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CASE REPORT

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Case Report: Prolonged Delirium Tremens

Sevtap TOLAMAN[1], Rasim TUNÇEL[2], Barış ÖRS[3], Damla İŞMAN HAZNEDAROĞLU[1]

[1]Department of Mental Health and Diseases, Ege University Faculty of Medicine, İzmir, Türkiye
[2]Department of Neurology, Ege University Faculty of Medicine, İzmir, Türkiye
[3]Ankara University Faculty of Medicine, Ankara, Türkiye

ABSTRACT

Delirium tremens represents the most severe condition of alcohol withdrawal, with the associated highest mortality rate. The primary treatment for cases of delirium tremens consists of benzodiazepines. Within the literature, prolonged cases of delirium tremens have been identified that do not respond to high-dose benzodiazepine treatments or respond late. Different treatment modalities, such as propofol, dexmedetomidine, and parenteral antipsychotic administrations, are being attempted in the management of these cases. In this case, a case of prolonged delirium tremens with insufficient response to benzodiazepine treatment will be presented.

Keywords: Alcohol withdrawal; antipsychotic; benzodiazepines; case report; delirium tremens

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INTRODUCTION

Alcohol plays a leading role in substance use disorders, and alcohol abuse is a matter of great concern due to the escalating rates of morbidity and mortality in the population (1). The lifetime risk of having delirium tremens (DT) in the population with alcohol use disorders is between 5% and 10% (2). The current DT diagnostic requirements involve an alteration in consciousness and cognitive function or perceptual disturbances that develop rapidly and the onset of these symptoms during or shortly after the cessation of heavy alcohol consumption.

Delirium tremens results in extended hospitalization, elevating healthcare costs and mortality risk (3). Death may occur because of hyperthermia, fluid loss, electrolyte imbalance, infection, or cardiovascular collapse. Identified risk factors for DT include low serum potassium, low platelet count, the presence of structural brain lesions, increased blood pressure, medical comorbidities, and a history of complicated withdrawal syndromes (DT and/or withdrawal seizures) (4,5). Mortality in hospitalized patients with DT can be reduced with adequate treatment (6), but there is no consensus on the most effective medications to address prolonged delirium.

The typical duration of delirium is three to five days, with some cases extending up to two weeks. However, exceptional instances of even longer DT have been reported (7). Most patients have underlying medical factors that contribute to their symptom's occurrence and prolongation. This case report presents a case of prolonged DT that was completely resolved after 56 days. The patient has given informed written consent for the case report to be published anonymously.

Highlights

- **In our case, the delirium tremens (DT) symptoms lasted for 56 days.**
- **The persistence of DT symptoms is not due to an organic etiology.**
- **Medical comorbidities may cause therapeutic challenges in cases of extended DT.**

CASE PRESENTATION

The patient was a married 42-year-old male who presented with fluctuating consciousness, disorientation, agitation, insomnia, visual hallucinations, disorganized behavior, and speech for approximately 3 weeks. He was a butcher who had lived with his wife at the time of admission. The patient's medical history was significant only for hepatosteatosis. A few months before this admission, he had been carrying out his responsibilities, but he started missing work due to alcohol consumption. He was identified as a "social drinker" who had been drinking for the last five years due to increasing marital and work-related stress. He had been drinking alcohol for the past 20 years. Upon admission, he disclosed that he started consuming 700 mL of country liquor (45% ethanol) daily in the morning. He declared having tolerance for alcohol, experiencing withdrawal symptoms, being unable to limit his alcohol use even while working, and having interpersonal and professional issues due to alcohol. He had not consumed any alcohol for

Correspondence Address: Damla İşman Haznedaroğlu, Ege Universiy Faculty of Medicine, Department of Mental Health and Diseases, İzmir, Türkiye • **E-mail:** damlaisman2004@hotmail.com
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approximately six weeks before admission. Twice in his life, he tried to stay alcohol-free or undergo alcohol detoxification. Both were unsuccessful. He had a history of smoking 20 pack-years, but no history of substance use. He had an uncle and father with a history of alcohol use disorder.

