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Spinal cord injury in tuberculous spinal epidural abscess patient with defiiency of vitamin D: a case report with literature review

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CASE REPORT

Bali Medical Journal (*Bali MedJ*) 2022, Volume 11, Number 3: 1478-1482
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Spinal cord injury in tuberculous spinal epidural abscess patient with deficiency of vitamin D: a case report with literature review

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ABSTRACT

Background: Spinal cord injury is not only caused by trauma but also by non-trauma, such as spinal epidural abscess (SEA). Tuberculous SEA is a rare infectious disorder, a delayed diagnosis associated with morbidity and mortality. The problems of tuberculous SEA is not only infection but also neurological deficit, axial back pain, pathological fracture, deformity, and socioeconomic and psychogenic problem. There are a few cases of SEA caused by *Mycobacterium tuberculosis*, mostly caused by *Staphylococcus aureus* with decreasing body's defense mechanism caused by a deficiency of vitamin D. This study aimed to evaluate the neurological recovery of non-traumatic spinal cord injury caused by spinal tuberculosis with spinal epidural abscess and vitamin D deficiency that treated with vitamin D as an adjuvant.
Case Report: A 31-year-old female came to the orthopedic clinic with paraparesis ASIA grade C, axial back pain, and spinal deformity for three months, with vitamin D levels of 15.5 ng/ml. MRI showed epidural abscess at the level of the 10th-11th thoracic vertebrae, spondylodiscitis of the 10th-11th thoracic vertebrae accompanied by paraspinal abscess of the 8th thoracic vertebrae to the 1st lumbar vertebra. The diagnosis was confirmed by cultured and histopathological examination. The treatment was surgery, followed by anti-TB drugs and daily vitamin D 5000 IU for one year. After one year of follow-up, the patient returned to work without a neurological deficit and axial back pain.
Conclusion: This report describes the importance of early diagnosis and proper treatment of spinal cord injury in tuberculous SEA with a deficiency of vitamin D.

Keywords: *Case Report, Deficiency of vitamin D, Mycobacterium tuberculosis, Spinal Epidural Abscess*.
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INTRODUCTION

Tuberculous Spinal Epidural Abscess (SEA) is a rare infectious disorder with an incidence of 1–2/10 000 of spinal epidural space caused by *Mycobacterium tuberculosis*. The problems are delayed diagnosis and treatment, infection, spinal cord injury, axial back pain, pathological fracture, deformity, and socioeconomic and psychogenic problems.[1-3] *Staphylococcus aureus* is the most common pathogen, whereas *mycobacterium tuberculosis* is a few pathogens of SEA.[4] Most TB patient has lower nutritional status. Vitamin D is an important nutrient for maintaining bone health and a potential immunomodulator of the innate immune response that acts as a cofactor for the induction of antimicrobial activity. It is very important for the body's defense system against tuberculosis infection, especially in the action of macrophages. Vitamin D has been found to play an important role in spinal TB healing through mechanisms inducing autophagy and increases spinal cord regeneration by angiogenesis, apoptosis and neurogenesis.[5] Vitamin D deficiency can increase the risk of tuberculous infection and the risk of developing tuberculous SEA.[5] Here we report a case of spinal cord injury in tuberculous SEA caused by *Mycobacterium tuberculosis* with a deficiency of vitamin D, a 31-year-old female patient. The diagnosis was done by Magnetic Resonance Imaging (MRI), histopathological examination and microbiological investigations on the aspirated pus.[1,2,4]

CASE PRESENTATION

A 31-year-old female came to the orthopedic clinic on January 14th, 2020, with a history of axial back pain. She couldn't walk for the past three months. The patient had no history of pulmonary tuberculosis. The patient was non-HIV, non-diabetic and had no history of trauma or surgery. On physical examination, the patient was febrile, with tenderness, kyphosis, and paraplegia inferior ASIA grade C. The pulse was 110/min, B.P. 110/80 mmHg. Laboratory investigations showed Hb 9.8 g/dL, total leukocyte count of 13,700/ml, lymphocyte count 2.740/ml, and vitamin D levels 15.5 ng/ml (normal 30-100 ng/ml).

