

Millard-Gubler Syndrome in a Patient with Pontine Infarction: A Case Report

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Abstract

Millard-Gubler Syndrome is a rare neurological condition caused by damage to the sixth and seventh cranial nerves, as well as the corticospinal tract in the brainstem. It is characterized by the presence of ipsilateral facial paralysis and contralateral hemiplegia. We report a 55-year-old male patient who presented with sudden onset of left-sided weakness. Imaging revealed a pontine infarct. The patient therefore, was diagnosed with Millard-Gubler Syndrome also known as Ventral Pontine Syndrome based on his symptoms and imaging findings. He was treated with Aspirin and Atorvastatin and was referred to neurology for further consultation and to physiotherapy for his weakness. This case report highlights the importance of prompt recognition and diagnosis of Millard-Gubler Syndrome in patients with pontine infarction. Early identification especially with the use of high-resolution MRI can facilitate appropriate management and treatment, ultimately improving patient outcomes.

Keywords

Millard Gubler Syndrome, Case Report, Ischemic Stroke, Pontine Infarction

1. Introduction

Brainstem contains the nuclei and fibers of cranial nerves III to XII, both motor and sensory tracts, and crucial vegetative structures for cardio-respiratory functions and wakefulness. Therefore, any injury can cause focal manifestations including cranial nerves deficits and more serious symptoms involving motor or sensory impairment, vegetative state and even death [1]. Brainstem damage is mostly due to ischemic stroke [2]. Furthermore, almost 7% of all ischemic strokes and 20% of brain stem strokes are pontine infarction that can be isolated

or not when presented in other parts of the brain [3]. Millard-Gubler syndrome (MGS) is due to the infarction of the ventral segment of the paramedian pons and is named after Adolphe Gubler and Auguste Millard defining the pontine stroke [4]. Clinical manifestations usually involve unilateral facial nerve and abducens nerve palsy with contralateral hemiplegia [5].

The aim of this report is to present an original case of Ischemic stroke leading to a Millard Gubler syndrome, and to discuss its presentation and management.

2. Case Presentation

A 55-year-old male presented to the emergency department of Fujairah hospital at 10 am complaining of left sided body weakness for the past five hours. He woke up at 5 am and found a considerable loss of strength in his left upper and lower limb. He is a driver by profession and found that he could not even drive his truck due to his weakness. He experienced dizziness as he was driving and there was slurring of his speech when talking to his supervisor. He also complained of a tight band like headache which started when he woke up. It did not radiate elsewhere, it was not associated with any other symptoms and was graded as mild to moderate in severity. He also reported decreased sensations on the left side of his body. The patient was fine the night before and had no such complaints when he went to sleep.

He did not have a similar complaint in the past. Regarding comorbidities, the patient has hypertension which was diagnosed 6 months ago. He was prescribed medication (valsartan 160 mg-amlodipine 5 mg) but he did not take it for the past 2 days. He does not have diabetes, dyslipidemia or any known heart diseases. Past surgical history is insignificant.

The patient is a chronic smoker with a 35 pack-year history. He consumed alcohol for 18 years but stopped 21 years back. His job is demanding and he drives for up to 18 hours per day.

On general physical examination the patient was conscious and oriented. However, facial asymmetry was noted with loss of the right nasolabial fold and left sided facial deviation when the patient was asked to open his mouth. Decreased range of motion of his left upper and lower limb was also observed. There were no other significant findings.

Neurological exam was performed and it revealed that higher mental functions were intact. Cranial nerve VII was tested; forehead wrinkles were absent on the right side of his face and there was a loss of the right nasolabial fold and a left sided facial deviation when the patient was asked to smile, clench his teeth and open his mouth. Other cranial nerves (II-XII) were intact. Motor examination of the left upper limb on inspection showed normal muscle bulk and the limb was kept close to his body. No abnormal movements such as tremors or fasciculations were noted. Hypertonia and hyperreflexia (grade +3) were present with a power grade of (4-)/5.

Motor examination of the left lower limb revealed hypertonia and hyperref-

lexia (grade +3) with power grade of 3/5. There was extensor plantar reflex (Babinski positive) in the left foot. Sensory and cerebellar examination was normal. He walked with a hemiplegic gait and tends to sway to his right.

Routine laboratory investigations were done (**Table 1**) and a complete blood count (CBC) revealed low hematocrit, RBCs and platelets. Random blood glucose was high [8.4 mmol/L (normal: 3.9 - 7.1 mmol/L)].

