Case Report

Brain Atrophy in a Patient with Delirium Tremens

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Abstract

Approximately 3-5% of individuals undergoing alcohol cessation or reduction after chronic overuse develop delirium tremens (DT), a severe manifestation of alcohol withdrawal syndrome. Long-term alcohol consumption has been associated with brain atrophy. This case report discusses the findings of an early-onset cerebral atrophy in an alcohol withdrawal patient with DT. A 53-year-old man presented to the emergency unit with nausea and flank pain following the ingestion of a cocktail of alcoholic beverages the day prior. Physical examination showed an elevated blood pressure (223/146 mmHg) with no history of seizure. Within two days, the patient had shown a deterioration of consciousness. He was transferred to the high care unit and referred to the psychiatry department for signs of agitation. Psychiatric assessment revealed that the patient was delirious and experiencing auditory and visual hallucinations. A multi-slice computed tomography scan revealed cerebral atrophy. This case highlights the co-occurrence of brain atrophy and DT, emphasizing the importance of neuroimaging in patients with a history of alcohol use. Early recognition of cerebral changes may facilitate timely intervention, improve management of severe withdrawal, and guide preventive strategies in alcohol use disorder.
Keywords: *delirium tremens, brain atrophy, alcohol withdrawal, alcohol use disorder, alcoholism.*

Atrofi Otak pada Pasien dengan Delirium Tremens

Abstrak

Delirium tremens (DT) merupakan bentuk berat dari sindrom putus alkohol dan terjadi pada 3-5% individu saat menghentikan atau mengurangi konsumsi alkohol yang berlebihan dan kronis. Konsumsi alkohol jangka panjang ditemukan berhubungan dengan atrofi otak. Laporan kasus ini membahas temuan atrofi serebri di onset yang lebih cepat pada pasien putus alkohol dengan DT. Laki-laki berusia 53 tahun datang ke unit gawat darurat dengan keluhan mual muntah dan nyeri perut samping satu hari setelah mengkonsumsi alkohol "oplosan". Pemeriksaan fisik menunjukkan tekanan darah tinggi (223/146 mmHg). Tidak ada riwayat kejang. Dalam dua hari, pasien menunjukkan penurunan kesadaran. Pasien dipindahkan ke High Care Unit dan dikonsultasikan ke departemen psikiatri untuk gejala agitasi. Saat pemeriksaan psikiatri, pasien didapati mengalami delirium, serta halusinasi auditorik dan visual. Multi-slice Computerized Tomography menunjukkan atrofi serebri. Laporan kasus ini berkontribusi dalam literatur kedokteran dengan memaparkan pentingnya pencitraan otak untuk deteksi dan intervensi dini pada individu dengan riwayat konsumsi alkohol, dalam mengantisipasi gejala putus alkohol berat dan memungkinkan penatalaksanaan yang tepat dan cepat.
Kata kunci*: delirium tremens, atrofi otak, putus alkohol, gangguan penggunaan alkohol, alkoholisme.*

Introduction

The prevalence of alcohol use is highest in high-income regions of Australasia and Europe, where more than three-quarters of adults consume alcohol, and lowest in Muslim-majority countries, including Indonesia, where the prevalence is only 3.7%.[1,2] Despite this, one of the most apparent alcohol problems in Indonesia is unrecorded alcohol consumption, which accounts for 83.3% of the total alcohol consumption.[2] Intoxication of methanol, ethylene glycol, and isopropanol potentially leads to irreversible organ damage, manifesting as visual impairments, gastrointestinal hemorrhage, pancreatitis, metabolic acidosis, and death within 6−24 hours.[3] Between 2008 and 2017, 840 deaths in Indonesia were attributed to unrecorded alcohol consumption.[2]

The harmful use of alcohol is driven by positive and negative reinforcement, leading to the desire for pleasure or gratification and the relief of stress or anxiety. These mechanisms are mainly driven by the upregulation of GABAergic (ɣ-aminobutyric acid-mediated) activity and the downregulation of glutamatergic activity. Chronic alcohol exposure induces adaptive changes in these neurotransmitter systems, leading to tolerance and dependence.[4] During alcohol cessation, approximately 50% of chronic drinkers experience withdrawal symptoms, including autonomic hyperactivity and cardiovascular instability. Severe withdrawal, characterized by hallucinations, seizures, or delirium tremens (DT), occurs in 3–5% of cases.[5]

