



A Case of Wernekink Commissure Syndrome

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This is a case of a lady who presented with slurring of speech and swaying while walking for 3 days. She is a known case of Diabetes Mellitus, Hypertension and dyslipidemia and is on treatment for it. Examination showed left side Internuclear ophthalmoplegia and bilateral cerebellar ataxia. MRI showed FLAIR hyperintensity in tegmentum of left caudal midbrain showing diffusion restriction. The patient was diagnosed with Wernekink commissure syndrome based on the clinical features and MRI findings. The patient was started on antiplatelets along with other treatment. The point of interest in this case is it is an extremely rare disease with only few case reports reported and its presentation with bilateral cerebellar symptoms makes it extremely difficult to localize the lesion. Unilateral INO in some cases may help to narrow down the differentials.

Keywords: Wernekink Commissure; INO; Cerebellum; MRI**Introduction**

Lhermitte first coined the term Wernekink commissure syndrome [2]. Decussation of superior cerebellar peduncle was first described by Friedrich Christian Gregor Wernekink and was named as the horseshoe-shaped commissure of Wernekink by his student Franz Joseph Julius Wilbrand [3]. Only a few cases have been reported till now. The symptoms occur due to damage to the decussation of superior cerebellar peduncles [1]. It is associated with bilateral cerebellar signs. Eye movement may be affected due to the damage of nearby medial longitudinal fascicle. Wernekink commissure is located anterior to the aqueduct and at the paramedian region of caudal midbrain. Commissure involves decussation of dentatorubrothalamic pathway which provides cerebrocerebellum connections through superior cerebellar peduncle in midbrain.

Objective

- To describe the symptoms, signs and investigation findings of Wernekink commissure syndrome
- To aid in the early diagnosis and localization of a patient presenting with bilateral cerebellar signs and symptoms, thereby increasing the quality of life of the patient.

Clinical scenario

A 59-year-old lady, with Diabetes Mellitus on Insulin and Oral antidiabetic drugs, Hypertension and Dyslipidemia on treatment presented with slurring of speech and associated swaying or imbalance while walking. There was no history suggestive of any head trauma, seizures, fever or loss of consciousness. There was no history of any sensory symptoms.

On examination patient was conscious and oriented. Dysarthria was present. Diplopia was present on rightward gaze. Left eye had adduction defect and Right eye showed horizontal nystagmus- features suggestive of left INO. Convergence was preserved. Power of bilateral upper and lower limbs was normal. However bilateral finger nose finger test, heel shin test was impaired on both sides (Left more than right). Bilateral dysdiadochokinesia was present. There was swaying to either side while walking. Tandem walking was impaired. All features were suggestive of bilateral cerebellar ataxia. The patient did not have any palatal tremor.

Routine blood investigations were within normal limits, except for deranged lipid profile and high HbA1c of 9.9 %.

MRI Brain FLAIR showed hyperintensity in tegmentum of left caudal midbrain showing diffusion restriction (Figure 1, 2, 3) [7-9].

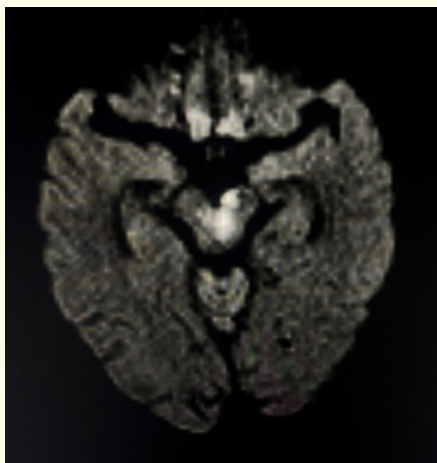


Figure 1: MRI Brain DWI [7].

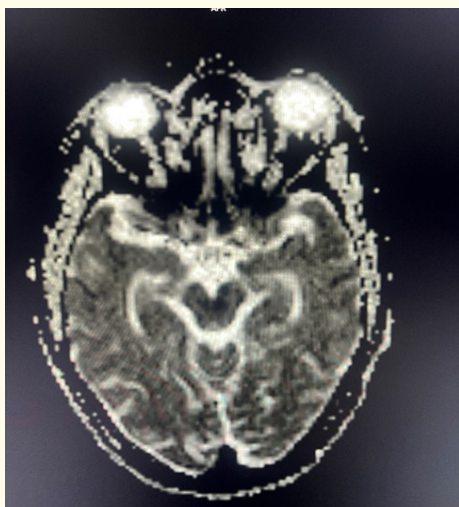


Figure 2: ADC [8].

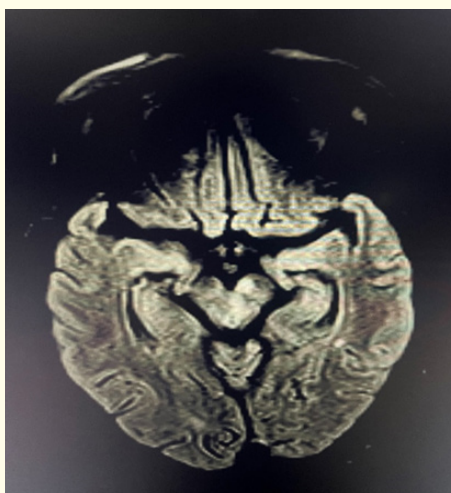


Figure 3: MRI FLAIR [9].

Patient was diagnosed as Wernekink commissure syndrome based on clinical features and investigation findings.

Discussion

Exact details regarding incidence and prevalence of Wernekink commissure syndrome are not available. Literature regarding Wernekink commissure syndrome mostly exists in the form of case reports in adult population.

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Wernekink commissure syndrome is characterized by bilateral cerebellar ataxia and eye movement defect [1]. The neurological defects are due to disruption in superior cerebellar peduncle, medial longitudinal fascicle and central tegmental tract [4]. Disruption of dentato-rubro-olivary and dentato-olivary tract may lead to hypertrophic olivary degeneration which may result in palatal tremor [5,6].

The Wernekink commissure is supplied by inferior paramedian mesencephalic arteries (a subset of interpeduncular fossa perforating branches that usually arise from the tip of the basilar artery, superior cerebellar artery and/or P1 segment of the posterior cerebral artery [2].

They can present as gait or truncal ataxia, dysmetria on finger nose test and heel shin test and dysdiadochokinesis. Marked dysarthria may be present. Eye movement findings like internuclear ophthalmoplegia and palatal tremors are also described.

Treatment and outcome

The patient was not a candidate for thrombolysis and was admitted for observation and medical management. The patient was treated with dual antiplatelets- Aspirin 150 mg per day and Clopidogrel 75mg per day along with high dose statin. Ophthalmology and Cardiology evaluation was done. Strict glycemetic control along with other supportive management was given. Over the course of stay in hospital, the patient became better, Gait improved. She was discharged and kept under follow up.

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