42-Year-Old Male Methamphetamine User With Dysarthria and Facial Droop

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A 42-year-old man presented to the emergency department after the sudden onset of left hand numbness and tingling, dysarthria, and right-sided temporal region pain that began 30 minutes previously. On his way to the hospital, the patient had developed bilateral flank pain. He denied any significant medical history or history of similar symptoms and stated he had been in his usual state of health until the sudden onset of symptoms that brought him to the emergency department. He denied taking any medications and had no history of stroke, head trauma, recent bleeding, chest pain, back pain, palpitations, fevers, or chills. On direct questioning, he admitted to methamphetamine use. He usually administered methamphetamine intravenously; his most recent use was 2 days before presentation. He denied any history of sexually transmitted diseases. He stated that he regularly lifted weights but had not had any neck strains.

The patient was afebrile and had a blood pressure of 120/76 mm Hg and a regular heart rate of 102 beats/min. Neurologic examination showed minimal left hand clumsiness, mild left lower facial weakness, and minimal dysarthria; fundi appeared normal. Cardiac examination revealed no murmurs, rubs, or gallops. Findings on chest examination were normal, with the exception of mild costovertebral angle tenderness. Findings on abdominal, testicular, and extremity examinations were normal.

1. Which one of the following best explains the patient’s neurologic symptoms?
   a. Subarachnoid hemorrhage
   b. Cerebral vasospasm
   c. Intracerebral hemorrhage (ICH)
   d. Partial simple seizure
   e. Acute ischemic stroke

Patients with subarachnoid hemorrhage usually have a “thunderclap” headache or lose consciousness, rarely present without headaches, and often lack focal neurologic deficits. On physical examination, they may have photophobia and nuchal rigidity. Our patient had neither a headache nor physical examination findings consistent with a subarachnoid hemorrhage. Cerebral vasospasm usually manifests with uncontrolled hypertension and typically requires an inciting event such as hypertensive urgency or a delayed response to a subarachnoid hemorrhage. Our patient’s blood pressure was within a normal range. He had no immediate inciting event and denied recent methamphetamine use that could have led to hypertensive urgency.

Intracerebral hemorrhage most commonly occurs either in older patients with a history of hypertension or in patients receiving anticoagulation therapy with warfarin. This patient was relatively young, had not received anticoagulation therapy, and had normal blood pressure. Patients with partial simple seizure typically do not have headaches and present with a focal neurologic deficit that resolves and then recurs; this did not fit the clinical picture of our patient. Acute ischemic stroke can present with headache and fluctuating neurologic deficits and is the diagnosis most consistent with our patient’s history.

Electrocardiography revealed a normal sinus rhythm in our patient. An emergent complete blood cell count and a coagulation panel were performed, with normal findings. Levels of electrolytes were normal, but levels of creatinine were elevated to 2.1 mg/dL. Findings on chest radiography were unremarkable.

2. Which one of the following would be most appropriate as the next step in the management of this patient?
   a. Computed tomography (CT) of the head without radiocontrast agent
   b. Computed tomography of the head with radiocontrast agent
   c. Magnetic resonance imaging (MRI) with and without contrast agent
   d. Administration of intravenous tissue plasminogen activator (tPA)
   e. Discharge from the emergency department with close outpatient neurology follow-up

Computed tomography of the head without radiocontrast agent is initially indicated in patients with suspected stroke and reliably distinguishes acute ICH from ischemia. It is nearly 100% sensitive for ICH and approximately 90% sensitive for subarachnoid hemorrhage. Computed tomography of the head with radiocontrast agent would be unnecessary
and perhaps unsafe in a patient with an elevated creatinine level. Although MRI is an option for evaluating patients with stroke, it is more expensive and time-consuming. The time involved in performing an MRI may preclude its use in a patient who is undergoing emergent evaluation for an ischemic stroke. If a diagnosis of ischemic stroke is made, treatment with intravenous tPA is indicated if the acute stroke is of less than 3 hours’ duration in patients with persistent neurologic deficits. Intravenous tPA is not indicated in patients with a history of ICH, ischemic stroke within the previous 3 months, recent head injury or intracranial surgery, recent surgery, recent gastrointestinal or genitourinary bleeding, specific laboratory abnormalities (platelet count, <50 × 10⁹/L; international normalized ratio, >1.7; blood glucose level, >400 mg/dL or <50 mg/dL), or radiographic evidence of acute ischemia, ICH, or any other intracranial abnormality that could explain the neurologic deficit (eg, an intracranial mass). Discharging the patient from the hospital at this time would be inappropriate because he has complex symptomology without an established diagnosis.

