MAYO CLINIC

82-Year-Old Woman With Acute-Onset Left-Sided Weakness

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See end of article for correct answers to questions.

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An 82-year-old woman with a history of type 2 diabetes mellitus, hyperlipidemia, and hypertension presented to the emergency department with sudden onset of left arm and leg weakness. She was sitting in church when she developed left-sided posterior neck and shoulder pain, followed 1 hour later by sudden-onset dense left-sided weakness. Emergency medical services were called, and they activated the prehospital stroke-notification protocol. The patient arrived at the emergency department approximately 30 minutes after the onset of the weakness. She had no previous history of these symptoms. She had no history of loss of consciousness or epileptiform activity. She denied any history of trauma. She also denied any recent history of fatigue, fever, chills, diaphoresis, or rigors. Her home medications included atenolol 50 mg twice daily, glimepiride 2 mg twice daily, lisinopril 20 mg daily, hydrochlorothiazide 25 mg daily, and acetaminophen as needed for joint pain in knees and hands.

On examination, she was visibly uncomfortable, owing to left-sided neck and shoulder pain. Her blood pressure was 172/73 mm Hg, pulse 70 beats per minute, respiratory rate 16 breaths per minute, and oxygen saturation was 97% while breathing room air. On examination, she had flaccid weakness of the left arm and leg. Sensation was preserved. The remainder of the neurological examination, including cranial nerves, was normal. No Horner syndrome was seen.

Her calculated National Institutes of Health (NIH) Stroke Scale was 8 (score of 4 for "no movement" of left arm and 4 for "no movement" of left leg). Blood tests revealed the following (reference ranges provided parenthetically): serum glucose 176 mg/dL (70 to 140 mg/dL); hemoglobin 12.2 g/dL (12.0 to 15.5 g/dL); platelet count 255 X10^9/L (150 to 450 X10^9/L); prothrombin time 10.8 sec (8.8 to 11.9 sec); international normalized ratio (INR) 1.1 (0.9 to 1.2); and activated partial thromboplastin time 27.2 sec (23.7 to 36.1 sec). A noncontrast computed tomography (CT) scan of the head did not reveal any acute intracranial abnormalities.

1. **Which *one* of the following would be the *most appropriate* next course of action?**
   a) Intravenous thrombolysis
   b) Thromboembolectomy
   c) Further imaging
   d) Lumbar puncture
   e) Admission for cardiac monitoring

The presentation could be concerning for an ischemic stroke. The incidence of acute ischemic stroke in this age group is significant and thus should be high on the differential diagnosis. A noncontrast head CT scan is often performed in the setting of acute stroke and is helpful to rule out hemorrhage. Ischemic stroke, however, may not be detectable early on, and a normal head CT scan does not rule out this possibility. What is atypical for ischemic stroke in this case is the associated neck and shoulder pain. The pattern of weakness can also help localize the lesion and thus guide further investigations; arm and leg weakness without associated face weakness would be less consistent with a cortical lesion.

Intravenous thrombolysis is considered in select patients with ischemic stroke within 4.5 hours of onset of symptoms. However, with her neck and shoulder pain occurring around the same temporal period as her hemiparesis, one would want to consider other etiologies that can present

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with pain and hemiparesis—such as aortic artery dissection, cerebral artery dissections, or spinal hematomas—before administering therapies such as intravenous thrombolysis. A thromboembolectomy would be considered in select patients with acute stroke and thrombus seen in the proximal cerebral arteries on CT angiogram. However, as a CT angiogram had not been performed yet, it would be premature to consider thromboembolectomy as the next course of action. Further imaging would be the most suitable choice in this situation given the atypical presentation of the left-sided hemiparesis.

A lumbar puncture would be indicated had there been clinical suspicion of subarachnoid hemorrhage (eg, presenting with a thunderclap headache) for this patient with a normal noncontrast CT scan of the head. Admission for cardiac monitoring would not be advised in this scenario, as time is of the essence to establish a diagnosis so that appropriate management can be instituted.

A telestroke consultation was initiated, and the consulted physician thought that the history did not fit a classic ischemic stroke, especially given the neck and shoulder pain. The decision was made to proceed with further imaging. A CT angiogram of the chest, neck, and head vessels was performed, which did not show any dissection of the aortic, vertebral, or carotid arteries.