A non-contrast axial CT scan of the head was done which showed:

- No focal cerebral area of abnormal density.
- Normal gray-white matter interface.
- No extra-axial or intra-cerebral collection of fresh blood.
- No midline shift.

Sinus bradycardia (heart rate 55 beats per minute) with T wave inversion at lead V4 and V5 were seen on ECG and an ECHO revealed left ventricular hypertrophy.

MRI of the brain showed acute infarction of the ventral pons (**Figure 1**):

- Hyperintensity in the right side of the pons (high DWI and low ADC values);
- Periventricular high FLAIR signal foci representing small vessel disease.

This led to a definitive diagnosis of an acute ischemic stroke which was an acute right pontine infarct.

Table 1. Laboratory findings.

	Results	Reference
Hemoglobin	12.5 g/dL	12.0 - 16.0 g/dL
WBC count	$8.69 \times 10^9/L$	$4.5 - 11.0 \times 10^9/L$
Blood Glucose	6.6 mmol/L	3.9 - 5.5 mmol/L
Potassium	3.16 mmol/L	3.5 - 5.0 mmol/L
Creatinine	44 $\mu\text{mol/L}$	60 - 110 $\mu\text{mol/L}$
Albumin	31.8 g/L	3.9 - 4.9 g/L

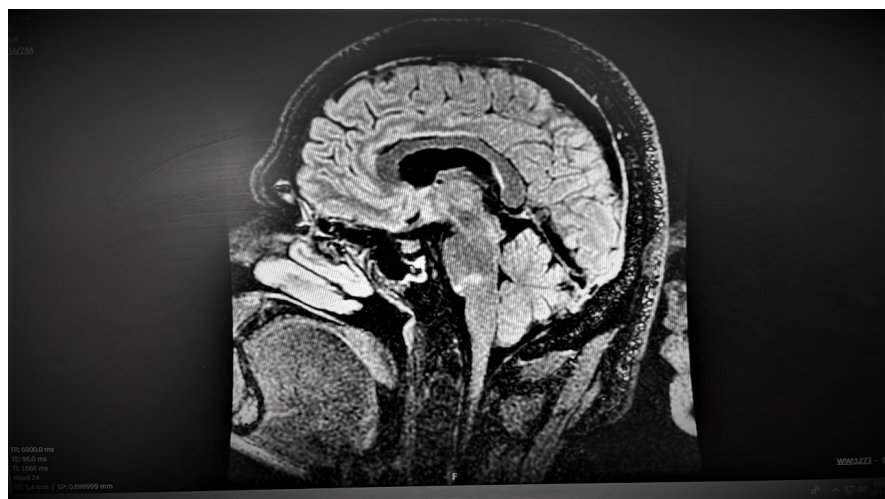


Figure 1. Brain MRI showing acute infarction of the ventral pons.

Since the patient presented to the hospital after the golden window period, thrombolysis could not be done. Instead the patient was admitted, aspirin (100 mg oral q24hrs) and atorvastatin (20 mg oral at bedtime) was given and he was referred to neurology for further consultation. Valsartan 160 mg-amlodipine 5 mg given to control his hypertension and he was referred to physiotherapy for his weakness.

The patient was educated about his condition and his management plan.

3. Discussion

Millard-Gubler syndrome (MGS) or Ventral Pontine syndrome is named after two French physicians Auguste Louis Jules Millard and Adolphe-Marie Gubler in 1858. As the name suggests, the infarction involves the ventral portion of the paramedian pons [5]. It is also known as facial abducens hemiplegia syndrome or the ventral pontine syndrome [6]. The syndrome involves the combination of ipsilateral abducens (CN VI) and/or facial (CN VII) palsy as a lower motor neuron lesion and a contralateral hemiplegia/hemiparesis of the upper and lower limbs (Figure 2) [7]. By contrast, pontine haemorrhage due to high blood pressure usually involves medially and damages CN VI nucleus and CN VII fibers, resulting thus to Foville syndrome [8]. Other neurological deficits such as contralateral hemianesthesia can also be present which was seen in our patient.