Plain X-ray of the thoracolumbar spine in lateral and AP views revealed kyphosis,

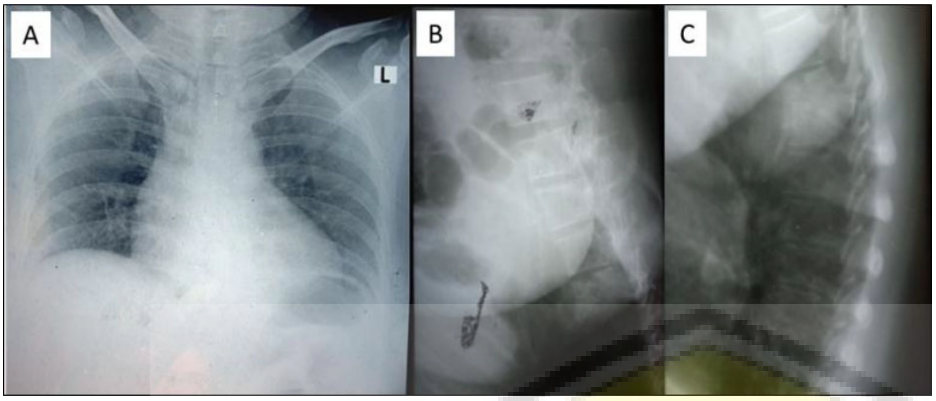

Figure 1. Plain X-ray. A. thoracic anteroposterior. B, C. Thoracolumbal spine in lateral view.

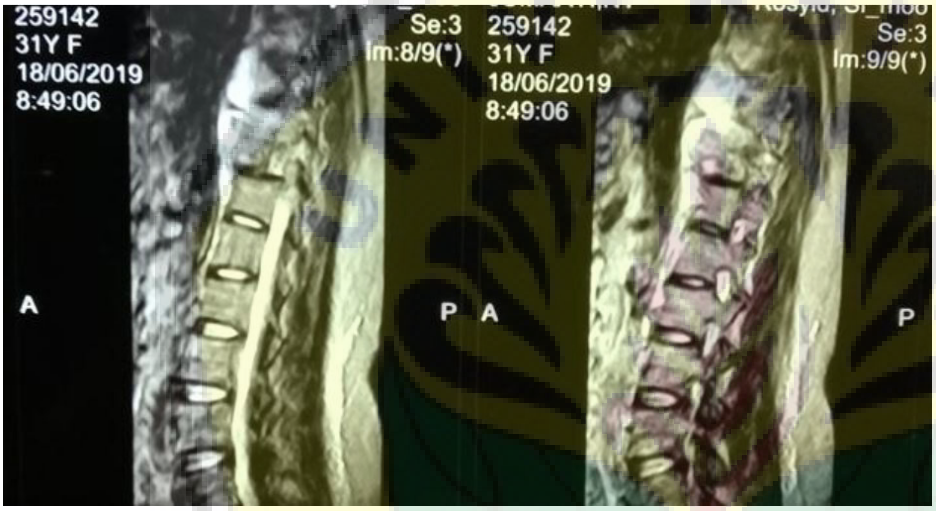

Figure 2. MRI of Thoracolumbal (sagittal view).

