ORIGINAL RESEARCH PAPER

Volume-7 | Issue-11 | November-2018 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

A CASE OF WALLENBURG'S SYNDROME WITH COMORBIDITIES

General Medicine

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ABSTRACT

Lateral medullary syndrome or Wallenburg's syndrome is a neurological disorder causing a range of symptoms due to ischemia in the lateral part of the medulla oblongata in the brainstem. The ischemia is a result of a blockage in the posterior inferior cerebellar artery or one of its branches. Lateral medullary syndrome is also called Wallenberg syndrome, posterior inferior cerebellar artery syndrome, PICA syndrome, vertebral artery syndrome, and Wallenberg's syndrome. Here I present a classical case of Wallenburg's syndrome which had all the features and responded to the treatment.

KEYWORDS

INTRODUCTION:-

This syndrome is characterized by sensory deficits that affect the trunk and extremities contralaterally (opposite to the lesion), and sensory deficits of the face and cranial nerves ipsilaterally (same side as the lesion). Specifically a loss of pain and temperature sensation if the lateral spinothalamic tract is involved. The cross body finding is the chief symptom from which a diagnosis can be made. Patients often have difficulty walking or maintaining balance (ataxia), or difference in temperature of an object based on which side of the body the object of varying temperature is touching. Some patients may walk with a slant or suffer from skew deviation and illusions of room tilt. The nystagmus is commonly associated with vertigo spells. These vertigo spells can result in falling, caused from the involvement of the region of Deiters' nucleus.[1]

Common symptoms with lateral medullary syndrome may include difficulty swallowing, or dysphagia. This can be caused by the involvement of the nucleus ambiguus, as it supplies the vagus and glossopharyngeal nerves. Slurred speech (dysarthria), and disordered vocal quality (dysphonia) are also common. The damage to the cerebellum or the inferior cerebellar peduncle can cause ataxia. Damage to the hypothalamospinal fibers disrupts sympathetic nervous system relay and gives symptoms that are similar to the symptoms caused by Horner syndrome – such as miosis, anhidrosis and partial ptosis. Palatal myoclonus, the twitching of the muscles of the mouth, may be observed due to disruption of the central tegmental tract. Other symptoms include: hoarseness, nausea, vomiting, a decrease in sweating, problems with body temperature sensation, dizziness, difficulty walking, and difficulty maintaining balance. Lateral medullary syndrome can also cause bradycardia, a slow heart rate, and increases or decreases in the patients average blood pressure.[2]

Case Report:-

The patient was a 47 yrs old married male presented in an altered state of consiousness and per the history given by the relatives, the presenting complaints were-

Fever for the past 02 days
Altered sensorium for the past 2 days
Inability to speak for 02 days
Inability to eat for 02 days

The patient presented with a history of fever for the past 02 days before admission which was moderate grade, continuous and associated with chills. There was no diurnal variation of the fever. There was an associated history of cough which was mildly productive in nature.

Patient also had altered sensorium for the past 02 days prior to admission which started all of a sudden with weakness of all the 4 limbs. Later on, the patient developed nasal regurgitation to liquids, had inability to speak and inability to eat.

History of urinary incontinence was present. Patient also had history of vomiting whenever he tried to eat.

There was no history of involuntary movements.
There was no history pertaining to the sensory system. There was also no history of dyspnoea or palpitations. There was nothing significant in the past history. There was no family history diabetes mellitus, hypertension, bronchial asthma, coronary artery disease. The patient was a vegetarian by diet, no addictions, but had incontinence of urine.

On general physical examination, the patient was conscious but drowsy. The general condition of the patient was not satisfactory. The patient was afebrile. The patient had a pulse rate of 62/min, regular, normal volume, normal character, no radiofemoral delay, peripheral pulses were well felt. The respiratory rate was 24/min, abdominothoracic. The BP was 130/70 mmHg in the right arm, supine position. There was no pallor, icterus, cyanosis, clubbing, lymphadenopathy, edema. The JVP was not raised. On CNS examination, the patient was drowsy, responding by following simple verbal commands.

Pupilis Rt Lt
2mm 5mm
Drooping of right eye lid was present.
Left sided VI CN palsy was present.
Right sided IX, X CN palsy was present.
DTRs :- Biceps, triceps, knee jerk on the right were exaggerated.
Plantars were bilateral flexor.
Reflexes on the left side were normal.
Sensory system could not be tested.

Weakness all 4 limbs were present. Power was grade IV in all the 4 limbs.

