Magna Scientia Advanced Research and Reviews

eISSN: 2582-9394

Cross Ref DOI: 10.30574/msarr

Journal homepage: https://magnascientiapub.com/journals/msarr/

(CASE REPORT)

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Two Foes, One Host: The Poignant Coexistence of Hansen's Disease and Hepatitis B

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Magna Scientia Advanced Research and Reviews, 2025, 15(01), 157-164

Publication history: Received on 30 August 2025; revised on 04 October 2025; accepted on 07 October 2025

Article DOI: https://doi.org/10.30574/msarr.2025.15.1.0122

Abstract

Leprosy (Hansen's disease) is a chronic infectious condition caused by Mycobacterium leprae, characterized by skin lesions and peripheral nerve involvement, often leading to functional and aesthetic complications. Chronic hepatitis B, caused by hepatitis B virus (HBV), is a hepatotropic infection that can range from asymptomatic carrier states to severe liver disease including cirrhosis and hepatocellular carcinoma. We report a rare case of a 29-year-old male from Uttar Pradesh presenting with a solitary hypopigmented patch over the right knee accompanied by hypoaesthesia, clinically and histopathologically confirmed as borderline tuberculoid leprosy. Incidentally, the patient was also diagnosed with chronic hepatitis B infection during initial evaluation. The patient has been on multidrug therapy for leprosy per WHO protocols while being monitored for HBV without antiviral initiation due to absence of active viral replication or significant liver damage. This dual coexistence underscores the complexities of managing patients with simultaneous infections that modulate immune function differently: leprosy impairing cell-mediated immunity and chronic HBV causing defective adaptive immune responses. Awareness of such co-infections is vital to tailor therapy and avoid drug-induced hepatic toxicity. Hepatitis B screening before starting leprosy treatment is emphasized to optimize management. This report adds to limited data on concurrent Hansen's disease and chronic hepatitis B, highlighting the need for further research into their immunopathological interactions and comprehensive clinical approach.

Keywords: Hansen's disease; Borderline tuberculoid leprosy; Chronic hepatitis B; Multidrug therapy; Immune dysregulation; Peripheral neuropathy

1. Introduction

Leprosy is a chronic infectious disease caused by Mycobacterium leprae. It is one of the oldest known infections in humans, becoming rare in modern times thanks to the efforts of the World Health Organization (WHO), but remaining mostly endemic in some regions of the world. The WHO's recent initiative 'Towards zero leprosy' is a forceful venture toward eliminating the condition definitely on a global scale by 2030 [1]. Though curable, leprosy remains an important cause of functional and aesthetic complications. Nevertheless, there is a gap in recognizing the condition which is rightly termed a 'great mimicker'.

M. leprae, the causative organism – the alcohol and acid resistant obligatory intracellular bacillus, was discovered by Gerhard-Henrik Armauer Hansen in 1873 in Norway, hence Hansen's disease. In 2008, a group of scientists discovered a second species Mycobacterium lepromatosis responsible for a similar clinical phenotype as M. leprae [2]. Both species are termed together M. leprae complex. The mode of transmission is through contact with infected skin and mucosa. Factors influencing transmission include host infectivity, proximity, duration and frequency of contact. M. leprae has a long incubation period ranging from two to five years with even longer durations reported [3].

Chronic hepatitis B is caused by the hepatitis B virus (HBV), a hepatotropic DNA virus that can replicate at high levels and cause minimal disease or severe liver injury. The clinical spectrum of chronic hepatitis B ranges from no symptoms

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to progressive hepatic fibrosis, advanced cirrhosis, and hepatocellular carcinoma. An estimated 296 million people have chronic hepatitis B, of whom 221 million live in low- and middle-income countries. Without intervention, deaths from HBV are expected to peak at 1.14 million by 2035 [4].

HBV generally causes a self-limiting illness in adults, with viral control mediated primarily by the adaptive immune response. Persistent viral replication cannot be contained by the depleted and defective HBV specific CD4 and CD8 T-cell response that results from the combination of high antigen-dose exhaustion and liver-tolerizing pathways, including inhibitory checkpoints (e.g., programmed death 1 [PD-1]), regulatory cells, and metabolic dysfunction [5].

