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Case Report

Pellagra-like presentation of borderline tuberculoid Hansen disease with a Type 1 lepra reaction: a diagnostic mimic

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Abstract:
Since immune responses, secondary infection and prolonged scratching can modify the clinical appearance of Hansen disease, this remains an important consideration for the diagnosis of chronic dermatoses. A 42-year-old man presented with intensely pruritic lesions of the neck, shoulders, upper chest and trunk since five years associated with chronic diarrhoea, progressive memory impairment and loss of weight. On examination, hyperpigmented, scaly and excoriated plaques were seen over the neck and upper trunk, multiple crusted or eroded papules and plaques were seen over the chest and there were additional scaly plaques seen over the posterior trunk and one lower limb. Photosensitive eruption was seen along with neurocognitive changes and diarrhoea, which led to a working diagnosis of pellagra; other diagnoses included psoriasis and pemphigus foliaceus. A bacterial culture obtained from an eroded lesion was positive for methicillin-sensitive Staphylococcus aureus. No clinical response was seen with nutritional supplementation, nicotinamide, systemic and topical corticosteroids, emollients and directed antibiotic therapy. Biopsy of skin from the chest showed that the dermis had well formed tuberculoid granulomas, while the epidermis showed parakeratosis, acanthosis and spongiosis. Near destruction of nerves and adnexal structures, epithelioid histiocytes, Langhans-type and foreign-body-type giant cells, a dense lymphocytic rim and prominent superficial dermal oedema were conspicuous features of granulomas, which were perivascular, perineural and periappendageal. This was consistent with finding a borderline tuberculoid Hansen disease and Type 1 lepra reaction. The case describes how a compelling clinical picture of pellagra may mimic leprosy and how careful histopathologic evaluation of a chronic dermatosis, when it fails to improve as expected, can help avoid the diagnosis of leprosy.

Introduction

Sustained efforts have been made for Hansen disease (leprosy) elimination globally, but still, it continues to be reported and mainly caused by Mycobacterium leprae. World Health Organization has stressed the need for continued surveillance and timely detection of the new cases for the interruption and elimination of the transmission and the disease of leprosy [1]. The clinical manifestation depends on the host cell-mediated immune response and is classically divided into tuberculoid and lepromatous disease with immunologically unstable borderline lesions in between the poles [2].

The relevance of borderline disease is highlighted by Type 1, or ‘reversal' reactions which can cause rapid inflammatory changes of already established lesions and can also be peripheral nerve reactions. These reactions are under the control of the stimulation of

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cell-mediated immunity, and recognition that is delayed can lead to avoidable neural damage [3]. In the atypical presentations, histopathology may be the critical test; epithelioid granulomas are typically found in the cases of tuberculoid and borderline tuberculoid disease, while oedema and giant-cell rich inflammation may occur in Type 1 reactions [4,5].

Pellagra is a systemic disorder that results from niacin deficiency or niacin metabolism disturbance and is defined by dermatitis, diarrhoea, dementia and, in late stages, death [6,7]. Often the dermatitis is hyper pigmented and photodistributed, so that a patient with chronic gastrointestinal and neurocognitive symptoms may reasonably be treated for pellagra prior to exploration of alternate diagnosis. We report an atypical case of borderline tuberculoid Hansen disease with a Type 1 reaction with the diagnosis confirmed by skin biopsy and treatment non-response.

Case presentation

A 42-year-old man came to the dermatology services with a 5-year history of severe itching on the neck, shoulders, chest and back. The clinical record shows that it started as small fluid-filled lesions which ruptured by themselves and continued to become thickened, excoriated and eroded with a lesion that was particularly persistent over the left shoulder. He also complained of chronic diarrhoea, gradual decrease in memory, loss of weight, and recurring fever & cough for many years. No history of diabetes mellitus, hypertension, thyroid disease or a previous chronic dermatosis was found.

On physical examination the neck and shoulders and upper chest were covered with multiple hyperpigmented, scaly plaques with prominent excoriation and focally erosions. Confluent lichenified scaling around the anterior neck, multiple discrete crusted or scaly papules and plaques over the upper chest, hyperpigmented scaly change over the posterior upper trunk and scaly plaque over the left ankle/dorsum of the foot were clinically documented. Hair, nails, oral mucosa and genital mucosa were documented as normal. A major limitation was the absence of a complete standardized sensory map, and peripheral nerve examination, from the available records, taking into account the final diagnosis.

