A case report of lichen nitidus: From classical to perforating

Dear Editor,

Lichen nitidus is a rare dermatosis characterised by multiple, discrete, pinpoint-sized, skin-coloured papules with flat, shiny surfaces, commonly occurring on the flexor surface of the upper limb, genitalia, chest, abdomen and dorsum of the hand. The histological characteristics comprise a dense, sharply circumscribed infiltrate composed of lymphocytes, epithelioid cells and Langhans giant cells. Elongated rete ridges adhere to and surround the infiltrate, in a "ball-in-claw" configuration that is usually confined to two or three dermal papillae. Lichen nitidus variants include the linear, spinous follicular, generalised, actinic and perforating types. Here, we report a case of lichen nitidus in which classical and perforating lesions appeared successively.

An otherwise healthy 24-year-old Chinese Han man presented with an asymptomatic papule on the upper limb that had persisted for 1 year. Physical examination revealed multiple, discrete, pinpoint-to-small, rice grain-sized, skin-coloured, firm, shiny, monomorphic papules on his left wrist [Figure 1a]. Skin biopsy revealed a dense, superficial lymphohistiocytic infiltrate bounded by elongated rete ridges in a broadened dermal papilla [Figure 1b]. He was diagnosed with lichen nitidus and administered topical flumetasone ointment twice daily and tazarotene gel every night for 6 months. The lesions subsided completely following the treatment. However, he returned to our clinic 10 months later with similar lesions on both palms. These lesions had coalesced into patches with umbilicated centres [Figure 1c]. Dermoscopy revealed a yellowish-brown central keratotic core with marginal lifting, surrounded by annular, pale white halo-like scales [Figure 1d]. Reflectance confocal microscopy showed three concentric areas with elliptical, poorly refractile centre, an irregular brightly refractile area and a peripheral halo with poorer refraction [Figure 1e]. Punch biopsy of the left palm revealed a dense lymphohistiocytic infiltrate covered by atrophic epidermis and bounded by elongated rete ridges in a broadened dermal papilla. Keratin and lymphohistiocytic cells extended to the surface via a transepithelial perforation channel [Figure 1f]. Masson-trichrome stain identified no transepithelial collagen migration. The final diagnosis was perforating lichen nitidus [Figure 1g]. He was prescribed betamethasone dipropionate cream twice daily and 0.1% tretinoin cream every night for 4 weeks. The lesions showed slight improvement and regular follow-up is ongoing.

Perforating lichen nitidus is a rare clinical variant first reported in 1981 by Bardach.[1] Only 13 cases, including ours, have been described to date.[1–11] The characteristics of the disease are summarised in Table 1, according to our systematic literature review. Perforating lichen nitidus occurs

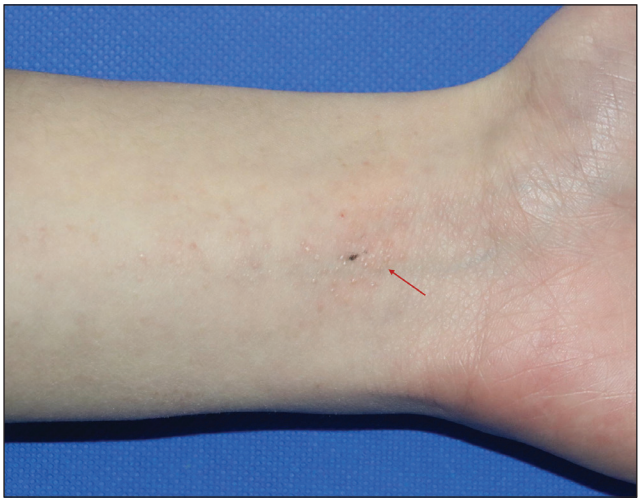

Figure 1a: Multiple, small, skin-coloured, shiny papules on the wrist (indicated by red arrow).

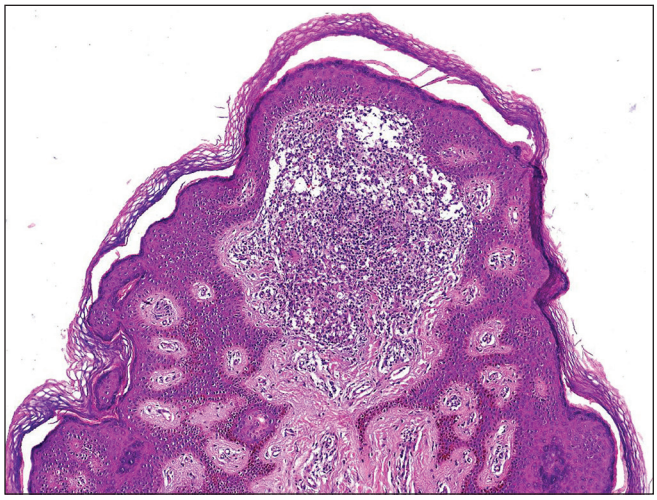

Figure 1b: A dense, superficial lymphohistiocytic infiltrate bounded by elongated rete ridges (H&E, 100x).

