A case of lateral medullary syndrome

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Abstract
Lateral medullary syndrome (LMS), known as Wallenberg's syndrome and posterior inferior cerebellar artery syndrome, is a rare cause of stroke. Variability of presentation is cause of underdiagnose for LMS.
In our case, 56year-old male, diabetic, hypertensive presented with sudden onset of weakness of right upper limb and lower limb, hoarse ness of voice, dysphagia and difficulty in walking after clinical and radiological evaluation, he was diagnosed as LMS.

Keywords: Lateral medullary syndrome, Cerebrovascular infarction, Medulla oblongata, Ataxia, Hoarseness of voice.

Introduction
Occlusion of the posterior inferior cerebellar artery (PICA) causes the lateral medullary syndrome and infarction of lateral medulla.

The typical signs and symptoms are crossed hemisensory disturbance (ipsilateral face, contralateral body), ipsilateral Horner syndrome, and ipsilateral cerebellar signs.

It is important to diagnose lateral medullary infarction because it is associated with vertebral artery dissection in 15% to 26% of cases.

The onset was sudden on most cases. Among non-sudden onset, first signs and symptoms are usually vertigo, headache, gait ataxia or dizziness. Sensory signs (as dysphagia, hoarseness and hiccups) are tended to occur later. The most common signs and symptoms are sensory symptoms/ signs, gait ataxia, dizziness and Horner sign. Sensory signs and symptoms are the most frequent manifestation. Moderately common signs and symptoms are dysphagia, hoarseness, vertigo, nystagmus, limb ataxia, nausea, vomiting and headache.

There are sensory signs affecting trunk and extremities opposite site of lesion and face and cranial nerves on the same site of lesion. The syndrome is characterized with loss of pain and temperature sensation on the contralateral side of body and ipsilateral side of face.

Involvement of nucleus ambiguous causes dysphagia, dysarthria. If spinal trigeminal nucleus is affected, this causes absence of pain on the ipsilateral side of the face as well as absence corneal reflex. The damage to the cerebellum or the inferior cerebellar peduncle causes ataxia. Damage to the hypothalamo-spinal fibers disrupts sympathetic nervous system giving rise to Horner's syndrome. Nystagmus and vertigo are the result of involvement of vestibular nuclei. So sudden onset of severe because of this. Damage of the cranial trigeminal tract causes palatal myoclonus

Case Report
56 Years old male came to casualty with c/o fever since 3 days and difficulty in walking.

Generalised weakness, hoarseness of voice, difficulty in swallowing (more to solids) since 3 days.

1. Patient is asymptomatic 3days before, patient developed weakness of right upperlimb and lower limb since 3days which was sudden in onset, no loss of consciousness, no deviation of mouth and no slurring of speech. Patient suddenly felt difficulty in walking and later noticed difficulty in swallowing. Weakness sudden in onset, unilateral right sided, is static, not progressive, associated with tingling and numbness.
2. Pt had intermittent burning sensation of both upper limb and lower limb of the effected side.
3. Pt developed fever since 3days. Low grade, not associated with chills and rigors, relieved with medication. Fever associated with Generalised weakness.
4. Patient had decreased appetite since 5 days. Developed difficulty in swallowing (solids), taking only liquid diet since 5days.associated with hoarseness of voice.
5. Patient doing his own daily activities, no memory deficit, no irrelevant talking, excessive sleepiness present.
6. H/O past illness:
7. K/C/O hypertension since 10years.
8. K/C/O Diabetes mellitus type 2 since 10years.
9. K/C/O CAD, CABG done 2014 (on regular medication) IHD, triple vessel disease, IWMI
10. (on nitroglycerin 2.5mg ; aspirin)
11. H/O smoking since childhood (nearly 40yrs), smoker since 40yrs stopped since 2014.
12. No relevant family history.

On Examination

1. Patient is conscious , coherent, cooperative
2. BP – 170/100mmHg ;
3. PR – 62bpm
4. RR- 17/min
5. GRBS :141mg/dl
6. CVS- S1 S2 heard. No thrills. No murmurs
7. RS – trachea central in position.
8. No dyspnea, no wheeze, vesicular breath sounds.

9. P/A – scaphoid in shape. No tenderness, no palpable mass. Normal hernial orifices. No free fluid and bruits.
10. CNS – conscious with normal speech.
11. Oriented to time, place and person,
12. nystagmus present in both eyes and mild ptosis of right eye+
13. All other cranial nerves are normal
14. Bulk and tone is normal in all 4 limbs, and power is reduced on right side
15. All superficial are normal, deep tendon reflexes are exaggerated
16. Plantar are flexor and withdrawl on right and left respectively
17. Sensory system intact
18. Cerebellar signs: couldn't ellicit tandem walking,2 point discrimination lost,and dysdiadokinesia impaired
19. No meningeal signs of irritation
20. No spine tenderness
21. Ataxic gait
22. Examination of other systems: NAD
23. The patient's blood pressure, heart rate, respiratory rate, SpO2 and body temperature were 140/70 mm/Hg, 92 beats/min, 16/min, 96% and 36°C, respectively.
24. In the laboratory tests: white blood cells were 11,000, hemoglobin was 11.4, hematocrit was 37.5, mean corpuscular volume was 84.1, plasma glucose was 142, blood urea was 50 plasma creatinine was 0.9 AST, ALT, Na, K and CI were 15, 14,124, 4.1and 103 respectively.
25. Urinary sodium 126, and serum osmolality 288
26. Magnetic resonance imaging of the brain, the right side of the medulla oblongata in the brain stem at the level of the FLAIR sequence was interpreted as signaling a high view

Discussion

Lateral medullary syndrome is a rare cause of stroke. Generally, lesions are related to multiple vessel involvement, dissection, and poor collateral circulation is larger than those associated with single-vessel disease, atherothrombosis/cardiac embolism, and good collateralization.

Conflicts of Interest

All contributing authors declare no conflicts of interest.

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