

**POSTERIOR INFERIOR CEREBELLAR ARTERY SYNDROME IN A PATIENT WITH
PRIOR CEREBROVASCULAR ACCIDENT**

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INTRODUCTION

Posterior Inferior Cerebellar Artery Syndrome also known as Lateral Medullary Syndrome or Wallenberg Syndrome is a clinical neurological condition in which an infarction or stroke occurs in the lateral medulla of brain stem resulting in blockage of the intracranial portion of the vertebral artery along with posterior inferior cerebellar artery, superior middle and inferior medullary arteries.^[1] It is characterized by several symptoms due to a neurological disorder of the brain stem affecting bodily functions.

Symptoms/abnormalities	Clinical features and structures affected
Vestibulocerebellar symptoms	Vertigo, Falling towards the side of the lesion, Diplopia, and Multidirectional nystagmus (inferior cerebellar peduncle and vestibular nucleus)
Autonomic dysfunction	Ipsilateral horner's syndrome, Hiccups
Sensory Symptoms	Initially abnormal stabbing pain over the ipsilateral face, loss of pain and temperature sensation over the contralateral side of the body (spinal trigeminal nucleus involvement)
Ipsilateral bulbar muscle weakness	Hoarseness, Dysphonia, Dysphagia, Dysarthria, Decreased gag reflex (nucleus ambiguus)

CASE PRESENTATION

A 58 years old male patient presented to the General Medicine OPD with complaints of slurring of speech and weakness of the right side of his body for 20 days. The patient is a known case of Hypertension and Diabetes Mellitus since 12 years, which is being managed with medications. He has a past history of Cerebrovascular Accident (CVA) 10 years back, for which he was admitted, treated and recovered without any neurological deficits. He has insignificant family history. On examination, the patient had gait abnormality (tendency to sway towards the left side while walking) and the power of his lower limbs was reduced bilaterally.

INVESTIGATIONS

Diagnosis of the disease is usually done by assessing vestibular-related symptoms in order to determine the location of the infarct in the medulla. Head Impulse, Nystagmus, Test of Skew (HINTS) examination of oculomotor function can also be performed, along with Computed tomography scan (CT-Scan) or Diffusion-

Weighted Imaging - Magnetic Resonance Imaging (DWI-MRI) to assist in stroke detection. Standard stroke assessment must be done to rule out a concussion or head trauma. Magnetic Resonance Imaging (MRI) of the brain and Magnetic Resonance Angiogram (MRA) circle of Willis of the patient showed a multi-infarct state with bilateral thalamic infarcts.

DIFFERENTIAL DIAGNOSIS

Based on the clinical and radiological evidence the patient was diagnosed with Posterior Inferior Cerebellar Artery Syndrome (Wallenberg syndrome or Lateral medullary syndrome). Differential diagnoses include Lacunar stroke, Middle Cerebral Artery (MCA) stroke, and Subarachnoid Haemorrhage (SAH).^[1]

TREATMENT

The prime goal of treatment is to minimise symptoms and impairments, maximise independence and function, prevent complications and reduce morbidity and mortality rate.

Management of disease depends on early detection and identification of its underlying cause. Speech therapy as supportive care helps in overcoming speaking and communication issues. Feeding tube and swallow therapy aids in assisting swallowing complications. Anti-epileptics such as gabapentin help in eliminating chronic neuropathic pain associated with the syndrome. Blood thinners such as heparin or warfarin reduce and dissolve the blockage in the artery. Long term treatment involves use of antiplatelets and anticoagulants to prevent the risk of subsequent episodes of stroke. Physical Therapy and Transcranial Magnetic Stimulation (TMS) has been shown to assist in rehabilitation. Surgical removal of clots can be an option in rare cases.

The patient was managed with intermittent oxygen inhalation and Ryle's tube (RT) feeding as swallowing was impaired. Anticoagulant and Antiplatelet agents such as Aspirin 150mg OD and Clopidogrel 75mg OD, Neuroprotectives like Citicoline 500mg BD were prescribed. Atorvastatin 40mg OD at night was used for the prevention of cardiovascular risks. Proton pump inhibitor, Rabeprazole was given 20mg IV OD to prevent ulceration and gastric irritation associated with aspirin. Analgesics such as Paracetamol 162.5mg and Tramadol 18.75 mg combination BD and Diclofenac 25mg IM BD was used for symptomatic relief of pain. The treatment was supplemented with supportive physiotherapy to achieve optimal health.

Table No.01 Medication Chart.

Proprietary Name	Generic Name	Dose	Dose/route	Frequency
Veloz	Rabeprazole	20 mg	IV	OD B/F
Ecosprin	Aspirin	150 mg	PO	OD
Atorva	Atorvastatin	40 mg	PO	OD HS
Clopivas	Clopidogrel	75 mg	PO	OD
Strocit	Citicoline	500 mg	PO	BD
Ultracet semi	Paracetamol and Tramadol	162.5mg and 18.75 mg	PO	BD
Inac	Diclofenac	25 mg IM	IM	BD
RT feeding with RT aspiration		200 ml	PO	Q3h/ Q4h
Physiotherapy		-	-	OD

OUTCOME AND FOLLOW UP

Wallenberg Syndrome has no specific outcome measures. Since the condition is most often due to the result of a stroke, outcomes are measured objectively to determine baseline measures and progression with treatment and interventions.

The patient responded well with the treatment and symptoms began to subside gradually. He was discharged after 15 days of hospitalization, with residual gait instability. The patient was prescribed medications and advised to follow up on regular basis.

DISCUSSION

Wallenberg syndrome is a rare syndrome having implications on one's activity of self-caring. Due to its varied etiological factors, causing stroke in one of the arteries of the brainstem, it is associated with damage in the lateral medulla of the brainstem which may cause damage to the cerebellum affecting the prognosis of the patient.^[1] The characteristic presentation of the condition is the crossed sensory symptoms (e.g., left side of the face and right side of the body) and Horner syndrome on

the ipsilateral side of the lesion.^[2] The most common cause of central Horner syndrome is Wallenberg syndrome which is present in up to 33% of these cases.^[3] This condition can be diagnosed clinically and confirmed with CT or DWI-MRI scan.^[1] The severity of the presentation, need for emergency intervention and subsequent monitoring will depend on the cause and size of the area affected in the brainstem. Affected individuals should receive urgent neuroimaging to exclude alternate diagnoses and contraindications for acute stroke therapies. Treatment is mainly symptomatic. Most cases require feeding tubes and will improve with speech/swallowing therapy.^[4]

CONCLUSION

Being a rare syndrome, the patient was managed in an optimal way. This case illustrates that with proper symptomatic treatment, clinical monitoring and effective post-stroke care, the prognosis for patient recovery can be positive. Most patients with this syndrome have minimal deficits at six months. Over 85% of these patients develop functional independence with

ambulation within a year following treatment; few might be left with significant neurological disabilities for years.

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