# LATERAL PONTINE STROKE SYNDROME PRESENTING AS A REPEAT STROKE: A CASE REPORT

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**ABSTRACT**

***Introduction:*** **The pons is the largest and a vital part of the brainstem between the midbrain and medulla oblongata. Due to the versatility of its function, various stroke syndromes have been described as a result of pontine infarction. Notable among these is the lateral pontine syndrome, which was first described in 1922 as a stroke when the anterior inferior cerebellar artery becomes occluded, leading to an infarction in the lateral inferior part of the pons, middle cerebellar peduncle, and the floccular region, resulting in ipsilateral limb ataxia, facial weakness, hearing loss, facial anaesthesia, contralateral hemihypoaesthesia, vertigo, and nystagmus. This case report aims to contribute to the limited existing literature on a rare brainstem stroke syndrome, which to the best of our knowledge, has not been reported in Africa**

***Case presentation:*** **The patient was a 66-year-old stroke survivor who developed sudden onset vertigo, dysarthria, right limb ataxia, facial weakness, and hearing impairment. Brain MRI revealed an acute infarct in the right lateral part of the lower pons.**

***Conclusion:*** **Secondary stroke prevention is vital for survivors. An MRI should be chosen if available when a brainstem stroke is suspected, especially in low-resource settings where patients can afford only one imaging session.**

## INTRODUCTION

Lateral Pontine Syndrome, also known as Marie Foix-Alajouanine Syndrome, is a type of brainstem stroke that occurs when the anterior inferior cerebellar artery becomes occluded, resulting in an infarction in the lateral inferior part of the pons, middle cerebellar peduncle, and the floccular region.[1] This affects the nuclei of CN V, VII, and VIII, spinothalamic tract, and cerebellar tracts, resulting in symptoms such as ipsilateral limb ataxia, ipsilateral facial palsy, ipsilateral hearing loss, vertigo, and nystagmus.[2,3]

The aetiopathogenic mechanism of pontine infarction has been described in some literature to include basilar artery branch disease (BABD), small-artery disease (SAD), and large-artery disease (LAD) of vertebro-basilar arteries.[4,5] Many pontine stroke syndromes have been described, depending on the artery or group of arteries occluded. These include Brissaud-Sicard syndrome (pontine perforating arteries), Facial colliculus syndrome (paramedian branches of basilar artery), Gasperini syndrome (long circumferential branch of the anterior inferior cerebellar artery (AICA), Gellé syndrome (pontine perforating arteries), Foville syndrome (Paramedian branches of basilar artery), Lateral pontine syndrome, also known as Marie-Foix syndrome (Anterior inferior cerebellar artery), Locked-In syndrome (Proximal and middle segment of basilar artery), Raymond syndrome (Paramedian branches of the basilar artery), Upper dorsal pontine syndrome also known as Raymond-Cestan syndrome(long circumferential branch of basilar artery), and Ventral pontine syndrome also known as Millard-Gubler syndrome (short circumferential branches of the basilar artery).[2]

To the best of our knowledge, there is no case report on the lateral pontine syndrome in Africa. However, some cases have been reported in other parts of the globe, either as a lateral pontine syndrome or a spectrum of anterior inferior cerebellar artery infarction.[1,6-8] We, therefore, report the case of a middle-aged man with clinical and radiological features in keeping with the lateral pontine syndrome following a partial recovery from a previous stroke. This report aims to underscore the importance of secondary stroke prevention and to highlight the significance of brain MRI when a

brainstem stroke is suspected. Possibly, some brainstem stroke syndromes described as rare may be identified, particularly in our setting, where most stroke patients typically undergo only a single imaging study, usually a brain CT scan, which would not have detected the infarction in this case. To the best of our knowledge, after searching the literature, lateral pontine stroke syndrome has not been reported from Africa.

## CASE PRESENTATION

We report the case of a 66-year-old man with systemic arterial hypertension and Type 2 diabetes mellitus of 8 years and a prior history of supratentorial ischaemic stroke in the right middle cerebral artery (MCA) territory, with mild residual weakness on the left side of one year. He presented to our facility with a sudden onset of vertigo and subsequently noticed dysarthria and postural imbalance of eight days duration. He had difficulty closing the right eye with ipsilateral facial weakness and numbness. There was accompanying dysarthria, worsening left-sided body weakness, and postural imbalance with the tendency to fall to the right on an attempt to walk. He also reported transient dysphagia and hearing impairment on the affected side. No loss of vision, vomiting, headache, loss of consciousness, or seizure. There was no history of falls or any trauma. There was no preceding fever, earache, ear discharge, skin rashes, or any symptom suggestive of upper respiratory tract infection. No neck pain or stiffness, no photophobia. He neither drinks alcohol nor smokes cigarettes.

