International Journal of Research in Dermatology
Damodaran D et al. Int J Res Dermatol. 2025 Jan;11(1):57-60
http://www.ijord.com

DOI: https://dx.doi.org/10.18203/issn.2455-4529.IntJResDermatol20243877

Case Report

Is this glove and boot protective: unraveling the mystery?

Devi Damodaran, Rajesh Rajagopalan*, Mohankumar Vethanayagam, Revathi Kandasamy

Department of Dermatology, Government Erode MCH, Perundurai, Tamil Nadu, India

Received: 27 September 2024
Revised: 08 December 2024
Accepted: 17 December 2024

***Correspondence:**
Dr. Rajesh Rajagopalan,
E-mail: rajeshderma@gmail.com

ABSTRACT

Pellagroid dermatitis, a rare manifestation of niacin deficiency exacerbated by chronic alcoholism, presents with distinct cutaneous and systemic symptoms. We report a case of a 42-year-old male with a history of daily alcohol consumption and heavy smoking, presenting with characteristic skin lesions predominantly over sun-exposed areas. The patient exhibited a spectrum of symptoms including dermatological manifestations, gastrointestinal disturbances, and neuropsychiatric symptoms. Diagnostic workup revealed hypoalbuminemia, mild elevated CRP, and microcytic hypochromic anemia, consistent with niacin deficiency. Treatment included niacinamide supplementation, B12 injections, proton pump inhibitors, and psychiatric counseling. Long-term management is essential to prevent relapse and associated complications.

Keywords: Pellagra, Dermatitis, Niacin deficiency, Alcoholism, Hyperpigmentation, B-complex vitamins

INTRODUCTION

Pellagra, a nutritional disorder caused by niacin (vitamin B3) deficiency, continues to be a relevant clinical concern, particularly in populations with limited access to a balanced diet. Described for the first time by Don Gaspar Casal in 1735, and later named by Frapolli in 1771, pellagra is characterized by the classic "4 D's" dermatitis, diarrhea, and dementia, which may progress to death if left untreated.[1] Although historically linked to diets heavy in maize, modern cases have been identified in individuals with chronic alcoholism, malabsorption syndromes, gastrointestinal surgeries, and specific genetic conditions, such as Hartnup disease.

Despite its potential for prevention through adequate niacin intake, pellagra remains a significant health issue in vulnerable populations worldwide.[2,3] By the early 20th century, South Carolina alone had reported over 30,000 cases of pellagra, with a mortality rate of 40%. Although much progress has been made in the understanding of the disorder, the clinical picture of pellagra is still complicated by diverse contributing factors. These include impaired niacin or tryptophan absorption, use of certain medications, and irregular eating patterns, which continue to make diagnosis challenging.[4] The characteristic cutaneous manifestations of pellagra include erythema, hyperpigmentation, hyperkeratosis, and in severe cases, bullous lesions. Alongside dermatologic signs, gastrointestinal and neuropsychiatric symptoms such as abdominal pain, cognitive decline, and depression are commonly observed.[1-3]

CASE REPORT

A 42-year, married male, working in garment company, presented with black-colored thickened, reddish to whitish skin lesions over both photo-exposed and unexposed areas (hands, forearms, legs, and feet face, back for one year). He reported burning and mild itching upon sunlight exposure, along with gastrointestinal symptoms (epigastric discomfort, abdominal pain, loose stools) and neuropsychiatric complaints (reduced sleep, headache, tremor). History of alcohol intake, smoking since the age of 13 and also having habit of inadequate, irregular, food in- take. Physical examination revealed

hyperpigmented scaly plaques predominantly on extensor surfaces of limbs, hypopigmented scaly plaques on elbows and dorsum of hands, diffuse hyperpigmentation on face, legs, and dorsum of feet, diffuse hyperpigmented nails (Figure 1 and 2), trunk multiple atropic pigmented scars over T4 &T5 dermatome left side (Figure 3), multiple hyperpigmented scaly papules and plaques on the back (Figure 4) and hyperpigmentation in buccal mucosa.

