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Prurigo pigmentosa associated with diabetic ketoacidosis : A case report and a literature review

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ABSTRACT

Prurigo pigmentosa is a rare inflammatory dermatosis characterized by recurrent pruritic erythematous papules arranged in a reticulated pattern, which resolve leaving characteristic net-like hyperpigmentation. While classically described in association with ketogenic diets, prurigo pigmentosa has been increasingly recognized as a cutaneous marker of ketosis from various etiologies, including diabetic ketoacidosis (DKA).

We report the case of a 19-year-old male with no prior history of diabetes who presented to the emergency department with altered consciousness, tachycardia, and a 48-hour history of pruritic reticulated erythematous papules on the trunk. Laboratory investigations revealed severe hyperglycemia (408 mg/dL), metabolic acidosis (pH 7.18, bicarbonate 8 mEq/L), and ketonuria, confirming the diagnosis of DKA as the initial presentation of type 1 diabetes mellitus. Dermoscopy showed a reticular pigmentation pattern with a negative network, erythema, and telangiectatic vessels. Histopathological examination revealed neutrophilic perivascular infiltration and spongiosis, consistent with prurigo pigmentosa. Following treatment of DKA with intravenous fluids and insulin, both the metabolic derangements and skin lesions resolved within one week without specific dermatologic therapy.

This case highlights prurigo pigmentosa as an important dermatologic clue to underlying ketosis, which may precede or coincide with the diagnosis of DKA. Clinicians should be aware of this association, as recognition of this distinctive eruption may facilitate early diagnosis of potentially life-threatening metabolic emergencies, particularly in patients with previously undiagnosed diabetes.

KEYWORDS :

Heart failure with reduced ejection fraction, Sacubitril/valsartan, ARNI, Reverse remodeling, Echocardiography, Quality of life, Real-world, Functional mitral regurgitation

MAIN ARTICLE

INTRODUCTION

Prurigo pigmentosa (PP) is a rare inflammatory dermatosis first described by Nagashima in 1971 in Japan, where it remains most prevalent [1,2]. The condition is characterized by chronic, recurrent, pruritic skin lesions evolving through three stages: erythematous urticarial papules; crusted erythematous papules, papulovesicles, and vesicles; and hyperpigmented macules [1]. Predilection sites are the chest, back, and neck in symmetrical distribution [1].

While the exact pathogenesis of PP remains unknown, a strong association with ketosis has been established [1,3]. The condition has been reported in association with various ketotic states, including ketogenic and low-carbohydrate diets, anorexia nervosa, fasting, severe diarrhea, and diabetic ketoacidosis (DKA) [3-6]. This association has led some authors to propose the term "keto rash" to describe PP occurring in the context of dietary ketosis [3].

Until 2020, 76% of PP cases reported worldwide were in patients of Asian origin, leading to debates on a predisposition in Asian individuals [1]. However, increasing recognition of the condition outside of Asia suggests that it may be underdiagnosed in other regions [1,5]. The mean age of onset is typically in the second to third decade of life, with a female predominance (male-to-female ratio of approximately 1:2 to 1:4) [1,5].

Diabetic ketoacidosis is a life-threatening metabolic emergency characterized by hyperglycemia, metabolic acidosis, and ketosis, most commonly occurring in patients with type 1 diabetes mellitus. While DKA presents with well-recognized systemic manifestations, cutaneous manifestations are uncommon and often overlooked. Prurigo pigmentosa represents a rare but clinically significant dermatologic sign of ketosis that may serve as an early diagnostic clue, particularly in patients with previously undiagnosed diabetes [7].

We report a case of PP as the presenting manifestation of DKA in a young male with no prior history of diabetes, and provide a comprehensive review of the literature on this rare association.

CASE PRESENTATION

A 19-year-old male with no significant past medical history presented to the emergency department with a one-week history of progressively worsening fatigue, shortness of breath, intermittent abdominal pain, and recurrent vomiting. He reported no recent illnesses, infections, or dietary changes. The patient denied following any specific diet, including ketogenic or low-carbohydrate diets, and had no known history of diabetes mellitus or other metabolic disorders. Family history was notable for type 2 diabetes in his maternal grandmother.

On physical examination, the patient appeared ill and was noted to be confused, with a Glasgow Coma Scale (GCS) score of 13/15. Vital signs revealed tachycardia (heart rate 130 beats per minute), tachypnea (respiratory rate 24 breaths per minute), blood pressure of 110/70 mmHg, and oxygen saturation of 98% on room air. The patient was afebrile (temperature 37.1°C).

