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A Rare Neurological Sequela: Pontine Infarct Conducing to Millard-gubler Syndrome

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Authors' contributions:

This work was carried out in collaboration among all authors. Authors HRSLV and NA gathered the case from emergency ward and author HRSLV arranged the theoretical and the presentation review of the case report. Author NA aided in reviewing the literature part and authors BAC and RLG chipped away at the case show alongside the remaining writers. Authors PK and MN aided in drafting the presentation, discussion, and summarized the conclusion part of the case report. Author PKY alongside author RLG dealt with the literature Searches and other authors in the arrangement of the manuscript. Every one of the Authors read and supported the last original copy. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Background: Ventral Pontine Syndrome, likewise called Millard Gubler Syndrome (MGS) or Facial abducens hemiplegia disorder is a neurodegenerative problem that is described by one-sided lesions influencing the basal piece of the caudal pons including fascicles of the abducens and the facial nerves and the pyramidal lot filaments. Clinical findings incorporate fringe Facial Nerve Paralysis and neuromuscular shortcoming or loss of motion of at least one appendage. This is an exceptionally uncommon condition assessed to influence 1 out of 10, 00, 000. Men (aged 40-60 years) are bound to foster the condition than ladies.

Case Nuances: A 45-year-old male patient had intensely passed a-sided facial droop 4 days prior to presenting with weakness on the left half of his upper and lower appendages. He, along with his attender likewise referenced that he had recently had a 2-minute loss of cognizance, discombobulation, and vertical shifting of the right eye both when he lost his consciousness. He had low serum Vitamin D and was determined to have Millard Gubler Condition as an Infarct in the pontine locale was recognized in imaging, and was dealt with restoratively and with Non-intrusive treatment.

Discussion: Millard-Gubler disorder might cause cerebellar ataxia if the paramedian pontine dead tissue region somewhat expands horizontally, influencing the centre cerebellar peduncles. Detached pontine localized necrosis might have decent progress whenever analyzed and overseen early.

Conclusion: MGS is typically found in instances of brainstem cancers, drain, tuberculoma, parasitic contamination, Ischemic stroke secondary to impediment of the basilar supply route, and injury.

Keywords: Millard gubler syndrome; ipsilateral facial paralysis; contralateral hemiplegia; abducens nerve palsy; pontine infarct.

1. INTRODUCTION

Millard-Gubler Condition (MGS) is characterized as a one-sided lesion of the ventrocaudal pons at the level of the facial nerve core, which might include the pontine center and the fascicles of VI, and VII cranial nerves. It prompts abducens nerve paresis and a contralateral hemiparesis that frequently influences the face, while an ipsilateral fringe facial nerve paresis may likewise happen when the sore stretches out adequately horizontally to harm the fascicle of the facial nerve [1-3].

This is a charming ventral pontine problem that shows up with the ipsilateral deficiency of the eye upon snatching (abducens nerve), ipsilateral facial muscle inadequacy (facial nerve), and contralateral hemiparesis or hemiplegia of the upper and lower limbs (pyramidal nerve strands) [4-6]. This is generally a direct result of an uneven lesion at the basal part of the caudal pons due to a tumor, hemorrhage, or sometimes, infarction [7-10]. Hemorrhage and localized necrosis are more typical in seasoned patients, while tissue development and illnesses are more ordinary in additional young individuals.

Pontine dead tissue addresses 7% of each and every ischemic stroke and 20% of Brainstem strokes [11]. Pontine areas of limited corruption can be confined or not isolated if they are present in that frame of mind of the cerebrum. The caudal pontine issue can have two instances of the presentation considering the piece of penetrating paramedian vessel affiliation: proximal and distal obstacle [12]. Likewise, infarction including the ventral piece of the paramedian pons will result in the ventral pontine condition, moreover called Millard-Gubler Syndrome [13].

The eponymous MGS, a representation of crossed hemiplegia, was named after Adolphe Gubler (1821-1879) and Auguste Millard (1830-1915), following their exact portrayal of the pontine stroke [14]. Millard-Gubler Condition is described as an uneven sore of the ventral pons at the level of the facial nerve center that presents as an uneven facial nerve and abducens nerve loss of motion with contralateral hemiplegia [15,16]. Ventral pontine conditions

are typically discretionary to demyelinating wounds, malignant growths, injury, irresistible causes, and rare areas of dead tissue [17]. Bell's palsy is an outcome of an extreme idiopathic periphery facial nerve loss of motion as a result of an uneven lower motor neuron lesion between the facial nerve centers and muscles.

