A Case Report on Lateral Medullary Syndrome

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***Abstract*— Wallenberg syndrome, also known as lateral medullary syndrome, is a relatively uncommon type of brainstem stroke resulting from an acute ischemic event that affects the lateral medulla region. This condition typically arises from the blockage of the intracranial portion of the vertebral artery or the posterior inferior cerebellar artery. The affected area includes the lateral medulla behind the inferior olivary nucleus and is usually seen in elderly patients with vascular risk factors. Individuals with Wallenberg syndrome present a distinct constellation of clinical symptoms, such as ipsilateral ataxia, Horner's syndrome, vertigo, dysphagia, dysarthria, and contralateral hemiparesis. This report discusses a 53-year-old female patient with Wallenberg syndrome who presented with persistent dizziness, vomiting, and loose stools over the past three to four days, along with a history of cerebrovascular accident (CVA). The clinical assessment suggested lateral medullary syndrome (LMS), and the diagnosis can be established based on clinical findings, which may be further confirmed through brain imaging using CT or MRI. Management typically consists of appropriate symptomatic and supportive treatment. The course of therapy adhered to hospital guidelines and benefited from the attending physician's expertise. Addressing the functional constraints associated with the syndrome proved effective in meeting her essential life needs.**

***keywords*— Ataxia, lateral medullary syndrome, hemiparesis, Posterior inferior cerebellar artery (PICA), Magnetic resonance imaging (MRI), Computed tomography (CT)**

I. INTRODUCTION

Gaspard Vieusseux initially described Wallenberg syndrome in 1808. Adolf Wallenberg provided a more thorough description of this illness in 1895, identifying it as infarction of the lateral medulla oblongata after vertebral artery (VA) or posterior inferior cerebellar artery (PICA) obstruction. Other names for Wallenberg syndrome include posterior inferior cerebellar artery syndrome and lateral medullary syndrome. The injury to the lateral portion of the medulla posterior to the inferior olivary nucleus causes various symptoms linked to this neurological condition. It is the most prevalent ischemic stroke condition with posterior circulation [1]. The aetiology of the lateral medullary syndrome includes atherosclerosis, Hypertension, Cardiogenic embolism, Dissection of vertebral arteries, Hypoplastic Vertebral disease, small vessel disease, and Vertebrobasilar dolichoectasia. The disease of the vestibular nuclei or vestibular–cerebellar connections may cause vertigo, nausea, vomiting, and symptoms such as skew deviation, diplopia, and severe gait ataxia [2].

II. CASE PRESENTATION

A 53-year-old female patient was admitted to the hospital after complaining of dizziness, vomiting, and loose stools for three to four days prior. These symptoms, together with a history of right lateral medullary infarction and an elevated blood pressure of 180/110 mmHg, constituted the foundation of her treatment plan. To reduce the danger of blood clot formation, the patient was given 0.4 mg of low molecular weight Heparin (LMWH), Tab. Telma 40 mg orally, and enteral feeding was eased by the placement of a Ryle tube, while urine drainage was accomplished using a Foley catheter. She was also given an IV cannula for Normal Saline at a rate of 30 ml/hour. Symptom-specific therapies included the administration of 16 mg of Inj. Vertin for dizziness and 40 mg of Inj. Pantop and 4 mg of Inj. Emeset for vomiting to stabilize her vitals upon admission to the intensive care unit. Over the last two months, the patient has had right-side hemiplegia, which is defined as paralysis on the right side of the body and a loss of vision in her left eye. She also has a medical history that includes a cerebrovascular accident (CVA), diabetes, and hypertension (HTN). This medical history was vital in determining the diagnosis and treatment approach. Upon clinical examination, the patient responded to vocal directions consciously and appropriately while retaining typical vital signs and behavior. Furthermore, the Glasgow Coma Scale (GCS) examination of (E4, M6, and V5) showed normal cognitive function. However, nystagmus was detected during the examination when the patient's attention was directed to the left. This evaluation gave vital information about the patient's neurological condition [3]. The physician has ordered a series of diagnostic tests to evaluate the patient's health further, including an MRI, ECG, 2D echocardiography, complete blood count (CBC), renal function tests, vitamin B12 assay, Anti-Nuclear Antibody (ANA) test, and serum electrolyte measurements. These carefully selected tests will provide vital information to aid the patient's diagnosis and treatment plan, establishing confidence in the accuracy of the diagnosis Table 1.

