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A Case Report on Wernicke–Korsakoff Syndrome

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Abstract

Background: Alcohol use is one of the most serious problems in public health and the Wernicke–Korsakoff Syndrome is one of the gravest consequences of alcoholism. Post–mortem studies suggest that Wernicke–Korsakoff Syndrome occurs in 12.5% of dependent drinkers and in 2% of the general population. Korsakoff Syndrome is an amnestic disorder generally followed by untreated Wernicke's Encephalopathy. Wernicke–Korsakoff Syndrome is most commonly a post–mortem diagnosis. **Aim and Objective:** To report a case of Korsakoff Syndrome since the clinical presentation is often undiagnosed or misdiagnosed. **Case description:** 50 year old male, drinking heavily since 25 years presented with complaints of forgetfulness, talking irrelevantly since last 18 months. Patient developed symptoms of confusion, ataxia, and altered behaviour 2 years back, for which he was treated as a case of encephalitis and not treated with thiamine. He later progressed to show symptoms of amnesia. On examination, patient showed recent memory deficit with anterograde and variable retrograde amnesia with confabulations. Neurological examination revealed absent deep tendon reflexes and signs of peripheral neuropathy. MRI brain showed global cortical atrophy. The sequence of events in this case study demonstrates the possible effects of long term alcohol use, namely Wernicke–Korsakoff Syndrome. Highlights of the medical model of Wernicke–Korsakoff Syndrome will be subsequently presented. Lastly, suggestions for treatment and prevention of further damage will be discussed.

Keywords: Chronic Alcoholism, Wernicke's Encephalopathy, Amnestic Syndrome, Thiamine Deficiency

1. Introduction

Alcohol use is one of the most serious problems in public health and the Wernicke–Korsakoff Syndrome is one of the gravest consequences of alcoholism. Post–mortem studies suggest that Wernicke–Korsakoff syndrome occurs in about 2% of the general population and 12.5% of dependent drinkers[1]. Korsakoff Syndrome is an amnestic disorder which generally follows untreated Wernicke's encephalopathy. Wernicke–Korsakoff Syndrome is most commonly a post–mortem diagnosis and it is often mislead[2].

2. Case

50 year old male, Hindu, married, studied up to 2nd standard, utensil vendor by occupation was admitted under the care of Department of Psychiatry with chief complaints of forgetfulness, tendency to falsify. These complaints were present since 18 months. Patient was consuming alcohol from past 25 years. Initially he started drinking around 90 ml of country made liquor per day for around 1 year; this quantity later increased to around 180 ml/day. His consumption increased to 540–720 ml per day. He attempted to abstain from alcohol on several occasions during which he experienced symptoms of tremors and palpitations. Around 2 years back he developed complaints of altered sensorium, ataxia and confusion. He was admitted in a private hospital where he was treated with antiviral drugs and there is no report of thiamine supplement given. After discharge, his condition worsened and he developed symptoms of forgetfulness. He was unable to recall who had visited him

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a day before and could not remember their names. He did not remember whether he had taken a bath or what what he had eaten for breakfast and on enquiry, he would give false answers. Whenever his wife or relative would try to talk to him, he would be inattentive towards them. When asked about recent events, he would give irrelevant answers, would repeat the same thing again and again. Significant family history shows alcohol dependence of his father who expired 20 years back because of alcohol related complication. History of alcohol dependence was seen in both elder brothers and two elder sons of the patient. Relatives denied of any other psychiatric illness in family.

2.1 On General Examination

Conscious, co–operative, oriented. Pulse 72/min regular. Blood Pressure: 120/78 mm Hg in RT upper arm in sitting position. Respiratory rate: 16/min. Temperature: 98 degree Fahrenheit. No cyanosis, clubbing pallor icterus or lymphadenopathy. No obvious signs of liver failure.

2.2 On Examination CNS

Examinations of all cranial nerves were within normal limits. Nutrition in all limbs was adequate; power was 5/5 in all four limbs. Tone was normal in all four limbs. Deep tendon reflexes were absent in all the limbs, no involuntary movements present, hypoesthesia was noted on both lower limbs. Cerebellar signs within normal limits. There were no signs suggestive of meningitis. Rest of systemic examination was normal.

2.3 Mental Status Examination

He was in clear sensorium, however could not sustain attention. He was well oriented to time, place and person. Memory: Registration 5/5, Recall 2/5, impairment in recent and immediate memory with predominant anterograde amnesia with islands of preservation and tendency to confabulate.

2.4 MMSE Score

20/30(according to education of patient, the cut off for MMSE is 21)[3].

2.5 Investigations

Routine and blood investigations were within normal limits.

2.6 CT and MRI

Generalized cerebral atrophy, wider third ventricles, and significantly larger lateral ventricles (Figure 1).

2.7 Diagnosis

Patient was diagnosed as a case of Amnestic Syndrome (Korsakoff syndrome), according to ICD 10 Classification with toxic nutritional neuropathy.

