International Journal of Research in Medical Sciences
Palraj A et al. Int J Res Med Sci. 2026 Sep;14(9):4078-4084
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20263131

Case Report

The ear, the nerves and the joints: a diagnostic puzzle in newly diagnosed HIV infection: a case report of HIV-associated inflammatory arthropathy mimicking Hansen disease and other infectious rheumatological mimics

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Received: 13 July 2026
Revised: 14 August 2026
Accepted: 19 August 2026

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ABSTRACT

A 53-year-old man with newly diagnosed HIV-1 infection (CD4+ T-cell count 178 cells/µL) developed a 3-month febrile inflammatory polyarthritis, dactylitis, painful auricular inflammation, nodular and ulcerative skin lesions, hypopigmented macules, and enlargement of bilateral common peroneal and ulnar nerves. Rheumatoid factor, anti-cyclic citrullinated peptide antibody and antinuclear antibody were negative. Slit-skin smears from two sites were negative for acid-fast bacilli (bacteriological index 0); the patient declined skin and lymph-node biopsies. Computed tomography of the chest showed bilateral cavitary consolidation, although sputum acid-fast smears and cartridge-based nucleic acid amplification testing were negative, mycobacterial culture was not performed, and bronchoscopic lavage was non-diagnostic. HIV-associated inflammatory arthropathy remained the working diagnosis. Arthritis and auricular inflammation improved during antiretroviral treatment, empirical antituberculous therapy and a tapering course of prednisolone, but Hansen disease and a lepra reaction could not be confidently excluded.

Keywords: HIV, Inflammatory arthritis, Dactylitis, Hansen disease, Leprosy reaction, Peripheral nerves

INTRODUCTION

Inflammatory arthritis in a person with HIV has a broad differential diagnosis. HIV-associated arthropathy may be the eventual diagnosis, but reactive arthritis, psoriatic arthritis, an unrelated rheumatic disease, immune reconstitution and infection must first be considered. This distinction is especially important when the presentation includes findings that are unusual for uncomplicated HIV arthropathy. We describe a man with febrile seronegative polyarthritis, dactylitis, auricular inflammation, tender nodules, hypopigmented lesions and thickening of several peripheral nerves-a combination that brought Hansen disease and a lepra reaction to the forefront of the differential diagnosis.[1-4]

CASE REPORT

A 53-year-old man from South India who worked as a plumber had been well until approximately 3 months before presentation. The illness began with pain and swelling of the right shoulder after machine-related work. An over-the-counter injection provided relief for approximately 2 days, after which severe pain recurred and disturbed sleep. Stiffness began on waking and persisted throughout the day, without substantial improvement with activity.

Over the following weeks, arthritis progressed to both shoulders, elbows and wrists; metacarpophalangeal, proximal interphalangeal and distal interphalangeal joints; both hips, knees and ankles; and the metatarsophalangeal and interphalangeal joints of the feet. Diffuse swelling of the right index finger was consistent with dactylitis. A daily high-grade fever usually began at approximately 19:00, with chills and rigors and temporary improvement after antipyretics. During the same interval, painful erythema and swelling affected the upper left ear. Throbbing pain disturbed sleep and partial auricular numbness followed, without hearing loss. There was no facial numbness or numbness elsewhere. Multiple predominantly subcutaneous nodules appeared on both forearms, some tender. Pruritic scalp scale developed, followed by hypopigmented facial and forehead lesions; there was no sensory loss over the lesions.

Approximately 6 weeks before presentation, HIV-1 infection was diagnosed during evaluation of prolonged fever and inflammatory arthritis using a standard serial serological testing algorithm. Baseline CD4+ T-cell count was 178 cells/µL; plasma HIV-1 RNA data were unavailable. Once-daily tenofovir disoproxil fumarate 300 mg, lamivudine 300 mg and dolutegravir 50 mg were initiated. Adherence was reported as good, without documented interruption before presentation. Approximately 15 days before presentation, he underwent a test injection described as Mantoux-like on the left forearm. He subsequently observed purulent ulcerative lesions at the right thigh, left shoulder, left gluteal region and right great toe. Most improved, but the gluteal ulcer persisted. This temporal association did not establish causation. The procedure and subsequent care occurred elsewhere; the test agent, indication and contemporaneous records were unavailable.