Table 1: lab investigations of abnormal values.

COMPLETE BLOOD PICTURE	
RBC	4.2Millions/cmm
MCV	67%
MCVC	42%
SERUM ELECTROLYTES	
Potassium(K^+)	3.3 mmol/L

The hematological study indicated abnormal values for the red blood cell count (RBC), mean corpuscular volume (MCV), and mean corpuscular hemoglobin concentration (MCHC). In addition, aberrant potassium levels were seen in the serum electrolytes, but the patient tested negative for antinuclear antibodies (ANA) and viral indicators. An MRI scan revealed ataxic hemiparesis, an acute infarction in the anterior lateral portion of the right pons, and mild bilateral ethmoidal sinusitis. **Fig. 1** and **Fig. 2**

Figure 1, the **left image** shows a hyperintense area in the lateral medulla on T2-weighted imaging, indicative of acute infarction, corresponding to the **right image** the T1-weighted image reveals a hypointense region due to oedema associated with the lateral medullary infarct.

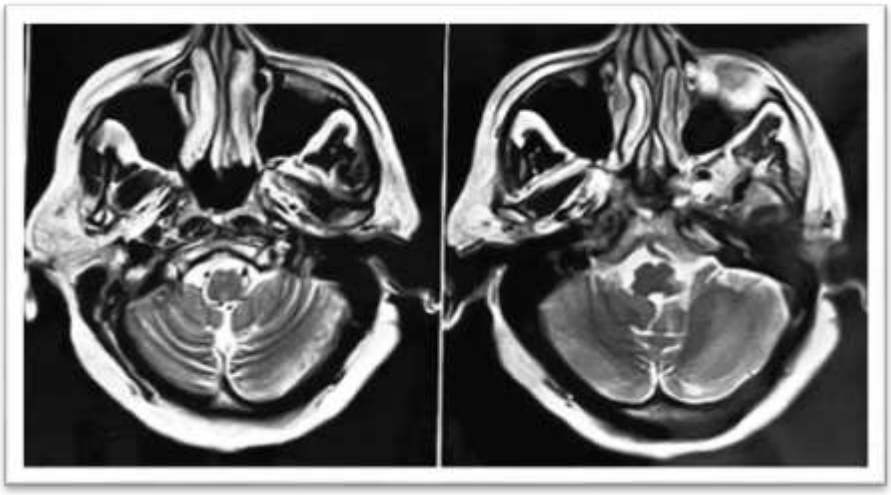
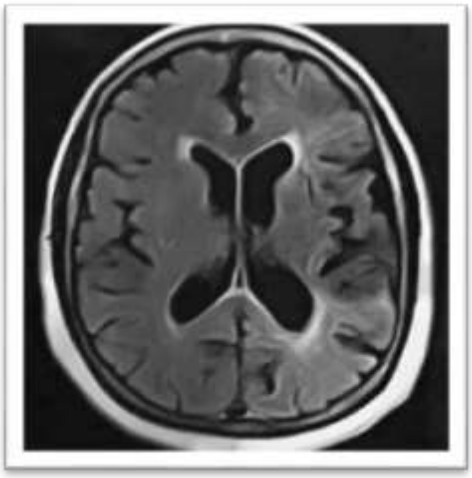


Figure 2, the MRI image demonstrates a hyperintense lesion on T2 weighted imaging within the region of the internal capsule, likely extending into the pons and consistent with a lacunar infarct. The electrocardiogram shows a normal sinus rhythm. Following



laboratory tests, the doctor diagnosed a left lateral medullary infarction with ataxic hemiparesis.