Accuracy in clinical assessment, the specific confinement of the lesion by imaging strategies, and information on any likely fundamental causes are important for the conclusion of Millard-Gubler's Syndrome [18-20]. The pathophysiology of the condition can be brought about by a large number of aetiologies including vascular occasions, growths, inflammatory sequences, and underlying irregularities, every one of which requires a particular way to deal with care.

Vascular etiologies have been perceived as significant reasons for Millard-Gubler's condition. Central brainstem areas of dead tissue result from ischemic strokes, which habitually result from blockages in the paramedic parts of the basilar conduit. Basic motor pathways and cranial nerve cores are disturbed because of the confined bloodstream, which prompts the mark of clinical appearance. Comparable lacks may likewise rise out of hemorrhagic strokes, which are brought about by blood vessel bursts in the brainstem. The condition shows itself distinctively relying upon when portions of the pons are defenceless.

Cancers and primary irregularities are additionally significant patrons. Millard-Gubler condition can be welcomed by oddities like arteriovenous malformations (AVMs), enormous contortions, and formative vascular irregularities. These peculiarities, which are now and again present from birth, force weight on adjacent brainstem districts, packing engine pathways, and cranial nerve cores. It ought to be noted that cancers, whether essential or metastatic and their development inside the pons cause the disorder's characterizing side effects by influencing close by tissues.

Irresistible and infectious cycles give more understanding of the condition's starting point. Whether it is an immune system or viral in the beginning, brainstem-related encephalitis harms neurons and causes cranial nerve brokenness. Demyelination may be a piece of incendiary pathways, which would slow down mind indicating. Furthermore, pathogenic microorganisms that attack the brainstem set up immunological responses that damage engine pathways and cranial nerve cores unexpectedly.

Millard-Gubler condition can be set off by injury, either straightforwardly from an effect or because of dying. If decisively situated inside the pons, occasions of demyelinating ailments like various sclerosis might give similar side effects [21]. Besides, ischemic episodes can cause transient ischemic assaults (TIAs) that unexpectedly re-enact the clinical attributes of the condition.

The vascular entanglements that are regularly connected to cerebrovascular mishaps assume a fundamental part in the rise of Millard-Gubler's condition. A significant component that builds the gamble of vascular injury and ischemic occasions in the brainstem is hypertension.

Smoking, atherosclerosis, and a past filled with cardiovascular infection can all deteriorate vascular issues, which could ultimately prompt the condition. Furthermore, diabetes mellitus, which influences the strength of veins, may be calculating vascular conditions that favor the improvement of Millard-Gubler disorder [22].

The potential gamble profile incorporates incendiary and immunological elements. Provocative responses that focus on the brainstem are more probable in immune system ailments like vasculitis or demyelinating sicknesses. Resistant reactions bringing about neuronal injury and cranial nerve brokenness can be set off by contaminations that influence the brainstem, like encephalitis.

The peril might be expanded by resistant regulating drugs or debilitated insusceptibility welcomed on by other clinical problems. In spite of the fact that corticosteroids reduce irritation and its basic causes, they are ordinarily used to treat Millard Gubler's disorder [23-24].

All things considered, this is the principal case to be represented from the Northern Coastal area of Andhra Pradesh. For this circumstance report, we present a patient with an extraordinary starting facial droop with no great explanation. An expansive workup was done to choose the presence of stroke and conceivable vein stenosis. The patient was found to have serious dead tissue at the left dorsal brainstem at the Ponto medullary crossing point which, close by his clinical presentation, featured the investigation of Millard-Gubler's condition.

2. CASE PRESENTATION

A 45-year-old male patient with no significant history was referred to the Emergency department with a major complaint of left upper and lower limb weakness that had been present for four days along with a mouth deviation to the left, and a left-sided facial droop in the last two days and was seen worsening gradually. He complained of Right eye-rolling, tingling, and numbness all over the limbs for 4 days, a change in the gait, and a projectile vomiting once that lasted up to 5 minutes after losing consciousness.

