

Case Report

Tuberculous Spondylitis in Elderly with Polyneuropathy

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Abstract

Background Tuberculous (TB) spondylitis or Pott's Disease is a manifestation of extrapulmonary tuberculosis infection caused by the hematogenous and lymphomatous spread of Mycobacterium Tuberculosis, causing infection in the vertebral bones. Approximately 10% of extrapulmonary TB cases are TB spondylitis. Pulmonary and extrapulmonary tuberculosis cases are considerable cases in developing countries. Indonesia is a country with the third-highest prevalence of TB in the world after China and India.

Objective The aim of this study was to report a case of TB spondylitis in a 66 years old woman with polyneuropathy at Bethesda Hospital Yogyakarta.

Case description A 66 years old female patient presented with complaints of pain and difficulty in moving the left lower limb 3 months ago. The patient had a history of hypertension 5 years ago, while the family history was not specific. The patient was generally in pain with full awareness and cooperation. Vital sign examination showed blood pressure 140/80 mmHg, pulse 82 beats per minute, and respiratory rate of 22 breaths per minute. The lumbosacral-MRI examination was performed without contrast material and L2-L5 TB Spondylitis, with L2-L3 Disc protrusion, was found.

Conclusion Tuberculous spondylitis (TB) may cause paradiscal inflammation, edema, and progressive bone destruction due to lysis of anterior bone tissue and secondary ischemia which will lead to collapse. Pain, difficulty moving, and local swelling are the most common complaints. Abnormalities in the curvature, misalignment of the vertebrae, deformity and bone destruction might be found in MRI radiological imaging. Treatment of TB spondylitis prioritizes administering of anti-TB drugs combined with immobilization using a girdle. The prognosis for TB spondylitis varies depending on the clinical manifestations.

Keywords: Tuberculosis Spondylitis, Potts Disease, Anti-Tuberculosis Medicine.

INTRODUCTION

Tuberculosis is a chronic infection caused by *Mycobacterium tuberculosis*, a rod-shaped bacterium with a length of 1-4/μm and a thickness of 0.3-0.6/μm. These bacteria have a wall composed of fatty acids (lipids), peptidoglycan, and arabinomannan. The lipids make bacteria acid-resistant. In tissue, the bacteria live as intracellular parasites in the cytoplasm of macrophages, since macrophages contain a lot of lipids. *Mycobacterium tuberculosis* is also an aerobic bacterium, so this bacterium has a predilection for oxygen-rich areas such as the brain (TB meningitis), bones (TB spondylitis), kidneys, and the lungs themselves, especially the apex of the lung or the upper lobe of the lung.¹ Tuberculosis infection of the vertebrae can be caused by disguised hematogenous spread, where the course of infection may spread mainly through arteries and veins, as well as additional routes. In the main route, the spread takes place systemically along arteries entering the vertebral body, via the lumbar segmental arteries that vascularize the respective half of the body. Each body is nourished by 4 arteries. The corpus is vascularized by the end arteries hence the

extent of the vertebral body infection frequently begins in the paradiscal area. Furthermore, the disguised hematogenic spread can also be through Batson's plexus which is an epidural and peridural vein that forms a webbing-like structure. Veins from the vertebral bodies drain into Batson's plexus in the perivertebral area. This plexus has anastomoses with plexuses at the base of the brain, chest wall, intercostals, lumbar, and pelvis. If there is backflow due to changes in pressure on the chest and abdominal walls, the bacilli can also spread. Another route of spread may result from a preformed paravertebral abscess that spreads from the anterior and posterior longitudinal ligaments to the adjacent vertebral bodies.^{2,3} Vertebrae that have been infected by *Mycobacterium Tuberculosis* will experience inflammation in the para discus region, spinal cord oedema, bone destruction due to lysis of bone tissue, ischemia, deformity, and peri-arthritis, causing the vertebrae to collapse and lose the mechanical strength that enables their function to support the weight.^{4,5}

Blood tests used for the evaluation of tuberculosis include the erythrocyte sedimentation rate (ESR) and

the level of C-reactive protein (CRP). In 60-83% of TB cases, the ESR usually increases by about 0.20 mm/hr. On radiological examination, the initial radiographic appearance of the vertebrae is vertebral osteoporosis, joint space narrowing, and unclear paradiscal margins of the vertebral bodies.⁶ Computed tomography (CT) scan of the vertebrae is useful for assessing the extent of bone destruction, posterior infection, and infections of the craniovertebral, cervical vertebrae, and sacroiliac joints, which are not readily visible on plain radiographs.⁷ Magnetic resonance imaging (MRI) is the gold standard for demonstrating an extension of disease to soft tissues, the spread of tuberculous abscess, and nerve compression.⁸ MRI is also useful for differentiating tuberculous spondylitis from pyogenic infection. Tuberculous spondylitis more commonly occurs in the thoracolumbar spine (40%)⁹ and presents a thin-walled, regular paravertebral abscess, while pyogenic spondylitis shows the thick and irregular-walled one.¹⁰

