HEME : Health and Medical Journal
pISSN : 2685 – 2772
eISSN : 2685 – 404x
Available Online at : https://jurnal.unbrah.ac.id/index.php/heme/issue/view/78

Beyond the Scales: Histopathologic Insights into Pityriasis Lichenoides Chronica

Utiya Nur Laili[1*], Alamanda Murasmita[2]

[1]Department of Dermatology and Venereology, Faculty of Medicine, Sebelas Maret University/ Dr. Moewardi General Hospital Surakarta - Indonesia
[2]Department of Dermatology and Venereology Sebelas Maret Hospital Kartasura - Indonesia
E-mail: utiya.laili@gmail.com

Abstract

Introduction: Pityriasis lichenoides is an inflammatory skin condition characterized by papular, squamous or necrotic lesions. Currently, studies that accurately correlate clinical subtypes with histologic features are rare. The aim of this study is to describe the clinical and histopathologic features of chronic pityriasis lichenoides especially in adults. **Case:** A 30-year-old male with complaints of reddish papules and nodules on the body and both hands since 10 years ago. The patient initially complained of reddish papules and nodules on the chest then increasingly spread to the whole body, back and both hands. The papules and nodules were slightly painful and did not itch. On histopathological examination, the epidermal layer showed hyperkeratosis, acanthosis, spongiosis and basal cell vacuolization, while the dermis layer showed perivascular infiltrate dominated by lymphocytes with eosinophil cells obtained 1 cell and a little melanin dropping. **Discussion:** The histopathologic impression supports the diagnosis of chronic pityriasis lichenoides. The histopathologic picture of the patient showed vacuolar interface dermatitis with parakeratosis, exocytosis, erythrocyte extravasation, necrotic keratinocytes, spongiosis and perivascular lymphocytic infiltration. This case report shows the characteristic histopathologic features of lichenoid (interface) dermatitis and lymphocytic vasculitis which are typical histopathologic markers in patients with chronic pityriasis lichenoides. **Conclusion:** Pityriasis lichenoides is essentially a lymphocytic vasculitis with associated inflammatory cell infiltration showing exocytosis into the epidermis with blurring of the dermoepidermal interface.

Keywords: pityiriasis, lichenoides, histopathology, lichenoid, interface

Abstrak

Pendahuluan: *Pitiriasis lichenoides adalah kondisi peradangan kulit yang ditandai dengan lesi papular, skuamosa, atau nekrotik. Saat ini, studi yang secara akurat mengkorelasikan subtipe klinis dengan gambaran histologis masih jarang. Tujuan penelitian ini adalah untuk menggambarkan gambaran klinis dan histopatologis pitiriasis lichenoides kronis, terutama pada orang dewasa.* ***Kasus:*** *Seorang pria berusia 30 tahun dengan keluhan papula dan nodul kemerahan pada tubuh dan kedua tangannya sejak 10 tahun yang lalu. Pasien awalnya mengeluhkan papula dan nodul kemerahan di dada, kemudian semakin menyebar ke seluruh tubuh, punggung, dan kedua tangannya. Papula dan nodul tersebut sedikit nyeri dan tidak gatal. Pada pemeriksaan histopatologi, lapisan epidermis menunjukkan hiperkeratosis, akantosis, spongiosis, dan vakuolisasi sel basal, sedangkan lapisan dermis menunjukkan infiltrat perivaskular yang didominasi oleh limfosit dengan sel eosinofil yang didapatkan 1 sel dan sedikit melanin dropping.* ***Pembahasan:*** *Gambaran histopatologi mendukung diagnosis pityriasis likenoides kronis. Gambaran histopatologi pasien menunjukkan dermatitis antarmuka vakuolar dengan parakeratosis, eksositosis, ekstravasasi eritrosit, keratinosit nekrotik, spongiosis, dan infiltrasi limfosit perivaskular. Laporan kasus ini menunjukkan gambaran histopatologi khas dermatitis likenoid (antarmuka) dan vaskulitis limfositik yang merupakan penanda histopatologi khas pada pasien dengan pityriasis likenoides kronis.* ***Kesimpulan:*** *Pityriasis lichenoides pada dasarnya adalah vaskulitis limfositik dengan infiltrasi sel inflamasi terkait yang menunjukkan eksositosis ke dalam epidermis dengan pengaburan antarmuka dermoepidermal.*

Kata kunci: *pityriasis, lichenoides, histopatologi, lichenoid, antarmuka*

I. INTRODUCTION

Pityriasis lichenoides is an inflammatory skin condition characterized by papular, squamous or necrotic lesions.[1] It is classified into three forms, namely pitiriasis lichenoides et varioliformis akuta (PLEVA), chronic pityriasis lichenoides and febrile ulceronecrotic Mucha-Habermann disease (FUMHD), however in the literature the terms acute and chronic refer to the clinical features of the lesions and not the course of the disease.[1,2] The etiology of pityriasis lichenoides is unknown, but some literature suggests hypersensitivity reactions to various antigens may trigger the development of the disease.

