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A Case Report of Lichen Amyloidosis

Bedour A. AlShammari [1], Alsadat Mosbeh [2], Abeer Albazali [3]

1. Internal Medicine, Farwaniya Hospital, Kuwait City, KWT 2. Dermatology/Dermatopathology, Faculty of Medicine, Al-Azhar University, Cairo, EGY 3. Dermatology, Farwaniya Hospital, Kuwait City, KWT

Corresponding author: Bedour A. AlShammari, bedourawadh@gmail.com

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Abstract

Lichen amyloidosis (LA) is a primary localized cutaneous amyloidosis that closely resembles other chronic dermatological disorders, potentially delaying diagnosis. We report the case of a 60-year-old man with hypertension and chronic kidney disease who was admitted in 2020 with left leg pain and swelling. Investigations ruled out deep vein thrombosis, while CT revealed diffuse subcutaneous edema. Cellulitis was suspected, and the patient was subsequently discharged with analgesia and outpatient follow-up with vascular surgery and dermatology. From 2021 onward, his symptoms were attributed to stasis eczema and lymphedema. Clinical examination revealed multiple hyperpigmented papules on the left lower limb, with additional involvement of the upper back, trunk, and arms. Laboratory investigations showed an eosinophil count of 20.2%, baseline creatinine levels, and normal electrolyte and liver function tests. Due to persistent symptoms, a skin biopsy was obtained from a lesion on the lower back. Histopathological examination revealed hyperkeratosis, acanthosis, elongated rete ridges, increased melanin deposition, and superficial perivascular lymphohistiocytic infiltrates with melanophages. Amyloid globules within the widened papillary dermis stained positively with Congo red, confirming the diagnosis of LA. This case highlights how LA may be misdiagnosed as stasis eczema or lymphedema for years. LA should be considered in patients presenting with persistent hyperpigmentation and papular eruptions involving the lower limbs.

Categories: Family/General Practice, Pathology, Dermatology
Keywords: congo red stain, cutaneous amyloidosis, histopathology, hyperpigmented papules, lichen amyloidosis, lymphedema, primary localized cutaneous amyloidosis, skin biopsy, stasis eczema

Introduction

Amyloidosis comprises a group of disorders characterized by the extracellular deposition of proteins in the form of insoluble fibrils. These deposits may be systemic, involving multiple organs, or localized to a single organ, such as the skin. Primary localized cutaneous amyloidosis (PLCA) is confined to the skin without evidence of systemic involvement and is classified into three forms: macular, lichen (LA), and nodular [1,2]. LA is a subtype of PLCA, which is relatively rare [1,2]. LA is more common in men and individuals with darker skin [1]. It typically presents as multiple pruritic, hyperpigmented, and hyperkeratotic papules, most commonly affecting the shins [1]. LA is a chronic dermatological condition with no malignant potential [1]. Treatment is generally unnecessary unless symptoms or cosmetic concerns warrant intervention [2]. A few reports have described a non-pruritic variant, which is rarer than the pruritic form of LA [3,4].

Due to its morphological similarities to other chronic dermatological conditions affecting the lower legs, including hypertrophic lichen planus, prurigo nodularis, and stasis dermatitis, LA is often underrecognized or misdiagnosed. Histopathological confirmation through skin biopsy is frequently required to establish the diagnosis [5]. We present the case of a 60-year-old man with chronic left leg swelling whose hyperpigmented papular lesions were misdiagnosed as stasis eczema and lymphedema for years before a skin biopsy confirmed LA. This case highlights the importance of considering LA in the differential diagnosis and performing a timely skin biopsy in patients with persistent, non-resolving hyperpigmented papules.

Written informed consent was obtained from the patient for publication of this case report, including the accompanying clinical and histopathological images.