Dermatological examination revealed numerous pruritic, erythematous papules and macules arranged in a distinctive reticular pattern, primarily distributed over the anterior and posterior trunk, with extension to the upper back and chest (Figure *1*). The patient reported that these lesions had appeared suddenly 48 hours prior to presentation and were intensely pruritic. The lesions were non-follicular, and no vesicles, pustules, or bullae were observed. The face, extremities, and mucous membranes were spared.

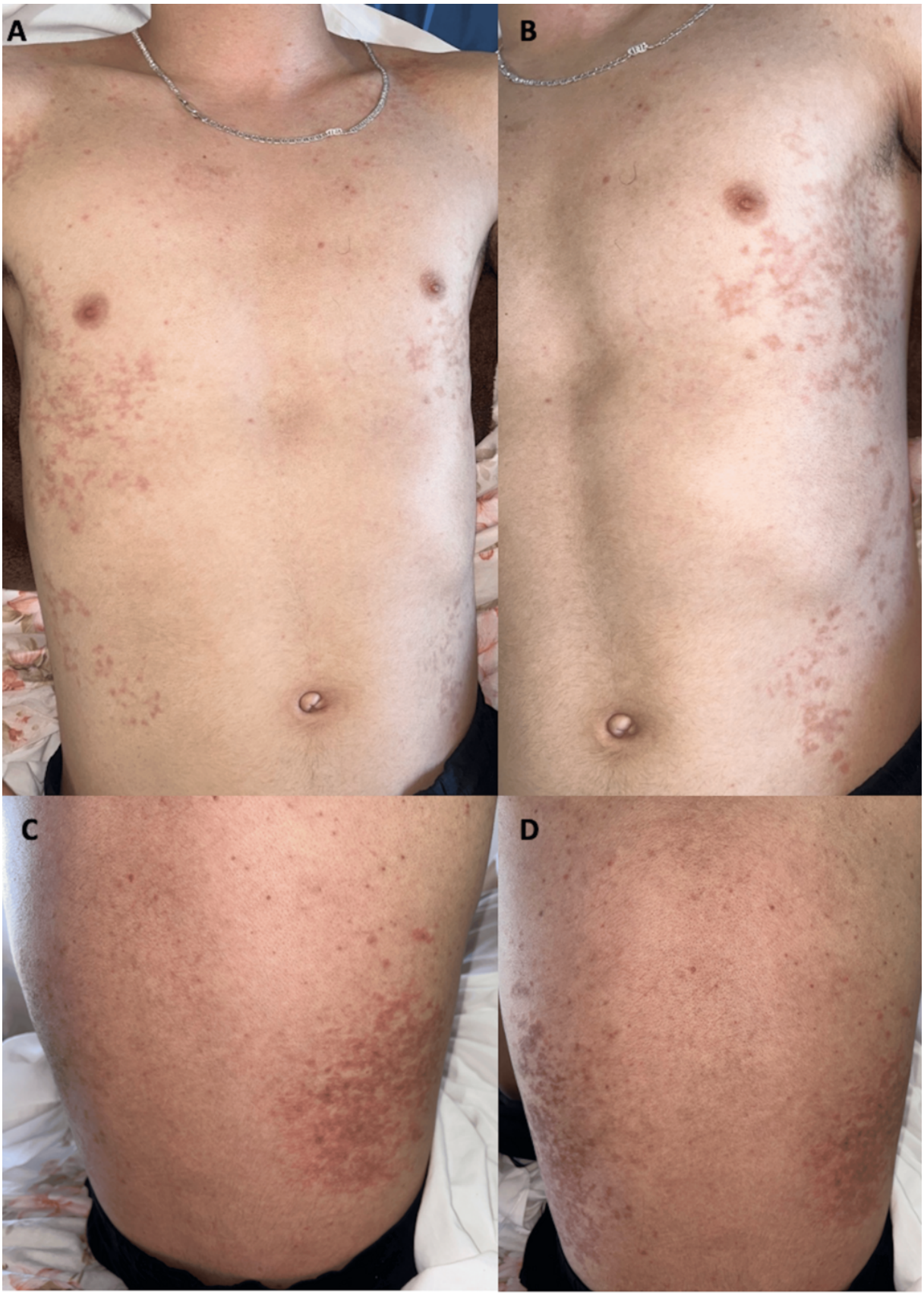

Figure 1: Early-stage clinical features of prurigo pigmentosa.