He also reported anorexia, a 10-kg weight loss within 1 month, dry cough and exertional dyspnoea for 10 days, the latter corresponding to New York Heart Association class II, and phlegm-like expectoration 4 days before presentation, without haemoptysis. There was no oral or genital ulceration, preceding enteric illness, inflammatory back pain, red eye, visual complaint or hearing loss. He reported a history of high-risk sexual exposure. There was no previous TB and no family history of psoriasis or connective-tissue disease. He was thin, with loss of buccal fat. There was no clinical pallor, icterus or cyanosis. Temperature was normal at examination, pulse 110/min, blood pressure 95/61 mmHg and respiratory rate 18/min. Clubbing, dyschromic nails and left leg oedema to mid-leg were present. Multiple right cervical nodes were matted; posterior auricular, left posterior cervical and bilateral submandibular nodes were also palpable. The posterior auricular nodes were tender; no axillary or inguinal lymphadenopathy was found.

Skin examination showed hypopigmented lesions on both hand dorsums without documented sensory loss; erythematous scaly palms, scalp scale with non-scarring alopecia, and multiple forearm nodules up to 4×4 cm. A tender 3×3 cm nodule lay near the right lateral epicondyle, and two tender erythema-nodosum-like lesions, the larger measuring 2×2 cm, were present on the left leg. A 4×3-cm hyperpigmented lesion on the right thigh contained a 0.5×0.5 cm central ulcer. Further ulcers measured 0.8×0.8 cm over the left shoulder, 1×1 cm over the dorsum of the right great toe and 3×2 cm over the left gluteal region. There was a painful erythematous scaly lesion over the left ear involving the auricle and lobule (Figure 1).

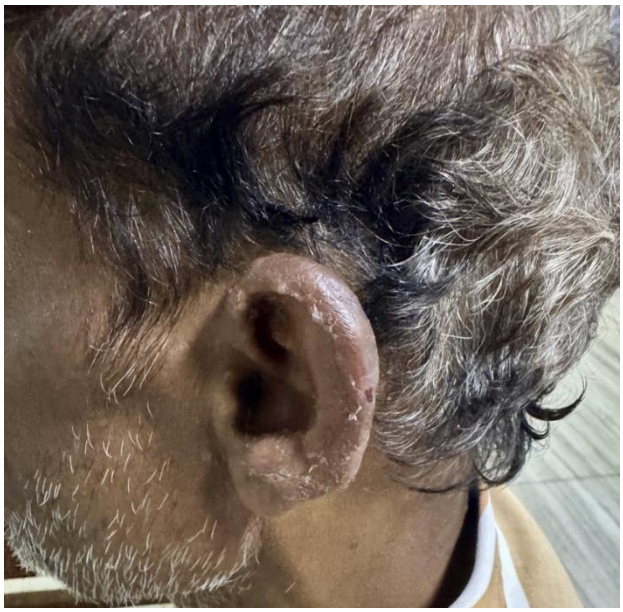

Figure 1: Left auricular inflammation with erythema, swelling and scaling involving the pinna and lobule.

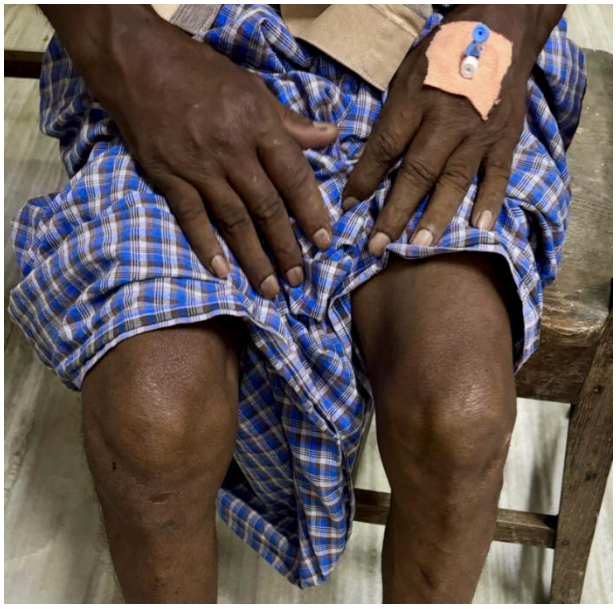

Figure 2: Clinical photograph showing right wrist swelling, bilateral hand-joint swelling, right index-finger dactylitis and right knee swelling in the setting of inflammatory polyarthritis.

