



LIFESTYLE MODIFICATION AND SUPPLEMENTATION IN A WOMAN WITH VITAMIN D DEFICIENCY AND NON SPECIFIC CHRONIC LOW BACK PAIN: A CASE REPORT

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Article History :

Received date : 2025/04/17

Revised date : 2026/05/04

Accepted date : 2026/06/01

Published date : 2026/07/21



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E-ISSN :

ISSN 3048-1368



P-ISSN

ISSN 3048-1376



ABSTRACT

Background: Non-specific chronic low back pain (CLBP) is a leading cause of disability worldwide. Vitamin D deficiency has been increasingly associated with CLBP through impaired bone metabolism, inflammation, and pain sensitization. Identifying and correcting vitamin D deficiency may improve clinical outcomes, particularly in patients without structural spinal abnormalities.

Case Illustration: A 26-year-old woman presented with a three-month history of severe low back pain (Visual Analog Scale [VAS] score 8). Physical examination and lumbosacral MRI revealed no significant abnormalities. Serum 25-hydroxyvitamin D was 11.6 ng/mL, confirming vitamin D deficiency. The patient received calcitriol 0.5 µg twice daily, vitamin D₃ 5,000 IU/day, calcium lactate 500 mg/day, nutritional counseling, daily 30-

minute sun exposure, and regular exercise. After two months, serum vitamin D increased to 22.7 ng/mL with pain improvement (VAS 5). At three months, vitamin D normalized to 34.1 ng/mL and the patient achieved complete pain resolution (VAS 0).

Discussion: The patient's clinical improvement supports the role of vitamin D optimization as part of a multimodal management strategy for non-specific CLBP. Vitamin D supplementation combined with lifestyle modification may reduce inflammation, improve musculoskeletal function, and enhance pain recovery, particularly in individuals with confirmed deficiency.

Conclusion: Screening for vitamin D deficiency should be considered in patients with unexplained chronic low back pain. Combined vitamin D supplementation and lifestyle modification may represent a safe and effective adjunctive treatment for vitamin D deficiency–associated CLBP.

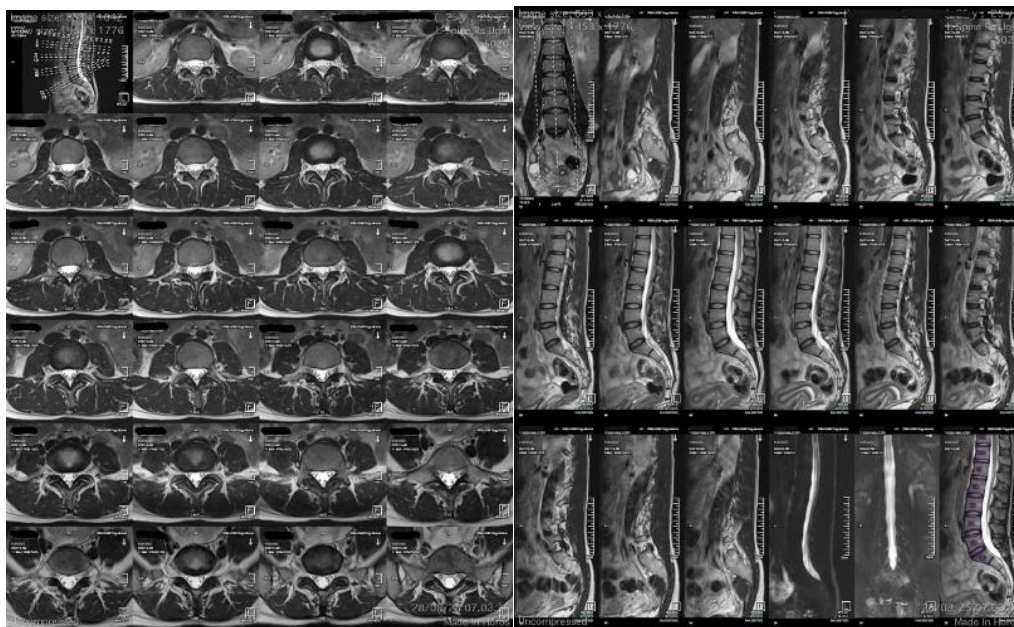
Keywords: vitamin D deficiency, chronic low back pain, supplementation, lifestyle modification

