Case Report

ANNALS OF MEDICINE & SURGERY

OPEN

Alcoholic pellagrous encephalopathy: a case report on atypical presentation and diagnostic dilemma in alcohol-related disorders

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Introduction: Alcohol use disorder is a global health concern with various complications, including pellagra, often overlooked due to its rarity. This case explores the neurological presentation of pellagra in a long-term alcohol and substance abuser, emphasizing the diagnostic challenges in resource-constrained settings.

Case presentation: A 36-year-old male with a history of substance abuse presented with multiple symptoms, including hallucinations and neurological deficits. His complex clinical history included alcohol dependence, seizures, and relapses. Physical and neurological examinations revealed characteristic signs of pellagrous encephalopathy. Laboratory findings confirmed anemia and a fatty liver.

Discussion: Alcoholic pellagrous encephalopathy (APE) presents a diagnostic challenge due to its atypical symptoms, overlapping with other alcohol-related disorders. Niacin deficiency, central to its pathogenesis, affects neurotransmitter synthesis, contributing to neurological symptoms. Diagnosis relies on clinical presentation, but laboratory tests for niacin levels can aid in confirmation. Neuroimaging can exclude alternative causes. This case underscores the importance of considering pellagrous encephalopathy in alcohol-related disorders with neurological symptoms.

Conclusion: This case underscores the importance of recognizing atypical presentations of APE in chronic alcohol-dependent individuals. Prompt diagnosis, nutritional correction, and addressing alcohol use are vital for successful management. Healthcare providers must be aware of the diagnostic complexities and socioeconomic barriers hindering timely intervention in APE.

Keywords: alcohol use disorder, case report, diagnostic challenges, niacin deficiency, pellagrous encephalopathy

Introduction

Alcohol use disorder (AUD) is a prevalent global health issue, characterized by chronic, and excessive alcohol consumption with detrimental physical, psychological, and social consequences. The spectrum of complications associated with AUD is extensive, including liver diseases, cardiovascular disorders, psychiatric comorbidities, and neurological manifestations[1]. The National Institute on Alcohol Abuse and Alcoholism defines excessive alcohol intake as the consumption of five unit (1 unit = 10 ml or 8 g) or more drinks in a single day or 15 unit or more drinks per week for men, and four unit or more drinks in a single day or eight unit or more drinks per week for women. Nevertheless, these thresholds may vary based on factors such as age groups, sex, and ethnic background[2,3].

Among many potential complications, pellagra, a deficiency disease caused by insufficient intake or absorption of niacin (vitamin B3), characterized by its typical rash and a triad of diarrhea, dermatitis, dementia – more preferably delirium, is a

HIGHLIGHTS

- Niacin (vitamin B3) deficiency can lead to the development of pellagra, a nutritional disorder characterized by various symptoms including dermatitis, diarrhea, delirium, and death if untreated.
- Alcoholic Pellagrous Encephalopathy is a rare neurological and neuropsychiatric manifestation of pellagra, challenging to diagnose, especially in chronic alcohol users with multiple substance use disorders.
- Diagnostic challenges arise due to overlapping symptoms with other alcohol disorders, limited access to healthcare, and socioeconomic factors affecting diagnosis and intervention.
- A 36-year-old substance abuser displayed unusual symptoms, including hallucinations, dermatitis, and cognitive impairment, along with a history of alcohol dependence and seizures.
- Timely recognition of alcoholic pellagrous encephalopathy, addressing socioeconomic barriers, and comprehensive care are essential for better outcomes in individuals with chronic alcohol dependence.

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Sponsorships or competing interests that may be relevant to content are disclosed at the end of this article.

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Annals of Medicine & Surgery (2024) 86:501–506

Received 10 September 2023; Accepted 1 November 2023

Published online 7 November 2023

http://dx.doi.org/10.1097/MS9.0000000000001497

condition often overshadowed by its more common counterparts in the context of alcohol-related disorders, which includes acute alcohol withdrawal, delirium tremens (DT), and Wernicke's encephalopathy. These counterparts often pose a diagnostic dilemma in the evaluation of the etiology for delirium especially with chronic alcohol consumers[2,3].

