Prurigo Pigmentosa: Review of the Literature, Impact of Ketogenic Diets and Proposal of a Hypothesis

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Abstract

Prurigo pigmentosa (PP) is characterized by an initially pruritic inflammatory rash on the torso and nape of the neck, which fades subsequently to leave a reticular macular hyperpigmentation. Among the list of suggested potential aetiological factors, ketosis remains an indisputable cause. However, the mechanism(s) linking ketosis and PP remain largely unknown. Recently, dermatologists have been confronted with an outbreak of PP induced by ketogenic diets (KD) and bariatric surgeries. Atypical forms (bullous variant) and localizations have been encountered. Herein, we review the relevant literature on PP, discuss its link to ketosis with current and future perspectives and propose a hypothesis for its aetiopathogenesis.

Key Words: *Bullous, confluent and reticulated papillomatosis, ketogenic diet, prurigo pigmentosa*

Introduction, Definition and History

Initially described by Nagashima *et al.* in 1971 and dubbed as 'prurigo pigmentosa' (PP) in 1978, PP is a rare, sporadic, pruritic recurrent inflammatory cutaneous disorder of unknown aetiopathogenesis.[1-8]

Recently, there has been a remarkable surge in PP prevalence in accordance with the popular ketogenic diets (KD) and bariatric surgeries.[9-12]

Methods

The aim of this article was to provide an updated concise review on PP and to discuss its metabolic link to ketosis.[5,11,13-15] In addition, we sought and proposed a hypothesis that could potentially explain this link, commented on current and future perspectives and highlighted the need for targeted research on ketosis and PP.

We conducted a PubMed search of English-language articles indexed for MEDLINE using the following key terms: Prurigo pigmentosa, ketoacidosis, diet, ketones, ketogenic, fasting, diabetes and bariatric surgery. We reviewed the abstracts and retrieved and summarized those articles most relevant for the scope of this review.

Epidemiology

The exact prevalence of PP is unknown.[4] Worldwide, about 400 cases have been reported hitherto.[7,13,16,17] It was once considered a rare disease; however, there is an upward escalation in its prevalence.[4,9-12] The disease is comparatively rare in the Western world; many cases have been described in Japan and other Eastern Asian countries.[4,5,9,13,18-21] This may be ascribed to the disorder's initial reporting from Japan, and unfamiliarity, misdiagnosis or underdiagnosis in the Western world, rather than implying a genetic predisposition of the Japanese population per se.[5,13,19,22] As the recognition of the disorder has amplified worldwide, cases of PP have been published from white, Hispanic and black populations, refuting the role of race or ethnicity in its development.[9-11,13,16-19,22-28] The disease has a predilection for young adult females (female/male: 2–6) and typically presents in spring or summer.[2,4,5,21,22,29-33] In approximately 85% of cases, the onset is between 11 and 30 years of age.[2,31,34] PP has not been reported in prepubertal children or the elderly.[19,22,35]

Clinical findings

PP is characterized by a distinctive staged clinical presentation, that is an acute early inflammatory phase, a mature phase and a chronic late resolution phase.[2-4,12,18,36]

The early inflammatory phase has an abrupt onset and a dynamic course, and usually lasts less than a week.[16,20,22,32,37] The characteristic lesions are

Access this article online

Quick Response Code:

Website: https://journals.lww.com/ijd

DOI: 10.4103/ijd.ijd_464_22

How to cite this article: Demircioğlu D, Durmaz EÖ, Demirkesen C, Şahin S. Prurigo pigmentosa: Review of the literature, impact of ketogenic diets and proposal of a hypothesis. Indian J Dermatol 2025;70:146-51.

Received: June, 2022. **Accepted:** October, 2024.

itchy erythematous macules, urticarial papules and plaques.[2,5,20,30,33,38,39] In the mature phase, blisters, papulovesicles, crust formation, scaling and excoriations may be encountered [Figures 1 and 2].[2,39] Bullous, pustular and urticarial variants have been reported.[5,11,34,35,40,41] New lesions are continuously formed, while the older ones coalesce and subside, leaving behind a mottled reticular pigmented macular network at the late phase [Figure 3].[2,26,29,30,33,38,39,42] The sites of predilection include the upper back and scapular regions, nape of the neck, clavicular regions and inter- and sub-mammary areas of the chest.[5,16,20,29,30] Rarely, the lesions may occur at other sites, such as the face, forehead, abdomen, lumbosacral area, pubis, antecubital fossae, shoulders and limbs.[4,7,20,29,30,39] Although the lesions are almost always symmetrically arranged, unilateral and segmental variants have been published.[5,15,19] Pruritus is the most bothersome symptom, elicited in 80% of patients.[13]