The nose, oral cavity and throat examinations were normal. Both common peroneal nerves were enlarged and tender. Both ulnar nerves were thickened, nodular and non-tender on palpation. Bilateral thenar wasting was present. Higher mental functions and cranial nerves were normal. Power was 5/5 in all four limbs, deep-tendon reflexes were normal and plantar responses were flexor bilaterally. Superficial sensation and vibration sense were intact, and gait was normal. A left corneal opacity measuring approximately 1×1 cm was attributed to previous ocular trauma. There was objective polyarthritis. Both wrists and bilateral metacarpophalangeal and proximal interphalangeal joints were swollen and tender; distal interphalangeal joints were also involved. The right index finger was dactylitic. The right knee and both ankles were swollen, warm, tender and movement-restricted (Figure 2).

Both metatarsophalangeal joints were swollen and tender. Shoulders, elbows, hips, the left knee and interphalangeal joints of the feet were tender. There was no sacroiliac or spinal tenderness. Chest auscultation revealed vesicular breath sounds with bilateral crepitations.

The jugular venous pressure was not elevated. Cardiovascular examination showed normal first and second heart sounds without a murmur, and the abdomen was soft, with no palpable organomegaly.

Table 1: Chronology of the illness.

Interval	Clinical events
Approximately 3 months before presentation	Right shoulder pain and swelling began after machine-related work; temporary improvement followed an over-the-counter injection. Severe nocturnal pain and all-day stiffness recurred.
Following weeks	Inflammatory arthritis progressed to large and small joints of all four limbs, including distal interphalangeal joints; right index-finger dactylitis and daily evening fever with chills and rigors developed.
Same interval	Painful left auricular inflammation, forearm nodules, scalp scale and hypopigmented facial lesions appeared.
Approximately 6 weeks before presentation	HIV-1 was diagnosed by a serial serological algorithm (CD4+ T-cell count 178 cells/µL; plasma HIV-1 RNA unavailable); once-daily tenofovir disoproxil fumarate 300 mg, lamivudine 300 mg and dolutegravir 50 mg were started.
Approximately 15 days before presentation	A Mantoux-like test performed elsewhere was followed temporally by ulcers at multiple distant sites; causation was not assumed and procedural records were unavailable.
Presentation	Extensive polyarthritis, dactylitis, auricular inflammation, nodules, ulcers, tender bilateral common peroneal enlargement and bilateral ulnar thickening were documented. Two slit-skin smears were negative (bacteriological index 0); the patient declined skin and lymph-node biopsies.
Inpatient treatment and approximately 1 week after discharge	CT showed bilateral cavitary consolidation; sputum AFB smears and CBNAAT were negative, mycobacterial culture was not performed, and bronchoalveolar lavage was unremarkable. Empirical HRZE was started with a planned 6-month course, dolutegravir was increased to 50 mg twice daily, ceftriaxone 2 g/day was given for 5 days, and prednisolone was tapered from 15 mg/day to discontinuation. Leg ulcers healed completely by discharge; arthritis and auricular inflammation improved.

Haemoglobin was 9.0 g/dl, haematocrit 29%, mean corpuscular volume 82 fl, leucocyte count 6,600/mm³ (neutrophils 66%, lymphocytes 19%) and platelet count 390,000/mm³. The anaemia was normocytic and normochromic; iron studies were not performed. ESR was 62 mm/h and C-reactive protein 48 mg/l. Serum creatinine was 0.84 mg/dl; liver function tests were within normal limits. Serum uric acid was 4.6 mg/dl. HBsAg and HCV testing were negative. Urinalysis was normal, without proteinuria, haematuria or active sediment. Rheumatoid factor, anti-cyclic citrullinated peptide antibody and antinuclear antibody by enzyme-linked immunosorbent assay were negative. Slit-skin smears from the left earlobe and margin of the right-thigh ulcer, stained by the modified Ziehl-Neelsen method, were negative for acid-fast bacilli, with a bacteriological index of 0 at both sites. Skin and lymph-node biopsies were advised, but the patient declined both procedures. Transthoracic echocardiography was normal. Computed tomography of the chest showed bilateral cavitary consolidation. Sputum acid-fast bacillary smears and cartridge-based nucleic acid amplification testing were negative. Mycobacterial culture was not performed. Bronchoalveolar lavage evaluation was reported as unremarkable. After consultation with the thoracic medicine team, empirical antituberculous therapy comprising isoniazid, rifampicin, pyrazinamide and ethambutol was initiated using national tuberculosis programme weight-band dosing, with an intended total duration of 6 months, despite the negative microbiological evaluation. A blood culture was negative and syphilis serology (VDRL) was non-reactive. Synovial-fluid analysis was not performed. Tenofovir disoproxil fumarate