The incidence and prevalence of pityriasis lichenoides need to be better documented. There are no specific risk factors associated with the disease, but there is a racial or geographical predisposition.[1,3] An epidemiologic study by Koh et al. in 2019 in Singapore showed the incidence of pityriasis lichenoides in 33% of patients with a mean age of 16 years or younger, with all patients being Chinese despite Singapore having a multiethnic population.[4]

The main clinical features of pityriasis lichenoides are erythematous and hyperpigmented papules with mica-like squamous, which can be hemorrhagic, acneiform or necrotic.[2] Predilection can be in the anterior or posterior trunk, extremities or generalized.[3] Widespread ulceronecrotic lesions are associated with high fever, susceptibility to secondary infection and a high mortality rate.[1] Systemic manifestations include interstitial pneumonitis, abdominal pain, malabsorption, etc.

Histopathological features of pityriasis lichenoides are generally characterized by vacuolar interface dermatitis with parakeratosis, exocytosis, erythrocyte extravasation, necrotic keratinocytes, spongiosis and perivascular lymphocytic infiltration.[5] A study by Lupu et al. in 2021 in France found a significant relationship between the clinical and histologic features. All patients with mild histologic patterns had only papules, whereas those with lymphomatoid patterns had isolated necrotic or mixed lesions. Mild and lymphomatoid patterns have similarities in the spectrum of epidermal injury and lymphocytic infiltrate density, but no study has yet found a relationship between histologic pattern and disease course.[1,3,6] Currently, studies that accurately correlate clinical subtypes with histologic features are rare. This case report aims to provide knowledge about the clinical and histopathologic features of chronic pityriasis lichenoides, especially in adults.

II. CASE REPORT

A 30-year-old man came to the dermatology and venereology polyclinic of Dr. Moewardi Surakarta General Hospital complaining of reddish pustules on his body and both hands 10 years ago. The patient initially complained of reddish pustules on the chest, then increasingly spread to the whole body, back, and both hands. The pustules felt a little painful and did not itch. These complaints increasingly interfere with his activities. The patient then went to the previous hospital and received antibiotic therapy, which was taken for 5 days every 3 months. The patient's complaints improved after taking the medicine, but sometimes they reappeared. The patient was then referred to the regional general hospital (RSUD), Dr. Moewardi Surakarta, for further treatment. The patient had a history of food allergies in the past medical history.

The patient had a history of food allergies such as chicken, eggs, and seafood, while a history of drug allergies, similar complaints, and other diseases such as diabetes mellitus and hypertension were denied. In the family history, the patient's mother had a history of allergic rhinitis, while the history of similar

complaints, drug/food allergies, and other diseases such as diabetes mellitus and hypertension was denied. On physical examination, the patient's general condition was compos mentis consciousness with blood pressure 128/74 mmHg, pulse 82x/min, respiratory rate 20x/min, temperature 36.6°C. The patient's nutritional status was 173 cm tall and 73 kg with a body mass index of 24.39 kg/cm2 (normoweight). On dermatologic examination, the anterior and posterior trunk regions and bilateral superior extremities showed papules, erythematous nodules, and discrete scattered multiple hyperpigmentation (Figure 1A-I).