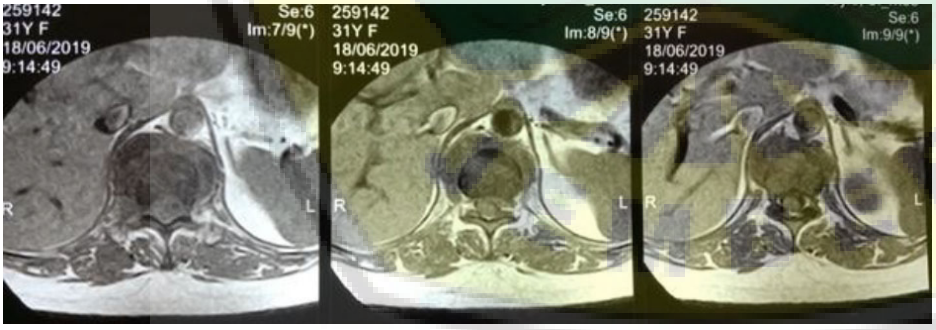

Figure 3. MRI of Thoracolumbal (coronal view).

destruction of bone, and compression fracture of spine 10th-11th (Figure 1), while chest x-ray was within normal limits (Figure 1A). MRI of the thoracolumbar spine of sagittal and coronal position showed epidural abscess at the level of the 10th-11th thoracic vertebrae compressing the spinal cord, spondylodiscitis of the 10th-11th thoracic vertebrae accompanied by subligamentous spreading and paraspinal abscess of the 8th thoracic vertebrae to the 1st lumbar vertebra (Figure 2 and 3).

The patient underwent circumferential decompression and fusion surgery. Surgical findings were compression of the spinal cord by abscess and bone fragments, granulation tissue and pus in the epidural space. The granulation tissue with a volume of 0.5 cc in white and gray irregular shapes was sent to the histopathology laboratory for identification. It was identified as a granulation tissue consisting of extensive caseous necrosis with several granulosa, histiocyte, epithelioid cells with an oval nucleus, lymphocytes, and multinucleated giant cell Langerhans as chronic granulomatous inflammatory (Figure 4). After three weeks of incubation, microbiological examinations of the pus using acid-fast bacilli in Ziehl-Neelsen staining and culture on Lowenstein Jensen's (LJ) media revealed the buff and tuff colonies of M. tuberculosis (Figure 5).

The patient has not had any treatment before for this disease. The patient's poor socioeconomic status made the patient not come to the physician. Though the late treatment from the onset of disease. The patient was started on Category I: Directly Observed Treatment Short Course (DOTS) therapy, i.e., 2H3R3Z3E3 (H: Isoniazid (600 mg), R: Rifampicin (450 mg), Z: Pyrazinamide (1500 mg), Ethambutol (1200 mg) for two months), followed by 4H3R3 (H: Isoniazid (600 mg), R: Rifampicin (450 mg)), for 10 months. A daily vitamin D intake of 5000 IU was consumed for one year. The patient started therapy on May 23rd, 2020 and responded well to the treatment when the last follow-up was taken one year after treatment (May 23rd, 2021). The patient returned to work, with complete recovery of the power of the lower limbs, without residual neurological deficits, axial pain, and spine deformity (Figure 6).

DISCUSSION

Vitamin D as a fat-soluble micronutrient is important for calcium and phosphate balance, vascular health, cell differentiation, and proliferation. The traditional role of vitamin D is to keep the balance of calcium and phosphate metabolism in order to support osteoblast control and maintain bone formation. Vitamin D's non-classical functions include inhibiting the proliferation of cancer cells, regulating cell death by preventing cell hyperplasia, and acting as an immune system immunomodulator.[5–7]

When the skin is not exposed to sunlight and is not receiving enough vitamin D from food sources, vitamin D insufficiency develops. Vitamin D insufficiency in this patient is thought to be brought on by inadequate nutrition and

sun exposure. The enzyme 25-hydroxylase in the endoplasmic reticulum of the liver converts sunlight (vitamin D3) and the vitamins D2 and D3 found in diet into serum 25(OH)D. In the proximal tubule of the kidney, 25 (OH) D is hydroxylated to 1,25 (OH) 2D. Since the active form of vitamin D is spread throughout the body, the amount of vitamin D 25-OH in the blood provides the most precise indication of the body's vitamin D status. Serum vitamin D 25-OH levels between 30 and 100 ng/ml are considered acceptable, while levels between 21 and 29 ng/ml indicate insufficiency and toxicity if they exceed 150 ng/ml.[5]

