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Clinical Communications

LEFT VENTRICULAR PSEUDOANEURYSM CAUSING MYOCARDIAL INFARCTION AND CEREBROVASCULAR ACCIDENT

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□ **Abstract—Although post-infarction mortality is most often due to ventricular dysrhythmias, the non-dysrhythmic causes of post-myocardial infarction death present a potential dilemma to the clinician. Non-dysrhythmic hemodynamic complications include cardiogenic shock, left ventricular free wall rupture, rupture of the interventricular septum, papillary muscle rupture, left ventricular pseudoaneurysm, and acute stroke. We present a rare case of a left ventricular pseudoaneurysm presenting with altered mental status, ultimately suspected to have caused the thromboembolic complications of acute myocardial infarction and cerebrovascular accident. © 2005 Elsevier Inc.**

□ **Keywords—ventricular pseudoaneurysm; aneurysm; myocardial infarction; complications**

INTRODUCTION

In the United States, nearly one-half million patients are hospitalized with acute myocardial infarction (AMI) annually. Post-infarction mortality rates range from 10% to 15%, most of which are secondary to acute ventricular dysrhythmias. However, the non-dysrhythmic hemodynamic complications of AMI—cardiogenic shock, left ventricular free wall rupture, rupture of the interventricular septum, papillary muscle rupture, left ventricular (LV) pseudoaneurysm, and acute stroke—are of significant concern (1). We present a rare case of a left ventricular pseudoaneurysm presenting with altered mental status, ultimately suspected to have caused the thromboembolic complications of AMI and cerebrovascular accident (CVA).

CASE REPORT

A 65-year-old woman was brought into the emergency department (ED) by ambulance with a change in mental status. The patient was in her usual state of health until she was found on the floor by her husband 9 h before her arrival in the ED. The patient was unable to recall the circumstances of the event. The patient's daughter reported similar symptoms of altered mental status 2 years prior when the patient suffered from a CVA. There were no complaints of chest pain, palpitations, shortness of breath, or difficulty breathing. She had not complained of any headaches. There were no recent illnesses, fever, chills, abdominal pain, nausea, vomiting or diarrhea. Bowel or bladder incontinence was not noted at the scene or in the ED.

The patient normally ambulated without assistance and had some mild residual right upper extremity weakness from the CVA in December 2001. The past medical history also included hypertension and hypercholesterolemia. The patient was taking acetylsalicyclic acid (ASA) 81 mg p.o. q.d., clopidogrel 75 mg p.o. q.d. and simvastatin 20 mg p.o. q.h.s. She had a 20 pack-year smoking history, and no surgical history.

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On examination, the patient appeared tired and was lethargic. The vital signs were: blood pressure 135/90 mm Hg, pulse 99 beats/min, respiratory rate 24 breaths/min, temperature 37°C p.o., with an oxygen saturation of 96% on 2 L/min of oxygen by nasal cannula. Pupils were equal, round and reactive to light. Extraocular movements and corneal reflexes were intact. Ear, nose and throat inspection was normal; the uvula was in the midline and the gag reflex was intact. Bronchial breathing was heard at the left lung base with bibasilar end-inspiratory crackles; no wheezing was heard. The heart sounds were normal, with no murmurs or extra heart sounds auscultated, but a left ventricular heave was appreciated. There was no cyanosis, clubbing or edema, and capillary refill was less than 2 s. The abdomen was soft and non-tender with normal bowel sounds. Rectal examination revealed normal tone and hemoccult negative stool.

Neurologically, the patient was awake and alert but slow to respond. Cranial nerves II–XII were intact. Right upper extremity (RUE) evaluation revealed biceps brachii strength of 2/5 (worsened from her baseline residual weakness), compared with 4/5 on the left. The remainder of the RUE examination revealed similar weakness. The lower extremity examination was limited due to poor compliance, but on reflex testing, toes were upgoing on the right and downgoing on the left. No clonus was appreciated.