He denied any history of Trauma, travel, and Upper Respiratory Tract Infection. He had never experienced these symptoms earlier and was found upon history taking that, the family history has nothing to do with this particular presentation. The patient had been consuming alcohol, was a chronic smoker with 20 pack-years, and consuming Gutka (Betel Quid) for over 10 years. His blood pressure and pulse rate were normal. Furthermore, there was no history of prior stroke, transient ischemic attack, diabetes, hypertension, head trauma, or dyslipidemia. On examination, he was fully conscious and oriented. He had horizontal left gaze palsy with horizontal Nystagmus, suggesting left-sided 6th cranial nerve palsy. He had mild left-sided facial palsy causing dysarthria speech. Right upper limb dysmetria was observed during the examination; otherwise, motor, sensory, and fundus examination results were normal.

Upon examination at arrival to the EMR, he was afebrile, had a pulse of 114 bpm, respiratory pace of 25 cycles per minute, circulatory strain of 191/103 mmHg, and oxygen saturation of 94% on room air. On assessment, his pupils were equivalent, round, and receptive to light, but was reported to be having eye dryness and irritation. Extraocular developments were flawless reciprocally. The facial sensation was flawless, however, there was a left upper and lower facial paralysis with the diminished unmistakable quality of the left nasolabial crease. Motor and Sensory assessment of bilateral upper and lower limits were ordinary and reflexes were flawless. He was oriented to individual, spot, time, and circumstance and his discourse was ordinary. The remainder of the physical assessment was unexceptional.

Lab examinations have shown no huge electrolyte anomalies. Because of worry about intense or sub-acute stroke, a non-contrast figured tomography angiography of the head, furthermore, and neck was done. This uncovered a sub-centimeter hypodensity in the left lentiform core, mirroring an age-uncertain lacunar infarct. There was additionally gentle stenosis of the proximal basilar course and moderate stenosis of the beginning of the right back cerebral course. There were no indications of the Hemorrhage. He was given Aspirin and Rosuvastatin in the Emergency Room. Tolerant hypertension was kept up for 24 hours, circulatory strain was then dealt with by oral antihypertensive agents. Thrombolysis was not shown since he was presented more than 5.5 hours after the beginning of the clinical presentation.

On examination of cranial nerves, right lateral rectus palsy (6th cranial nerve) and right orbicularis palsy with a deviation of mouth to the left side (7^{th} cranial nerve), and sub-acute infract involving the right pontomedullary junction and partial blockage of the left vertebral artery is apparent on the MRI scan.
In the patient's ECHO study, there was a collapse of the interventricular artery. Bell's palsy, lateral rectus palsy, and hemiparesis are the patient's diagnoses which can also be recognized as affected with Millard Gubler's Syndrome.

The patient was in this manner conceded for the management of an ischemic stroke. Differential diagnoses were confirmed as Bell's paralysis and Millard-Gubler Syndrome. Serological tests for Hepatitis B, Hepatitis C, and HIV were negative. To preclude Lyme infection as the reason for facial nerve paralysis, Lyme sickness IgG/IgM antibodies were requested, which returned negative. The conclusion of Millard-Gubler's condition was made in light of imaging discoveries of intense pontine dead tissue and neurological discoveries of ipsilateral facial paralysis. He started taking the recommended medications on Day 1 (T. Aspirin 150 mg BD, T. Atorvastatin 40 mg BD, T. Gabapentin 400 mg & Nortriptyline 10 mg BD, T. Vertin 16 mg TID, Cap. Nuhez OD) and continued taking them on Days 2, 3, and 4 as well. However, on day 3, the prescription additionally includes an injection of Thiamine 100mg TID, Paracetamol 1gm SOS (patient having temperatures above 100°F), and methylcellulose drops. Anti-platelets in addition to statins have an anti-inflammatory effect and are also advised for stroke patients. The pain caused by nerve injury or neuropathic pain diminishes with the use of Nortriptyline and gabapentin. Vertin is a medication used to

address balance issues. Thiamine is a supplement for the nerves. Methylcellulose drops are used to reduce tears and irritation.

He was treated with dual antiplatelet treatment with Aspirin and Clopidogrel. Actual restoration was started. By the eighth day of confirmation, his symptoms had totally settled. He was then discharged, and following 30 days of anti-platelet treatment, he was asked to take Aspirin along with the statin. On trial for about two months, he had no remaining fresh/past symptoms or new neurological side effects.

