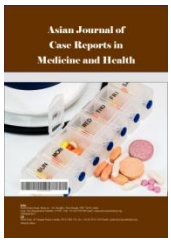

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A Case of Protracted Alcohol Withdrawal Syndrome: Resistant Lilliputian Hallucinations with Tremor

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Authors' contributions

This work was carried out in collaboration among all authors. Author AGSD wrote the original draft. Authors AGSD, KR and BAG conceptualized, validated, reviewed, edited, supervised and the final approval of the version to be submitted. All authors read and approved the final manuscript.

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Case Report

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ABSTRACT

In Sri Lanka WHO estimates 3.1% of the population to have alcohol used disorder and predicts that nearly 50% of them can present with alcohol withdrawal syndrome.

We describe a 51 year old male with the back ground history of alcohol abuse for 20 years duration and series of adverse life events facilitating him to develop severe alcohol used disorder and depression. When he presented to us, he exhibited features of major depression with suicidal thoughts and evidence of alcohol withdrawal syndrome according to DSM V criteria. He also had minor cognitive impairment according to MMSE.

After two months of inward treatments, he continued to have disturbing Lilliputian hallucinations, tremors and sleep disturbances which made us to diagnose him having protracted alcohol withdrawal syndrome after considering the other possibilities like major depression with psychotic symptoms, lewy body dementia and psychotic disorder with mood symptoms. His cognitive impairment can be explained by reduced attention and concentration together with low literacy, therefore the diagnosis of pseudo dementia secondary to depression was made.

This case illustrated us the multi-faceted presentations of alcohol used disorder which ultimately posing significant burden to the patient, family, society and also to the free health care system in the country.

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Keywords: Alcoholism; protracted alcohol withdrawal; depression; visual hallucination.

ABBREVIATIONS

AUD : Alcohol used disorder;
AWS : Alcohol withdrawal syndrome;
DSM V : Diagnostic and statistical manual of mental disorders, 5th edition;
NMDA : N-methyl-D-aspartate;
PAW : Protracted alcohol withdrawal;
TBIL : Traumatic brain injury.

1. INTRODUCTION

Alcohol is an organic compound which can cause dependency and numerous negative health consequences not only physically, but also psychologically, socially and spiritually. WHO estimates, 3.1% of population in Sri Lanka to have Alcohol used disorder (AUD) in the year 2016. Nearly half of the patients with AUD can present with alcohol withdrawal syndrome (AWS) which directly posing a significant burden to the free health care system in the country [1].

Alcohol withdrawal syndrome is diagnosed when there are two or more withdrawal features such as hand tremors, insomnia, autonomic hyperactivity, psychomotor agitation, nausea or vomiting, transient hallucinations, anxiety and generalize tonic clonic seizures causing functional impairment or significant distress, following several hours to few days after cessation or reduction in alcohol use which has been heavy and prolonged, when there is no alternative cause apparent [2]. The presence of seizures, altered level of consciousness, extreme autonomic activity and hallucinations hints the presence of severe AWS. The hallucinations can occur with normal vital signs in alcoholic hallucinosis. The natural cause of disease is that the AWS usually disappears in 2-7 days where as in severe forms, it needs medical treatment which otherwise carry even a risk of death.

Protracted alcohol withdrawal (PAW) can be defined as persistence of withdrawal symptoms beyond the typical time frame, usually considered after 14 days [3]. It can persist up to even 1 year or so. The identification of the syndrome is important as it is associated with significant distress to the patient resulting in relapse of alcohol drinking to alleviate those bothering symptoms. There are no well defined criteria either in Diagnostic and Statistical manual of Mental disorders (DSM) or in International Classification of Diseases (ICD). However in keeping with the current understanding of the disease, one study done in 2005, a PAW was diagnosed if a patient shows re-appearance and increase of typical alcohol withdrawal symptoms during the careful taper of benzodiazepines or clomethiazole after at least 14 days of treatment and has immediate reduction of these symptoms on increment of the doses when there are no other psychiatric or somatic disorder which explain the symptoms, including no use of sedatives or hypnotics in the last 6 months [4]. The epidemiological data with regard to PAW and its impact to the health system and society is yet to be presented not only from Sri Lanka but also globally.

Furthermore this case highlights the importance of comprehensive assessment by professional psychiatry team, not to misdiagnose this presentation as a primary psychotic disorder in which the treatment approach is significantly different.

2. CASE PRESENTATION

A 51-year-old male who was a father of 3 children, a cook in a restaurant was abusing alcohol for more than 20 years duration. He used to take in average 15 units of alcohol daily in the form of arrack containing 40% of alcohol. Occasionally, when he had financial difficulties he also used to take cheap illicit alcohol to counteract craving and withdrawal symptoms. He had several attempts of quitting which failed due to lack of determination and adverse life events which maintained his behavior. However, he never obtained medical or psychological assistance in quitting alcohol.

