International Medical Case Reports Journal

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CASE REPORT

Lichen Planus Pigmentosus as a Cutaneous Extrahepatic Manifestation of Chronic Hepatitis C Virus Infection: A Case Report

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Abstract: Lichen planus pigmentosus is an uncommon dermatological condition characterized by hyperpigmented and dark-brown macules. It is a rare variant of lichen planus, one of the most common cutaneous extrahepatic manifestations of chronic hepatitis C virus infection. The cutaneous extrahepatic manifestations may indicate an undetected chronic hepatitis C virus infection, showing the need for thorough evaluation in patients presenting with these dermatological conditions. This case report presents a case of lichen planus pigmentosus in a 60-year-old male, initially complained of an itchy dark brown macule on his right temple, which progressively enlarged, covered almost the entire face, and became thicker in several areas. Then, he was diagnosed with chronic hepatitis C virus infection and liver cirrhosis. Dermoscopy examination showed gray-brown dots, globules, and peri-eccrine gray-brown pigmented dots. Laboratory tests revealed low ferritin levels, a positive antinuclear antibody test, but a negative antinuclear antibody profile. The histopathological examination showed basal vacuolar changes on the epidermis and interface dermatitis extending to the area of the hair follicle infundibulum, with melanin pigment deposits between the fibro-collagen connective tissue accompanied by inflammatory and peri-adnexa lymphocytes, and fibrosis around the hair follicles in the dermis, supporting the diagnosis of lichen planus pigmentosus. The patient was treated with sun avoidance and photoprotection, mometasone furoate 0.1% cream twice a day, and direct-acting antiviral agents. Improvement was observed clinically and supported by spectrophotometry examination. This case highlights lichen planus pigmentosus as a potential indicator of previously undetected hepatitis C virus infection, recommending hepatitis C virus testing for patients with lichen planus pigmentosus.

Keywords: diffuse hyperpigmentation, liver disease, viral infection

Introduction

Hepatitis C virus (HCV) infection affects approximately 50 million people worldwide, causing not only liver disease but also health issues beyond the liver, known as extrahepatic manifestations (EHMs).[1,2] One of the EHMs is cutaneous EHMs (cEMHs). Although HCV infection is often asymptomatic, cEHMs may serve as a primary indicator in certain patients with HCV infection, potentially revealing previously undetected HCV infections.[3,4] Approximately 74% of HCV patients will experience HCV-related EHMs in their lifetime, with about 17% of the patients experiencing at least one cEHM.[2,4] The most common cEHMs are mixed cryoglobulinemia, lichen planus (LP), and porphyria cutanea tarda.[3,4] One rare variant of LP is lichen planus pigmentosus (LPP) characterized by hyperpigmented, dark-brown macules in sun-exposed and flexural folds.[5]

Several associations between HCV infection and LP have been reported.[6,7] Lichen planus has been observed in 7.05% of patients infected with HCV,[8] while the prevalence of HCV infection in patients with LP ranged up to 62%.[9] There are only a few case reports of LPP as the cEHMs. A study by Al-Mutairi et al[7] also reported 60.6% of 33 LPP patients had positive serology for HCV infection. Therefore, a dermatologist must be able to perform a comprehensive assessment of

Received: 10 January 2025
Accepted: 16 April 2025
Published: 17 May 2025

individuals presenting with these dermatological disorders as the initial symptoms. An HCV test is advisable for patients with LP or other cEHMs due to the significant epidemiological and pathogenic association.[3] This case report aims to report a case of LPP as the cEHM of chronic HCV infection.