Dermatoscopy

In the early stage, dermatoscopy displays erythematous papules with overlying whitish scales (representing dyskeratosis), brownish-black to blue-grey dots and globules, punctate or linear vessels, and blue-white veil-like structures. Late resolving lesions demonstrate brownish reticular pigmentation with islands of brown-grey dots (representing dermal pigment incontinence), and prominent peripheral linear blood vessels.[15,43]

Histopathology and immunohistochemical findings

The histopathological scene is also staged and variable depending on the time of biopsy.[2,17,19,22,44] The portrait is dominated by neutrophils in the early inflammatory phase, whereas lymphocytes and eosinophils play the leading roles in the mature phase. Epidermal reaction to injury and dermal melanophages become more pronounced as the disorder progresses to the late phase.[13,27,37]

The histopathological landscape in the early phase consists of epidermal thinning, inter- and intracellular oedema, focal spongiosis, scanty necrotic basal keratinocytes, neutrophil exocytosis, rare neutrophil microabscesses, dermal papillary oedema, dilatation of superficial blood vessels, and superficial perivascular and interstitial infiltrate mainly composed of neutrophils.[2,4,17,19,22,27,29,30,33,36-40] Neutrophil microabscess formation in the epidermis is archetypal, and neutrophils adhere to oedematous or necrotic keratinocytes in a clumped or single-cell manner.[2] In more mature and bullous PP, the panorama is dominated by numerous necrotic keratinocytes (focal; and rarely overall epidermal necrosis), marked spongiosis, ballooning, profound vacuolar alteration and reticular degeneration of the basal layer, intraepidermal or subepidermal vesiculation, lymphocyte exocytosis, and a patchy lichenoid lymphocytic and eosinophilic infiltrate [Figure 4].[2,4-6,16,17,19,27,33,37,39] The portrayal in the late phase comprises acanthosis, marked parakeratosis, hyperkeratosis, perivascular infiltrate of histiocytes and lymphocytes, pigmentary incontinence and melanophages in the papillary and reticular dermis.[2,9,16,19,23,27,29,37,40,44]

Immunohistochemical studies disclose a predominance of CD4+ T cells in the dermal infiltrate and CD8+ T cells in the epidermis [Figure 5], intense lymphocyte function-associated antigen 1 (LFA-1) immunostaining of dermal and epidermal lymphocytes, increased CD1+ cell population in the epidermis and marked expression of intercellular adhesion molecule 1 (ICAM-1) (the ligand for LFA-1) and human leukocyte antigen - DR isotype by lymphocytes in the lower epidermis.[45] Interleukin-6 (IL-6) is significantly increased in the lesional skin.[13]

Direct immunofluorescence (DIF) and indirect immunofluorescence (IIF) studies consistently yield negative or nonspecific findings.[5,6,23,34,35]

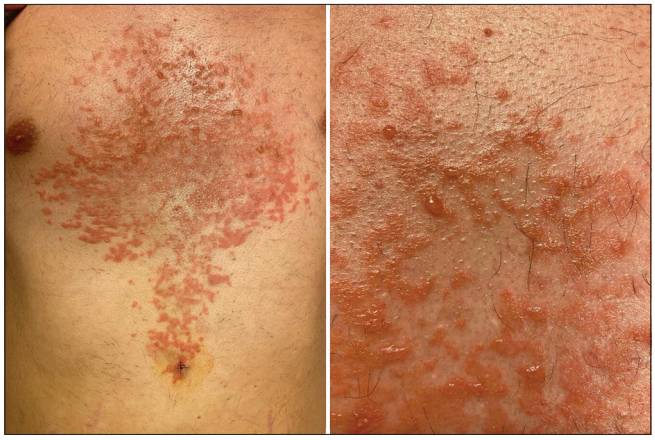

Figure 1: Bullous prurigo pigmentosa. Reticular erythematous papules and bullae on the chest and abdomen (left). Closer view of bullous lesions (right)

