ST Elevation Myocardial Infarction Followed by Acute Ischemic Stroke in a Case of Occult Malignancy

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Abstract

Although acute myocardial infarction (AMI) is mostly caused by atheromatous plaque rupture in the coronary artery, coronary artery embolism is an important non-atherosclerotic cause of AMI. In one registry database, coronary artery embolism accounted for four to five percent of patients with ST-segment-elevation myocardial infarction (STEMI). The most frequent underlying disease was atrial fibrillation followed by malignancy. Here, we report a 50-year-old woman who presented with STEMI, caused by coronary artery embolism. Two weeks after this event, she had acute onset slurred speech and computed tomography angiography of the brain revealed recent and acute ischemic stroke at different territories of the cerebral artery. Further investigation revealed ovarian malignancy. This case reminds us that in patients with coronary artery embolism, comprehensive study of the etiology should be undertaken.

Keywords: acute myocardial infarction, ischemic stroke, malignancy, coronary artery embolism

Introduction

Coronary artery embolism is a relatively rare cause of acute ST-elevation myocardial infarction (STEMI). In one registry database, coronary artery embolism accounted for four to five percent (53/1232) of patients with STEMI.[1] The most frequent underlying disease was atrial fibrillation (28.3%) followed by malignancy (15.0%).[1] Coronary embolism was also associated with a higher risk of death than atheromatous plaque rupture.[1] We report on a fifty-year old woman with the initial diagnosis of STEMI, followed by ischemic stroke, whereby the ischemic stroke could have originated from ventricular thrombi developed after acute myocardial infarction (AMI).[2] We comprehensively studied the etiology of the young stroke and found hidden ovarian malignancy in the patient. This case highlights the importance of comprehensive etiology survey for coronary artery embolism related to myocardial infarction.

Case report

This fifty year-old woman had no specific

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systemic disease and her daily living was totally independent. She never smoked and drank alcohol only socially. She came to our emergency room because of persistent anterior chest pain radiating to her back for several hours.

In our emergency room, she had clear consciousness. The initial vital signs were heart rate 95 beats per minute, blood pressure 159/86 mmHg and body temperature 37.3°C. Neck examination revealed jugular venous level was 0.5 cm above the clavicle while sitting upright and rales were heard over the bilateral lower chest. Initial electrocardiogram (ECG) showed lead I, aVL and V2 to V6 ST-elevation with reciprocal change in the inferior leads, leads II, III, and aVF with ST-depression (Figure 1-A). Chest radiography (CXR) showed bilateral interstitial and alveolar edema (Figure 1-B). The initial laboratory tests showed elevated Troponin T (244.90 ng/L). Tentative diagnosis was acute extensive anterior MI with acute pulmonary edema. Loading doses of antiplatelet (aspirin 300 mg and ticagrelor 180 mg) and anticoagulant (heparin 3000 U) were given. In addition, immediate coronary angiography was performed.

The coronary angiogram showed total occlusion of the third diagonal branch of the left anterior descending artery (LAD) with TIMI 0 flow (Figure 2-A). Therefore, aspiration thrombectomy was done several times (Figure 2-B). In addition, distal intra-catheter thrombolytic agent, urokinase 120000 U, was given via thrombo-suction catheter. The final angiogram showed good blood flow with minimal amount of thrombus left (Figure 2-C).

Echocardiography was performed after primary percutaneous coronary intervention (PCI) and revealed left ventricular ejection fraction of 40% with regional wall motion abnormality. In addition, survey for coronary artery embolism was done. Anti-phospholipid antibodies (lupus anticoagulant, anti-cardiolipin antibody and Aβ2GP I antibody), antinuclear antibodies (ANA), protein C, protein S and homocysteine were within