Case Report

A 60-year-old male was consulted from the Internal Medicine Department with a chief complaint of diffuse itchy dark brown macules over the face and ears for two years. The patient initially complained of itchiness on his right temple, which developed into a dark brown macule. One year later, the hyperpigmentation darkened and spread to the forehead and entire face. Four months before consultation, the hyperpigmentation darkened and thickened on the forehead and both temples. The itchiness persisted intermittently. Eighteen months and twelve months prior to consult, the patient complained of hematemesis and was hospitalized, but had never been tested for anti-HCV due to the limited facilities at the previous hospital. Two days before the consult, the patient experienced a recurrence of hematemesis, became apatic, and was hospitalized in the high-care unit. The patient was exposed to sunlight for approximately two hours a day without any sun protection. There is no family history of similar skin disorders. The patient had family history of hepatitis, the patient's father had a history of jaundice and was diagnosed with hepatitis. The history of HCV infection in the patient's mother and wife is unknown.

On physical examination, the patient had skin type IV according to the Fitzpatrick classification, with diffuse hyperpigmented macules and plaques without any Wickham's striae on the face and ears, sparing the tip of the nose and the ala of the nose (Figure 1). Wood's lamp examination showed no accentuation. Dermoscopy examination revealed reticular gray-brown dots and globules, as well as peri-eccrine gray-brown pigmented dots (Figure 2). The histopathological examination showed basal vacuolar changes in the epidermis and interface dermatitis extending to the area of the hair follicle infundibulum, with numerous melanin pigment deposits between the fibro-collagen connective tissue accompanied by several inflammatory lymphocytes, peri-adnexa lymphocytes, and fibrosis around the hair follicles in the dermis, confirming the diagnosis of LPP (Figure 3). Laboratory tests showed reactive anti-HCV, low ferritin levels, elevated serum glutamic oxaloacetic transaminase (SGOT) levels, normal alpha-fetoprotein levels, as well as non-reactive anti-human immunodeficiency virus (anti-HIV) and hepatitis B surface antigen (HBsAg). The antinuclear antibody (ANA) test was positive with a titer of 1:100 and a fine-speckled pattern. However, the ANA profile test showed negative results. The abdominal ultrasonography examination indicated liver cirrhosis with suspected portal hypertension. The upper gastrointestinal tract endoscopic examination showed grade 2 esophageal varices and moderate portal hypertensive gastropathy. Based on these findings, the patient was diagnosed with LPP associated with chronic

Figure 1 Clinical manifestation showed hyperpigmentation macules and plaques on the face and ears, from front view (**A**), right side (**B**), and left side (**C**).

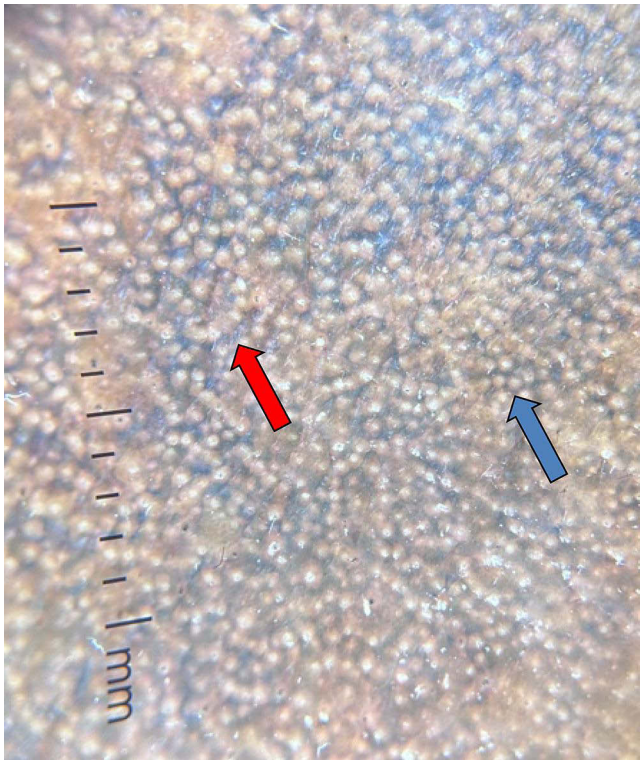

Figure 2 Dermoscopy examination revealed reticular gray-brown dots and globules (blue arrow) and peri-eccrine gray-brown pigmented dots (red arrow).