Six weeks before admission, the patient was presented to an outpatient addiction clinic for alcohol cessation due to hepatosteatosis. The initial treatment consisted of diazepam 50 mg/day, folic acid 10 mg/day, thiamine 250 mg/day, disulfiram 500 mg/day, and naltrexone 50 mg/day. On the fourth day of regular medication use, owing to intense sedation, a gradual reduction in the diazepam dose to 10 mg/day in two weeks and disulfiram and naltrexone treatments were discontinued. However, the patient and his healthcare provider were not able to reduce the dose of diazepam gradually. They continued to use only diazepam 5 mg/day for three days.

The patient presented with symptoms of DT with fluctuating consciousness, disorientation, agitation, insomnia, visual hallucinations, disorganized behavior, and speech. The patient presented to the emergency room three weeks after the onset of the symptoms. The possibility of DT led to the initiation of treatment with oral diazepam 20 mg/day, haloperidol 2 mg/day, propranolol 80 mg/day, disulfiram 500 mg/day, naltrexone 50 mg/day, folate 5 mg/day, pantoprazole 40 mg/day, and thiamine 3 mg/day. Due to intense agitation, 5 mg of intravenous diazepam was administered in the emergency room. As the delirium symptoms and intense agitation persisted, the patient was transferred to our inpatient ward of Addiction Psychiatry.

On admission, the patient's self-care was inadequate. The patient was confused and alert. He was found to have tremors in his chin and extremities. His blood pressure was 164/111 mmHg, and his pulse rate was 83 beats per minute, physical examination was otherwise normal. Electrocardiography (ECG) revealed no abnormalities. The neurological assessment indicated mild dysarthric speech and ataxic gait. Upon mental status examination, the patient was found to be cooperative, distracted, and disoriented regarding time, place, and person. The patient was dysthymic and tense. Short- and long-term memories were not intact. No delusions were found in the thought content and there was a lack of insight. The patient had visual hallucinations.

The patient was determined to be at risk of moderate-to-severe alcohol withdrawal syndrome due to a history of alcohol dependence. The Clinical Institute Withdrawal Assessment of Alcohol Scale, Revised (CIWA-Ar) scores were 36. Consequently, treatment was initiated with oral quetiapine 50 mg/day, propranolol 40 mg/day, thiamine 500 mg/day, and folate 10 mg/day, diazepam 40 mg/day as the highest dose he could tolerate.

Admission laboratory work revealed elevated serum gamma-glutamyl transpeptidase (GGT): 505 U/L, alkaline phosphatase (ALP): 206 U/L, aspartate aminotransferase (AST): 38 U/L, total bilirubin: 1.49 mg/dl, direct bilirubin: 0.93 mg/dL, and mean corpuscular volume (MCV): 103.3 fL. The renal function test results, serum electrolytes, and complete blood count (CBC) were all within normal ranges. Urine screening for other substances yielded negative results. Abdominal ultrasonography indicated grade 2 hepatosteatosis with no abnormalities. Further investigations, including the hepatotropic viral serology panel (HBsAg, AntiHBc IgG, AntiHBs, Anti HCV, Anti-HIV), autoimmune hepatitis panel ((antinuclear antibody (ANA) , anti-neutrophil cytoplasm antibodies (ANCA)), liver profile (soluble liver antigen (SLA), liver kidney antibody (LCA-1), liver kidney microsomal antibody) LKM, Anti-mitochondrial M2 antibody (AMA-M2), anti-smooth muscle antibody (ASMA), antimitochondrial antibody (AMA), antigastric parietal cell antibody (GPC), and magnetic resonance cholangiopancreatography (MRCP), revealed no pathology. Contrast-enhanced cranial magnetic resonance imaging (MRI) showed no acute central pathology.

The patient was diagnosed with prolonged DT. On the third day of admission, quetiapine XR 200 mg was added for insomnia. On the fourth day, haloperidol 2.5 mg/day was initiated for intense nocturnal agitation. After confirming insufficient thiamine replacement, intravenous thiamine replacement was administered in 1000 cc of saline for three days at a dose of 250 mg/day, followed by oral thiamine replacement at 250 mg/day. During the first week of admission, intensive agitation at night led to the administration of parenteral haloperidol, chlorpromazine, and biperiden. The haloperidol dose was increased to 12.5 mg/day in routine treatment, and the diazepam dose was reduced to 30 mg/day. By the end of the second week, agitation had decreased, and the patient's orientation had begun to stabilize, leading to a reduction in haloperidol to 10 mg/day and diazepam to 27.5 mg/day.