300 mg and lamivudine 300 mg once daily were continued. Following initiation of rifampicin-containing antituberculous therapy, dolutegravir was increased from 50 mg once daily to 50 mg twice daily. Prednisolone was initiated at 15 mg once daily and tapered through 10 mg and 5 mg daily before discontinuation over approximately 2 weeks. No disease-modifying antirheumatic drug was used. In view of fever and bilateral cavitary consolidation, empirical intravenous ceftriaxone 2 g once daily was administered for 5 days and then stopped. Pus culture from a skin ulcer showed no growth. The chronology of the illness is presented in Table 1.

Follow-up

At discharge, the leg ulcers had healed completely. Antiretroviral therapy and empirical antituberculous therapy were continued. At review approximately 1 week after discharge, the arthritis and auricular lesion had improved clinically. Longer follow-up, objective joint counts, repeat nerve examination, immunovirological response and radiological outcome were unavailable.

DISCUSSION

HIV-associated inflammatory arthropathy was the working diagnosis. Although the best-known pattern is an acute asymmetric oligoarthritis of the lower limbs, a symmetric rheumatoid-like polyarthritis and, less often, chronic destructive arthritis are well described. The timing of the polyarthritis around the diagnosis of HIV and the negative rheumatoid factor and anti-cyclic citrullinated peptide antibody supported this possibility. Seronegativity, however, is not specific, and the nodules, auricular lesion and palpable nerve thickening required an explanation of their own. The early clinical improvement was also difficult to interpret because ART, prednisolone, ceftriaxone and antituberculous treatment overlapped.[5–7]

Dactylitis, distal interphalangeal involvement and scalp and palmar scaling suggested a spondyloarthritis phenotype. Sexually acquired reactive arthritis usually follows genitourinary infection and tends to affect the lower limbs asymmetrically, often with enthesitis, dactylitis or mucocutaneous disease; the full urethritis-conjunctivitis-arthritis triad is uncommon and is not required for diagnosis.[8] Psoriatic arthritis can likewise produce distal interphalangeal arthritis and dactylitis.[9,10] In this patient, however, neither psoriasis nor typical psoriatic nail disease was confirmed, and there was no axial or entheseal involvement. These distinctions can be blurred in HIV: reactive arthritis may have a psoriasiform eruption or palmoplantar keratoderma, and psoriasis may be unusually extensive. The distribution of arthritis, a carefully elicited infection history and the evolution of the skin lesions are therefore more useful than serology alone. Syphilis serology was non-reactive, but his sexual history warranted nucleic-acid testing for Chlamydia trachomatis and Neisseria gonorrhoeae. The absence of diarrhoea, urethritis, conjunctivitis, enthesitis, sacroiliitis and inflammatory back pain, and the strikingly symmetric and widespread synovitis, made reactive arthritis less likely. Dermatological confirmation or biopsy of a scaly lesion would have been necessary before diagnosing psoriatic arthritis. Hansen disease was the most difficult alternative to dismiss. A diagnosis rests on at least one cardinal sign: definite sensory loss in a compatible skin lesion, thickening or enlargement of a peripheral nerve with sensory or motor impairment, or demonstration of bacilli. The hypopigmented lesions and bilateral thickening of the common peroneal and ulnar nerves were highly suggestive. Sensory and motor function was examined systematically, but neither definite anaesthesia within a lesion nor a deficit corresponding to an enlarged nerve was found. Slit-skin smears from two sites were negative. Taken together, these findings argued against multibacillary disease but did not exclude paucibacillary or predominantly neural leprosy.[11–13] A lepra reaction could account for much of the systemic illness. Type 1 reactions chiefly inflame existing lesions and nerves, whereas erythema nodosum leprosum may cause recurrent tender nodules, fever, oedema, lymphadenitis, neuritis, arthritis and dactylitis.[14-18] The latter pattern closely resembled this case. A biopsy of an active, untreated nodule or plaque for routine histology and Fite–Faraco staining, with Mycobacterium leprae polymerase chain reaction where available, would have been the most useful discriminator; however, the patient declined skin biopsy. Leprosy-associated immune-reconstitution inflammatory syndrome was less convincing as an explanation for disease onset because the fever, arthritis and skin disease began before ART, even though the baseline CD4+ T-cell count was only 178 cells/μL. Plasma HIV-1 RNA and subsequent immunovirological response were unavailable.[19] The painful auricle and seronegative arthritis also raised relapsing polychondritis. Classic auricular chondritis usually spares the non-cartilaginous lobule and is often followed by inflammation of the nose, airway, eyes or audiovestibular structures.[20–22]

