

Spinal epidural hematoma: an important stroke mimic

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Abstract

Tissue plasminogen activator (tPA) is, on occasion, given to patients who do not suffer from acute cerebral ischemia. As the underlying conditions often mistaken for acute ischemic stroke tend to produce transient dysfunction, and are generally seen in individuals younger than stroke patients, the outcome of such mistaken treatment is generally benign. We will describe two elderly patients with acute hemiparesis caused by spinal epidural hematomas (SEDH), both of whom were initially considered candidates for tPA. The literature review and discussion will emphasize features allowing the distinction between these unusual hemiparetic presentations of SEDH and acute brain ischemia, and briefly review other cervical lesions that may rarely present with hemiparesis.

Keywords

Tissue plasminogen activator, spinal epidural hematoma, stroke mimics

Key Points

1. Cervical spinal epidural hematoma (SEDH) usually presents with quadriparesis, but may occasionally present with hemiparesis, mimicking acute stroke.
2. Imaging studies routinely used in acute cerebral ischemia do not rule out spinal hemorrhage.
3. Severe posterior cervical pain, absence of unequivocal facial weakness, and contralateral loss of pain and temperature sensation suggest cervical SEDH as a possible diagnosis in patients presenting with acute hemiparesis.
4. Cervical SEDH is an important stroke mimic to consider, since the consequences of thrombolysis in this setting would be catastrophic.

Introduction

Tissue plasminogen activator (tPA) is occasionally given to patients whose neurological symptoms prove not to be the result of acute cerebral ischemia. Outcome is usually benign in such cases, because the underlying conditions – usually epilepsy, migraine or psychogenic illness, produce transient dysfunction. In such patients, complications following tPA are few. Intracranial hemorrhage has never been reported,¹ probably because there is no acute or chronic cerebrovascular pathology.

Administration of tPA to a patient with CNS hemorrhage would be a serious error. Fortunately, such errors are extremely rare, because intracranial hemorrhage is rapidly and reliably detected by plain cranial CT. However, spinal hemorrhage is not ruled out using any imaging modality used to investigate acute cerebral ischemia.

Spinal epidural hematoma (SEDH) usually presents with quadraparesis or paraparesis, but acute hemiparesis has been documented repeatedly^{2–4} and might raise acute cerebral ischemia as a diagnostic consideration. Thus, SEDH is also a potential

“stroke mimic,” but one in which thrombolytic therapy would be catastrophic. In addition to exacerbating the SEDH, it would also complicate emergency surgery. SEDH has rarely occurred *de novo* after intravenous tPA administration in proven acute cerebral ischemia.^{5–6}

We present two patients initially diagnosed as ischemic stroke, and considered candidates for intravenous tPA, who turned out to have SEDH as the cause of their acute hemipareses.

Case 1

A 96 year old woman awakened with upper back, left shoulder and neck pain. Weakness of her left limbs developed 45 minutes later. Her past medical history included hyperlipidemia, hypertension and a prior stroke that had resolved. She was taking aspirin and clopidogrel. Neurological exam revealed age-appropriate mental status and a mild droop in the left nasolabial fold, with otherwise normal cranial nerves. The left extremities were weak: 4+/5 in the proximal arm, grip and leg 3/5; right extremities had normal strength. Sensation was normal to pinprick

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and cold, without neglect. The left plantar was extensor. Tendon reflexes were slightly brisker on the left; ankle jerks were absent. Cranial CT showed evidence of previous cerebral infarction but no acute abnormality. Coagulation studies were normal. tPA was not administered because of advanced age, uncertainty regarding time of onset, and concern over the cause of pain. MRI of the brain (MRIb) showed no acute lesion. MRI of the cervical spine (MRIcsp), after MRIb results became known, revealed a left sided SEDH from C5 to C7 (Figures 1 & 2). This was removed at laminectomy. On discharge, she was able to mobilise with a walker.

Case 2

An 81 year old woman abruptly developed pain in the posterior neck and both shoulders that radiated to her right arm. At another hospital, weakness of the right limbs and a negative cranial CT scan were documented. Coagulation studies (prothrombin and partial thromboplastin times and platelet count) were normal. tPA was not administered because of uncertainty about time she was last known to be well. Her past medical history included hypertension, diabetes, hyperlipidemia, and coronary artery bypass surgery. She had been taking aspirin. After transfer to our institution, neurological exam revealed a dense right hemiparesis: minimal shoulder and foot movement remained; strength of the left limbs was normal. Tone was normal. Pinprick sensation was reduced to a T2 level on the left; vibration and position were normal. Plantars were silent. Only the left biceps jerk could be obtained. However, cranial nerve examination showed no relevant abnormality and her mental state and speech were normal. MRIcsp revealed a thick right sided SEDH from C3 to the superior aspect of C6. This was immediately removed at laminectomy. On discharge, she was able to mobilise with a walker.

