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Case Report: A tuberculosis spondylitis pediatric patient with paraplegic

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Abstract : *Skeletal tuberculosis (TB) accounts for approximately 10% of all extrapulmonary TB with the spine as the most common skeletal site affected. Spinal tuberculosis accounts for almost 50% cases of skeletal TB. Thoracolumbar junction remains to be the most affected region.* ***Case :*** *A 3-years old and 2 months old girl was admitted to the hospital with the chief complaints of paraparesis, inability to walk for 3 months and back pain. X-ray of the lumbosacral revealed spondylitis TB. The patient was planned to undergo surgery due to neurological deficit secondary to spinal destruction.* ***Conclusion*** *This case showed that there was a relationship of Tuberculous spondylitis causing Paraplegia.*

Keywords - *Tuberculosis, Spondylitis, Paraplegia, Pott disease, Mycobacterium Tuberculosis*

I. INTRODUCTION

Mycobacterium tuberculosis mainly affects the lungs. Other commonly affected organ systems include respiratory system, gastrointestinal (GI) system, lymphoreticular system, skin, central nervous system, musculoskeletal system, reproductive system, and liver, therefore it is called a multi-systemic disease.[1] Globally, an estimated 10.0 million people developed active TB disease in 2019, with 1.4 million TB deaths. According to WHO, TB global incidence rate declines of about 2% per year for most of the past two decades. The incidence of extrapulmonary TB was low however its decline rate is not as significant as pulmonary TB.[2]

Skeletal tuberculosis (TB) accounts for approximately 10% of all extrapulmonary TB with the spine as the most common skeletal site affected. Spinal tuberculosis accounts for almost 50% cases of skeletal TB. Thoracolumbar junction remains to be the most affected region of the spinal column followed by lumbar spine and the cervical spine.[3] Tuberculosis remains a significant cause of both illness and death in developed countries, especially among individuals with a suppressed immune system. Children are also vulnerable, and tuberculosis was responsible for one million illnesses in children in 2015, according to the WHO.

Spinal TB is usually secondary to hematogenous spread from a primary site of infection (most commonly the lungs). The paradiscal vessels typically supply the subchondral bone on either side of the disc space and therefore, the most common site of vertebral involvement is paradiscal. The clinical presentation of spinal tuberculosis is variable. The manifestations depend upon the duration of illness, severity of the disease, site of the lesion, and presence of associated complications including deformity and neurological deficit. Back pain in tuberculosis can be related to the active disease itself (secondary to inflammation), bone destruction and instability.[4]

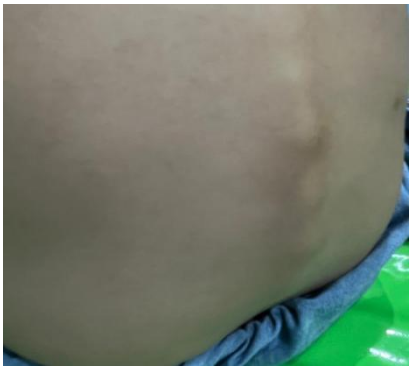
Early diagnosis allows rapid therapeutic intervention and prevention of possible complications. Disk space narrowing and vertebral body destruction are the most common changes seen on plain radiographs, which may however be normal at the earliest stage of the disease. The management of STB consists of supportive care, chemotherapy, and surgery. Anti tuberculosis drug remains the mainstay of therapy throughout the treatment process. The fundamental principle of chemotherapy in TB is that any regime chosen must include multiple drugs and must be given for a prolonged period of time. Therapy should be continued for a duration of 6 to 9 months.[5]

The following report describes a case of pulmonary TB accompanied by spinal TB with paraplegia in a 3 years 2 months old girl patient.

II. CASE REPORT

A girls aged 3 years and 2 months was admitted to our hospital with the chief complaint pf inability to walk for 3 months, initially the patient was limping when walking, which then progressed into unable to walk and sit. The patient also had back pain. Food and water intake were decreased. She had received complete vaccination. There was a positive history of contact with a TB-infected neighbor. Moreover, there were history of cough longer than 3 weeks. There was no history of fever longer than 2 weeks, weight loss, or trauma. Family history of similar complaint was denied. The patient was referred from Neurology division with paraparesis secondary to extrapulmonary tuberculosis.

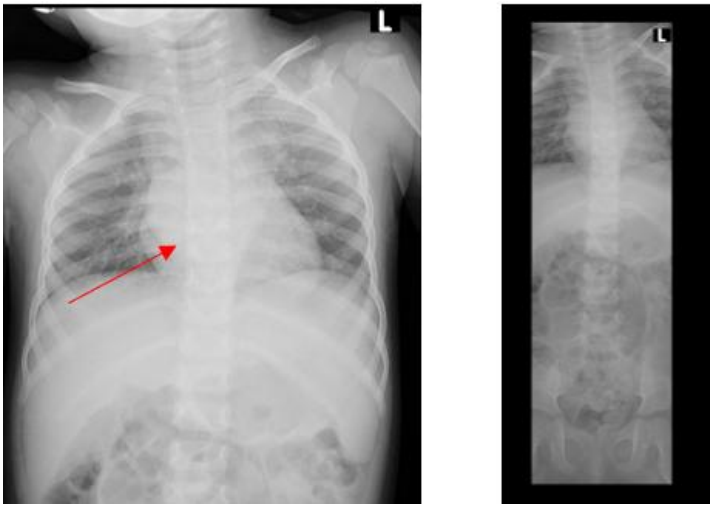
On presentation to the Emergency Department, the general condition was moderately ill and vital signs within normal limit and pain scale of 2 NRS (Numerical Ratio Scale). General examinations revealed sunken eyes, and gibbus on the 9^{th} thoracal vertebrae with tenderness (Picture. 1), otherwise normal. Her puberty status was A1M1P1. Neurological tests showed on both lower extremities, we found low motoric strength (2/2), hypotonus, decreased physiological reflexes, with normal sensory function. Pathological reflexes were negative. There was a positive urinary retention. Tuberculosis score was 7 of 13, with a contacts of smear-positive TB patients (3), positive Mantoux test (3), and chronic cough (1), indicating of clinically-diagnosed TB.