Here, we discuss a 29-year-old male, who presented with a solitary hypo to normopigmented patch over right knee, associated with hypoaesthesia. Confirmed with biopsy to be a case Borderline Tuberculoid Leprosy. Evaluation during the same time revealed the presence of Hepatitis B infection in the patient. Patient has been on follow up for last two years.

2. Case report

A 29-year-old male, married, resident of Uttar Pradesh, right-handed, initially presented with complaints of light-coloured numb patch over right knee for 08 months. Patient was apparently asymptomatic till 08 months back, when he noticed a single coin sized light coloured patch over right knee, insidious in onset, gradually progressed in size over next 08 months. He did not complain of any light-coloured numb patches elsewhere over the body, fever, arthralgia, myalgia, multiple crops of red raised painful lesions, deep sharp shooting pain down the limbs, any sensory loss elsewhere over the body, any features suggestive of motor weakness, redness of eyes, epistaxis, hoarseness of voice, gynaecomastia, pain abdomen, testicular pain or atrophy. He was evaluated clinically, bacteriologically and histopathologically and confirmed as a case of Hansen's disease (Borderline Tuberculoid); and started on 1st line Multibacillary Multidrug Therapy. During hospital say, he was incidentally found to be HBsAg positive. He was evaluated further.

Dermatological Examination at initial presentation: Solitary, ill defined, hypo to normopigmented patch approx. 5 cm x 4 cm present over medial aspect of right knee, associated with hypoaesthesia to fine touch, temperature and pain and overlying hypotrichosis. No nerve to patch/ satellite lesion present. Peripheral Nerve Examination: Left ulnar nerve Gd I thick and non-tender. Musculoskeletal Examination: Power in all muscle groups Grade 5/5.

Dermatological Examination six months into treatment (Figure 1): Ill-defined area of size approx. 5 cm x 4 cm present over medial aspect right knee, associated with hypoaesthesia to fine touch, temperature and pain with overlying hypotrichosis. No nerve to patch/ satellite lesion present. 02 healed hyperpigmented biopsy scars seen over the hypoaesthetic area.

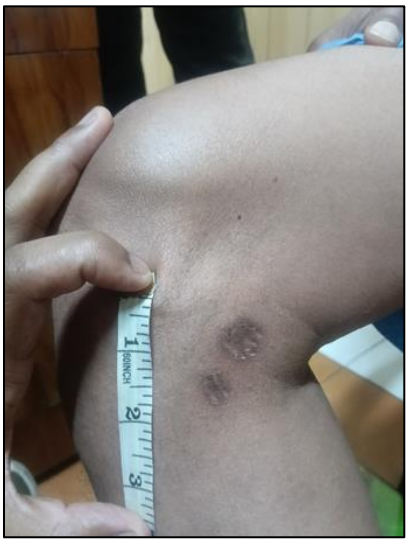
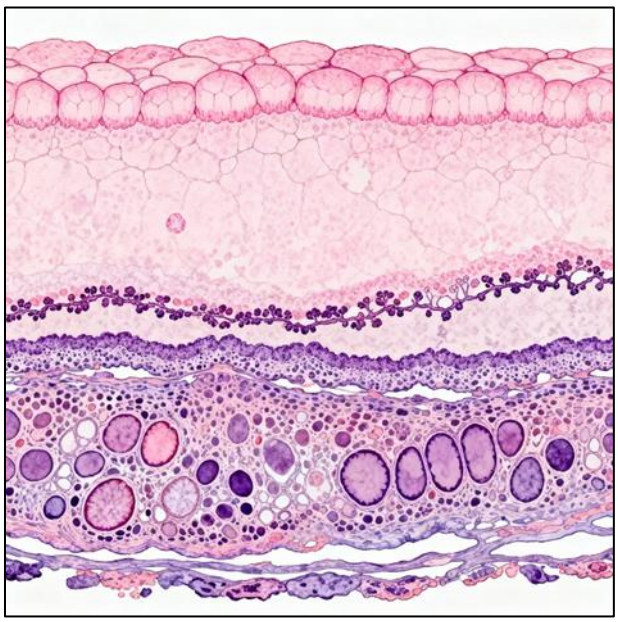


Figure 1 Ill-defined hypoesthetic, hypopigmented patch approx. 5x4 cm over right knee with decreased sensation to touch, temperature, and pain, associated with overlying hair loss and two healed biopsy scars, no nerve involvement nearby

Peripheral Nerve Examination: Left ulnar nerve Gd-I thick and non-tender. Musculoskeletal Examination: Power in all muscle groups Grade 5/5.

