



Clinical Reasoning: An 83-year-old woman with acute right-sided weakness and chest pressure

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SECTION 1

An 83-year-old right-handed woman presented to the emergency department with right arm and leg weakness. The patient had been feeling well until 1 hour before arrival when she noted that her right hand became clumsy while typing. When she tried to stand, she found that she could not walk because of right leg weakness. The patient denied other complaints until specifically asked about chest pain or pressure. Accompanying her weakness was a moderate-severity sensation of chest pressure without pain or shortness of breath. Because of her symptoms, she called a friend who took her to the emergency department.

The patient's medical history is notable for hypertension, coronary artery disease status post coronary artery bypass grafting surgery and bioprosthetic aortic valve replacement 6 years prior, and melanoma in remission. She takes aspirin 81 mg daily as well as antihypertensive medications. She has no history of stroke, metastatic brain disease, or seizures.

Questions for consideration:

1. Based on the patient's medical history and current presentation, what are the differential diagnoses?
2. How should this patient be triaged?

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SECTION 2

Patients with multiple, acute life-threatening complaints pose a significant challenge in diagnosis and triage. One complaint may overshadow an equally important issue. In this patient's case, her obvious hemiparesis was her chief complaint, but when asked specifically she disclosed the presence of chest pressure. Based on this patient's history, the differential diagnosis should include acute ischemic stroke, acute myocardial infarction (MI), aortic dissection, hemorrhagic stroke, and CNS metastasis of melanoma or other tumor.

Upon her arrival at the emergency department, the patient was triaged as an acute stroke and an emergent cardiology consult was placed. The initial neurologic evaluation demonstrated an alert and oriented patient with intact mental status, no evidence of aphasia, no dys-

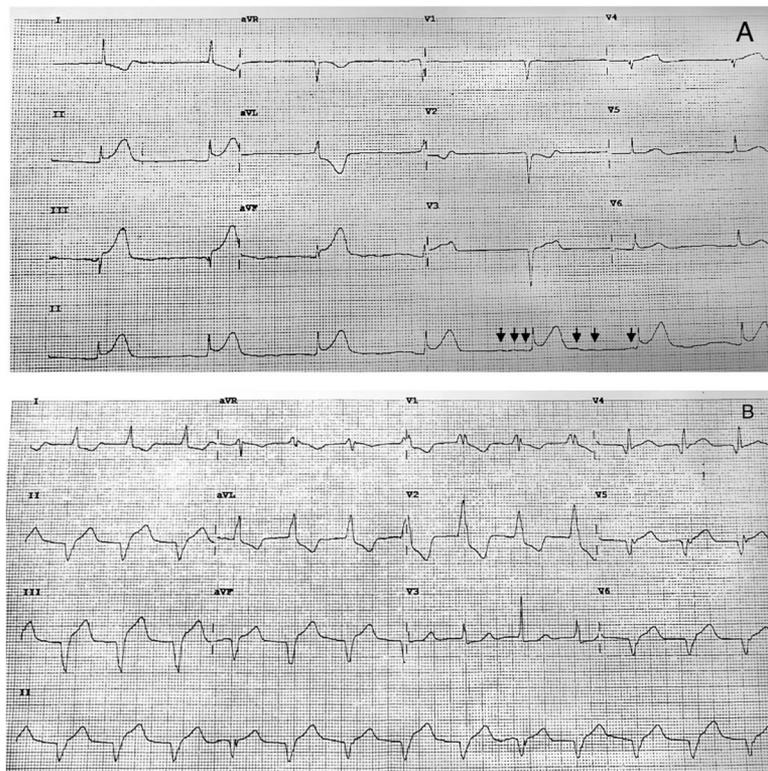
arthria or cranial nerve deficits, moderate right arm and leg weakness, as well as associated right arm partial sensory loss to pin prick. The patient's National Institutes of Health Stroke Scale (NIHSS) score based on her initial examination was 5. Her initial EKG revealed a heart rate of 42 beats per minute with a new junctional escape rhythm, underlying atrial fibrillation, and ST segment elevations in the inferior leads (figure, A). The initial troponin T and creatine kinase MB fraction levels were 0.01 ng/mL and 3.4 ng/mL, respectively.

Questions for consideration:

1. How frequently do acute stroke and acute MI occur together?
2. What is the appropriate next step in working up the patient?

