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A Case of Acute HIV Infection and Tuberculous Leprosy

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Introduction

Tuberculoid leprosy is a chronic infection caused by either Mycobacterium leprae or Mycobacterium Lepromatosis. Leprosy remains a significant global health concern, with approximately 133,802 cases reported in 2021, primarily concentrated in Southeast Asian and African regions [1]. Notably, no definite cases of leprosy have been acquired in the UK since 1954, with the last recorded case of UK-acquired leprosy dating back to 1925 [2,3]. The most frequently reported countries of birth among leprosy patients in all decades are India, followed by Pakistan, Bangladesh, Sri Lanka, and Nigeria [4]. Nevertheless, it is important to consider leprosy as a potential diagnosis in migrants from various parts of the world who have underlying comorbidities such as HIV [3].

Case Presentation

A 39-year-old male patient visited his General Practitioner due to extensive skin lesions affecting his face, hands, chest, and back. These lesions were described as painless, well-defined, scaly, and hairless, with raised edges (Figure 1,2). He also reported joint pain during physical exertion and occasional abdominal discomfort with no apparent change in his bowel habits. He denied experiencing night sweats, unintentional weight loss, or loss of appetite. His medical history was unremarkable, and he was not taking any regular medications.

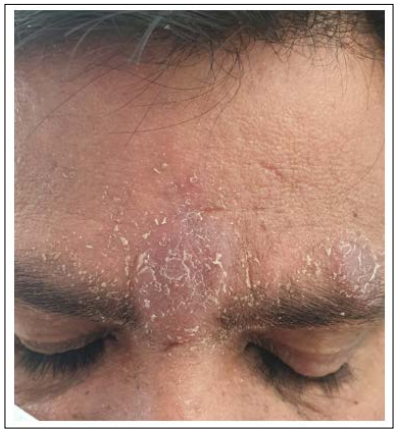

Figure 1: Glabella Facial Scaly Plaque

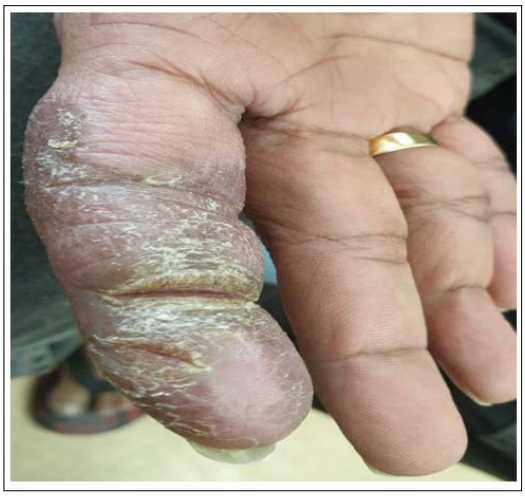

Figure 2: Finger Swollen by Inflammatory Plaque

The patient had migrated from India a few years ago and was employed in an office-based job in the UK, while his wife and child resided in his home country. He identified as a bisexual male and reported engaging in sexual relationships with multiple male partners prior to seeking medical attention.

Investigations

Given his clinical presentation and history, his GP ordered viral serological tests and viral load assessments, which confirmed positive results for Human Immunodeficiency Virus (HIV), Epstein Barr Virus (EBV), and Cytomegalovirus (CMV). Consequently, he was referred to a tertiary sexual health clinic for a comprehensive investigation where he underwent additional virology screening, which confirmed positive serology for Syphilis and rectal Chlamydia Trachomatis (CT). At the time of his HIV diagnosis, his CD4 cell count was 120 cells/mm^3, and his viral load was 162,000 copies/ml. He also tested positive for hepatitis B surface antigen, and core antigen. His autoimmune screening was positive for Anti-mitochondrial antibodies with non-M2 pattern. His biochemistry tests showed that he was Vitamin D and Vitamin B12 deficient, hence he was started on vitamin supplement. TSH was

raised with normal T3 and T4, hence this was considered secondary to autoimmune phenomenonn.