He was away from home in Maldives and worked as a cook for more than 2 years when he returned to Sri Lanka 5 years ago. Soon after returning, his wife was diagnosed with a terminal malignancy which resulted in death, after 18 months of treatment at Cancer Institute, Maharagama, Sri Lanka. And he lost his elder child 2 years ago, at the age of 7 years from Dengue fever. Furthermore, he had a severe head injury following road traffic accident which needed neurosurgical interventions and 3 months of hospital stay during the last year. However he recovered with no focal neurological symptoms or signs or gross cognitive deficits.

With these series of events he further addicted to alcohol and he did not have any control over it. He had craving and started taking alcohol in the mornings also to relieve the withdrawal symptoms like restlessness and tremors. He also lost the interest of life and his job, less energetic and had passive thoughts of suicide when he first presented to a psychiatric unit 8 months ago. He was diagnosed with moderate depression and alcohol withdrawal syndrome. He was detoxified with benzodiazepines and thiamine during the ward stay and started on venlafaxine 75 mg/ 112.5 mg and risperidone 3 mg mane for which he had reasonable compliance. But he was not compliant with behavioral modifications and was not registered in any alcohol cessation programme.

He was bringing up his children with great difficulty due to financial constraints as well as due to irreplaceable loss of his beloved wife. Meanwhile he lost his second child due to Dengue fever 2 months ago. He felt guilty and worthless and had an attempt of suicide during the funeral ceremony by means of hanging in the bed room under influence of alcohol. Due to the ceremonial event he was not produced to psychiatric care.

He presented this time himself complaining progressively poor sleep, loss of energy and low mood for last 2 months duration which severe enough to keep him absent from his job. He also claimed loss of weight. However he continued to take alcohol, sometimes binging which exceeded 30 units per day. He was tremulous and agitated by mid noon of the day without alcohol. He last drank 34 hours earlier to the presentation.

He biologically looked much older than for his age. He was anxious and had postural coarse tremors in bilateral hands. He was seeing animals and snakes of normal size and colour at distance and when coming closer becoming smaller which make him scared and disturbing sleep. He had no evidence of ophthalmoplegia, nystagmus and variations in blood pressure, pulse rate, temperature. MMSE was 20 (orientation: 10/10, registration: 3/3, attention: 2/5, recall: 1/3, language: 4/8, copying: 0/1). However he had studied only up to grade 4 and was unable to write any language. He had not had any falls, incontinence or memory impairment of note before this presentation. He could not retain or concentrate on any task given for more than 5 minutes during occupational therapy.

He was started on IM thiamin 100 mg daily, chlordiazepoxide 30 mg tds, venlafaxine 150 mg mane and 75 mg nocte and risperidone 2 mg daily. His mood was settling gradually after 3 weeks of treatment, but he continued to have sleep disturbances, coarse hand tremors and disturbing visual hallucinations as described above. His appetite, social engagement and attention were also improved with occupational therapy including group activities and cookery sessions.

3. DISCUSSION

In accordance with the Diagnostic and Statistical manual of Mental disorders, 5th edition (DSM V), it was evident that above person had severe AUD which is complicated by AWS and severe depression. The entity of Alcohol used disorder is described as the presence of at least 2 out of 11 criteria over a 12 months period causing significant distress or impairment on the person's life. Drinking more or longer than intended, persistent desire or failed attempts to reduce or stop drinking, lot of time spent for drinking and recovering from the effects of alcohol, craving to drink, interference to responsibilities due to drinking, continued drinking despite social problems related to alcohol, continued drinking despite physical or psychological problems related to alcohol, giving up previously pleasurable activities due to drinking, persistent drinking in hazardous situations, features of tolerance and evidence of withdrawal features are considered as 11 criteria. Base on the number of criteria present the severity classified as mild AUD when 2 to 3 criteria are met, moderate AUD when 4 to 5 criteria are met and severe AUD when more than 5 criteria are fulfilled [1].

The comorbid occurrence of depression and AUD is common and it can have either a causal relationship or can act as a maintaining factor to each other. The United States NESARC study found that the major depression was 3.7 times more likely to occur in person with AUD than those without AUD [5]. Moreover, it can influence the treatment and outcome of either disease [6]. The causal relationship of depression by alcohol is observed by remission of depression by detoxification and abstinence from alcohol [7]. The vice versa also true where a person with depression who use alcohol to relieve its symptoms need treatment for depression to achieve full remission from AUD. And it is also shown that the occurrence of AUD

and depression together compared to either disorder alone pose a higher risks with regard to dissatisfaction in life, lower global functioning and suicidal attempts [7]. Moreover, insomnia which can be a symptom of depression itself is a risk factor to craving alcohol [8].

The evidences for relationship between AUD and the adverse life events such as bereavement of loved ones, care giving for cancer patients, widowhood is not uniform. And those relationships are further manipulated by the age, sex and personality of the patient, the external support received during the difficult period etc. [9,10]. The effect of traumatic brain injury (TBI) on AUD also is not consistent. While it is shown to have reduction in alcohol use immediately after the TBI mainly due to physical barriers and immediate strong determinations to stop alcohol, there is significant subset around 25% in some studies, who resume alcohol use after TBI. Generally, TBI does not trigger development of new cases of AUD at its own merits [11].

