Clinical Reasoning: A 76-year-old man with acute-onset left-sided weakness and numbness

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SECTION 1

A 76-year-old right-handed man with no known medical problems presented 2 hours after falling at work due to the acute onset of left-sided weakness and numbness. He had experienced left knee pain the previous day, but denied vertigo, headache, neck pain, chest pain, abdominal pain, or back pain. On examination, his blood pressure was 230/118 mm Hg; pulse and respiratory rate were normal. He was anxious with normal mentation, no carotid bruits, normal cardiac

rate and rhythm, clear lung fields, normal cranial nerves including facial strength, weakness in his left leg (2/5 power) and arm (4/5 power), sensory loss to painful stimuli in his left face, arm, and leg, and normal deep tendon reflexes with plantar responses bilaterally.

Questions for consideration:

1. Where does this process localize?
2. What is the differential diagnosis?
3. What is the next step in management?

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SECTION 2

In the setting of normal cranial nerve function, the combination of left arm and leg weakness, leg weaker than arm, and left face, arm, and leg numbness typically localizes the lesion to the right anterior cerebral artery distribution, posterior limb of the internal capsule, or thalamus. The differential diagnosis of sudden-onset weakness and numbness includes vascular etiologies (e.g., stroke, vasospasm), seizure with postictal paralysis, hypertensive encephalopathy, migraine, and functional. CNS tumors and demyelination can also present with similar symptoms, but they typically are subacute in onset. Thus, a stroke code was activated to evaluate vascular etiologies and to determine whether tissue plasminogen activator (tPA) treatment was indicated. CT of the patient's head showed neither intracranial hemorrhage nor acute hypodensities. The only contraindication to tPA was the patient's elevated blood pressure, so antihypertensive medications were started prior to

tPA administration. Ten minutes after the tPA infusion began, the patient sat upright, grabbed his throat, gasped for air, and lost consciousness for approximately 10 seconds. In response to sternal rub, he regained alertness with no new neurologic deficits, but was noticeably more tachypneic (30 breaths per minute, normal oxygen saturation, pulse 80 beats per minute) than he had been prior to tPA administration. One minute later, the event repeated; but this time the patient was observed to have fixed downward gaze deviation during his transient loss of consciousness. Again, he became alert within 10 seconds without additional neurologic deficits, but he reported new lower abdominal pain.

Questions for consideration:

1. What is the differential diagnosis of acute mental status change and abdominal pain after receiving tPA?
2. What is the next step in management?

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(A) Aortogram reveals a Stanford type A aortic dissection (arrow), which extends from the aortic root to the origin of the great vessels. (B) Diffusion-weighted MRI of brain demonstrates restricted diffusion in the right lateral thalamus and posterior limb of the internal capsule (arrow).

SECTION 3

For neurologists, the major concern in administering tPA is intracranial hemorrhage, but acute gastrointestinal, retroperitoneal, and pericardial bleeding are also risks. Anaphylaxis, orolingual angioedema, or laryngeal edema can compromise airways in <1% of patients receiving tPA. In this patient, rapid airway and respiratory assessment did not reveal angioedema or laryngeal edema, and his stable blood pressure and normoxia made anaphylaxis, hemorrhage, or acute respiratory failure unlikely. The differential diagnosis then focused on understanding the patient's new-onset abdominal pain, respiratory distress, and syncope. A significant intra-abdominal or retroperitoneal hemorrhage could explain abdominal pain, but it would typically be associated with tachycardia and hypotension. Early brainstem hemorrhage, bilateral thalamic hemorrhage, or basilar artery occlusion can present with waxing and waning mental status and tachypnea, but none of these would explain the abdominal pain. Similarly, a seizure would not explain his abdominal pain and is unlikely in the absence of postictal confusion. A pulmonary embolism can cause acute loss of consciousness and tachypnea, but the time course of resolving and recurring syncope would be unusual. Aortic dissection and hemorrhagic pericardial effusion with or without tamponade typically present with chest pain, but patients may also experience abdominal pain, shortness of breath, and syncope.¹

After the patient's second syncopal event, the tPA infusion was promptly discontinued. Focused assessment with sonography was negative for intra-abdominal or retroperitoneal hemorrhage. Repeat head CT was unchanged, but an aortogram revealed a large, unruptured Stanford type A aortic dissection extending from the aortic root to the origin of the great vessels (figure, A).