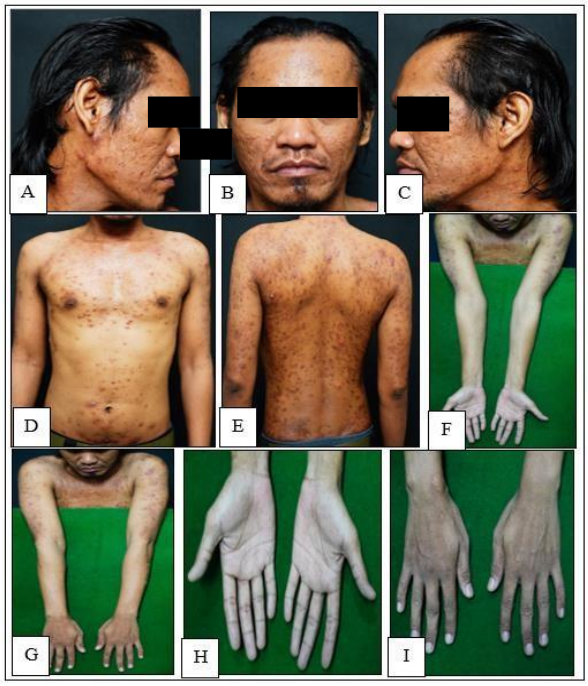

FIGURE 1. (A-I). THE GENERALIZED REGION SHOWS DISCRETE SCATTERED MULTIPLE ERYTHEMA PATCHES WITH OVERLYING SCUAMA

Differential diagnoses of the patient are chronic pityriasis lichenoides, lichen planus, and psoriasis gutata. Laboratory examination showed anti-HIV, anti-HCV, and non-reactive HbsAg. The patient underwent a skin biopsy examination followed by a histopathological examination with hematoxylin staining; with the result, the epidermal layer showed hyperkeratosis, acanthosis, spongiosis, and basal cell vacuolization, while the dermis layer showed perivascular infiltrate dominated by lymphocytes with eosinophil cells obtained one cell and a little melanin dropping. The histopathologic impression supported the diagnosis of chronic pityriasis lichenoides (Figure 2A-C).

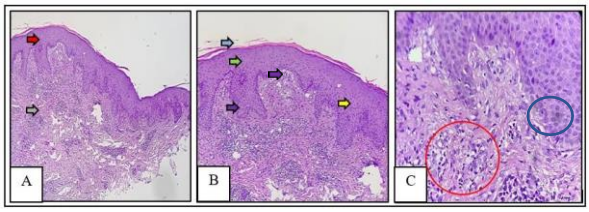

FIGURE 2. (A) EPIDERMAL LAYER (RED ARROW) AND DERMIS LAYER (GRAY ARROW) (HE 4X). (B) THE EPIDERMIS LAYER SHOWS HYPERKERATOSIS (BLUE ARROW), ACANTHOSIS (GREEN ARROW), SPONGIOSIS (YELLOW ARROW) AND BASAL CELL VACUOLIZATION (PURPLE ARROW) (HE 10X) (C) THE DERMIS LAYER SHOWED PERIVASCULAR INFILTRATES DOMINATED BY LYMPHOCYTES (RED CIRCLES) WITH 1 EOSINOPHIL CELL AND A SLIGHT DROPPING OF MELANIN (BLUE ARROW) (HE 40X).

Based on the results of the history, physical examination and supporting examination result, the patient was diagnosed with chronic pityriasis lichenides. The patient then received azithromycin 500mg/24h tablet orally for 5 days, desoxymethasone 0.05% cream applied to the reddish lesion area 2 times a day (day and night) and soft u derm® (urea 10%) applied to the dry area 2 times a day (day and night).

III. DISCUSSION

Pityriasis lichenoides is an inflammatory skin condition characterized by papular, squamous, or necrotic lesions.[1] Pityriasis lichenoides is classified into three forms, namely pityriasis lichenoides et varioliformis akuta (PLEVA), pityriasis lichenoides chronica, and febrile ulceronecrotic Mucha-Habermann disease (FUMHD), however in the literature the terms acute and chronic refer to the clinical features of the lesions and not the course of the disease.[1,2] Specific pathogens that cause pityriasis lichenoides include adenovirus, Epstein-Barr virus, Toxoplasma gondii, parvovirus B19,

Staphylococcus aureus, and Streptococcus pyogenes.[2,7] Other diseases that cause pityriasis lichenoides include upper respiratory tract infections, varicella, viral gastroenteritis, streptococcal pharyngitis, and early-phase human immunodeficiency virus (HIV).[8] Substances such as tegafur, astemizole, and radiocontrast iodide have also been implicated in pityriasis lichenoides.[4,9] Some studies have also shown a predominant T cell clonality in the lesions of pityriasis lichenoides, which has resulted in its categorization as a type of lymphoid dyscrasia.4 Acute pityriasis lichenoides will subside after discontinuing the suspected agent, but chronic pityriasis lichenoides will have frequent recurrences.[5]

An epidemiologic study by Koh et al. in 2019 in Singapore showed an incidence of pityriasis lichenoides in 33% of patients aged 16 years or younger, with all patients being Chinese, despite Singapore having a multiethnic population.[4] The mean time to presentation from symptom onset was similar in both age groups, with children presenting earlier during the disease.[1,4] In Koh et al., 2021 study in Singapore, the mean time to recovery in adults was 8 months, while in children, the mean time to recovery was 21 months.[1,4]