Case Presentation

A 60-year-old male with a past medical history of hypertension and chronic kidney disease was initially admitted in 2020 with left leg pain and swelling. An extensive work-up was performed, but the findings were inconclusive. This included an ultrasound Doppler of the lower limb, which was negative for deep vein thrombosis. A CT scan of the leg was obtained (Figure *1*), revealing diffuse subcutaneous edema. The differential diagnosis at that time included, but was not limited to, superficial and deep cellulitis. The patient was subsequently discharged with pain control and recommendations to follow up at the vascular surgery and dermatology outpatient clinics. Since 2021, the vascular surgery and dermatology clinics had initially diagnosed the condition as stasis eczema and lymphedema. On examination, several hyperpigmented papules were noted on the left lower limb, with a few also present on the right lower limb (Figure *2*). His vital signs and laboratory investigations are summarized in Table *1*. The laboratory work-up

was unremarkable except for a serum creatinine level of 117 µmol/L, which was at baseline. A complete blood count revealed eosinophilia (20.2%), while liver function tests were within normal limits.

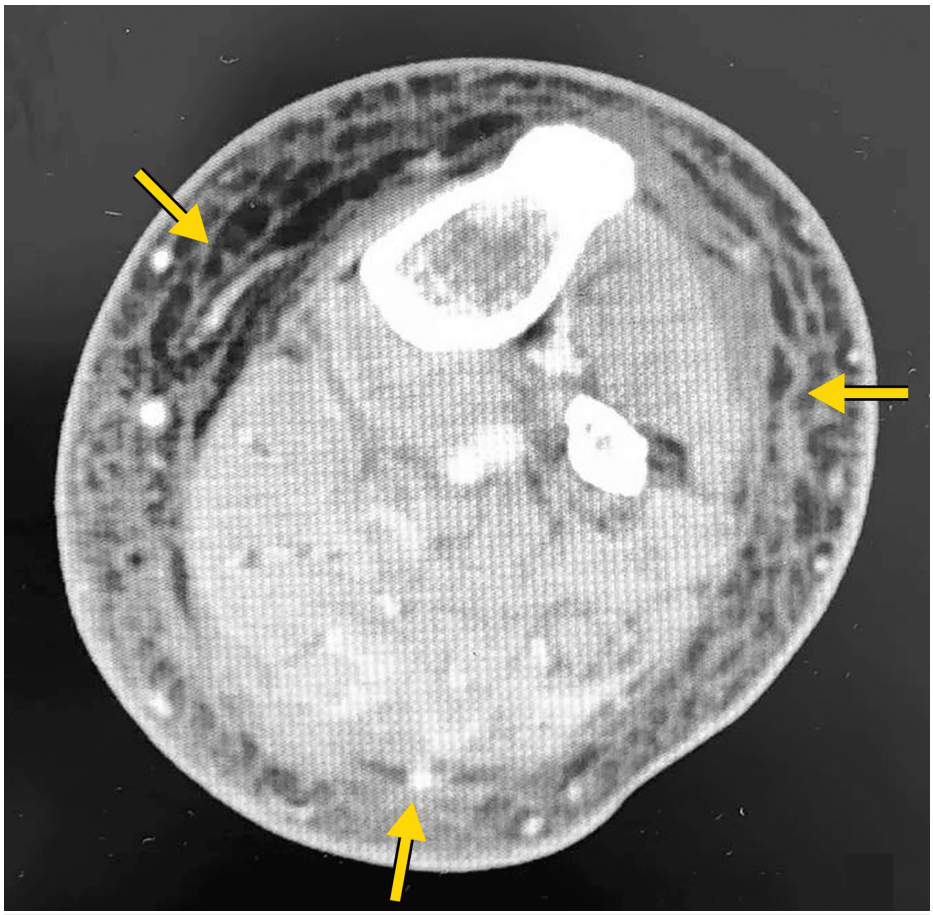


FIGURE 1: Axial contrast-enhanced CT of the left lower leg showing diffuse subcutaneous edema with reticular stranding of the subcutaneous fat (arrows)