No acute intracranial pathology was evident on CT of the brain without contrast agent. By the time the CT was completed, the patient’s neurologic symptoms had resolved, but the bilateral flank pain persisted. Because the patient’s neurologic deficits had resolved, fibrinolytic treatment was not given. Further diagnostic work-up was indicated.

The vascular neurology service was consulted; in accordance with their recommendations, the patient began taking one 325-mg tablet of aspirin orally and underwent an MRI of the brain and magnetic resonance angiography of the head and neck. These studies revealed 3 small acute cortical infarcts in the right frontal parietal region and poor filling of the right common carotid arteries (both internal and external) compared with those on the left. The erythrocyte sedimentation rate was 1.

3. Which one of the following diagnoses best explains all of these findings?
   a. Endocarditis
   b. Atrial fibrillation
   c. Isolated carotid artery dissection
   d. Systemic vasculitis
   e. Aortic dissection

Although the patient is an admitted intravenous drug user, he had no fevers, no leukocytosis, a normal erythrocyte sedimentation rate, and no murmur, making a clinical diagnosis of endocarditis unlikely. There was no history of palpitations, the heart rate was regular, and electrocardiography showed no rhythm disturbances, making a diagnosis of atrial fibrillation unlikely. Although an isolated carotid artery dissection would explain the patient’s neurologic symptoms, this diagnosis would not explain his renal findings. Systemic vasculitis is another consideration because it can involve any organ system and mimic other diseases. However, this patient’s acute presentation would argue against vasculitis involving the central nervous system because vasculitis usually presents as a subacute illness that evolves over weeks to months and is typically accompanied by an elevated sedimentation rate. Vasculitis is not commonly limited to renal and neurologic involvement with no other clinical manifestations. Acute aortic dissection with extension into the carotid and renal arteries could explain this patient’s acute clinical findings.

A diagnosis of aortic dissection was suspected even though the patient continued to deny chest and back pain. No difference was observed in pulse pressures in his arms.

4. Which one of the following is the most appropriate test to confirm the suspected diagnosis in this patient?
   a. Transthoracic echocardiography
   b. Abdominal CT with contrast agent
   c. Computed tomographic angiography
   d. Transesophageal echocardiography (TEE)
   e. Chest radiography

Transthoracic echocardiography has a low sensitivity and specificity for the diagnosis of aortic dissection. Image quality can be affected by obesity, emphysema, mechanical ventilation, or small intercostal spaces. Because of its poor sensitivity, this modality is not generally used in diagnosis. Abdominal CT and CT angiography both have an extremely high sensitivity and specificity for acute aortic dissection. They are noninvasive, readily available in most emergency departments, and quickly completed. However, both modalities require the use of an intravenous contrast agent, and our patient already had an elevated creatinine level of 2.1 mg/dL. Thus, neither is an ideal first-line test in our particular patient. Transesophageal echocardiography has a high sensitivity and specificity for acute aortic dissection, overcomes many of the limitations of transthoracic echocardiography, provides a higher degree of anatomic detail, can be performed at the bedside, and does not require arterial access, intravenous contrast agent, or ionizing radiation, making it the appropriate next step in confirming a diagnosis in our patient.

Chest radiography is a good starting point because it can reveal a widened mediastinum; however, it is not very sensitive for aortic dissection and so cannot be used to exclude this condition. In our patient, chest radiography would not be a good selection because findings on previous chest radiography were normal.

