Case Report

Wernekink Comissure Syndrome: a rare midbrain syndrome secondary to stroke

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Abstract

Wernekink commissure involves the decussation of superior cerebellar peduncle (SCP) in midbrain. We report an elderly hypertensive, diabetic man who developed slurred speech, ataxia, and internuclear ophthalmoplegia. MRI examination revealed an unusual ischemic stroke involving Wernekink commissure. This rare stroke pattern involving decussation of SCP occurs in the setting of small arterial disease. The association between the anatomic location of the stroke and clinical findings is noteworthy.

Introduction

The midbrain is a common site for stroke lesion. The broad clinical spectrum of signs and symptoms is observed. Moreover, the arterial blood supply to the midbrain is complex. There are overlaps between arterial territories and individual variations. The arterial system supplying the midbrain is also terminal. There is much variation and numerous classifications of the blood supply to the brain stem.[1,2]

Limb movements, consciousness, cognition and oculomotor capacities are the functions mainly involved in midbrain strokes and their combinations tend to be characteristic. Other neuro-opthalmological features, including skewed deviations, tonic ocular-tilt reactions, intermittent corectopia, internuclear ophthalmoplegia, seesaw nystagmus have been reported.[3] Involvement of the thalamo-mesencephalic artery can be associated with behavioural and neuropsychological disturbances. Subthalamic small deep infarcts can be associated with abnormal movements and asterixis.[4] Delayed athetoid or clonic movements may occur.[5] Rarely infarcts restricted to the upper midbrain may give rise to peduncular hallucinosis. The term hallucinosis is used in order to distinguish these features from the hallucinations occurring in patients with delirium. In peduncular hallucinosis, the patient often presents with abnormal perceptions and hallucinations without any evidence of a psychiatric disorder.[6,7]

We report a rare midbrain stroke, Wernekink commissure syndrome which has been characterized by bilateral cerebellar syndrome, occasionally associated with ocular motor sign or internuclear ophtalmoplegia. Distribution of the lesion is at Wernekink commissure.[8]

Case Report

A seventy-year-old male, having history of diabetes mellitus, hypertension, peripheral vascular disease and cataract, was admitted to stroke service for sudden onset poor balance and slurred speech. He denied any nausea, vomiting or vertigo. On arrival to the emergency, his vital signs were normal except a blood pressure of 160/70 mmHg. He was alert and oriented but speech was markedly dysarthric and he had internuclear ophtalmoplegia on left side. He also had an involuntary clonic type intermittent jaw opening movements. Power was normal, but there was markedly diminished fine finger movement on left side and poor coordination bilaterally, more in the left hand. Sensation was normal. He had marked truncal ataxia and was unable to walk without assistance. CT brain CT showed haemorrhage in midbrain. (Figure 1A)

Stroke work- up was negative except cholesterol of 271 mg/dl. The MRI confirmed a stroke just below the brachium conjunctivum. (Figure 1B) These symptoms were felt to be due to the disruption of the Wernekink commissure.

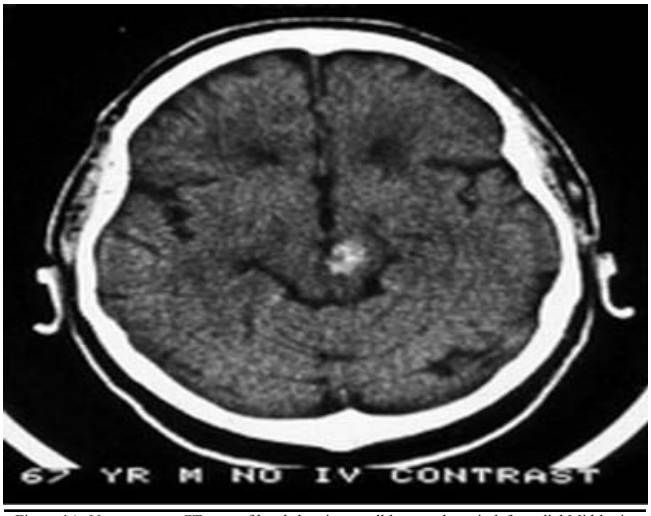

Figure 1A. Non- contrast CT scan of head showing small haemorrhage in left medial Mid-brain.

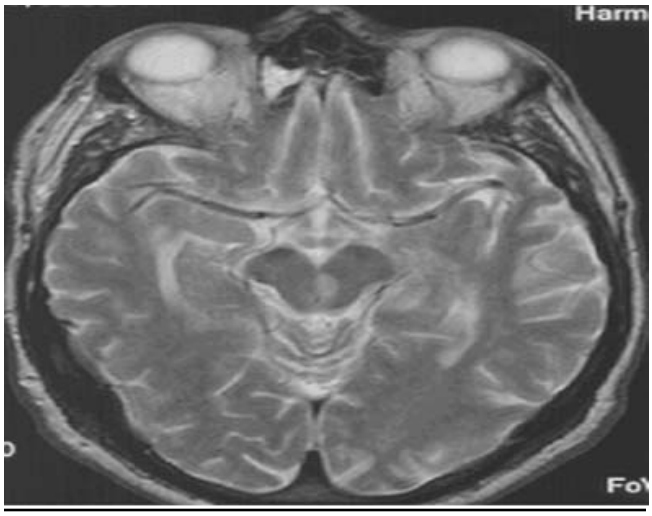

Figure 1B. Proton density image in axial plane showing infarction in medial mid brain.

Following day, patient became very confused and disoriented and started having hallucinations. He thought that he was at school and small children were crowding in his room. The hallucinations were mostly visual but not auditory. His cognition, speech and gait began to improve gradually after two weeks.

Discussion

The Wernekink commissure syndrome is extremely rare and first described by Lhermitte in 1958.[8] It is characterized by bilateral cerebellar syndrome and occasionally associated with internuclear ophtalmoplegia or ocular signs. It may be due to disruption of the Wernekink commissure. No case report has been reported since 1958. This may be a common sign in multiple sclerosis but it is not usually seen in stroke cases. We report the second case in the literature.

Midbrain lacunar strokes have a wide spectrum of clinical expressions and they are sometimes difficult to diagnose. Ataxia of the limbs related to cerebellar infarct in the territory of the superior cerebellar artery can be associated with various combinations of rostral midbrain syndromes. If the territory of the thalamo-mesencephalic artery is involved, akinetic mutism, disorientation to time and place, memory disturbance (anterograde amnesia) are frequent. The midbrain signs can be due to either emboli fragmenting into the rostral basilar artery or to infarction of the midbrain territory of the superior cerebellar artery.[9] In this case, lesion most likely related to paramedian small arterial lacunar stroke. Extensive cardiac and arterial examinations must be carried out in these patients, using the same techniques as in carotid-territory infarcts.

References

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