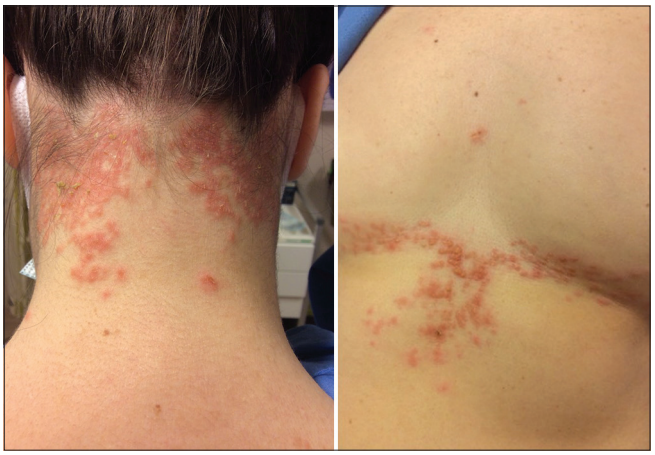

Figure 2: Vesiculobullous prurigo pigmentosa. Reticulated erythematous papules with crusting on the nape of the neck (left). Blistering erythematous papules on the inframammary areas (right)

Differential diagnosis

The list of differential diagnostic considerations embraces pigmented contact dermatitis, lichen planus pigmentosus, prurigo melanotica, reticular erythematous mucinosis, erythema ab igne, macular amyloidosis, Riehl's melanosis, acute systematic lupus erythematosus (SLE), Darier's disease, urticaria, drug eruption, erythema multiforme, pityriasis lichenoides et varioliformis acuta, atopic dermatitis, Dowling-Degos disease, Galli-Galli disease, urticaria pigmentosa, erythema dyschromicum perstans, poikiloderma vasculare atrophicans and confluent and reticulated papillomatosis of Gougerot and Carteaud.[2,4,6,12,25,19,30,31,39] Bullous PP should be distinguished from bullous SLE, linear IgA bullous dermatosis, pemphigus, bullous lichen planus and dermatitis herpetiformis.[2,6]

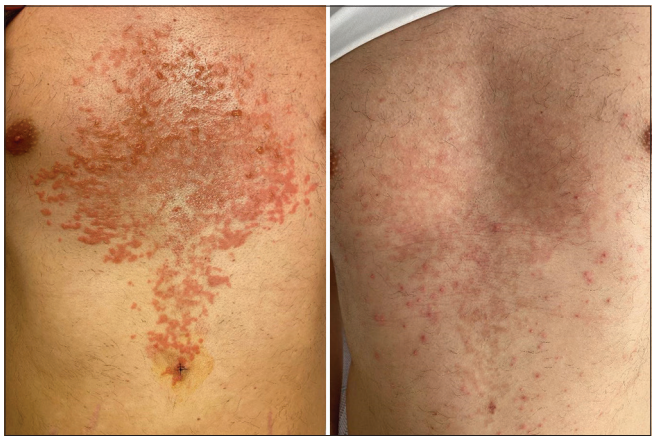

Figure 3: Bullous prurigo pigmentosa (left). Resolution with pigmentation after 2 weeks of doxycycline treatment at a dose of 100 mg/day (right)

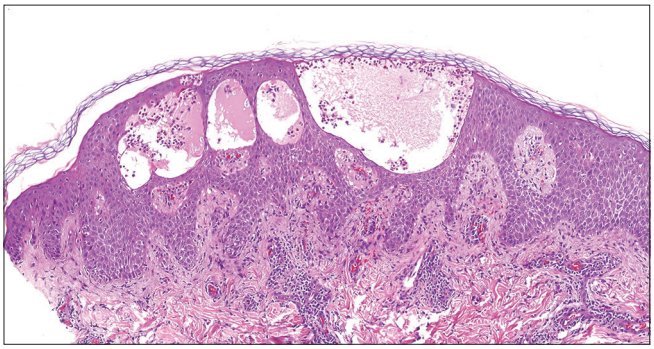

Figure 4: Bullous prurigo pigmentosa. Intraepidermal vesicles, containing both eosinophils and neutrophils, prominent eosinophilic and neutrophilic spongiosis, perivascular and interstitial dermal infiltration, composed of eosinophils, lymphocytes and histiocytes (H and E ×20)

