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Case Report

Clinical manifestations and prognosis of tuberculous spondylitis in an adolescent with disseminated tuberculosis: a case report

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Indonesia is one of the countries with the highest number of tuberculosis (TB) cases globally. Around 10-20% of adolescents with TB infection progress to pulmonary TB, and less than 0.5% develop miliary or central nervous system TB. TB spondylitis occurs in only 5.6% of extrapulmonary TB patients. The clinical manifestations of disseminated and TB spondylitis are heterogeneous and insidious, with several potential risk- and prognostic factors. We report the case of a 16-year-old male admitted with abdominal distension, paraplegia, and urinary retention. He was diagnosed with disseminated TB with TB spondylitis. This case was unique because the patient had no classic symptoms of pulmonary TB. This report focuses on the diagnosis, comprehensive management, and prognosis of TB spondylitis, as well as the risk factors for disseminated TB. The management consisted of antituberculous agents and surgery. The prognosis is influenced by the patient's age, severity of kyphosis deformity, number of vertebrae involved, lesion site, and patient's health status, including nutritional status. **[Paediatr Indones. 2024;64:176-83; DOI: 10.14238/pi64.2.2024.176-83**].

Keywords: *adolescent; tuberculosis; spondylitis; risk factors; therapy; prognosis*

Indonesia is one of the countries with the highest tuberculosis (TB) burdens in the world, with 9% of the TB cases occurring in the children.[1] Commonly, TB infection in adolescents will only be a latent infection.[2] Around 10-20% of adolescents with TB infection will develop pulmonary TB, and less than 0.5% become miliary or central nervous system (CNS) TB.2 Besides pulmonary TB, disseminated TB is also caused by *Mycobacterium tuberculosis* infection that spreads from the blood into two or more non-contiguous sites after primary infection or reactivation of the dormant bacteria.[3] This condition is considered a significant cause of morbidity and mortality for adolescents in developing countries.[3] It can involve multiple extrapulmonary organs, such as the pleura (TB pleuritis), the meninges (TB meningitis), genitourinary organs (genitourinary TB), lymph nodes (peripheral lymphadenopathy), bones and joints (osteoarticular TB), the skin (scrofuloderma), and the peritoneum (TB peritonitis).[1,4]

Osteoarticular TB accounts for 10% of all extrapulmonary TB cases.[5] Spinal involvement in TB spondylitis is usually caused by the hematogenous spread of *M. tuberculosis* from a primary source into the dense vasculature of the cancellous bone of the vertebral bodies.[6] The incidence of TB spondylitis in children varies from 58% in Korea, 33% in India, and 26% in Hong Kong.[7] The clinical manifestations of TB spondylitis are fever, back pain, paraparesis, disturbance of sensory function, and autonomic dysfunction (bowel and bladder dysfunction).[8] The clinical manifestations of disseminated TB and TB

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spondylitis are heterogeneous and insidious, with several potential risk factors and prognostic factors. Nowadays, diagnosis and management of disseminated TB and TB spondylitis remain challenging. This case report highlights the diagnosis, comprehensive management, and prognosis of TB spondylitis, as well as the risk factors for developing disseminated TB.

The case

A 16-year-old male was admitted to Cipto Mangunkusumo Hospital with a two-week history of abdominal distension and back pain. His legs were paralyzed and numb, and he was unable to urinate. He had enlarged lymph nodes on his neck that had ruptured five months ago, had mild pain and curvature of the backbone, and had lost 10 kg of weight in the last 3 months.

He had been vaccinated with the BCG vaccine. His history of growth, development, and puberty was normal. He had no close contact with a TB patient and no history of smoking, allergy, alcohol consumption, or substance use. He had dropped out of school and worked in the market while frequently sleeping there.

On physical examination, he appeared moderately ill. His *Glasgow Coma Scale* was E4M6V5 with normal vital signs. His body weight was 45 kg, and his height was 160 cm, consistent with severe malnutrition with normal stature. He had three palpable masses on the neck, each 3.5 x 3.5 cm in size, with multiple ulcerations, necrotic patches and plaques, irregular skin border, and bridging skin or scrofuloderma (**Figure 1**). His tuberculin test was 12 mm. He had abdominal distension in the suprapubic region with a palpable pain mass (15 x 15 cm) without referred pain. There was no chessboard phenomenon or organomegaly. He showed cachexia, severe muscle wasting, and prominent ribs.