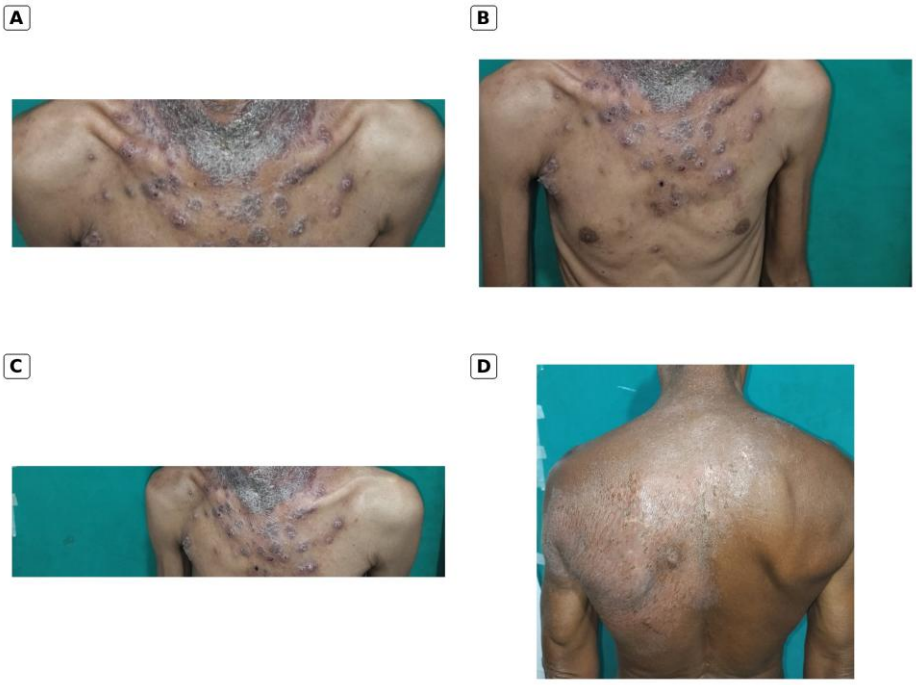

Figure 1. De-identified clinical photographs showing the distribution and morphology of the eruption. (A) Anterior neck and upper chest with confluent hyperpigmented scale and multiple papules/plaques; (B, C) lateral views demonstrating extensive cervical and upper chest involvement; (D) posterior trunk showing broad hyperpigmented scaly change. Facial identifiers were removed by cropping; lesion morphology was otherwise unaltered.

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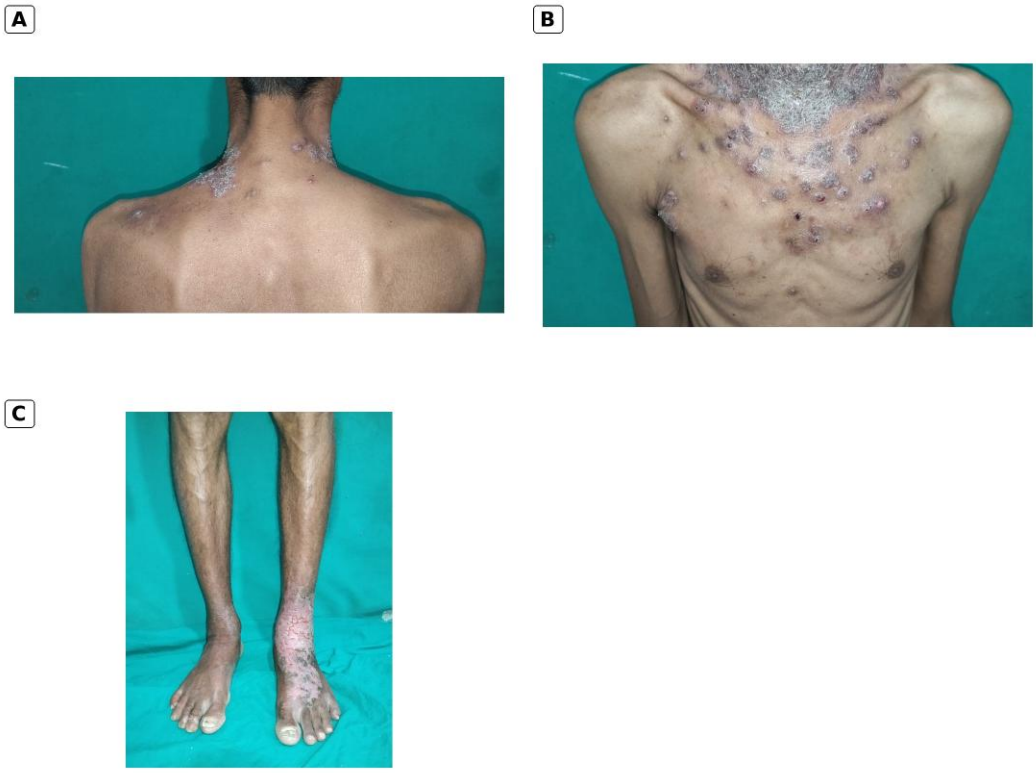

Figure 2. Additional de-identified clinical views. (A) Posterior neck and upper back lesions; (B) closer anterior view showing thick scaling and multiple crusted/eroded papules and plaques; (C) asymmetric scaly and lichenified plaque over the left ankle and dorsum of the foot. The images were cropped only to protect identity.

