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Acute Alcoholic Hallucinosis: A Review

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Abstract
Acute alcoholic hallucinosis is a psychotic disorder characterized by a predominance of auditory hallucinations with delusions and affective symptoms in the clinical picture. Classically, it develops as part of the alcohol withdrawal syndrome. The prevalence of acute alcoholic hallucinosis ranks second among alcohol-related psychoses after alcohol delirium. The study aimed to systematize the scientific data on the history of alcoholic hallucinosis, its pathogenesis, clinical presentation, and treatment approaches. A literature search was performed in PubMed, Scopus, Google Scholar, and eLibrary. The following words and combinations were used as search strings: (alcoholic hallucinosis OR alcoholic psychosis OR alcohol-related psychosis OR alcohol-induced psychosis OR alcohol-induced psychotic disorder OR complicated alcohol withdrawal syndrome) NOT (animal OR rat OR mouse). The relevant information concerning the history of acute alcoholic hallucinosis, its pathogenesis, clinical picture, and treatment approaches was systematized and summarized. This review presents relevant findings regarding acute alcoholic hallucinosis. Limitations of the review include the use of heterogeneous and mostly descriptive studies and studies on small cohorts of patients.

Introduction

The nosological construct of alcoholic hallucinosis has received significant interest in the scientific literature and has been interpreted differently during the development of psychiatric medicine [1, 2]. The history of the investigation of alcoholic hallucinosis began in 1847, when Claude Marcel introduced the term *folie d'ivrogne* (*drunken madness*) to refer to a symptom complex similar to alcoholic hallucinosis. Marcel is also credited with being the first to distinguish it from delirium tremens [3]. He described 20 clinical cases, three of which were reported in detail in his thesis *De la folie causée par l'abus des boissons alcooliques*: "Acute psychoses in persons who have abused alcohol for many years with auditory hallucinations of a threatening and/or obscene nature, with possibly also frightening visual hallucinations, and delusions secondary to the contents of the hallucinations. The pa-

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tients were orientated, but had feelings of great anguish and could at any time attempt suicide. The age of onset was between 25 and 45 years, the duration of illness varying between a few weeks and several months. Prognosis was good" [4].

In 1900, Carl Wernicke coined the term *alcoholic hallucinosis* and described its main features, which included the presence of verbal hallucinations of threatening and abusive content, as well as the feeling of fear [5]. Emil Kraepelin, describing the clinical picture of alcoholic hallucinosis, differentiated it from delirium tremens and stated: "The picture of disease has a certain resemblance to delirium tremens, in the fantastic delusions, the vividness of the hallucinations, the remarkable combination of insane ideas with a sense of illness and the alcoholic origin. It differs from delirium chiefly in its much longer duration, in the absence of any disturbance of the patient's idea of his position, and in the predominance of the hallucinations of hearing compared with the prevalence of hallucinations of sight in delirium tremens. The unrest, too, is generally less, and the tremors are not nearly so pronounced. For these reasons, it seems best to clearly distinguish this illness from delirium tremens, although the two are certainly nearly related. We call this disease alcoholic mania. Its course may extend over several weeks or months. The outcome is generally complete recovery, but there is no inconsiderable number of cases in which incurable states of weakness remain, usually with particular permanent delusions and hallucinations." [6].

