Case report

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An Unusual Relationship Between Auditory Hallucinations, Alcohol Drinking, and Alcohol-Induced Premature Liver Damage in a Patient With Schizophrenia: a Case Report

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Keywords

Alcoholism; hallucinations; hepatitis, alcoholic; hypertension, portal; schizophrenia

Abstract

Aim: This case report explores the relationship between psychotic symptoms and excessive alcohol consumption in schizophrenia, examining its role in self-medication, premature liver damage, and treatment challenges. **Case report:** We describe a 31-year-old male with schizophrenia, who was nonadherent to antipsychotic treatment, and who repeatedly manifested excessive alcohol consumption during the exacerbations of psychotic symptoms, claiming that his alcohol drinking was for the purpose of self-medicating for auditory hallucinations. Despite temporary perceived relief, the patient developed severe alcohol-related liver damage, including alcoholic hepatitis and portal hypertension, at a remarkably early age. This case challenges the classical self-medication hypothesis, as alcohol use likely worsened both psychiatric and physical outcomes. **Conclusions:** This report highlights the complex relationship between psychosis and alcohol misuse, underscoring the need to explore alternative mechanisms linking auditory hallucinations to alcohol consumption. It also emphasizes early recognition of alcohol-related liver damage in schizophrenia. When liver function is impaired, careful selection of antipsychotic treatment is critical. The atypical antipsychotic amisulpride, with its minimal hepatic metabolism, appears to be a promising option for controlling psychotic symptoms and reducing alcohol cravings in such cases.

Introduction

Tobacco, alcohol, cannabis, and cocaine are substances widely used by individuals with schizophrenia [1,2]. One meta-analysis that included many individuals with schizophrenia (N = 165.811) reported a 24.3 % preva-

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lence of alcohol use disorder (AUD), which is three-fold higher compared to in the general population [1,3].

This high comorbidity between schizophrenia and AUD could be due to pleiotropy, or shared genetic liability [1,4,5]. Recently published genome-wide association studies for AUD (N = 77.822) and schizophrenia (N = 46.827) identified 55 independent genetic variants that increase the risk of both disorders [5]. The high prevalence of AUD among patients with schizophrenia might also be explained by the self-medication hypothesis, which suggests that patients use alcohol in attempts to alleviate the symptoms of schizophrenia or side effects from antipsychotic medications [4,6,7].

Temporal psychotic disorders caused by excessive alcohol consumption (so-called alcohol-induced psychotic syndrome, alcohol-induced psychosis, and alcoholic hallucinosis) have been described in the literature and clinical practice [8-10]. Patients with alcohol-induced psychotic syndrome manifest hallucinations, delusions, and other symptoms resembling those of schizophrenia [9,10]. On the other hand, there is a lack of reports indicating that patients with psychotic disorders excessively consume alcohol for the purpose of self-medication from either productive (positive) or negative symptoms.

Here we report the case of a 31-year-old male with schizophrenia, who manifested excessive alcohol consumption during repeated episodes of the exacerbation of psychotic symptoms. He claimed that his alcohol consumption was for the purpose of self-medicating for auditory hallucinations. Excessive alcohol consumption impacted the patient's liver at an early age, causing premature severe alcohol-related liver damage in the form of alcoholic hepatitis and portal hypertension, at an early age.

Case report

A 31-year-old male Caucasian Croatian patient was admitted to the Department of Psychiatry after an initial half-day observation at the Emergency Department. He had been unemployed for the last 4 weeks, was unmarried and had no children, was in an emotional relationship, and had a positive family history for paranoid schizophrenia. At the Emergency Department, the patient presented with alcoholic pre-delirium syndrome, characterized by moderate-to-severe tremor of extremities and symptomatic epileptic seizures, and also complained that had recently been experiencing auditory hallucinations. Computed tomography scan of the brain revealed no abnormalities of the brain parenchyma. Laboratory findings indicated mildly elevated levels of the liver enzymes aspartate aminotransferase (AST; 103 U/L) and alanine aminotransferase (ALT; 67 U/L), and a significantly elevated level of gamma-glutamyl transferase (966 U/L) [11]. During observation at the Emergency Department, the patient was treated with crystalloid infusions (1 - L infusions of 0.9 % saline, and 1 - L infusions of 5 % glucose) with four ampoules of diazepam 10 mg, which slightly decreased the tremor, and no repetitive epileptic seizures were observed.