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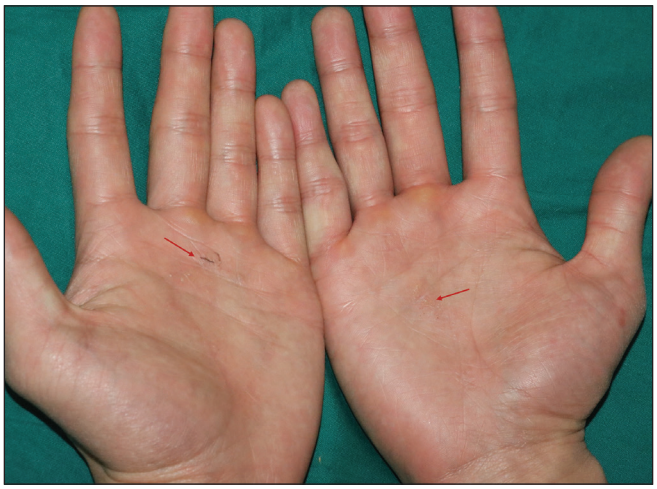

Figure 1c: Umbilicated papules coalesced into patches on both palms (indicated by red arrow).

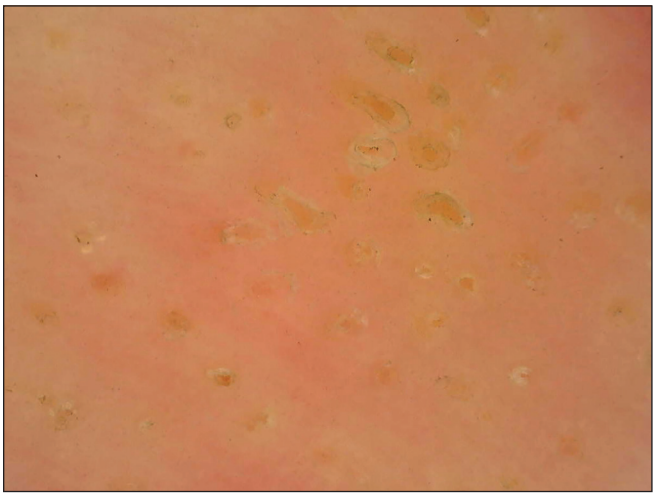

Figure 1d: Dermoscopy: yellowish-brownish central keratotic core with marginal lifting, surrounded by annular, pale white halo-like scales (50x).

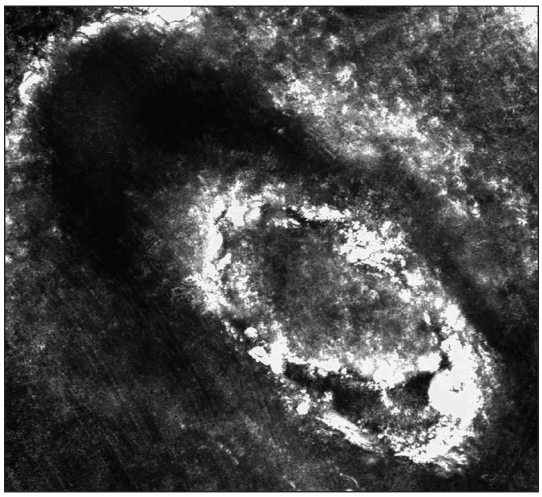

Figure 1e: Reflectance confocal microscopy: three concentric areas with elliptical poorly refractile centre, an irregular brightly refractile area and a peripheral halo with poorer refraction.