Investigation Reports: Hb: 17.9 g/dl, TLC: 5070/Cumm, DLC: N-50.9%, L-6.4%, E- 2.2%, Platelet: 1,20,000/Cumm, BUN: 19 mg/dl, S. Creat: 0.67 mg, Bilirubin- 0.3 mg/dl, AST: 39U/L, ALT- 58U/L, S. Cholesterol- 171mg/dl, Urine RE/ME, Blood Sugar: F/PP- 151/220 mg/dl, HIV – NR, HbsAg –positive, HbeAg- Negative. HBV DNA – Target Not Detected. Anti HBc positive.

Skin Biopsy at initial presentation (Graphical representation in Figure 2)**:** H&E stained section of skin biopsy shows a normal epidermis. The dermis shows a mild perivascular lymphocytic infiltrate. No periadnexal inflammatory infiltrate, granuloma or necrosis noted.



ZN stain- No acid fast bacilli seen. Impression: With these histological features, Indeterminate Leprosy cannot be excluded.

Figure 2 Representation of Skin Biopsy showing H&E-stained section of the skin biopsy revealing a normal epidermis with a mild perivascular lymphocytic infiltrate in the dermis, and no presence of periadnexal inflammation, granuloma, or necrosis

2.1. Clinical Diagnosis: Hansen's Disease (Borderline Tuberculoid)

Treatment: The patient has been on a consistent regimen of multidrug therapy over the past six months, which includes Cap Rifampicin 600 mg once monthly, Cap Clofazimine 300 mg monthly along with 50 mg daily, and Tab Dapsone 100 mg at bedtime. This combination therapy is in line with the World Health Organization's recommended treatment protocol for Hansen's disease, aimed at effectively controlling the infection and preventing disease progression while being monitored closely for potential side effects associated with these medications. Since patient does not fulfil criteria for initiation on antivirals for Hepatitis B infection, he has not been initiated on Tab Tenofovir/ Tab Entecavir and is on regular follow up.

3. Discussion

This case is notable due to the rare coexistence of Hansen's disease and chronic hepatitis B infection, conditions that are individually common but seldom reported together. The dual infection highlights complex interactions between immune system dysfunction caused by both diseases. Leprosy impairs cell-mediated immunity, while chronic hepatitis B persists due to impaired adaptive immune responses, making this combination important for understanding immune dysregulation. Managing these coexisting infections requires careful consideration of drug effects, especially hepatotoxicity, and emphasizes the need for hepatitis B screening before starting leprosy treatment.

The World Health Organization has set a goal to eliminate HBV as a public health threat by 2030. No country is currently on track to achieve this target. Major obstacles impeding elimination efforts include pervasive stigma, which can cause

feelings of shame, denial, self-isolation, and depression. As a result, people with chronic HBV may avoid testing and treatment and may conceal their infection. Additional challenges are limited access to care and overly complex, restrictive clinical practice guidelines, all of which hinder effective diagnosis, management, and prevention of HBV infection worldwide.

On the other hand, Leprosy is ubiquitous in tropical countries, particularly underdeveloped and developing countries. Despite control efforts, including the widespread use of MDT and stabilization of reported new case detection rates in the last few years, leprosy remains endemic in many developing countries. Skin lesions are usually the first clinical manifestation observed. If appropriate medical treatment is not received, leprosy may progress to cause permanent damage to the skin, nerves, limbs, and other organs [6]. Table 1 shows Comparison of classifications of leprosy proposed by World Health Organization and Ridley–Jopling.

Table 1 Comparison of classifications of leprosy proposed by World Health Organization and Ridley–Jopling

Classification		**Brief description**		
WHO (1987)	Ridley–Jopling [7]	Number of skin lesions	Neurological damage	Bacteriology: microscopic criteria
Paucibacillary				
1 skin lesion	Tuberculoid (TT)	Unique and infiltrated lesions	No neurological damage	Negative
2–5 skin lesions	Borderline tuberculoid (BT)	Stasis and hypopigmented lesions few or many lesions of varying size	Little or no neurological damage	Negative/ 1+
Multibacillary				
>5 lesions	Borderline borderline (BB)	Multiple lesions and maculopapular	Late thickening of the nerve	≧2+
	Lepromatous (BL)	Multiple lesions, maculopapular, and nodules	Late thickening of the nerve	≧2+
	Lepromatous (LL)	Multiple lesions, maculopapular, and nodules	Late thickening of the nerve	≧2+