Here the lesion was unilateral and scaly, involved the lobule, and was not accompanied by disease at another cartilaginous site; accepted clinical criteria were not met. VEXAS syndrome was another reasonable consideration in a man older than 50 years with fever, anaemia, chondritis-like inflammation, skin lesions, arthritis and lung abnormalities. However, his anaemia was normocytic rather than macrocytic, his platelet count was raised rather than reduced, and the illness was neither recurrent nor steroid dependent. The prominent peripheral nerve thickening was also atypical.[23–25] UBA1 testing would become appropriate if otherwise unexplained inflammation recurred with macrocytosis, cytopenias or marrow abnormalities.

Table 2: Differential diagnosis of the combined articular, cutaneous, auricular and neural phenotype

Diagnosis	Supporting features	Limitations and required discriminators
HIV-associated inflammatory arthropathy	Seronegative acute/subacute polyarthritis around HIV diagnosis; CD4+ T-cell count 178 cells/µL; ART initiated.	Working diagnosis, but does not independently explain dactylitis, nodules, auricular disease or multiple enlarged nerves. Improvement under ART, prednisolone and empirical antituberculous therapy was non-specific; plasma HIV-1 RNA was unavailable and longer objective follow-up is required.
Reactive arthritis	Sexual risk, dactylitis, distal and lower-limb disease, possible psoriasiform scale.	No documented urethritis, enteritis, eye disease, enthesitis or axial disease. Syphilis serology was non-reactive; gonorrhoea/chlamydia nucleic-acid testing was not documented.
Psoriatic arthritis	Dactylitis, distal interphalangeal involvement, scalp/palmar scale, RF negativity.	Psoriasis and typical nail dystrophy were not confirmed; nerve enlargement is discordant. Dermatology assessment and lesion biopsy are required.
Hansen disease/lepra reaction	Endemic setting; hypopigmented lesions; bilateral common peroneal and ulnar enlargement; tender nodules, fever, oedema, lymphadenopathy, arthritis and dactylitis.	No definite lesion anaesthesia or mapped nerve deficit; slit-skin smears were negative and skin biopsy was declined. Reconsider lesion/nodule biopsy, stains and M. leprae PCR if the patient later consents.
Relapsing polychondritis	Auricular inflammation with seronegative arthritis.	Unilateral scaly auricular and lobular lesion; no nasal, airway, ocular or audiovestibular involvement; criteria not met.
VEXAS syndrome	Man aged >50 years with fever, anaemia, chondritis-like disease, skin lesions, arthritis and pulmonary abnormalities.	Normocytic anaemia (mean corpuscular volume 82 fL), thrombocytosis and a short non-refractory course argue against it; consider marrow/UBA1 testing only if unexplained inflammation recurs.
TB/Poncet disease	HIV with CD4+ T-cell count 178 cells/µL, weight loss, cough, clubbing, matted nodes, erythema-nodosum-like lesions, polyarthritis and bilateral cavitary consolidation.	Sputum smears and CBNAAT were negative; mycobacterial culture was not performed; bronchoalveolar lavage was unremarkable and lymph-node biopsy was declined. Empirical antituberculous therapy was started, but microbiological confirmation and synovial exclusion of direct infection were lacking; document radiological and clinical response.
Septic or disseminated infection	Fever with rigors, tachycardia, ulcers and warm effusions in HIV.	Pus culture showed no growth and a blood culture was negative. Ulcers improved during 5 days of empirical ceftriaxone. Chronic symmetric arthritis remained atypical, but synovial-fluid studies were not performed.

AFB, acid-fast bacilli; ART, antiretroviral therapy; CBNAAT, cartridge-based nucleic acid amplification test; CT, computed tomography; PCR, polymerase chain reaction; RF, rheumatoid factor; TB, tuberculosis; VEXAS, vacuoles, E1 enzyme, X-linked, autoinflammatory, somatic.