INTRODUCTION

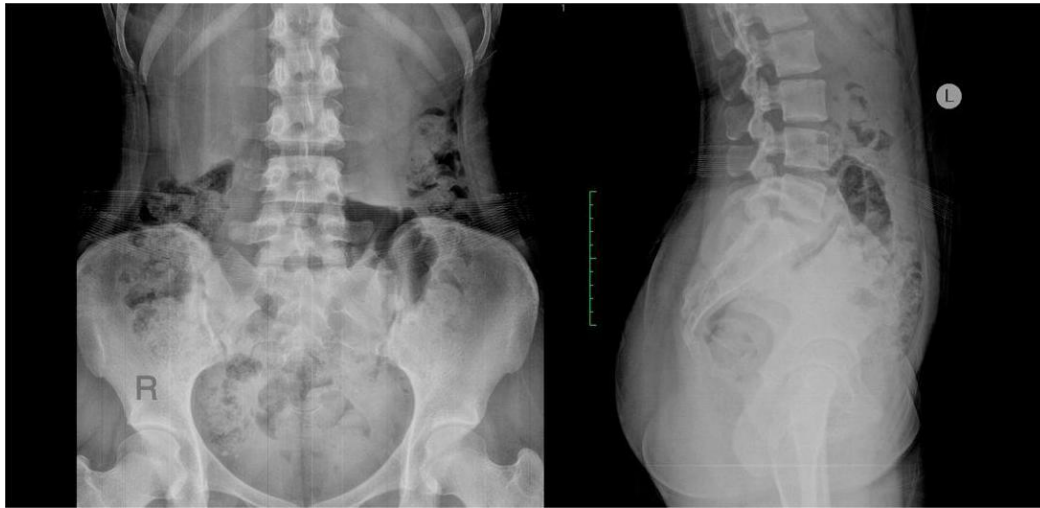
Chronic low back pain (CLBP) is defined as pain located between the costal margins and the inferior gluteal folds persisting for ≥ 3 months, with or without neurological deficits, and remains one of the leading causes of disability worldwide, with a global prevalence exceeding 600 million cases and a projected rise to more than 840 million by 2050^{1,4,5}. Non-specific CLBP, in which no clear structural or radiological cause can be identified, accounts for the majority of cases and continues to challenge clinicians seeking safe, modifiable therapeutic targets¹. Emerging evidence points to vitamin D status as one such target: patients with CLBP are more likely to have vitamin D deficiency, and lower serum 25-hydroxyvitamin D concentrations correlate with greater pain intensity and a higher inflammatory burden^{2,6,7}. Vitamin D deficiency has been shown to heighten both peripheral and central pain sensitization, an effect explained by its influence on neurite and astrocyte outgrowth, its modulation of glial and opioid-mediated pain pathways, and its more recently described capacity to suppress mitochondria-associated ferroptosis and neuroinflammatory signaling within dorsal horn neurons^{2,7,8}. Large ancillary analyses of major supplementation trials and cohort studies have produced mixed results regarding the analgesic benefit of vitamin D repletion, suggesting that its effect depends on baseline deficiency severity, pain chronicity, and concurrent lifestyle factors rather than supplementation alone^{3,9,10}. Vitamin D deficiency nevertheless remains highly prevalent, particularly among women of reproductive age in tropical, sun-abundant countries such as Indonesia, where limited sun exposure practices and inadequate dietary intake continue to drive high rates of hypovitaminosis D^{17,18,19}. We present a case of a young woman with severe non-specific CLBP and vitamin D deficiency who achieved complete pain resolution after three months of combined vitamin D supplementation and lifestyle modification, illustrating the value of correcting vitamin D status as part of a low-risk, non-pharmacological CLBP management strategy alongside guideline-recommended exercise therapy^{20,21}.