HCV infection, upper gastrointestinal tract bleeding due to ruptured esophageal varices, grade II encephalopathy, and suspected liver cirrhosis with Child-Pugh C due to HCV infection.

The patient used sunscreen with a sun protection factor (SPF) 45 for protection against ultraviolet (UV) B and protection grade of UVA (PA) ++ every morning and reapplied every two hours. The patient was also treated with a combination of mometasone furoate 0.1% cream twice daily, propranolol 10 mg twice a day, lactulose syrup 15 cc once a day, and direct-acting antiviral agents (DAA) for chronic HCV infection with liver cirrhosis that consists of a combination of daclatasvir 60 mg and sofosbuvir 400 mg once a day for 24 weeks. The improvements were observed clinically supported by spectrophotometer examination.

Discussion

LPP and LP have similar immunopathogenesis, potentially triggered by various factors such as viral infections, exposure to UV light, or the use of certain oils on the hair or skin.[10] LP is considered a T-cell mediated autoimmune response to exogenous or self-altered antigens presented by antigen-presenting cells (APC) such as dendritic cells or keratinocytes, and attacked by the cytotoxic cluster of differentiation (CD)8+ T cells.[11] Several studies have utilized polymerase chain reaction (PCR)-based methods to demonstrate the presence of HCV ribonucleic acid (RNA) in mucosal and cutaneous lesions, indicating compartmentalization of HCV within tissues.[12] In this process, CD8+ T lymphocytes identify and target epidermal keratinocytes, leading to intense pigmentary incontinence. The lichenoid reaction occurs within a short period with a significant hydropic degeneration of basal keratinocytes. However, there is a lack of compensatory increased proliferation of keratinocytes during this phase. Consequently, livid papules swiftly transition into brown macules, predominantly characterizing the clinical presentation of the disease.[13]

The typical progression of LPP begins with manifestations on the face and neck. Initially, the lesions manifest as small, brown, oval-shaped macules with diffuse borders. As the condition progresses, the macules coalesce to form large hyperpigmented patches.[6,14] The most frequent pigmentation was diffuse (56%), reticular (16%), and blotchy (12%).[9] Wickham's striae are usually absent.[14] LPP is usually asymptomatic, but some lesions may be associated with itching or