In the third week, fluctuating orientation prompted a neurological consultation for differential diagnoses of prolonged delirium or Wernicke encephalopathy. Contrast-enhanced cranial MRI and EEG were performed, but no pathology was detected. In the third week, the patient developed a cough and sputum, leading to consultation with a pulmonologist. High-resolution computed tomography (HRCT) and COVID PCR (polymerase chain reaction) tests showed no pathology, and the patient was diagnosed with acute bronchitis. Levofloxacin 500 mg/day and N-acetylcysteine 600 mg/day were added to the treatment, and antibiotic therapy was planned for ten days due to the identification of Haemophilus influenzae non-type B in the sputum culture. After the bronchitis fully resolved, the patient was still in delirium.

By the fourth week, the patient's agitation decreased, and the orientation toward the person and place began to stabilising. The haloperidol and diazepam doses were gradually reduced. On the 36th day of admission, the patient demonstrated a complete orientation to the person, place, and time. The patient's delirium symptoms resolved 56 days after onset. Following psychotropic-free monitoring, a short cognitive examination revealed good orientation, attention, and instant memory. Montreal Cognitive Assessment (MoCA) test score was 21of 30 (According to the version used, the cut-off point is 21). Based on the tests administered and behavioral observation during evaluation; the psychologist's overall opinion was that the patient did not have a cognitive impairment.

After psychotropic-free monitoring, the patient was discharged without medication. He remained conscious and oriented. There were no notable findings on the physical examination. DSM-5 axis, the patient was discharged in remission with the following diagnoses: alcohol dependency and alcohol withdrawal delirium. During follow-up in the outpatient clinic, weeks later, the patient displayed compulsive behaviors such as checking belongings, doors, and locks and asking confirmation questions to their spouse. The treatment plan was adjusted to include sertraline 50 mg/day, and follow-up was planned.

DISCUSSION

Multiple medical complications arise with DT treatment, commencing at the time of diagnosis. The majority of DT cases typically resolve within 5 to 7 days. There are few reported cases in the literature where DT persists beyond the specified duration (8). The inability to taper down a high dose of diazepam is considered a precipitating factor for delirium in our case history. Thus, the absence of treatment for three weeks following the onset of delirium symptoms suggests that this may contribute to the exacerbation and prolonging of the symptoms.

The prolonged duration was a significant factor in the case in question, even though the onset and progression of the confusional state, as described here, were consistent with DT. Therefore, it is therapeutically imperative to consider any other medical and surgical issues that could

have contributed to the patient's altered mental state. Particular emphasis should be placed on cerebral lesions, such as subdural hemorrhagic encephalopathy, infections, and metabolic or endocrine disorders. Serum electrolytes, specifically serum magnesium concentration, are the most important factor in predicting the duration of DT (9). The serum magnesium levels were within the normal range for our case. The presence of normal cranial MRI results, the absence of nystagmus-like ophthalmopathy, and mild ataxic signs ruled out Wernicke's encephalopathy. Further investigations, such as hepatotropic viral serology and autoimmune hepatitis panel, ruled out any underlying medical conditions that could cause prolonged DT.

In case of protracted DT cases, benzodiazepines are required at higher dosages and for longer durations (10,11). According to the patient's prior alcohol withdrawal treatment, the diazepam dose was limited to 40 mg/day, and the haloperidol dose was planned to be between 5–10 mg per day for agitation and hallucinations.

It was also observed that the extension of the delirium process during the hospitalization period, along with the presence of medical comorbidities such as infection, complicates and protracts the treatment process. By the third week of hospitalization, acute bronchitis had been detected. A combination of antibiotherapy and psychotropic agents was utilized in our case.

This is one of the few cases of prolonged DT reported in the literature. The information currently available suggests that alcohol withdrawal delirium is the cause of delirium, but the extended duration of the altered mental state precludes a definitive diagnosis.

Informed Consent: The patient has given informed written consent for the case report to be published anonymously.

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