The main clinical features of pityriasis lichenoides are erythematous and hyperpigmented papules with mica-like scales that can be hemorrhagic, acneiform, or necrotic.[2] Predilection can be in the anterior or posterior trunk, extremities, or generalized.[3] Widespread ulceronecrotic lesions are associated with high fever, susceptibility to secondary infection, and a high mortality rate.[1] Systemic manifestations include interstitial pneumonitis, abdominal pain, malabsorption, central nervous system symptoms, and rheumatologic symptoms. Ulcerations tend to heal with post-inflammatory hypopigmentation and atrophic scars. Histologic examination of skin biopsy specimens remains the standard for identification of pityriasis lichenoides, although definitive diagnosis is difficult.[3]

Pityriasis lichenoides belongs to parapsoriasis. Parapsoriasis is a heterogeneous group of asymptomatic squamous dermatoses with some clinical similarities to psoriasis. Three distinct entities have been recognized and included in the concept of parapsoriasis, namely pityriasis lichenoides, chronic superficial dermatitis (small plaque parapsoriasis (SPP) and digitate dermatosis), and large plaque parapsoriasis (LPP) (atrophic parapsoriasis, retiform parapsoriasis and patch-stage mycosis fungoides). The histopathological features of pityriasis lichenoides are generally characterized by vacuolar interface dermatitis with parakeratosis, exocytosis, erythrocyte extravasation, necrotic keratinocytes, spongiosis and perivascular lymphocytic infiltration.[5] In pityriasis lichenoides, erythrocyte infiltration is rare, but a study by Agarwala and Hafeez in 2021 in Palestine showed a case of pityriasis lichenides-like drug reaction with large numbers of eosinophils present in the inflammatory infiltrate.[5] This is a rarely reported histopathologic presentation of drug hypersensitivity reactions.[5,10]

Pityriasis lichenoides is essentially a lymphocytic vasculitis with associated inflammatory cell infiltration showing exocytosis into the epidermis with blurring of the dermoepidermal interface.[11,12] Some epidermal keratinocytes undergo apoptosis, resulting in confluent necrosis in the epidermis.[3] Pityriasis lichenoides chronica is similar to PLEVA, with the histopathologic difference being that the inflammatory process and degree of epidermal changes are more prominent in PLEVA than in pityriasis lichenoides chronica to involve the superficial vascular plexus and more erythrocytes in the epidermis and dermis. The conclusion in both is a lichenoid reaction (interface) with lymphocytic vasculitis (lymphocytic lichenoid vasculitis).[11] In this

patient, a histopathological picture in the epidermal layer shows hyperkeratosis, acanthosis, spongiosis, and vacuolization of basal cells, while in the dermis layer appears perivascular infiltrate dominated by lymphocytes with eosinophil cells obtained one cell and a little melanin dropping. This leads to a histopathological picture of pityriasis lichenoides characterized by vacuolar interface dermatitis with parakeratosis, exocytosis, erythrocyte extravasation, necrotic keratinocytes, spongiosis, and perivascular lymphocytic infiltration.

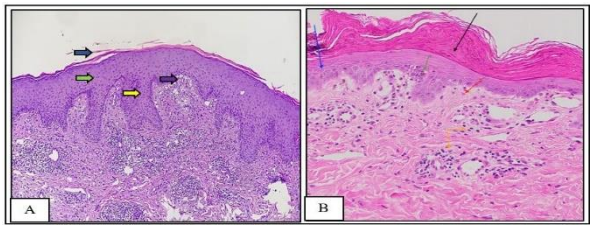

FIGURE 3. (A) HYPERKERATOSIS (BLUE ARROW), ACANTHOSIS (GREEN ARROW), SPONGIOSIS (YELLOW ARROW), AND VACUOLIZATION OF BASAL CELLS (PURPLE ARROW) (HE 10X) WERE OBSERVED IN THE EPIDERMAL LAYER (B) IN THE LITERATURE, THE HISTOPATHOLOGICAL EXAMINATION OF PITYRIASIS LICHENOIDES SHOWS FOCAL FEATURES OF PARAKERATOSIS (BLACK ARROW), SPONGIOSIS (BLUE ARROW), PERIVASCULAR LYMPHOCYTE INFILTRATION (ORANGE ARROW), KERATINOCYTES UNDERGOING APOPTOSIS (GREEN ARROW) AND MULTIPLE ERYTHROCYTE EXTRAVASATION (RED ARROW) (HE 20X) (ADAPTED FROM CLAREY, 2020).