Transesophageal echocardiography revealed a dissection flap in the proximal ascending aorta. Although the
patient had acute renal insufficiency, discussion with the cardiothoracic surgery and vascular neurology services led to a decision to perform CT of the thorax and abdomen with contrast agent to further evaluate the extent of dissection. This study was performed, revealing a type A aortic dissection that extended through the thoracic aorta. The 2 left renal arteries arose from the false lumen, and a superior right renal artery arose near the junction of the true and false lumens.

5. Which one of the following would be the most appropriate intravenous therapy in this patient’s initial management?
   a. Enalaprilat
   b. Esmolol
   c. Hydralazine
   d. Heparin
   e. Nitroprusside

   Initial management of all aortic dissections involves lowering the blood pressure and decreasing the velocity of left ventricular contraction. These interventions decrease aortic shear stress and minimize the propagation of the dissection. Enalaprilat affects the renal vasculature and may cause a dangerous decrease in blood pressure in renal arteries that already feed into the false lumen. β-Blockers are the initial agent of choice because they decrease both the volume of contraction and systemic blood pressure. Esmolol is an appropriate choice given its short half-life in the event pressure drops too much. Hydralazine is commonly used to quickly lower blood pressure. However, it is often accompanied by a reflex tachycardia that can increase the derivative of raw blood pressure, increasing the pressure gradient of the vessel. Heparin is contraindicated in aortic dissection because of increased risk of aortic rupture and catastrophic bleeding. Heparin is also relatively contraindicated in acute stroke because of the risk of hemorrhagic conversion. Nitroprusside is an arterial and venous dilator that can be rapidly titrated to effect. It should not be used unless accompanied by β-blockers because vasodilation alone may induce reflex activation of the sympathetic nervous system, leading to enhanced ventricular contraction and increased aortic shear stress.

   The patient was treated with intravenous esmolol and taken to surgery. He underwent successful repair of a thoracoabdominal aortic dissection with ascending transverse arch repair and aortic valve root resuspension with deep hypothermic circulatory arrest. Although his hospital stay was complicated by difficulty weaning him from the ventilator, he eventually recovered. His renal function returned to baseline with a creatinine level of 1.2 mg/dL. He was instructed on the importance of blood pressure control and the necessity of avoiding methamphetamine use. After a 15-day hospital stay, he was discharged and experienced a successful outpatient recovery.

DISCUSSION

The incidence of aortic dissection, an uncommon but potentially catastrophic entity, is approximately 2.6 to 3.5 per 100,000 person-years.1 If the dissection is not treated, mortality rates during the first 24 to 48 hours are as high as 1% to 2% per hour; therefore, early and accurate diagnosis and treatment are crucial for survival. The peak incidence of aortic dissection is in the sixth and seventh decade of life, with men affected twice as often as women.3

The pathophysiology of aortic dissection involves weakening of any of the 3 contiguous tissue layers of the aortic wall: intima, media, and adventitia. Weakening of these layers usually leads to a tear in the intima that permits entry of blood between the intima and adventitia. Driven by persistent intraluminal pressure, the dissection process extends a variable length along the aortic wall. The blood-filled space between the dissected layers of the aortic wall becomes the false lumen.

Aortic dissections are classified by whether they originate in the ascending aorta, within centimeters of the aortic valve, or in the descending aorta, just distal to the origin of the left subclavian artery. Three major classification systems are used to define the location and extent of aortic involvement: DeBakey types I, II, and III; Stanford types A and B; and the anatomic categories proximal and distal. DeBakey types I and II involve the ascending aorta and are grouped together for simplicity with Stanford type A and the anatomic category proximal. DeBakey type III involves the descending aorta and is grouped with Stanford type B and the anatomic category distal. Most intimal tears occur in the ascending aorta.