(A–D) Erythematous macules and papules arranged in a reticular pattern on the trunk.

At 72-hour follow-up, the skin lesions had evolved significantly, with the erythematous papules transitioning to hyperpigmented macules while maintaining the characteristic reticulated pattern (Figure *2*). This biphasic evolution, from inflammatory papules to reticular hyperpigmentation, is pathognomonic of prurigo pigmentosa.

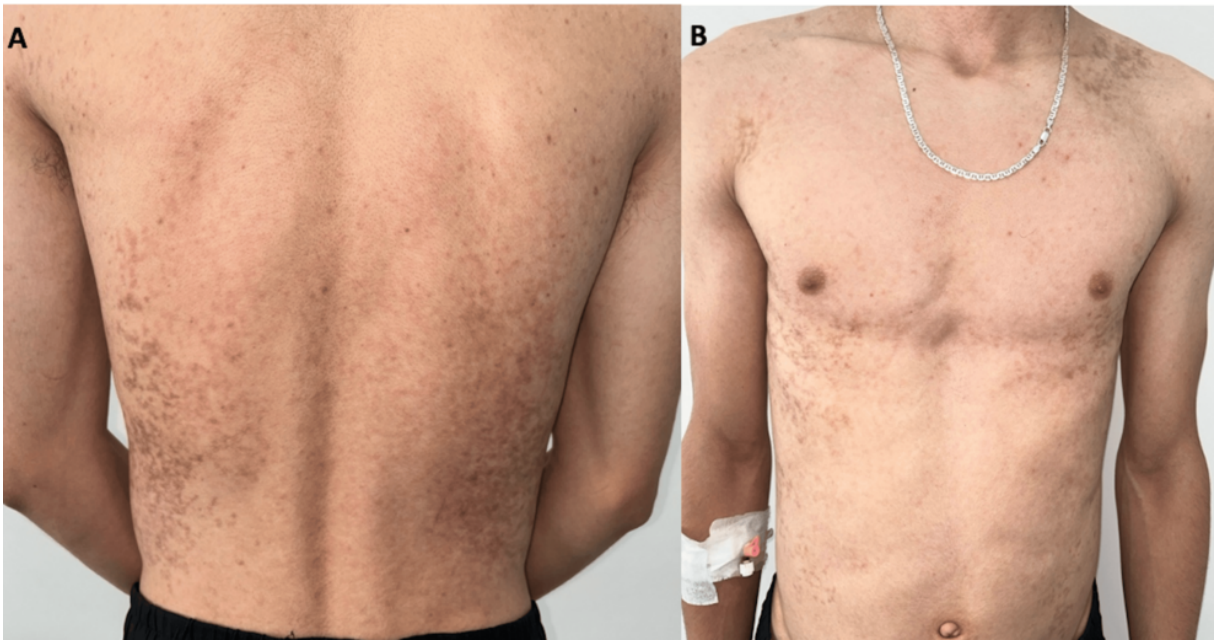

Figure 2: Late-stage clinical features of prurigo pigmentosa at 72 hours.
(A) Posterior trunk showing reticulated hyperpigmented macules.
(B) Anterior trunk showing similar reticulated hyperpigmented macules.

Dermoscopic examination revealed several characteristic features consistent with the transitional phase of PP, including reticular pattern of brown pigmentation, negative pigment network with accentuated skin markings, areas of background erythema, fine telangiectatic vessels at the periphery of lesions, and absence follicular plugging (Figure *3*).