Brain volume loss happens physiologically with age.[6] However, long-term alcohol exposure may precipitate cerebral degeneration.[7] Although the rate of unrecorded alcohol consumption in Indonesia is high, literature on pre-elderly cerebral atrophy in DT is limited.[2] This case report discusses a case of DT with findings of cerebral atrophy at a relatively young age of 53 years.

Case Description

A 53-year-old man was urgently transported to the emergency unit of Hospital X in October 2023 with complaints of nausea, profuse vomiting, and flank pain following the ingestion of a cocktail of unknown alcoholic beverages the previous day. He was an unmarried tour guide and photographer working at a popular tourist destination in Yogyakarta, Indonesia. The patient reported a history of alcohol consumption since the age of 25, drinking approximately every other day. He was unable to estimate the amount consumed per sitting but admitted a preference for expired or unrecorded alcoholic beverages to achieve heightened effects. There was no known history of psychiatric disorders or chronic medical conditions, and he was not on routine medication.

Within two days, the patient's level of consciousness deteriorated. He was transferred to Hospital Y for further evaluation. On admission, he was somnolent, with a Glasgow Coma Scale (GCS) score of E3V2M4. Pupils were dilated with decreased light reflex. Laboratory tests confirmed metabolic acidosis and acute kidney injury. A multi-slice computed tomography (MSCT) scan revealed a round-shaped infarction in the left thalamus, a well-defined hypodense lesion, and cerebral atrophy characterized by prominent gyri, sulci, and Sylvian fissures (Figure 1).

The patient was admitted to the high care unit (HCU) under the joint management of internal medicine, neurology, and cardiology specialists. His condition gradually improved, and he was transferred to a regular ward the following day. Ophthalmology evaluation was performed for visual impairment, and psychiatric consultation was requested due to agitation.

Psychiatric evaluation revealed that the patient appeared appropriate for his age and gender but demonstrated poor self-care. Due to agitation, non-cooperative behavior, and hyperactivity, hand restraints were applied as part of the management strategy. His orientation to place, time, and space was impaired. Affect was inappropriate, with a dysphoric mood. Thought form was non-realistic, content was centered on a desire to return home, and thought progression was coherent but occasionally irrelevant.

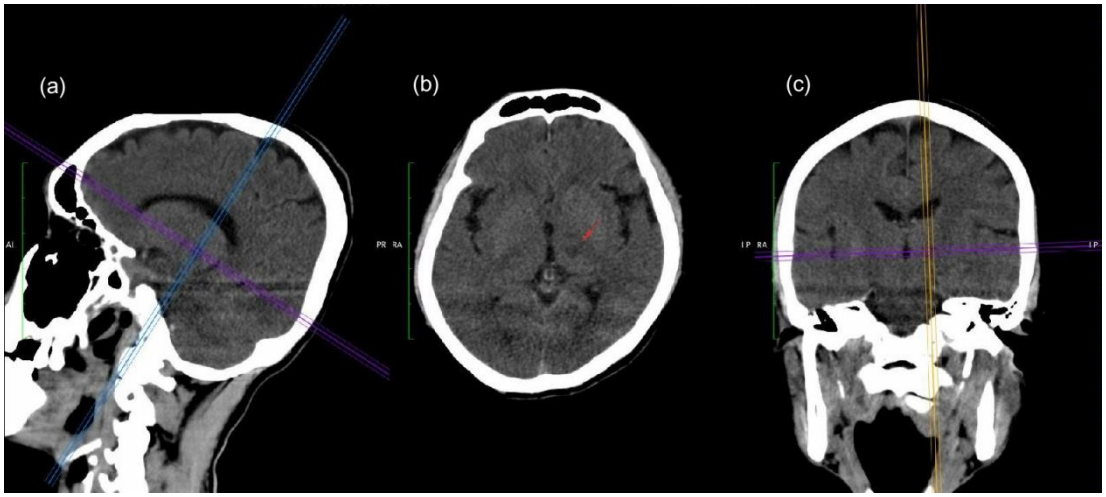

Figure 1. Non-Contrast Head MSCT Scan
(a) Sagittal view: demonstrates cerebral atrophy marked by deep sulci and gyri;
(b) Axial view reveals a hypodense lesion in the left thalamus; and
(c) Coronal view confirms cerebral atrophy marked by deep sulci and gyri