Pellagra is classically recognized for its dermatological features, but pellagrous encephalopathy, a neurological and neuropsychiatric presentation, is a rare and challenging manifestation, particularly when occurring in conjunction with AUD. Alcoholic pellagrous encephalopathy refers to a condition where a person who abuses alcohol develops neurological and neuropsychiatric symptoms due to a deficiency of vitamin B3 (niacin), known as pellagra[2,4]. Alcohol is the 11th leading cause of Disability Adjusted Life Years (DALYs) in Nepal. According to a 2013 WHO STEPS survey, 17% of the population in Nepal had consumed alcohol in the past month, with a higher prevalence among males (28%) than females (7.1%). Binge drinking was observed in 18.6% of males and 2.9% of females. The cultural acceptance of alcohol varies among ethnic groups in Nepal, and alcohol consumption has increased across different age groups and ethnicities. Despite a relatively high rate of alcohol abstinence, those who do consume alcohol in Nepal drink nearly five times more on average (28.8 l of pure alcohol) compared to the global average (6.2 l)[7,8].

However, it is essential to recognize that the diagnostic landscape of AUD and its associated complications can be influenced by a multitude of factors, including the socioeconomic status of the patient, limited drug availability, and diagnostic tools. The socioeconomic condition of the patient can pose diagnostic challenges, affecting access to healthcare, diagnostic tools, and timely management. In resource-constrained settings, individuals with AUD, particularly those from low socioeconomic backgrounds, may face barriers to receiving comprehensive care, potentially delaying accurate diagnoses and effective interventions[5,6,9,10]. This case report explores the intricate relationship between alcohol misuse and pellagra, highlighting the atypical neurological presentation of pellagrous encephalopathy in a patient with a prolonged history of alcohol dependence and multiple substance use disorders. The work has been reported in line with the Surgical CAse REport (SCARE) 2020 criteria[11].

Case presentation

A 36-year-old male, of low socioeconomic status, presented to the hospital accompanied by his brother-in-law, with a history of substance abuse spanning over two decades. He had a gradual onset of symptoms, primarily driven by substance use and peer influence. His history included 21 years of nicotine consumption, 18–19 years of alcohol consumption (2–3 l daily), 5–6 years of cannabis use, and 4–5 years of consumption of oral tablets (tramadol, dicyclomine, and sirup cyproheptadine). He reported decreased food intake for 2–3 months, generalized weakness for 1 month, tremors, impaired vision, and an inability to walk for 15–20 days. He also experienced auditory and visual hallucinations, altered sensorium, and delusional thinking for the past 10–12 days.

Approximately one and a half years ago, the patient was hospitalized for tremors, confusion, altered sensorium, and hallucinations, and diagnosed with DT and seizures. Despite a period of abstinence, he relapsed due to peer influence suggesting a history of complicated withdrawal. Over the past year, he experienced multiple episodes of hallucinations, fearfulness, sleep disturbances, and tremors with two episodes of alcohol-induced psychotic disorder. His alcohol consumption averaged 1.5–2 l daily.

In the past 2 months, he had reduced food intake, developed hyper-pigmented, pruritic upper limb lesions, and experienced generalized weakness, tremors, visual impairment, and gait issues. He also displayed fluctuating consciousness, sleep disturbances, restlessness, disorientation, and misidentification of time and place. There were no gastrointestinal symptoms. Two to three days before these symptoms, his alcohol consumption had decreased by 50%, leading to fearfulness, hallucinations, disturbed sleep, and fluctuating consciousness within 24 h. His last alcohol intake occurred 13–14 days ago. His overall functioning was significantly impaired, with decreased self-care, heightened levels of anxiety and restlessness. He experienced a substantial decline in overall functioning, which was accompanied by reduced self-care abilities, as well as heightened levels of anxiety and restlessness.