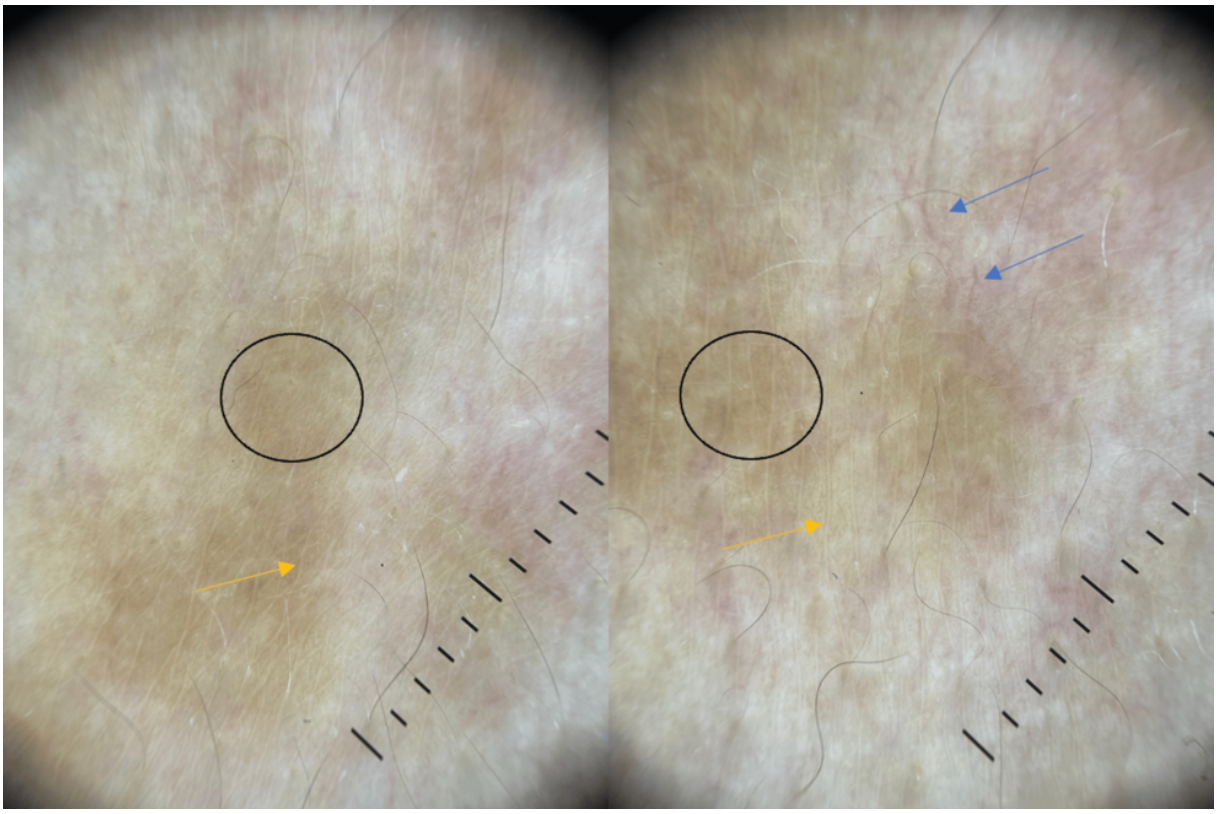

Figure 3: Dermoscopic features of prurigo pigmentosa.

Dermoscopy reveals areas of homogeneous brown pigmentation (circles), dilated vessels (blue arrows), and accentuated skin markings (yellow arrows).

Laboratory investigations revealed severe metabolic derangements consistent with diabetic ketoacidosis, as summarized in Table *1*.

Parameter	Result	Reference Range
Blood glucose	408 mg/dL (22.6 mmol/L)	70-100 mg/dL (3.9-5.6 mmol/L)
Arterial pH	7.18	7.35-7.45
Bicarbonate (HCO_3^-)	8 mEq/L	22-28 mEq/L
Serum ketones (beta-hydroxybutyrate)	5.2 mmol/L	<0.6 mmol/L
Anion gap	24 mEq/L	8-12 mEq/L
Urine glucose	3+	Negative
Urine ketones	3+	Negative

Glycated hemoglobin (HbA1c)	12.8%	<5.7%
Serum sodium	128 mEq/L (corrected: 134 mEq/L)	136-145 mEq/L
Serum potassium	5.1 mEq/L	3.5-5.0 mEq/L
Serum creatinine	1.4 mg/dL	0.7-1.3 mg/dL
White blood cell count	14,200/μL	4,500-11,000/μL

Table 1: Laboratory findings at initial presentation.

A 4-mm punch biopsy was obtained from an erythematous papule on the upper back, one week after the onset of the eruption. Histopathologic findings demonstrated slight to moderate acanthotic changes in the epidermis accompanied by orthokeratotic hyperkeratosis. Scattered dyskeratotic or apoptotic keratinocytes were identified, with no significant spongiotic changes. The dermoepidermal interface exhibited mild basal vacuolar degeneration and minor irregularity. A sparse perivascular lymphocytic inflammatory infiltrate was present in the superficial dermis, with no apparent neutrophilic component (Figure *4*). These features were consistent with the intermediate stage of prurigo pigmentosa.