There was no neck stiffness present. There was decreased sweating on the right side of the face. GCS was E4M4V1 = 9. On other systems examination, crepitations were heard on the right side more than the left side. Bilateral throat conducted sounds were present. Cardiovascular system and per abdomen examination were normal.

On Investigation :-

Viral markers : Negative
Dengue Serology : Negative
Typhi dot IgG, IgM : Negative
Malarial Ag : Negative
Blood Culture & Sensitivity : No growth
Sputum for AFB : Negative

Sputum Culture & Sensitivity showed the growth of MDR K. pneumoniae which was sensitive to colistin, imipenem, meropenem, Polymyxin-B, tigecycline.
BT: 3 min, CT : 6 min

Day	1	2	4	6	8	10	12	15	17
CBC	12.6	12.1	11.9	12.2	11.9	11	1.06		7.9
Hb									
TLC	17,130	13,770		8,900	10,500		23,600		5,370
Polys	83	76		81	97		75		60
Urine Ex	Normal			4-6 Pus	cells				
Sr Creat	1.1		0.8			1.3	0.9	0.8	0.8
Bl Urea	31		28			41	33	19	24
Sr AST	22		15					40	
Sr ALT	16	13	13					29	

ABG
PH : 7.42 RBS :- 120 mg%
PCO2:- 34 mmHg ECG :- Normal
Po2 :- 67 mmHg CXR :- NHO right midzone and left lower

zone
HCO3:- 22.3
MRI Brain : Subacute infarct in the right posterolateral medulla.
USG Abd : Normal

The patient was found to have an infarct in the right posterolateral medulla which was responsible for the neurological clinical features of the patient. He was also found to have right sided aspiration pneumonia. He also had urinary tract infection. Treatment was started for the same. Patient was started on-

- Inj Piperacillin+Tazobactam 4.5 IV TDS
- Inj Levofloxacin 500 mg IV OD
- Inj Pantoprazole 40 mg iv OD
- Inj Emeset 4 mg iv BD
- Inj Mannitol 100 ml iv TDS
- Tab Atorvastatin 10mg HS
- Tab Dolo sos
- RT insertion was done
- Foleys cathetermination was done
- Position change every 2 hourly was advised. After the availability of the sputum culture and sensitivity report which showed MDR K.pneumoniae, Inj.Zosyn was stopped and Inj Imipenum was started. The patient responded to the treatment and the sepsis slowly recovered and the patient Wallenburg Syndrome also improved immensely with partial recovery of the CN palsies and the weakness in the limbs also improved. Over the next few days as the patient became better and started oral feeds, and foley's catheter removed, the patient was finally discharged from the hospital in good general condition.

DISCUSSION:-

It is the clinical manifestation resulting from occlusion of the posterior inferior cerebellar artery (PICA) or one of its branches or of the vertebral artery, in which the lateral part of the medulla oblongata infarcts, resulting in a typical pattern. The most commonly affected artery is the vertebral artery, followed by the PICA, superior middle and inferior medullary arteries. Since lateral medullary syndrome is often caused by a stroke, diagnosis is time dependent. Diagnosis is usually done by assessing vestibular-related symptoms in order to determine where in the medulla that the infarction has occurred. Head Impulsive Nystagmus Test of Skew (HINTS) examination of oculomotor function is often performed, along with computed tomography (CT) or magnetic resonance imaging (MRI) to assist in stroke detection. Standard stroke assessment must be done to rule out a concussion or other head trauma. Treatment for lateral medullary syndrome is dependent on how quickly is it identified. Treatment for lateral medullary syndrome involves focusing on relief of symptoms and active rehabilitation to help patients return to their daily activities. Many patients undergo speech therapy. Depressed mood and withdrawal from society can be seen in patients following the initial onslaught of symptoms.

In more severe cases, a feeding tube may need to be inserted through the mouth or a gastrostomy may be necessary if swallowing is impaired. In some cases, medication may be used to reduce or eliminate residual pain. Some studies have reported success in mitigating the chronic neuropathic pain associated with the syndrome with anti-epileptics such as gabapentin. Long term treatment generally involves the use of antiplatelets like aspirin or clopidogrel and statin regimen for the rest of their lives in order to minimize the risk of another stroke. Warfarin is used if atrial fibrillation is present. Other medications may be necessary in order to suppress high blood pressure and risk factors associated with strokes. A blood thinner may be prescribed to a patient in order to break up the infarction and reestablish blood flow and to try to prevent future infarctions.[3]

CONCLUSION:-

In order to diagnose Wallenburg's syndrome, one has to be vigilant for it's varied presentations. Such patients can have many complications like in this case the patient had aspiration pneumonia and UTI which ultimately responded to the treatment.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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