Course and prognosis

PP has a chronic, waxing and waning course with frequent recurrences and spontaneous remissions.[2,5,6,13,22,23,39] Recurrent PP occurs in almost half of patients usually in spring or summer time, and remains strictly confined to pigmented areas.[2,4,30,32,36,39,45] In all patients, the net-like hyperpigmentation persists for months to years, even after successful therapeutic alleviation of the inflammation and pruritus.[2,5,11,19,22,44]

Treatment

All therapeutic regimens effective in PP are thought to act through anti-inflammatory, anti-neutrophilic and immunomodulatory properties.[2,4,5,9,27,28,30,33,34,36-38,44] Minocycline 100-200 mg per day for 3-5 weeks is the treatment of choice.[2,6,19,21,22,24,26,30-32,40] Tetracycline 2 × 500 mg/day or doxycycline 100-200 mg/day for 2–8 weeks are alternatives.[9,17,22,26,27,33,36] Dapsone 25-100 mg/day is dramatically effective, although adverse effects may preclude its use[2,22,30,31] Potassium iodide, trimethoprim-sulfamethoxazole and macrolide antibiotics (clarithromycin, azithromycin and roxithromycin) are useful in some cases[4,18,32,34,38,46] Oral retinoids are reserved for refractory cases[28,34] As expected from a predominantly neutrophilic disorder, colchicine has been reported as a beneficial therapeutic regimen[46] Phototherapy (NB-UVB) is another therapeutic option in patients with allergies or contraindications to systemic medications[15]

Aetiopathogenesis

The aetiopathogenesis of PP is an enigma. Both endogenous and exogenous factors have been implicated in its incitement or exacerbation.[5] Suggested potential causes are presented in Table 1. Neutrophil-mediated inflammation with the production of oxygen intermediates is suspected to be the main pathogenetic event[2,30,38]

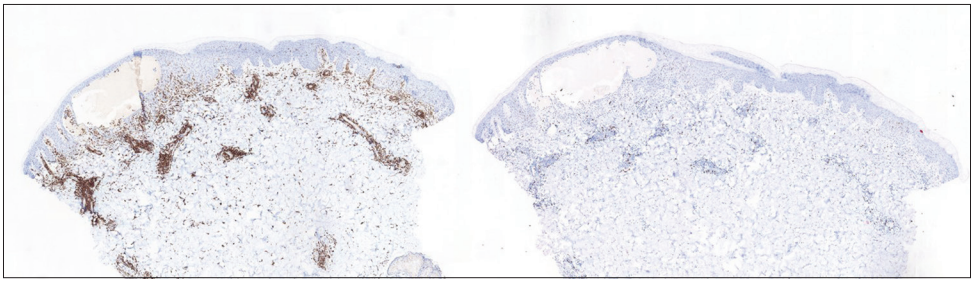

Figure 5: Prurigo pigmentosa. Prominence of CD4+ T lymphocytes within the dermal infiltration. CD4+ (left) and CD8+ (right) T-cell immunostaining

Table 1: Suggested causes of prurigo pigmentosa.

ENDOGENEOUS	Genetic factors	* Sporadic; familial predisposition unproven.
		* Only one report of PP occurrence in monozygotic twins.
	Hormonal factors	* Based on female predilection, worsening during pregnancy and menstruation, association with polycystic ovary syndrome.
	Allergic contact dermatitis	* Speculations involve paraamino compounds (e.g.: TCP) used in cloth manufacturing, nickel, chrome in detergents, chromium in acupuncture needles and trichlorophenol. Patch tests positive for chrome in two cases.
		*The absence of positive history and patch test findings in most cases disputes contact allergy proposal.
	Atopic tendency	* Atopy may be present.
	Rheumatologic disorders	* A PP-like persistent rash observed in patients with Sjogren syndrome and adult-onset Still's disease.
EXOGENEOUS	Physical factors	* Physical trauma, mechanical irritation, rubbing, friction from clothes, sweating, summer heat, sunlight exposure, exercise, wearing wet clothing.
	Drugs	* Bismuth, carbamazepine, clomipramine HCl and bromazepam implicated, but not proven.
	Environmental contaminants	* Speculation relies on the frequency of Japanese cases.
	Infections	* H. pylori and Borrelia species (*Borrelia garinii* and *Borrelia afzelii*) implicated in PP occurrence.
		* PP is speculated to represent a manifestation of Lyme disease.
	Dietary changes and ketoacidosis	* Uncontrolled DM with ketoacidosis, insulin-dependent DM, pregnancy with hyperemesis gravidarum, fasting, starvation, carbohydrate or calorie-restricted strict dieting, ketogenic dieting, Paleolithic (paleo) dieting, Atkins dieting, intermittent fasting (Ramadan), anorexia nervosa, obesity, sudden weight loss, prolonged and excessive exercise, alcohol intake, primary biliary cirrhosis, bariatric surgery (laparoscopic sleeve gastrectomy), gastric surgery.
		* Gut dysbiosis suspected as a pathogenetic factor.