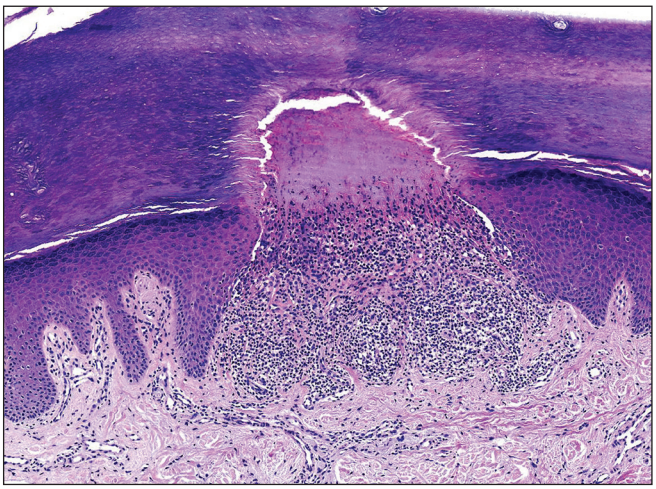

Figure 1f: A dense lymphohistiocytic infiltrate covered by atrophic epidermis and bounded by elongated rete ridges in a broadened dermal papilla. Keratin and lymphohistiocytic cells extended to the surface via a transepithelial perforation channel (H&E, 100x).

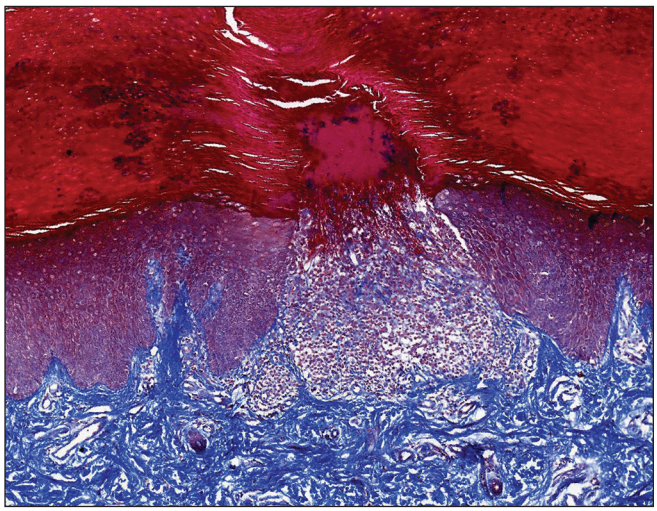

Figure 1g: No extrusion of collagen fibres in the perforating channel (Masson stain, 100x).

most commonly in children and young adults, with 10 (76.9%) patients aged 10–30 years. The 13 reported cases comprised eight Asians, four Caucasians and one African-American. Perforating lichen nitidus lesions are similar to classical lichen nitidus lesions except for the appearance of central umbilicated papules seen in seven (53.9%) cases. According to previous reports[9–11] and our findings, dermoscopic findings of perforating lichen nitidus demonstrated a well-defined, light-brown keratin plug surrounded by a ring-shaped, silvery-white area, whereas classical lichen nitidus lesions usually defined with a round, elevated, shiny and smooth appearance.[12] The most important histopathological feature is a transepidermal perforating channel that can extrude the dermal material into the stratum corneum. The treatment options for perforating lichen nitidus include topical glucocorticoids and tretinoin; however, in one patient, the lesions healed spontaneously.[4]

Table 1: The clinical characteristics of all reported cases of perforating lichen nitidus

Cases	Ethnicity	Age (year)/ Duration	Sex	Diseased parts	Lesion appearance	Dermoscopy	Therapy response
Bardach[1]	Caucasians	8/Few months	Male	Trunk and extremities	Flat, shiny papules	None	None reported
Banse-Kupin *et al.*[2]	African American	22/1 month	Male	Trunk, extremities and penis	Flesh-coloured, dome-shaped papules	None	None reported
Itami *et al.*[3]	Asian	32/3 years	Male	Hand and fingers	Discrete, pinhead-sized or half-rice corn-sized, flesh-coloured papules	None	None reported
Yoon *et al.*[4]	Asian	18/2 years	Female	Hands, wrists, forearms, elbows and knees	Skin-coloured, flat shiny papules	None	Improvement after topical steroid for 1 year
Yoon *et al.*[4]	Asian	20/11 months	Female	Wrists, elbows, knees and dorsal feet	Flat shiny papules, some of them were umbilicated	None	Spontaneous clearance
Arrue *et al.*[5]	Caucasians	35/>20 years	Male	Palms, feet and lateral border fingers	Skin-coloured, shiny, firm, monomorphic papules	None	No response to topical corticosteroid
Vijaya *et al.*[6]	Asian	14/6 months	Female	Dorsum of the hands and feet	Small, shiny papules, some of them were umbilication	None	None reported
Zhang *et al.*[8]	Asian	15/5 years	Male	Right palm	Small, skin-coloured papules with central umbilication	None	None reported
Zussman *et al.*[7]	Caucasians	25/1 year	Male	Dorsal digit	Small papules grouped into patches	None	No response to topical acid wraps or clobetasol spray
Martinez-Mera *et al.*[9]	Caucasians	30/1 year	Male	Dorsal of hands and fingers	Umbilicated papules grouped into patches	Light-brown keratin plug surrounded by a whitish, annular cloud-like area	Slightly improvement to topical corticosteroid for several weeks
Li *et al.* [11]	Asian	10/2 years	Male	Upper extremities, trunk, lower jaw, and nape	Numerous, flesh-coloured, shiny papules, some of them were umbilicated	Whitish-brownish keratin plug in the centre, whitish annular cloud-like area and a peripheral brown pigmentation	Slightly improvement to topical corticosteroid for 2 weeks, but relapsed after discontinuation
LeWitt *et al.*[10]	Asian	32/2 months	Male	Dorsal of hands and feet, palms, soles	Small white to pink molluscum-like umbilicated papules	Depressed central keratotic core rimmed by fine white scale	Significant improvement with topical corticosteroid for 6 weeks
Present case	Asian	24/10 months	Male	Palms	Umbilicated papules grouped into patches	Yellowish-brownish keratotic core surrounded by pale-white halo-like scales	Slight improvement with topical corticosteroid for 4 weeks