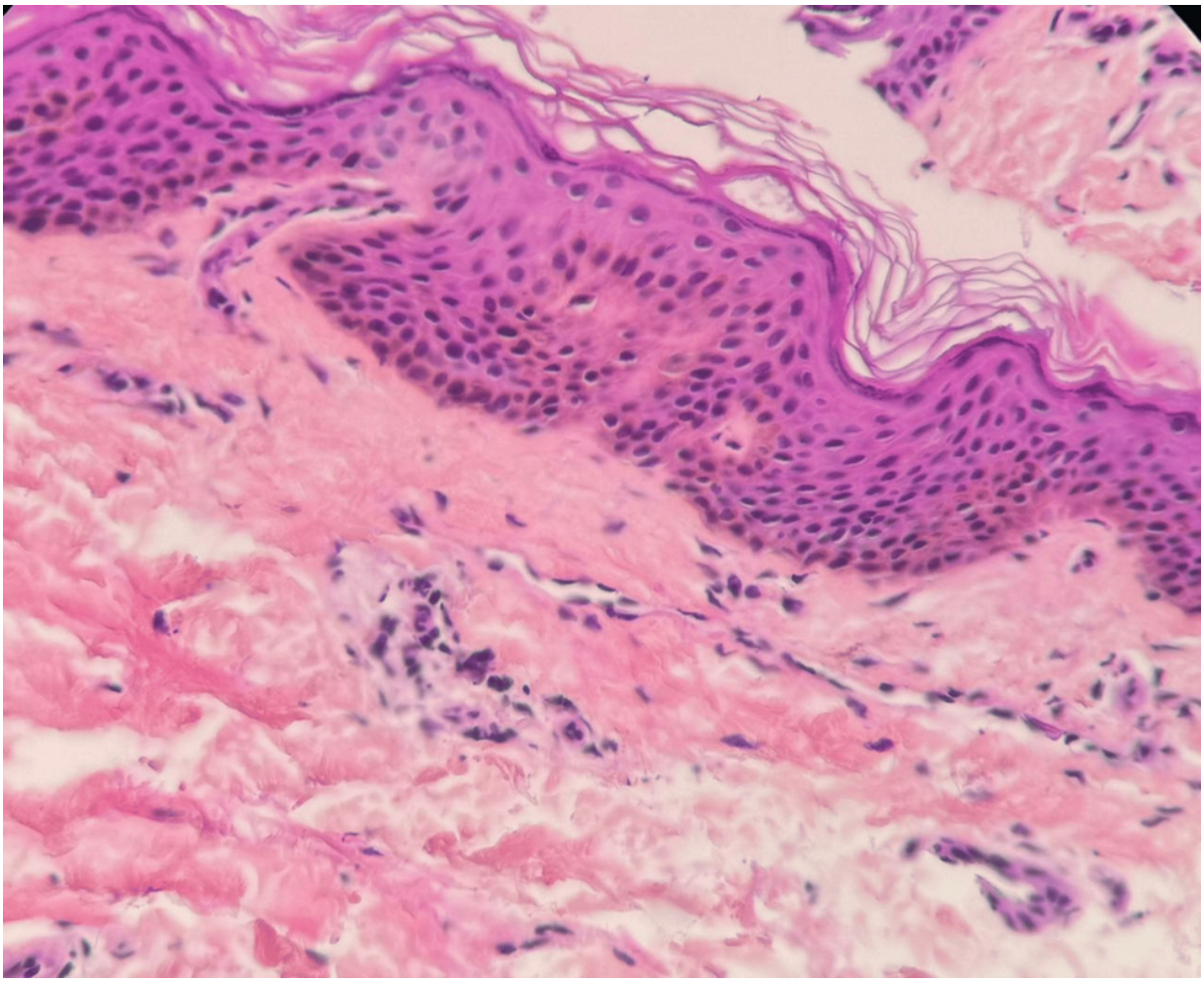

Figure 4: Histological section of an inflammatory papule.

Low-power view (hematoxylin and eosin stain, ×100 magnification) showing mild spongiosis of the epidermis with a superficial perivascular inflammatory infiltrate in the dermis.

The patient was admitted to the intensive care unit and treated according to standard DKA protocols, including aggressive intravenous fluid resuscitation with 0.9% normal saline, continuous intravenous insulin infusion (0.1 units/kg/hour), potassium replacement, and close monitoring of blood glucose, electrolytes, and acid-base status.