Discussion

To our knowledge, there is only a single case report of a patient with a SEDH being given tPA,⁷; in this case the suspected arterial pathology was vertebral dissection. This patient was unusual in that there was flaccid weakness in three limbs. D'Souza and colleagues⁸ report a "close call" in a patient who presented with acute hemiparesis and interscapular pain radiating to the arm. When a minor facial asymmetry was confirmed as chronic, further examination revealed a partial Brown-Sequard syndrome, and the correct diagnosis. Both of these cases demonstrate the necessity for continuing evaluation of suspected stroke patients presenting with atypical features.

Our patients, and those from the literature, provide several instructive points concerning SEDH as a stroke mimic. In these hemiparetic elderly patients, the initial

diagnostic consideration was acute cerebral ischemia. Features which suggested the correct localization included the finding of a partial Brown-Sequard syndrome in case 2, and severe neck pain in both cases. The latter is distinctly unusual in cerebral ischemia, except in the case of extracranial carotid or vertebral dissection. However, the pain accompanying dissections is rarely prominent in the shoulder or arm(s), is usually lateralized, and in the case of carotid dissections, is more prominent in the anterior neck area.

The early symptoms of SEDH can fluctuate, and even change sides,⁹ behavior that might suggest fluctuating perfusion of the brain, or a brainstem lesion. Indeed, the fluctuations seen in patients with SEDH have been attributed to fluctuating spinal cord perfusion caused by pressure on spinal arteries.⁹ Very high cervical SEDH produces respiratory distress^{3,10} and extension into the posterior fossa may suggest brainstem ischemia by affecting lower cranial nerves. Fortunately, the higher a cervical SEDH extends, the more likely it is to be seen on non-contrast head CT. In cases where an emergent MRI cannot be obtained, spinal CT may enable identification of large SEDH.

Acute stroke assessment may be confounded by the finding of minor facial asymmetry as in our first case. In the absence of confirmation that it predates the acute presentation the clinician must formulate the case both with, and without, considering it. It should also be emphasized that, while assessment of position and vibration sensations is not required by the NIH stroke scale, these modalities may prove quite useful in demonstrating a full Brown-Sequard syndrome. Even within the constraints of the NIH Stroke Scale, the finding of sensory loss to pinprick and temperature contralateral to the hemiparetic side should direct the clinician to suspect a spinal localization.

Other non-traumatic cervical spinal pathologies may rarely present with rapid-onset hemiparesis, or a partial Brown-Sequard syndrome. These include cervical spondylosis,¹¹ spinal cord infarction,¹² demyelinating disease, and intraparenchymal cord hemorrhage. Among these causes, the authors have seen several cases of acute hemiparesis following forceful neck flexion-extension in patients with pre-existing cervical spondylosis and compromised diameter of the cervical canal. Other cord syndromes - central cord syndrome or quadriparesis - are more likely to follow such an event. Demyelinating disease as a cause of rapid-onset hemiparesis would not be a diagnostic problem in patients with well established multiple sclerosis, but might be potentially misleading in patients with a first demyelinating event (i.e. a clinically isolated syndrome). Among the other entities conceivably causing diagnostic confusion, only parenchymal cord hemorrhage would be a firm contraindication to use of tPA, and for this rare situation, the same considerations mentioned for SEDH would apply.