Laboratory studies were as follows: Na 143 mmol/L, K 3.7 mmol/L, Cl 108 mmol/L, CO_2 22 mEq/L, BUN 20 mg/dL, Cr 1.4 mg/dL, Gluc 162 mg/dL, WBC 19.8×10^3, Hb 11.9 g/dL, Hct 36.1%, Plts 353×10^3, PT 13.3 s, INR 1.4 units, and PTT 29 s. Two bedside cardiac markers drawn 90 min apart were positive for cardiac ischemia: 1) CKMB $>$ 125 mg/mL (normal 0–10 mg/mL), myoglobin (myo) $>$ 500 mg/mL (normal 0–170 mg/mL), Troponin I 19.2 mg/mL (normal 0–1.0 mg/mL), and 2) CKMB $>$ 125 mg/mL, myo $>$ 500 mg/mL, TNI 28.4 mg/mL. Urinalysis had a specific gravity 1.05, pH 5.0, trace glucose and moderate blood—old 4 red blood cells were seen on microscopy. Serum CPK was 874 UI/L (normal 45–235).

The chest X-ray (CXR), Figure 1, revealed cardiomegaly with an abnormal contour of the left ventricle as well as blunting of the left costovertebral angle; the mediastinum was wide, measuring 9 cm. Electrocardiogram (EKG), Figure 2, demonstrated normal sinus rhythm (NSR) at 87 beats per minute with right bundle branch block (RBBB), bi-atrial enlargement, and T wave inversions in leads V4–V6. There were no previous EKGs for comparison.

Computed tomography (CT) of the head revealed a left cerebellar infarct. A CT scan of the chest with and without contrast revealed a large left ventricular pseudoaneurysm measuring 9.0 × 7.5 cm without evidence of leak or aortic dissection (Figure 3). Fifty percent of the aneurysmal cavity was filled with thrombus. As well, the thoracic CT scan revealed a small subsegmental right pulmonary artery embolism.

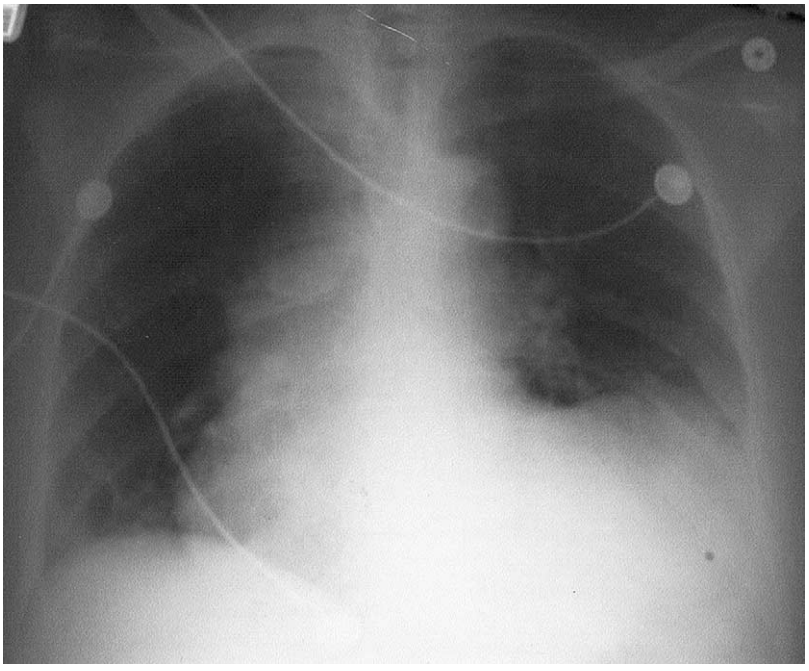

Figure 1. Chest X-ray: wide mediastinum, cardiomegaly, abnormal contour left ventricle and blunting of the left costophrenic angle.