

Acute myocardial infarction caused by a floating thrombus in the proximal ascending aorta

Woong Jeon, Seung-Jin Lee, Sang-Ho Park, Se-Whan Lee, Won-Yong Shin, and Dong-Kyu Jin

Division of Cardiology,
 Department of Internal Medicine,
 Soonchunhyang University Cheonan
 Hospital, Cheonan, Korea

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Correspondence to Seung Jin Lee, M.D.

Division of Cardiology,
 Department of Internal
 Medicine, Soonchunhyang
 University Cheonan Hospital, 31
 Suncheonhyang 6-gil,
 Dongnam-gu, Cheonan 31151,
 Korea
 Tel: +82-41-570-3896
 Fax: +82-41-574-5762
 E-mail: drlsj3@gmail.com

To the Editor,

Acute myocardial infarction (AMI) is generally a consequence of complicated coronary atherosclerosis with thrombotic occlusion of the arterial lumen. Nevertheless, in some cases reported in literature, AMI was caused instead by ostium obstruction of the left or right coronary artery by a free-floating thrombus in the ascending aorta [1].

Development of aortic thrombosis in the absence of atherosclerosis, dissection or aneurysm is rare. Herein, we report a case of a 45-year-old man who died 3 days after undergoing transfemoral cerebral angiography (TFCA) from AMI caused by a floating thrombus in the left sinus of Valsalva, which had obstructed the left coronary artery.

A 45-year-old man presented to the

Emergency Department complaining of a headache in the left occipital and left periorbital areas that had persisted for 7 days, and a gradual onset of memory disturbance a few hours prior to presentation. He was a nonsmoker with no medical history of hypertension, dyslipidemia, diabetes mellitus, ischemic heart disease, or stroke. Upon admission to the Neurology Department of our institution, he was hemodynamically stable with a blood pressure of 130/80 mmHg and a heart rate of 80 beats per minute. Upon physical examination, he had no focal motor or sensory deficits, but experienced naming and memory impairment. Results from chest radiography and electrocardiography were normal (Fig. 1A). Laboratory findings were non-specific

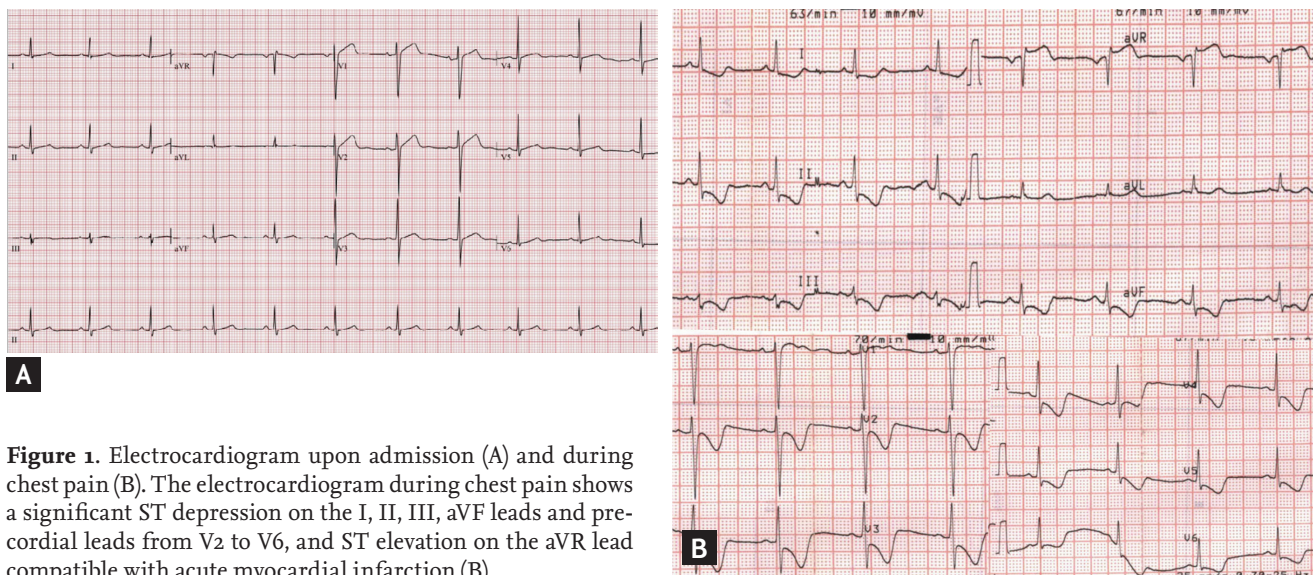


Figure 1. Electrocardiogram upon admission (A) and during chest pain (B). The electrocardiogram during chest pain shows a significant ST depression on the I, II, III, aVF leads and precordial leads from V2 to V6, and ST elevation on the aVR lead compatible with acute myocardial infarction (B).

