CASE REPORT

Accelerated CD4+ T-cell recovery following vitamin D supplementation in a virologically suppressed adult living with HIV - A Case Report

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ABSTRACT

Background: Immune non-response despite sustained HIV viral suppression remains a clinical challenge. We report a patient with a prolonged plateau in CD4 count who experienced a marked rise following correction of vitamin D insufficiency.

Case presentation: A 48-year-old man with chronic HIV infection, fully adherent to antiretroviral therapy and virologically suppressed for at least two years (2022-2024), developed disseminated osteoarticular tuberculosis involving the left hip and shoulder. After completing prolonged anti-tuberculosis therapy, his viral load remained suppressed (40 copies/mL) but CD4 count plateaued at 426 cells/mm3. Serum 25-hydroxyvitamin D was 25 ng/mL (insufficient). Vitamin D3 60,000 IU weekly was initiated. After six months, CD4 count increased to 1,071 cells/mm3 while viral suppression was maintained, with no change in antiretroviral regimen or other concurrent therapies.

Conclusion: Vitamin D supplementation temporally coincided with accelerated CD4 recovery in a virologically suppressed patient with prior poor immunological recovery. Causality cannot be inferred from a single case; controlled studies are required to clarify whether vitamin D repletion benefits immune non-responders.

INTRODUCTION

1,25-dihydroxycholecalciferol is the most active metabolite of vitamin D and is best known for its role in calcium and bone metabolism.[1] Evidence also supports immunomodulatory functions of vitamin D in innate immunity, including host responses to intracellular pathogens such as Mycobacterium tuberculosis, fungi, and viruses.[2] With effective antiretroviral therapy (ART), many people living with HIV (PLHIV) achieve sustained viral suppression. However, a subset of patients demonstrate incomplete immune reconstitution, with persistently low CD4 counts despite virological control (immune non-response).[3]

Hypovitaminosis D has been associated with impaired CD4 recovery during ART. In a longitudinal observational study, vitamin D deficiency or insufficiency was associated with a greater deficit in absolute CD4 cell recovery.[4] In vitro data also suggest that vitamin D may reduce CD4+ T-cell susceptibility to HIV infection.[5] This

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report describes a case of accelerated CD4 recovery following vitamin D supplementation in a patient with sustained viral suppression yet previously poor CD4 recovery.

Case presentation

Patient information

A 48-year-old man with a 9-year history of HIV infection (diagnosed in 2013) was on ART with excellent adherence. In 2021, his regimen was changed from tenofovir disoproxil fumarate/emtricitabine/efavirenz to tenofovir alafenamide/emtricitabine/dolutegravir.

Clinical findings

He presented with severe pain in the left hip and shoulder, accompanied by muscle spasms in the left thigh and inability to walk. Examination revealed marked left hip tenderness exacerbated by external rotation. The left shoulder was tender on rotational movement. Abduction and adduction were possible but limited.

Diagnostic assessment

Magnetic resonance imaging (MRI) demonstrated avascular necrosis of the left femoral head. Tuberculosis was confirmed by GeneXpert (Xpert MTB/RIF) testing of aspirated pus and excised femoral head tissue, detecting rifampicin-sensitive Mycobacterium tuberculosis.

Therapeutic intervention

He commenced first-line anti-tuberculosis therapy (rifampicin, isoniazid, ethambutol, and pyrazinamide) for an intensive phase of two months, followed by a continuation phase with rifampicin and isoniazid. Because of slow resolution of joint collections, the continuation phase was extended beyond the standard duration to a total of 22 months. At 24 months, he developed a cold abscess in the left shoulder with persistent collections in the left hip, raising concern for drug-resistant tuberculosis. Microbiological confirmation was not possible due to difficulty obtaining sputum and challenging joint

Timeline

Date/period	Key events and findings
2013	HIV diagnosed; started ART.
2021	Switched to tenofovir alafenamide/emtricitabine/dolutegravir.
2022	Diagnosed with disseminated osteoarticular tuberculosis (left hip and shoulder); commenced anti-tuberculosis therapy.
2022-2024	Sustained viral suppression documented, including during active tuberculosis.
36 months of TB treatment	Viral load 40 copies/mL; CD4 plateaued at 426 cells/mm3; patient concerned about persistently low CD4 count.
Post-TB treatment	25-hydroxyvitamin D measured at 25 ng/mL; vitamin D3 60,000 IU weekly started.
6 months later	CD4 increased to 1,071 cells/mm3 with continued viral suppression (Figure 1).

aspiration. Adherence was reviewed and judged to be good based on the tuberculosis treatment card, with the patient's spouse acting as treatment observer. Clinical suspicion for resistance was considered low because he remained systemically well (no fever, night sweats, or weight loss) with a normal body mass index. A pragmatic clinical decision was made to add levofloxacin and extend treatment for a further 12 months. Serial imaging (Figure 2) was used to monitor therapeutic response.