CASE ILLUSTRATION

A 26-year-old woman, initial “K,” presented to the Orthopedic Spine Clinic at Gadjah Mada University Academic Hospital with a three-month history of low back pain rated as 8 on the Visual Analog Scale (VAS), which had worsened by her first visit in February 2025. Her vital signs were within normal limits. Physical examination revealed a body weight of 45 kg, height of 158 cm (BMI 18.0 kg/m²), waist circumference of 72 cm, and upper arm circumference of 23 cm. Provocative tests, including Lasegue’s and Patrick’s (FABER) tests, were negative, and there was no lumbar tenderness. Lumbosacral X-ray and non-contrast MRI findings were unremarkable. Laboratory results showed a serum vitamin D level of 11.6 ng/mL, confirming vitamin D deficiency. She was diagnosed with vitamin D deficiency–related low back pain and was started on calcitriol 0.5 µg every 12 hours, calcium lactate 500 mg daily, and vitamin D₃ 5000 IU daily. Nutritional targets were set at 1125 kcal/day (25 kcal/kg/day) and 45 g of protein/day (1 g/kg/day). She was also advised to have daily sun exposure for 30 minutes and to exercise for at least 30 minutes per day, with monthly reassessment of serum vitamin D levels.



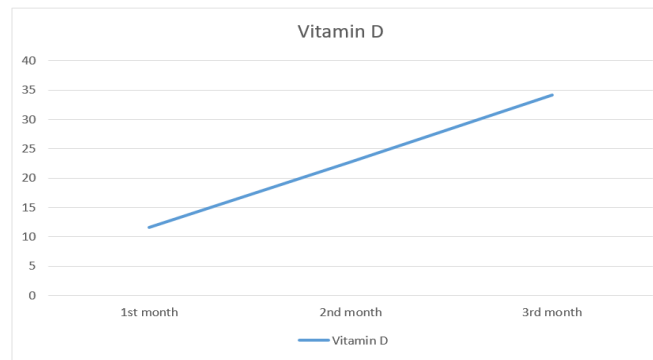
Normal MRI



Normal Lumbosacral Ap/Lat

At the two-month follow-up, her vitamin D level had increased to 22.7 ng/mL, and her low back pain improved to VAS 5. Physical examination showed a weight of 46 kg (BMI 18.4 kg/m²), waist circumference of 72.5 cm, and upper arm circumference of 23.2 cm. The same supplementation regimen was continued, and a repeat vitamin D test was scheduled for the following month.

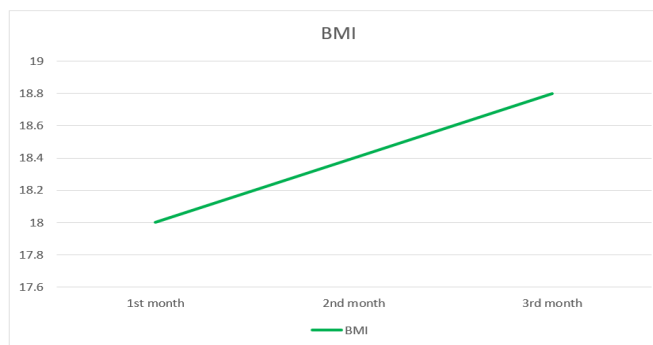
By the third month, the patient reported complete resolution of low back pain, and her vitamin D level had normalized to 34.1 ng/mL. At that time, she weighed 47 kg (BMI 18.8 kg/m²), with a waist circumference of 73 cm and upper arm circumference of 23.5 cm. The patient was declared to have achieved normal vitamin D levels, and no further follow-up was required. She was counseled to maintain a healthy lifestyle and balanced diet to preserve optimal musculoskeletal health and prevent recurrence of vitamin D deficiency.