Vitamin D is an immunomodulator by lowering levels of inflammatory proteins and increasing antimicrobial, which destroys *Mycobacterium tuberculosis*. The 1,25(OH)2D form is an active metabolite of vitamin D that can affect the phagocytic ability of macrophages to suppress intracellular tuberculous growth after binding to the vitamin D receptor (RVD) expressed in macrophages. If there is a deficiency of 1,25 (OH) 2D serum levels, the ability to phagocytose macrophages will decrease so that tuberculous infection can develop into tuberculous disease.[5-7]

There is a ton of research demonstrating the link between Vitamin D deficiency and tuberculosis.[5-7] Vitamin D3 supplementation, administered daily and consistently for six months, increases the expression of VDR during transcription and enhances the efficacy of anti-TB medication therapy.[6] The mechanisms of vitamin D in controlling autophagy are stated by activation of VDR in the expression of cathelicidin and increasing nitric oxide (NO). Cathelicidin also requires Vitamin D (the Ca2+ mobilizing agent) to reach the antigen of MTB in immature macrophages and

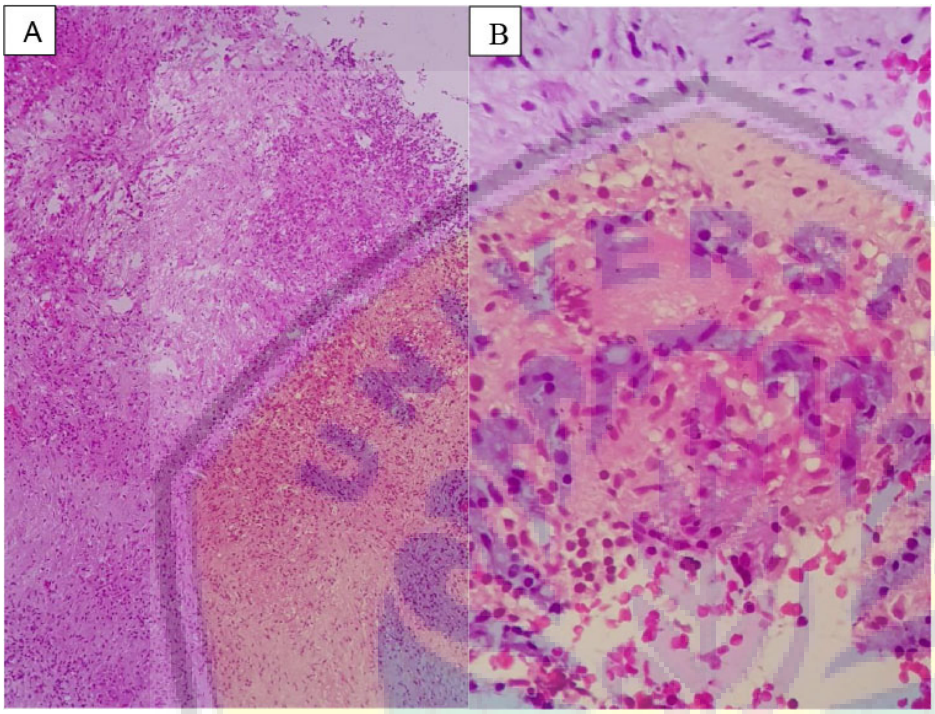

Figure 4. Histopathological observations support the diagnosis of *M. tuberculosis* with the presence of granulation tissue, caseous necrosis, and multinucleated Langerhans giant cells. Observation with a light microscope objective magnification 10x, 40x.

