International Journal for Multidisciplinary Research (IJFMR)
E-ISSN: 2582-2160 ● Website: www.ijfmr.com ● Email: editor@ijfmr.com

Behavioural and Clinical Aspects of Chronic Alcohol Dependence Syndrome: A Case Report

Gannavarapu Kumara Swamy[1], Chinnapureddy Koushika[2], Bolneni Sathvika[3]

[1,2,3]Department of Pharmacy Practice, Malla Reddy Pharmacy College, Maisammaguda, Dhulapally, 500100.

ABSTRACT

Alcohol Dependence Syndrome (ADS) refers to a chronic progressive disease, which has several characteristics, including loss of control over consumption, tolerance, and withdrawal symptoms, as well as persistent habit despite adverse physical or social consequences. Alcoholism, in its more chronic forms, causes both physical and mental changes in the body. This includes withdrawal, nervousness, tremors, anxiety, and hallucination, as well as psychosomatic changes, which result in biochemical abnormalities in the organism. The following points will highlight a case presentation for a 33-year-old male patient who has been a chronic drinker for many years, with symptoms linked to ADS withdrawal seizure and psychiatric illness, as well as biochemical abnormalities stemming from hepatic changes brought about by alcohol intake in this person. Alcohol Dependence Syndrome, as well as Withdrawal Seizure, affects this person on numerous grounds.

KEYWORDS: Alcohol Dependence Syndrome, chronic alcoholism, withdrawal seizures, neurobiological adaptation, psychiatric comorbidity.

INTRODUCTION

The Alcohol Dependence Syndrome (ADS) is a progressive, relapsing, and chronic condition characterized by a loss of control of one's alcohol use, tolerance, withdrawal symptoms, or continued use of alcohol in the face of physical, mental, or social detrimental effects. ADS has been described by Edwards as a multidimensional dependence syndrome that has several components, which include craving, salience of drinking, loss of control, tolerance, and withdrawal, and these components may vary from person to person in their presence and intensity [1].

Worldwide, ADS continues to be underdiagnosed and diagnosed late in its stages. Epidemiological studies have indicated the contribution of alcohol dependence to the burden of disease as a significant public health concern because of its effects on the family, work, and community performance [3]. Ethanol biotransformation results in the formation of a toxic intermediate product called acetaldehyde, which facilitates heavy drinking behaviour along with inducing structural damage to the liver, indicated by high levels of liver enzymes [4].

Neurobiological changes that occur within rewarding circuitry, stress circuits, and inhibitory regulatory mechanisms contribute to compulsive drinking behaviours and enhance the vulnerability for recurrent episodes within patients with ADS [5]. Comorbid disorders like depression, anxiety disorders, impulsivity,

loss of judgment, and suicide have been often reported and exacerbate the condition, mainly when patients with ADS are intoxicated and undergoing withdrawal symptoms [6]. The factors associated with personality disorders, vulnerability for relapse, and chronic management needs were often recorded within patients with ADS [2, 7, 8, 9].

CASE PRESENTATION

The 33-year-old male patient was complaining of agitation, mumbling, and insomnia when brought to the psychiatry outpatient department [OPD]. He has been a chronic alcoholic for the past few years, but after an altercation with his brother-in-law three weeks ago, he attempted suicide three times. However, despite this, he reduced his liquor intake for a week before being brought to us. His previous intake was 180 milliliters, but for the past four years, it has gradually progressed to 750 milliliters. However, this liquor is taken in the first 180 milliliters in the morning, and the rest is taken whenever time is free. It was taken only two days ago, with an intake of approximately 90 ml. He has also been a chronic smoker for the past ten years. However, his intake is 1-2 cigars a day. He has also, over the past three days, turned to chewing tobacco instead of smoking it. His intake is approximately 25 gm. He is also complaining of irritation, headaches, heaviness in his head, and reduced appetite. However, apart from this, he is also complaining of seizures. However, these seizures have occurred under two circumstances. First, it was ten years ago, when he had a car accident, because of which he sustained injuries to his head. Secondly, it was at three in the morning, three weeks ago, for thirty seconds. It continued for 20 seconds for the third time, roughly a week ago. However, then his total body stiffened, and his eyes turned upward, but ever since then, for the past week, he has not suffered from any of his other usual episodes of losing his bladder, biting his tongue, foaming, or being mentally dazed after his seizure. He has his brother, father, and himself belonging to his family, wherein all three are his family members who are chronic alcoholics. However, his father is an additional member of his family who has tuberculosis. Owing to the higher intake of his favourite drinks, his lab work is abnormal, which shows that his liver is dysfunctional. According to his history, his direct bilirubin level is slightly higher, but his Gamma-Glutamyl Transferase [GGT] level is substantially higher, with his Aspartate Aminotransferase [AST] level being moderately higher as well. If his blood is tested, then this too shows substantially the same, whereby this too shows substantially the same phenomenon, which has been witnessed, that is, observed, under dependence syndrome. He was checked for tremors and then sent for admission for further tests. Pharmacologic therapy was started with Tab. Librium 25 mg in divided doses as a long-acting benzodiazepine for the management of withdrawal symptoms and prevention of complications such as seizures and delirium tremens. Inj. Lorazepam was given intramuscularly for acute episodes of agitation and severe withdrawal symptoms requiring immediate sedation. Tab. Quetiapine 25 mg was prescribed for associated anxiety, insomnia, and any perceptual disturbances. Tab. Gabapentin 400 mg was given as an adjunct for the relief of withdrawal symptoms, reduction of anxiety, and assistance in craving control. The patient was given Inj. Optineuron 100 ml intravenous infusion containing B-complex vitamins, especially thiamine, for the prevention of Wernicke's encephalopathy and other neurological complications associated with chronic alcohol use.