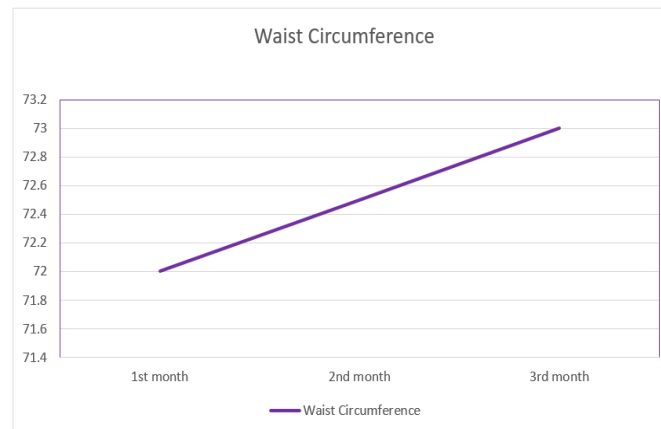
Monitoring graph vitamin D



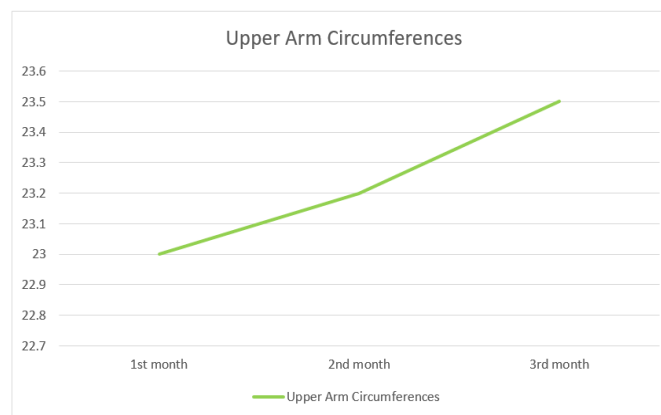
Monitoring graph weight



Monitoring graph BMI



Monitoring graph waist
circumference



Monitoring graph upper arm
circumference

DISCUSSION

Vitamin D deficiency is increasingly recognized as a contributing factor in the pathogenesis of non-specific low back pain^{1,2,6}. As a steroid hormone, vitamin D regulates calcium and phosphorus homeostasis, which is essential for maintaining bone mineralization, structural integrity, and normal bone turnover^{9,11}. In deficiency states, impaired intestinal calcium absorption triggers compensatory skeletal mobilization of calcium, and prolonged deficiency can precipitate osteomalacia or worsen pre-existing osteopenia and osteoporosis, both of which are associated with vertebral microarchitectural weakness and mechanical back pain^{11,12,13}. This calcium-phosphate imbalance simultaneously stimulates parathyroid hormone (PTH) secretion; chronically elevated PTH accelerates bone turnover and can produce microfractures within vertebral trabecular bone, further contributing to axial pain^{11,13}.

Beyond its skeletal role, vitamin D exerts broad immunomodulatory and anti-inflammatory effects. Through vitamin D receptor signaling, it downregulates pro-inflammatory cytokines such as TNF- α , IL-1 β , IL-6, and IL-8, while promoting anti-inflammatory mediators including IL-10, thereby dampening the low-grade systemic inflammation implicated in chronic pain states^{14,15}. At the neuronal level, vitamin D modulates nociceptive signaling through calcium-channel regulation, suppression of TRPV1-mediated transduction, glial cell inhibition, and endogenous opioid peptide gene expression; more recent mechanistic work has also demonstrated that vitamin D3 attenuates neuropathic pain by suppressing mitochondria-associated ferroptosis via inhibition of the PKC α /NOX4 signaling pathway in the spinal cord^{7,8}. These combined skeletal, immunological, and neuronal mechanisms provide biological plausibility for the clinical improvement observed in this patient's pain following vitamin D repletion.

Nevertheless, the evidence for vitamin D supplementation as a stand-alone analgesic intervention remains inconsistent: a recent updated meta-analysis of randomized controlled trials found only a modest, non-significant reduction in pain scores with supplementation alone³, and a large ancillary analysis of the VITAL trial similarly found no significant effect of vitamin D on pain prevalence or severity in a general older-adult population⁹. These findings suggest that

supplementation is most beneficial in patients with confirmed deficiency and pain of suspected vitamin D-related origin, rather than as a universal analgesic, and that its effect may be potentiated when combined with adequate dietary calcium and protein intake, regular physical activity, and safe sun exposure^{10,16,20}. Current clinical practice guidelines for chronic low back pain also consistently recommend structured exercise therapy as a first-line, non-pharmacological intervention, which may act synergistically with vitamin D optimization to improve musculoskeletal loading capacity and reduce pain^{20,21}.