CT: computed tomography

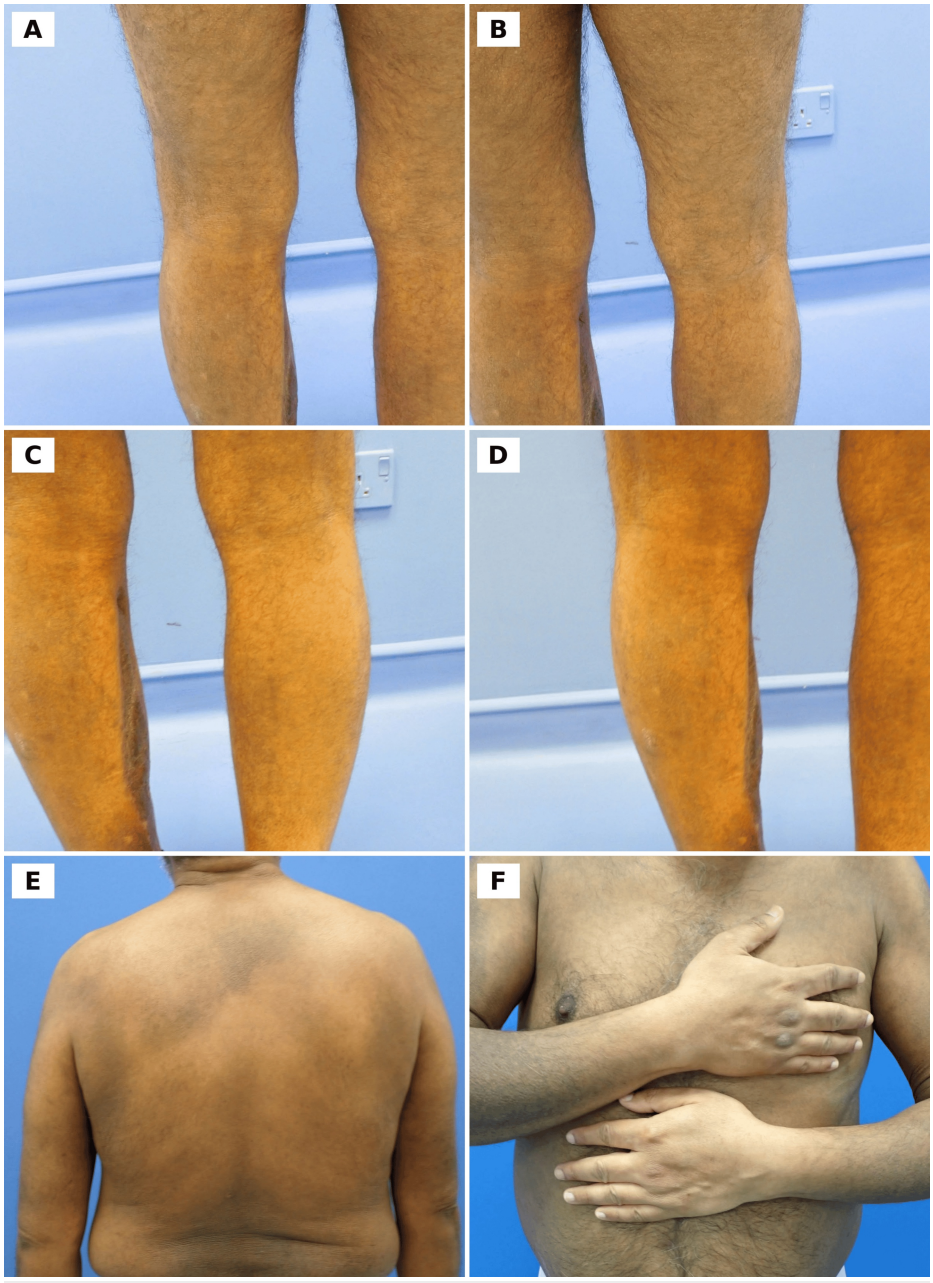


FIGURE 2: Clinical photographs of the patient demonstrating multiple discrete and confluent hyperpigmented papules and plaques