Ketosis and PP

Among the list of potential aetiological factors of PP, ketosis has gained attention within the last decade and has become a trending topic. There has been a 'miniepidemic' of PP due to popular celebrity-endorsed KD and bariatric surgeries[3,5,7,11,35] Public awareness of the condition has also boosted, and fans of the paleo and KD have coined the term 'keto rash' for this condition[10,12] As dermatologists, we have recently been confronted with an outbreak of PP prompted by KD. We have observed not only classical PP, but atypical bullous variants and localizations as well [Figures 1-3].

The relevant literature shows that ketosis is the main culprit in PP provoked by diet changes and bariatric surgeries and patients with PP exhibit increased levels of urine and/or serum ketones[2,4,5,7,9,11,13,14,26,35,41,47,48] Diet changes are associated with PP occurrence in at least 50% of the cases, and KD is responsible for 40% of diet-provoked PP cases[9,13] Interestingly, PP recurs with each KD provocation and spontaneously resolves following resuming of a carbohydrate-enriched (>50 g/day) regular balanced diet[3,5,7,11-13,16,35] Thus, dietary adjustment alone may completely alleviate PP lesions in patients on restrictive diets, dispensing the need for medication use or phototherapy.[15]

A KD consists of reduced carbohydrate (5–10%; usually <50 g/day) and increased fat (75–80%) and protein (15–25%) content in the diet.[11,49] KD seems to bring health and well-being in all aspects of medicine.[50] Traditionally, it has been employed to treat epilepsy. Over the last decade, it has gained a worldwide reputation for its ability to provide rapid and substantial weight loss, and to help control of obesity, metabolic syndrome, diabetes, hypertension, cardiovascular disease and autoimmune disorders.[50,51] Based on small clinical trials, population studies and basic science research, a promising adjunctive therapeutic role for KD has been proposed in dermatologic disorders as well (such as acne, autoinflammatory syndromes, diabetic skin disease, cutaneous neoplasms and wound healing).[50]

KD is known to harbour anti-inflammatory properties through NLRP3 inflammasome suppression.[49,50] However, KD may also have detrimental effects on skin and paradoxically trigger or aggravate skin inflammation. A recent study showed that KD may exacerbate psoriasis through neutrophil and IL-17A-mediated cutaneous inflammation.[49] Ketone bodies (beta-hydroxybutyrate, acetoacetate, acetone) are believed to accumulate and induce a predominantly neutrophil-mediated perivascular inflammation, which might also pave the way to PP development.[2,5,11,13,16,17,48,52] Ketone bodies

have been shown to upregulate ICAM-1 expression on epidermal keratinocytes and LFA-1 expression on leucocytes.[16] ICAM-1/LFA-1 interaction is believed to be the pivotal event by which leucocytes can adhere to keratinocytes and endothelial cells, permitting leucocyte tissue migration and indirectly producing keratinocyte cytotoxicity.[16,26,36,45] Alternatively, ketone bodies may enter the cells and alter intracellular processes, directly leading to keratinocyte cytotoxicity.[5,17]

Conclusion, Current and Future Perspectives and Proposal of a Hypothesis

Based on this evidence, there is an ultimate cause-effect relationship between KD and PP. However, the exact pathogenic mechanisms remain unclear. Although there is no medical publication, the sweating has been conveyed to elicit PP and there are self-reports of oversweating after starting a KD. Ketone bodies are known to alter body odour and excreted in sweat during exercise or emotional sweating.[52,53] In our opinion, alterations in the cutaneous microbiome (skin dysbiosis) due to increased ketone excretion in sweat, possibly in patients with a genetical predisposition, might transpire PP pathogenesis. Gut dysbiosis has been suspected as a pathogenetic factor in PP, and hence, several studies have investigated the alterations in gut microbiome by KD. So far, no study has assessed KD-induced alterations in the cutaneous microbiome. Although PP has been proposed to represent a reactive inflammatory condition against follicular colonization by bacteria,[54] there are no data on the type and spectrum of bacterial colonies involved.