Paul Eugen Bleuler in his *Lehrbuch Der Psychiatrie* (*Textbook of Psychiatry*, 1916) described alcoholic hallucinosis as "alcoholic insanity" (*Alkoholwahnsinn*), providing two other names in brackets: "Kraepelin's hallucinatory insanity of drunkards" (*halluzinatorischer Wahnsinn der Trinker Kraepelins*) and "Wernicke's acute hallucinosis of drunkards" (*Akute Halluzinose der Trinker Wernickes*) [7]. Bleuler pointed out that alcoholic hallucinosis is in many aspects the opposite of delirium tremens: "It manifests itself chiefly in auditory hallucinations, which have a peculiar character: in most patients it is a case of the voices of several or many people not present who discuss the patient in a dramatically elaborate dialogue; that is, they discuss him in the third person; much more rarely do they speak to him. These voices threaten him, remind him of his sins, scold him, make plans as to how they will catch him and perhaps torment and torture his family also. Some egg the other on, or some of them side with the patient, try to defend him and save him. In very acute cases the connection is usually less organized; in place of more quiet scenes there is a confusion of voices. Of the hallucinations of the other senses visual deceptions occur most readily. Delusions of persecution correspond to the hallucinations. At the same time the patients remain oriented and in spite of the delusions they generally remain clear. The dominant affect is anxiety, which is not lacking in any case and usually dominates the whole behavior." [7]. Bleuler stressed the similarity of the described psychotic disorder to schizophrenia and suggested that it was schizophrenia triggered by alcohol abuse. Compared to Kraepelin's clinical picture of alcoholic hallucinosis, the outcome is definitely worse and chronic in its development.

In the following years, the nosological nature of alcoholic hallucinosis continued to be debated. Some authors regarded the disorder as an independent nosological entity with a specific etiology, clinical presentation, and course; others supported the hypothesis that it constitutes a form of schizophrenia triggered by alcohol abuse, as proposed by Bonhoeffer with the model of exogenous psychosis, induced by alcohol and substances [8]; others denied the existence of acute alcohol hallucinosis as a separate disorder or considered it a form of alcohol withdrawal or delirium tremens with less pronounced physical symptoms [3, 9–11].

In 1989, two articles by Glass were published, summarizing the accumulated literature at that time [12, 13]. Evidence was listed to support the identification of acute alcoholic hallucinosis as a separate disorder from paranoid schizophrenia: acute onset; average duration of the disorder about 3 months; history of alcohol dependence; family history of alcoholism and no evidence of a positive family history of schizophrenia; the age of onset of 40–50 years; coherent thought processes; presence of auditory, visual, and tactile hallucinations; and anxious affect congruent with delusional disorders.

In the 10th revision of the International Classification of Diseases (ICD-10), alcoholic hallucinosis is coded as F10.52 ("Alcohol-induced psychotic disorder, with hallucinations") and as 6C40.60 in ICD-11. According to the ICD-10 diagnostic criteria, the diagnosis of F10.52 ("Alcohol-induced psychotic disorder, with hallucinations") is a condition in which the psychotic symptoms develop within 2 weeks of alcohol consumption and must persist for more than 48 h. The psychotic disorder is characterized by vivid hallucinations (typically auditory, but often in more than one sensory modality), perceptual distortions, delusions, psychomotor disturbances (excitement or stupor), and an abnormal affect, which may range from intense fear to ecstasy. The sensorium is usually

clear, but some degree of clouding of consciousness, though not severe confusion, may be present. The duration of the episode may be up to 6 months.

Material and Methods

Data Sources

The review was conducted in accordance with Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. Two reviewers (VYS and MSZ) independently searched PubMed, Scopus, Google Scholar, and eLibrary without any time restrictions. The following words and combinations were used as search strings: (alcoholic hallucinosis OR alcoholic psychosis OR alcohol-related psychosis OR alcohol-induced psychosis OR alcohol-induced psychotic disorder OR complicated alcohol withdrawal syndrome) NOT (animal OR rat OR mouse). Both reviewers independently assessed publications as well as extracted and checked the data for accuracy. In addition, we performed further secondary searches by using the reference listing of all eligible papers.

Results

Prevalence of Acute Alcoholic Hallucinosis and Its Pathogenesis

According to Tsuang et al. [14] who studied a cohort of 643 patients, the prevalence of acute alcoholic hallucinosis is 7.5% of all hospitalized patients with alcohol dependence. Meanwhile, Soyka estimated that acute alcohol hallucinosis has been detected in 0.4% of patients hospitalized for alcohol dependence [15]. In a population-based study, Perälä et al. [16] found a lifetime prevalence of acute alcoholic hallucinosis of 0.41%. In the subpopulation of individuals with alcohol dependence, the prevalence rate was 4.0%. The highest lifetime prevalence of acute alcoholic hallucinosis was found in the 45–54 year age group (1.8%). According to the literature, it has been found that younger age of onset of alcohol dependence, low socioeconomic status, the family burden of alcohol use disorders and other mental disorders, as well as repeated hospital admissions due to alcohol use disorders are associated with a higher risk of acute alcoholic hallucinosis [17].