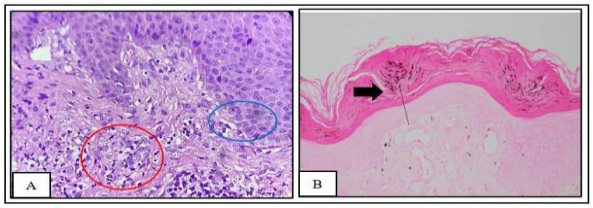

FIGURE 4. (A) THE DERMIS LAYER SHOWS PERIVASCULAR INFILTRATES DOMINATED BY LYMPHOCYTES (RED CIRCLES) WITH 1 EOSINOPHIL CELL AND A SLIGHT DROPPING OF MELANIN (HE 40X). (B) IN THE EPIDERMAL LAYER OF THE STRATUM CORNEUM, MELANIN DEPOSITION IS VISIBLE (BLACK ARROW) (FONTANA MASSON 40X) (ADAPTED FROM CLAREY, 2020).

Another differential diagnosis in this case is lichen planus. Lichen planus is a chronic inflammatory disease affecting the skin, oral and genital mucosa, scalp, and nails.[13] Lichen planus in the acute phase is often characterized by polygonal, purple, flat, itchy, and shiny erythematous papules and plaques with predilection for the flexor surfaces of the wrists and forearms, dorsal side of the wrists, front of the feet, neck and sacrum areas. On the surface of the papules, fine white mesh-like lines called Wickham striae can be seen.[13,14] A rare macular variant of lichen planus, it commonly affects adults with skin types III-V, including Indian, Latin American, and Middle Eastern populations.[13] Lichen planus is usually triggered by trauma (Koebner phenomenon).[13] Disease onset generally occurs between the third and fourth decades of life, affecting more women.[15] Lichen planus has an immunopathogenesis characterized by altered cellular immune responses mediated by T lymphocytes, where CD8+ T lymphocytes recognize and attack epidermal keratinocytes, causing profound pigmentary incontinence.[13] Typical histopathologic features of lichen planus include vacuolar degeneration of the epidermal basal cell layer, Civatte bodies (apoptotic basal keratinocytes), sawtooth rete ridge, band-like lichenoid or perivascular lymphocytic infiltrates in the upper dermis, incontinence of superficial pigment and melanophages, artifactual gaps between the epidermis and dermal papillae (Max-joseph cleft). Other less common findings include hyperkeratosis and epidermal atrophy.[12] Romiti et al. reported the histopathologic features of lichen planus lesions in 9 patients, showing atrophic epidermal features with basal cell vacuolar degeneration occurring in 78% of patients, basement membrane thickening (50%), interfollicular inflammatory infiltrates (66.6%) and interfollicular melanophages (100%). Perifollicular changes found in all cases included melanophages (100%), inflammatory infiltrates (42.8%), lichenoid infiltrates (25%), vacuolar degeneration of

the follicular basal cell layer (57%) and basement membrane thickening (25%).[16] On histopathological examination of patients with HE staining of dorsal skin biopsies in the epidermal layer, hyperkeratosis, acanthosis, spongiosis, and vacuolization of basal cells were seen, while in the dermis layer, perivascular infiltrates dominated by lymphocytes with eosinophil cells obtained one cell and a little melanin dropping. In contrast, in lichen planus, interfollicular inflammatory infiltrates and perifollicular changes were found in all cases, including melanophages (100%), Civatte bodies, band-like lichenoid, Max-joseph cleft, so the diagnosis of lichen planus could be excluded.

Another differential diagnosis is psoriasis gutata. Psoriasis gutata is a clinical variant of psoriasis consisting of erythematous papules 1 to 5 mm in size with smooth scaling followed or preceded by an acute infection such as streptococcal pharyngitis.[17] The incidence of psoriasis in the world is about 2% of the world's population. In the United States and Canada, the prevalence reaches 4%.[18]

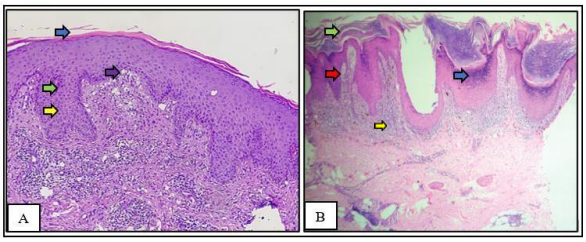

FIGURE 5. (A) IN THE EPIDERMAL LAYER, HYPERKERATOSIS (BLUE ARROW), ACANTHOSIS (GREEN ARROW), SPONGIOSIS (YELLOW ARROW), AND VACUOLIZATION OF BASAL CELLS (PURPLE ARROW) WERE OBSERVED (HE 10X) (B) HISTOPATHOLOGICAL EXAMINATION OF HYPERTROPHIC LICHEN PLANUS IN THE LITERATURE SHOWED CIVATTE BODIES (BLUE ARROW), SAWTOOTH RETE RIDGE (RED ARROW), HYPERKERATOSIS (GREEN ARROW), BAND-LIKE LICHENOID (YELLOW ARROW) (ADAPTED FROM JOSHI, 2019).