III. TREATMENT ADVISED

The patient's comprehensive treatment plan includes a carefully curated list of medications to address various health concerns. To prevent blood clots, the patient was administered a combination of Clopidogrel and Aspirin 150/75mg PO/OD. In addition, Atorvastatin 40mg/PO/OD was given to help reduce the patient's cholesterol levels. To treat uncontrolled blood pressure, Nifedipine XL-10mg/PO/OD was administered to relax the blood vessels. In addition, the patient was given Beta-Histine 16mg/PO/BD to reduce vertigo symptoms. Pantoprazole 40mg IV/OD was administered to prevent Aspirin-induced stomach irritation and ulceration. Furthermore, Ondansetron 4mg/PO/BD was used to treat vomiting episodes, and Rejunex Forte 1Amp/IV/OD was used to aid in general recovery and brain health. To treat low serum potassium levels, the patient was given Inj. KCL IV/OD was initially

used, followed by syrup KCL 15ml/PO/TID to stabilise potassium levels. Following four days of treatment, the patient's symptoms improved significantly, resulting in her discharge with appropriate guidance and medication, giving the audience hope for a possible recovery.

IV. DISCUSSION

Gaspard Vieusseux first recorded Wallenberg Syndrome in 1808, and Adolf Wallenberg went into great depth about it in 1895. This syndrome results from a lateral medulla oblongata infarction caused by a blockage in the vertebral artery (VA) or posterior inferior cerebellar artery (PICA) [4]. It is also known as lateral medullary syndrome and is characterised by various symptoms such as vertigo, nausea, vomiting, and neurological impairments caused by lateral medulla injury. Given its prevalence as the most common ischemic stroke disorder affecting the posterior circulation, a thorough understanding of its etiology is critical for efficient treatment. This case report describes the clinical manifestations and therapeutic management of a 53-year-old female patient with Wallenberg Syndrome, which is caused by infarction of the lateral medulla oblongata due to occlusion of the vertebral artery (VA) or posterior inferior cerebellar artery (PICA). The patient, who had a history of hypertension and a previous cerebrovascular accident, displayed symptoms such as dizziness, vomiting, right-sided hemiplegia, and blurred vision, all of which are symptomatic of the syndrome. Magnetic resonance imaging (MRI) indicated an acute infarction in the right pons, with laboratory results indicating hypokalemia and abnormal hematological indices. Standard electrocardiogram (ECG) readings and negative antinuclear antibody (ANA) tests helped eliminate other causes. The comprehensive treatment regimen included anticoagulants (Clopidogrel and Aspirin). Combining this with usual practice in the hospital, such a pattern optimises medical needs confined to the patient. Among patients receiving clopidogrel following acute infraction, concomitant therapy with proton pump inhibitors other than pantoprazole was associated with a loss of the beneficial effects of clopidogrel and increased risk of reinfarction [5]. Nifedipine, an antihypertensive agent, is administered to patients as an early diagnosis of high blood pressure. Additionally, this medication serves to mitigate the risk of recurrent stroke. statins such as Atorvastatin were started as the triglyceride level was elevated. In patients with hyperlipidemia, treatment with HMG CoA reductase inhibitors decreases the risk of stroke when compared with other agents (fibrates and resins). Therefore, it seems plausible that the protective effects of statins are not mediated by cholesterol lowering but by anti-atherothrombotic properties and physiotherapy further helped the patient to get better. symptom-specific pharmacotherapy, such as Beta-Histine for vertigo, is a vasodilator available in Europe, Central and South America, and Canada it is reported to act by improving microvascular circulation in the stria vascularis of the cochlea or by inhibiting vestibular nuclei activity and Pantoprazole for gastrointestinal complications prevention, were used. Potassium supplements were used to correct electrolyte abnormalities. The patient's subsequent significant improvement and stabilisation demonstrate the usefulness of this comprehensive strategy in managing Wallenberg Syndrome, emphasizing the importance of addressing both ischemia episodes and concomitant metabolic dysregulation for optimal recovery.

V. CONCLUSION

The case report highlights the complexities of Wallenberg Syndrome, caused by a lateral medulla oblongata infarction due to vertebral or posterior inferior cerebellar artery occlusion. A complete therapy plan that included anticoagulants, antihypertensives, statins, symptom-specific medicines, and electrolyte correction showed significant success in stabilizing the patient and reducing symptoms. This instance demonstrates the importance of a comprehensive strategy for improving patient outcomes.

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