Pearls & Oy-sters:

Localization in acute stroke management

Thinking straight when it comes down to crunch time

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PEARLS Localization of the stroke syndrome with possible elucidation of the underlying pathophysiology is of paramount importance before initiating IV recombinant tissue plasminogen activator (rtPA).

OY-STERS A high NIH Stroke Scale score in isolation, without due consideration of the underlying stroke mechanism, should not be used as a selection criteria for acute stroke therapy with IV rtPA.

Dedicated and localized imaging studies should be performed if the clinical picture is not typical of a stroke syndrome, especially so if IV rtPA is being considered.

CASE HISTORY A 56-year-old woman presented to the emergency department of our hospital with acute-onset weakness of the right upper and lower extremities of 2.5 hours' duration. This was associated with mild pain and vague sensory symptoms involving the homolateral side. The NIH Stroke Scale score at presentation was 9. The initial CT scan showed no evidence of an intracranial bleed. CT angiogram showed normally opacified extracranial carotids, extracranial vertebrals, and intracranial vasculature. On examination, the heart rate was 72 beats/min and regular, and the blood pressure was 148/80 mm Hg. The Acute Stroke Team of our hospital was activated for IV thrombolysis.

On further neurologic assessment, the patient had no features of an expressive or receptive aphasia. There were no signs of any inattention or neglect. The extraocular movements were complete with normally reacting pupils. There was no impairment of sensation over the face or any evidence of facial nerve palsy. Palatal movements were complete and symmetrical with no deviation of the tongue. Muscle tone on the right was decreased with grade 0/5 power of the right upper extremity and 1–2 power of the right lower extremity. Deep tendon reflexes on the right were absent with an upgoing plantar on the right. Muscle power and reflexes were normal on the left. Sensory system examination revealed mildly reduced proprioception and vibration sense on the

right. Pain and temperature sense was grossly normal on both sides.

In view of the absence of any cortical or cranial nerve signs, the possibility of lower medullary/upper cervical cord pathology was considered. Historical clues for a vertebral artery dissection were negative, including chiropractic neck manipulation or recent neck trauma. A focused examination for possible medial medullary or Brown-Séquard syndrome was done; however, there was no involvement of the hypoglossal nerve or a crossed sensory pattern to corroborate the above clinical diagnoses.

Further discussion with the neuroradiologist and a closer look at the CT angiogram revealed an area of hyperdensity within the cervical spinal canal (figure, A). An urgent MRI scan of the spine was done, which revealed an epidural hematoma extending from the inferior border of C2 to C5 causing cord compression that was predominantly located on the right (figure, B and C). An urgent C2-C4 laminectomy was done with evacuation of the blood clot. A bleeding epidural vein was seen adjacent to the clot.

Postoperatively the patient underwent rehabilitation with subsequent improvement of her neurologic deficits.

DISCUSSION Stroke syndromes commonly present as hemi-sensorimotor deficits with a varying combination of other neurologic signs.¹ Large hemispherical strokes are frequently associated with cortical signs such as language dysfunction when the dominant hemisphere is involved or visuospatial abnormalities when the nondominant hemisphere is involved. Subcortical stroke syndromes involving the centrum semiovale can present with differential weakness of the upper and lower extremities depending on the arterial territory involved, whereas those involving the internal capsule present with dense deficits. Brainstem stroke syndromes are classically associated with crossed hemiparesis wherein there are ipsilateral cranial nerve signs and contralateral corticospinal signs.

Two rather uncommon causes of a hemi-sensorimotor syndrome with minimal to absent

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(A) CT angiogram of the brain shows an area of hyperdensity within the cervical spinal canal anterior to the C3-C4 posterior processes. (B) T2-weighted sagittal image of the cervical spine showing an epidural collection anterior to the spinous processes of C3 and C4 vertebrae with resultant compression and anterior displacement of the cord. (C) T2-weighted axial image of the cervical spine at the level of C3 demonstrating a posteriorly based epidural collection on the right with cord compression.

cranial nerve signs are the medial medullary syndrome and a high cervical spine Brown-Séquard syndrome.

The medial medullary syndrome or the “syndrome of Dejerine” results from infarction of the anteromedial part of the medulla. This results in facial-sparing hemiparesis and hemisensory loss, of the posterior column type, contralateral to the side of infarct and weakness of the tongue ipsilateral to the infarct. These signs are attributable to the involvement of the pyramidal tract rostral to their decussation, the medial lemniscus, and the fibers and nucleus of the hypoglossal nerve. This triad of clinical features may not be seen in all patients with a medial medullary syndrome. And to further confound the clinical picture, some patients may have additional signs depending on the rostrocaudal and mediolateral involvement of the ischemic territory. A prospective clinical-imaging study done on 86 patients with medial medullary infarction demonstrated that most patients had a combination of a motor and sensory neurologic deficit involving the contralateral side followed by a varying combination of brainstem features, which included in decreasing frequency dysarthria, vertigo, nystagmus, limb ataxia, and dysphagia. Of note, an ipsilateral tongue deviation was seen in only 3 of the 86 patients.² This discrepancy was explained by a more rostral involvement of the ischemic territory sparing the hypoglossal nuclei. Similar observations have been made by several other investigators.³

Brown-Séquard syndrome involving the cervical spine classically presents with a hemi-sensorimotor syndrome with weakness and posterior column sensory loss on the side ipsilateral to the cord pathology and sensory loss of a spinothalamic type on the contralateral side. Differential involvement of these tracts can lead to a partial Brown-Séquard syndrome

depending on the location and extent of the hemicord pathology. This syndrome is usually associated with trauma, slowly growing epidural tumors, cervical disk diseases, cervical epidural hematomas, and very rarely spinal cord infarction.