His medications before presentation included antihypertensives, antidiabetics, a statin, and an antiplatelet

On examination, he was conscious. His pulse was 72 per minute, regular with normal volume, and BP was 150/90 mm of Hg. He had normal cognitive function and absent signs of meningeal irritation. The pupils were normal in shape and size and reactive both directly and consensually. The corneal reflex was impaired on the right, normal on the left, and gag/swallowing reflexes were intact. He had right facial nerve palsy, lower motor neuron type (Figure 1), hearing loss, and bidirectional nystagmus. He was dysarthric, and power was 4- on the left upper limb and 4+ on the lower limb with hyperreflexia. The power on the right limbs was normal with brisk reflexes. He had hypoesthesia on the right side of his

**Figure 1:** Right facial nerve palsy, lower motor neurone type

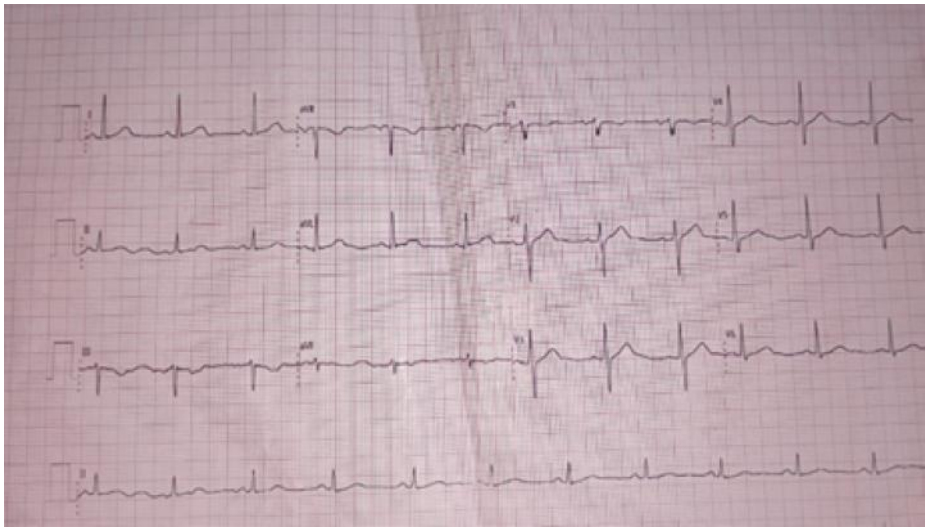

**Figure 2:** Normal ECG

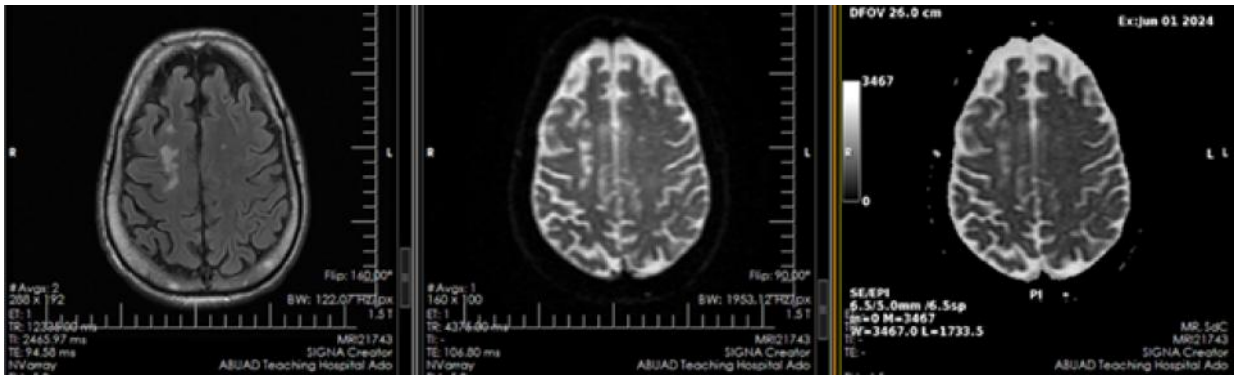


**Figure 3A:** Hyperintensities in the right centrum semi-ovale seen on FLAIR, DWI, and ADC, no diffusion restriction

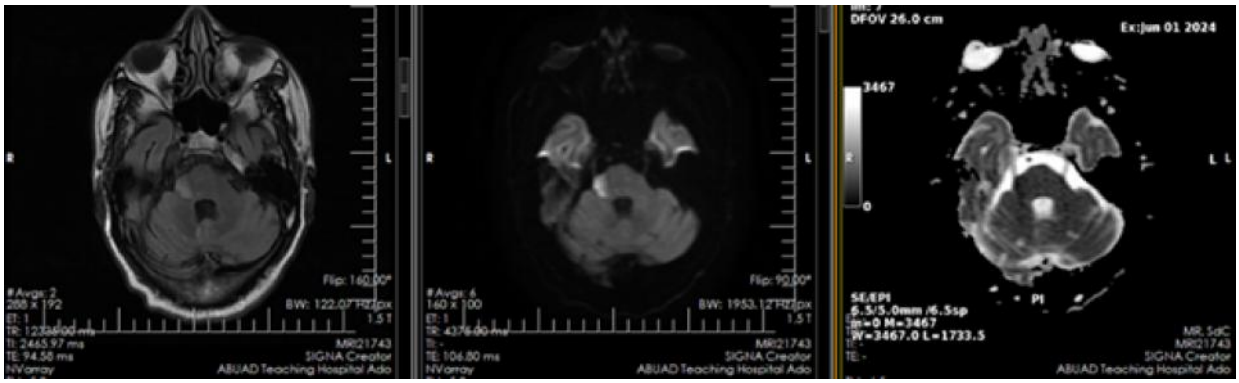


**Figure 3B:** Hyperintensity in the right mid-lateral aspect of the pons on FLAIR and DWI, with diffusion restriction on ADC

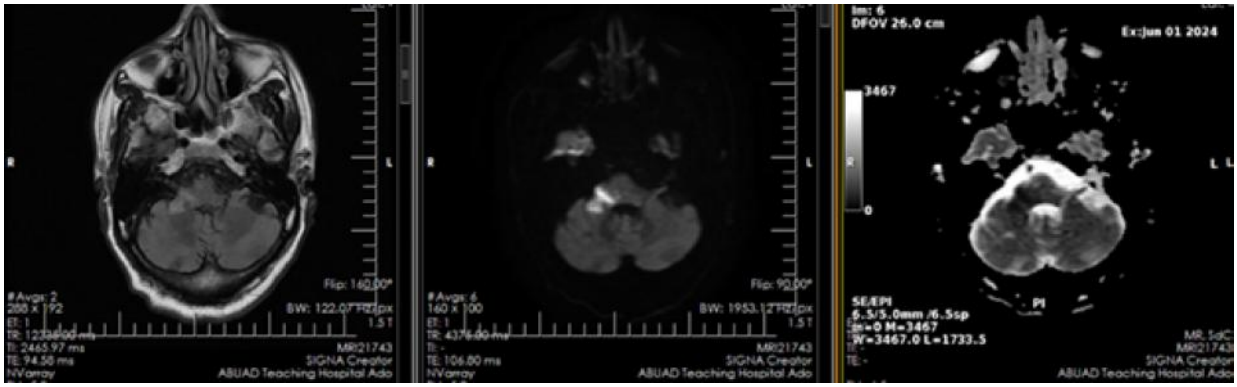