2. **Which *one* of the following would be the *most appropriate* next imaging investigation?**
   a) Noncontrast MRI scan of the cervical spine
   b) Noncontrast MRI scan of the head
   c) Plain x-ray of the neck
   d) Conventional cerebral angiography
   e) Ultrasound of the soft tissues of the head and neck

An MRI cervical spine scan without contrast material would be the most appropriate next step. The neurological presentation of unilateral weakness without facial involvement, together with the neck pain, localizes the lesion to somewhere in the neck region. A CT angiogram was already done to exclude a carotid or vertebral artery dissection. Thus, an MRI scan of the cervical spine should be done next to assess the cervical spinal cord. A noncontrast MRI scan of the head would be more sensitive in diagnosing an acute ischemic stroke than a noncontrast CT scan of the head, but without also including the neck, pathologies in the neck might be missed. A plain x-ray of the neck would be able to look at obvious bony abnormalities of the cervical spine but would not be able to assess the soft tissues. A conventional cerebral angiography (done as a digital subtraction angiography) is a catheter-based angiography that remains the gold standard for evaluating cerebral vessels for arterial stenosis, dissection, or vascular malformations. Our clinical suspicion is pointing us toward a lesion in the neck region, thus making a conventional cerebral angiography unnecessary. An ultrasound of the soft tissues of the head and neck would be useful to assess soft tissue neck masses and help with lymph-node assessment. In newborns and infants, ultrasonography is a well-established method of investigating the spinal canal and cord because the incompletely ossified and predominantly cartilaginous spinal arches in this age group create a good acoustic window.[1] However, in adults, mature ossified bone prevents useful examination of the spinal cord. Thus, although an ultrasound of the soft tissues can help us investigate the cause of the neck pain, it will not help us visualize pathologies in the cervical spinal cord, which is our focus for the likely cause of the acute neurological deficit.

The stroke physician recommended a noncontrast MRI scan of the cervical spine. This showed an abnormal heterogeneous posterior left epidural fluid collection extending from C2 to C5 with associated mass effect upon the cervical cord.

3. **Based on the *clinical picture and imaging findings*, what would be the *most likely* diagnosis?**
   a) Epidural abscess
   b) Epidural cyst

c) Epidural hematoma
d) Epidural tumor
e) Dural arteriovenous fistula

The left epidural fluid collection would appear to be the culprit causing the patient's symptoms. Ascertaining the cause of the fluid collection would determine the next plan of management.

An epidural abscess is unlikely, given that abscesses tend to happen more slowly, leading to progressive neurological symptoms, and the patient did not have any other signs of infection such as fever, chills, or rigors. An epidural cyst is also unlikely, given that it tends to grow more slowly, and the symptoms tend to be more insidious in onset. An epidural hematoma would best fit the description of the MRI findings and the timing of the presentation (acute onset of pain and neurological deficit). An epidural tumor would be a solid structure (with or without fluid components) rather than a pure fluid collection and symptoms would occur in a more insidious manner. Patients with spinal dural arteriovenous fistula typically present with slowly progressive myelopathy caused by spinal-cord congestion rather than bleeding. The symptoms are usually slowly progressive over days to weeks, but rapid symptoms can occur if bleeding ensues. However, in this case, this would not be the best answer, as the collection is located in the epidural layer.

The patient continued to have left-sided hemiparesis.

4. **What would be the *next best* plan of management?**
   a) Surgical referral for decompression
   b) Observation and reimaging in 24 hours
   c) Intravenous thrombolysis
   d) Clopidogrel 300 mg orally
   e) Anticoagulation with enoxaparin

Surgical referral for evaluation and decompression would be the most appropriate next step. The diagnosis of spinal epidural hematoma requires prompt referral to a spine surgery team for evaluation. Patients with significant and/or progressing neurological deficits require urgent surgical intervention, usually a laminectomy, and evacuation of the blood. Observation and reimaging would not be an option, as the patient had a significant neurological deficit. Timely decompression of the hematoma is essential to avoid permanent loss of neurologic function. If the patient already had significant neurological improvement before assessment, observation and reimaging would be a reasonable option. Administration of thrombolytics, antiplatelet agents (aspirin), or anticoagulation (enoxaparin) is contraindicated and could cause the hematoma to enlarge, further compressing the spinal cord.

The patient was transferred to a tertiary medical center for assessment by the spine surgery team. During her transfer, she began to recover her neurological function. By the time she arrived at the tertiary center and was assessed by the surgical team, she only had mild weakness of the left upper and lower extremity, NIH Stroke Scale of 2 (score of 2 for "some effort against gravity" of the left arm).