(A) and (B) Posterior view of both thighs and knees, showing hyperpigmented papules with a rippled, thickened appearance over the left posterior thigh and knee. (C) Posterior view of both lower legs, with more extensive involvement of the left calf. (D) Close-up posterior view of the left lower leg, demonstrating coalescing hyperpigmented papules and overlying hyperkeratosis. (E) Upper back, showing an area of reticulated hyperpigmentation over the interscapular region. (F) Anterior trunk and involvement of both arms

Parameter	Patient value	Reference range
Vital signs		
Temperature (°C)	37	36.1–37.2
Heart rate (beats/min)	82	60–100
Blood pressure (mmHg)	134/70	90–120/60–80
Respiratory rate (breaths/min)	18	12–20
SpO_2 on room air (%)	99	≥ 95
Complete blood count		
Hemoglobin (g/L)	16.4	135–175
White blood cell count (×10^9/L)	8.2	4.0–11.0
Eosinophils (%)	20.2 (H)	1–6
Platelets (×10^9/L)	340	150–400
Renal function and electrolytes		
Sodium (mmol/L)	142	135–145
Potassium (mmol/L)	4	3.5–5.0
Urea (mmol/L)	13 (H)	2.5–7.1
Creatinine (µmol/L)	117 (H; baseline)	60–110
Liver function tests		
ALT (U/L)	20	7–40
AST (U/L)	60	10–40
ALP (U/L)	85	40–129
Total bilirubin (µmol/L)	12	3–21
Albumin (g/L)	33	35–50

TABLE 1: Vital signs and laboratory investigations at presentation

H: high; SpO_2: peripheral oxygen saturation; ALT: alanine aminotransferase; AST: aspartate aminotransferase; ALP: alkaline phosphatase

Due to persistent symptoms, the patient returned and underwent a skin biopsy of a lesion on his lower back. Histopathological examination of the biopsy specimen revealed superficial perivascular inflammatory infiltrates composed of lymphohistiocytic cells admixed with melanophages (Figure *3*). The epidermis demonstrated hyperkeratosis and acanthosis, with elongated rete ridges and increased melanin deposition. The widened and dilated papillary dermis contained amyloid globules that stained positively with Congo red (Figure *4*), which was suggestive of LA.

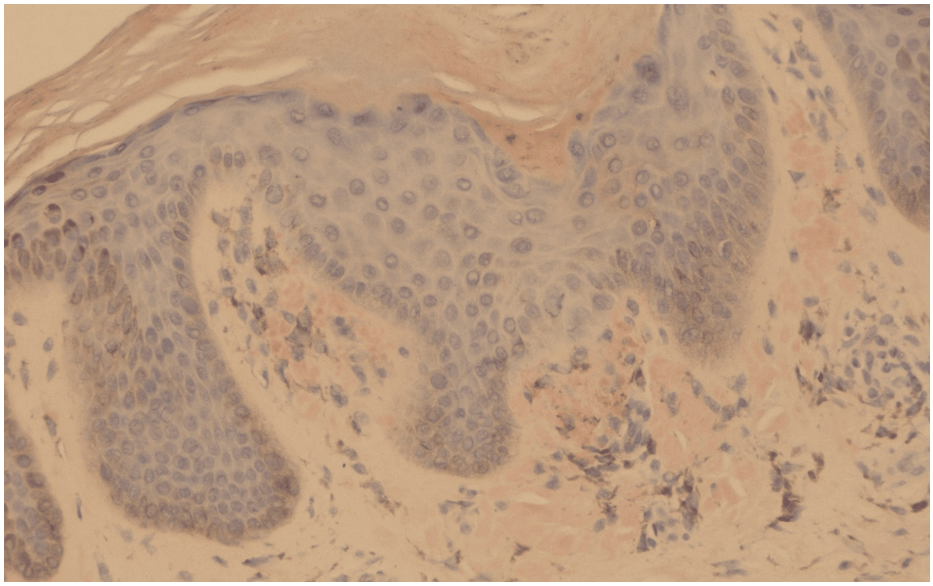


FIGURE 3: Congo red stain positive for amyloid globules in papillary dermis at 200x

