

## A Rare Case Report of A Patient With Weber's Syndrome Landed Into Millard–Gubler Syndrome.

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**Abstract:** This is a case report of a lady who presented with Weber's syndrome. She had left oculomotor nerve palsy and right hemiparesis. The patient subsequently made a good recovery, after 1 year same patient landed into millard-gubler syndrome now she had ipsilateral paralysis of the abducent and facial palsy and contralateral hemiplegia of the extremities.

### I. Introduction

A complete oculomotor (3rd) nerve lesion results in ipsilateral ptosis, pupillary dilatation, loss of pupillary and accommodation reflexes and lateral deviation of the eye.[1] If the 3rd nerve palsy is associated with contralateral hemiplegia, the condition is described as Weber's syndrome.[2] However, this syndrome may be reversible as described in this report. After 1 year same patient landed into millard-gubler syndrome, it is characterized by ipsilateral facial palsy probably owing to involvement of the root fibers and contralateral hemiplegia resulting from involvement of the corticospinal fibers. This is the first case report of pure Weber's syndrome ultimately landed into millard-gubler syndrome

### Case Report

A 68-year-old lady noticed weakness of the right arm and leg and diplopia on waking in the morning. She had been a diabetic and hypertensive for the past 20 years. Her medications included gliclazide, metformin, insulin, amlodipine, telmisartan and aspirin.

On presentation, she had a right hemiparesis and a left 3rd nerve palsy (drooping of eyelid/ lateral deviation of the eye/ diplopia on looking to the right) without involvement of the pupils. Higher mental functions, Sensory system and Cerebellar signs were intact. A Computerized Tomograph (CT) scan of the brain showed possible lacunar infarcts in the basal ganglia. Over the next week her hemiparesis resolved completely and her diplopia was getting better. Two weeks later, she had no more diplopia and was discharged with no neurological sequel. She subsequently had a Magnetic Resonance Imaging (MRI) of the brain which established the diagnosis. The MRI showed numerous small T2W signal hyperintensities within the cerebral white matter and the lower mid-brain, consistent with infarcts. The diagnosis of Weber's syndrome without pupillary involvement was made on the basis of a crossed hemiplegia: left 3rd nerve palsy and right hemiparesis. Now after 1 year same patient developed a throbbing headache and showed paresthesia on the left side of the body, vertigo and right facial weakness. On admission, her blood pressure was 188/100, she was alert and intelligence was normal, she showed a peripheral facial nerve palsy on the right left sided hemiparesis, deep tendon reflexes are exaggerated and Babinski's reflex was equivocal on left side. Objective sensory loss and cerebellar ataxia was not seen, routine blood and urine examinations were normal. T2 weighted MRI showed a high-signal area in right ventral pons. A ventral arteriogram showed occlusion of both vertebral arteries.

### II. Discussion

Weber's syndrome was described in 1863 by the German physician Hermann Weber.[2] He described a 52-year-old man who presented with right limb weakness and left oculomotor palsy (involving the pupils) caused by a hemorrhage in the left cerebral peduncle. The clinical findings of Weber's syndrome include an ipsilateral 3rd nerve palsy and a contralateral limb weakness due to a lesion in the mid-brain (crus cerebri). Subsequently, cases have been reported wherein mid-brain lesions can produce a 3rd nerve lesion sparing the pupils, be it due to infarct, tumor or bleed.[3],[4],[5],[6]

The 3rd nerve nuclei are located in the mid-brain and are approximately 10 mm in length from the rostral to caudal extent.[3] It consists of: (a) the Edinger-Westphal nucleus located in the upper mid-brain supplying fibers to the pupils and (b) the motor nucleus located in the lower mid-brain supplying the extra-

ocular muscles, except the lateral rectus and the superior oblique.[7] Fascicles from the nuclei run forward and laterally through the red nuclei and converge at the inter-peduncular fossa before emerging from the mid-brain. As the nuclei and fascicles are spread across a relatively wide area, mid-brain lesions can lead to partial 3rd nerve lesions. Hence a lesion of the lower mid-brain affects the extraocular muscles but spares the pupils, whereas lesions involving both the upper and lower parts of the mid-brain are associated with pupillary dilatation.

Millard–Gubler syndrome is named after two French physicians, Auguste Louis Jules Millard (1830–1915), who first identified the disorder in 1855, and Adolphe Marie Gubler (1821–1879), who described the disease in a medical paper one year later as a lesion of the pons. It is also called ventral pontine syndrome.<sup>[1]</sup> Symptoms result from the functional loss of several anatomical structures of the pons, including the sixth and seventh cranial nerve and fibers of the corticospinal tract. Paralysis of the abducens (CN VI) leads to diplopia, internal strabismus (i.e., esotropia), and loss of power to rotate the affected eye outward, and disruption of the facial nerves (CN VII) leads to symptoms including flaccid paralysis of the muscles of facial expression and loss of the corneal reflex. Disruption of the corticospinal tract leads to contralateral hemiplegia of the extremities. This syndrome is easier to diagnose today thanks to the technical advances in brain imaging (CT, MRI). It can also be identified based on the symptoms described above.

In this case, the sudden onset of left 3rd nerve palsy and contralateral hemiplegia indicates a mid-brain vascular lesion. There was a difference between the CT scan and the MRI scan findings which reflects the better sensitivity of the MRI scans to detect lesions of the brainstem. The pathological lesion in this patient indicating Weber's syndrome was shown on the MRI scan as an ischemic lesion in the lower part of the mid-brain where the motor part of the 3rd nerve nucleus is located, whereas there was no pathological lesion in the upper mid-brain where the Edinger-Westphal nucleus is found. Although the MRI scan showed multiple infarcts in the cerebral white matter, they were probably due to her long-standing diabetes and hypertension and these were not associated with functional or neurological deficits.

Finally, this report demonstrates a crossed hemiplegia with 3rd nerve palsy (pupil-sparing) due to an infarct in the lower part of the mid-brain as documented by the MRI scan. The other interesting feature to note in our report is that the patient recovered completely and was discharged with no neurological sequel. This indicates that some of these patients may have a good prognosis. Patient recovered completely but left his medications after 6 months. Now patient presented to us as a case of Millard-Gubler syndrome this is the first case report of pure Weber's syndrome ultimately landed into Millard-Gubler syndrome.

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