Questions for consideration:

1. How can painless aortic dissections be detected during a stroke code?
2. What is the next step in management?

GO TO SECTION 4

SECTION 4

Aortic dissection occurs when blood penetrates through the inner intimal layer into the media, resulting in longitudinal extension along the artery. The dissection may involve the aortic arch and ascending aorta (Stanford type A or DeBakey I/II) or the descending aorta (Stanford type B or DeBakey III). One-third to one-half of these patients display neurologic symptoms secondary to cerebral or spinal infarction.^{2,3} A total of 80%–90% of all patients with acute aortic dissections present with severe tearing chest pain that radiates to the back¹; 40% of patients report abdominal pain. Painless aortic dissections are rare, however, occurring in only 6%–10% of patients.¹ Detection of painless aortic dissections during a stroke code is challenging because the codes are focused on ruling out intracerebral hemorrhage and expedited delivery of tPA. While CT angiography of the head and neck can often detect ascending dissections, most acute stroke protocols do not include angiography because of the low incidence of these dissections. This puts more emphasis on the initial physical examination, which can be tailored to screen for common features of aortic dissection: a diastolic heart murmur or asymmetry in upper extremity blood pressures or pulses is observed in 40%, 30%, and 20% of patients, respectively.¹ While none of these findings was detected in this patient, their presence should prompt additional consideration of aortic dissection.

Once the diagnosis was made with an aortogram, the patient's blood pressure was lowered to a goal systolic blood pressure of 110 mm Hg. Platelets, clotting factor status, and fibrinogen were obtained to evaluate for post-tPA coagulopathy, but replacement factors were not immediately required. Cardiothoracic surgery was consulted for emergency surgical repair, and a supracoronary tube graft hemi-arch repair of his type A aortic dissection was performed.

Subsequent brain MRI revealed restricted diffusion in the right lateral thalamus and posterior limb of the internal capsule (figure, B), right frontal subcortical white matter, left centrum semiovale, right parietal lobe, right occipital lobe, and bilateral cerebellar hemispheres. Spinal imaging showed a small focus of T2 hyperintensity in the patient's right ventrolateral gray matter between levels T2 and T5, consistent with a spinal cord infarct. Magnetic resonance angiography of his brain and neck was negative for dissection, flow-limiting stenosis, or aneurysm. While the patient had numerous CNS lesions, his initial presentation of left-sided numbness and weakness best localized to the right thalamocapsular infarct.⁴ These strokes were most likely caused by emboli generated from his thrombogenic dissected aorta. However, because neuroimaging was obtained following

aortic repair, some of the embolic strokes observed on MRI might have been secondary to this surgery.

The patient's syncope and tachypnea after receiving tPA was a dramatic and unexpected complication. However, these are not uncommon symptoms for type A aortic dissections,^{1,3} and with involvement of his aortic root, both of these symptoms could be explained by acute aortic valve insufficiency with subsequent reduction in cardiac output and cerebral hypoperfusion.

The patient did well after surgery and was discharged to a rehabilitation facility on postoperative day 10 with improving arm and leg strength.

DISCUSSION This case reminds us that painless aortic dissections are rare but important causes of acute cerebral or spinal cord infarction and can lead to a challenging therapeutic decision. Acute stroke is managed by rapid administration of thrombolytics, but tPA in the setting of an aortic dissection is associated with a mortality rate of 71%.⁵ Mortality is often secondary to extension of the aortic dissection and subsequent pericardial hemorrhage and tamponade. Administration of tPA also results in a hypocoagulable state that significantly complicates emergency cardiothoracic surgery. While there are no specific guidelines regarding tPA administration in patients with stroke secondary to an aortic dissection, the significant mortality risk outweighs any benefit to most patients. In cases of severe stroke (e.g., acute basilar artery occlusion), where the mortality risk of inaction approximates that of extending the aortic dissection, the decision has to be made on an individual basis. It is therefore critical to establish the correct diagnosis rapidly during a stroke code. Most patients with an aortic dissection will report chest or abdominal pain, which should prompt additional imaging (vascular or chest X-ray) prior to administration of tPA. However, in the small group of patients with painless aortic dissection, it can be difficult to establish a diagnosis with examination or routine studies alone. While the sensitivity is limited, checking for a diastolic murmur and symmetric upper extremity blood pressures and pulses may reduce inadvertent tPA administration to patients with aortic dissection during stroke codes. In the event an aortic dissection is discovered after initiating tPA, it is critical to discontinue the tPA infusion promptly, maintain strict blood pressure control, assess coagulation status, and consult the cardiothoracic surgery team for repair.

AUTHOR CONTRIBUTIONS

Dr. Renthal: concept, design, and writing of the manuscript. Dr. Alberts: critical revision of the manuscript for important intellectual content. Dr. Shang: critical revision of the manuscript for important intellectual content.

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DISCLOSURE

W. Renthal reports no disclosures relevant to the manuscript. M. Alberts is a speaker and consultant for Genentech. T. Shang reports no disclosures relevant to the manuscript. Go to Neurology.org for full disclosures.

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