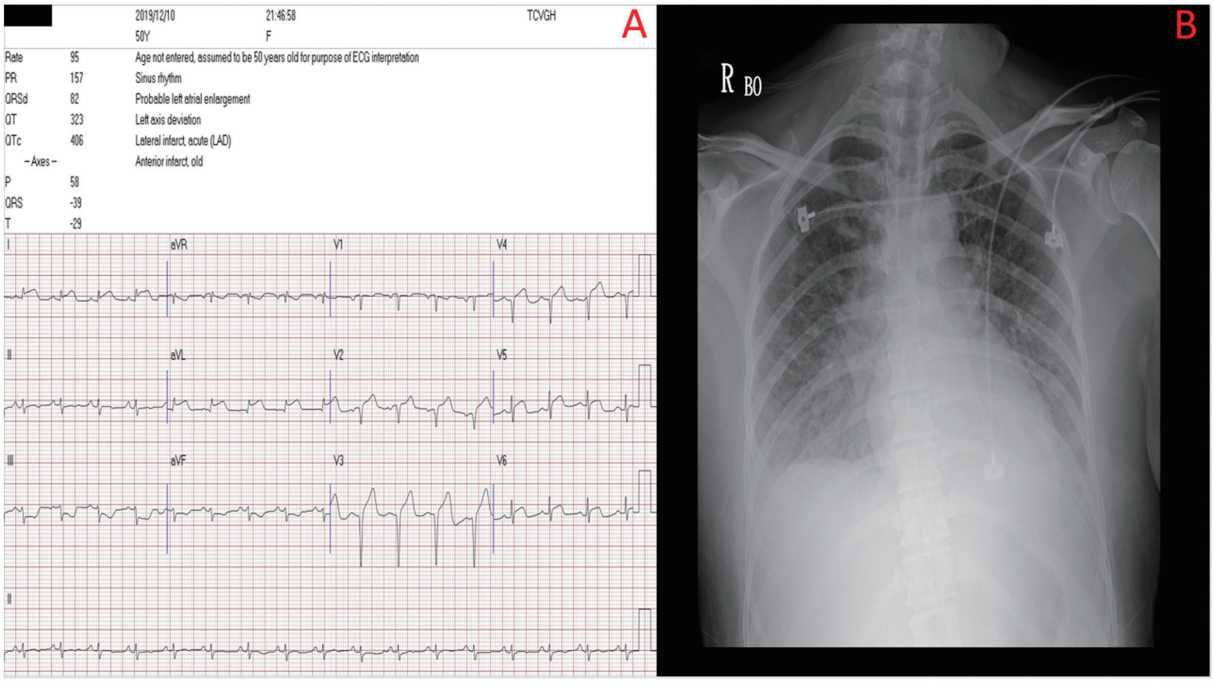

Figure 1-A. Electrocardiograms (ECG) and 1-B Chest X-ray (CXR)
ECG shows lead I, aVL and V2 to V6 ST elevation
CXR shows pulmonary edema