His initial treatment involved lorazepam from a local clinic, but he was eventually referred to the ER and diagnosed with DT. The patient had a family history of alcohol-related disorders, with his brother currently in a rehabilitation center, and a history of frequent arguments with his wife under the influence of substances. Despite a challenging presentation, his premorbid personality was characterized as euthymic and well-adjusted.

On examination, the patient presented with physical and neurological symptoms, including tremors, tremulousness, horizontal and vertical nystagmus, and difficulty in motor tasks indicative of cerebellar dysfunction. His Clinical Institute Withdrawal Assessment for Alcohol (CIWA) score was 22 out of 67, and a vital signs showed slightly elevated blood pressure and heart rate. A local examination revealed erythematous scaly lesions on his upper limbs (as shown in Fig. 1). Cerebellar tests were mostly unsuccessful, with an inability to perform tasks like the finger-to-nose test, alternate hand movement, and straight-line walking. His mental state showed increased psychomotor activity, anxiety, limited insight, impaired attention, loss of recent memory, and impaired orientation to time and place.

The laboratory investigations included a complete blood count, liver function test, thyroid function test, renal function

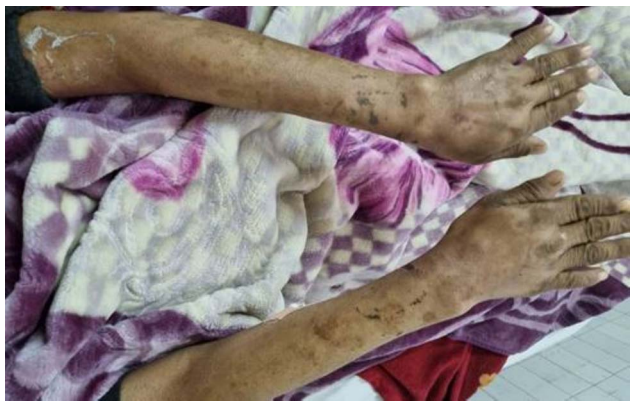

Figure 1. Erythematous scaly itchy lesion over bilateral upper limbs.

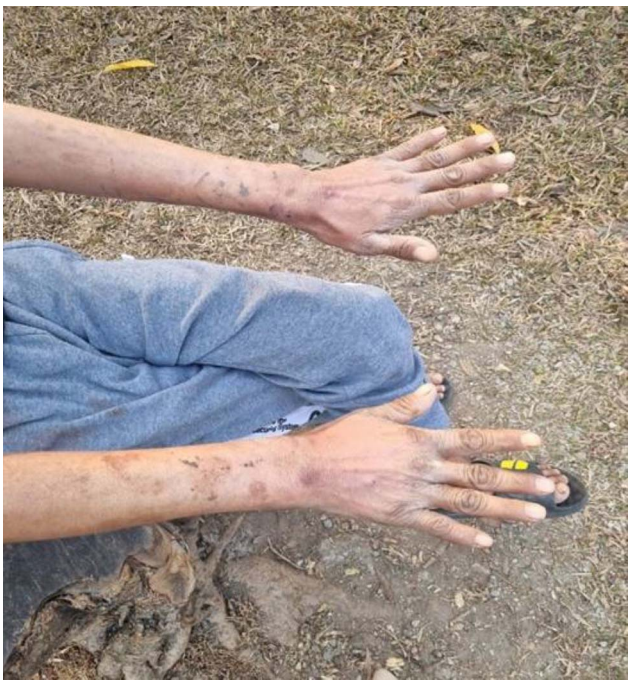

Figure 2. Day 7 of admission and vitamin B3 supplementation.