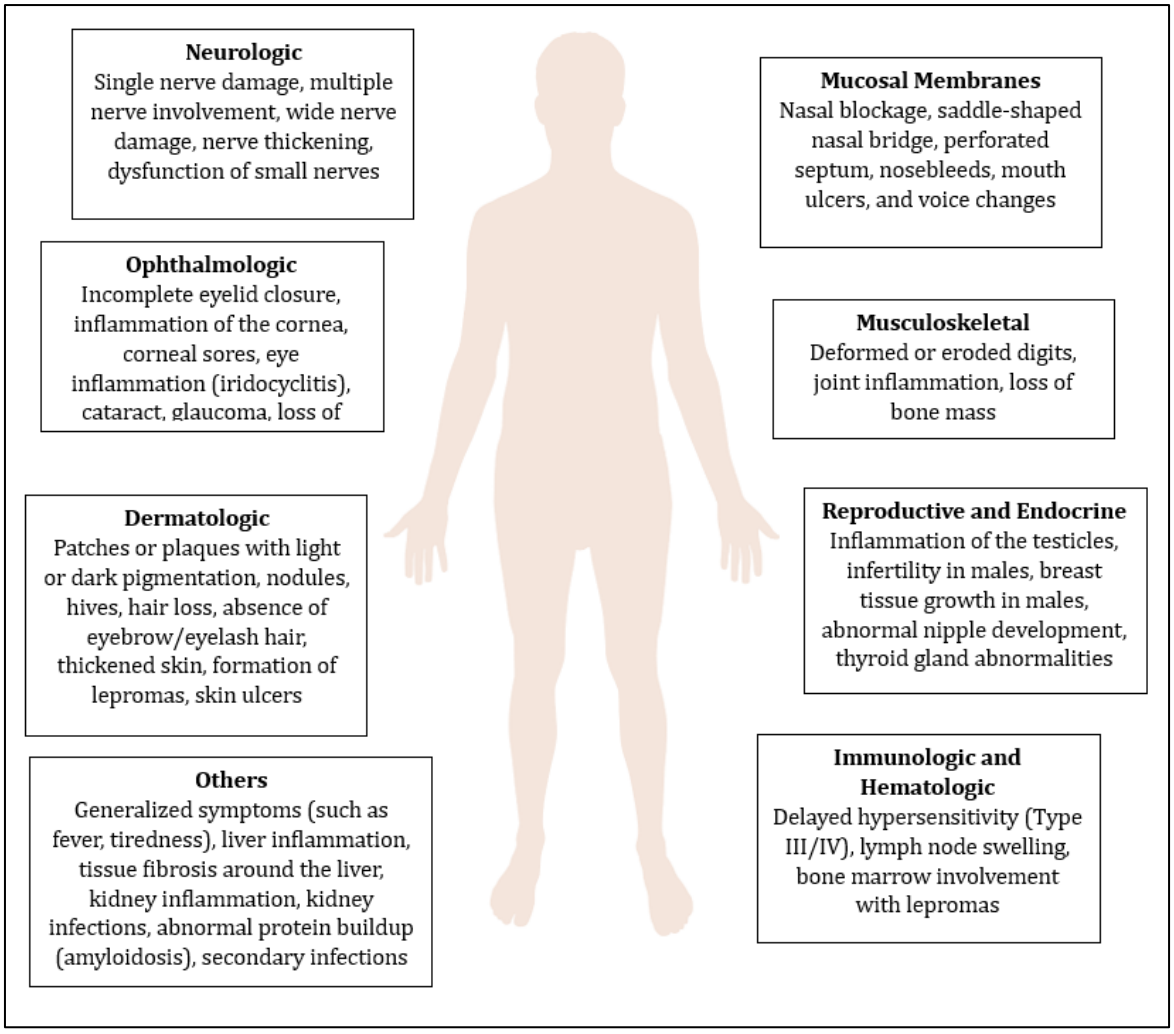


Figure 3 Clinical Manifestations of Leprosy

Diagnosis of Leprosy: Diagnosis is established by linking characteristic skin findings, especially areas of reduced pigmentation, with evidence of peripheral nerve involvement. Confirmation is achieved through skin slit smears or biopsy of skin or nerve tissue. Nerve conduction studies are essential to detect peripheral nerve dysfunction and help determine the location and distribution of nerve pathology [8]. These studies also inform the selection of a site for nerve biopsy. Advanced imaging techniques such as nerve ultrasound and MRI can assist in evaluating nerve involvement, but their regular use is restricted by access and the specialized skills required for these procedures.