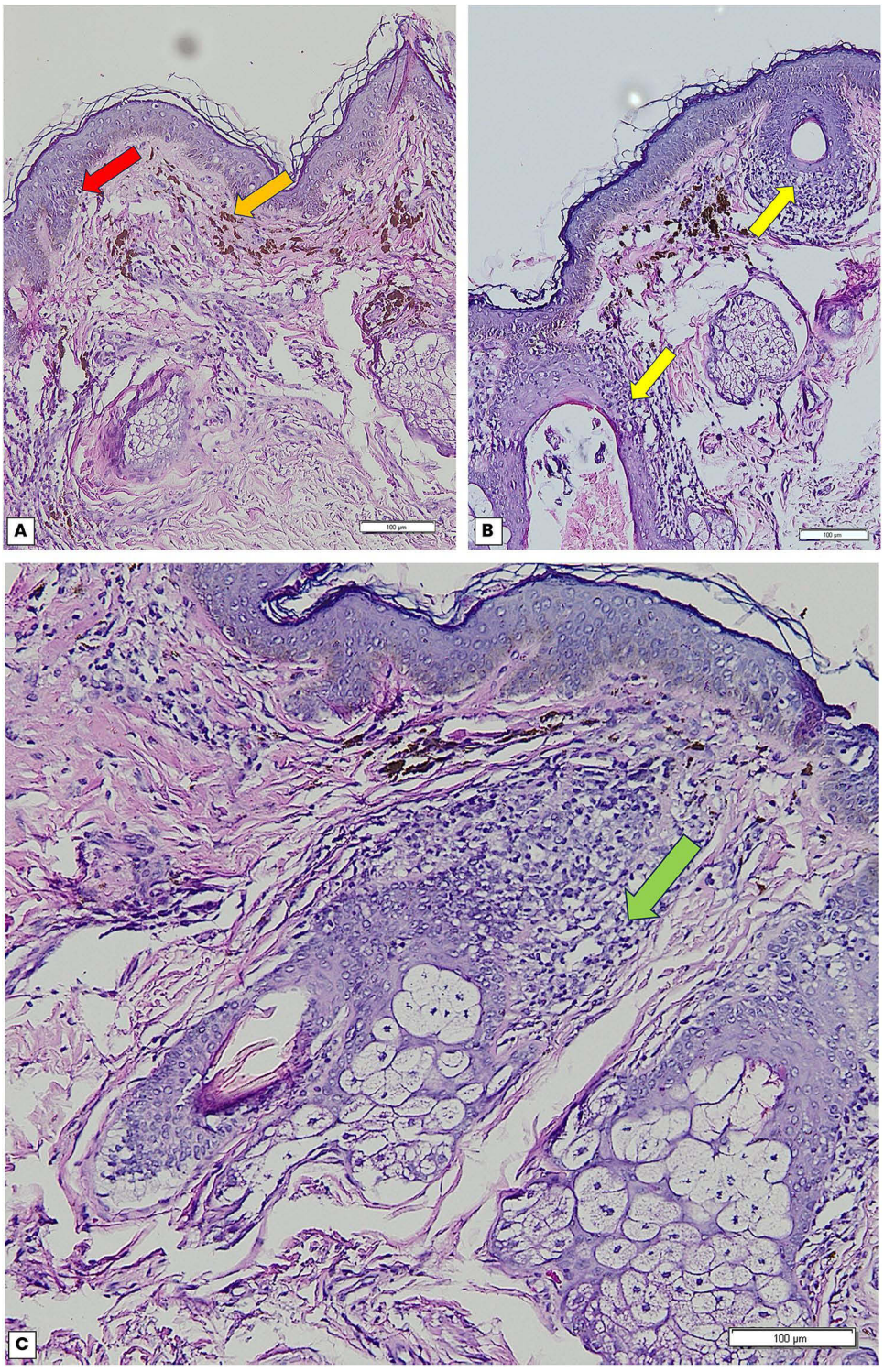

Figure 3 Histological findings from the right cheek showed epidermis with basal vacuolar change (red arrow) and melanin deposits (Orange arrow) (**A**), interface dermatitis (yellow arrow) (**B**), and lymphocyte infiltration on peri adnexa (green arrow) (**C**).

burning sensation.[6] Some consider itching to be an indicator of activity and advancement of the disease.[15] The lesion of this patient began as hyperpigmented macules on his right temple that became diffuse hyperpigmented patches on his entire face with several diffuse hyperpigmented plaques without any Wickham's striae. The patient also complained of itching before the skin lesion appeared and the itching still arises and disappears erratically.

Dermoscopic examination of LPP lesions revealed pigmentation in various nonspecific patterns, including black, brown, or violet dots, globules, and blotches as predominant pigment structures. Other findings include peri-eccrine and perifollicular gray-brown pigmented dots and globules.[16] In this case report, the dermoscopy examination revealed reticulate blue-black dots and peri-eccrine gray-brown pigmented dots and globules.

The histopathological examination of LPP showed vacuolar degeneration of the epidermal basal cell layer, band-like lichenoid or perivascular lymphocytic infiltrates (interface dermatitis) in the papillary dermis, as well as superficial pigmentary incontinence and melanophages. Other less common findings are hyperkeratosis and epidermal atrophy. In older lesions, the histopathological findings showed marked pigmentary incontinence, melanophages, a decrease in vacuolar degeneration, and slight perivascular lymphocytic infiltrate.[7,15,17] In this case report, the histopathological examination from the right cheek revealed that in the epidermis, there were basal vacuolar changes and interface dermatitis extending to the area of hair follicle infundibulum. In the dermis, there were numerous melanin pigment deposits between the fibro-collagen connective tissue accompanied by several inflammatory lymphocytes, peri adnexa lymphocytes, and fibrosis around the hair follicles. These findings supported the diagnosis of LPP.