The clinical and histological settings of PP are reminiscent of either an allergic contact dermatitis (where a LMW antigen induces delayed-type hypersensitivity reaction) or a superficial infection (with clonal proliferation of antigen-specific CD4+ and CD8+ T cells). We postulate that a microorganism (bacteria or fungus) residing within or colonizing the skin and feeding on ketones and/or lipids might be responsible for PP development, either directly or indirectly through antigenic mimicry (cross-reaction). Clinically, a staged presentation (initially inflammatory lesions, later resolution with pigmentation), reticulation (Koebnerization?), instant response to antibiotics and site-specific recurrence (antigenic recall?) of PP lesions might point to a superficial infection or epidermal self-antigen (such as HSP's). Histopathologically, a staged presentation (neutrophils initially and lymphocytes later), an epidermal CD8+ T-cell and dermal CD4+ T-cell infiltration may imply that innate and in due course adaptive immune systems are activated. A CD8+ T-cell attack directly to a microorganism or indirectly to an epidermal self-antigen and production of IL-8 and other cytokines by the infiltrating CD4+ T cells and keratinocytes (creating a neutrophil-mediated inflammatory loop) might underlie PP pathogenesis. We presume that both autoinflammation and autoimmunity might be involved. In this scenario, Th17 subset of CD4+ T cells, responsible for neutrophilic and monocytic inflammation for defence against extracellular bacteria and fungi, and stimulated via IL-1 signalling, might play a crucial role.

Future research should assess ketone excretion and antimicrobial peptides (dermcidin) in eccrine sweat, ketone utilization by cutaneous microbiome, fatty acid composition of sebum, cutaneous microbial diversity and dysbiosis (particularly focusing on Malassezia spp. and Cutibacterium), innate (TLR, NLR, NLRP-3 inflammasome activation, caspase-1, IL-1, IL-6, IL-8, TNF-α, TGF-β, IL-10 and IL-18) and adaptive (particularly Th17 cytokines IL-17A-F, IL-22, IL-20 and those involved in Th17 lineage differentiation such as IL-23) immune system components in patients with PP.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Nagashima M, Ohshiro A, Shimizu N. A peculiar pruriginous dermatosis with gross reticular pigmentation. Jap J Derm 1971;81:78-91.
2. Yang J, Jiao S, Zhang M. Use of minocycline for the treatment of prurigo pigmentosa with intraepidermal vesiculation: A case report. J Int Med Res 2021;49:3000605211015593.
3. Alkeraye S, AlMuqrin A, AlQahtani SM, AlSwayyed M, Alhuzimi A. Twenty-five year-old female with sudden onset itchy skin eruption over her upper back and chest three weeks after starting a ketogenic diet. Ann Saudi Med 2019;39:444-5.
4. Yau EKY, Yau KC. A young gentleman with reticulated hyperpigmentation: A case of prurigo pigmentosa. Hong Kong J Dermatol Venereol 2012;20:121-4.
5. Almaani N, Al-Tarawneh AH, Msallam H. Prurigo pigmentosa: A clinicopathological report of three middle eastern patients. Case Rep Dermatol Med 2018;2018:9406797.
6. Wang X, Xu C. Bullous prurigo pigmentosa. Open Life Sci 2019;14:214-6.
7. Alsebayel MM, Ghobara YA, Al-Issa A. Facial prurigo pigmentosa after bariatric surgery. Cureus 2020;12:e6909.
8. Chao LL, Lu CF, Shih CM. Molecular detection and genetic identification of Borrelia garinii and Borrelia afzelii from patients presenting with a rare skin manifestation of prurigo pigmentosa in Taiwan. Int J Infect Dis 2013;17:e1141-7.
9. Goh SW, Adawiyah J, Md Nor N, Yap F, Ch'ng P, Chang CC. Skin eruption induced by dieting-An underdiagnosed skin disease in Malaysia. Malays Fam Physician 2019;14:42-6.
10. Lonowski S, Levins P. A unique case of prurigo pigmentosa related to ketogenic diet. Dermatol Online J 2020;26:13030/qt56p0z9rp.
11. Alshaya MA, Turkmani MG, Alissa AM. Prurigo pigmentosa following ketogenic diet and bariatric surgery: A growing association. JAAD Case Rep 2019;5:504-7.