Initial assessment of HBV infection begins with patient history, physical examination, evaluation of liver disease activity, and interpretation of different hepatitis markers and/or their combinations such as HBsAg, HB core antigen (HBcAg), HBeAg, HB surface antibody (anti-HBs/HBsAb), HB core antibody (anti-HBc), antiHBc IgM, HB e antibody (anti-HBe), and focus on the detection of antigens and antibodies (WHO 2017b). The Hepatitis B Foundation (HBF) recommends screening all adults for HB with the triple serological marker panel that involves HBsAg, anti-HBs, and anti-HBc total (HBF 2018b). To classify the phases of the infection in HBV infected patients, the followings should be performed: i) the assays for HBsAg, HBeAg/anti-HBe, HBV DNA; ii) liver blood tests including aspartate aminotransferase (AST), alanine transaminase (ALT), and iii) transient elastography (Fibroscan) as a noninvasive test or needle liver biopsy as an invasive method for the presence of cirrhosis (EASL 2017)

Figure 4 represents graphically, when to treat Hepatitis B patients with antivirals, adapted from National Viral Hepatitis Control Programme (NVHCP), Ministry of Health and Family Welfare, Government of India. Technical guidelines for diagnosis and management of hepatitis B [9]. Our patient, despite being hepatitis B surface antigen (HBsAg) positive, lacked significant fibrosis, cirrhosis, or persistently elevated alanine aminotransferase (ALT) levels, and the HBV DNA viral load was below thresholds that recommend treatment. Therefore, antiviral drugs were not initiated in this case, consistent with current treatment guidelines.