**Figure 3C:** Hyperintensity in the right inferior lateral aspect of the pons and middle cerebellar peduncle on FLAIR and DWI, with diffusion restriction on ADC

face and the left side of his body. He had right upper and lower limb ataxia. No loss of joint position sense. The random blood sugar level at presentation was 11.5mmol/L, and the HbA1c level was 7.9%. The total cholesterol was3.62mmol/L, triglyceride - 0.91mmol/L, HDL – 1.16mmol/L, and LDL – 2.05mmol/L. The full blood count, renal function test, and ECG were normal.

Brain MRI shows old infarcts – (hyperintensities in the right centrum semi-ovale on FLAIR, DWI, and ADC sequences, no diffusion restriction) (Figure 3A); hyperintensity in the right mid-lateral aspect of the pons on FLAIR and DWI, with diffusion restriction on ADC (Figure 3B), hyperintensity in the right inferior lateral aspect of the pons and middle cerebellar peduncle on FLAIR and DWI, with diffusion restriction on ADC. (Figure 3C)

## DISCUSSION

The diagnosis of pontine stroke syndrome in this patient was based on history taking and neurological examination. Our patient had risk factors for atherosclerosis, including hypertension, diabetes, and dyslipidemia. The patient's calculated ASCVD score was 7.5% with a 10-year risk of 73.8%, putting him at