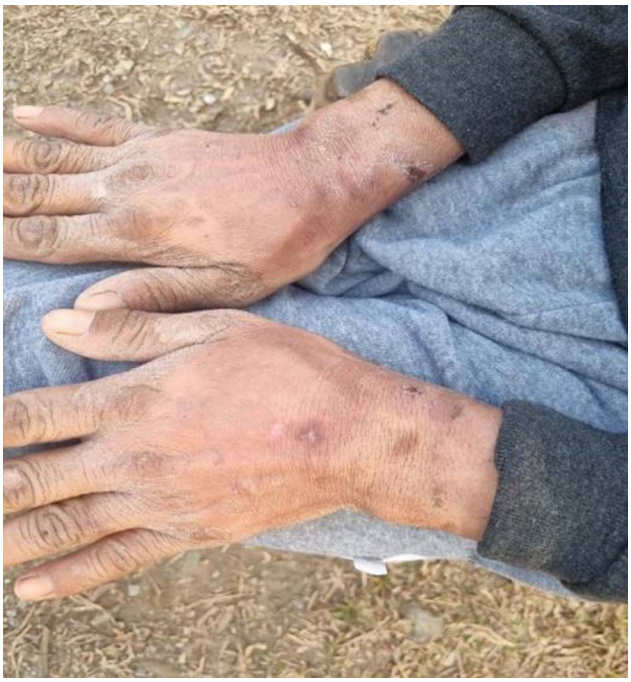

Figure 3. Day 10 of admission.

test, serum electrolytes, random blood sugar, and coagulation profile. The relevant findings included macrocytic anemia with red blood cell count 3.20, Hemoglobin (Hb) 10.9 gm/dl, mean corpuscular volume 105.6 fl and the rest of the other findings being normal including nonreactive results for HIV, HBsAg, and HCV. Additionally, an abdominal ultrasound revealed a fatty liver with slightly deranged values of alanine aminotransferase and aspartate aminotransferase.

Following an initial evaluation, the patient was provisionally diagnosed with Alcohol dependence syndrome (ADS), characterized by complicated withdrawal and DT, coupled with concurrent anemia and a fatty liver. Additionally, the patient exhibited pellagrous dermatitis. Differential diagnoses included ADS with Wernicke-Korsakoff Syndrome (WKS) and pellagrous dermatitis, considering the history of complicated withdrawal (DT and seizures) and multisubstance dependence (nicotine, cannabis, and opioids). Furthermore, ADS with pellagrous encephalopathy was considered due to the ongoing neurological symptoms of the patient.

The management plan of the patient included the administration of diazepam and intravenous fluids on the first day, with thiamine and niacinamide supplementation. Subsequently, on the second and third days, the patient required lower doses of diazepam to maintain sedation. By the fourth and fifth days, sedation was no longer necessary as the patient became mostly sedated without medication. On the fifth day, the transition to oral diazepam began, and it was gradually tapered and eventually discontinued. Additionally, the patient received an oral formulation of vitamins containing 50 mg of nicotinamide (two tablets thrice daily) and oral thiamine at a dosage of 300 mg per day, as advised by the dermatology team. The physical and neurological symptoms of the patient showed improvement, and the skin lesions (as shown in Figs 2 and 3) and delirium resolved. Overall, the management process posed challenges related to diagnostic limitations, drug availability issues, and patient-related factors, but the cooperation and progress of the patient were promising. Diazepam was eventually stopped on the 8th day of admission.

Several diagnostic challenges were encountered during the management of this patient. The lack of access to radio imaging, primarily due to the low socioeconomic status of the patient, hindered a comprehensive assessment of their condition. Additionally, the absence of serum and urine analysis for niacin limited our ability to monitor and adjust treatment effectively. Drug-related challenges, such as the unavailability of appropriate dose formulations for injectable and oral nicotinamide and a shortage of parenteral thiamine, added complexity to the treatment process. Moreover, patient and patient-party challenges, including a short hospital stay, inadequate motivational interviewing, and instances of lost follow-up, further complicated the management of the patient's condition.