No specific dermatologic treatment was initiated for the skin lesions. Within 24 hours of treatment initiation, the metabolic acidosis began to resolve, and the patient's mental status improved. The pruritus associated with the skin lesions decreased significantly by day 3 of hospitalization. By day 7, the erythematous component of the lesions had completely resolved, leaving only residual reticular hyperpigmentation.

The patient was discharged on a basal-bolus insulin regimen with diabetes education and close endocrinology follow-up. At one-month follow-up, the hyperpigmentation had faded significantly, and no new lesions had developed. The patient's HbA1c at three-month follow-up was 7.2%, indicating improved glycemic control.

DISCUSSION

This case illustrates prurigo pigmentosa as a rare but clinically significant cutaneous manifestation of diabetic ketoacidosis, serving as an important diagnostic clue in a patient with previously undiagnosed type I diabetes mellitus. The chronological correlation between cutaneous manifestations and metabolic derangement, coupled with complete lesion resolution following normalization of ketone levels, provides compelling evidence for a causal relationship between ketosis and PP in this patient.

Epidemiology and Clinical Features

Prurigo pigmentosa was first described by Nagashima et al. in 1971 and has been reported predominantly in Japanese and Korean populations [1,2]. In a study of 20 central European patients, Michor-Tscharre et al. found a mean age at diagnosis of 24.05 years (range 15-51 years) with a female predominance (15 female, 5 male) [1]. The most common sites were the breast (80%), back (65%), and neck (55%), with the abdomen involved in 30% of patients [1]. Lesions appeared in a reticular arrangement in all patients, and pruritus was a consistent symptom [1]. The clinical presentation of PP is characterized by three distinct phases: an early/inflammatory phase with intensely pruritic erythematous urticarial papules arranged in a reticulated pattern predominantly on the trunk; a transitional phase with crusted erythematous papules, papulovesicles, and vesicles developing central hyperpigmentation while peripheral erythema persists; and a late/resolved phase where inflammation resolves completely, leaving characteristic net-like hyperpigmented macules that may persist for weeks to months [1,2].

Importantly, early, fully developed, and late-stage lesions can coexist, a finding observed in 75% of patients in the Michor-Tscharre et al. study [1]. Our patient demonstrated this classic biphasic evolution, with erythematous papules at presentation transitioning to hyperpigmented macules within 72 hours. The prolonged persistence of hyperpigmentation

(at least three months in our case) is consistent with the natural history of post-inflammatory pigmentary changes in PP and does not indicate ongoing disease activity [1,2].

Association with Ketosis

The association between PP and ketosis is well-established, with multiple case reports and series documenting the condition in various ketotic states [1-8] In a study by Oh et al., ketosis was confirmed in 6 of 16 patients with PP, and 8 patients showed a chronological relationship between the appearance of skin lesions and dieting or fasting [5]. Hijazi et al. reported that PP is now established to be linked to ketoacidotic states [6]. Table *2* summarizes the published cases of PP associated with diabetic ketoacidosis and diabetes mellitus.

Author (Year)	Age/Sex	Diabetes Type	Prior DM Diagnosis	Lesion Distribution	Treatment	Time to Resolution
Kobayashi et al. (1996)	16/F	Type 1	No	Trunk	DKA treatment (insulin)	Resolution with ketosis correction
Oh et al. (2012)	Multiple	Variable	Variable	Trunk, neck	Minocycline ± ketosis correction	Variable
Hijazi et al. (2014)	Mean 23.5/F	N/A	N/A	Trunk, upper extremities	Variable	Variable
Page et al. (2021)	Median 29.5	N/A	N/A	Variable	Doxycycline	Complete regression
Michor-Tscharre et al. (2023)	Mean 24.05	1 patient with diabetes	Variable	Breast, back, neck	Variable	Variable
Present case	19/M	Type 1	No	Trunk	DKA treatment alone	Inflammatory lesions: 1 week; Hyperpigmentation: >3 months

Table 2: Published Cases of Prurigo Pigmentosa Associated with Diabetes Mellitus and Ketosis

This table summarizes published cases of prurigo pigmentosa associated with ketosis and diabetes mellitus [1,5,6,7,8]

Review of the literature reveals that PP associated with ketosis predominantly affects young females, often in the context of dieting or fasting [4]. Our case is notable for occurring in a male patient with DKA as the initial presentation of type 1 diabetes mellitus, which is less commonly reported. The immediate arrest of new lesion development upon restoration of metabolic homeostasis, without specific dermatologic therapy, strongly implicates ketosis as the underlying trigger for PP, whereas the prolonged persistence of hyperpigmentation reflects the natural evolution of post-inflammatory pigmentary changes rather than ongoing disease activity, particularly in individuals with darker phototypes [1,9].