A skin biopsy on the glabella area revealed a dermis characterized by numerous granulomas composed of epithelioid histiocytes and a sparse lymphocytic infiltrate. Stains for tuberculosis (TB) and fungal infections were negative. Considering the inconclusive histological findings, his presentation was initially considered to be an acute HIV seroconversion episode, possibly accompanied by co-infections with CMV and Syphilis. Therefore, treatment for co-infections (Syphilis and CT) was initiated. His rectal Chlamydia was treated with a two-week course of oral doxycycline mad was given oral form of Valganciclovir for CMV Viraemia until his CMV viral load became undetectable. He further received single intramuscular dose of Benzathine 2.4 million units for latent or possible tertiary Syphilis, along with Septrin due to his low CD4 count of 120 cells/mm^3. These combined treatments led to some resolution of the facial rash.

Subsequently, the patient was started on a triple antiretroviral therapy regimen, Biktarvy, due to the advanced stage of HIV and associated immunosuppression. The patient displayed excellent adherence to Highly Active Antiretroviral Therapy (HAART) and did not experience any hyperactive immune response. After five months, his viral load became undetectable (<40 copies/ml), and his CD4 cell count improved to 220 cells/mm^3.

Six months after his initial visit to the Sexual Health Clinic, the patient attended urgent care due to a sudden left-sided facial droop and complete immobility on the left side of his face. A CT head scan ruled out acute intracranial events, and he was diagnosed with Bell's palsy. He was discharged the same day with a 10-day regimen of Prednisolone at 50 mg once daily and Acyclovir at 400 mg administered five times daily.

Four weeks later, he unexpectedly passed away at his home. A post-mortem examination revealed an extensive high-grade B-cell lymphoma with negative findings for Epstein-Barr virus (EBER) and Kaposi's sarcoma-associated herpesvirus (KSV). The tumour masses were observed in various organs, including the ileum, kidneys, oesophagus, lungs, liver, and spleen. The jejunum and mesentery displayed high-grade B-cell lymphoma (CD20+, CD79a+), affecting all layers of the bowel wall. Turbid fluid was found in the peritoneal cavity, along with pale tumour deposits in the pelvis. Additionally, splenomegaly was noted, likely due to syphilis.

During the autopsy, skin samples and a review of the skin biopsy slides revealed a diagnosis of tuberculoid leprosy, characterized by widespread peri-neurovascular granulomatous inflammation that had caused damage to the deep dermal nerves (Figure 3,4). Importantly, the histological sample did not show the presence of leprosy bacilli.

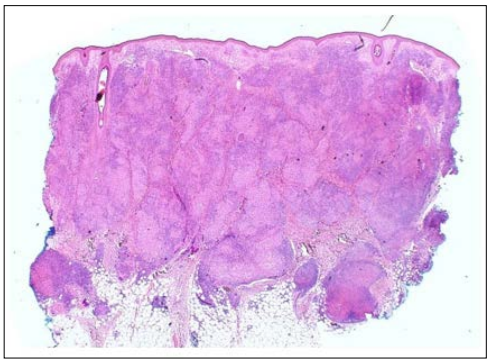

Figure 3: Biopsy Glabellar Skin. Low Power View, Pandermal Granulomatous Inflammation. H&E

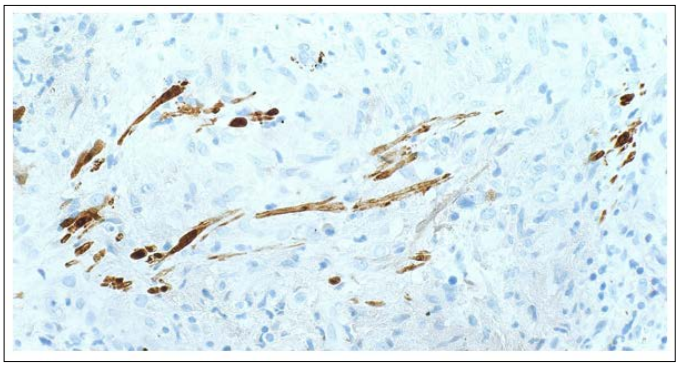

Figure 4: Biopsy Skin. S100 Immunostain Shows Fragmented Schwann Cells within a Nerve Destroyed by Granuloma

Discussion

This case report traces the clinical journey of a young male patient who initially presented with a skin rash and was later diagnosed with HIV, multiple sexually transmitted infections, and tuberculoid leprosy. Since Leprosy was categorised as a notifiable disease in 1951, the cases in the UK have been reported at a rate of around 10 per year. Over time, the incidence within UK has fallen significantly and this has led to concerns of underreporting due to the rarity of the disease and limited awareness of its clinical presentation. Between 1951 and 1960, 373 cases were reported, compared to 75 cases between 2011 and 2020 [2].