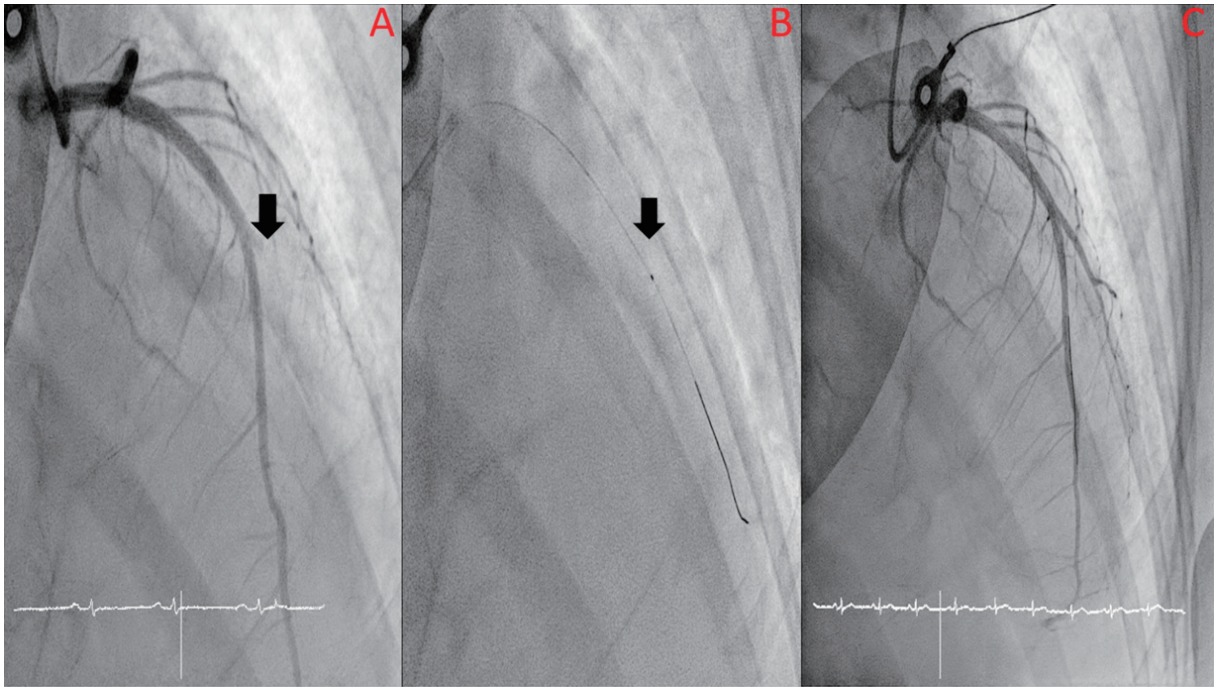

Figure 2. Coronary angiogram and the process of percutaneous coronary intervention
Figure 2-A shows diagonal branch total occlusion with TIMI 0 flow.
Figure 2-B shows aspiration thrombectomy, and the black arrow is the radio-opacity of the thrombosuction catheter.
Figure 2-C shows the final angiogram with good blood flow.

normal range. The patient was discharged with standard medical treatment for acute coronary syndrome, including aspirin, ticagrelor, valsartan, bisoprolol and atorvastatin.

However, she developed acute onset of slurred speech 10 days after discharge. Neurologic examination showed left upper extremities muscle power decrease, 4+ and impairment in coordination. Brain computed tomography angiography (CTA) revealed occlusion of the left middle cerebral artery (MCA) M2 branch, stenosis of the right MCA bifurcation and low density of change at right cerebellum. Recombinant tissue plasminogen activator, rt-PA, was not given because the NIHSS score was four. Brain magnetic resonance angiography (MRA) was done and the diagnosis of acute and recent stroke was confirmed (Figure 3). Serial ischemic stroke survey, including transcranial color-coded sonography (TCCS), carotid ultrasonography, transesophageal echocardiography (TEE), Holter's monitor ECG, and tumor marker screening were done. TCCS revealed normal flow velocities in the intracranial large arteries, and carotid ultrasonography showed normal bilateral common carotid, internal carotid, subclavian and vertebral arteries. TEE revealed no intramural and left atrium appendage thrombus. Holter's monitor ECG showed no paroxysmal atrial fibrillation. However, tumor marker screening showed elevated CA-199 and CA-125 serum levels as follows: CA-199: 115.8 Unit/ml (< 34.0 Unit/ml) and CA-125: 131.5 Unit/ml (< 35 Unit/ml). CEA, CA-153, AFP and SCC were within normal range. We consulted a gynecologist for malignancy survey. A gynecologic sonography revealed a 14 cm huge pelvic cyst, and malignancy could not be excluded. The patient was discharged after finishing the examinations. She had clear consciousness but still slurred speech. The prescription of antiplatelets was stopped in