Pathophysiology

The exact pathophysiological mechanisms underlying prurigo pigmentosa remain largely unknown [1,2]. While a strong association with ketosis has been established through clinical observations, the precise mechanisms by which ketone bodies trigger the characteristic inflammatory response are not fully elucidated.

Established associations:

The cause of PP is still unknown, but ketosis, friction, and sweating are associated risk factors. Eruption of lesions while dieting occurred in 20% of patients in the Michor-Tscharre et al. study, and malnutrition, particularly when being on a ketogenic diet, as well as severe diarrhea contributing to nutrition deficiency, might trigger the onset of PP. In 20% of patients, affected skin was associated with external stimuli, which might be explained by Koebnerization [1].

Several mechanisms have been proposed to explain the pathogenesis of PP, though none have been definitively proven [1,2]. It has been hypothesized that elevated levels of ketone bodies (β-hydroxybutyrate, acetoacetate, acetone) may directly trigger inflammatory cascades in the skin, potentially through activation of innate immune pathways or promotion of neutrophil

chemotaxis [2]. Acetone, a volatile ketone body excreted through the skin, has been proposed as a potential contact irritant, particularly in areas of friction and sweating such as the trunk, which may explain the characteristic distribution of lesions [1,2]. In the context of restrictive dieting, deficiencies in certain micronutrients have been implicated, although this mechanism appears less relevant in acute DKA [2].

Recent immunohistochemical studies have provided new insights into the cellular composition of PP infiltrates. Michor-Tscharre et al. demonstrated the presence of myeloid cell nuclear differentiation antigen (MNDA)-positive cells with characteristic elongated "band" nuclei in all examined cases, indicating the involvement of neutrophils and their precursors (myelocytes and promyelocytes) in the pathogenesis [1]. Pirrone and Böer-Auer further characterized this finding, demonstrating promyelocytic differentiation in PP infiltrates and drawing an analogy to histiocytoid Sweet syndrome, suggesting that PP may share pathogenic mechanisms with other neutrophilic dermatoses [10]. This finding supports the classification of PP as a neutrophilic dermatosis and suggests an acute, eruptive inflammatory process. Additionally, a predominance of CD8+ T-lymphocytes in the epidermis has been observed in 86% of cases, suggesting a possible role for cytotoxic T cells in the pathogenesis, although the exact mechanism remains unclear [1].

The most compelling evidence for the central role of ketosis in PP pathogenesis comes from the consistent observation that correction of the underlying ketotic state typically leads to cessation of new inflammatory lesions within days, often without specific dermatologic therapy [1,9]. In patients with ketosis-induced PP, administration of insulin or an increase in carbohydrates can halt the development of new lesions [1]. This was clearly observed in our patient, in whom no new inflammatory lesions appeared after initiation of DKA treatment, despite the persistence of post-inflammatory hyperpigmentation for at least three months.

Dermoscopic Features

Dermoscopy is a valuable non-invasive tool for the diagnosis and monitoring of PP. Wang et al. conducted a retrospective study of 20 patients with PP and identified stage-specific dermoscopic features [11]. Early lesions show pink oval lesions with punctate or linear vessels, surrounded by pale yellow rings around the skin lesions. Fully developed lesions

display pink lesions with brown pigment granules in the center and linear vessels at the edge. Late lesions present with grainy grayish-brown or yellowish-brown pigmentation surrounding the hair follicle, merging with each other [11]. In our patient, dermoscopy revealed features consistent with the transitional phase, including reticular pigmentation, a negative network, background erythema, and telangiectatic vessels. These findings, combined with the clinical presentation and histopathology, supported the diagnosis of PP.

Differential Diagnosis

The differential diagnosis of PP includes several conditions that may present with reticulated erythema or pigmentation [2,5] :

Condition	Key Distinguishing Features
Confluent and reticulated papillomatosis (Gougerot-Carteaud)	Papillomatous surface, no pruritus, responds to minocycline
Erythema ab igne	History of heat exposure, fixed location, telangiectasias
Livedo reticularis	Vascular pattern, no papules, associated with systemic disease
Urticaria pigmentosa	Darier sign positive, mast cell infiltrate on biopsy
Allergic contact dermatitis	Exposure history, vesicles, eczematous changes
Dermatitis herpetiformis	Associated with celiac disease, IgA deposits on DIF
Pigmented purpuric dermatosis	Petechiae, hemosiderin deposits, no neutrophilic infiltrate

Table 3: Differential Diagnosis of Prurigo Pigmentosa

This table summarizes the key differential diagnoses for prurigo pigmentosa and their distinguishing features [2,5].