Leprosy, caused by M. leprae, can lead to damage in the respiratory system, skin, eyes, and nerves, resulting in anaesthesia and potential loss of extremities due to unnoticed injuries or infections. Tuberculoid leprosy patients typically exhibit a robust cell-mediated immune response to M. leprae, leading to a few skin lesions with well-organized lymphocytes and CD4-rich granulomas. In contrast, lepromatous leprosy patients have weaker immunity, predominantly featuring CD8 cells and experiencing widespread skin lesions with uncontrolled bacilli growth. In non-endemic regions like the UK, diagnosis of lepromatous leprosy is usually made through skin smears from skin lesions or ear lobes to detect acid-fast bacilli (AFB) under microscopy [4]. In contrast, the skin or peripheral nerve biopsy is used to investigate histological findings in tuberculoid cases since the effective immune response has eliminated the AFB.

In the context of HIV and leprosy co-infection, it's interesting to note that leprosy infection is only subtly influenced by HIV co-infection. HAART for HIV has led to an increased occurrence of leprosy within Immune Reconstitution Inflammatory Syndrome (IRIS), where an individual's health deteriorates in response to recovery of their immune system and thus, reacts to latent infections. The prevalence of IRIS among HIV-positive individuals varies, ranging from 3% to over 50%, influenced by factors like the patient's AIDS-defining illnesses and risk factors [5]. HAART can boost CD4+ cell production and immunity which, in turn, can reveal co-infections such as TB, CMV, and leprosy. Leprosy-associated IRIS is frequently observed as borderline tuberculoid leprosy, often accompanied by a Type 1 leprosy reaction, sometimes involving neuritis and/or lesion ulceration. Favourable outcomes are achieved with standard leprosy treatment and HAART maintenance [6].

Importantly, our patient exhibited a positive response to HAART and did not present with opportunistic infections commonly linked to advanced HIV immunosuppression, such as Pneumocystis pneumonia. Thorough history showed that the onset of leprosy symptoms preceded the initiation of antiretroviral therapy, making it unlikely to be a case of unmasking Immune Reconstitution Inflammatory Syndrome (IRIS).

Therefore, it is probable that the patient's leprosy and HIV conditions were coincidental rather than directly linked.

Conclusion

HIV is known for its diverse range of clinical presentations in affected individuals. We presented a male patient with a complex medical profile. He initially presented with extensive skin lesions, and his subsequent diagnostic journey revealed HIV infection, multiple sexually transmitted infections, tuberculoid leprosy, and a haematological malignancy. As HIV and leprosy co-infections become more prevalent, this case serves as an important reminder that a thorough diagnostic approach is required to suspect Leprosy especially among the individuals travelling from leprosy endemic region, such as India, Pakistan, Bangladesh, Sri Lanka, and Nigeria. Effort should be made to increase awareness of clinical symptoms and presentation of Leprosy to minimise risks of under-diagnosis and under-reporting of Leprosy.

Declaration of Interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

1. Global leprosy (2022) (Hansen disease) update, 2021: moving towards interruption of transmission weekly epidemiological record' Who reference number: WER 97: 429-450.
2. UK Health Security Agency (2023) Memorandum on leprosy 2 GOV UK https://www.gov.uk/government/publications/leprosy-memorandum/memorandum-on-leprosy-2023#ref9.
3. Lockwood DN, McIntosh A, Armstrong M, Checkley AM, Walker SL, et al. (2022) 'Diagnosing and treating leprosy in a non-endemic setting in a national centre, London, 1995 to 2018' PLoS Neglected Tropical Diseases 16: e0010799.
4. Lockwood DN, Lambert SM (2010) Leprosy and HIV, where are we at? Lepr Rev 81:169-175.
5. Pereira GA, Stefani MM, Araujo Filho JA (2004) Human immunodeficiency virus type 1 (HIV-1) and Mycobacterium leprae co-infection: HIV-1 subtypes and clinical, immunologic and histopathologic profiles in a Brazilian cohort. Am J Trop Med Hyg 71: 679-684.
6. Chen KH, Lin CY, Su SB, Chen KT (2022) Leprosy: A Review of Epidemiology, Clinical Diagnosis, and Management. J Trop Med 4: 2022-8652062.