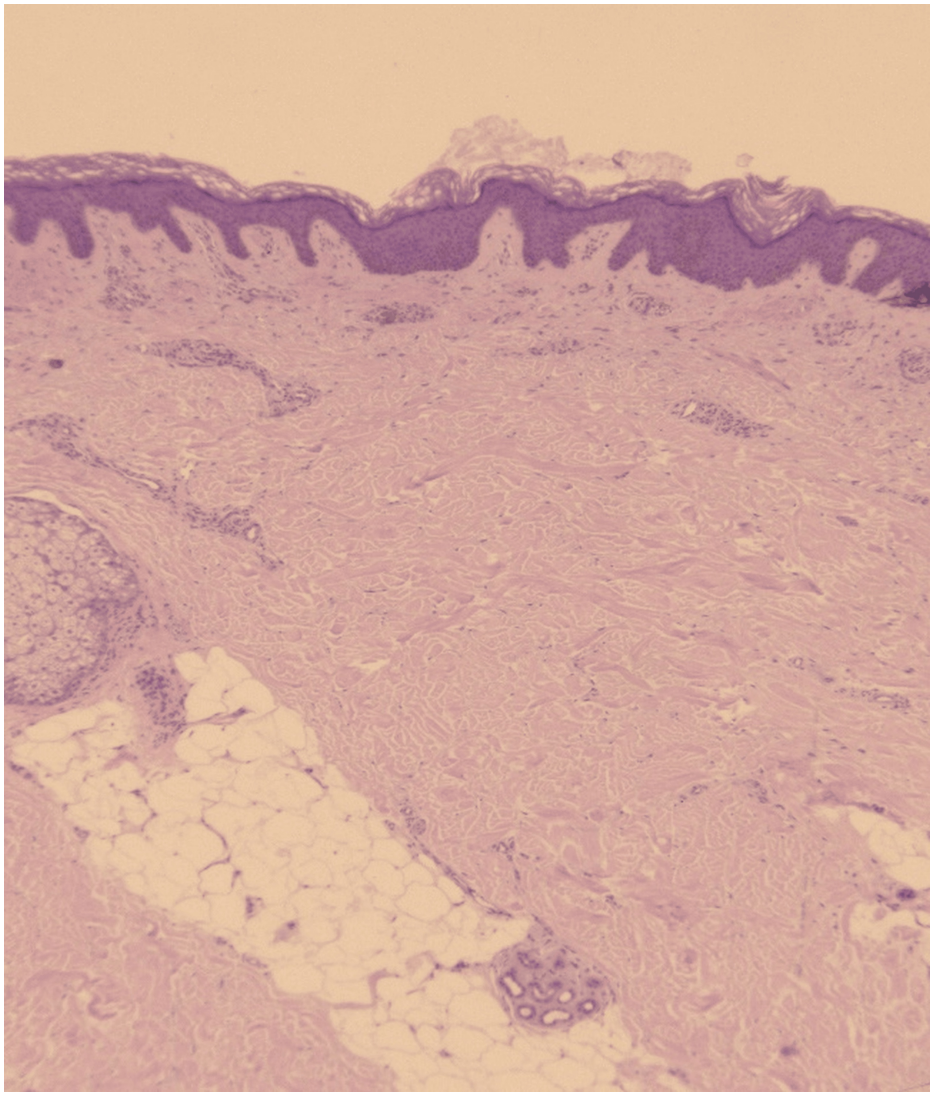


FIGURE 4: H&E stain of lesion at 10x

The image reveals hyperkeratosis and acanthosis with elongated ridges, widened dermal papillae, and superficial perivascular inflammatory infiltrates in the form of lymphohistiocytes mixed with melanophages

H&E: hematoxylin and eosin

Following histopathological confirmation of the diagnosis, treatment was initiated with clobetasol propionate 0.05% ointment applied twice daily under a hydrocolloid dressing overnight for four weeks, followed by gradual tapering. Cetirizine 10 mg daily was prescribed for symptomatic relief of pruritus. Follow-up was arranged for eight weeks later, at which time the patient demonstrated improvement in both pruritus and pigmentation.