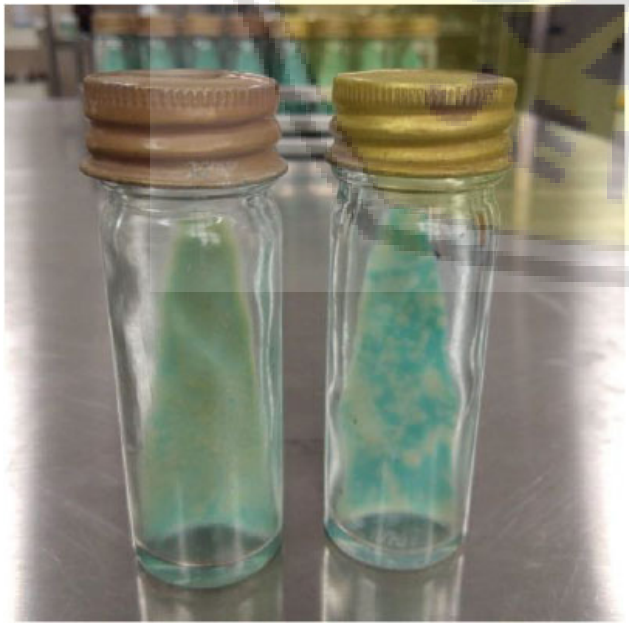

Figure 5. Lowenstein Jensen's media showed colonies of *M. Tuberculosis*

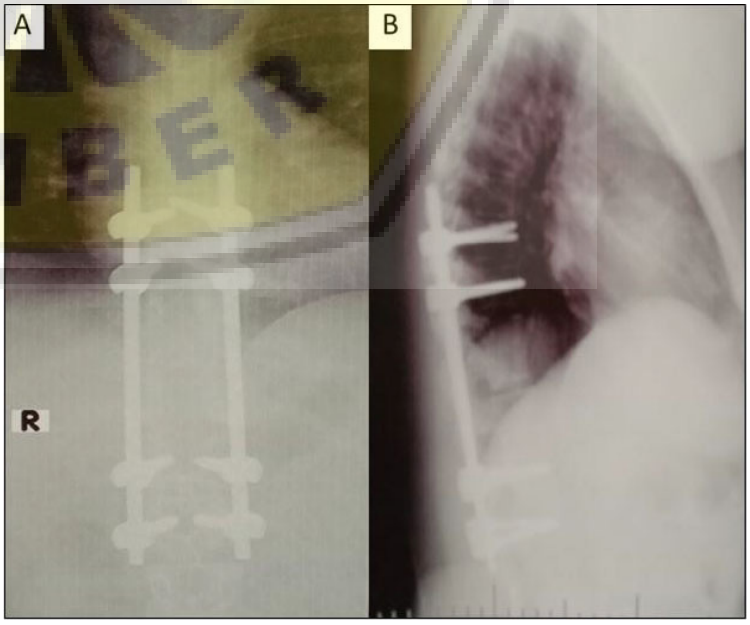

Figure 6. Evaluation after one-year treatment.

the help of Ca2+/calmodulin-dependent kinase (CaMKK)-β.[6,7] By raising the activity of nitric oxide synthase, which causes alterations in the mitochondrial membrane and releases of cytochrome C, vitamin D heightens apoptosis.[7]

By raising vascular endothelial growth factor (VEGF) expression and release in vascular smooth muscle cells (VSMC) in vitro by direct binding of the VDR, a transcription factor, to the VEGF promoter, the active form of vitamin D also enhances angiogenesis.[6] Vitamin D deficiency might affect the expansion of immature neurons, differentiation, and protein expression within neurons, decreased glutamate and glutamine levels and expanded Gamma-Amino Butyric Acid (GABA) levels neurogenesis. This mechanism explains that supplementation of vitamin D might enhance the neurogenesis process.[7] In this case, report, after daily vitamin D treatment, the patient gets normal recovery from SEA and spine TB.