During the initial psychiatric examination, the patient denied the presence of auditory hallucinations but confirmed that had been experiencing auditory hallucinations daily over the past 3–4 weeks. He verbalized that the auditory hallucinations had been manifesting in the form of unknown male and female voices, commenting and criticizing his behaviour, and occasionally threatening the patient in a manner related to assault, rape, and even murder. He further explained that his excessive alcohol drinking co-occurred with the appearance of auditory hallucinations (over the past 3 - 4 weeks). He stated that his daily alcohol use had usually been limited to 9 - 10 beer units (equalling 90 - 100 g of alcohol), while he had not been drinking at all for the last two days due to physical weakness [12]. The patient verbalized that had been drinking alcohol alone at his home, usually from early afternoon to bedtime. He categorically denied having experienced other hallucination types (e.g., visual hallucinations) or delusional ideas, and claimed that his excessive alcohol consumption over the past 3 - 4 weeks had been for the purpose of self-medication. When specifically asked to explain the role of alcohol consumption for "self-medication", he explained that drinking alcohol caused the voices to occur "less frequently", and to be "more silent" and sometimes even "less harassing" (specifically, as related to threats).

Upon admission to the Department of Psychiatry, during further psychiatric examination, the patient verbalized that he had been repeatedly experiencing periods of auditory hallucinations over the past 4 years, which had usually occurred several times per year and generally lasted for 3 - 4 weeks. He stated that during these periods, he had been excessively drinking alcohol for the purpose of self-medication, and explained that excessive alcohol drinking during the "hallucination phase" caused the hallucinations to "go away" for a prolonged period. He verbalized that in addition to beer, whiskey was another common alcoholic drink that he consumed during the "hallucination phase", and explained that when he had been consuming whiskey, he had not been consuming beer, and that his daily consumption of whiskey had been limited to 10 whiskey units (equalling 100 g of alcohol) [12]. He claimed that during the period when he had not been experiencing hallucinations, he had not been drinking alcohol and that he had been living a rather functional life, e.g., he had been working and socializing. The latter was also confirmed by his parents and girlfriend.

During the patient's current hospital psychiatric treatment, his tremor was relieved by diazepam 80–100 g daily during the first 3 days, and then the antipsychotic amisulpride 800 mg daily was introduced as therapy. However, amisulpride therapy resulted in an increased plasma prolactin level (2206 mIU/L); therefore, the amisulpride dose was titrated from 800 mg to 500 mg daily [13]. After several days of amisulpride treatment, the patient verbalized the disappearance of auditory hallucinations, and claimed that this remained stable on lower medication doses. Furthermore, he described no extrapyramidal adverse effects

upon amisulpride treatment, and reported that he felt that amisulpride was more suitable than the medications prescribed previously. After one month of in-hospital psychiatric treatment, the patient continued to receive psychiatric treatment in the outpatient clinic during the next 40 days. During treatment in the outpatient clinic, the patient repeatedly reported no auditory hallucinations or other psychotic symptoms, he also claimed alcohol abstinence and denied any alcohol craving. Importantly, several routine laboratory check-ups indicated negative blood alcoholaemia, and gradual decreases in the liver enzymes AST, ALT, and GGT. In parallel with psychiatric improvement, the patient reported improvement in everyday functioning. When he announced that he had been given a new job opportunity, his day hospital treatment was ended, following careful consultation with medical staff.

In his medical history, it was recorded that our patient had previously been hospitalized and treated at the Department of Psychiatry for three weeks in 2019, under a diagnosis of unspecified psychosis, which had been manifesting with auditory hallucinations. Indeed, at that time, hallucinations in the form of unknown male voices verbalizing death and torture had been causing the patient such high emotional distress that he had even made a telephone call to the police department. During the "hallucination phase" prior to the hospital admission, the patient had also been reporting excessive alcohol drinking—specifically, the consumption of 10 whiskey units daily. At hospital admission, elevated liver enzymes had been recorded (AST, 206 U/L; ALT, 144 U/L; and GGT 302 U/L), though to a lesser extent compared to those recorded at his current admission [11]. During the previous hospitalization, the patient had been administered the antipsychotic aripiprazole 30 mg daily and haloperidol 2.5 mg daily, and had received a long-acting aripiprazole injection (400 mg) at discharge. However, the patient had not showed up at scheduled ambulatory check-ups, and he claimed that he had stopped taking his medications a few weeks after hospital discharge. During his current psychiatric treatment, the patient was asked about the reason for medication nonadherence, and he only shortly answered that the prescribed medications "depressed his everyday functioning".

