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Case Report

Alcohol Induced Cerebellar Ataxia: A Case Report

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ABSTRACT

Consumption of alcohol excessively causes dependence and leads to psychological discomfort. Motivation to drink is influenced by a variety of neurobiological and environmental variables. The proclivity of an individual to imbibe is thought to reflect a balance between alcohol's positive reinforcing (i.e., rewarding) effects, such as euphoria and anxiety reduction (i.e., anxiolysis), and the drug's aversive effects, which are typically associated with negative consequences of alcohol consumption (e.g., hangover or withdrawal symptoms). Neuroadaptive alterations caused by continuous alcohol use and abuse (such as tolerance and physiological dependence) are regarded to be critical in the shift from regulated alcohol use to more frequent and excessive, uncontrollable drinking which induces cerebellar degeneration considered as common type of acquired toxic ataxia. Excessive use leads to degeneration of the midline cerebellum. Progressive trunk and gait ataxia is characteristic, with little involvement of upper limbs, eyes or speech (a corollary of relative cerebellar hemispheric sparing). Abrupt termination of alcohol use after a period of heavy drinking may result in alcohol withdrawal seizures. Generalized tonic–clonic seizures are the most common and severe form of seizure in this conditions. Current case report presents complaints of astasia and slurred speech with few episodes of seizures. The diagnosis indicated Mild cerebellar atrophy. Patient was advised to undergo de-addiction and cure.

Key words: Cerebellar ataxia, Alcohol, Cerebellar atrophy, GABA, astasia,seizures.

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INTRODUCTION

Chronic alcohol abuse induces cerebellar degeneration considered as common type of acquired toxic ataxia. The onset of cerebellar deficit symptoms occurs in mid-age population with persistent abstinence in alcoholics.[1] Alcoholic cerebellar degeneration is characterized by ataxia of stance and gait, whereby a wide-based gait with shortened steps is adapted to compensate for the loss of balance and unsteadiness experienced. Dysdiadochokinesis, terminal dysmetria, and decomposition of movements are observed in varying degrees.

Speech would be slowed and slurred, however is not usually characterized by scamming (variability of pitch and loudness) and a horizontal nystagmus is sometimes present.[2]

This case report presents alcoholic patient with vulnerability of diffused cerebellar atrophy based on the normal neurological examination.

CASE DESCRIPTION

A 31year old male patient was admitted in general medicine ward with chief complaints astasia and slurred speech. Past history revealed that he is alcoholic from past 10 years with no history of medications for de-addiction and presence of 1 or 2 seizure episodes in last 2 years. Present illness showed difficulty in walk (wide-based gait), numbness of limbs, non-coordinated movement, insomnia, fear, anxiety due to this he was referred to psychiatry unit.

On examination patient was conscious, oriented to person, but not oriented to place and time. Psychomotor agitation is normal but tremors present. His blood pressure 120/70mmHg, pulse rate 80bpm, saturation 97% on RA, respiratory rate 18cpm, random blood sugar levels 297 mg/dl, central nervous system: higher mental function (HMF) is consciousness, not oriented, presence of ataxia gait, intellect was intact. His speech was coherent, irrelevant at time, dysphonia, dysarthria, dysphasia or aphasia events are observed. Also with intact cerebellar signs of ataxia with swaying bilaterally, dysmetria, Dysdiadochokinesis, dysesthesia, steppage gait, nystagymus.

During his laboratory investigations the RBC's were found to be 2.68×10^6/μL, Hb 8.2g/dl, HCT 23.5%, urea 30mg/dl, serum creatinine 0.7mg/dl, sodium 128mmol/L, potassium 3.3mmol/L, amylase 234U/L. Computed Tomography of brain was done which revealed mild diffuse cerebellar atrophy (Figure 1).

On the day of admission, patient was treated with Inj. Ceftriaxone 1g IV BD, Inj. Pantoprazole 40mg IV OD, Inj. Ondansetron 4mg IV SOS, IVF -2pint normal saline, 1 pint DNS with multivitamins supplements infusion rate at 75ml/hr, Inj. Vitcolol-C in 100ml NS OD, Inj. Thiamine 300mgIV stat, Tab B-Complex PO OD. Patient prognosis was getting better on day. Patient was stable and conscious, oriented at time of discharge.

DISCUSSION

Alcohol is a cause of late cortical cerebellar degeneration of the anterior lobe.[3] Alcoholic patients typically exhibit ataxia of the lower limbs, ataxia of gait, and trunk instability. Less frequent clinical findings include nystagmus, dysarthria and upper limb in-coordination.[4]

Ethanol might have induced cerebellar ataxia in our patient by its effects on γ-aminobutyric acid (GABA), N-methyl-D-aspartate (NMDA), or serotonin receptors. GABA is a major neurotransmitter in the cerebellum, and ethanol strongly acts on GABA-A receptors, modulating chloride flux. However, although the effects of alcohol on the CNS are better understood and a genetic inheritance might play a determinant part, it is still not clear why the spinocerebellum is selectively vulnerable.[5]

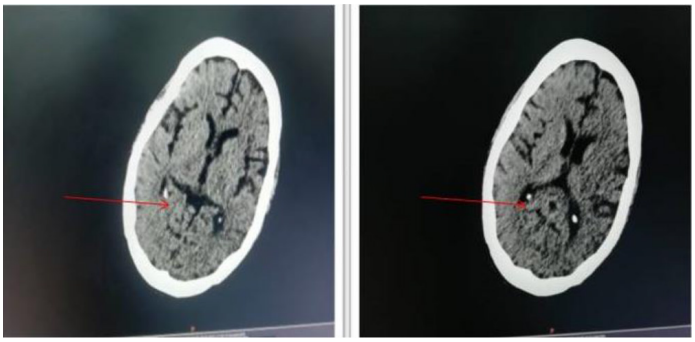

Figure 1: Alcohol induced Cerebral ataxia.

The acquired ataxia defines alcohol as major toxic cause of ataxia and excessive use leads to degeneration of the midline cerebellum. Progressive trunk and gait ataxia is characteristic, with little involvement of upper limbs, eyes or speech (a corollary of relative cerebellar hemispheric sparing).

Many neurobiological and environmental factors influence to drink an individual's propensity to imbibe is thought to reflect a balance between alcohol's positive reinforcing (i.e., rewarding) effects, such as euphoria and reduction of anxiety (i.e., anxiolysis), and the drug's aversive effects, which typically are associated with negative consequences of alcohol consumption (e.g., hangover or withdrawal symptoms).

Neuroadaptive changes that result from continued alcohol use and abuse (which manifest as tolerance and physiological dependence) are thought to be crucial in the transition from controlled alcohol use to more frequent and excessive, uncontrollable drinking.[6]

It is therefore essential that patients undergo an alcoholism cure. In addition vitamin B1/ thiamine were supplemented. In cases with a rapid onset of ataxia parenteral application of 100mg vitamin B1/thiamine is recommended for several days until supplementation is continued with an oral vitamin B1/ thiamine preparation.[7]

CONCLUSION

Ethanol causes severe damage to the cerebellum and it is important to identify potential neuroprotective agents to ameliorate ethanol toxicity. Ataxia progresses in alcoholism hence it is essential for the patient to undergo de-addiction and cure.

Patient Consent

Patient consent was taken for publishing his report without revealing his identity.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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