A gibbus deformity was observed in the vertebrae. He had no abnormalities of the cranial nerves; upper extremity muscle strength was normal. He had weakness of both lower limbs with motor strength of 2222/2222, abnormal sensibility up to the level of the L1-L2 vertebrae, increased physiological reflexes, negative clonus, and negative pathological reflexes.

Laboratory examination showed anemia (hemoglobin 10.5 g/dL), leukocytosis (16,030/mm^3) with lymphocytosis (87.7%), elevated ureum level (175.4 mg/dL), elevated serum creatinine level (3.4 mg/dL), hyponatremia (126 mEq/L), hyperkalemia (6.6 mEq/L), elevated C-reactive protein (17.3), and erythrocytes in urinalysis (>50 pg/dL). HIV screening was non-reactive. The result of the rapid polymerase chain reaction (PCR) test for M. tuberculosis was positive only for sputum. Acid-fast bacilli (AFB)

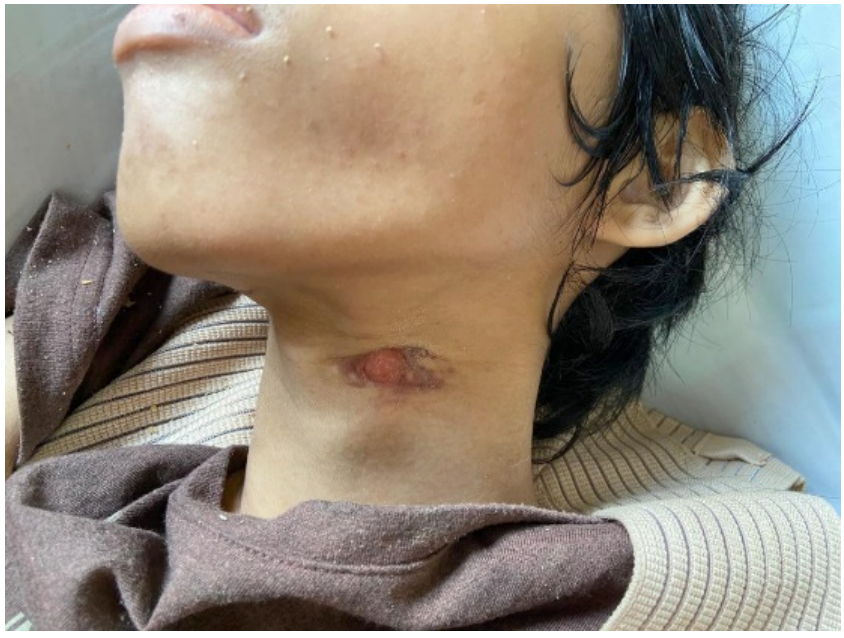

Figure 1. The ulcerated lesion on the neck

smears of sputum, urine, and metatarsal abscess were negative. No AFB culture was performed. Chest X-ray showed bilateral and paracardial reticulonodular infiltrates.

Gadolinium-enhanced thoracic magnetic resonance imaging (MRI) (**Figure 2**) showed destruction of the T3-T4 vertebral bodies, resulting in kyphotic deformity and severe spinal cord compression with anterolateral paravertebral abscess formation at the level of T2-T5. There was also destruction and fusion of the T10-T11 vertebral bodies with moderate spondylolisthesis of T10, resulting in kyphotic deformity and severe spinal cord compression with anterolateral paravertebral abscess formation at the level of T9-T12.

Histopathology examination of the skin lesion showed epitheloid granuloma, lymphocytes, plasma cells, and Langerhans cell histiocytosis, corresponding with scrofuloderma. Abdominal ultrasonography revealed overdistension of the bladder to the upper abdominal region, suspected as neurogenic bladder and bilateral grade II hydronephrosis with bilateral proximal hydroureter. A urinary catheter was placed to facilitate micturition.

Contrast brain computed tomography (CT) scan showed multiple small isodense lesions in the right lentiform nucleus, right internal capsule, and right cerebellum, suggestive of tuberculoma (**Figure 3**).