The patient reported auditory and visual hallucinations, describing seeing *wayang* (traditional Indonesian puppets) in his room and conversing with an imaginary companion. Establishing a mental connection was challenging; attention could be gained, but was easily lost. Insight was assessed at level 1.

Based on these findings, the patient was diagnosed with delirium. He was treated with intramuscular olanzapine 10 mg per 24 hours as needed for agitation, oral thiamine 100 mg daily, and supportive psychotherapy. Over the course of treatment, his condition improved, and he was discharged after a nine-day hospital stay.

Discussion

Alcohol acts as a central nervous system depressant by enhancing inhibitory neurotransmission and suppressing excitatory pathways. Specifically, alcohol potentiates γ-aminobutyric acid (GABA) receptor activity while inhibiting N-methyl-D-aspartate (NMDA) receptors, thereby reducing glutamatergic excitatory signaling. Chronic exposure induces adaptive changes, including downregulation of GABA receptor activity and upregulation of NMDA receptor activity. Abrupt cessation of alcohol use removes both inhibitory and excitatory modulation, resulting in autonomic hyperactivity within 6–8 hours.[5]

In the present case, the patient exhibited nausea, profuse vomiting, hypertension, and tachycardia—classic features of autonomic hyperactivity. His long-standing history of alcohol consumption, combined with recent cessation, strongly suggested alcohol withdrawal. Withdrawal symptoms typically peak within 72 hours and may progress to seizures or DT in 3–5% of cases.[5]

Several predictors of DT have been identified, including advanced age, high daily alcohol intake, prior history of DT or alcohol-induced psychosis, cognitive deficits, and severe physical or laboratory abnormalities.[8,9] Daily alcohol intake is a stronger predictor of DT than age alone. Prior episodes of DT, alcohol-induced psychosis, and cognitive impairment are also significant risk factors. The severity of physical and laboratory findings is crucial in anticipating DT, as conditions such as hypertension, tachycardia, hyperthermia, thrombocytopenia, and electrolyte imbalances increase the likelihood of progression to severe withdrawal.[8,10]

In this case, the patient had a long and intense history of alcohol consumption and presented as a pre-elderly male. One day prior to DT diagnosis, examination revealed markedly elevated blood pressure (223/146 mmHg), tachycardia (110–130 beats per minute), and severe hyperkalemia (serum potassium 8

mmol/L). Although platelet counts remained within normal limits, they had declined from 192,000/µL to 154,000/µL, approaching baseline. No prior history of alcohol-related disorder behavior was reported.

Indicators of alcohol withdrawal in this case included cessation of heavy alcohol use, autonomic hyperactivity (hypertension and tachycardia), profuse vomiting, and agitation. Delirium features, including impaired attention and awareness, poor orientation, and hallucinations, followed these symptoms. The patient reported visual and auditory hallucinations, describing seeing *wayang* (traditional Indonesian puppets) and conversing with an imaginary companion. Given that both DSM-5 criteria for alcohol withdrawal and delirium were met (Table 1), the patient was diagnosed with DT.[11]

The current case presented structural lesions in the brain, as shown on the MSCT scan, as impressions of left thalamic infarction and cerebral atrophy (Figure 1). While there are various publications on DT, data about pre-elderly cerebral atrophy in DT is limited,[12,13] especially in Indonesia, where unrecorded alcohol consumption is prevalent.[2] A study conducted by Maes et al.[15] revealed that the Sylvian fissures and interhemispheric fissures were significantly larger in DT patients compared to non-DT alcohol-dependent patients, which consecutively represented temporal subcortex atrophy and frontal cortex atrophy.[14]