Discussions

The presented case involves a 36-year-old male with a long-standing history of substance abuse, including alcohol, nicotine, cannabis, and oral tablet/sirup intake. His initial presentation to the hospital was marked by a gradual onset of symptoms, including generalized weakness, tremors, impaired vision, and an inability to walk, which progressed to auditory and visual hallucinations, altered sensorium, delusional thinking, and decreased food intake. These symptoms, although severe, were atypical for an individual with a history of chronic alcohol abuse, leading to a diagnostic challenge.

Alcoholic pellagrous encephalopathy is a rare and complex neurological disorder that arises in individuals with a history of chronic alcohol abuse, characterized by severe niacin (vitamin

B3) deficiency. It presents a diagnostic challenge due to its atypical symptoms and overlaps with other alcohol-related disorders, such as ADS and WKS[7,8].

Alcoholic pellagrous encephalopathy presents as a niacin deficiency-related condition with characteristic symptoms, known as the '4 D's': dermatitis marked by rough, red, and sun-exposed areas of the skin becoming inflamed (similar to the above findings), persistent diarrhea, dementia (including memory loss and confusion) more preferably delirium, and potential fatality if left untreated, emphasizing the need for prompt medical intervention[2,4].

Specific to the encephalopathy, there may be additional neurological signs such as tremors, ataxia (lack of muscle control), and other alterations in mental state. It is important to note that symptoms can vary widely, and some patients may present without the classic signs, which further complicates diagnosis[12].

In cases of alcoholic pellagrous encephalopathy, it is quite common to observe confusion, hallucinations, altered clarity of consciousness, oppositional hypertonus, ataxia, and myoclonus, with the latter appearing in roughly half of the patients. Despite this, such symptoms are not typically witnessed in other varieties of alcohol-induced encephalopathies that often concurrently occur with alcoholic pellagra[13].

In those struggling with chronic alcohol consumption, pellagra often manifests without the characteristic rash, unlike in our case, or exclusively with delirium[14]. This situation can cause pellagrous encephalopathy to be missed as a standalone diagnosis or as a concurrent ailment alongside DT and Wernickes' encephalopathy (WE). Considering pellagra as a potential differential or accompanying diagnosis is crucial because it is a manageable source of disease and possible death among these individuals[15].

Niacin (vitamin B3) deficiency can lead to the development of pellagra, a nutritional disorder characterized by various symptoms. A significant decrease in the food intake of the patient could have contributed to the nutritional deficiency in our case. Niacin primarily functions as a coenzyme in numerous biological redox reactions in the form of nicotinamide. In regards to the mechanism of niacin, it contributes to the functioning of the Nicotinamide Adenine Dinucleotide Phosphate (NADP)-dependent kynurenine hydroxylase. Without sufficient niacin, as seen in pellagra patients, the activity of this enzyme is inhibited. Niacin deficiency leads to inadequate production of Nicotineamide Adenine Dinucleotide (NAD) and NADP coenzymes, impairing energy production in cells, including neurons, and affecting the synthesis of important neurotransmitters in the brain. This disrupts the metabolic activity of the brain, causing neurological symptoms and other disease processes contributing to pellagrous encephalopathy[16].

Primary causes include a dietary deficiency of tryptophan (an essential amino acid) and niacin. Secondary causes encompass conditions like chronic alcoholism, chronic colitis, anorexia nervosa, medications, and more. Pellagra can be diagnosed clinically, based on presenting symptoms, although testing for niacin levels or urinary metabolites is available. With treatment, the dermatologic and gastrointestinal symptoms generally resolve quickly, while the response to neurocognitive symptoms can be variable. While, in our case, the patient was out of delirium from the 4th day of admission onwards[14].

Alcohol consumption can trigger pellagra due to the resulting malnutrition, which reduces the availability and absorption of niacin, its precursors, and other essential nutrients. Moreover, alcohol can amplify the negative impact of these nutritional deficiencies through its unique effects. These include metabolic changes due to alcohol metabolism, hindered transformation of tryptophan to niacin, induced zinc deficiency, disruptions in the synthesis of heme, and alterations in glutamate and GABA neuronal activity[16]. This establishes an intricate relationship between chronic alcohol abuse and pellagra manifesting different clinical pictures including various neuropsychiatric symptoms.