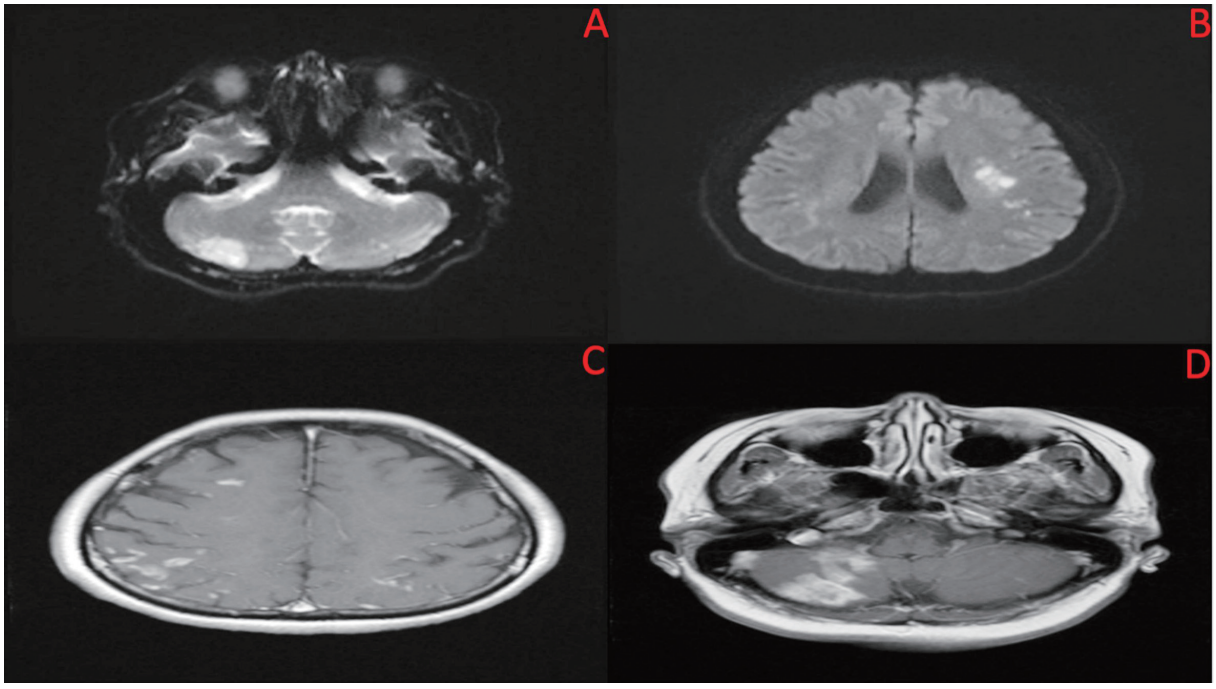

Figure 3. Brain MRA
Figure 3-A (Left upper picture) shows hyper-intensity at right cerebellum (DWI).
Figure 3-B (Right upper picture) shows hyper-intensity at left cerebral cortex (DWI).
Figure 3-C (Left lower picture) shows hyper-intensity at right cerebrum (T1 contrast).
Figure 3-D (Right lower picture) shows hyper-intensity at right cerebellum (T1 contrast).
These findings imply acute and recent ischemic events involving different territories.

preparation for pelvic tumor operation, and the anticoagulant enoxaparin was added for multiple embolic events.

However, the patient developed worsening neurological symptoms including speech disturbance ten days after being discharged. She had also had intermittent fever up to 38°C in the days before. Brain MRA showed an increased new area of infarction compared with the previous exam. Abdominal computed tomography showed cystic tumor arising from the right ovary with abdominal lymphadenopathy (Figure 4). At this stage, she had gone into hospital to prepare for the operation. However, she developed aspiration pneumonia complicated with hypoxic respiratory failure and another episode of blood stream infection with decompensated heart failure during hospitalization. She received endotracheal intubation twice but was weaned from the ventilator successfully. She received the operation for ovarian tumor while in a relatively stable condition. Frozen specimen of the right ovary revealed clear cell carcinoma. Therefore, the debulking operation, hysterectomy, bilateral salpingo-oophorectomy, bilateral pelvic lymph node dissection, para-aortic lymph node dissection, presacral lymph node dissection and omentectomy were performed. She recovered well after the operation and also received her first adjuvant chemotherapy, paclitaxel 96 mg. On this occasion she stayed in the hospital for fifty-seven days. We shifted anticoagulation from enoxaparin to warfarin before she was discharged. She received serial chemotherapy and targeted therapy afterward, and kept up the rehabilitation program. Although there were some neurological sequelae,

