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Pontine Hemorrhage presenting with Millard Gubler Syndrome with cerebellar ataxia in a 52-year-old male Nigerian hypertensive.

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ABSTRACT

Millard-Gubler syndrome (MGS) is a brainstem syndrome characterized by ipsilateral sixth and seventh cranial nerve palsies and contralateral hemiparesis. Pontine stroke is the commonest cause of MGS even though other non-vascular etiologies have been described in the literature. MGS resulting from pontine infarction is associated with relatively good prognosis, even though the overall prognosis is influenced by other factors.

We report a case of 52-year-old hypertensive with both clinical and imaging evidence of Millard -Gubler syndrome with cerebellar ataxia following a pontine hemorrhage. He managed conservatively and was discharged after about three weeks on admission. He has remained clinically stable since after discharge.

List of abbreviations:

MGS Millard –Gubler syndrome
ICH Intracerebral hemorrhage
MRI Magnetic Resonance Imaging
HIV Human immunodeficiency virus

INTRODUCTION

Millard -Gubler Syndrome (MGS) is a rare type of brainstem syndrome specifically involving ventral pontine area, affecting the 6th and 7th cranial nerves as well as the corticospinal tracts and was first described in 1958(1). The core features of MGS consist of ipsilateral lateral rectus palsy, ipsilateral facial palsy, and contralateral hemiparesis of the upper and lower limbs. Generally, stroke is the common cause of this syndrome, even though other non-vascular etiologies have been profusely documented in the literature (1–3)

Brainstem stroke is the most lethal form of all strokes, and globally contributes significantly to a significant cause of mortality and morbidity(4,5). Although ischemic accounts for the majority of the vascular events in the brain stem, pons appear to be the commonest site of spontaneous brainstem hemorrhage (6). Spontaneous brainstem hemorrhage constitutes about 1/10th of all intracerebral hemorrhages with an annual incidence of roughly 2-4/100,000 per year (6–8) and pons is most common site of bleed (60-80%) (4,9) and individuals in their 4th and 5th decades are mostly involved with more males affected. Vascular lesion to the brain stem disrupts a plethora of physiological functions such as respiration, cardiac rhythm, blood pressure control, consciousness, and the sleep-wake cycle.

We report a case of a middle-aged man, known hypertensive, who presented to us with clinical features of Millard Gubler syndrome following anterior pontine hemorrhage and was conservatively managed successfully.

CASE REPORT

We presented a 52-year-old male state security operative and a known hypertensive who was admitted into our facility with a new onset acute right upper and lower limb weakness with associated left-sided gaze impairment and facial palsy. There was a preceding history of generalized, severe, and throbbing headache, which was transiently responsive to analgesics. He also had accompanying episodes of projectile vomiting, which later resolved spontaneously. Apart from dysarthria, he did not experience dizziness or dysphagia and consciousness was preserved.

Patient had significantly used marijuana and tobacco for more than 5 years up until the time of his sickness. He, however, took alcoholic beverages occasionally. However, he denied history of intravenous drug use.

Apart from hypertension, he did not volunteer a history of type 2 diabetes and random blood sugar tested on admission was normal. On further questioning, he disclosed poor adherence to his antihypertensives medications despite enrolling into a health insurance program. He had never experienced a transient ischemic attack in the past. Family history of hypertension or type diabetes mellitus could not be ascertained. Patient was married in a monogamous family with three (3) adult children (2 females and a male).

At presentation, he was fully conscious, but restless and diaphoretic and had severely elevated blood pressure (250/170 mmHg) with displaced heaving apex. Oxygen saturation was 95% at the room air. Other significant findings noted were left abducens and facial nerve palsies with intact left adduction and preserved upward gaze. The contralateral cranial nerves were intact (see Table 1 below). However, there was weakness (power grade 4/5) of the contralateral upper and lower limbs. In addition, he had left appendicular cerebellar signs such as ataxia and dysmetria. Other systems were essentially intact.