Following tuberculous spondylitis, diabetes mellitus, alcoholism, intravenous drug addiction, and HIV/AIDS are the most often occurring probable SEA causes. Age, early diagnosis, and the extent of neurological involvement are the three main prognostic markers in SEA patients.[8] Tuberculous spondylitis accounts for less than 1% of all tuberculosis infections, the extreme form of skeletal tuberculous is considered, with potential neurological symptoms due to compression of the neural structures by abscess, bone fragments, fibrosis, and vascular problem.[8,9] It can also contribute to spinal deformity, substantial destruction, and instability of the vertebral structure.[10,11]

Tuberculous SEA is a rare disease with an incidence of 1-2 per 10,000,[11,12] late complication of tuberculous spondylitis, compressing the spinal cord result in spinal cord injury.[1,2,9] The tuberculous SEA has been reported in developing countries associated with deficiency of vitamin D, diabetes mellitus, and HIV/AIDS.[3] The extradural space has affluent blood flow, most commonly, hematogenous infections occur via hematogenous spread from urinary tract infection, soft tissue infection, respiratory tract infections, or through the direct extension of infected tissue.[3] Any level of the spine, including the lumbar and cervical spine, may be affected by tuberculous spondylitis, though the multilevel lower thoracic spine region is where it is most frequently found. The disease typically spreads via the hematogenous route or direct extension from the paraspinal to the spine. As in our patient, noncontiguous infection sites as well as three adjacent vertebral bodies may be affected.[13]

Staphylococcus aureus is the most typical cause of SEA. Mycobacterium tuberculosis is just the root cause of 2% of these illnesses. Mycobacterium TB enters the spinal canal through the epidural space and seeds the sterile region between the dura mater and vertebral wall, causing a suppurative infection in this small space. Pyogenic infection and abscess formation happen after bacteria penetrate the epidural space.[3,13] The following mechanisms contribute to the spinal cord injury in tuberculous SEA: I direct abscess compression, (ii) thrombosis and thrombophlebitis of surrounding veins, (iii) disruption of arterial blood flow, and (iv) inflammation brought on by bacterial toxins and mediators.[13-15]

About 9% of patients with SEA had two or more noncontiguous patches of pyogenic collections, despite the fact that hematogenous spreading is the most common cause of SEA.[15,16] As our patient showed tuberculous spondylodiscitis of the 10th-11th thoracic vertebrae spine accompanied by epidural abscess at the level of the 10th-11th thoracic vertebrae, subligamentous spreading, and paraspinal abscess of the 8th thoracic vertebrae to the 1st lumbar vertebra spine.

Spinal cord injury in tuberculous SEA requires urgent surgical intervention, including circumferential decompression and fusion, followed by anti-TB drugs as the treatment choice.[17–19] Vitamin D deficiency requires daily administration of oral vitamin D to improve the immune system.[8] The length and initial severity of the neurologic deficit are correlated with the extent of neurologic recovery following surgery. The mortality rate of this case is around 2%-20%. Failure of early diagnosis and treatment caused by the clinical presentation of the tuberculous SEA is more insidious than tuberculous spondylitis.[17–19]

CONCLUSION

Spinal cord injury due to compression of the neural structures by tuberculous of SEA is a severe form of the tuberculous spine associated with deficiency of vitamin D. Case report describe the importance of early diagnosis and proper treatment, especially vitamin D administration as an adjuvant of surgery and oral anti TB drugs to get an excellent result.

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ETHICAL STATEMENT

Informed written consent was obtained from the patient for the publication of this report and any accompanying images.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

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AUTHOR CONTRIBUTIONS

Conceptualization: INS. Visualization: MYN. Writing - original draft: INS, AW, MYN. Writing - review & editing: NNJ, AP.

CARE CHECKLIST (2016) STATEMENT

The authors have read the CARE Checklist (2016), and the manuscript was prepared and revised according to the CARE Checklist (2016)

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