DISCUSSION

This case study brings to focus the Alcohol Dependence Syndrome (ADS), which is a progressive and relapsing disorder influenced by the complex interplay of biological, psychological, and social factors. This patient clearly exhibits the characteristic features of the Alcohol Dependence Syndrome, such as the

progressive consumption of alcohol, inability to control consumption, tolerance, withdrawal manifestations, and consumption despite the associated problems, according to the dependence syndrome proposed by Edwards. It leads to tolerance, compulsive drinking, and physical dependence because of neuroadaptive alterations in reward and stress pathways, especially in dopaminergic, GABAergic, and glutamatergic pathways. In the proposed case study, if there is no alcohol, withdrawal manifestations such as tremors, seizure disorders, irritability, and insomnia would follow.

These seizures can most likely be categorized under alcohol withdrawal seizures and usually occur within 24 to 48 hours after stopping alcohol consumption in large quantities. Furthermore, the previous brain injury in the patient can make their neurons even more vulnerable to these withdrawal effects. This increased vulnerability is because, during alcohol withdrawal, the neurons become highly susceptible to alterations in their functional properties. Regarding diagnostic findings, the laboratory results include elevated GGT, mildly to moderately elevated AST, and mildly elevated bilirubin, which indicate an alcoholic liver caused by alcohol consumption [4].

Psychiatric morbidity is an important part of ADS, which can be assessed by the suicide attempt that was undertaken by the patient just now. The presence of alcohol dependence syndrome is accompanied by mood instability, impulsiveness, anxiety, and depression, which will culminate in suicide if it is not dealt with comprehensively [6]. By default, antisocial personality syndrome has not been stated in this patient, but personality disorder can be equated with higher levels of severity, compliance, and relapses, along with early onset in the case of antisocial personality in ADS patients [2,7].

In public health terms, AIDS continues to be common but less frequently diagnosed, especially in primary healthcare, where it imposes a considerable burden on society [3]. The risk of relapse is increased if there is early age at start of drinking, familial predisposition, protracted heavy drinking, nicotine dependence, psychiatry, and psychosocial stressors, all contributing factors for this patient. Stress, negative affect, and conditioning are highly predictive of relapse during initial phases of detoxification [8]. The conceptual framework containing dependence views relapse often as a symptom of chronic vulnerability instead of lack of willpower [9]. Also important are the long-term management strategies that are multidisciplinary. Medically supervised detoxification with benzodiazepines is crucial to prevent seizures; in addition, pharmacological interventions according to liver function as a means to prevent relapses, as well as psychosocial interventions like cognitive behavioural therapy, involving families, as well as careful follow-ups [5]. This case highlights ADS as a complex condition that is both chronic and recurrent in character [1].

CONCLUSION

Alcohol Dependence Syndrome is a chronic and relapsing condition that has major medical and psychological implications. This particular case is a good example of the manifestations that may follow chronic and excessive alcohol intake. There are potential sequelae of alcohol withdrawal seizures and liver damage, as well as dramatic psychological and behavioural manifestations such as suicidal ideas. The earlier the condition is diagnosed and the proper treatment is implemented, the better.

ABBREVIATIONS

ADS: Alcohol Dependence Syndrome
OPD: outpatient department
GGT: gamma-glutamyl transferase

AST: aspartate amino transferase
GABA: gamma-aminobutyric acid
AIDS: Acquired immunodeficiency syndrome

DECLARATION OF CONSENT

I hereby affirm that the patient has examined the clinical facts and granted written informed consent for their publication. The patient was guaranteed that their identity would be kept secure and that only anonymized data would be utilized. All ethical requirements pertaining to patient privacy were meticulously adhered to. Consent documentation has been securely preserved.

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