These structural changes, including neuronal and axonal loss and demyelination, are attributed to alcohol consumption and are immediately evident in the frontal lobe, as evidenced by widening of the third ventricle.[15,16] Although the frontal lobe is more sensitive to alcohol than other parts of the brain, the decrease of gray matter volume is also apparent in parietal and insular cortex, temporal and cingulate regions, brain stem, putamen, and amygdala. Moreover, white-matter microstructural alterations are evident in tracts connecting extensive networks and deeper white-matter systems. These changes are detectable in individuals who consume 10-20 ml (8-16 g) of ethanol daily, and worsen with the severity of alcohol use.[7]

Management of DT focuses on controlling agitation, preventing seizures, and reducing mortality. In this case, intramuscular olanzapine (10 mg daily as needed) was administered. Olanzapine, an atypical antipsychotic, acts by blocking 5-HT2 serotonin and D2 dopamine receptors. It has demonstrated efficacy in alleviating alcohol withdrawal symptoms and reducing alcohol cravings.[17] Its sedative effect, combined with a lower risk of extrapyramidal symptoms, respiratory depression, and circulatory disturbances, made it a suitable choice.[18–20] Oral thiamine (100 mg daily) was also prescribed to prevent thiamine deficiency and Wernicke's encephalopathy. Supportive psychotherapy and correction of metabolic disturbances were provided concurrently.[21]

Table 1. The DSM-5 Diagnostic Criteria for Delirium Tremens[11]

Alcohol Withdrawal	Delirium
1. Cessation or diminution of prolonged and heavy alcohol use 2. A minimum of two out of eight symptoms subsequent to reduced alcohol use: ● Autonomic hyperactivity ● Hand tremors ● Insomnia ● Nausea or vomiting ● Temporary hallucinations or illusions ● Psychomotor agitation ● Anxiety ● Generalized tonic-clonic seizure	1. Decreased attention and awareness over a short period of time 2. Disruptions in cognition, including memory, orientation, language, visuospatial abilities, perception, and all functions that deviate from the baseline and fluctuate in severity 3. No evidence of coma or occurrence of neurocognitive disorders

Patients with a history of alcohol consumption often seek medical attention for complaints unrelated to alcohol. However, alcohol withdrawal upon hospitalization might trigger withdrawal symptoms. Despite its rare prevalence, severe withdrawal symptoms and DT are very serious and life-threatening. There are many risk factors leading to DT. However, initial history taking can be challenging in agitated patients, especially when the patient's guardian lacks detailed knowledge of the patient's situation. Therefore, this case report highlights the importance of investigating the patient's history of alcohol consumption, which involves cerebral atrophy or other possible brain abnormalities. This allows for the timely administration of appropriate therapy to prevent morbidity and mortality.

Limitations of this report include reliance on a single case, absence of detailed prior medical history, and lack of follow-up data. Future studies with larger cohorts and longitudinal designs are needed to clarify the relationship between DT, alcohol withdrawal, and cerebral atrophy, and to establish optimal strategies for early detection and management.

Conclusion

This case report highlights brain atrophy as a relative predictor of DT. It emphasizes the significance of medical professionals being vigilant toward individuals with a history of alcohol use. Timely identification of withdrawal manifestations is essential to anticipate progression to severe stages, allowing immediate and precise intervention to reduce morbidity and mortality.

Conflict of Interest

The authors declare that there is no conflict of interest.

Consent Form

Written informed consent was obtained from the patient.

Authors' Contributions

AFK conceptualized and designed the study and provided supervision and mentorship. BDLD, PSP, ANK, and MRG collected data. Data analysis and interpretation were performed collaboratively by AFK, BDLD, PSP, ANK, and MRG. AFK and KS were responsible for validating and verifying the results. The initial draft of the manuscript was prepared by BDLD, PSP, ANK, MRG, and KS, and AFK and KS critically revised it for important intellectual content. All authors read and approved the final version of the manuscript for publication.

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