Diagnosing pellagrous encephalopathy in alcohol-related disorders can be challenging due to its overlap with conditions like WE. Diagnosis relies on clinical presentation, especially in chronic alcohol abusers, with attention to the classic four D's' (dermatitis, diarrhea, dementia, and death). Additionally, assessing niacin deficiency through laboratory tests measuring niacin or its metabolite, N1-methyl nicotinamide, in urine can aid in diagnosis, which appeared as a limitation in our case[2,11].

In a study by Lopez *et al.*[13] the patient had a similar history of chronic alcohol consumption and presented with signs of pellagra, delusions, and visual hallucinations. Successful treatment with niacin resulted in the improvement of neurological and dermatological injuries, as well as psychotic symptoms. However, the patient was also taking Olanzapine, so the full contribution of niacin treatment in improving psychotic symptoms cannot be fully ascertained. However, the effectiveness of treatment with niacinamide in our case has been reflected.

Neuroimaging like MRI, although not specific to pellagrous encephalopathy, can be employed to exclude alternative causes of the patient's symptoms like WES. However, due to the economic barrier it could not be performed. Maintaining a heightened level of suspicion is vital when dealing with neurological symptoms in chronic alcoholics, as their presentations may not consistently align with the typical clinical profile[17,18]. Electroencephalography (EEG) plays a crucial role in the diagnostic evaluation of APE. Electroencephalography results in pellagrous encephalopathy often display widespread slowing, primarily in the theta range. These findings point to neurocognitive issues resembling delirium rather than dementia in pellagrous encephalopathy[6].

In the presented case, diagnosing the condition of the patient proved to be a formidable challenge due to its atypical presentation and overlapping clinical features with various alcohol-related disorders. Initially, the possibility of alcohol withdrawal delirium (DT) was considered, but several factors, including the gradual onset of symptoms, protracted delirium after alcohol abstinence, and minimal alcohol withdrawal physical signs, conflicted with this diagnosis. Additionally, the presence of neurological signs further complicated the assessment. The consideration of Wernicke-Korsakoff syndrome (WKS) was also contemplated, supported by poor dietary intake, oculomotor deficits, altered mental status, and cerebellar signs. However, the absence of radiological reporting and the presence of dermatitis posed challenges. APE emerged as a potential diagnosis, supported by the presence of dermatitis and delirium, improvement with nicotinamide supplementation, but the absence of other neurological signs such as myoclonic jerks, gastrointestinal symptoms, serum niacin, and urine niacin levels not being assessed due to the unavailability of diagnostic tools created a diagnostic barrier.

In a similar study by Sharma *et al.*[18], a 56-year-old chronic alcoholic presented with progressive weakness in all four limbs, cognitive impairment, spastic quadriparesis, extensor plantars, and cerebellar features. MRI revealed subdural effusion,

subarachnoid hemorrhage, and cortical atrophy. Initially treated with thiamine, the patient's condition worsened. Low serum niacin indicated alcoholic pellagra encephalopathy. Niacin therapy led to significant improvement, despite multiple complicating factors. In contrast, in this study, due to certain barriers like low socioeconomic status and resource unavailability the required investigations like MRI and serum niacin could not be performed. However, with the similar treatment strategies the patient responded well. In light of the diagnostic complexities surrounding pellagrous encephalopathy, clinicians may contemplate a therapeutic trial involving niacin supplementation. A positive response to this treatment can serve as a valuable diagnostic indicator. However, the response to the treatment favored the diagnosis of APE as supported in the study mentioned above.