3. DISCUSSION AND CONCLUSION

Millard-Gubler disorder, one of the traditional pontine-crossed conditions, was first depicted in examination examples by the French School of Nervous System Science in the 1900s. Initially, the reason for the neurologic shortage was accepted to be tumoral. Our case report stresses the conceivable vascular reason for the condition. For our patient’s situation, the exact anatomic area was related to imaging. The neuro-anatomic pathways present at the inferior pontine level, in the focal locale of the ventral pons course the corticospinal and corticobulbar plots. The average lemniscus lies posteriorly on each side of the middle raphe. Cores of the VI and VII nerves are tracked down in the dorsal piece of the pons: their filaments go through the pontine tegmentum and arise anteriorly at the cerebello-pontine point. The spinothalamic plot possesses a situation in the anterolateral tegmentum, average to the slipping parcel and core of the V nerve. The lesion causes Millard-Gubler disorder, which contains the corticospinal parcel and the fascicular intrapontine part of the VII nerve, causing ipsilateral loss of motion of the facial nerve and contralateral hemiplegia. The average lemniscus and the spinothalamic lot are saved in this disorder, subsequently making sense of the shortfall of tactile disability.

The association of the pyramidal parcels for the most part appears in hemiplegia or hemiparesis of the upper or lower furthest points notwithstanding facial paralysis. For our patient’s situation, there was no motor shortfall in the limits. The patient whined about left-sided furthest point paralysis which had settled when of assessment, and no tactile shortfall in the limits was available on beginning or resulting assessments. The one-sided facial shortcoming was unmistakable for this situation, and the show was regular of fringe facial paralysis, with the inclusion of the upper and lower appendages. One of the elective conclusions considered was Bell's palsy, which is a finding of rejection. Nonetheless, the presence of contralateral tangible side effects, but transient, wouldn't be normal in Ringer's paralysis.

Other intracranial pathologies like growths, vascular mutations, and neurocysticercosis were avoided employing imaging. Herpes zoster reactivation is one more likely reason for facial nerve paralysis yet would give torment and vesicles on assessment in impacted dermatomes, which were absent for this situation. Also, proof of intense pontine localized necrosis on imaging affirmed the finding of Millard-Gubler's condition.

Our patient had a full recuperation in the span of multi week of presentation. This is reliable with the clinical course depicted if reports, particularly those including little intense infarcts. Brainstem infarcts are normally found behind the scenes of different gamble factors like hypertension, diabetes, and hyperlipidemia.

Our patient had beforehand undiscovered hypertension yet no other critical clinical history. Our case exhibits the utility of imaging in diagnosing Millard-Gubler's condition and recognizing it from other differential judgments, particularly in cases with a flighty patient profile and show.

On the off chance that the patient had not been dealt with, facial nerve issues could prompt trouble eating. It could display signs remembering soft spots for the contrary appendage and the face, which may some of the time be painstakingly settled. While affirming the finding and recognizing it from other potential analyses like vascular abnormalities, growths, or other intracranial masses, an X-ray can be incredibly gainful. With strong supportive management, complete recovery is achievable.

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CONSENT

Every author proclaims that informed consent was acquired from the patient, and the signed consent form is submitted alongside the manuscript and submission form.