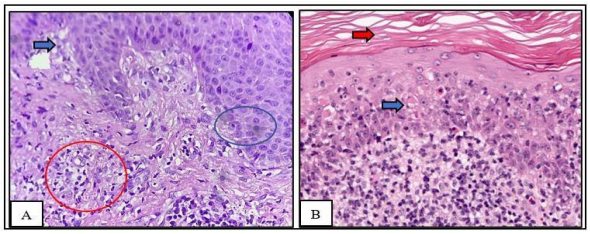

FIGURE 6. (A) IN THE EPIDERMAL LAYER, BASAL CELL VACUOLIZATION (BLUE ARROW), IN THE DERMIS LAYER, LYMPHOCYTIC INFILTRATION (RED CIRCLE), MELANIN DROPPING (BLUE CIRCLE) (HE 40X) (B) HISTOPATHOLOGICAL EXAMINATION OF LICHEN PLANUS IN THE LITERATURE SHOWS THE EPIDERMIS LAYER APPEARS HYPERKERATOSIS (ORTHOKERATOSIS) (RED ARROW), CIVATTE BODIES (BLUE ARROW) (HE 40X) (ADAPTED FROM JOSHI, 2019).

Onset can peak at 20-30 and 50-60 years.[19] Psoriasis gutata accounts for less than 30% of all psoriasis cases.[20] It occurs equally in both sexes. Cytotypes and epidermal molecules of the innate and adaptive immune systems are involved in the pathogenesis and progression of the disease. When triggers are present, keratinocytes can produce antimicrobial peptides such as cathelicidin (LL-37), defensins, and cytochrome 100 (S100) protein.[21] Keratinocytes also behave as secretive cells, as they can produce chemokine-20, further responsible for recruiting T-cells into the skin. Keratinocytes then express toll-like receptors (TLRs), which allow them to respond specifically to environmental microbial challenges. In humans, TLR1, TLR2, TLR4, and TLR6 are expressed on the cell surface, while TLR3, TLR7, TLR8, and TLR9 are expressed in cellular components. Toll-like receptors allow to sense molecular patterns associated with danger-associated molecular patterns (DAMPs) and pathogen-associated molecular patterns (PAMPs).[22] During terminal differentiation of keratinocytes, keratins (K) 5 and 14 are expressed in the proliferative epidermal compartment, while K1/K10 in the suprabasal differentiation layer.[23] Specific keratins have been recognized as regulators of many cell functions and as mediators of the maintenance of epidermal integrity after stress.[21,24] Keratins16 and K17 are indicated as "psoriatic keratins" in the differentiated

layer of the epidermis because they are associated with epidermal hyperproliferation.[18] This pathophysiology gives rise to various types of psoriasis.[25] The supporting examination to establish a diagnosis often used to establish various types of psoriasis is histopathological examination.[6,26] The histopathological picture of gutata psoriasis in the epidermal layer appears acanthotic, often with an elongated rete ridge (psoriasiform), there are alternating hypo- and hypergranulose zones in the epidermis, thinning of the suprapapillary plate, areas of parakeratosis in the stratum corneum with neutrophil mounds (Munro microabscesses), collections of neutrophils in the stratum spinosum (Kogoj spongiosiform) and often found keratinocytes that undergo apoptosis.[19,27] In the dermis layer, a lymphocytic infiltrate dominates in the upper and middle dermis and dilated and tortuous blood vessels in the dermis papillae.[28] The results of the histopathological examination in this patient with hematoeoxycillin staining of the dorsal skin biopsy in the epidermis layer showed hyperkeratosis, acanthosis, spongiosis, and vacuolization of basal cells, while in the dermis layer, there was a perivascular infiltrate dominated by lymphocytes with eosinophil cells obtained one cell and a little melanin dropping. This patient did not have alternating hypo- and hypergranulose zones in the epidermis, thinning of the suprapapillary plates, areas of parakeratosis in the stratum corneum with neutrophil mounds (Munro microabscesses), neutrophil collections in the stratum spinosum (Kogoj spongiosiform) so that the diagnosis of psoriasis gutata could be excluded.