Discussion

This case report highlights the diagnostic complexity and challenges involved in recognizing LA. The exact etiology of LA remains incompletely understood. It is considered a form of primary localized cutaneous amyloidosis in which amyloid is deposited within the skin without systemic involvement [5]. Chronic pruritus and scratching are proposed contributors to the development of LA, with histological features of lichen simplex chronicus frequently accompanying amyloid deposits [6]. However, this theory does not fully explain all cases, particularly the generalized or non-pruritic variants [3,4].

Additional proposed triggers include ultraviolet radiation, local trauma, and other environmental factors [7]. Genetic susceptibility may also play a role, particularly in cases with a positive family history, in which mutations involving genes in the interleukin-31 pathway, such as OSMR and IL32RA, may predispose individuals to chronic pruritus and subsequent keratinocyte damage [8,9]. Although most diagnosed cases are sporadic, up to 10% of cases are believed to be inherited in an autosomal dominant pattern with variable

penetrance [7].

The currently favored etiological hypothesis is the keratinocyte degeneration/apoptosis theory [5,10]. This theory proposes that repeated epidermal injury, classically attributed to chronic scratching and friction, induces keratinocyte damage and apoptosis, resulting in the accumulation of keratin filaments in the papillary dermis. These deposits subsequently degenerate to form amyloid fibrils, producing the characteristic amorphous, eosinophilic material in the papillary dermis [10]. The characteristic distribution of LA provides an important diagnostic clue. Lesions typically present as multiple, coalescing, hyperkeratotic, pruritic papules arranged in a bilateral, symmetrical distribution [1].

The most characteristic site of involvement is the anterior aspect of the legs, with possible extension to adjacent extensor areas. However, lesions may also involve the thighs, forearms, and upper back, particularly in more extensive disease [3,5]. LA has also been hypothesized to result from dysregulation of sweating, with decreased sweat production proposed as a potential trigger for amyloid deposition [11]. A published case report describes a "thermosensitive" distribution of LA in which areas with higher cutaneous temperatures are spared [12].

Despite its bothersome symptoms, LA remains a benign, skin-limited disorder without systemic involvement or a risk of internal organ disease [1,5]. There is no single definitive cure, and management typically requires a combination of therapies aimed at reducing pruritus and interrupting the itch-scratch cycle [2]. Treatment options include topical corticosteroids, keratolytic agents, and laser or light therapy [2,7].

Conclusions

LA is a rare and often underrecognized condition that may be mistaken for numerous other disorders. While clinical assessment and laboratory investigations provide valuable diagnostic information, histopathological examination remains essential for establishing a definitive diagnosis. Treatment of LA can be challenging due to the lack of a universally effective therapeutic modality and the high risk of recurrence. Management typically involves a multimodal approach aimed at alleviating pruritus, reducing scratching, and improving the overall cosmetic appearance of the lesions. Greater awareness of this condition is essential to facilitate timely and accurate diagnosis, minimize misdiagnosis, and ultimately improve patient outcomes and quality of life.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Bedour A. AlShammari, Abeer Albazali

Drafting of the manuscript: Bedour A. AlShammari

Critical review of the manuscript for important intellectual content: Bedour A. AlShammari, Alsadat Mosbeh, Abeer Albazali

Acquisition, analysis, or interpretation of data: Alsadat Mosbeh, Abeer Albazali

Supervision: Abeer Albazali

Disclosures

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