However, the first clinical impression was in favor of pellagra, given the accentuation of the eruption on exposed areas and the association with chronic diarrhoea and cognitive symptoms. Psoriasis in an immunocompromised patient and pemphigus foliaceus were also taken into consideration. Methicillin-sensitive Staphylococcus aureus (MSSA) was isolated from pus culture from an eroded shoulder lesion. A complete blood count, liver function tests, renal function tests, viral markers, chest radiography, CBNAAT and Mantoux testing had not revealed another unifying diagnosis. Laboratory data obtained were mild anaemia (Hb 11.3 g/dL), normal TSH (0.54 µU/mL), normal total leukocyte count (5.0 × 109/L) and platelet count (204 × 109/L), random glucose (72 mg/dL), HbA1c (5.9%), serum vitamin B12 (184.73 pg/mL), and 25-hydroxyvitamin D (13.51 ng/mL). These abnormalities were not responsible for the complete clinicocutaneous syndrome.

Empirical treatment comprised of oral nicotinamide 350 mg three times a day, multivitamin supplementation, zinc, vitamin B12, oral prednisolone 30 mg/day, topical corticosteroids, and emollients, as well as amoxicillin-clavulanate for secondary bacterial infection and empirical deworming. The skin lesions (also on the chest) didn't resolve as anticipated, so a skin biopsy was collected from the chest.

Histopathology

Sections showed epidermal parakeratosis, acanthosis and spongiosis. The dermis contained well-formed tuberculoid granulomas that were more diffuse in the superficial dermis and discrete in the deeper dermis, with prominent superficial dermal oedema. In the deeper dermis, granulomas were distributed around blood vessels, nerves and cutaneous appendages and produced near-destruction of adjacent nerves and adnexal structures. The granulomas were composed of epithelioid cells with Langhans-type and foreign-body-type giant cells, surrounded by a dense rim of lymphocytes and occasional plasma cells. The subcutaneous tissue was unremarkable; acantholysis, neutrophilic collections in the upper epidermis and cellular atypia were not identified. The integrated histopathological impression was borderline

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tuberculoid Hansen disease with a Type 1 lepra reaction.

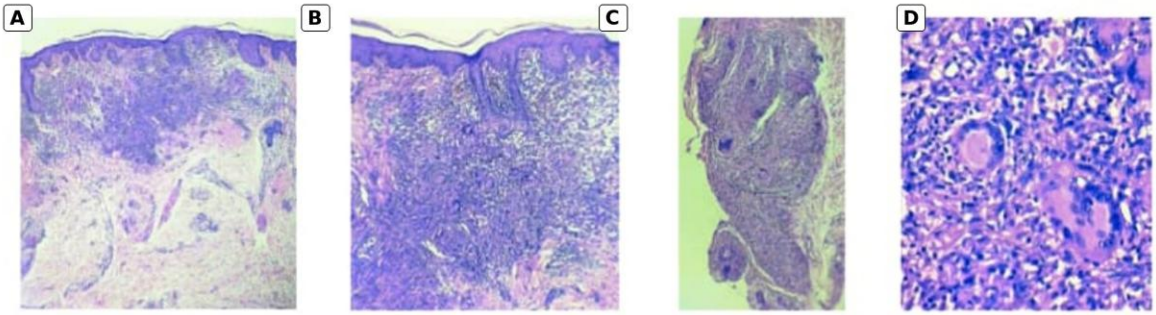

Figure 3. Histopathology images extracted from the diagnostic pathology report. The submitted report described parakeratosis, acanthosis and spongiosis with well-formed tuberculoid granulomas, prominent superficial oedema, and perivascular, perineural and periappendageal granulomatous inflammation with near-destruction of nerves and adnexal structures. Original microscopic magnifications were not stated in the source report and are therefore not assigned here.