In a study by Egorov et al. [18], it was found that, despite a general downward trend in the incidence of alcohol-related psychoses in recent years, patients with such disorders continue to make up a significant proportion (15–30%) of those who undergo treatment in psychiatric hospitals, and the incidence of alcoholic hallucinosis is 14–27% of the total number of cases of alcohol-related psychoses. Sedain found that alcoholic hallucinosis was diagnosed in 12.4% of patients hospitalized for alcohol use disorders [19]. Soyka et al. [20] analyzed the prevalence of acute alcoholic hallucinoses by referring to hospital statistics from a major health insurance provider in Germany. The authors concluded that the prevalence of acute alcoholic hallucinoses in the general population was low, but they noted a high rate of repeated hospital admissions for patients with this psychotic disorder. According to a recent study by Narasimha et al. [21], the prevalence of alcoholic hallucinosis was 0.9% of all hospitalized patients diagnosed with alcohol dependence.

Published studies have suggested several neurobiological mechanisms that may account for the occurrence of hallucinations in patients with alcohol dependence [22]. They include dysfunction of the dopaminergic system [23, 24], dysfunction of the serotonergic system [23, 25], the decrease in concentration of inhibitory neurotransmitter amino acids and increase in concentration of excitatory (glutamine, asparagine) neurotransmitter amino acids [26], changes in beta-carboline activity rates [27], and possible damages of auditory and sensory conducting pathways [28].

It was established experimentally that alcohol administration to rats is accompanied by enhanced dopamine release from nigrostriatal and mesolimbic pathways [24]. Moreover, the level of homovanillic acid in cerebrospinal fluid (which is an indirect indicator of the central dopaminergic neurons' activity) was shown to correlate significantly with the frequency of auditory and visual hallucinations occurring in alcohol withdrawal [29]. Aliev described an increase in plasma concentrations of glutamate and aspartate in patients with acute alcoholic hallucinoses, indicating the hyperactivity of excitatory neurotransmission, which may be aggravated by a decrease in inhibitory GABAergic activity when cessation of alcohol consumption [26]. Since plant extracts containing beta-carboline can cause hallucinations, the increased plasma levels of beta-carboline detected in patients with acute alcoholic hallucinoses may have potential pathogenetic significance [27]. Finally, toxic damage to the sensory and peripheral auditory pathways resulting from excessive alcohol use has been suggested as a possible contributing factor to the development of hallucinations, similar to the described mechanism of tinnitus [28].

Based on the results of ^{18}F-fluorodeoxyglucose positron emission tomography imaging performed in 4 patients with acute alcoholic hallucinosis, Soyka et al. [30, 31] suggested a possible thalamic dysfunction. One patient was found to have reduced glucose metabolism in

frontal (by 28–30%), thalamic (by 50%), and cerebellar (by 36%) regions. Another patient showed a 10–18% reduction in ^{18}F-fluorodeoxyglucose uptake in the right thalamic region compared to the left thalamic region [30].

The description of a clinical case of a 56-year-old patient with acute alcoholic hallucinosis showed that single-photon emission computed tomography revealed abnormal blood flow in the frontal lobe, the left basal ganglia, and the thalamus [32]. Against the background of effective diazepam and haloperidol therapy, there was a normalization of blood flow in the left basal ganglia and the left thalamic area. The described changes in blood flow were not confirmed in a study by Jordaan et al. [33]. The authors found increased blood flow in the area of the right calcarine sulcus in patients with acute alcoholic hallucinosis compared to healthy volunteers and revealed a tendency to increased blood flow in the frontal and temporal lobes, as well as in the right side of the globus pallidus. Thus, the accumulated data of neuroimaging studies performed on patients with acute alcoholic hallucinosis to date are inconsistent but generally reflect dysfunction at the neuroanatomical level [22].