Spinal cord infarction is a rare cause of acute myelopathy accounting for only 1% of all strokes and 5% to 8% of acute myelopathies.⁴ There are several potential mechanisms of spinal cord infarction; atherosclerotic disease and surgical procedures involving the aorta are the most common association. There are several distinct spinal cord ischemic syndromes based on the vascular anatomy of the spinal cord. The anterior spinal artery syndrome is the most common and patients present with acute-onset symmetrical weakness and loss of sensation, involving the spinothalamic modalities, with a distinct spinal level. This is due to disruption of the lateral corticospinal and lateral spinothalamic tracts in the anterior two-thirds of the spinal cord, which is distributed by the anterior spinal artery. Posterior spinal artery syndrome is rare and patients present with acute-onset loss of sensation of a posterior column type. A partial Brown-Séquard syndrome due to occlusion of the sulcommissural artery is an extremely rare cause of spinal cord infarction.

Spinal epidural hematoma is an extremely rare and devastating neurologic emergency that needs to be considered in any patient presenting with acute weakness of presumed spinal cord pathology. It usually occurs as a complication of trauma or postoperatively, especially so if the patient has an underlying coagulopathy. Spontaneous or idiopathic spinal epidural hematoma represents about 40% of all spinal epidural hematomas and occurs at an estimated incidence of approximately 0.1 per 100,000 patients.^{5,6} The

hematoma in these patients can be of arterial or venous origin. Venous bleed occurs as a result of sudden increase in the intrathoracic or intraabdominal pressure leading to rupture of the thin-walled epidural veins. Alternatively, extreme movements of the neck can lead to tearing of the arteries. Our patient demonstrated a hematoma secondary to a bleeding epidural vein. The clinical manifestations depend on the longitudinal and transverse extent of the bleed within the spinal canal. The most common longitudinal localizations are within the cervicothoracic and thoracolumbar junctions. On a transverse plane, about 75% of the hematomas are located posterior to the spinal cord with only 5% being anterior.^{7,8} Symptoms evolve quite rapidly over a few hours and are usually preceded by localized neck or back pain, which at times have a radicular nature. Most patients predominantly have motor-sensory deficits, with associated bladder and bowel dysfunction to a lesser extent.⁹ Brown-Séquard syndrome is a recognized presentation of cervical epidural hematoma. Prompt recognition of this association with early surgical evacuation can lead to good long-term outcomes.¹⁰

Our case highlights the importance of localizing stroke syndrome and elucidating possible stroke mechanisms before initiating acute stroke therapy with IV rtPA. This can be extremely challenging, especially so when we act within a very tight therapeutic window with ever more emphasis on the fact that “time lost is brain lost.” Early localization of the stroke syndrome and discussion with the neuro-radiologist prompted us to obtain an urgent MRI scan of the cervical spine with subsequent referral to the neurosurgeon for urgent decompressive surgery. Initiation of IV rtPA would have had catastrophic consequences otherwise!

AUTHOR CONTRIBUTIONS

Dr. Joy Vijayan and Dr. Teoh Hock Luen helped with the formulation of the article and the compilation of literature. Dr. Eric Ting and Dr. Chou Ning helped with clinical assessment of the patient and provided guidance on the formulation of the article.

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REFERENCES

1. Balami JS, Chen RL, Buchan AM. Stroke syndromes and clinical management. *Q J Med* 2013;106:607–615.
2. Kim JS, Han YS. Medial medullary infarction: clinical, imaging, and outcome study in 86 consecutive patients. *Stroke* 2009;40:3221–3225.
3. Toyoda K, Imamura T, Saku Y, et al. Medial medullary infarction: analyses of eleven patients. *Neurology* 1996;47:1141–1147.
4. Rubin MN, Rabinstein AA. Vascular diseases of the spinal cord. *Neurol Clin* 2013;31:153–181.
5. Holtas S, Heiling M, Lonntoft M. Spontaneous spinal epidural hematoma: findings at MR imaging and clinical correlation. *Radiology* 1996;199:409–413.
6. Kreppel D, Antoniadis G, Seeling W. Spinal hematoma: a literature survey with meta-analysis of 613 patients. *Neurosurg Rev* 2003;26:1–49.
7. Groen RJ, Ponssen H. The spontaneous spinal epidural hematoma: a study of the etiology. *J Neurol Sci* 1990;98:121–138.
8. Liao CC, Lee ST, Hsu WC, et al. Experience in the surgical management of spontaneous spinal epidural hematoma. *J Neurosurg* 2004;100(1 suppl spine):38–45.
9. Vandermeulen EP, Van Aken H, Vermeylen J. Anticoagulants and spinal-epidural anesthesia. *Anesth Analg* 1994;79:1165–1177.
10. Cai HX, Liu C, Zhang JF, et al. Spontaneous epidural hematoma of thoracic spine presenting as Brown-Séquard syndrome: report of a case with review of the literature. *J Spinal Cord Med* 2011;34:432–436.

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