There are four documented cases of co-existence of classical and perforating lichen nitidus, including ours, suggesting that different types of lichen nitidus can occur in the same patient.[4,6,11] In addition, we performed the first reflectance confocal microscopy feature analysis of perforating lichen nitidus. The poorly refractile area in the centre corresponds to the histopathology of perforated keratin and lymphohistiocytic cells in the surface. The irregular, brightly refractile area correlates on histopathology with the fissure of the stratum corneum overlying the infiltrate. The peripheral halo with poorer refraction correlates with the adjacent stratum corneum. The reflectance confocal microscopy characteristics of perforating lichen nitidus differ from those of the classical type.[13]

Several mechanisms, including an abnormality of epidermal proliferation and differentiation, alteration of connective tissues, mechanical disruption, participation of immunological factors and binding of altered dermal constituents or foreign bodies to some unidentified receptors have been proposed to explain the transepithelial perforation phenomenon.[3,14] However, given the paucity of documented cases, it is difficult to clarify the specific mechanism of perforation in lichen nitidus. Hence, the identification of more cases of this phenomenon will lead to a better understanding of its aetiology, pathophysiology and clinical course in future.

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Dermoscopic and in vivo reflectance confocal microscopic features of endogenous ochronosis

Dear Editor,

Endogenous ochronosis (EO) is a rare hereditary metabolic disease caused by the decreased activity of the enzyme, homogentisate 1,2 dioxygenase. Bluish-grey discoloration of the skin and mucosa is typically seen in this disorder.[1] The features of ochronosis by in vivo reflectance confocal microscopy (RCM) have scarcely been reported and that too, in only the exogenous type. The present case demonstrates the clinical features, dermoscopic findings, RCM findings and the response to the treatment in a case of EO. We were unable to find any previous reports of RCM features of EO.

A 49-year-old male presented with pigmented skin lesions on his ears and pain and limitation of movement in both knees since five years. There was no history of intake of systemic drugs or topical medications. There were bluish-grey nodules seen on both the auricles [Figures 1a and 1b] and yellowish-brown discoloration of the conjunctivae [Figures 1c and 1d]. The left knee was swollen and had a limited range of motion [Figure 1e]. The magnetic resonance imaging and X-ray of the knee revealed osteophytic degenerative changes [Figure 1f]. Liver and kidney function tests, complete blood count, and rheumatoid factor were within normal limits. ANA was positive at a titer of 1/160. The patient's urine darkened after two hours of exposure to the air. The level of homogentisic acid detected in urine or blood could not be evaluated for technical reasons. The homozygous mutation was detected in the HGD gene (exon 10, c.731_749del p.Thr244ArgfsTer30). Renal or cardiovascular involvement was not detected.

Dermoscopic examination of the ear lesions revealed a homogeneous brown to grey-blue pigmentation and linear vessels on a pinkish background [Figure 2a and 2b]. On reflectance confocal microscopy (Vivascope 3000; CALIBER ID, Rochester, NY, USA), the epidermis exhibited a normal honeycombed pattern with inflammatory and dendritic cells. The dermo-epidermal junction had a normal ridged pattern with normal-edged papillae. The dermis had hypo-refractile irregular, disorganised dark areas of different shapes and sizes [Figures 3a and 3b]. There were thin irregular collagen fibres with increased inflammatory cells and vessels between these dark areas. There were no atypical cells.

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