Clinical Presentation

The authors traditionally describe the onset of alcoholic hallucinosis as gradual, when the patients develop sleep disorders, which are later joined by anxiety [34]. Classically, acute alcoholic hallucinosis develops as part of the alcohol withdrawal syndrome; thus, Gofman considers alcohol withdrawal syndrome as a prodromal period of alcoholic hallucinosis development and emphasizes that he has never observed the onset of acute alcoholic hallucinosis outside the end of drinking or during a time when withdrawal symptoms have already completely disappeared [34]. In most cases, acute alcoholic hallucinosis occurs within 2 days after the last alcohol consumption, and, unlike delirium tremens, the duration of the preceding binge may be significantly shorter, sometimes amounting to less than a week [35, 36]. Meanwhile, Aliev noted an increased frequency of cases of acute alcoholic hallucinosis against the background of continuing binge drinking [26].

As a rule, acute alcoholic hallucinosis occurs against the background of unconsciousness, when the patients suddenly experience auditory hallucinations [37, 38]. A characteristic feature of verbal hallucinations occurring in the debut of acute alcoholic hallucinosis is the absence of multiple voices: patients usually hear one or two voices, whose utterances are often repeated and represent an annoying repetition of individual words or short phrases. Often, these verbal hallucinations take the form of dialogic speech, a conversation taking place somewhere close to the patient. Another characteristic of auditory hallucinations is their neutrality, i.e., the content of the hallucinations does not concern the patient directly, and the topic of conversation of the voices is everyday matters, mundane activities, and everyday relationships. This allows the patient to remain only a witness to the hallucinatory events rather than a direct participant in them. In turn, most often the loss of the neutral character of hallucinations indicates the transition of the debut of acute alcoholic hallucinosis to the advanced stage [39].

Gofman describes other variants of the debut of acute alcoholic hallucinosis: for instance, a seizure-like onset in which there are several abortive psychotic episodes separated by lucid intervals followed by an extended and longer psychotic disorder; and an onset with the appearance of elementary sensory distortions: acoasms (patients hear squeaks, whistles, rings, buzzes, and musical sounds), later to be replaced by phonemes [39]. The transition of acute alcoholic hallucinosis to the advanced stage is accompanied by the development of advanced verbal and auditory hallucinosis, which is often combined with delusion. These voices, in most cases, come from the real surroundings of the patient and have a condemning character [34, 40]. A distinctive feature of verbal hallucinations is a reference by voices to the patient in the third person that makes him an involuntary listener, forced to be present when discussing his actions and behavior (occurring, as a rule, in a negative way and often cynical in nature) [39, 40]. It is not uncommon that some voices judge the patient, scold him, and make mocking remarks, while others, on the contrary, are friendly to him, protect him, and justify him and, as a result, he listens to a whole discussion about himself [34]. With the further intensification of the hallucinatory influx, the reference to the patient in the third person may be replaced by a direct reference of voices to the patient. According to Gofman, the content of verbal hallucinations most often corresponds to one of four main themes: stalking the patient to physically destroy him; alcohol abuse; immoral behavior; and the patient's sexual relationships [39].

Along with auditory hallucinations, patients often experience visual hallucinations, usually related to the content of verbal hallucinations and representing a kind of illustration of what the patient hears; tactile hallucinations may also present rarely [40]. This is another characteristic that differentiates alcohol hallucinosis from delirium tremens. Delusions in patients with acute alcoholic hallucinoses are typically related to the content of

hallucinations. In most cases, delusions of persecution are present, with the motives of persecution being trivial and mundane. A combination of delusions of persecution and poisoning ideation is also common.

Acute alcoholic hallucinosis is also characterized by affective disorders, which are adequate to the content of delusions and manifest predominantly in the form of fear, characterized by depth and constancy and combined with anxiety [40]. The occurrence of melancholy at the peak of hallucinosis is also possible when the fear reaches its apogee and turns into despair [39]. All phenomena develop against a background of relatively clear consciousness or only mild disorientation with regard to time [34]. If the hallucinosis persists for 1–2 weeks, there is a danger of developing chronic hallucinosis.