ETHICAL APPROVAL

As per international standards or university standards written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Ropper AH, Samuels MA, Klein JP, Prasad S: Disorders of motility: paralysis and weakness. Principles of Neurology. McGraw-Hill, New York. 2019;71.
2. Matlis A, Kleinman Y, Korn-Lubetzki I: Millard-gubler syndrome. AJNR Am J Neuroradiol. 1994;15:179-81.
3. Onbas O, Kantarci M, Alper F, Karaca L, Okur A: Millard-gubler syndrome: MR findings. Neuroradiology. 2005;47:35-7. Available:10.1007/s00234-004-1312-1
4. Kesikburun S, Safaz I, Alaca R. Pontine cavernoma hemorrhage leading to Millard-Gubler syndrome. Am J Phys Med Rehabil. 2011;90:263. Available:10.1097/PHM.0b013e3181e29e8e
5. Ceballos-Lizarraga R, Palomino-Díaz C, Romero-Figueroa JÁ. Wall-eyed monocular internuclear ophthalmoplegia (WEMINO) and Millard-gubler syndromes in a patient with isolated pontine infarction: topographic, oculomotor, and radiological analysis of two very uncommon conditions. Case Rep Neurol. 2019;11:230-7. Available:10.1159/000501794
6. Li XT, Yuan JL, Hu WL. Vertebrobasilar artery dissection manifesting as Millard-Gubler syndrome in a young ischemic stroke patient: a case report. World J Clin Cases. 2019;7:73-8. Available:10.12998/wjcc.v7.i1.73
7. Prasad R, Kapoor K, Srivastava A, Mishra O. Neurocysticercosis presenting as Millard Gubler syndrome. J Neurosci Rural Pract. 2012;3:375-7. Available:10.4103/0976-3147.102634
8. Lewis PD. The classical brain stem syndromes (translations of the original papers with notes on the evolution of clinical neuroanatomy). Med Hist. 1973;17:106.
9. Dourado Leite R, Freitas C, Guimaraes S. Vertical muscle transposition with silicone band belting in VI nerve palsy. BMJ Case Rep. 2016;2016. Available:10.1136/bcr-2016-216722
10. Ayele BA, Tadesse Y, Guta B, Zenebe G. Millard-Gubler syndrome associated with cerebellar ataxia in a patient with isolated paramedian pontine infarction - a rarely observed combination with a benign prognosis: a case report. Case Rep Neurol. 2021;13:239-45.
11. Huang J, Qiu Z, Zhou P, Li J, Chen Y, Huang R et al. Topographic location of unisolated pontine infarction. BMC Neurol. 2019;19(1):186.
12. Evans MR, Weeks RA. Putting pontine anatomy into clinical practice: the 16 syndrome. Pract Neurol. 2016;16(6):484–7.
13. Ahmad H, Bukhari MH, Asghar M. Infarction of ventral pons presenting as millard-gubler syndrome: A case report. J Rawalpindi Med Coll. 2019;23:128–9.
14. Walusinski O. Adolphe Gubler (1821–1879) or Parisian neurology outside La Salpêtrière in the age of Jean-Martin Charcot. Rev Neurol (Paris). 2019;175(4):207–16.
15. Kannengiesser K, Mahlke R, Petersen F, Peters A, Kucharzik T, Maaser C. Instant evaluation of contrast-enhanced endoscopic ultrasound helps to differentiate various solid pancreatic lesions in daily routine. World J Clin Cases. 2019;7(1):19–27.
16. Yang L, Qin W, Li Y, Yang S, Gu H, Hu W. Differentiation of pontine infarction by size. Open Med (Wars). 2020;15(1):160–6.
17. Ortiz de Mendivil A, Alcalá-Galiano A, Ochoa M, Salvador E, Millán JM. Brainstem stroke: anatomy, clinical and radiological findings. Semin Ultrasound CT MR. 2013;34(2):131–41.
18. Matlis A, Kleinman Y, Korn-Lubetzki I. Millard-Gubler syndrome. AJNR Am J Neuroradiol. 1994;15(1):179-81. PMID: 8141053. PMCID: PMC8332095.
19. Chakraborty U, Santra A, Pandit A, Dubey S, Chandra A. Space occupying lesion presenting as Millard-Gubler syndrome. BMJ Case Rep. 2022;12;15(4):e248590.
20. Silverman IE, Liu GT, Volpe NJ, Galetta SL. The Crossed Paralyses: The original brain-stem syndromes of millard-gubler, foville, weber and raymond-cestan. Arch Neurol. 1995;52(6):635–638.

21. Gardeła G, Kuśmiderski J, Słowik S. Millard-gubler syndrome in a young man after hemorrhage from arteriovenous hemangioma to the brain stem. Neurologia i Neurochirurgia Polska. 1989;23(2):149-152.
PMID: 2615889.
22. Satake M, Kira J, Yamada T, Kobayashi T. Raymond syndrome (alternating abducent hemiplegia) caused by a small hematoma at the medial pontomedullary junction. J Neurol Neurosurg Psychiatry. 1995;58(2):261.
DOI: 10.1136/jnnp.58.2.261.
PMID: 7876876.
PMCID: PMC1073342.
23. Richard J. Madonna, Shruti Pandya, The Millard-gubler syndrome, Clinical Eye and Vision Care.1995;7(1)33-38.
ISSN 0953-4431.
24. Dominika Dulak, Imama A. Naqvi. Neuro anotamy cranial nerve 7 July 24;2023.

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