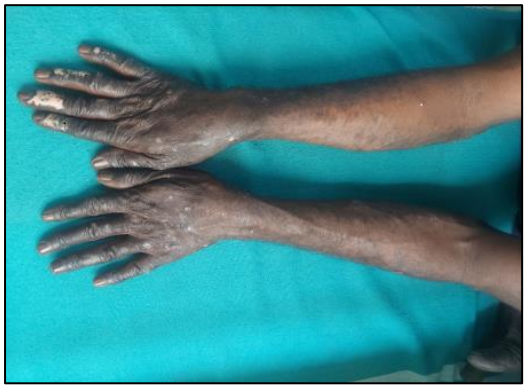

Figure 1: Hyper & hypo pigmented scaly plaques on the dorsum of hand with diffuse hyper pigmented nails.

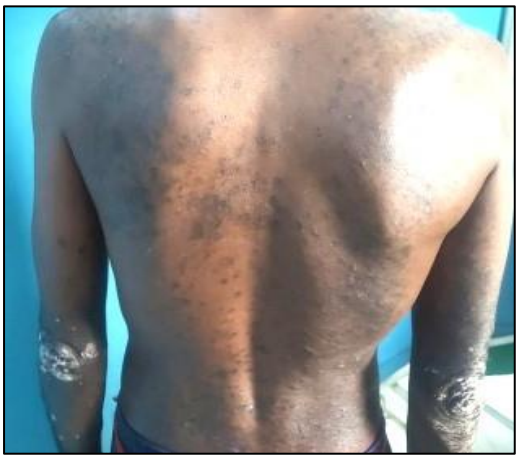

Figure 2: Multiple hyper pigmented scaly papules and plaques on the back.

Basic laboratory investigations showed hypo-albuminemia, mild elevated CRP, and microcytic, hypochromic anemia and other values within normal limits. Skin biopsy done on right forearm, histopathology shows epidermal hyperplasia with hyperkeratosis, parakeratosis, acanthosis, lymphocytic perivascular inflammatory infiltrates, upper part of dermis shows increased dermal collagens and few areas showing collagen destruction (HPE Figure 5 and 6), these findings suggestive of pellagroid dermatitis. Patient treatment initiated with oral nicotinamide 100 mg every 8 hours, tapered upon symptom resolution, supplemented with B12 injections, proton pump inhibitors, acamprosate, chlordiazepoxide along with psychiatric counseling. Long-term management emphasized niacin-rich diet, B-complex vitamins, zinc, and magnesium supplementation

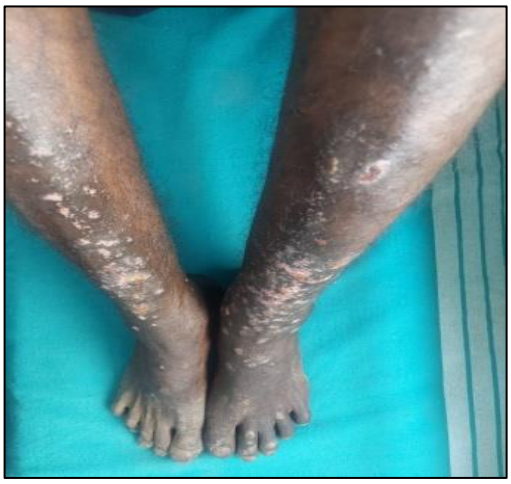

Figure 3: Hyper pigmented scaly plaques on the extensor aspects of lower limbs.

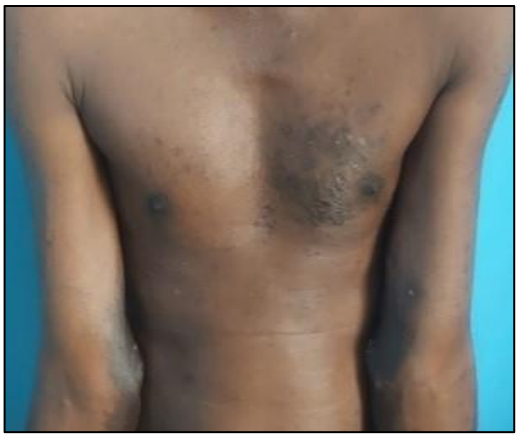

Figure 4: Multiple atrophic pigmented scar over T4 and T5 dermatome on left chest.

