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Case Report

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Wernicke encephalopathy presenting with rare clinical findings: bilateral VI cranial nerve palsy and pontine lesion

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ABSTRACT

Wernicke encephalopathy (WE) is a serious acute neurological condition that is commonly associated with vitamin B1 (thiamine) deficiency due to alcohol abuse, albeit it can also occur in non-alcoholic settings. In cases with thiamine deficiency, WE is diagnosed by the presence of at least two of the classic clinical findings, including ophthalmoplegia, ataxic gait, and confusion. In this case report, we present a rare clinical manifestation of WE involving bilateral sixth cranial nerve palsy, ataxia, and pontine involvement on magnetic resonance imaging (MRI). A 36-year-old male patient presented with a five-day history of diplopia and a one-month history of hallucinations and insomnia. His medical history revealed chronic alcohol dependence, including recent consumption of homemade alcohol prior to the onset of symptoms. Neurological examination showed restricted lateral gaze in both eyes and an ataxic gait. Brain MRI (T2-weighted axial sections) demonstrated a hyperintense lesion in the midline of the pons. The patient was started on thiamine therapy at a dose of 500 mg/day for five days, followed by maintenance therapy with 200 mg/day for five days and then 100 mg/day for an additional five days. On neurological examination prior to discharge, right eye movements had normalized, while restricted lateral gaze persisted in the left eye. MRI findings in WE include involvement of the ventral thalamus, hypothalamus, mammillary bodies, periaqueductal region, and the floor of the fourth ventricle. Early parenteral thiamine administration remains the cornerstone of treatment. Our patient presented with bilateral sixth cranial nerve palsy and ataxia, along with a hyperintense signal in the midline of the pons on T2-weighted brain MRI. This case has been presented to highlight a rare clinical presentation of WE.

Keywords: Wernicke encephalopathy, diplopia, pontine lesion

INTRODUCTION

Wernicke encephalopathy (WE) is a serious acute neurological condition that is commonly associated with vitamin B1 (thiamine) deficiency due to alcohol abuse, albeit it can also occur in non-alcoholic settings.[1] While the prevalence of WE is estimated at approximately 2% in the general population, it is often underdiagnosed and undertreated. Reports suggest that up to 80% of cases are not identified until postmortem examination.[2,3] In cases with thiamine deficiency, WE is diagnosed by the presence of at least two of the classic clinical findings, including ophthalmoplegia, ataxic gait, and confusion. WE typically develops 4 to 6 weeks following the onset of thiamine deficiency, and the classic triad is observed in only 16–33% of patients during the early phase.[1] Timely recognition and treatment initiation are crucial. Untreated WE may result in irreversible brain damage or death.[3] In this case report, we present a rare clinical manifestation of WE involving bilateral sixth cranial nerve palsy, ataxia, and pontine involvement on magnetic resonance imaging (MRI).

CASE

A 36-year-old male presented with a five-day history of diplopia and a one-month history of hallucinations and insomnia. His medical history revealed no chronic illnesses. He had a history of chronic alcohol dependence and reported regular alcohol consumption on a weekly basis for the past 15 years, including recent intake of homemade alcohol prior to the onset of symptoms. On neurological examination, the patient was alert, cooperative, and oriented. Speech was normal, and there was no nuchal rigidity. Pupils were isocoric with normal light reflexes (+/+), and bilateral restricted lateral gaze was observed. There was no facial asymmetry; muscle strength was preserved; bilateral plantar reflexes were flexor. The patient's gait was ataxic. Cranial computed tomography (CT) and diffusion-weighted magnetic resonance imaging (MRI) revealed no abnormalities. Axial T2-weighted MRI images of the brain showed a hyperintense lesion in the midline of the pons (Figure 1, 2). The patient was admitted to the ward with a prediagnosis of WE. The patient was

started on thiamine therapy at a dose of 500 mg/day for five days, followed by maintenance therapy with 200 mg/day for five days and then 100 mg/day for an additional five days. On neurological examination prior to discharge, right eye movements had normalized, while restricted lateral gaze persisted in the left eye. The patient was referred to the physical therapy and rehabilitation clinic, where balance training was initiated to address ataxic gait. A psychiatric consultation was also obtained, and mirtazapine 15 mg once daily was added to the treatment regimen for insomnia. The patient was discharged on the 11th day with continued maintenance thiamine therapy.