because blood chemistry test results were almost within the normal limits, with the exception of a slightly increased lactate dehydrogenase of 260 IU/L. Coagulation markers, such as prothrombin and activated partial thrombin times, were also within normal limits. Cardiac enzymes, including creatine kinase-MB and troponin T, were within normal limits, and pro-BNP was 38.2 pg/mL. Brain magnetic resonance imaging revealed a 3.5 × 2.5 cm intracranial hemorrhage in the left anterior temporal lobe with surrounding edema (Fig. 2). The left anterior temporal location of the intracranial hemorrhage was unusual and required further evaluation to pinpoint the specific cause of bleeding, such as arteriovenous malformation. A neurologist at our institution performed TFCA, but did not find any abnormal cerebral vasculature. The procedure took 30 minutes and unfractionated heparin was not used during this time due to a risk of further bleeding from the cerebral hemorrhage. Conventional treatments aimed at decreasing the intracranial pressure, including intravenous mannitol (125 mL every 8 hours) and dexamethasone (5 mg every 24 hours), were administered. Following treatment, the patient's vital signs stabilized, and the only remaining symptom was a mild headache. Three days after TFCA, he underwent transthoracic echocardiography (TTE) at a routine check-up. TTE identified a highly mobile 2.5 × 1.5 cm mass in the proximal ascending thoracic aorta attached by a pedicle to the sinus of Valsalva, near the ostium of the left main coronary artery. The mass was swinging in the aortic flow, had a characteristic hard echogenic surface but an echolucent inner body, and appeared fragile (Fig. 3). The sinus of Valsalva and proximal ascending aorta were

neither dilated nor calcified, and no dissection or macroscopic atherosclerosis was observed. The aortic valve was tricuspid and normal, and the patient did not complain of chest pain. He was referred to our department, where transesophageal echocardiography (TEE) was planned for further investigation. Coronary angiography was not considered because the patient did not complain of chest pain and the risk of embolism was high. However, while preparing for the TEE, the patient complained of sudden chest pain. Electrocardiography was immediately performed, which revealed an ST depression on the I, II,

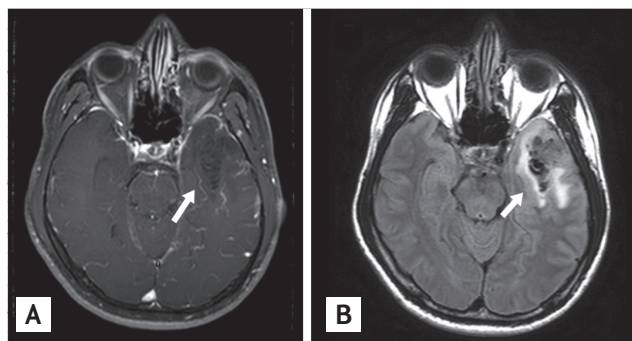


Figure 2. T1 weighted image (A) and T2 flair image (B) of brain magnetic resonance imaging showing an acute intracranial hemorrhage on the left anterior temporal lobe with surrounding edema (arrows).