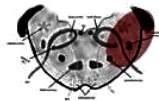
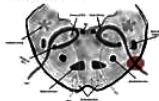
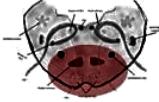

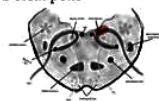

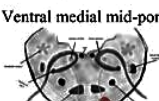
Components of MGS [6]

- 1) Contralateral hemiparesis/hemiplegia of the extremities due to pyramidal (corticospinal) tract involvement.
- 2) Ipsilateral lateral rectus palsy (due to CN VI involvement). This causes diplopia when the patient looks towards the side of the lesion and esotropia.
- 3) Ipsilateral peripheral facial nerve palsy (due to CN VII involvement).

In our case, the patient had left hemiparesis and right sided facial palsy owing to his right sided pontine infarct. Given his past history of hypertension, a CT scan was done to rule out a hemorrhagic stroke. In this case, a long standing history of smoking, alcohol ingestion, his age and high blood sugar suggests a more probable diagnosis of an ischemic stroke, for which an MRI was done. Occlusion of the basilar artery is a cause of ventral pontine infarction [9] and small vessel disease affecting the pontine branches of the basilar artery was seen on MRI as well. MRI have a key role in distinguishing vascular etiologies of a ventral pontine infarction from other disorders including malformations, tumors, demyelinating disorders and tuberculosis [10].

In addition, previous evidence reported multiple risk factors associated with the occurrence of pontine infarction including dyslipidemia, hypertension, diabetes and others [11]. Similarly, our patient is an old male who had hypertension in addition to be a chronic smoker and alcohol heavy drinker suggesting the cause of the atherosclerotic origin of the pontine infarction. Therefore, it is important to identify at high risk of developing an ischemic stroke and the significance of applying secondary stroke prevention plans.

Table 1. Classical Pontine Stroke Syndromes

Syndrome	Location	Structures Affected	Clinical Presentation
Marie-Foix (Lateral Pontine) Syndrome ^{1,2*}	Lateral pons 	Corticospinal tract, spinothalamic tracts, facial nerve, vestibulocochlear nucleus, cerebellar tracts	Contralateral hemiplegia and loss of pain and temperature sensation Ipsilateral limb ataxia, facial paralysis, hearing loss, vertigo, and nystagmus
Millard-Gubler Syndrome ³	Ventral pons 	Corticospinal tract and the fascicular intrapontine portion of the VII nerve	Unilateral facial and/or abducens palsy with contralateral weakness of the arm and leg
Locked-in Syndrome ⁴	Ventral pons 	All motor fibers running from grey matter in the brain via the spinal cord to the body's muscles and damage to the centers in the brainstem important for facial control and speaking	Quadriplegia and anarthria with preservation of consciousness
Brissaud-Sicard Syndrome ⁵	Anterolateral and inferior pons 	Corticospinal tract, facial nucleus or nerve root	Ipsilateral facial cramps, contralateral hemiparesis
Facial Colliculus Syndrome ⁶	Dorsal pons 	Facial colliculus, comprising of the nucleus of the abducens nerve, the facial nerve, the paramedial reticular formation, and the medial longitudinal fasciculus	Ipsilateral lower motor neuron pattern of facial nerve palsy, ipsilateral lateral rectus palsy, and frequently, conjugate gaze palsy due to an associated contralateral medial rectus palsy
Gasperini Syndrome ⁷	Lateral caudal pons 	Ipsilateral facial nerve nucleus, abducens fibers, acoustic fibers, spinal trigeminal tract, spinothalamic tract	Peripheral facial palsy, abducens nerve palsy, hypacusis, facial hypesthesia, contralateral hemihypesthesia for pain and temperature
Raymond Syndrome ⁸	Ventral medial mid-pons 	Abducens nerve, nondecussated corticofacial fibers, nondecussated corticospinal tract	Ipsilateral abducens impairment, contralateral facial paresis, and contralateral hemiparesis

*Though the classical picture of Marie-Foix syndrome involves the corticospinal and spinothalamic tracts, cerebellar tracts, and CN 7 and CN 8, some sources also indicate involvement of the spinal trigeminal tract and nucleus 615. <https://doi.org/10.1093/qjmed/hc992>

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Figure 2. Different types of syndromes and their characteristics.

4. Conclusion

An acute ischemic stroke which was an acute right pontine infarct in our adult patient may have a good prognosis, if diagnosed and managed early. High-resolution MRI is important in early detection. Future studies regarding Millard-Gubler syndrome are needed to assist the interprofessional management team.

Human Ethics

Informed consent was obtained from the patient to report this case report

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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