The duration of pharmacological therapy for cases of tuberculous spondylitis is still in research, The World Health Organization (WHO) guideline (2021) recommends undergoing OAT treatment for 9 months. The anti-tuberculosis drugs (OAT) given are isoniazid, rifampin, pyrazinamide, ethambutol, or streptomycin given in the initiation phase for 2 months, followed by isoniazid and rifampin for 7 months in the continuation phase. Surgical intervention can be performed in situations such as lack of response to therapy or recurrence, severe weakness, and persistence or worsening of neurological deficit even after the start of therapy, instability, severe pain, and deformity.¹¹ Complications that can occur are severe kyphosis due to the bone damage that occurs, leading to considerable bone destruction. This will eventually result in paraplegia in the lower extremities known as Pott's paraplegia.¹² The prognosis of TB spondylitis varies depending on the clinical manifestations that occur. Prognosis improves when treatment is delivered promptly, high mortality in children aged less than 5 occur up to 30%.¹³

CASE DESCRIPTION

Patients Information

A 66-years-old female patient visited the Neurology Clinic of Bethesda Hospital Yogyakarta with complaints of pain and difficulty in moving the left lower extremity 3 months ago. The patient also complained of pain that radiated from the waist to the left lower extremity. The patient had a previous history of hypertension 5 years ago, no history of tuberculosis infection, and no specific family history.

Clinical Finding

The general condition of the patient appeared to be in pain with full awareness and cooperation. Examination of vital signs showed blood pressure of 140/80 mmHg, pulse 82 beats per minute, and respiratory rate 22 breaths per minute. In this patient,

there was a neurological deficit in which there was a decrease in motoric strength in the left leg.

Diagnosis Assessment

On the first visit, the patient was diagnosed with Unspecific Polyneuropathy and treated with analgesics and mecobalamin. The patient's condition did not improve, a Lumbosacral-MRI examination without contrast material was performed. Based on the MRI images, it was found that there was an impression of lumbar spondyloarthrosis with lordotic alignment rigidity and L4-L5 listhesis, presence of deformity at L3-L4 level, narrowing at L2-L3 level and disc material protrusion as seen in Fig 1 and 2. We concluded these finding to be Spondylitis TB L2-L5, with disc protrusion L2-L3.



Figure 1. MRI Lumbosacral (T1 Sagittal Section)

Red circle shows lumbar spondyloarthrosis with lordotic alignment rigidity and L4-L5 listhesis, presence of deformity at L3-L4 level



Figure 2. MRI Lumbosacral (T1 Sa Sagittal Section) Red circle shows narrowing at L2-L3 level and protrusion of disc material

DISCUSSION

Tuberculous spondylitis is extrapulmonary tuberculosis that requires early detection to prevent further neurological complications. The most common site of TB spondylitis is the thoracolumbar vertebra, followed by the thoracic vertebrae, lumbar vertebrae, and cervical vertebrae. In this patient, the location of the lesion was in the lumbar vertebral segment. The main complaints (83-100% of cases) are back pain caused by disruption of the intervertebral discs and spinal instability, nerve compression or pathological fracture, exacerbated by spinal movement, coughing, and weight-bearing. Typical symptoms of lung tuberculosis such as cough, fever, and weight loss, were relatively rare (32%). This may be due to *Mycobacterium tuberculosis* which is dormant in the vertebrae during primary infection.¹⁴ Primary focus can be active or dormant, obvious or latent, either pulmonary or extrapulmonary. Typical symptoms of pulmonary TB and medical history that support a radiological diagnosis (Primary Tuberculosis) are not primary symptoms. Tuberculosis bacilli can travel from the lungs to the spine via Batson's paravertebral venous plexus or via lymphatic drainage to the para-aortic lymph nodes. Patients with TB spondylitis also have complications of neurological deficits. In this patient, there was a neurological deficit in which there was a decrease in motoric strength in the left leg. This condition was caused by compression of the paraspinal abscess in the spinal cord. Infection of the vertebrae was followed by a chronic inflammatory response characterized by epithelioid cells, Langhans cells, lymphocytes, and inflammatory exudate, which together formed a characteristic histopathological lesion called a tubercle. This infection usually begins in the anterior cancellous bone within the vertebral body followed by the destruction of the vertebral body, extending down the anterior longitudinal ligament and forming an abscess near the vertebral body.

Based on national and international guidelines, the standard treatment protocol for TB spondylitis is OAT (isoniazid, rifampin, ethambutol, and pyrazinamide) with a treatment duration of 9-12 months, and can be extended to 24 months in severe cases. In this patient, surgical intervention was not recommended due to the elderly age, although the

patient also met the indications for surgical intervention such as the presence of neurological deficits and spinal deformities. Various studies have shown that the majority (82-95%) of patients with TB spondylitis respond very well to OAT treatment. Almost all OATs penetrate well into the target lesion. The prognosis for neurologic deficits is good in 75% to 95% of treated TB spondylitis patients. Many factors influence recovery from paraplegia resulting from tuberculous spondylitis. These factors include the general physical condition of the patient, including immunological status, age, spinal cord status, level, and, number of vertebrae involved. Other factors influencing recovery were the degree of spinal deformity (almost no recovery even after radical decompression surgery in patients with kyphosis greater than 60 degrees), duration and degree of paraplegia, time to start treatment, type of treatment, and drug sensitivity.^{15,16}

CONCLUSION

Tuberculous spondylitis (TB) can cause inflammation of the paradiscus, edema, progressive bone destruction, which is caused by lysis of anterior bone tissue, and secondary ischemia that will cause collapse. Pain, moving difficulty, and local swelling are the most frequently found symptoms and signs. MRI radiology imaging presented abnormalities in curvature, vertebral alignment, deformity, and bone destruction. Treatment of TB spondylitis is based on anti-TB drugs combined with immobilization by wearing a corset. The prognosis of TB spondylitis varies depending on the clinical manifestations that occur.

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