According to the International Registry of Acute Aortic Dissection (IRAD),4 the most important predisposing factors for acute aortic dissection are systemic hypertension (72%) and atherosclerosis (31%). However, these factors were less important in young patients; IRAD analysis showed that only 34% of patients younger than 40 years had a history of hypertension and only 1% had a history of atherosclerosis. Our patient was found to be normotensive on presentation, as were most of the younger patients in the IRAD analysis. Interestingly, our patient was an admitted methamphetamine user, and an association between methamphetamine use and aortic dissection has been documented by 1 case report series.5 The median age in that series was 39.5 years, with blood pressures ranging from the normotensive to hypertensive range on initial presentation. Other predisposing factors found in the IRAD analysis included pre-existing aortic aneurysm, vasculitis, bicus-
Rapid aortic valve, coronary artery bypass grafting, Turner syndrome, cardiac catheterization, previous aortic valve replacement, disorders of collagen, and trauma.

The clinical manifestations of acute aortic dissection are diverse. Although the IRAD analysis revealed that chest pain was the single most common presenting symptom, occurring in 72.7% of patients, other presenting symptoms included cerebrovascular accident, heart failure, syncope, pulse deficit, aortic regurgitation, and shock/tamponade. In one study, painless acute aortic dissection accounted for 6.4% of all presentations and was associated with a less favorable outcome than painful acute aortic dissection. The initial aortic tear and subsequent extension of a false lumen along the aorta can occlude blood flow from the true lumen of the aorta into any of the arteries that originate from the aorta. Depending on which arteries become occluded, patients can present with a variety of clinical sequelae, including acute myocardial infarction (coronary artery dissection), syncope, hemiplegia (carotid artery occlusion), anuria/flank pain (renal artery flow disruption), paraplegia or quadriplegia (occlusion of vessels feeding the anterior spinal artery), and death.

Diagnosis of aortic dissection is primarily made on the basis of history and physical examination findings. Chest radiography classically shows a widened mediastinum. However, the IRAD analysis showed no findings of widened mediastinum or abnormal aortic contour in 21.3% of patients presenting with acute aortic dissection. The imaging modalities of choice in diagnosis of acute aortic dissection are CT and TEE. Both modalities have a sensitivity and specificity greater than 90%. More readily available in most emergency departments, CT is completed quickly. Readily available in many larger centers, TEE can be completed quickly at the bedside, which makes it ideal for evaluating unstable patients.

When all modalities are available, CT should be considered first in the evaluation of suspected aortic dissection in light of its accuracy, safety, speed, and convenience. When CT identifies a type A dissection, the patient may be taken directly to the operating room, where TEE can be performed to assess the anatomy and competence of the aortic valve without delaying emergent surgery. Patients with uncomplicated type B dissections may be managed medically. According to IRAD data, type B dissections have a 10% in-hospital mortality rate with medical management alone and a 60% to 80% 5-year survival rate.

Patients with type A dissections require urgent surgery to avoid death as a consequence of intrapericardial rupture, cardiac tamponade, or myocardial infarction. IRAD data showed mortality rates as high as 1% to 2% per hour after symptom onset if the dissection was left untreated. The objective of surgical therapy for aortic dissection is to dissect the damaged segment, excise the intimal tear, and obliterate the entry into the false lumen. The procedure is performed primarily via median sternotomy. Repair involves dissection from inside the aorta to determine the extent of the damage to the intima and media and to remove the damaged segments. The aortic wall is rebuilt and continuity of the aorta is re-established by placement of a Dacron prosthetic sleeve graft between the ends of the aorta.

Our case is a perfect illustration of how an acute aortic dissection can present clinically in a nonspecific manner. The unifying diagnosis of acute aortic dissection explained the patient’s transient neurologic symptoms, head pain, and costovertebral angle tenderness with renal insufficiency. Misdiagnosis of an acute aortic dissection as unstable angina, myocardial infarction, or an acute nonhemorrhagic stroke can lead to disastrous iatrogenic consequences should the patient receive therapy with either intravenous heparin or fibrinolytic agents. Clinicians must be aware of the varied presentations of an acute aortic dissection and have a high index of suspicion for prompt evaluation of this potentially fatal clinical entity.

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REFERENCES


Correct answers: 1. e, 2. a, 3. e, 4. d, 5. b