In 2021, our patient had also been hospitalized for 3.5 weeks at the Department of Gastroenterology and Hepatology, due to an episode of severe alcoholic hepatitis and portal hypertension. At hospital admission, his Maddrey's Discriminant Function (DF) was 34.6, Glasgow Alcoholic Hepatitis Score was 7, and Model for End-Stage Liver Disease score was 26 [14,15]. A Maddrey's DF score of $\geq$ 32 indicates severe alcoholic hepatitis, with a 1-month mortality rate of up to 30–50 % [14].

At admission to the Department of Gastroenterology and Hepatology, the patient had been complaining of malaise, exercise intolerance, nausea, anorexia, and weight loss of ~9 kg over several months. He also verbalized that two weeks prior to hospital admission, he had been noticing yellow discoloration of the sclera and skin, dark urine discoloration, and pruritus of the skin. When he was asked about alcohol consumption, he answered that he had been drinking strong alcoholic drinks (mostly whiskey), in amounts equalling 10 units daily, but he did not specify the duration of that drinking period. Laboratory findings at hospital admission revealed elevated levels of AST (174 U/L) and GGT (1169 U/L), hyperbilirubinemia (403 μmol/L), macrocytic anemia (haemoglobin of 103 g/L, and MCV of 103.5 fL), hypoalbuminemia (22.0 g/L), and abnormal coagulation parameters (prothrombin time: 58 % activity, and international normalized ratio test: 1.3) [16-19]. Abdominal ultrasound revealed significant hepatomegaly (~20 cm in the midclavicular line), with an enlarged left liver lobe; hyperechoic liver parenchyma; thickening of the gallbladder wall; and an increased diameter of the portal vein and its roots, suggestive of portal hypertension. Since the patient's clinical and laboratory scores (Maddrey's DF, Glasgow Alcoholic Hepatitis score, and MELD score) were suggestive of severe alcoholic hepatitis, during hospitalization, he received treatment with corticosteroids (methylprednisolone 40 mg), together with albumin infusions, enteral nutrition, and other supportive therapy, which led to improvement of patient's general condition, in parallel with decreases in serum bilirubin and liver enzymes. At ambulatory check-up at five weeks after hospital discharge, the patient reported further improvement of his physical condition and weight gain of ~4 kg. He denied the presence of nausea and pruritus of the skin yellow discoloration of the sclera and skin was significantly decreased. The patient also reported alcohol abstinence, and denied the presence of any psychotic symptoms, although he said that he did not visit the psychiatrist and had not been taking any psychiatric medications.

Discussion

Here we report the case of a patient with schizophrenia, who was nonadherent to antipsychotic treatment, and who excessively consumed alcohol for the purpose of self-medication (specifically, to alleviate auditory hallucinations). Written informed consent was obtained from the patient following a detailed explanation of the objectives and protocol of the study, which was conducted in accordance with the ethical principles stated in the Declaration of Helsinki. The self-medication hypothesis states that patients with schizophrenia consume alcohol and other addictive substances to alleviate negative symptoms and extrapyramidal side effects of antipsychotic medications [4,6,7,20]. Alcohol and other addictive substances raise brain dopamine levels by stimulating mesolimbic dopamine neurons [4,6,7,21], and decreased dopamine levels in specific brain areas are associated with negative symptoms and extrapyramidal side effects of antipsychotic medications that block postsynaptic dopamine D2 receptors [22]. On the other hand, hallucinations, delusions, and other positive symptoms of schizophrenia are associated with increased brain dopamine levels, rather than with decreased brain dopamine levels [23]. Therefore, our current report that

a patient with schizophrenia excessively consumed alcohol to alleviate auditory hallucinations does not support the self-medication hypothesis of schizophrenia [20]. It is possible that other yet-undefined factors may affect the relationship between auditory hallucinations and excessive alcohol consumption in patients with schizophrenia. For instance, alcohol reportedly increases gamma-aminobutyric acid (GABA) neurotransmission, and decreased brain GABA levels predispose patients with schizophrenia to hallucinate [24,25].