On day 9 of hospitalization, soft tissue swelling was observed in the left dorsum of the foot. Findings on anterolateral foot X-ray showed osteomyelitis and abscess at the first metatarsal with subcutaneous emphysema (**Figure 4**). Pseudomonas aeruginosa was grown on culture of the pus from his skin lesion.

On day 24 of hospitalization, a decubitus ulcer developed on the sacrum region. A plastic surgery consult was ordered, and the patient was diagnosed with grade III pressure injury on the sacrum and medial of the left plantar pedis.

After 24 days of hospitalization, the established diagnoses were TB spondylitis with compression fractures of thoracic vertebrae 3-4 and 10-11, scrofuloderma, multiple brain tuberculoma, and pulmonary TB, as part of disseminated TB, neurogenic bladder, acute kidney injury, grade II bilateral hydronephrosis with hydroureter, osteomyelitis of the first left metatarsal, grade III pressure injury in the sacrum region and left medial plantar pedis, and severe malnutrition with normal stature.

His pharmacotherapy included 3 adult fixed-dose combinations of anti-TB tablets once a day (rifampicin 150 mg, isoniazid 75 mg, pirazimamide 400 mg, and ethambutol 275 mg), 10 mg of methylprednisolone IV four times a day (1 mg/kgBW/day), ampicillin-

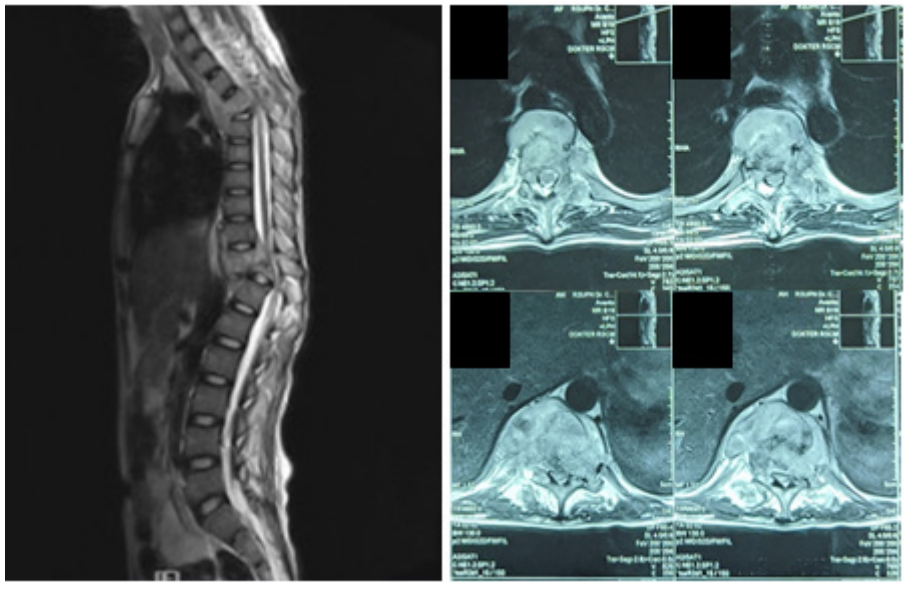

Figure 2. Spinal MRI results: (a) axial T2WI MRI showing paravertebral abscess formation and nerve compression; (b) sagittal T2WI MRI depicting two compression sites (red arrows).
MRI=magnetic resonance imaging; T2WI=T2-weighted imaging

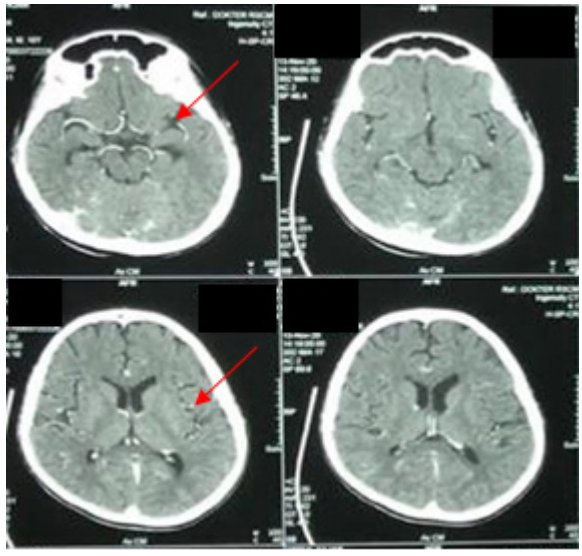

Figure 3. Contrast brain CT scan showing multiple small isodense lesions on the right lentiform nucleus, right internal capsule, and right cerebellum (red arrows)