a very high risk of future CVD. The lateral pontine syndrome is characterized by contralateral hemihypoaesthesia and loss of discriminative sensation with ipsilateral Horner's syndrome, facial hemianesthesia, ataxia, ipsilateral lower motor neuron–type facial paresis, deafness, and vertigo.[1,9] All the above clinical features but Horner's syndrome, were present in this index patient. The patient's contralateral hemihypoaethesia is due to the lesion involving the spinothalamic tract, while facial hemianaesthesia is due to the involvement of the main sensory nucleus of the trigeminal nerve. The ipsilateral facial weakness results from facial nerve involvement, which emerges from the ventrolateral part of the pons. The four cranial nerve nuclei in the pons are trigeminal, abducens, facial, and vestibulocochlear. However, based on the topographical arrangement of the cranial nerve nuclei in the pons, in which the sixth cranial nerve nucleus is in the midline (others are lateral), the patient did not have lateral gaze palsy. Our patient had hearing loss, which could be encountered in lateral pontine syndrome due to the involvement of the cochlear nuclei in the lateral inferior part of the pons as it joins the medulla. A case of lateral pontine syndrome without hearing loss was reported a few years ago,[5] it is worth noting that the incidence of hearing loss is highly variable, with reports ranging from 30% to 100% of patients.[10] Our patient had ataxia due to the involvement of the middle cerebellar peduncle at the lower pons. Horner's syndrome is also one of the expected clinical features of lateral pontine syndrome, although it was not seen in our patient. This is because the clinical and radiological features of our patient were more suggestive of AICA occlusion leading to inferior pontine stroke syndrome, where Horner's syndrome is not typical.[7] Lastly, the limb weakness reported in our patient could be from the stroke he had a year before this ictus. However, some patients with lateral pontine stroke syndrome may have contralateral hemiparesis if the corticospinal tract is involved.[4,7,11]

Brain MRI was delayed due to financial constraints, and he was managed expectantly in the medical ward until the test revealed the diagnosis as presented above. It is important to choose brain MRI as the imaging modality when evaluating patients who have a brainstem stroke in resource-poor settings to avoid the financial stress of paying twice, as the conventional non-contrast brain CT for stroke in general may be unrevealing when the lesion is in the posterior cranial fossa.[12]

In conclusion, the case presented depicts clinical and neuroradiological features of the right lateral pontine stroke syndrome, which occurred about a year after a supratentorial ischaemic stroke in the right MCA territory. This underscores the importance of secondary stroke prevention and the preference for brain MRI when patients have features of brainstem stroke syndrome.

## REFERENCES


1. **Regmi PR,** Amatya I. A rare case of lateral pontine syndrome-Marie Foix syndrome. Journal of Patan Academy of Health Sciences. 2021 Apr 29;8(1):69–72.
2. **Gowda SN,** Munakomi S, De Jesus O. Brainstem Stroke. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 [cited 2024 Jun 11]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK560896/
3. **Balami JS,** Chen RL, Buchan AM. Stroke syndromes and clinical management. QJM. 2013 Jul;106(7):607–615.
4. **Huang J,** Qiu Z, Zhou P, *et al.* Topographic location of unisolated pontine infarction. BMC Neurol. 2019 Aug 5;19(1):186.
5. **Kumral E,** Bayülkem G, Evyapan D. Clinical spectrum of pontine infarction. J Neurol. 2002 Dec 1;249(12):1659–1670.
6. **Yoshioka T,** Tokumitsu S, Goto K, Kuroiwa S, Okajima T. [Lateral inferior pontine syndrome: a case report]. Rinsho Shinkeigaku. 1989 Jul;29(7):933–935.
7. **Amarenco P.** Anterior Inferior Cerebellar Artery Territory Infarcts: Mechanisms and Clinical Features. Arch Neurol. 1993 Feb 1;50(2):154.
8. **Kumral E,** Kisabay A, Ataç C. Lesion patterns and etiology of ischemia in the anterior inferior cerebellar artery territory involvement: a clinical - diffusion weighted - MRI study. Eur J Neurol. 2006 Apr;13(4):395–401.
9. **Cuoco J,** Hitscherich K, Hoehmann C. Brainstem Vascular Syndromes: A Practical Guide for Medical Students. Edorium Journal of Neurology. 2016 Sep 12;3:4–16.
10. **Chen MM,** Chen SR, Diaz-Marchan P, *et al.* Anterior Inferior Cerebellar Artery Strokes Based on Variant Vascular Anatomy of the Posterior Circulation: Clinical Deficits and Imaging Territories. J Stroke Cerebrovasc Dis. 2018 Apr;27(4):e59–64.
11. **Ortiz de Mendivil A,** Alcalá-Galiano A, Ochoa M, *et al.* Brainstem stroke: anatomy, clinical and radiological findings. Semin Ultrasound CT MR. 2013 Apr;34(2):131–141.
12. **Hwang DY,** Silva GS, Furie KL, Greer DM. Comparative Sensitivity of Computed Tomography vs. Magnetic Resonance Imaging for Detecting Acute Posterior Fossa Infarct. J Emerg Med. 2012 May;42(5):559–565.