Case Report

Wernicke's encephalopathy- a case report

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Abstract

Wernicke's encephalopathy is a neuropsychiatric disorder characterized by acute onset, nystagmus and oculomotor abnormalities, and a confusional state. Chronic alcohol intake is still the most common reason. Wernicke's encephalopathy, if not recognized and treated, can become irreversible. Common findings in Magnetic Resonance Imaging (MRI) include: symmetric T2 Weighted Image (T2) hyperintensities in peri-aqueductal gray matter, dorsal medial thalamus and mammillary bodies. This case highlights neurological deficits, persistent memory and disorientation.

Key words: Alcohol use, Wernicke encephalopathy, MRI findings

Introduction

Wernicke, in 1881, had first descried this condition in two chronic alcoholics and one case of persistent vomiting after sulfuric acid poisoning.[1] Although it can occur in non-alcoholic cases, long term alcohol use is the commonest cause. The classic triad of confusion, ataxia and oculomotor abnormalities may be present in one third of the cases only.[2] In those cases where typical signs and symptoms are not present, lately, MRI findings are reported to be useful in the diagnosis.[3] Given some variations in presentation and persistent of memory symptoms, it is hoped that this case will help in early identification and treatment of such cases.

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Case report

A 52 years male presented in emergency department with visual hallucination, persecutory idea, restlessness and non-fluctuating disorientation for 4 days. He had multiple episodes of vomiting 9 days back for 2 days. He had weakness in lower limbs, inability to walk, ataxic gait and forgetfulness for last three months. He was mostly bed-ridden and he complained of double-vision, dizziness and burning or tingling sensation of lower limbs.

He had history of alcohol consumption for last 25 years with average daily consumption of more than 1 liter of alcohol. He demonstrated craving, tolerance, loss of control, withdrawal features at least for last 2 years. There was no history suggestive of complicated withdrawal. The food intake in last one year was significantly decreased.

During admission, vitals were stable with normal systemic examination. He showed mild intentional tremor, symmetrical mild weakness, diminished deep tendon reflexes in all limbs, horizontal nystagmus, impaired finger-nose and heel-shin test, and dysdiadokinesia. Cranial nerves and sensory test were within normal limit. He was confused at the time of presentation but no fluctuating sensorium. Concentration was impaired.

Disorientation to time and place was present. Memory was impaired. He had poor insight. Signs of alcohol withdrawal were not noticed.

His liver enzymes were elevated including gamma glutamyl transferase (504 U/l).

His magnetic resonance imaging of brain showed 'symmetrical area of T2 and Fluid-attenuated Inversion Recovery (FLAIR) hyper-intensity in bilateral mammillary bodies, medial thalami and peri-aqueductal grey matter with mild diffuse brain atrophy'.

He was admitted in psychiatry department and treated with parental thiamine 500 mg a day for two days followed by 100 mg three times a day, along with intravenous fluids. The nystagmus, double-vision, in-coordination, weakness of lower limbs, ataxia improved but memory problems, disorientation and loss of insight remained.

Discussion

Alcohol does not have thiamine although it has some calories. Moreover, alcohol impairs absorption of thiamine, storage in liver, decreases its phosphorylation to its active metabolite and increases thiamine need for the metabolism of alcohol. With less food intake and no supplementation, the thiamine begins to get deficient. The body storage capacity for thiamine is 30-50 mg and with average daily need of 1-2 mg, it is expected to deplete in about a month. It has been argued, therefore, that some patients have already sustained irreversible brain damage at the time of presentation who progress to Korsakoff's syndrome[4] and it is likely that our patient fell into that category due to decreased food intake, continued alcohol drinking, and without thiamine supplementation for nearly a year.

Thiamine is an important co-enzyme for pyruvate dehydrogenase to be used in Krebs cycle. It is hypothesized that thiamine deficiency results in focal lactic acidosis and increased blood-brain-barrier permeability coupled with excitotoxic effects of N-methyl-D-Aspartate activation with resultant cell death, proliferation of astrocytes and activation of microglia causing symmetrical damage in the thalamus, mammillary bodies, cerebellum, and pons. These symmetrical features are commonly seen in MRI as hyperintensities

in these areas.[5] In a review of MRI findings in Wernicke's encephalopathy, the typical areas of T2 and FLAIR hyperintensities were reported to be thalami, mammillary bodies, tectal plate and peri-aqueductal regions.[3] In keeping with this, our patient's MRI findings revealed similar symmetrical area of T2 and FLAIR hyperintensities in bilateral mammillary bodies, medial thalami and peri-aqueductal gray matter.

The predisposing factors to thiamine deficiency are weight loss in past year, reduced body mass index, general clinical impression of nutritional status, high carbohydrate intake, recurrent episodes of vomiting in past month and co-occurrence of other nationally related conditions. The predisposing factors to neurotoxicity of alcohol are genetic predisposition to alcohol dependence, frequency of alcohol use, severity of dependence, frequent episodes of acute intoxication, withdrawal symptoms, concurrent use of cocaine and alcohol-related liver damage. And, the early signs and symptoms of thiamine deficiency were considered as loss of appetite, nausea/ vomiting, fatigue/ weakness/ apathy, giddiness/ diplopia, insomnia/ anxiety/ difficulty in concentration and loss of memory.[6] Our patient had general clinical impression of poor nutritional status, vomiting in last few days in predisposing factors to thiamine deficiency; all predisposing factors to neurotoxicity except concurrent use of cocaine and all symptoms described as early signs and symptoms of thiamine deficiency described above.

The operational criteria developed by Caine et al. (1997) for Wernicke's encephalopathy required two out of the four signs, namely: dietary deficiencies, oculomotor abnormalities, cerebellar dysfunction and either altered mental state or mild memory impairment.[7] Our patient exhibited the symptoms from all four domains. The frequencies of signs and symptoms of WE in different studies were summarized by Thomson et al.[6] The range of frequencies of signs were as follows: 34-100% for confusion, 12.5-37% for ataxia or staggering, 8-100% for nystagmus, 32-75% for apathy/ lethargy, 5-50% for disorientation, 4-16.5% for diplopia and oculomotor abnormalities except ophthalmoplegia and 12.5-52% for peripheral neuropathy. Another study noted horizontal nystagmus in 18%, cerebellar ataxia in 21% and absence of deep tendon reflexes in eighty two percent.[8] Among the symptoms described, our patient had confusion, lethargy, loss of appetite, horizontal nystagmus, diplopia, diminished tendon reflexes, cerebellar signs, hallucination, impaired concentration, disorientation and impaired

memory, however with no ophthal-moplegia and cranial nerve involvement.

Conclusion

This case highlights decreased food intake for a year with continued drinking; development of the gradually increasing weakness, unsteadiness, and memory difficulties; precipitation of Wernicke's encephalopathy by vomiting; persistence of memory dysfunction, poly-neuropathy, and disorientation despite parenteral thiamine supplementation.

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