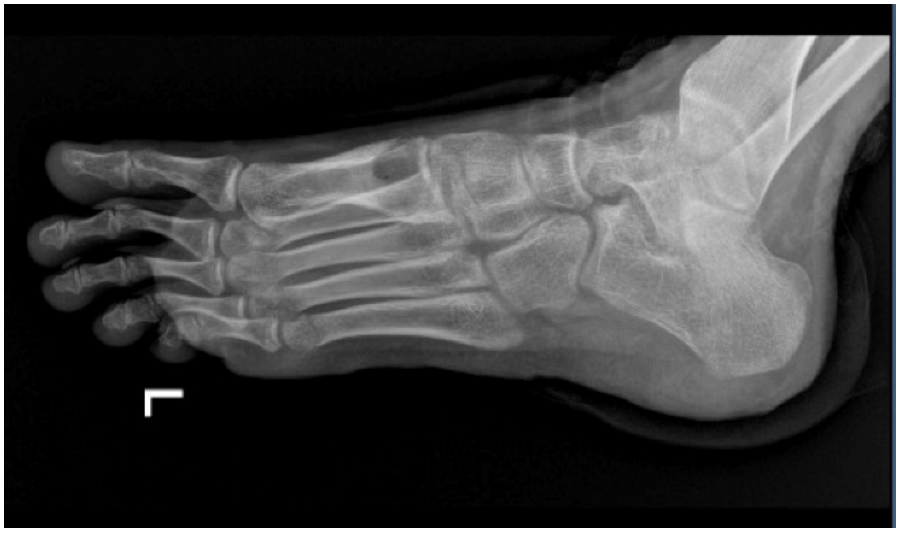

Figure 4. Anterolateral left foot X-ray showing osteomyelitis and abscess at the first metatarsal

sulbactam IV 2 g per 6 hours (200 mg/kgBW/day), 10 mg of vitamin B6 once a day, and 1 mg of folic acid once a day. His non-pharmacological therapies included a diet of 1,500 kcal, 250 mL of high-calorie milk 1-2 times a day, urinary catheterization, back slab application, wound toilet (normal saline, vaseline album, and fusidic acid), physiotherapy, regular mobilization, and placement on a decubitus bed.

On day 38 of hospitalization, he underwent nerve decompression, posterior instrumented fusion, and kyphotic deformity in a staged fashion. We exposed the lower thoracic vertebrae for the first surgery and inserted pedicle screws bilaterally on T8, T9, T12, and L1. The orthopedic team performed nerve decompression by laminectomy on T10 and T11 (**Figure 5**), ensuring no thecal sac compression occurred. Furthermore, a Smith-Petersen osteotomy was done to correct the deformity and posterolateral fusion. No mycobacterial specimen were collected for culture during the first surgery. He was discharged after 47 days of hospitalization and was followed up in the outpatient clinic to continue the anti-TB agent, next surgery preparation, and regular rehabilitation.

He was discharged after the first surgery

and underwent the second surgery within three months using a similar procedure. He underwent total treatment with VI posterior debridement, decompression laminectomy, posterior stabilization of T2, T3-5, T6, and deformity correction. There was no complication after the surgery. His body weight increased to 52 kg after four months of anti-TB treatment, and he no longer had bladder dysfunction. After the second surgery, he was still in a wheelchair with paraplegia, and his motor strength increased to 3333/3333.

Discussion

Disseminated TB is the wide spread of M. tuberculosis through the blood.[4] It is affected by several factors, including the patient's adolescent age, the reactivation process of TB, and complications of primary TB infection. Disseminated TB is rare but commonly occurs in children younger than 2-3 years or those in an immunocompromised state.[9] Disseminated TB affects less than 2% of immunocompetent TB patients.[4] The bacteria are disseminated into distant organs such as the pulmonary apex, liver, spleen, skin, and others through lymphohematogenous spreading.[10]

Differing from past studies on disseminated TB,4,5 our case is unique as the patient was an adolescent with disseminated TB affecting the lungs (pulmonary TB), vertebrae (TB spondylitis), skin (scrofuloderma or cutaneous TB), and brain (multiple brain tuberculomas).