The ANA test can be positive in chronic HCV infection patients with a prevalence of 20.2%.[18] Two of 100 LPP patients also had positive ANA without any signs or symptoms indicative of autoimmune disease.[7] The patient in this case report had a positive ANA test at titers 1:100 in a fine speckled pattern with a negative ANA profile as well as no signs or symptoms indicative of autoimmune disease. Therefore, the positive ANA test in the patient was considered as a result of the chronic HCV infection or the LPP.

According to the European Dermatology Forum Guidelines on the management of LP,[19] the management of cutaneous LP aims to reduce itching and shorten the duration between the onset of the disease and the resolution of the lesions. The first-line treatments are topical steroid or triamcinolone intralesional injection, systemic corticosteroid, acitretin, and oral cyclosporin with additional oral antihistamines or topical antipruritic agents. However, there were no available treatment options that demonstrated consistent responses or distinct superiority over other methods for LPP—the evidence regarding the treatment efficacy was limited to a few case series.[20] Since LPP has been linked to sun exposure, the patient should avoid sun exposure and use photoprotection.[12] Ohshima et al[21] reported that the use of mometasone furoate 0.1% ointment twice a day for 9 months resulted in gradual improvement of the lesions. In this case report, the patient did sun avoidance and used sunscreen with an SPF 45 and PA ++ every morning and reapplied every two hours. The patient was also treated with mometasone furoate 0.1% cream twice a day as well as DAA that consist of a combination daclatasvir 60 mg and sofosbuvir 400 mg once a day for 24 weeks. The improvement of the hyperpigmentation in the patient showed one week after the treatment observed with spectrophotometry examination. This tool objectively assesses the degree of brightness (L*), erythema (a*), and tanning (b*). The pigmented skin was shown to present with lower L* values than less pigmented skin. Based on the spectrophotometry examination, the patient's skin became brighter with an increased L* value in all areas.

The patient in this case report is an Asian population with hyperpigmentation macules on his right temple four months before experiencing hematemesis and 21 months before being diagnosed with liver cirrhosis due to HCV infection. The dermoscopic and histopathological examination of the skin lesion also confirmed the diagnosis of LPP. These findings support that LPP may be an early indicator of previously undetected HCV infection.

Conclusion

This case report demonstrates a case of LPP associated with chronic hepatitis C virus infection as the cutaneous extrahepatic manifestation. In conclusion, an HCV test is advisable for patients with LP or other cEHMs as it may act as an indicator to uncover previous asymptomatic HCV infections.

Abbreviations

ANA, antinuclear antibody; CD, cluster of differentiation; cEHM, cutaneous extrahepatic manifestation; DAA, direct-acting antiviral agents; EHM, extrahepatic manifestation; HBsAg, hepatitis B surface antigen; HCV, hepatitis C virus; LP,

lichen planus; LPP, lichen planus pigmentosus; PCR, polymerase chain reaction; RNA, ribonucleic acid; SGOT, serum glutamic oxaloacetic transaminase; SPF, sun protection factor; UV, ultraviolet.

Ethics Statement

The publication of images was included in the patient's consent for the publication of the case. Institutional approval was obtained to publish the case details from Dr. Hasan Sadikin Hospital Ethical Committee with ethical approval number DP.04.03/D.XIV.6.5/373/2024.

Consent Statement

The authors certify that they have obtained all appropriate patient consent forms. The patient signed a consent form for the publication of the case details and images.

Acknowledgments

The authors would like to thank the staff of the Dermatology and Venereology Department and the Department of Anatomical Pathology, Faculty of Medicine, Universitas Padjadjaran – Dr. Hasan Sadikin General Hospital.

Disclosure

The authors report no conflicts of interest in this work.

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