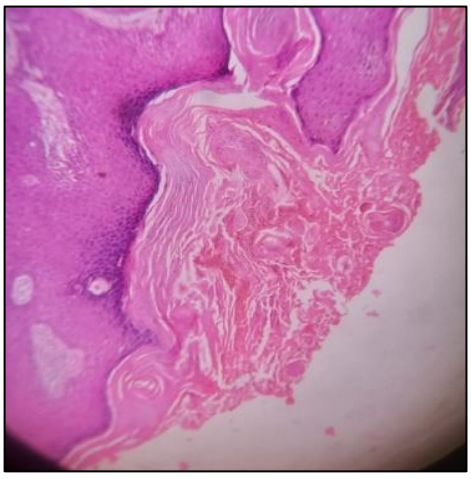

Figure 5: HPE - epidermal hyperplasia with hyperkeratosis parakeratosis acanthosis lymphocytic peri vascular inflammatory infiltrates.

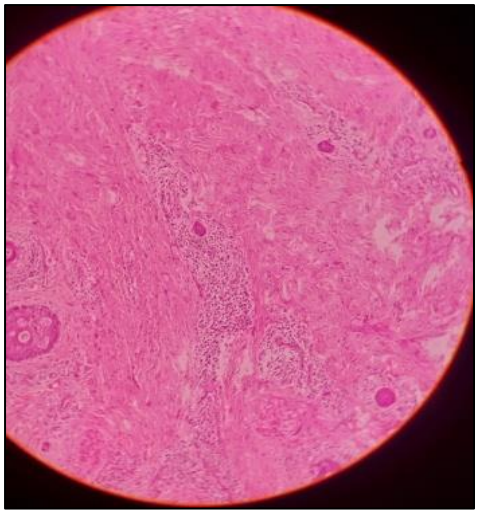

Figure 6: HPE - dermis - increased dermal collagens and few areas showing collagen destruction.

DISCUSSION

Pellagra was first described by Don Gaspar Casal in 1735 and named by Frapolli in 1771.[1] Niacin, a water-soluble B-complex vitamin, serves as a crucial component of coenzymes such as nicotinamide adenine dinucleotide (NAD) and nicotinamide adenine dinucleotide phosphate (NADP). These enzymes play pivotal roles as hydrogen donors and acceptors in metabolic processes involving carbohydrates, proteins, and fatty acids.

Deficiency in NAD can lead to dermatitis, diarrhoea, and dementia, often resulting in death (4 "D"). Pellagra can manifest across all age groups without sex predilection, particularly in populations consuming diets high in grains, maize and corn. Pellagroid dermatitis, association with niacin deficiency often compounded by chronic alcoholism, presents a complex clinical picture encompassing dermatological, gastrointestinal, and neuropsychiatric manifestations.[2] Pellagra may also be associated with conditions like carcinoid syndrome with reduced dietary intake and impaired absorption of niacin or tryptophan being primary predisposing factors.[3]

Other contributing factors include, irregular meal patterns, inadequate intake, and malabsorption due to inflammatory disorders such as Crohn's disease or gastrointestinal surgeries (gastroenterostomy, subtotal gastrectomy, or gastric bypass surgery). Certain medications like 5-fluorouracil, isoniazid, and azathioprine can interfere with tryptophan conversion to niacin, exacerbating the deficiency.[4,5]

Genetic predisposition, such as mutations in the SLC6A19 gene, also predisposes susceptible individuals to pellagra. Hartnup disease, characterized by cerebellar ataxia, psychiatric symptoms, aminoaciduria, and pellagra-like dermatitis, further exemplifies the spectrum of niacin-related disorders.

The characteristic cutaneous manifestations of pellagra include a bilateral, symmetrical rash predominantly localized to sun-exposed areas such as the dorsum of hands, "V" of the neck, face, radial aspects of forearms, and exposed skin on legs and feet.[6-8] Initially presenting as painful or pruritic erythema and edema, severe cases may exhibit vesicles and bullae. Chronic lesions are marked by pronounced hyperpigmentation, hyperkeratosis, and skin dryness and roughness.

Additional signs include the "glove or gauntlet, boots" and "Casal's necklace" sign, as well as symmetrical rash " butterfly distribution" on the face, cheilitis, glossitis, oral or perianal ulcers, and the "half and half " nail sign.[1-3] Gastrointestinal symptoms such as abdominal pain, headache, irritability, fatigue, insomnia, apathy, depression, impaired memory, and psychosis can also manifest.[7] Investigation-reduced niacin urinary metabolites such as N-methylnicotinamide and 2-pyridone levels (Normal >17 micromol/day , in niacin deficiency less than 5.8 micromol /day) Diagnosis of pellagra relies on typical clinical presentation and rapid response to niacin supplementation.