5. **What is the *most likely* etiology of this patient's hematoma?**
   a) Bleeding secondary to antiplatelet medications
   b) Bleeding secondary to anticoagulant medications
   c) Bleeding secondary to trauma
   d) Bleeding secondary to cervical-spine instrumentation
   e) Spontaneous bleeding

There was no identifiable risk factor and no precipitant for the patient's epidural hematoma. After careful history taking, once the patient was in the tertiary center, we did not identify any medications that could have predisposed her to a hematoma: specifically, antiplatelet and anticoagulant medications. We also clarified the patient's history and determined that there was no preceding trauma, even trivial trauma (such as a sudden neck jerk while driving caused by braking). She also denied any recent cervical spine instrumentation or manipulation

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(such as a manipulation from a chiropractor). Thus, this was classified as a spontaneous cervical epidural hematoma.

Serial clinical examinations done over 48 hours showed that her neurological function had normalized (NIH Stroke Scale score of 0). Because of resolution of the patient's neurological impairment, surgery was not performed, and she was discharged home with appropriate follow-up.

DISCUSSION

Unilateral weakness is a worrisome presentation. Although the etiologies of unilateral weakness are diverse, one of the most common causes is stroke. With an incidence rate of 670 to 970 per 100,000 patients per year[2] in this patient's age group, this would be the first diagnosis that comes to mind. However, other etiologies that can mimic acute stroke should also be considered, such as seizure with postictal paresis (Todd paralysis), migraine aura, hypertensive encephalopathy, spinal-cord disorders, and functional deficit. Studies have reported a misdiagnosis rate of stroke of approximately 10%.[3] Common reasons for misdiagnosis include preconceived notions about patients (for example, assuming that young people do not get strokes), being misled by the patient's use of words (for example, the patient says "I am having another migraine attack") or misinterpretation of diagnostic testing (for example, a false normal noncontrast CT scan of the head in early stroke or using a CT scan to diagnose a cerebellar or brain-stem infarction).[3] Careful clinical history and physical examination can help elucidate the etiology or may call to light features that would not be consistent with a particular diagnosis.

When assessing a patient with a neurological deficit, a good approach would be to localize the lesion based on the pattern of the presentation, then to think of an etiology that could affect that area, and finally guide your imaging modality based on the likely etiology. In this case, the patient had negative results of a noncontrast CT scan of the head, which would be expected in an acute ischemic stroke. However, the patient had concurrent neck and shoulder pain, which does not fit the classic presentation of an acute ischemic stroke. Also, the pattern of weakness does not fit with a typical cortical stroke, as there was sparing of the face. The hemiparesis with sparing of the face points toward a subcortical internal capsule lesion, a medullary pyramid lesion, or a spinal-cord lesion. With the associated neck pain, the focus was to investigate etiologies localized to the neck region.

The incidence of acute aortic dissection ranges from 2.6 to 3.5 patients per 100,000 patients per year,[4-6] with the incidence of spontaneous internal carotid artery dissection 1.72 per 100,000 patients per year.[7] These conditions can present with chest and neck pain, respectively, with neurological deficits. This, along with the patient's history of hypertension (being on multiple antihypertensive medications), would warrant investigation to effectively exclude the diagnosis. The CT angiogram of the chest, neck, and head vessels ruled out this diagnosis in our patient.

Spontaneous spinal epidural hematomas are rare, with 1 study estimating the incidence to be 0.1 patients per 100,000 patients per year.[8] It is most frequent after the fourth or fifth decade, although it has been reported to occur in all age groups.[9] The pathophysiology is uncertain, but theories include rupture of the epidural venous plexus and rupture of the spinal epidural arteries as the source of hemorrhage. Sudden-onset neck and shoulder pain followed shortly thereafter by a neurological deficit would be the most common presentation of a cervical epidural hematoma. The characteristic and rapidity of the developing neurological deficit would depend on the level of the lesion and area of the spinal cord affected as well as the severity and rapidity of the bleed. Precipitating factors include antiplatelet therapy, anticoagulation therapy, vascular malformations, trauma, and iatrogenic spinal instrumentation. However, there are reported cased on spontaneous spinal epidural hematoma in the literature.[9-11] Treatment usually involves surgical intervention with evacuation of the

hematoma to alleviate the compression of the spinal cord by the hematoma. The neurological recovery is significantly correlated with the timing of surgery,[12] and thus early intervention is key. Conservative management (as in this case) is uncommon but can be considered when neurological symptoms improve before medical evaluation. The hypothesis for the spontaneous recovery of the neurological deficit appears to relate to the spreading of the hematoma throughout the epidural space, thus decreasing the pressure on the spinal cord and its nerve roots.[10]

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CORRECT ANSWERS: 1. c. 2. a. 3. c. 4. a. 5. e