The high background prevalence of vitamin D deficiency among Indonesian women of reproductive age driven by limited sun exposure practices, dietary insufficiency, and melanin-related attenuation of cutaneous UVB synthesis underscores the importance of screening for hypovitaminosis D in young patients presenting with otherwise unexplained chronic low back pain, particularly when structural imaging is unremarkable^{17,18,19}. In this case, a multimodal approach combining calcitriol, calcium, and vitamin D3 supplementation with structured lifestyle modification, including daily sun exposure and regular exercise, was associated with progressive normalization of serum vitamin D and complete resolution of pain over three months, supporting an integrated rather than isolated therapeutic strategy^{2,16}.

The favorable outcome observed in this case is likely attributable to the synergistic effects of vitamin D repletion and lifestyle modification rather than supplementation alone. Adequate sunlight exposure, regular physical activity, and improved nutritional intake enhance musculoskeletal health by promoting muscle strength, bone remodeling, and functional recovery. Exercise therapy remains the cornerstone of non-specific CLBP management according to current clinical guidelines, while vitamin D optimization may improve the physiological response to rehabilitation in patients with documented deficiency. This integrated approach may explain the progressive reduction in pain intensity and normalization of serum vitamin D concentrations over the three-month follow-up period.¹⁷ Although evidence regarding vitamin D supplementation as a stand-alone treatment for chronic low back pain remains inconsistent, recent studies suggest that its greatest benefit is observed in patients with confirmed vitamin D deficiency rather than in unselected populations.^{2,19} This case supports a targeted screening strategy for hypovitaminosis D, particularly among young

women in tropical countries where vitamin D deficiency remains prevalent despite abundant sunlight. Early recognition and correction of vitamin D deficiency, combined with guideline-recommended non-pharmacological interventions, may reduce pain severity, improve functional outcomes, and potentially decrease unnecessary diagnostic procedures and prolonged analgesic use. Nevertheless, larger prospective studies are needed to establish the long-term effectiveness and optimal treatment protocols for vitamin D-related chronic low back pain.^{20,21}

CONCLUSION

Supplementation with lifestyle modification and adequate dietary intake can increase vitamin D levels, thereby reducing low back pain.

KEYWORDS

Vitamin D deficiency, chronic low back pain, supplementation, lifestyle modification.

REFERENCES

1. Kanaujia V, Yadav R, Verma S, Jain S, Patra B, Neyaz O. Correlation between vitamin D deficiency and non-specific chronic low back pain: retrospective observational study. *Journal of Family Medicine and Primary Care*. 2021;10(2).
2. Xu H, Zhang S, Yi Y, Chen H, Hu T, Wang S, Wu D. Relationship between vitamin D and nonspecific low back pain may be mediated by inflammatory markers: retrospective study. *Pain Physician*. 2021;24:E1015-E1023.
3. Lee TH, Tsai RY, Ho CH, Chen CC, Li CY. Updated meta-analysis reveals limited efficacy of vitamin D supplementation in chronic low back pain. *In Vivo*. 2024;38(6):2955-2967.
4. GBD 2021 Low Back Pain Collaborators. Global, regional, and national burden of low back pain, 1990–2020, its attributable risk factors, and projections to 2050: a systematic analysis of the Global Burden of Disease Study 2021. *Lancet Rheumatology*. 2023;5(6):e316-e329.