Thus, to establish a correct diagnosis of the acute alcoholic hallucinosis, a detailed history is important. Specifically, it is imperative to determine the patient's alcohol use history. Acute alcoholic hallucinosis must be differentiated from delirium tremens and schizophrenia. Furthermore, other causes of altered mental status should also be evaluated, including infection, trauma, and metabolic causes such as liver disease, thiamine deficiency, and electrolyte imbalance [41]. Therefore, CT imaging of the brain, urinalysis, urine drug screen, lab evaluation including electrolytes, liver function tests, ammonia, and toxicology screening may be indicated [42]. Finally, dual disorders are common in patients with alcohol use disorder, and a specific diagnostic assessment is mandatory. The list of the most common mental comorbidities in dual disorders includes anxiety disorders, post-traumatic stress disorder, attention deficit with/without hyperactivity disorder, and personality disorders (mainly antisocial and borderline) [43].

In recent years, several studies on the pathomorphosis of acute alcoholic hallucinosis, i.e., the changes in clinical presentation of disease over time, have been published. Thus, according to a study by Alikulov and Teshabaeva, pathomorphosis is expressed in the following ways: decrease in duration of acute alcoholic hallucinosis; higher frequency of abortive acute alcoholic hallucinosis compared to typical; and clinical presentation of acute alcoholic hallucinosis in males and females is found identical in terms of psychosis duration and structure [44].

In 2018, a study conducted by Nemkova and Gofman investigated changes in the clinical presentation of acute alcoholic hallucinosis during the last 50 years [45]. According to its results, a change in the clinical presentation of acute alcoholic hallucinosis was established, which the authors attributed to the extensive use of modern antipsychotic drugs. When studying the pathomorphosis of acute alcoholic hallucinosis over the past 50 years, the highest frequency of abortive hallucinosis (characterized by short duration and absence of intense hallucinatory onset) compared to typical acute alcoholic hallucinosis was found in recent years. The authors found that typical acute alcoholic hallucinosis is second most frequent, with a clinical presentation consisting of verbal hallucinosis and sensory delusions of persecution combined with anxiety, fear, and melancholy. In addition, the average duration of a typical acute alcoholic hallucinosis was found to be 78 ± 12.5 h, while in cases of abortive hallucinosis it was 9 ± 5 h.

Similar conclusions were obtained by Uvarov et al., who investigated the pathomorphosis of acute alcoholic psychoses [46]. An increase in the proportion of alcoholic hallucinosis in the total number of acute alcohol-related psychoses was found in comparison between 2020 and 1980 (42.5% and 25%, respectively). The authors also attributed the described pathomorphosis to the widespread use of modern neuroleptics. Moreover, in order to explain these changes, they discussed social factors: the past 2 decades have been marked by dramatic social transformations with profound and complex implications for psychiatry, influencing mental health risk factors, dynamics in clinical encounters, styles of help-seeking behavior, and clinical outcomes, contributing to the emergence of alcohol and substance-related psychotic phenomena [47].

Treatment

The clinical features of acute alcoholic hallucinosis (such as psychomotor agitation accompanied by the feeling of fear and an influx of frightening hallucinations, as well as a high frequency of suicide attempts and sudden manifestations of aggression) often necessitate hospital treatment [39, 48]. Patients with acute alcoholic hallucinosis typically benefit from antipsychotic medication, sometimes in combination with sedatives [48, 49]. Moreover, given that the use of antipsychotics in the context of alcohol withdrawal could precipitate convulsions, the administration of benzodiazepines should be mandatory [50]. Studies state that the condition usually resolves within 18–35 days with antipsychotic and/or benzodiazepine treatment, while a minority of patients may have persistent symptoms for 6 months or more which is considered to be a chronic hallucinosis [51].