Declaration of competing interests: Nothing to declare

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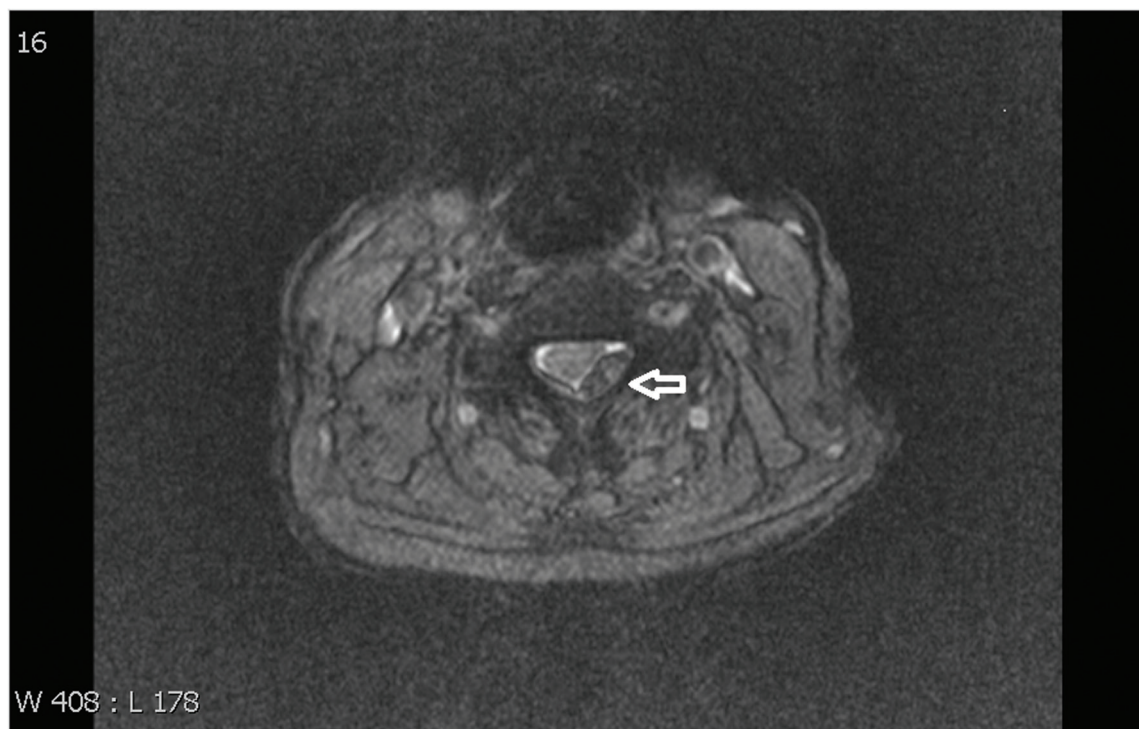


Figure 1. Cranial MRI - arrow indicates poster-laterally located SEDH in patient 1

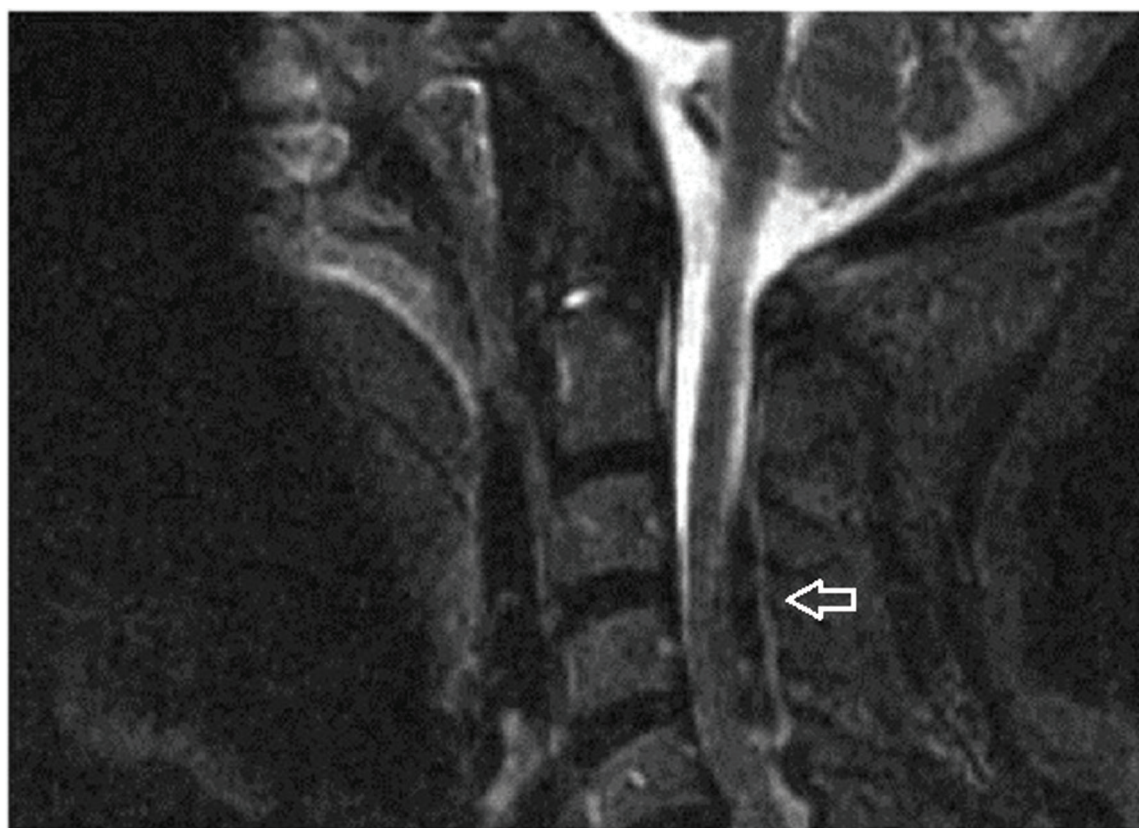


Figure 2. Coronal MRI image with SEDH indicated by arrow

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