His investigation results, including serology tests for HIV, Hepatitis B virus and Hepatitis C Virus, were all negative; urinalysis revealed moderate proteinuria (2+); full blood count parameters equally showed normal findings; both initial and repeat serum electrolyte urea and creatine results showed normal parameters as shown in Table 1. His Electrocardiogram (ECG) result showed inferior lateral ischemic infarcts, left ventricular hypertrophy with strain pattern, reciprocal changes, and left axis deviation (LAD).

Brain magnetic resonance imaging (MRI) showed pontine hemorrhage with background white matter microvascular infarcts as shown in Figure 2

Patient was managed conservatively in accordance with the recommended guidelines for managing hemorrhagic stroke, including management of hypertension, intracranial pressure, and comorbidities. He received oral antihypertensive drugs, 20% intravenous mannitol and multivitamins. Neurorehabilitation was also activated.

His clinical state improved significantly within two weeks of hospitalization and was discharged a week later to the Neurology Outpatient Clinic for follow up. The patient has remained stable since after discharge and his visual symptoms have also improved remarkably.

Table 1. Patient's serum electrolyte, urea, & creatinine results

Parameter	Result 1	Result 2	Reference
Sodium	140	138	128-148mmol/L
Potassium	2.9	4.3	3.4-4.8mmol/L
Bicarbonate	24	22	24-30mmol/L
Urea	4.9	3.8	1.5-6.6mmol/L
Creatinine	108	113	60 – 120µmol/L
Chloride	104	107	98-108mmol/L
Calcium	2.38	3.0	2.02-2.6mg/L
Uric acid	274	288	208-428µmo/L

Figure 1. Left abducens (CN 6) and facial (CN 7) nerve palsies (on admission)

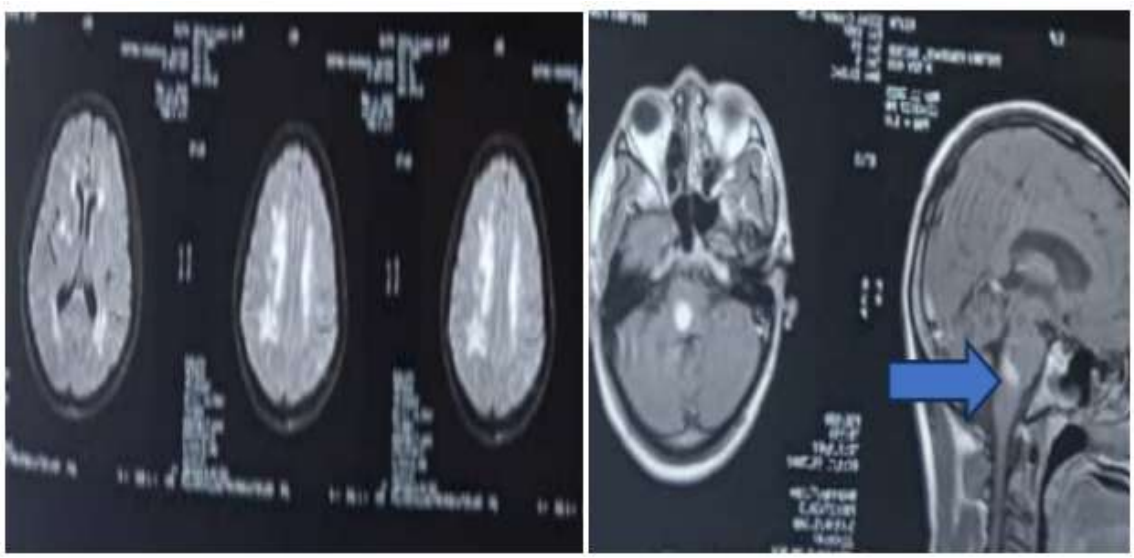

Figure 2. MRI result of the patient showing pontine hyperintensity (blue arrow) and white matter hyperintensity (yellow arrow).