5. Zhang C, Lv B, Yi Q, Qiu G, Wu F. Global, regional, and national burden of low back pain in working-age population from 1990 to 2021 and projections for 2050. *Frontiers in Public Health*. 2025;13:1559355.
6. Jiang X, Zhou R, He Y, Zhu T, Zhang W. Causal effect of serum 25-hydroxyvitamin D levels on low back pain: a two-sample Mendelian randomization study. *Frontiers in Genetics*. 2022;13:1001265.
7. Li M, et al. Vitamin D deficiency-associated neuropathic pain examined in a chronic pain management program. *The Permanente Journal*. 2024;28(4).
8. Zhang W, Yu S, Jiao B, Zhang C, Zhang K, Liu B, Zhang X. Vitamin D3 attenuates neuropathic pain via suppression of mitochondria-associated ferroptosis by inhibiting PKC α /NOX4 signaling pathway. *CNS Neuroscience & Therapeutics*. 2024;30(9):e70067.
9. Soens MA, Sesso HD, Manson JE, Fields KG, Buring JE, Lee IM, Cook NR, et al. The effect of vitamin D and omega-3 fatty acid supplementation on pain prevalence and severity in older adults: a large-scale ancillary study of the VITAL trial. *Pain*. 2024;165(3):635-643.
10. Carballo-Casla A, de Paz-Cantos S, Ortolá R, García-Esquinas E, Sotos-Prieto M, Banegas JR, Rodríguez-Artalejo F. Dietary vitamin D intake, pain incidence, and pain changes in older adults: the Seniors-ENRICA-1 cohort. *Nutrients*. 2022;14(18):3776.
11. Chen X, Shen L, Gao C, Weng R, Fan Y, Xu S, Zhang Z, Hu W. Vitamin D status and its associations with bone mineral density, bone turnover markers, and parathyroid hormone in Chinese postmenopausal women with osteopenia and osteoporosis. *Frontiers in Nutrition*. 2024;10:1307896.
12. Vilaca T, Velmurugan N, Smith C, Abrahamsen B, Eastell R. Osteomalacia as a complication of intravenous iron infusion: a systematic review of case reports. *Journal of Bone and Mineral Research*. 2022;37(6):1188-1199.
13. Jan de Beur SM, Minisola S, Xia WB, Abrahamsen B, Body JJ, Brandi ML, et al. Global guidance for the recognition, diagnosis, and management of tumor-induced osteomalacia. *Journal of Internal Medicine*. 2023;293(3):309-328.
14. Sîrbe C, Rednic S, Grama A, Pop TL. An update on the effects of vitamin D on the immune system and autoimmune diseases. *International Journal of Molecular Sciences*. 2022;23(17):9784.

15. Saedmocheshi S, Amiri E, Mehdipour A, Stefani GP. The effect of vitamin D consumption on pro-inflammatory cytokines in athletes: a systematic review of randomized controlled trials. *Sports (Basel)*. 2024;12(1):32.
16. Elliott TM, Gordon LG, Webb AR, Kift R, Foeglein A, Neale RE. Making the sunshine vitamin – how much sun exposure is needed to maintain 25-hydroxy vitamin D concentration? *Photochemistry and Photobiology*. 2024;100(3):746-755.
17. Octavius GS, Shakila A, Meliani M, Halim A. Vitamin D deficiency is a public health emergency among Indonesian children and adolescents: a systematic review and meta-analysis of prevalence. *Annals of Pediatric Endocrinology & Metabolism*. 2023;28(1):10
18. Octavius GS, Daleni VA, Angeline G, Virliani C. A systematic review and meta-analysis of prevalence of vitamin D deficiency among Indonesian pregnant women: a public health emergency. *AJOG Global Reports*. 2023;3(2):100189.
19. Cui A, Zhang T, Xiao P, Fan Z, Wang H, Zhuang Y. Global and regional prevalence of vitamin D deficiency in population-based studies from 2000 to 2022: a pooled analysis of 7.9 million participants. *Frontiers in Nutrition*. 2023;10:1070808.
20. Hayden JA, Ellis J, Ogilvie R, Malmivaara A, van Tulder MW. Exercise therapy for chronic low back pain. *Cochrane Database of Systematic Reviews*. 2021;9:CD009790.
21. George SZ, Fritz JM, Silfies SP, Schneider MJ, Beneciuk JM, Lentz TA, et al. Interventions for the management of acute and chronic low back pain: revision 2021. *Journal of Orthopaedic & Sports Physical Therapy*. 2021;51(11):CPG1-CPG60.