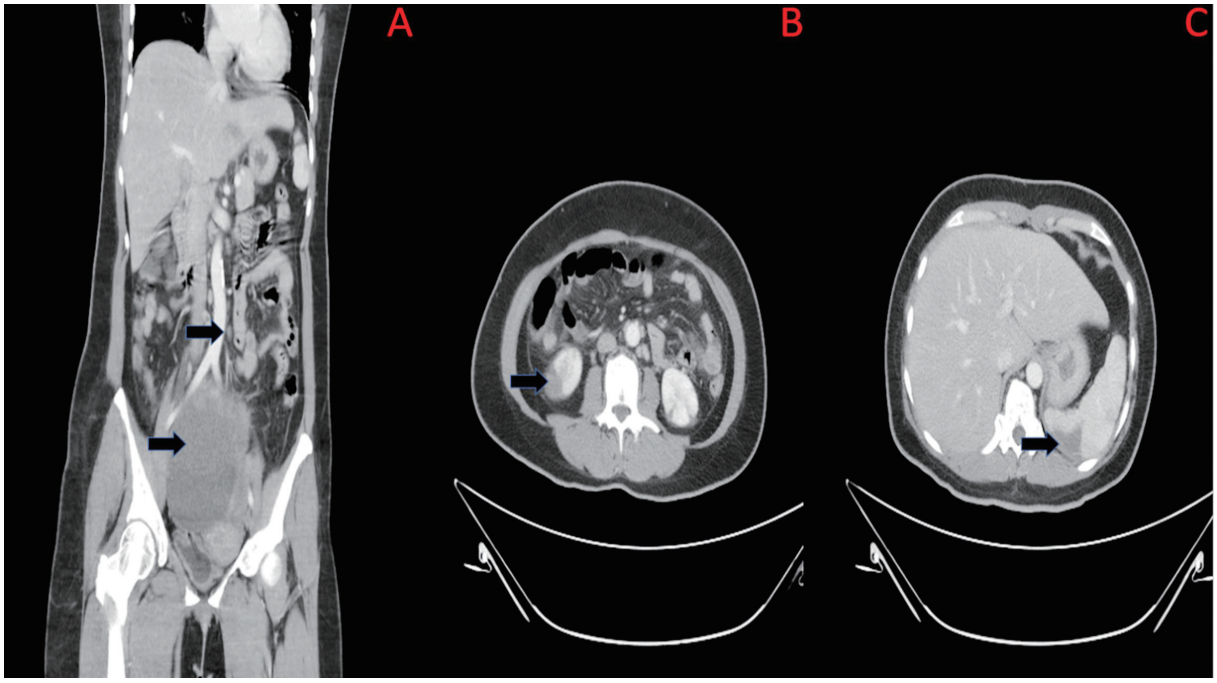

Figure 4. Abdominal CT with contrast
Figure 4-A shows cystic tumor arising from the right ovary. The upper black arrow shows para-aortic lymphadenopathy, and the lower black arrow shows tumor location.
Figure 4-B: The black arrow shows left renal infarction.
Figure 4-C: The black arrow shows spleen infarction.

she can now carry on most daily activities with minimal assistance.

Discussion

AMI is usually due to the rupture of atherosclerotic plaques. Nonetheless, mechanisms other than atherosclerosis, such as coronary spasm and embolism may result in myocardial infarction.[3] Coronary angiogram showing abrupt filling defect only at the occlusion site, without any other irregularity or stenosis is the typical feature of coronary artery embolism.[4] The diagnosis can be made by intravascular imaging, such as optical coherence tomography.[5,6] Once the diagnosis has been made, it is crucial to do comprehensive study to find the etiology of the coronary artery embolism, including atrial fibrillation, dilated cardiomyopathy, infective endocarditis, intracardiac tumor, malignancy, systemic autoimmune disease or antiphospholipid syndrome.[1]

Although one observational study did show patients to have increased incidence of cancer after myocardial infarction (MI),[7] there has been no evidence showing that cancer patients have higher risk of MI. The rate of STEMI was comparable in cancer patients and cancer-naive patients in one national registry.[8] On the other hand, one multicenter registry study demonstrated ovarian clear cell carcinoma (CCC) patients have higher risk of cerebral infarction compared to non-CCC patients.[9] The pathogenesis of cerebrovascular disease (CVD) is diverse because of the different types of malignancy. Vasogenic edema, cerebral vein thrombosis, intravascular lymphomatosis, non-bacterial thrombotic endocarditis and disseminated intravascular coagulation have been proposed.[10] Besides, cancer patients with CVD have significantly poorer prognosis than patients

without CVD.[10] To conclude, the pathogenesis of the multiple embolic events in this case is not fully understood and may best be attributed to a hypercoagulable state with in-situ thrombosis formation consistent with another, similar case analysis.[4]

There is no randomized controlled trial offering insights on how to manage cancer related multiple artery embolic events. However, according to a systemic review on management of cancer related stroke,[10] it is reasonable to use anticoagulation if there is no contraindication. Besides, treatment of the malignancy would surely be the fundamental factor in dealing with the problem.

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