The characteristic clinical evolution (inflammatory papules to reticular hyperpigmentation), association with ketosis, and histopathological findings (neutrophilic infiltrate, spongiosis) help distinguish PP from these mimickers [2,5].

Treatment

The management of PP depends on the underlying etiology and the severity of symptoms [2,5]. In cases associated with DKA or dietary ketosis, correction of the underlying metabolic abnormality is the primary treatment, and resolution of ketosis typically leads to cessation of new inflammatory lesions and resolution of pruritus within days, as observed in our patient, although post-inflammatory hyperpigmentation may persist for months [1,9]. Tetracycline antibiotics, including doxycycline (100 mg twice daily) or minocycline (100 mg twice daily), are considered first-line pharmacologic treatments for PP, likely due to their anti-inflammatory rather than antimicrobial properties [1,2,5]. Treatment with doxycycline in 9 patients in the Page et al. study resulted in complete regression of the lesions, and patients respond well to minocycline treatment, with a mean duration of use of 2.4 weeks [1,8]. Kim et al. reported similar efficacy with minocycline in their Korean cohort of 50 patients [12]. Dapsone has been reported effective in refractory cases, likely through inhibition of neutrophil chemotaxis, and macrolide antibiotics such as erythromycin and azithromycin have been used with variable success [2].

In our patient, correction of DKA alone led to immediate cessation of new inflammatory lesions and complete resolution of pruritus within one week. Although oral doxycycline was offered to potentially accelerate fading of the residual hyperpigmentation, the patient declined this treatment as the pigmentary changes were not cosmetically concerning to him and his symptoms had resolved. This case demonstrates that in ketosis-associated PP, correction of the underlying metabolic abnormality may be sufficient to halt disease activity, and specific dermatologic therapy may not be necessary when the patient is asymptomatic. The residual reticular hyperpigmentation, which persisted for at least three months, represents post-inflammatory pigmentary changes that fade gradually over time without intervention.

Clinical Implications

This case has several important implications for clinical practice. First, PP may serve as an early cutaneous marker of ketosis, potentially preceding the diagnosis of DKA or diabetes mellitus, and clinicians should maintain a high index of suspicion for underlying metabolic disorders in patients presenting with reticulated erythematous eruptions. Second, patients

presenting with PP, particularly those without a history of ketogenic dieting, should undergo screening for diabetes mellitus, including fasting glucose, HbA1c, and urinalysis for ketones. Third, collaboration between dermatologists and endocrinologists is essential for optimal management of PP associated with DKA. Fourth, as demonstrated in our case, specific dermatologic therapy may not be necessary when pruritus resolves with ketosis correction alone, and the decision to initiate tetracycline therapy should be individualized based on symptom severity and patient preference regarding residual hyperpigmentation. Fifth, patients should be informed that while the active inflammatory phase and pruritus resolve rapidly with ketosis correction, residual hyperpigmentation may persist for several months and does not indicate treatment failure or ongoing disease activity. Finally, PP may remain underdiagnosed outside of Asia, and increased awareness is necessary, especially because fasting is a common religious practice in many regions [1].

CONCLUSIONS

This case highlights prurigo pigmentosa as an uncommon yet clinically important dermatologic manifestation of diabetic ketoacidosis, particularly in individuals with no prior history of diabetes. The distinctive clinical presentation, pruritic reticulated erythematous papules evolving to net-like hyperpigmentation, combined with characteristic dermoscopic and histopathological features, should prompt clinicians to investigate for underlying ketosis. The rapid identification and treatment of DKA led to immediate cessation of new inflammatory lesions and complete resolution of pruritus, confirming resolution of active disease, although residual reticular hyperpigmentation persisted for at least three months as expected with post-inflammatory pigmentary changes. Although tetracycline therapy was offered, the patient declined as the residual hyperpigmentation was not bothersome, demonstrating that correction of the underlying ketotic state may be sufficient treatment in selected patients. Increased awareness of the association between PP and ketosis among dermatologists, emergency physicians, and endocrinologists may improve outcomes by enabling early recognition and treatment of potentially life-threatening metabolic emergencies.

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