Considering the potential of TB disease in adolescents, the epidemiological data described that 80-90% of TB in the pediatric population would not progress to TB disease if the primary infection occurred over the age of 10 years (adolescent period).[2,11] Only 10-20% of these adolescents develop pulmonary TB, and less than 0.5% of them develop miliary disease or CNS TB.2,11 The common cause of disseminated TB is a complication from a primary infection that usually occurs within 2-6 months after the initial infection.[10] This condition usually occurs in infants and young children.[10]

Another possible cause of disemminated TB is the reactivation of latent TB infection.[10] Several risk factors for dormant bacteria reactivation in patients with latent TB include HIV infection, close contact with TB patients, organ transplantation recipients, chronic renal failure requiring dialysis, anti-tumor necrosis factors (anti-tumor necrosis factor) use, corticosteroid therapy, underweight, smoking, and others.[12] Immunocompromised states, such as an individual's health status (autoimmune disease, malnutrition, and liver or kidney disease), immunosuppressive substances use, or HIV infection, increase the risk of infection and disease, mainly disseminated disease with extrapulmonary TB.[13] It is unlikely that our patient's risk of disseminated TB was associated with reactivation of dormant

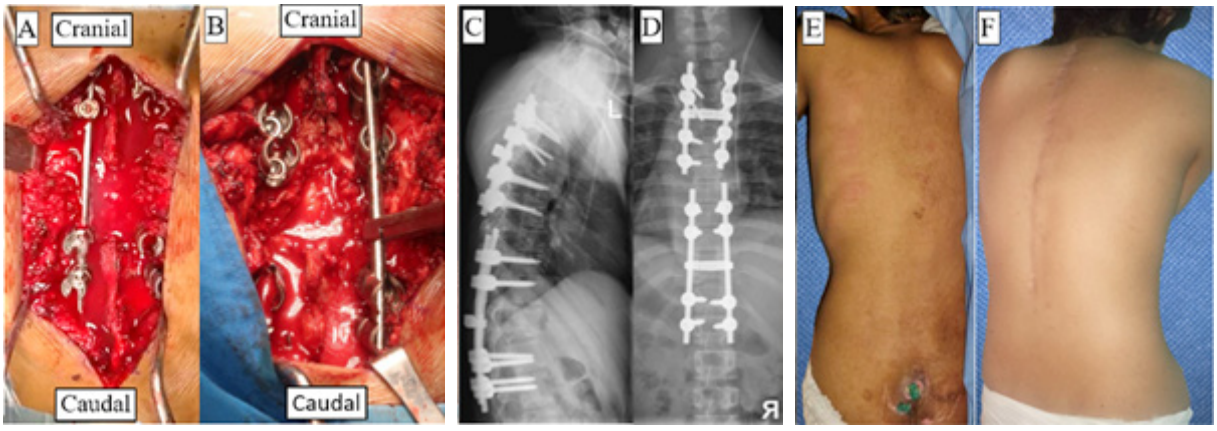

Figure 5. Sequences of (a) intraoperative image from the first surgery depicting granulation tissue material from the anterior vertebral body; (b) intraoperative pictures from the second surgery on upper thoracic vertebrae depicting laminectomy on T3 and T4 Vertebrae; (c and d) postoperative images after the second surgery were completed; (e and f) comparison of the preoperative and postoperative clinical conditions showing excellent scar healing and deformity improvement.

bacteria. He had no family history of TB, smoking history, autoimmune disease, corticosteroid or other immunosuppressant agent use, or HIV infection. Severely malnourishment occurred after the emergence of palpable masses on his neck. Therefore, malnutrition was not the risk factor for the reactivation of TB in this case. Thus, disseminated TB in our patient might have been caused by primary TB. The probability of TB infection increased as he lived and worked in the traditional market with lack of sanitation and potential contact with individuals with pulmonary TB.