12. Maco MW, Lee E, Wu Y, Lee R. Treatment of Prurigo Pigmentosa with diet modification: A medical case study. Hawaii J Med Public Health 2018;77:114-7.
13. Mufti A, Mirali S, Abduelmula A, McDonald KA, Alabdulrazzaq S, Sachdeva M, *et al*. Clinical manifestations and treatment outcomes in prurigo pigmentosa (Nagashima disease): A systematic review of the literature. JAAD Int 2021;3:79-87.
14. Teraki Y, Teraki E, Kawashima M, Nagashima M, Shiohara T. Ketosis is involved in the origin of prurigo pigmentosa. J Am Acad Dermatol 1996;34:509-11.
15. Wang HF, Zhou XF, Deng XF, Li YC, Zhang L, Gu HZ, Zhai ZF. Dermoscopy and reflection confocal microscope features of pigmented prurigo. Skin Res Technol 2023;29:e13258.
16. Hartman M, Fuller B, Heaphy MR. Prurigo pigmentosa induced by ketosis: Resolution through dietary modification. Cutis 2019;103:E10-3.
17. Onaygil E, Songur A, Kutlubay Z, Demirkesen C. Early stage prurigo pigmentosa: A case report. Turk Patoloji Derg 2018;34:182-5.
18. Erbagci Z. Prurigo pigmentosa in association with Helicobacter pylori infection in a Caucasian Turkish woman. Acta Derm Venereol 2002;82:302-3.
19. Toberer F, Enk A, Fink C, Haenssle HA. Prurigo Pigmentosa in a 21-year-old caucasian man: A clinicopathological correlation. Acta Derm Venereol 2017;97:1130-1.
20. Lin SH, Ho JC, Cheng YW, Huang PH, Wang CY. Prurigo pigmentosa: A clinical and histopathologic study of 11 cases. Chang Gung Med J 2010;33:157-63.
21. Shevchenko A, Valdes-Rodriguez R, Hsu S, Motaparthi K. Prurigo pigmentosa: Case series and differentiation from confluent and reticulated papillomatosis. JAAD Case Rep 2017;4:77-80.
22. Corley SB, Mauro PM. Erythematous papules evolving into reticulated hyperpigmentation on the trunk: A case of prurigo pigmentosa. JAAD Case Rep 2015;1:60-2.
23. Joyce AP, Horn TD, Anhalt GJ. Prurigo pigmentosa. Report of a case and review of the literature. Arch Dermatol 1989;125:1551-4.
24. Yanguas I, Goday JJ, González-Güemes M, Berridi D, Lozano M, Soloeta R. Prurigo pigmentosa in a white woman. J Am Acad Dermatol 1996;35:473-5.
25. Schepis C, Siragusa M, Palazzo R, Ussia AF, Cavallari V. Prurigo pigmentosa treated with minocycline. Br J Dermatol 1996;135:158-9.
26. Gür-Toy G, Güngör E, Artüz F, Aksoy F, Alli N. Prurigo pigmentosa. Int J Dermatol 2002;41:288-91.
27. Baykal C, Buyukbabani N, Akinturk S, Saglik E. Prurigo pigmentosa: Not an uncommon disease in the Turkish population. Int J Dermatol 2006;45:1164-8.
28. Akoglu G, Boztepe G, Karaduman A. Prurigo pigmentosa successfully treated with low-dose isotretinoin. Dermatology 2006;213:331-3.
29. Dijkstra JW, Bergfeld WF, Taylor JS, Ranchoff RE. Prurigo pigmentosa. A persistent lichenoid reaction to bismuth? Int J Dermatol 1987;26:379-81.
30. Kim MH, Choi YW, Choi HY, Myung KB. Prurigo pigmentosa from contact allergy to chrome in detergent. Contact Dermatitis 2001;44:289-92.
31. Cota C, Donati P, Amantea A. Prurigo pigmentosa associated with an atopic diathesis in a 13-year-old girl. Pediatr Dermatol 2007;24:277-9.
32. Shannon JF, Weedon D, Sharkey MP. Prurigo pigmentosa. Australas J Dermatol 2006;47:289-90.
