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Pellagra In Alcohol Dependence Syndrome: A Diagnostic Challenge With Neuropsychiatric Presentation - A Case Report

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Abstract:
Background: *Pellagra is a nutritional disorder caused by niacin deficiency, commonly linked to a poor dietary intake, alcohol dependence, or malabsorption. Although rare today, alcohol use disorder remains a significant risk factor due to impaired nutrient absorption and metabolic disturbances. This report discusses a 40-year-old man with chronic alcohol dependence who presented with characteristic symptoms of pellagra. Dermatological evaluation confirmed skin lesions consistent with niacin deficiency. Prompt treatment with niacin supplementation and nutritional intervention led to significant improvement. This case highlights the importance of recognizing pellagra as a differential diagnosis in an alcohol-dependent individual presenting with neuropsychiatric and dermatological manifestations.*
Keywords: *Pellagra, Niacin Deficiency, Alcohol Dependence Syndrome, Nutritional Deficiency, Casal's Necklace.*

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I. Introduction

Pellagra is a medical condition caused by niacin (vitamin B3) deficiency, historically prevalent in regions with diets deficient in niacin or tryptophan. While the incidence has declined, individuals with alcohol dependence remain vulnerable due to poor nutrition, malabsorption, and metabolic disruptions. Pellagra's classic triad – dermatitis, diarrhoea, and dementia – can mimic other conditions, often leading to misdiagnosis if not considered early in the clinical assessment (1). Prevalence of pellagra varies globally but remains a notable concern among alcohol dependence individuals, with studies suggesting rates as high as 10 – 30 % in chronic alcohol users, particularly in resource-limited settings (2). Identifying and treating this condition promptly is crucial, as untreated cases may progress to irreversible neurological impairment or death (3). Niacin (vitamin B3) is critical in energy production, DNA repair, and cellular metabolism. It is derived from dietary sources or synthesized endogenously from tryptophan. Chronic alcohol use impairs niacin absorption in the gastrointestinal tract, reduces tryptophan conversions, and accelerates niacin depletion (4). Additionally, alcoholic induced hepatic dysfunction may further hinder niacin metabolism, exacerbating deficiency. The resulting niacin deficit disrupts NAD/ NADP dependent enzymatic pathways, impairing oxidative metabolism, mitochondrial function, and skin barrier integrity. This metabolic dysfunction manifests as the characteristic triad of pellagra: dermatitis, diarrhoea and dementia (5).

II. Case Report

A 40-year-old married male from a lower socioeconomic and urban background presented to our psychiatry outpatient department with a 15-year history of alcohol use and a two-month history of irritability, forgetfulness, and social withdrawal. He had been consuming alcohol in a dependence pattern over the 7 to 8 years, averaging 18 units of spirits daily, which escalated to 24 to 36 units daily over the past two years. His food intake decreased significantly resulting in unintentional weight loss, reduced appetite, and occasional diarrhoea. He denied any medical comorbidities or use of medications. On initial examination, he was found to have tachycardia, generalized tremors, and excessive sweating. He had hyperpigmented, rough and scaly plaques over his neck (Casal's necklace), forearms and dorsal hands. Cognitive assessment revealed impaired attention, disorientation, and memory deficits. Laboratory tests revealed haemoglobin level 10.2g/dl, MCV of 103fL, and low serum niacin levels. Liver function tests showed mildly elevated transaminases, while folate, thiamine, and vitamin B12. Peripheral smear, blood sugar level, kidney function test, and serum electrolytes were within normal

levels. ECG and imaging taken, and no abnormalities were noted. The differential diagnosis included pellagra, psoriasis, seborrheic dermatitis, vitamin B12 deficiency, and Wernicke's encephalopathy. Psoriasis was excluded due to the absence of sharply defined plaques, while seborrheic dermatitis was ruled out as there were no greasy scales or scalp involvement. Vitamin B12 levels were normal, and there were no features of Wernicke's encephalopathy such as ataxia, ophthalmoplegia, or confusion. A dermatological consultation confirmed pellagra-related lesions. The patient was treated with a tapering dosage of Lorazepam for detoxification and initially thiamine 500mg in 100 ml Normal saline thrice a day, given along with niacin 300 mg /day, multivitamin supplements, and nutritional guidance. Alcohol cessation counselling was initiated, and the patient agreed to participate in a de-addiction program. His skin lesions, cognitive impairment, and gastrointestinal symptoms improved significantly within four weeks. He maintained abstinence during follow–up and continued nutritional support.

Table 1: Summary of case details

Parameters	Details
Age/ sex	40-year-old male
Alcohol history/ Duration	Consuming nearly 24 – 36 units of spirits daily / 15 years
Presenting Symptoms	Irritability, forgetfulness, social withdrawal, diarrhoea, weight loss
Examination Findings	Hyperpigmented, rough, scaly plaques on the neck, forearms, and dorsal hands (Casal's necklace), cognitive impairment.
Differential Diagnoses	Pellagra, Psoriasis, Seborrheic dermatitis, Vitamin B12 deficiency, Wernicke's encephalopathy
Investigations	Hb: 10.2 g/dl, MCV: 103 Fl, low serum niacin, Normal vitamin B12, folate, and thiamine levels
Dermatology Opinion	Confirmed Pellagra – related skin lesions
Treatment	Niacin 300 mg/ day, Multivitamin supplementation, Nutritional guidance, Alcohol cessation counselling
Outcome	Improved skin lesions, cognitive functions, and gastrointestinal symptoms after four weeks. Maintained abstinence during follow-up.

III. Discussion

Pellagra remains a potential diagnosis in an individual with chronic alcohol dependence, particularly when neuropsychiatric and dermatological symptoms coexist. Chronic alcohol use depletes niacin reserves by impairing tryptophan conversion, reducing gastrointestinal absorption, and increasing the risk of niacin deficiency (vitamin B3) (6). Pellagra's dermatological features often mimic other conditions, with symmetric photosensitive lesions typically appearing on sun-exposed areas (4). Neurological manifestations may include confusion, irritability, or even psychosis, which can resemble alcohol withdrawal or other mental health disorders. Gastrointestinal symptoms such as diarrhoea and glossitis further complicate diagnosis (7).

Prompt diagnosis is essential as untreated pellagra can progress to coma or even death. Niacin supplementation, combined with nutritional correction and alcohol cessation, significantly improves prognosis. In this case, dermatological consultation played a key role, confirming the diagnosis and guiding the treatment. This case emphasizes the need for clinicians to consider pellagra in alcohol dependent individuals presenting with cognitive decline, skin lesions, and gastrointestinal disturbances. Early recognition and appropriate management can prevent life-threatening complications.

IV. Conclusion

This case highlights the importance of recognizing pellagra as a potential diagnosis in patients with chronic alcohol dependence. Timely diagnosis and niacin supplementation resulted in significant clinical improvement. Dermatological evaluation was pivotal in confirming the diagnosis and ensuring targeted treatment. Clinicians should maintain a high index of suspicion for pellagra in vulnerable populations to prevent long-term complications.

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