This case had interesting clinical manifestations of disseminated TB. Disseminated TB is a miliary disease caused by the release of massive M. tuberculosis into the bloodstream and affecting two or more organs.[10] The organs that typically exhibit greater numbers and extent of lesions than other organs are the lung, spleen, liver, and bone marrow.[10] Disseminated TB mostly develops systemic symptoms (such as anorexia, weight loss, and low-grade fever) and rarely develops respiratory symptoms (dyspnea and cough).[10] The physical signs are generalized lymphadenopathy, organomegaly, and rales or wheezing.[10] Different from the literature, the clinical manifestations of disseminated TB in this case are localized lymphadenopathy, while the site of infection are vertebrae (bone) and the brain. We did not explore the possibility of multi-drug resistant (MDR) TB or underlying fungal infection.

A previous study found that TB spondylitis only affected four of 72 pediatric subjects with extrathoracic TB (5.6%).[14] TB spondylitis causes bone destruction, spinal deformities, and neural complications. *M. tuberculosis* spreads through the ending arterioles in the vertebral body, located adjacent to the anterior of the vertebral endplate. Therefore, the clinical manifestation of TB spondylitis commonly involves the anterior part of the vertebral body. The extension of TB infection will disrupt the cortex, spreading to the anterior longitudinal ligaments and the periosteum of the vertebral bodies. TB infection stops the blood supply to the vertebrae and gradually destroys the avascular intervertebral disc, which results in disc space narrowing and spondylodiscitis. Severe vertebral destruction may occur in the pediatric population because most children have cartilaginous bones. The deformity (angulation) of the vertebrae is also more significant in children than in adults due to the restricted growth of the anterior column, which is in line with the unlimited growth of the posterior column.[8]

A study involving 37 children aged 4 to 15-years with TB spondylitis found that TB spondylitis symptoms included systemic symptoms (fever, night sweats, anorexia, and weight loss) and neurological symptoms (back pain, fever, bilateral weakness of lower extremities or four extremities, and bowel and bladder dysfunction).[8] A review article also described that the clinical manifestations of TB spondylitis included fever of unknown origin (for three months), painless superficial lymphadenopathy, cough for more than 30 days, recurrent diarrhea that was not resolved by therapy, mass in the abdomen, signs of fluid accumulation in the abdomen, deformity of vertebrae (kyphosis) in 80% of cases caused by the formation of gibbus, paraplegia in the early onset or even in the healed patient (late-onset), and cold abscess.[6]

In line with previous studies, our patient first presented with lymphadenopathy in the neck, followed by systemic symptoms such as fever and body weight loss. The deformity of vertebrae occurred after 3 months of the onset of lymphadenopathy. Paraplegia occurred 2 months after the onset of the vertebrae deformity. The deformity and diminished motoric functions were followed by sensory and autonomic dysfunction (urination and defecation), and abdominal distention. The vertebrae X-ray and MRI with contrast found destruction of vertebrae thoracal 3-4 and vertebrae thoracal 10-11. These findings align with the literature, which depicts what was known as atypical spondylitis tuberculosis in which non-contiguous levels were affected by the disease.[16] In contrast, our patient did not develop respiratory symptoms such as cough and dyspnea. The literature describes that the anterior aspect of the vertebral body is the common area of spondylitis lesions, while the MRI shows that the lesions also involve the lateral portion of the vertebrae. However, extension of the TB infection to the soft tissue was not observed in this patient.

Several problems occur during hospitalizations, such as the presence of hematuria, which later found bilateral nephrolithiasis, soft tissue swelling of the left dorsalis pedis, osteomyelitis, and an abscess at the first metatarsal with subcutaneous

emphysema because of *Pseudomonas aeruginosa*, decubital ulcer of the sacrum, and several psychiatric problems, including loss of attention, hypothymic for medication, and rehabilitation. Therefore, we performed several additional supportive examinations such as genitourinary ultrasound, skin biopsy, and foot X-ray. These several problems resulted in longer hospitalizations and delayed the definitive correction for kyphotic deformity.