DISCUSSION

We presented this case report of a middle-aged male, known hypertensive , patient of ours who was admitted into our facility on account of left facial nerve and gaze palsy, left appendicular cerebellar ataxia and contralateral hemiparesis and was thought to have Millard -Gubler Syndrome (MGS). Magnetic Resonance Imaging helped to clarify our diagnosis by demonstrating a pontine hemorrhage and deep periventricular hyperintensities (suggesting microvascular infarcts). Generally, a diagnosis of Millard -Gubler Syndrome (MGS) is usually made in a patient with suspicious symptoms in combination with the appropriate brain imaging. We followed the same sequence to establish this diagnosis in our patient

Millard-Gubler syndrome is a rare brainstem disorder resulting from a lesion to the ventral pons (1). The classical features of this syndrome comprise ipsilateral facial nerve palsy, ipsilateral abducens palsy and contralateral hemiplegia (1,10). Millard-Gubler syndrome (MGS) is an eponym after two French physicians, Auguste Louis Jules Millard and Adolphe-Marie Gubler who first described the features of this syndrome in 1858.The first description of this condition was in association with a pontine mass (1,2). Since then, other etiologies have been described with stroke accounting for most of the cases of MGS ((11–14)) Our patient presented with all the classical features MGS in addition to the left limb ataxia and dysmetria. Cerebellar features can sometimes be associated with this syndrome depending on the extent of the lesion, Interruption of the cerebellopontine fibres is though to be responsible for the emergence of these features in MGS (15). Another case of MGS with cerebellar features was reported by Ayele et al in Ethiopia in a 55-year- old male that suffered a pontine infarction.(15) . It was curious this patient did not have hypertension as well as other traditional cardiovacular risks, even though he was not extensively investigated for other rare causes of ischemic stroke such as monogenic arteriopathies. However, for our patient, there was a lon standing history of hypertension (16).

Compared to brainstem hemorrhage, pontine infarction appears to have been more frequently described in various case reports of MGS than the former. For both ischemic and haemorrhagic strokes, uncontrolled hypertension is the main driver of these events, and poor adherence to to the prescribed treatments remains a major cause. Our patient admitted to a history of poor drug compliance and that reflected in his high admission blood pressure. As mentioned above, hypertension remains the most important risk factor of stroke, irrespective of the subtype (16,17), and measures to address stroke risks should be part of the strategies towards steming stroke tide. These measures should be incorporated into stroke education for patients.

Our patient was a middle-aged man in his 50’s and in this age bracket, the commonest cause of Millard-Gubler syndrome (MGS) is vascular, whether haemorrhagic or ischemic stroke (5). In terms of the vascular territory involved, occlusion or rupture of the short circumferential branch of the basilar artery formed by the confluence of the vertebral arteries could be the reason for the emergence of symptoms of MGS (4). Sometimes, compression of pontine arteries by subarachnoid haemorrhage or a space-occupying lesion can also lead to MGS, (8,18) Rarely, a brain stem cavernous vascular malformation may cause MGS, especially if a recent bleed has happened (3,8) . For our patient, although it was clear that uncontrolled hypertension caused his brainstem bleed, the specific vessels involved could not be ascertained since an angiography of the brain was not conducted. In the younger age group, common causes of MGS include tumours, infections (e.g., neurocysticercosis),(13) viral infection (e.g., rhombencephalitis), demyelinating disease (e.g., multiple sclerosis), and immune-mediated inflammatory disorders, such as neuro-Behçet's disease (11).

Primary brainstem hemorrhage is usually associated with the worst prognosis of all types of brainstem ICH, and prognosis will depend on factors such as age, presence of coma, blood glucose, Glasgow Coma Scale (GCS), hemorrhage size, location, and extent of hemorrhage (6,8). Our patient had some of these variables in his favor, especially the fact he never lost consciousness right from the admission, and his blood sugar level was also within normal limit. These factors as well as other possible covert variables might have influenced his excellent clinical outcome despite suffering a potential life-threatening condition.

Finally, our patient made a significant clinical improvement within three weeks of (6) hospitalization and was successfully discharged. Good clinical out have also been documented following brainstem stroke in other case reports. (9,10).

CONCLUSION

This case report has achieved two important purposes. Firstly, it has provided an opportunity to showcase a rare ventral pontine syndrome in a Nigerian patient who suffered a pontine bled and was successfully managed. It has also reechoed the strategic role of brain imaging, especially magnetic resonance imaging, in confirming diagnosis and differentiating it from other differential diagnoses. Complete recovery is usually expected with supportive treatment and in the absence of any major complications.

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