A case study of a 58-year-old man with acute alcoholic hallucinosis successfully treated with risperidone up to 4 mg daily was published in 2007 [48]. A study by Uvarov

(2009) confirmed the effectiveness of risperidone in patients with acute alcoholic hallucinosis and demonstrated its advantages over haloperidol: in the group of 15 patients, risperidone had a significant effect on such symptoms as hallucinations and delusions from days 2–3 of treatment, while in the group of 15 patients receiving haloperidol, these symptoms disappeared by days 4–5 of therapy [52].

Meanwhile, a study by Nemkova conducted on 100 patients with acute alcoholic hallucinosis suggested that the most effective antipsychotic was haloperidol administered parenterally at a dose of 5–10 mg. The mean duration of acute alcoholic hallucinosis was 29 ± 15 h against the background of haloperidol therapy, whereas for olanzapine administered in a dose of 10 mg parenterally, it was 49 ± 19 h, and for risperidone prescribed 4 mg orally, this parameter was 53 ± 23.5 h [53].

A systematic review of the treatment of acute alcoholic hallucinosis (2018) included 15 studies (4 of them double-blind randomized) and 10 clinical case descriptions [54]. None of the publications included had used second-generation antipsychotics. Six studies examined the use of one or more first-generation antipsychotics, including haloperidol, chlorpromazine, trifluoperazine, reserpine, thiotixene, and levopromazine. Three studies investigated the use of a regimen that included antipsychotics, benzodiazepines, and vitamin B_1 but did not specify which antipsychotics were used. Anticonvulsants were used in three studies (lamotrigine, sodium valproate, and phenobarbital, respectively). In one study, hypnotics such as barbamyl and chloral hydrate were used. In two studies, other drugs acting on GABA receptors (piracetam and clorazepate) were prescribed. One study investigated the use of electroconvulsive therapy (ECT).

The results of this systematic review indicated heterogeneous results, even for the same medication. For instance, the proportion of patients with complete remission achieved with chlorpromazine ranged from 0 to 68%, and partial remission was reported in 0–32%. The best results were obtained for haloperidol: complete remission was achieved in 68–90% of cases and partial remission was achieved in 0–30% of patients. Treatment duration ranged from 3 to 546 days, but in most publications, it was short, and a longer treatment duration was not associated with more favorable treatment outcomes.

Descriptions of ten clinical cases included in the review reported 13 patients receiving 21 different treatment regimens. All patients received first- or second-generation antipsychotics. Three received benzodiazepines and vitamin B_1 additionally. Five patients reported complete remission with risperidone and two with olanzapine. No remission was reported in patients treated with chlorpromazine, perazine, flupentixol, trifluoperazine, or quetiapine. Of the three cases treated with haloperidol, only one achieved partial remission. ECT was used in two of the clinical cases, both of which achieved complete remission. However, it should be noted that the last report on the use of ECT in the treatment of acute alcoholic hallucinations was published in 1956 [55].

While discussing their findings, the authors of the systematic review concluded that there was sufficient evidence to suggest that patients with acute alcoholic hallucinosis responded to therapy with antipsychotics. However, the authors point out that there is no reliable evidence of the superiority of any particular medication and conclude that both first- and second-generation antipsychotics appear to be effective.

In terms of side effects and possible interactions, a recent review regarding the role of atypical antipsychotic drugs in dual disorders suggested some advantages for second-generation antipsychotics, specifically for D2 partial agonists [43]. However, these results should be confirmed in alcoholic hallucinosis.

Conclusions

Our review presents relevant findings regarding acute alcoholic hallucinosis. Limitations of the review include the use of heterogeneous and mostly descriptive studies, as well as the investigation of small cohorts of patients.

The authors previously published a review on the topic in Russian [56], which may be of interest to Russian readers since it describes the problem through the prism of Russian clinical perspective and Russian psychiatric scientific schools, which have some originality and are not always consistent with the recommendations of European and American psychiatric associations.

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Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

Valentin Yurievich Skryabin took primary responsibility for conceptual development, organization, and writing. Giovanni Martinotti and Johan Franck contributed to conceptual development, drafted and edited portions of the manuscript, and handled formatting tasks. Mikhail Sergeevich Zastrozhin made substantial contributions to conceptual development and manuscript editing.

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