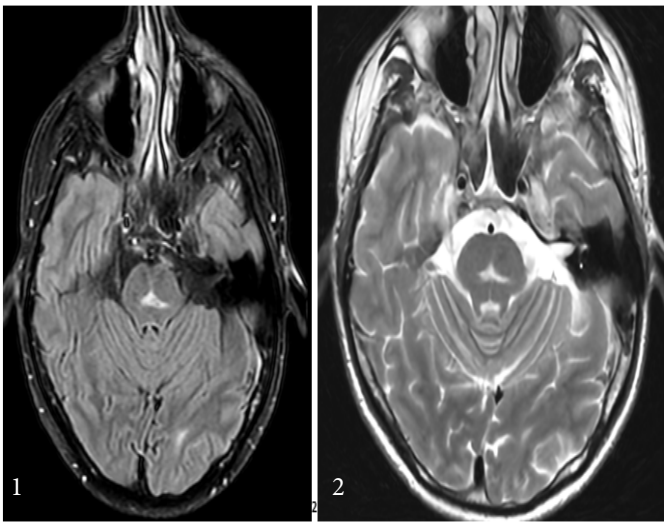

Figure 1, 2: Axial T2-weighted MRI images of the brain showed a hyperintense lesion in the midline of the pons

DISCUSSION

Although alcoholism accounts for approximately half of all cases and is the most common cause of thiamine deficiency in WE, other causes include hyperemesis gravidarum, uremia, starvation, hemodialysis, and a history of gastric surgery.[4] In our case, the patient had a history of chronic alcohol use. During the acute phase of WE, characteristic symptoms include mental status changes, ophthalmoplegia, and ataxia, while the chronic phase may involve Korsakoff syndrome (KS), which is characterized by amnestic disorders and confabulations.[5] Kuzume et al.[6] reported a rare case of WE diagnosed with bilateral sixth cranial nerve palsy, in which neurological symptoms improved following thiamine treatment. Similarly, our patient was diagnosed based on bilateral sixth cranial nerve palsy and ataxia, and symptom improvement was observed with thiamine therapy. MRI has a sensitivity of 53% and a specificity of 93% in the diagnosis of WE and is considered the standard imaging modality for confirming the diagnosis. On T2-weighted MRI, WE is suggested by hyperintense lesions in regions associated with high carbohydrate metabolism, most notably the ventral thalamus, hypothalamus, mammillary bodies, periaqueductal area, and the floor of the fourth ventricle.[7,8] Additional areas of involvement may include the cerebellum, dorsal medulla, pons, cranial nerve nuclei, red nucleus, dentate nucleus, putamen, caudate nucleus, fornix, splenium of the corpus callosum, and pre- and postcentral gyri.[7] In our case, a hyperintense signal change was observed in the midline of the pons on T2-weighted sequences. In a study involving 41 WE patients, MRI was performed on 36, and one-third of them demonstrated T2/FLAIR hyperintensities suggestive of WE. A normal MRI does not exclude the diagnosis of WE.[8] Early parenteral thiamine administration remains the cornerstone of treatment. The literature generally recommends intravenous thiamine at doses ranging from 200 to 500 mg every eight hours for at least several days; however, there is no definitive evidence regarding the optimal dose or duration of treatment.[9]

CONCLUSION

Our patient presented with bilateral sixth cranial nerve palsy and ataxia, along with a hyperintense signal in the midline of the pons on T2-weighted brain MRI. Parenteral thiamine therapy was initiated in accordance with approaches recommended in the literature, and improvement in ophthalmoplegic symptoms was observed. This case has been presented to highlight a rare clinical presentation of WE.

ETHICAL DECLARATIONS

Informed Consent

The patient signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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