The patient had a history of chronic alcohol consumption and presented with signs of pellagra, delusions, and visual hallucinations. Successful treatment with niacin resulted in the improvement of neurological and dermatological injuries, as well as psychotic symptoms. However, the patient was also taking Olanzapine, so the full contribution of niacin treatment in improving psychotic symptoms cannot be fully ascertained.

The management of pellagrous encephalopathy within the context of AUDs entails several essential approaches. Firstly, nutritional correction is paramount, involving the rectification of niacin deficiency through high-potency niacin supplements and comprehensive nutritional support. Secondly, addressing the AUD is crucial, encompassing detoxification and the establishment of sustained alcohol abstinence. Thirdly, medical intervention may be necessary to tackle medical symptoms such as seizures or psychoses, potentially requiring medication. Finally, sustained care remains indispensable, ensuring ongoing support for nutritional rehabilitation, maintaining alcohol abstinence, and addressing concurrent mental health issues[2,8,9].

The management involved the administration of diazepam for alcohol withdrawal symptoms, intravenous fluids, thiamine, and niacinamide supplementation. The gradual improvement in the physical and neurological symptoms, along with the resolution of pellagrous dermatitis (as shown in Figs 2 and 3), highlighted the effectiveness of appropriate treatment. However, it is essential to acknowledge the challenges faced during treatment, including drug availability issues and patient-related factors.

As per WHO in adults with pellagra, recommended dosages encompass the oral or intravenous/intramuscular administration of niacin or niacinamide (nicotinamide). Oral dosing permits a daily intake of up to 500 mg, varying based on the severity of the niacin deficiency. Intravenous or intramuscular administration offers flexibility, with dosage ranging from 50 to 100 mg intramuscularly up to five times daily, or 25–100 mg via a slow intravenous infusion twice daily, contingent on the extent of niacin deficiency. The maximum daily dose should not exceed 500 mg. Patients should exercise caution by refraining from alcohol consumption during niacin administration to mitigate the risk of pruritus and flushing. Notably, these recommendations are intended as guidelines and necessitate supervision and prescription by a qualified healthcare professional[19].

This case report emphasizes the need for heightened awareness among healthcare providers regarding the atypical presentations of APE in individuals with chronic alcohol dependence. Early recognition of symptoms, access to necessary diagnostic tools, and prompt initiation of treatment can lead to better outcomes. Furthermore, efforts should be made to address socioeconomic barriers and resources unavailability that may hinder the diagnostic process and treatment accessibility that stood as a potential limitation in the present case.

Conclusions

Alcoholic pellagrous encephalopathy is a rare condition that can manifest with atypical symptoms, making it challenging to diagnose, especially in individuals with a history of chronic alcohol abuse. This case report highlights the diagnostic dilemmas, socioeconomic barriers, and clinical complexities associated with this condition and underscores the importance of early recognition and appropriate management to improve patient outcomes. Additionally, it serves as a reminder of the need for comprehensive care and addressing barriers to diagnosis and treatment in individuals with alcohol-related disorders in a limited resource setting.

Ethical approval

The study is exempt/waived from ethical approval in our institution as it poses minimal risk to the patient and the study is for educational purpose/activities.

Consent

Written informed consent was obtained from the patient for publication and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Sources of funding

Not applicable.

Author contribution

Mr. T.N.Y.: literature review, follow-up the patient, writing the manuscript, and final approval of the manuscript; Dr P.B.C.: literature review, follow-up the patient, writing the manuscript, and final approval of the manuscript; Dr A.B.: literature review, follow-up the patient, writing the manuscript, and final approval of the manuscript; Dr S.L.: literature review, follow-up the patient, writing the manuscript, and final approval of the manuscript; Mr R.K.: literature review, follow-up the patient, writing the manuscript, and final approval of the manuscript.

Conflicts of interest disclosure

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Research registration unique identifying number (UIN)

Not applicable.

Guarantor

Mr. Tek Nath Yogi.

Data availability statement

Not applicable.

Provenance and peer review

Not commissioned, externally peer-reviewed.

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