33. Ekmekci TR, Altunay IK, Koslu A. Prurigo pigmentosa treated with doxycycline. Dermatol Online J 2006;12:9.
34. Requena Caballero C, Nagore E, Sanmartín O, Botella-Estrada R, Serra C, Guillén C. Vesiculous prurigo pigmentosa in a 13-year-old girl: Good response to isotretinoin. J Eur Acad Dermatol Venereol 2005;19:474-6.
35. Al-Dawsari NA, Al-Essa A, Shahab R, Raslan W. Prurigo pigmentosa following laparoscopic gastric sleeve. Dermatol Online J 2019;25:13030/qt2b20c2w8.
36. Gürses L, Gürbüz O, Demirçay Z, Kotiloglu E. Prurigo pigmentosa. Int J Dermatol 1999;38:924-5.
37. Böer A, Ackerman AB. Prurigo pigmentosa is distinctive histopathologically. Int J Dermatol 2003;42:417-8.
38. Miyachi Y, Yoshioka A, Horio T, Imamura S, Niwa Y. Prurigo pigmentosa: A possible mechanism of action of sulfonamides. Dermatologica 1986;172:82-8.
39. Boer A, Asgari M. Prurigo pigmentosa: An underdiagnosed disease? Indian J Dermatol Venereol Leprol 2006;72:405-9.
40. Matsumoto C, Kinoshita M, Baba S, Suzuki H, Kanematsu S, Kanematsu N. Vesicular prurigo pigmentosa cured by minocycline. J Eur Acad Dermatol Venereol 2001;15:354-6.
41. Hanami Y, Yamamoto T. Bullous prurigo pigmentosa in a pregnant woman with hyperemesis gravidarum. J Dermatol 2015;42:436-7.
42. Cox NH. Prurigo pigmentosa. Br J Dermatol 1987;117:121-4.
43. Mehta H, Bishnoi A, Vinay K, Kaushik A, Kumaran MS, Kollabathula A, *et al*. Prurigo Pigmentosa: Dermoscopic evaluation. Dermatol Pract Concept 2021;11:e2021115.
44. Asgari M, Daneshpazhooh M, Chams Davatchi C, Böer A. Prurigo pigmentosa: An underdiagnosed disease in patients of Iranian descent? J Am Acad Dermatol 2006;55:131-6.
45. Teraki Y, Shiohara T, Nagashima M, Nishikawa T. Prurigo pigmentosa: Role of ICAM-1 in the localization of the eruption. Br J Dermatol 1991;125:360-3.
46. An I, Ucmak D, Ibiloglu I, Demir V, Akdeniz S. Colchicine may be of therapeutic benefit in prurigo pigmentosa. Pediatr Dermatol 2018;35:e202-3.
47. Murao K, Urano Y, Uchida N, Arase S. Prurigo pigmentosa associated with ketosis. Br J Dermatol 1996;134:379-81.
48. Mogilnicka I, Bogucki P, Ufnal M. Microbiota and malodor-etiology and management. Int J Mol Sci 2020;21:2886.
49. Locker F, Leitner J, Aminzadeh-Gohari S, Weber DD, Sanio P, Koller A, *et al*. The Influence of ketogenic diets on psoriasiform-like skin inflammation. J Invest Dermatol 2020;140:707-10.e7.
50. Fomin DA, Handfield K. The ketogenic diet and dermatology: A primer on current literature. Cutis 2020;105:40-3.
51. Osmani S, Smidt AC, Phan CM, Johnson DW. Acquired acrodermatitis enteropathica from a ketogenic diet. JAAD Case Rep 2021;9:75-7.
52. Schranner D, Kastenmüller G, Schönfelder M, Römisch-Margl W, Wackerhage H. Metabolite concentration changes in humans after a bout of exercise: A systematic review of exercise metabolomics studies. Sports Med Open 2020;6:11.
53. Smeets MAM, Rosing EAE, Jacobs DM, van Velzen E, Koek JH, Blonk C, *et al*. Chemical fingerprints of emotional body odor. Metabolites 2020;10:84.
54. Kafle SU, Swe SM, Hsiao PF, Tsai YC, Wu YH. Folliculitis in prurigo pigmentosa: A proposed pathogenesis based on clinical and pathological observation. J Cutan Pathol 2017;44:20-7.