3. Discussion

Thiamine deficiency often leads to Wernicke's encephalopathy which is associated with high morbidity and mortality. It is generally underdiagnosed. Diagnosis frequently occurs during post mortem examination. It occurs in about 2% of the general population and 12.5% of dependents[1]. It is important not only to correct the thiamine and magnesium deficiencies, but also to correct all other nutritional deficiencies in order to give the patient the best opportunity to recover normal brain function. In view of the diagnostic difficulties, clinicians should have a low threshold for making a 'presumptive' diagnosis of Wernicke 's encephalopathy. It is better to give too much thiamine too soon than to give too little too late. All clinical signs and symptoms are rarely present in Wernicke 's encephalopathy, hence a high index of suspicion needs to be maintained at all times. Variable doses

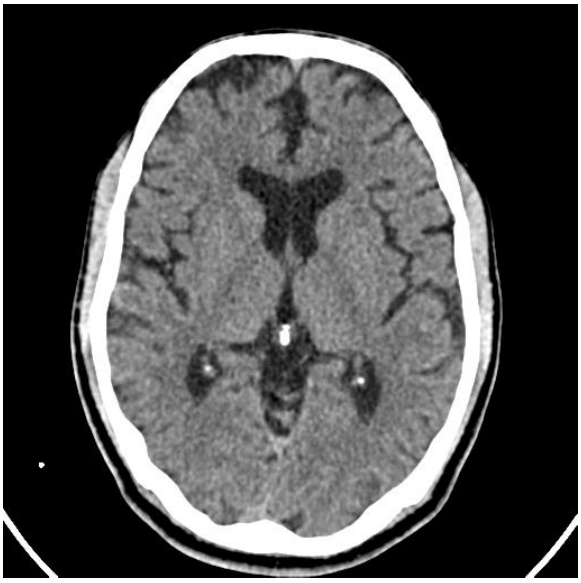

Figure 1. CT scan image of the patient showing generalized cerebral atrophy.

of thiamine are recommended to prevent Wernicke's encephalopathy. For example: heavy drinkers who are otherwise healthy can be given oral thiamine of about 300 mg/day during detoxification; Patients with high risk of Wernicke's Encephalopathy may be given 250 mg of thiamine parenterally once a day for three to five days and in a suspected or established cases of Wernicke's encephalopathy. Intramuscular or intravenous thiamine of more than 500 mg - three to five days depends on response[4].

According to a study conducted by Victor et al. in patients with Korsakoff Syndrome, 25% did not show any improvement, 50% improved over time while rest 25% showed recovery[5]. Some patients of Korsakoff may have a genetic predisposition. The characteristic neuropathology includes: (i) micro-haemorrhages, (ii) gliosis, and (iii) neuronal loss in the paraventricular and peri-aqueductal grey matter. Memory dysfunction can be attributed to lesions in the mammillary bodies, the mammillo–thalamic tract and the anterior thalamus rather than lesions in the medial dorsal nucleus of the thalamus. Korsakoff syndrome is characterised by severe impairment of episodic memory. The learning of new semantic memories is variably affected while implicit memory is preserved[6]. In a calm and well structured environment, Korsakoff patients are capable of learning new material, particularly if the new information is cued[7]. Very few long–term studies have been conducted in patients with Korsakoff Syndrome. However, the available data suggests that patients with Korsakoff Syndrome can have a normal life expectancy, provided they remain abstinent from alcohol[8].

4. References

1. Harper C. Thiamine (vitamin B1) deficiency and associated brain damage is still common throughout the world and prevention is simple and safe. Eur J Neurol: the official journal of the European Federation of Neurological Societies. 2006; 13(10):1078–1082.
2. Torvik A, Lindboe CF, Rogde S. Brain lesions in alcoholics: a neuropathological study with clinical correlations. J Neurol Sci. 1982; 56(2–3):233–248.
3. Crum RM, Anthony JC, Bassett SS, Folstein MF. Population–based norms for the mini–mental state examination by age and educational level. JAMA: J Am Med Assoc. 1993; 269(18):2386–2391.
4. Lingford–Hughes AR, Welch S, Nutt DJ. Evidence–based guidelines for the pharmacological management of substance misuse, addiction and comorbidity: recommendations from the british association for psychopharmacology. J Psychopharmacol. 2004; 18(3):293–335.
5. Victor M, Adams RD, Collins GH. The Wernicke–Korsakoff syndrome: a clinical and pathological study of 245 patients, 82 with post–mortem examinations. Contemp Neurol Ser. 1971; 7:1–206.
6. Fama R, Pfefferbaum A, Sullivan EV. Visuoperceptual learning in alcoholic Korsakoff syndrome. Alcohol Clin Exp Res. 2006; 30(4):680–687.
7. Lennane KJ. Management of moderate to severe alcohol–related brain damage (Korsakoff syndrome). The Med J Aust. 1986; 145(3–4):136, 141–143.
8. Kopelman MD, Thomson AD, Guerrini I, Marshall EJ. The Korsakoff syndrome: clinical aspects, psychology and treatment. Alcohol and alcohol. 2009; 44(2):148–154.