The treatment principles for TB spondylitis are anti-TB drugs for 9-12 months, spinal immobilization, and surgery if needed.[6] The patient took four regiments of antituberculous agent and surgery for debridement, nerve decompression, and kyphotic deformity. After the TB lesion is healed, the vertebral deformity may improve or worsen as the patient grows.7 Therefore, the patients should be monitored and evaluated every year to ensure the condition of kyphosis until the final maturation of the bone.[7] The rate of deformity progress depends on the severity of kyphosis deformity, the level of lesion, and the patient's age before the initial treatment.[7] Severe kyphotic deformity commonly occurs in children under 10-year-old with more than three vertebral bodies involvement and localization of the lesion in the thoracic vertebrae.[8]

A previous study reported the prognosis of vertebrae deformity in children with TB spondylitis after the disease healed.[7] Vertebrae deformity worsened in 39% of the patients and improved spontaneously in 44% of patients. Meanwhile, 17% of the patients still had the same degree of vertebrae deformity before initial treatment. The prognosis was worse in patients aged under 7 years, with the involvement of three or more dorsal or dorsolumbar vertebrae. A persistent vertebrae deformity causes biomechanical stresses on the proximal or distal vertebrae and spinal cord. The patient will develop severe canal stenosis and late-onset paraplegia. In addition, severe deformity of the vertebrae will cause costo-pelvic pain, resulting in disturbance of cardiorespiratory regulation as the worst effect.

Two previous studies showed that kyphotic deformity could be corrected via anterior, posterior, or a combination thereof, which described debridement, abscess drainage, nerve decompression, and fusion as the surgical treatment of choice.[17,18] Our patient had more than three vertebral body involvements and a TB lesion at the thoracic vertebrae. These two risk factors induce severe kyphotic deformity with severe spinal cord compression. The spinal cord compression causes paraplegia symptoms, disturbance of sensory function, and urinary incontinence. Furthermore, toppling one vertebral body over other vertebrae (**Figure 2**, yellow arrow) indicated severe deformity potential if a surgical correction was not performed.

Resolution of paraplegia and other neurologic symptoms is also the goal of the therapeutic targets in TB spondylitis management. Anti-TB drugs can heal lesions and recover the neural components in paraplegia and bowel and bladder dysfunction. It can also improve the paraplegia symptoms. Decompressive surgery is often reserved for those who fail to recover after 3-4 weeks of anti-TB drug consumption or develop a neurological deficit with conservative treatment.

However, paraplegia can occur a few years after the TB lesion has healed, termed as late-onset paraplegia. After 10 years of successful treatment, patients with a history of severe kyphotic deformity can gradually develop upper motor neuron lesions. This neural deficit is caused by the reactivation of TB adjacent to the apex of kyphosis deformity. Late-onset paraplegia is also caused by the progression of kyphosis deformity with increased internal bulging that causes a stretched spinal cord.[7]

Our patient's TB prognosis was influenced by his nutritional status, which was severely malnourished. A cohort study in 456 pulmonary TB patients, found that those who were underweight at the baseline had a higher risk of anti-TB treatment failure after two months.[20] We provided adequate nutrition with a high-calorie diet during and after hospitalizations.

After 4 months of therapy, our patient showed a good response to the anti-TB drugs and two-stage surgery. He gained weight, improved motor strength, and had no complaints of bladder dysfunction. Tight monitoring of anti-TB drug consumption, improvement of nutritional status, and routine control of the medical rehabilitation specialist and orthopedic specialist for the next vertebrae deformity repair surgery while monitoring the possibility of kyphosis deformity progression and late-onset paraplegia are also needed.

In conclusion, disseminated and TB spondylitis are rare yet crucial cases in the adolescent population,

which are commonly caused by TB primary infection. Several variations in systemic and neurologic symptoms have been found in TB spondylitis patients. The prognosis of TB spondylitis is influenced by the patient's age, severity of kyphosis deformity, amount of vertebrae involvement, site of the lesion, and patient's health status, such as nutritional status and other comorbidities.

Diagnosis of TB spondylitis in this patient was developed from clinical manifestations (severe malnutrition, multiple lymphadenopathy, sclofuroderma, gibbus, paraplegia and numbness, disruption of urination, and characteristic lesion of upper motor neuron), and his laboratory examinations showed: positive M. tuberculosis PCR, positive Mantoux test, skin biopsy, brain CT scan (tuberculoma) and vertebrae MRI (abscess formation at the paravertebral anterolateral) although the TB contact was not clear. Elaborating the medical history and characteristics of extrapulmonary TB such as sclofuroderma and gibbus, the possibility of MDR-TB, and screening for possible TB in the family are essential to prevent the spread of TB.

Conflict of interest

None declared.

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