Clinical timeline

Time	Key event
~5 years before diagnosis	Pruritic vesicular eruption began over neck/shoulder region and evolved into chronic scaly, excoriated and eroded plaques.
Over subsequent years	Chronic diarrhoea, progressive memory difficulty, weight loss and intermittent fever/cough accompanied the dermatosis.
Initial dermatology assessment	Pellagra was favored; psoriasis and pemphigus foliaceus were also considered. Secondary bacterial infection was evaluated.
During initial treatment	MSSA was isolated from pus culture. Nicotinamide, nutritional supplementation, corticosteroids, emollients, antibiotics and deworming were administered without the expected cutaneous response.
Diagnostic reassessment	Skin biopsy from the chest was performed because of persistent disease.
Histopathologic diagnosis	Perineural/periappendageal tuberculoid granulomas with nerve/adnexal destruction and dermal oedema supported borderline tuberculoid Hansen disease with a Type 1 lepra reaction.

Discussion

The diagnostic significance of the case is the power of the initial competing diagnosis. The diagnosis of pellagra was not simply based on a non-specific rash, but the patient had the "classical" clinical domain of a photo-distributed dermatitis, chronic diarrhoea and progressive neurocognitive symptoms. This constellation is still a point of emphasis in modern reviews, though not always, [6,7]. At this point a therapeutic trial of nicotinamide and nutritional replacement was clinically justifiable. However, lack of meaningful change was a stark indicator that the working diagnosis was incomplete or wrong.

Borderline tuberculoid Hansen disease tends to have fewer distinct, well-demarcated plaques and sensory alterations and asymmetrical nerve involvement. This patient's morphology was much less typical. The eczematous and crusted appearance of this Type 1 reaction was likely aggravated by prolonged excoriation/lichenification and secondary infection with MSSA and inflammatory change. Type 1 reactions are most common in borderline cases and can cause increased erythema, oedema and clinical activity of established lesions [3]. On a histological

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basis, oedema and increased granulomatous inflammation are known features of the reversal reactions [4].

The critical factor was not the granulomatous dermatitis alone, but the distribution around nerves and adnexal structures, accompanied by destructive involvement of the nerves. With nonclassic clinical presentations, the organization of epithelioid granulomas and inflammation around nerves in the skin are significant clues in the diagnosis of tuberculoid-spectrum leprosy [4,5]. Well formed tubercular mass, perineural / periappendageallocalisation, near destruction of nerves and appendages, dense lymphocytic rims as well as prominent oedema were in favour of borderline tubercular disease with a Type 1 reaction in the present biopsy. The lack of acantholysis excluded pemphigus foliaceus and the granulomatous perineural pattern ruled out pellagra.

The leprosy is a well-studied diagnostic mimic. The disease may be confused with sarcoidosis [8], mycosis fungoides [9], granuloma annulare [10] and cheilitis granulomatosa [11]. One common theme of these reports is that, when the morphology and response to treatment are incongruent, examinations of the tissue can provide insight into the clinical situation that is undetected in the clinic. A pellagra-like presentation seems to be uncommon and the co-occurrence of cognitive symptoms and diarrhoea with this patient added to the anchoring bias of nutritional deficiency.

This case also demonstrates a secondary positive microbiological result which can affect the diagnostic process. The MSSA isolated from an eroded lesion was clinically relevant and could explain only superinfection, and not the chronic distribution, scaling or systemic symptom complex. Likewise, low level of vitamin D and low-normal level of vitamin B12 were nutritional observations, but did not prove pellagra. So the diagnostic turning point was a genuine re-evaluation and not yet more empiricism in the treatment of the condition.

There are limitations. None of the source records included contained all the standardized sensory examination, peripheral nerve palpation findings, and bacillary index. They also did not report on the subsequent multidrug-therapy regimen, or long-term neurologic or dermatologic outcome. These elements are missing, and they should not be reconstructed. However, histopathological evidence of perineural and periappendageal tuberculoid granulomatous inflammation was very characteristic and set the main diagnostic teaching point.

Conclusion

In endemic areas, Hansen disease should be included in the differential diagnosis of chronic inflammatory/dehydrated appearing dermatoses, especially when the clinical profile is atypical for the assumed diagnosis, or when the clinical course is prolonged. In this patient, a convincing pellagra-like syndrome did not lead to an early diagnosis until a destructive perineural tuberculoid granuloma was revealed by skin biopsy and features of a Type 1 reaction were seen. Early biopsy of persistent atypical lesions may reduce the delay in diagnosis and can help prevent permanent nerve damage.

Declarations

Patient consent: Written informed consent was documented for publication of the case and accompanying clinical details. All clinical photographs included in this manuscript were cropped to remove facial identifiers; no patient name, registration number, address or other direct identifier is shown.

Competing interests: The authors declare no competing interests.

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Data availability: The clinical information supporting this report is contained within the patient record and diagnostic pathology report. Additional patient-level data are not publicly shared to preserve confidentiality.

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