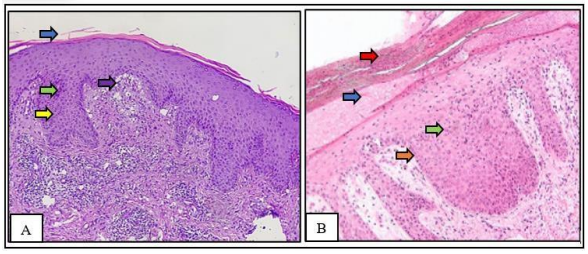

FIGURE 7. (A) HYPERKERATOSIS (BLUE ARROW), ACANTHOSIS (GREEN ARROW), SPONGIOSIS (YELLOW ARROW), AND VACUOLIZATION OF BASAL CELLS (PURPLE ARROW) (HE 10X) WERE OBSERVED IN THE EPIDERMAL LAYER (B) HISTOPATHOLOGICAL EXAMINATION OF PSORIASIS GUTATA FROM THE LITERATURE IN THE EPIDERMAL LAYER SHOWED HYPERKERATOSIS (RED ARROW), HYPOGRANULOSIS (BLUE ARROW), ACANTHOSIS (GREEN ARROW), RETE RIDGE ELONGATION (ORANGE ARROW) (HE 10X) (ADAPTED FROM GROLLEAU, 2024).

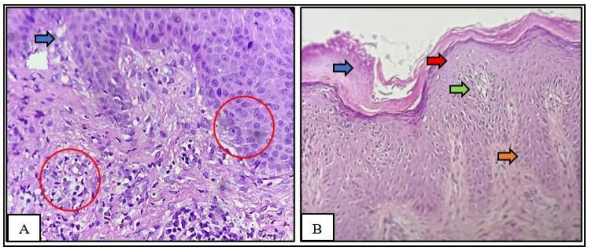

FIGURE 8. (A) IN THE EPIDERMAL LAYER, BASAL CELL VACUOLIZATION (BLUE ARROW), IN THE DERMIS LAYER, LYMPHOCYTIC INFILTRATION (RED CIRCLE), MELANIN DROPPING (BLUE CIRCLE) (HE 40X) (B) HISTOPATHOLOGICAL EXAMINATION OF PSORIASIS GUTATA FROM THE LITERATURE IN THE EPIDERMIS LAYER SHOWED HYPERKERATOSIS (BLUE ARROW), HYPOGRANULOSIS (RED ARROW), ACANTHOSIS (GREEN ARROW), RETE RIDGE ELONGATION (ORANGE ARROW) (HE 40X) (ADAPTED FROM ROUAI, 2021).

REFERENCES

[1]. Lupu J, Chosidow O, Wolkenstein P, Bergqvist C, Ortonne N, Ingen-Housz-Oro S. Pityriasis lichenoides: a clinical and pathological case series of 49 patients with an emphasis on follow-up. Clin Exp Dermatol. 2021;46(8):1561–6.

[2]. Elbendary A, Youssef R, Abdel-Halim MRE, Abdel Halim D, El Sharkawy DA, Alfishawy M, et al. Role of streptococcal infection in the etiopathogenesis of pityriasis lichenoides chronica and the therapeutic efficacy of azithromycin: a randomized controlled trial. Arch Dermatol Res. 2023;315(3):521–30.

[3]. Fernandes NF, Rozdeba PJ, Schwartz RA, Kihiczak G, Lambert WC. Pityriasis lichenoides et varioliformis acuta: a disease spectrum. Int J Dermatol. 2010;49(3):257–61.

[4]. Koh W, Koh MJ, Tay Y. Pityriasis lichenoides in an Asian population. Int J Dermatol. 2013;52(12):1495–9.

[5]. Agarwala S, Hafeez F. Pityriasis lichenoides-like drug reaction with numerous eosinophils. Int J Clin Exp Pathol. 2021;14(9):1010.

[6]. Balan R, Grigoraş A, Popovici D, Amălinei C. The histopathological landscape of the major

psoriasiform dermatoses. Archive of clinical cases. 2019;6(3):59.

[7]. Sechi A, Pierobon E, Pezzolo E, Germi L, Trevisan G, Zardo D, et al. Abrupt onset of Sweet syndrome, pityriasis rubra pilaris, pityriasis lichenoides et varioliformis acuta and erythema multiforme: unravelling a possible common trigger, the COVID-19 vaccine. Clin Exp Dermatol. 2022;47(2):437–40.

[8]. Filippi F, Patrizi A, Sabattini E, Varotti E, Bertuzzi C, Pileri A. Pityriasis lichenoides triggered by measles-mumps-rubella vaccine injection. JDDG: Journal der Deutschen Dermatologischen Gesellschaft. 2020;18(7):758–60.

