform slices of the heart [2]. Contrast-enhanced cardiac MRIs are not as good as cardiac CT scans, because they lack diagnostic accuracy for detection of thrombi in the LAA [6]. In addition, MRIs are not as fast available as CTs are. Cardiac CTs are noninvasive promising modalities, which play a crucial role in the detection of intracardiac thrombus formation and follow-up subsequent complications [2]. In addition, CT angiography (CTA) with late-phase scanning allows enough time for filling in the LAA. It reduces false positive findings from slow blood mixing using a contrast medium [7].

The clinical significance for LAA (not simply an atrial outgrowth) is that over 15% strokes originate from the heart, and from the LAA in particular [8]. In patients with atrial fibrillation, blood clots arise from the LAA in greater than 90% of cases [8]. They dislodge (forming emboli), and contribute to ischemic damage to the brain, kidneys, or other organs supplied by the systemic circulation [9]. Actually, LAA dysfunction with spontaneous echo contrast (SEC) showing on TEE [10] and local blood stasis resulting from post-RFA (Radiofrequency ablation) in the curing dysrhythmia, are also associated with a high incidence of thrombi formation and thromboembolic events [11].

It is important for a physician to identify LAA dysfunction, and then to treat it. LAA obliteration is an experimental treatment to prevent strokes in atrial fibrillation, no matter surgical or percutaneous intervention [12]. Long-term anticoagulatory medication is also another conservative treatment.

By this case, cardiac CT could be a potential and promising modality to detect and evaluate LAA abnormalities, and even its subsequent complications.

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<th>Table 1. Time table of every admission</th>
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Severe complications of left atrial appendage thrombi

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3. Halpern EJ. Triple-rule-out CT angiography for evaluation of acute chest pain and possible acute coronary syndrome. Radiology 2009; 332-345