Tuberculosis was a major competing diagnosis. Fever, rapid weight loss, cough, clubbing, matted cervical nodes, HIV and erythema-nodosum-like lesions were concerning in a TB-endemic setting, and the bilateral cavitary consolidation strengthened that concern.[26] Against this, sputum acid-fast smears and cartridge-based nucleic acid amplification testing were negative, mycobacterial culture was not performed, and bronchoalveolar lavage was unrevealing. Lymph-node biopsy was advised but declined. The thoracic medicine team nevertheless advised empirical antituberculous therapy. Poncet disease is an aseptic, non-erosive inflammatory arthritis that accompanies active TB elsewhere in the body and improves with treatment of the infection.[27] In this case, pulmonary TB was not microbiologically confirmed, an inflamed joint was not aspirated, and the early response occurred while prednisolone was being given. Poncet disease therefore remained possible but unproven; sustained clinical improvement together with radiological resolution of the lung lesions would have been needed to support it.

The high fever with rigors, tachycardia, ulcers and several warm, swollen joints also demanded direct exclusion of infection before immunosuppression. Bacterial, gonococcal, mycobacterial and fungal arthritis may be

atypical or polyarticular in an immunocompromised host. A blood culture was negative, but aspiration of the knee or another accessible effusion for cell count, crystals, Gram stain and culture would have made the final diagnosis more secure. Mycobacterial and fungal studies could then have been added according to the synovial findings and clinical course.[28]

Other infectious mimics were less likely but not fully assessed. Disseminated gonococcal infection may present with fever, migratory joint symptoms, tenosynovitis and a sparse pustular eruption; secondary syphilis can combine generalized skin disease, lymphadenopathy and musculoskeletal symptoms. Syphilis serology was non-reactive, but gonococcal and chlamydial nucleic-acid testing was not documented. Bacteraemia, infective endocarditis and disseminated fungal infection merit attention when fever, clubbing, nodules, ulcers and polyarthritis coexist. The negative pus culture neither established nor excluded a systemic bacterial infection: it sampled only one skin lesion. A blood culture was negative, but synovial cultures were not obtained. With a CD4+ T-cell count of 178 cells/µL, opportunistic infection had to remain in the differential diagnosis.[28]

In a similar presentation, the evaluation should proceed along several tracks at the same time. Synovitis should be documented and a readily accessible warm joint aspirated before glucocorticoids if the patient's condition allows. The articular pattern-symmetric or asymmetric, peripheral or axial, with or without dactylitis and enthesitis-should then guide rheumatological testing. Infection should be investigated according to the clinical setting, including blood cultures, sexual-health screening, TB imaging and microbiology, and fungal studies when indicated. At the same visit, skin lesions and palpable nerves should be mapped carefully, with sensory, motor and autonomic findings recorded for each affected nerve. Biopsy should target a fresh, untreated lesion. Follow-up is equally important: objective joint counts, inflammatory markers, immune and virological response, nerve findings, and the radiological course of the lung lesions would help separate diagnoses that look similar at presentation.[28] The differential diagnosis of the combined articular, cutaneous, auricular and neural phenotype is presented in Table 2.

CONCLUSION

This case highlights that inflammatory arthritis in newly diagnosed HIV infection should not be assigned too quickly to HIV-associated arthropathy when peripheral nerve enlargement, hypopigmented lesions, nodules and auricular inflammation are present. The main advance is the bedside diagnostic lesson: HIV-associated inflammatory arthropathy, Hansen disease, lepra reactions, tuberculosis-associated rheumatic disease and disseminated infection can converge in the same clinical space, and the distinction depends on careful skin and nerve examination, targeted microbiology, tissue sampling where feasible and longitudinal follow-up. In such patients, a rheumatological label should follow the exclusion of treatable infection rather than replace it.

ACKNOWLEDGEMENTS

The authors would like to express gratitude to Dr. Nitheesh Premhar for assistance with data collection. The authors also thank the clinical and nursing staff involved in the patient's care.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Palraj A, Subramanian ARP, Ramadoss I, Veeran VK. The ear, the nerves and the joints: a diagnostic puzzle in newly diagnosed HIV infection: a case report of HIV-associated inflammatory arthropathy mimicking Hansen disease and other infectious rheumatological mimics. Int J Res Med Sci 2026;14:4078-84.