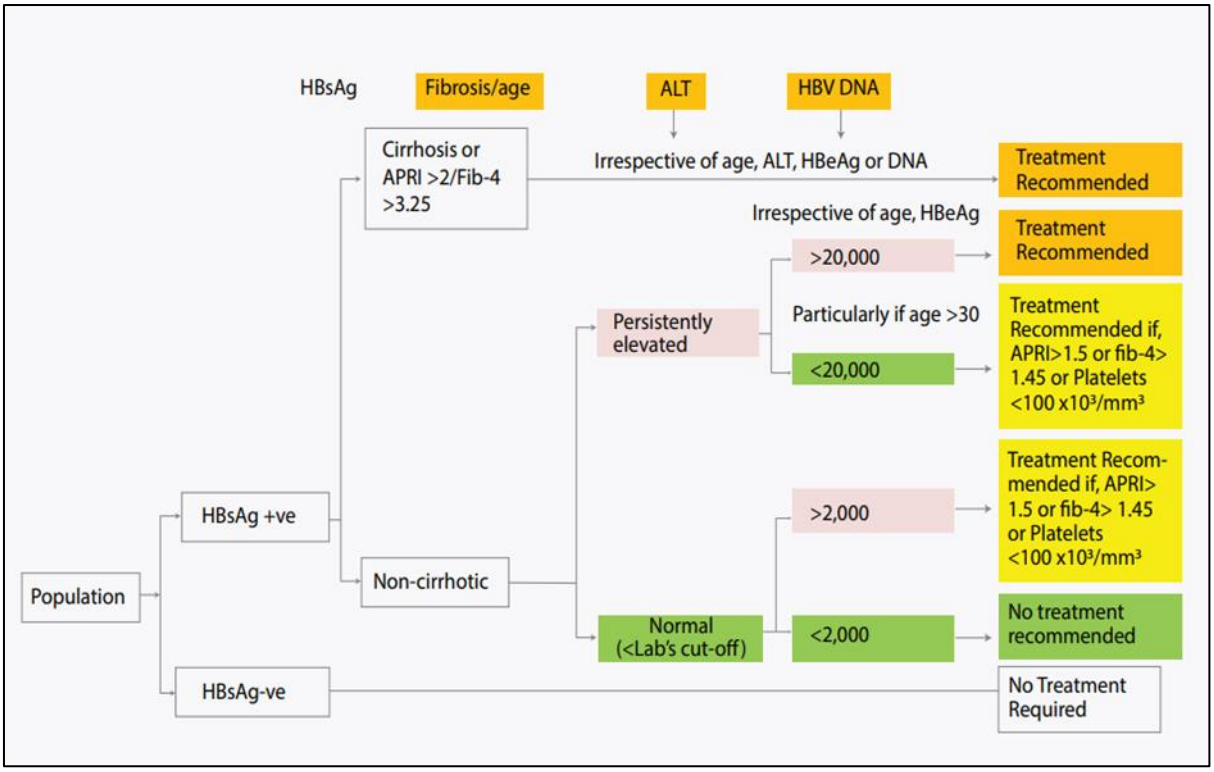


Figure 4 When to treat Hep B patients; HBsAg: Hepatitis B surface antigen; ALT: Alanine aminotransferase; HBV DNA: Hepatitis B virus deoxyribonucleic acid (viral load); APRI: Aspartate aminotransferase to platelet ratio index; Fib-4: Fibrosis-4 score; HBeAg: Hepatitis B e antigen (Adapted from National Viral Hepatitis Control Programme (NVHCP), Ministry of Health and Family Welfare, Government of India. Technical guidelines for diagnosis and management of hepatitis B)

Treatment Of Hepatitis B: For chronic hepatitis B, tenofovir or entecavir—both having a high barrier to resistance—are recommended in eligible adults, adolescents, and children aged 12 years or older. Tenofovir is preferred in women of childbearing age, during potential pregnancy, and in those with prior lamivudine exposure. Entecavir is recommended for children aged 2–11 years and may be favored in patients over 60 years or with bone disease, osteoporosis, fragility fractures, or renal impairment with low eGFR, proteinuria, or hypophosphatemia.

According to WHO guidelines, leprosy is managed using a multidrug therapy regimen tailored to the patient's age and classified into paucibacillary and multibacillary types [10]. The primary drugs used are rifampicin, clofazimine, and dapsone (diaminodiphenyl sulfone). Paucibacillary leprosy is treated with this combination for six months, whereas multibacillary cases require the same regimen for twelve months.

Abbreviations

- WHO: World Health Organization
- MDT: Multidrug Therapy
- HBV: Hepatitis B Virus
- HBsAg: Hepatitis B Surface Antigen
- HBeAg: Hepatitis B e Antigen
- APRI: Aspartate Aminotransferase to Platelet Ratio Index
- ALT: Alanine Aminotransferase
- PB: Paucibacillary
- MB: Multibacillary
- MDT: Multidrug Therapy
- PCR: Polymerase Chain Reaction
- HIV: Human Immunodeficiency Virus
- DNA: Deoxyribonucleic Acid

- AST: Aspartate Aminotransferase
- TLC: Total Leukocyte Count
- DLC: Differential Leukocyte Count
- ACE: Angiotensin-Converting Enzyme
- eGFR: Estimated Glomerular Filtration Rate
- ZN stain: Ziehl-Neelsen stain
- IgM: Immunoglobulin M
- IgG: Immunoglobulin G
- FPP: Fasting Plasma Glucose
- PD-1: Programmed Death 1

4. Conclusion

The presented case highlights the rare coexistence of Hansen's disease and chronic hepatitis B infection, emphasizing the complexities in diagnosis and management when these two conditions occur simultaneously. This dual infection provides valuable insights into the interplay between immune dysregulation in leprosy and chronic viral persistence in hepatitis B, necessitating careful therapeutic considerations to avoid hepatic complications. The case underscores the importance of baseline hepatitis B screening in patients diagnosed with leprosy before initiating multidrug therapy, ensuring individualized treatment plans and close follow-up. This report contributes to the limited literature on such dual infections, promoting heightened clinical awareness and further research into their immunopathological interactions and optimal management strategies.

Compliance with ethical standards

Disclosure of conflict of interest

No conflicts of Interest. In accordance with the ICMJE uniform disclosure requirements, the authors declare the following:

Statement of ethical approval

Ethical approval was not applicable for this case report as it does not constitute a research study. Patient informed consent for publication of clinical details and images was obtained.

Statement of informed consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Financial relationships

The authors report no financial relationships, either current or within the past three years, with organizations that could have an interest in this work.

Other relationships

The authors also declare no other relationships or activities that could be perceived as influencing the submitted work.

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