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Figure Electrocardiograms



(A) Initial EKG. This EKG demonstrates a junctional rhythm with underlying atrial fibrillation (arrows indicate P waves). The prominent ST-segment elevations in the inferior leads are consistent with acute myocardial infarction. (B) Reperfusion rhythm. Both EKGs are standard 25 mm/s, 10 mm/mV recordings.

SECTION 3

Acute MI and acute ischemic stroke may occur simultaneously. Ischemic stroke is associated with elevated troponin T without electrographic signs of myocardial ischemia in approximately 6% to 10% of cases.^{1,2} ST-elevation MI may, in uncommon instances, lead to or occur concurrently with ischemic stroke in approximately 3% of cases.² Stroke, especially stroke involving the insula, may induce an increase in sympathetic tone, which is postulated to cause arrhythmia and myocardial cell damage.³ The patient in this case had inferior ST elevations, but normal cardiac enzymes suggesting very early MI.

The workup proceeded with a noncontrast CT scan of the brain demonstrating no hemorrhage or large territory infarction. CT angiography of the aortic arch to the circle of Willis demonstrated no dissection, aneurysms, or large artery occlusion. A high degree of aortic arch and bilateral carotid atherosclerosis with calcification was noted on CT angiography. Reevaluation of the patient 1 hour after presentation was notable for worsening right-sided weakness with near complete right-sided paralysis giving her an NIHSS score of 7.

The patient was diagnosed with simultaneous acute MI and ischemic stroke, likely both secondary to new-onset atrial fibrillation. The case was discussed among the cardiology, emergency medicine, and neurology services. Cardiac catheterization was deferred in favor of IV recombinant tissue plasminogen activator (rtPA) because it was thought that emergent percutaneous coronary intervention for an inferior location MI could safely be deferred. The patient received stroke-dosed IV rtPA within 2 hours of symptom onset. She subsequently had complete resolution of neurologic deficits within 3 hours of administration of rtPA. The patient's chest pressure resolved and her ECG evolved to a sinus reperfusion rhythm with right bundle branch block and hemi-fascicular block (figure, B). Within 1 hour of rtPA administration, her ECG reverted to normal sinus rhythm with complete resolution of signs of inferior infarction. The troponin T level peaked 8 hours later to 6.45 ng/mL.

Question for consideration:

1. Does acute MI exclude this patient from receiving IV rtPA?

DISCUSSION Thrombolytic agents have been used successfully to treat MI and ischemic stroke, although percutaneous interventions with angioplasty and coronary stenting are considered superior to thrombolysis in MI when available within a reasonable timeframe.⁴ However, the only Federal Drug Administration–approved therapeutic to treat acute ischemic stroke is IV rtPA. Current guidelines for IV rtPA administration exclude patients with MI in the 3

months before presentation, but do not address simultaneous MI.⁵ The great concern with administering thrombolytics to patients with acute or subacute MI is the risk of cardiac rupture, which has rarely been reported in patients with stroke.⁶ In patients treated with thrombolysis for acute MI, the rate of rupture is between 1% and 8% with the highest risk being ascribed to anterior wall infarctions, which are most likely to rupture.⁷ Of note, the IV rtPA treatment dose is higher and the time window for treatment longer (6 hours vs 3–4.5 hours) when IV rtPA is administered for acute MI rather than for stroke. The simultaneous occurrence of a disabling ischemic stroke and ST-elevation MI poses a therapeutic challenge in deciding whether to pursue percutaneous coronary intervention or opt for thrombolysis.

In this case, the decision to pursue IV rtPA was based on the knowledge that thrombolytics have been used in cardiac care for many decades at higher doses and with good efficacy to treat acute MI. IV rtPA seemed to be the superior intervention because it could potentially treat both the cardiac and the cerebral ischemia, which in this case was likely secondary to fibrin-rich emboli generated from new-onset atrial fibrillation. The location of the MI, which involved the inferior wall and spared the anterior wall, also placed the patient at lower risk for cardiac rupture. We chose to use the stroke-based dosing for rtPA rather than the higher acute MI dosing to reduce the risk of intracranial hemorrhage. This case demonstrates that in the proper clinical setting, IV rtPA can be successfully used to treat simultaneous acute MI and ischemic stroke.

AUTHOR CONTRIBUTIONS

G.G. Curiale: Drafting/revising the manuscript, acquisition of data. J.L. Schindler: Drafting/revising the manuscript, study concept or design, analysis or interpretation of data.

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DISCLOSURE

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