Differential diagnosis includes disseminated DLE, chronic lichenoid eczema, lichenoid psoriasis, photosensitive drug eruption, PCT, chronic actinic dermatitis, pyridoxine deficiency, Hartnup's disease. Skin biopsy can be done to rule out other disease but it is not routinely done.[1,9-11] Histopathological features-initial lesions shows psoriasiform epidermal hyperplasia with hyperkeratosis, parakeratosis, and lymphocytic perivascular inflammatory infiltrates. End stage epidermal atrophy, hypermelanosis, vascular ectasia, and sebaceous atrophy can be seen.[1]

The clinical presentation of pellagroid dermatitis in our patient included characteristic skin lesions "glove and boots sign" and these lesions progressed from erythematous and oedematous stages to chronic phases marked by hyperpigmentation, hyperkeratosis, skin roughness and along with accompanying symptoms of burning sensation and mild pruritus exacerbated by sunlight exposure, are hallmark features of pellagra.

Beyond the dermatological manifestations, our patient exhibited systemic symptoms like epigastric discomfort, abdominal pain, and frequent loose stools, suggestive of gastrointestinal involvement. Neuropsychiatric symptoms such as reduced sleep, intermittent headaches, and tremor further underscored the multifaceted impact of niacin deficiency on neurological function.

These findings align with previous literature describing pellagra's triad of dermatitis, diarrhoea, and dementia, highlighting the need for comprehensive assessment and management. The primary treatment involves replacing nicotinamide 100 mg thrice a day and tapered until all symptoms resolve.[1] For severe cases, parenteral administration of 1 gm of nicotinamide 3 to 4 times per

day is recommended. Along with supplementing other B-vitamins, Zinc, Magnesium and diet rich in niacin, calories, and proteins is advised.

CONCLUSION

Early recognition and prompt treatment of pellagra are vital to prevent irreversible complications. Healthcare providers must remain vigilant for nutritional deficiencies in patients presenting with dermatological and multi-systemic symptoms. This case highlights the importance of maintaining a balanced diet, particularly in high-risk populations such as chronic alcoholics. Long-term follow-up and patient education are key to preventing relapse and ensuring the best possible outcomes.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Karthikeyan K, Thappa DM. Pellagra and skin. International J Dermatol. 2002;41:8.
2. Oldham MA, Ivkovic A. Pellagrous encephalopathy presenting as alcohol withdrawal delirium: a case series and literature review. Addiction Science & Clinical Practice. 2012;7:1-9.
3. Rubin de Celis Ferrari AC, Glasberg J, Riechelmann RP. Carcinoid syndrome: update on the pathophysiology and treatment. Clinics. 2018;73:490.
4. Garg G, Khopkar U. Ethionamide-induced pellagroid dermatitis resembling lichen simplex chronicus: A report of two cases. Indian J Dermatol, Venereol and Leprol. 2011;77:534.
5. Kura MM, Ramteke MN, Dhillon SK. Second-line antitubercular therapy with ethionamide and pyrazinamide causing pellagroid dermatitis presenting as diffuse palmoplantar keratoderma. JAAD Case Reports. 2021;17:77-80.
6. Wan P, Moat S, Anstey AJ. Pellagra: a review with emphasis on photosensitivity. British Journal of Dermatology. 2011;164(6):1188-200.
7. Sharma G, Sandhu JK. A man with well-demarcated, erythematous and scaly plaques over sun-exposed sites. Cosmoderma. 2022;5:2.
8. Luthe SK, Sato R. Alcoholic pellagra as a cause of altered mental status in the emergency department. J Emergency Med. 2017;53(4):554-7.
9. So JK, Hamstra A, Calame A, Hamann CR, Jacob SE. Another great imitator: allergic contact dermatitis differential diagnosis, clues to diagnosis, histopathology, and treatment. Current Treatment Opt Allerg. 2015;2:333-48.
10. Doménech MV, Sanz ES, Marco CP, del Caz MP, Cerdá OB, Albert JS. Pellagra. A challenging differential diagnosis in burn injuries. J Tis Viabi. 2014;23(1):37-41.
11. Ladoyanni E, Cheung ST, North J, Tan CY. Pellagra occurring in a patient with atopic dermatitis and food allergy. J European Acad Dermatol and Venereol. 2007;21(3):394-6.

Cite this article as: Damodaran D, Rajagopalan R, Vethanayagam M, Kandhasamy R. Is this glove and boot protective: unraveling the mystery?. Int J Res Dermatol 2025;11:57-60.