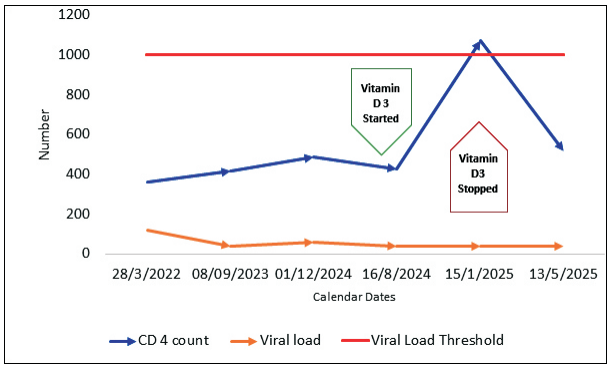

Figure 1: Trends of CD4 Counts, viral load status against time points of Vitamin D supplementation

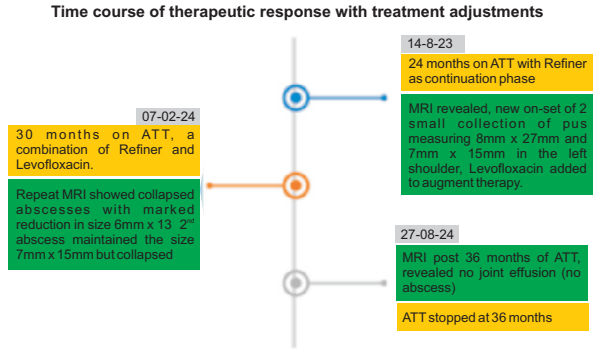

Figure 2: Time course of therapeutic response with treatment adjustments

Follow-up and outcomes

At completion of anti-tuberculosis therapy (36 months), HIV viral load remained suppressed (40 copies/mL), yet CD4 count had plateaued at 426 cells/mm3. The patient remained concerned about his persistently low CD4 count. Serum 25-hydroxyvitamin D was 25 ng/mL, consistent with insufficiency (reference range 30-100 ng/mL). Vitamin D3 60,000 IU weekly was initiated. Six months later, CD4 count increased to 1,071 cells/mm3 while viral suppression was maintained, without changes to ART or other concurrent treatments.

DISCUSSION

This case suggests a temporal association between vitamin D repletion and substantial immunological recovery in a patient with stable virological control but persistently low CD4 count. Vitamin D deficiency has been linked to impaired T-cell proliferation and macrophage dysfunction.[6]

Mechanistically, vitamin D has been reported to influence immune activation markers and chemokine receptor expression and may modulate pro-inflammatory cytokines implicated in HIV pathogenesis and immune activation.[7] Vitamin D has also been proposed to have protective effects in HIV-1 infection.[8]

However, the broader evidence base remains mixed. Some supplementation studies have not demonstrated significant improvements in immune recovery among PLHIV, and heterogeneity in dosing, baseline vitamin D status, and study populations limits generalisability.[9] Randomised trial data have also been variable.[10]

Causality cannot be inferred from a single observation. Potential confounders include unmeasured changes in diet, reduced systemic inflammation following tuberculosis resolution, and regression to the mean. Vitamin D was assessed only once, and follow-up measurements of vitamin D, calcium, and phosphate were not documented; these limit interpretation and safety assessment. Despite these limitations, clinicians may consider evaluating and correcting vitamin D insufficiency on a case-by-case basis, particularly in patients with suboptimal CD4 responses, while awaiting more definitive evidence.

CONCLUSION

Vitamin D supplementation temporally coincided with accelerated CD4 recovery in a virologically suppressed patient with previously poor immunological recovery. The observation is hypothesis-generating and should be interpreted cautiously. Larger, controlled studies are needed to clarify whether vitamin D repletion improves immune reconstitution in immune non-responders with HIV.

Patient perspective

The patient expressed concern regarding persistently low CD4 counts despite long-term viral suppression. He reported reassurance after the subsequent rise in CD4 count following initiation of vitamin D supplementation.

Informed consent

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

Conflict of interest

Authors declared they have no conflicts of interest.

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