Another important finding of the current study is that our patient's excessive alcohol consumption led to the development of alcoholic hepatitis and portal hypertension at 29 years of age. Research and clinical data indicate that the typical age at presentation of alcoholic hepatitis is between 40 – 50 years and the age at presentation of portal hypertension is typically over 50 years [26,27]. Notably, gene pleiotropy may contribute to the high comorbidity between schizophrenia and AUD [1,4,5]. Accordingly-and in line with our patient with schizophrenia manifesting alcoholic hepatitis and portal hypertension at early age-we speculate that alcoholics with schizophrenia may be more vulnerable to the detrimental effects of alcohol. This speculation is supported by the fact that our patient claimed intermittent alcohol consumption, mostly during exacerbations of psychotic symptoms, while alcoholic hepatitis and portal hypertension are usually linked to everyday drinking behaviour [28]. Additionally, our patient claimed that his daily consumption of alcohol during the exacerbation of psychotic symptoms did not exceed 100 g/day, while patients with alcoholic hepatitis usually report a history of alcohol use of over 100 g/day, with 150 – 200 g/day being common [26].

Since many medications are metabolized in the liver, liver disease can interfere with the metabolization of antipsychotic medications [29]. The only antipsychotic medications that are not primarily metabolized by the liver are sulpiride, amisulpride, and risperidone's active metabolite, paliperidone (9 - hydroxyrisperidone) [30-32]. We chose amisulpride as a treatment option for our patient because research and clinical evidence suggest that amisulpride is more efficacious compared to other antipsychotic medications, and sulpiride is considered a low-potent antipsychotic medication [33].

Amisulpride, a substituted benzamide derivative, is a second-generation (atypical) antipsychotic medication, which shows high affinity for dopamine D2 and D3 receptors, and no affinity for serotonin, muscarinic, or alpha-adrenergic receptors [34]. Amisulpride predominantly works in the limbic system, explaining its lower risk of extrapyramidal adverse effects relative to other antipsychotic medications [35]. At low doses (100 – 300 mg/daily), amisulpride preferentially binds to D2/D3 presynaptic autoreceptors, increasing dopaminergic transmission in the prefrontal cortex, which is believed to lead to improvement of negative symptoms. On the other hand, amisulpride doses of 400 - 800 mg/daily result in antagonism of postsynaptic D2/D3 receptors, leading to improvement of positive symptoms of schizophrenia, with less extrapyramidal syndrome development [35].

Amisulpride is also believed to modulate neural response in cases of substance dependence, via antagonism of postsynaptic D2/D3 receptors [36,37]. In one study, functional magnetic resonance imaging was used to examine the effects of single administration of 400 mg amisulpride to abstinent alcoholics. Amisulpride medication was associated with reduced cue-induced activation of the thalamus, a brain region closely connected with frontostriatal circuits that regulate behaviour and may influence relapse risk [36]. In another study, ambulatory patients diagnosed with alcohol, heroin, cocaine, or cannabis dependences were treated with amisulpride doses ~ 500 mg/daily, and assessed at 0, 3, 6, and 9 months. Patients who completed the treatment reported overall improvement in their psychological distress, decreased craving, and improved psychological and social functioning [38].

To our knowledge, our present report is the first to indicate that a patient with schizophrenia excessively consumed alcohol to self-medicate while experiencing psychotic symptoms. Although his alcohol consumption was intermittent (during exacerbations of psychotic symptoms) and at doses not exceeding 100 g/day, it resulted in the development of alcoholic hepatitis and portal hypertension at an age significantly earlier than typically observed in the general population.

The main shortcoming of this report is that the data regarding alcohol consumption were mainly based on patient self-report. Therefore, it cannot be excluded that the patient may have also consumed alcohol, to a variable amount, while was free of psychotic symptoms. There exists a need for prevention strategies in the field of psychosis related to the control of addictive behaviours (specifically, alcohol dependence) and medication nonadherence.

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Conflict of Interest

None to declare.

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