[9]. Hrin ML, Bowers NL, Jorizzo JL, Feldman SR, Huang WW. Methotrexate for pityriasis lichenoides et varioliformis acuta (Mucha-Habermann disease) and pityriasis lichenoides chronica: A retrospective case series of 33 patients with an emphasis on outcomes. J Am Acad Dermatol. 2022;86(2):433–7.

[10]. Zhang J, Lei Z, Xu C, Zhao J, Kang X. Current perspectives on severe drug eruption. Clin Rev Allergy Immunol. 2021;61(3):282–98.

[11]. Clarey DD, Lauer SR, Trowbridge RM. Clinical, dermatoscopic, and histological findings in a diagnosis of pityriasis lichenoides. Cureus. 2020;12(6).

[12]. Gru AA, Salavaggione AL. Lichenoid and interface dermatoses. In: Seminars in Diagnostic Pathology. Elsevier; 2017. p. 237–49.

[13]. Daye M, Işık B, Kılınç F. Lichen planus due to hirudotherapy. Turkiye Parazitol Derg. 2021;45(2):149–52.

[14]. Kusari A, Ahluwalia J. Lichen Planus. N Engl J Med. 2018 Aug;379(6):567.

[15]. Cheng HM, Chuah SY, Gan EY, Jhingan A, Thng STG. A retrospective clinico-pathological study comparing lichen planus pigmentosus with ashy dermatosis. Australasian Journal of Dermatology. 2018;59(4):322–7.

[16]. Romiti R, Biancardi Gavioli CF, Anzai A, Munck A, Costa Fechine CO, Valente NYS. Clinical and histopathological findings of frontal fibrosing alopecia-associated lichen planus pigmentosus. Skin Appendage Disord. 2017;3(2):59–63.

[17]. Dupire G, Droitcourt C, Hughes C, Le Cleach L. Antistreptococcal interventions for guttate and chronic plaque psoriasis. Cochrane Database of Systematic Reviews. 2019;(3).

[18]. Garritsen FM, Kraag DE, De Graaf M. Guttate psoriasis triggered by perianal streptococcal infection. Clin Exp Dermatol. 2017;42(5):536–8.

[19]. Grolleau C, Bettuzzi T, Wang L, Wolkenstein P, Sbidian E, Ortonne N. Guttate psoriasis shares histological features with pustular psoriasis and often shows neutrophil exocytosis and pustules. Pathology. 2024;56(4):606–8.

[20]. Jindal R, Chauhan P, Sethi S. Dermoscopic characterization of guttate psoriasis, pityriasis rosea, and pityriasis lichenoides chronica in dark skin phototypes: an observational study. Dermatol Ther. 2021;34(1):e14631.

[21]. Armstrong AW, Read C. Pathophysiology, clinical presentation, and treatment of psoriasis: a review. JAMA. 2020;323(19):1945–60.

[22]. Mihu C, Neag MA, Bocşan IC, Melincovici CS, Vesa ȘC, Ionescu C, et al. Novel concepts in psoriasis: histopathology and markers related to modern treatment approaches. Romanian Journal of Morphology and Embryology. 2021;62(4):897.

[23]. Deng Y, Chang C, Lu Q. The inflammatory response in psoriasis: a comprehensive review. Clin Rev Allergy Immunol. 2016;50:377–89.

[24]. Huang YW, Tsai TF. HLA-Cw1 and psoriasis. Am J Clin Dermatol. 2021;22(3):339–47.

[25]. Kamiya K, Kishimoto M, Sugai J, Komine M, Ohtsuki M. Risk factors for the development of psoriasis. Int J Mol Sci. 2019;20(18):4347.

[26]. Nurfaiqoh E, Evanti AM, Primisawitri PP, Irawanto ME. Relationship between severity of psoriasis vulgaris based on Psoriasis Area And Severity Index (PASI) scores and depression. Journal of Pakistan Association of Dermatologists. 2023;33(1):101–7.

[27]. Fitriani F, Dharmawan N, Hakim FA, Wasita B, Widhiati S. The difference of histopathological profile of early onset and late onset psoriasis in tertiary hospital in Central Java, Indonesia. Journal of Pakistan Association of Dermatologists. 2023;33(2):587–92.

[28]. Niculet E, Radaschin DS, Nastase F, Draganescu M, Baroiu L, Miulescu M, et al. Influence of phytochemicals in induced psoriasis. Exp Ther Med. 2020;20(4):3421–4.