However, the most bothering symptom in our patient was visual hallucinations which made him anxious and contributing to sleep disturbance. Visual hallucination is a perception of an external visual stimulus in the absence of any external stimulus. It can be caused by disturbances in brain anatomy, brain chemistry, prior experiences and psychodynamics. It is categorized as simple and complex. The simple hallucinations are also called as "elementary" or "non-formed" and include colors, lights, lines, shapes, or geometric designs. The complex hallucinations are also called as “formed” and include images of people, animals, objects or sceneries. The psychotic illnesses, alcohol withdrawal, dementia, narcolepsy, delirium are amongst the causes for complex visual hallucinations and migraine, retinal pathologies, occipital lobe pathologies are amongst the causes for simple visual hallucinations. Seizures and visual loss can present with either simple or complex visual hallucinations. The causes we entertained for visual hallucinations in our patient were as follow (Table 1).

The specific type of visual hallucinations in our patient was Lilliputian hallucinations, in which things, animals or people are seen smaller than in real life [12]. Although it was initially described as specific to alcohol or drug-related toxicity, later it was also described in infective, neurodegenerative causes, schizophrenia and seizure disorders as well [12,13]. However the commonly seen disorder with Lilliputian hallucinations is AWS.

Since the above patient demonstrated the Lilliputian hallucinations, hand tremors and sleep disturbances well beyond (more than 8 weeks) the usual time period and it was resistant to treatment with benzodiazepines, the diagnosis of protracted alcohol withdrawal (PAW) was considered. Alcohol consumption increases inhibitory GABA neurotransmitter function on GABA- A receptors and decreases excitatory glutamate neurotransmitter function on N-methyl-D-aspartate (NMDA) receptors. In normal individuals, the excitatory effect of glutamate on NMDA receptor, gives a positive contribution in memory, learning and generation of seizures. Therefore, the overall effect of alcohol is sedative. With prolong use of alcohol there is adaptive decrease in functions of GABA receptors and adaptive increase in functions of NMDA receptors. During the withdrawal less GABA and more glutamate with potentiated receptors causes hyper excitability of CNS causing autonomic over activity, seizure and delirium [14]. The adapted glutaminergic response said to be responsible not only for the acute withdrawal state, but also for the protracted withdrawal [15].

The well-established treatment of AWS is with benzodiazepine which is effective in preventing agitation, seizures and delirium tremens while it is cross- tolerant with alcohol. However, it has its own potential of addiction when used longer period as in PAW. In such instances anti-convulsants like carbamazepine, valporic acid and gabapentin are good alternatives by decreasing withdrawal seizure; craving; kindling while having no or less abuse potential and less sedative effects at the expense of their own side effect profiles. Anti-convulsants also have positive effects on mood symptoms such as depression, anxiety and irritability [16, 17, 18]. Hence, one of the anticonvulsants is being considered in above patient too, as he was continuing to have withdrawal symptoms and signs. Trazodone, an anti-depressant also has promising results on sleep efficiency in PAW [19].

Table 1. Differential diagnosis for visual hallucinations in our patient

Differential diagnosis	Features support the diagnosis	Features against the diagnosis	Remarks
Alcohol withdrawal syndrome	The onset after abrupt withdrawal of alcohol (within 6- 24 hrs) The presence of other withdrawal features like tremors, sleep disturbances and anxiety	Persists after typical time period- even more than 8 weeks Not improved with treatment with benzodiazepines	Protracted alcohol withdrawal syndrome is a possibility
Major depressive disorder with psychotic features	Psychotic symptoms were started with evidence of major depression- low mood, anergia, anhedonia, loss of appetite, guilt, worthlessness, suicidal ideas were present Had previous history of depression	Hallucinations were not consistent with depressive themes of guilt and worthlessness [ex: nihilistic type delusions, persecutory type delusions]	
Psychotic disorder with mood symptoms and AUD	Presence of visual hallucinations and low mood	No past history of psychotic illnesses Only visual hallucinations No first rank symptoms	
Lewy body dementia	Presence of parkinsonism features- slowness, hand tremors Presence of visual hallucinations Presence of cognitive impairment	No falls No fluctuations of attention Functional till 2 months ago. The cognitive decline is rapid	The cognitive impairment is mainly due to reduced attention and concentration which can be due to pseudo dementia in depression and language deficit due to poor education

4. CONCLUSION

This case illustrated us the multi-faceted presentations of AUD which modified by patients psycho-social background. Therefore it demands the comprehensive evaluation by expertise from several disciplines including a psychiatrist, physician, occupational therapist and social worker. It is very important to have the thorough evaluation by professional psychiatric team at the very beginning of the presentation, not to label the patient to have a psychotic disorder due to the presence of prominent visual hallucinations. It also demands long term, fine-tuned, patient centered rehabilitation programme together with timely adjusted pharmacological interventions.

CONSENT

All authors declare that written informed consent was obtained from the patient for publication of this case report.

ETHICAL APPROVAL

As per international standard, written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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