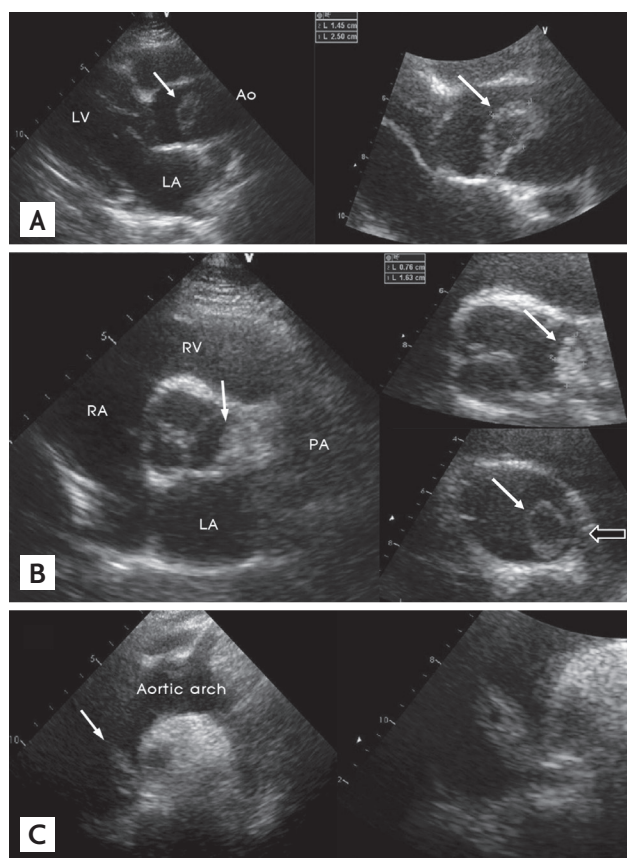


Figure 3. Transthoracic echocardiogram. (A) Parasternal long axis view (left) and magnified view of the aortic root (right) showing a pedunculated floating thrombus with an echogenic surface and echolucent inner body (arrows). (B) Parasternal short axis view of the aortic valve level (left) and magnified view of the aortic valve (upper right) and proximal ascending aorta (lower right) showing a floating thrombus (white arrows) near the ostium of the left coronary artery (black arrow). (C) Suprasternal notch view (left) and magnified view of the ascending aorta (right) showing the root of the thrombus (arrow) near the ostium of the left coronary artery. LV, left ventricle; LA, left atrium; Ao, aorta; RV, right ventricle; RA, right atrium; PA, pulmonary artery.

III, and aVF leads, precordial leads from V₂ to V₆, and ST elevation on the aVR lead (Fig. 1B). Considering the high risk of left main coronary artery obstruction, we decided to perform emergency surgery. However, while preparing for the operation, the patient experienced sudden cardiac arrest. Cardiopulmonary resuscitation was performed for 2 hours, but did not induce a response and, unfortunately, the patient died. The family denied a request for autopsy.

Virchow's thrombus formation triad includes abnormalities in blood flow, blood constituents and vessel walls [1]. In general, an aortic thrombus occurs when atherosclerotic plaques are eroded or disrupted. Nader et al. [2] attributed thrombi etiologies to local erosive lesions and age-related degenerative changes to the aortic wall. Thromboses of the aortic arch are infrequent sources of systemic emboli, but mobile thrombi in the aortic arch in young patients without diffuse atherosclerosis have been reported previously. The high prevalence of inserted thrombi in the wall opposite the ostia of the aortic arch may be related to the fact that areas of low wall shear stress are predisposed to the development of atherosclerotic plaques, which, even when minute, are likely sites for thrombi attachment. However, unless underlying diseases or causes such as chest trauma, atherosclerosis, hypercoagulable state, chemotherapy, or instrumentation are present, a thrombus (in particular, one involving the ascending aorta) is rare. Thrombi originating from the sinus of Valsalva, responsible for coronary flow disturbance, have been reported by several authors [1,3]. In a literature review by Knoess et al. [4], younger women (mean age, 45.5 years; range, 30 to 59) with no history of cardiac symptoms and no atherosclerotic changes were reported to be predisposed to free-floating thrombus formation, with no predilection for occlusion of either the right or left coronary artery.

A free-floating thrombus can cause acute coronary syndrome whether it interferes with coronary flow itself or its emboli occlude coronary flow. In the present case, while we could not perform coronary angiography or demonstrate a thrombus in the coronary artery, we hypothesized that the typical electrocardiographic changes observed during the presence of chest pain were sufficient evidence of a left coronary artery obstruction.

Because our patient was a young man with no risk factors of cardiovascular disease, the cause of thrombus

formation in the aortic root remains unclear. However, we assume that the TFCA performed 3 days prior to cardiac arrest was one cause of thrombus formation. Since heparin was not injected during the procedure, the risk of thrombus formation associated with the catheter or guide wire was high, and possible mechanical damage of the aortic wall by the catheter or guide wire may have potentiated thrombus formation. Another possible explanation is the presence of a hypercoagulable state. If TFCA was performed in a hypercoagulable state, the potency of thrombus formation would double. Because cardiac arrest occurred 2 hours after the patient was referred to our department, we were unable to perform an extensive serologic survey for hypercoagulable and vasculitis disorders (including antiphospholipid antibodies, antinuclear antibody, lupus anticoagulant, and protein C/S). However, a diagnosis of hypercoagulable disorder or systemic disease was less likely due to the absence of previously unexplained arterial or venous thromboses in the patient and members of his family.

Ostium obstruction of the left main trunk has been previously reported in the context of paradoxical embolism through a patent foramen ovale [5]. It is unlikely that our patient was in the same situation because we could not find any evidence of a foramen ovale in TTE, and the patient did not complain of dyspnea or suspected pulmonary embolism.

While we did not have time to perform TEE, its diagnostic value has been stressed by several authors [3]. Optimal treatment for ascending aortic thrombosis remains to be defined. Anticoagulant therapy appears to be a logical strategy, but persistence of the thrombus despite anticoagulant therapy and a highly mobile thrombus are indicative of operative therapy. If the thrombus location and shape are likely to occlude the coronary artery, surgery should be performed immediately.

To the best of our knowledge, this is the first report on AMI due to an ascending aortic thrombus related to TFCA. A free-floating thrombus is rare, but should be considered in the differential diagnosis of AMI, especially in younger patients without definite cardiovascular risk factors.

Keywords: Myocardial infarction; Thrombosis; Aorta

Conflict of interest

No potential conflict of interest relevant to this article was reported.

Acknowledgments

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