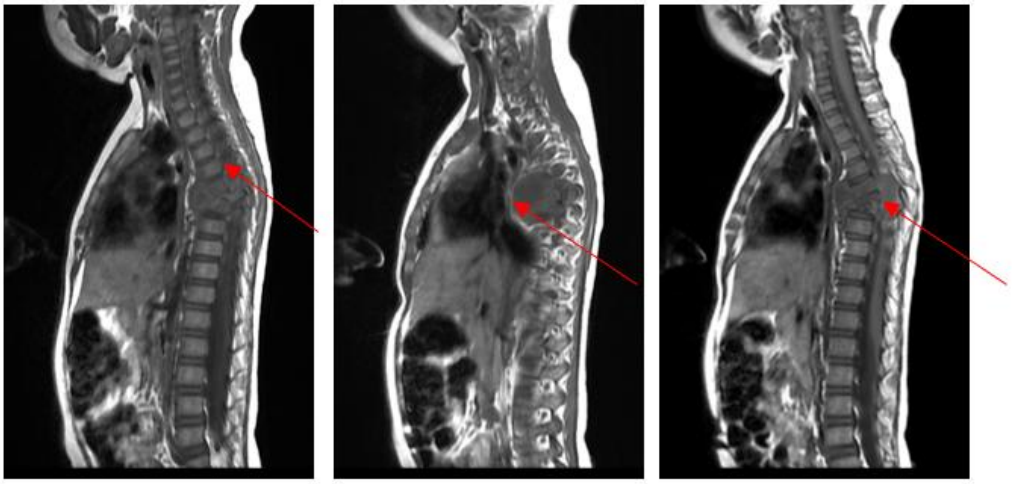
Picture 1. Gibbus on the 9^{th} thoracal vertebrae

In the supporting examination, x-ray anteroposterior view of chest and lumbosacral showed spondylitis TB (Picture 2). The result of the supporting examination were also obtained hemoglobin 10,2 gr/dl and WBC 10.300/mm^3. Gastric lavage was performed with a negative result. MRI with contrast showed a CV T7 destruction with bone marrow edema on CV T6 concur with spondylitis TB and gibbus deformity (Picture 3)

The patient received intensive phase anti-tuberculosis (Isoniazid 120 mg, Rifampicin 180 mg, Pyrazinamide 240 mg, and Ethambutol 240 mg) followed by maintenance phase anti-tuberculosis (Isoniazid 120 mg and Rifampicin 180 mg)



Picture 2. AP/Lateral view of chest x-ray and lumbosacral x-ray



Picture 4. Thoracal MRI with contrast

III. DISCUSSION

Tuberculosis is an infectious bacterial disease caused by *Mycobacterium tuberculosis* (Mtb), transmitted through the respiratory route and most commonly affects the lungs, but can damage any tissue. The pathogen persists in many infected individuals in a latent state for many years and can be reactivated to cause disease. The risk of progression to TB disease after infection is highest soon after the initial infection and increases dramatically for persons co-infected with HIV/AIDS or other immune-compromising conditions.[6]

Tuberculosis (TB) spine results in bone loss as well as disturbed growth potential, hence spinal deformities may progress as the child grows. The growth potential is also disturbed when the disease focus is surgically intervened. Surgery is indicated for complications such as deformity, neurological deficit, instability, huge abscess, diagnostic dilemma and in suspected drug resistance to mycobacterium tuberculosis. The child on antitubercular treatment needs to be periodically evaluated for weight gain and drug dosages need to be adjusted accordingly. The severe progressive kyphotic deformity should be surgically corrected. Mild to moderate cases should be followed up until maturity to observe progression/improvement of spinal deformity. The surgical correction of kyphotic deformity in active disease is less hazardous than in a healed kyphosis. The internal kyphectomy by extra pleural approach allows adequate removal of internal salient in paraplegic patients with healed kyphotic deformity.[7]

Malnutrition has been associated with increased risk of respiratory infections. Several mechanisms may underlie the increased risk and severity of respiratory infections in malnourished children. Malnourished children has a higher risk of tuberculosis infection.[8]

Since 2000 when the World Health Organization (WHO, 2017) estimated that the global incidence rate for tuberculosis has fallen by 1.5% every year. Furthermore, mortality arising from tuberculosis has significantly and steadily declined. The World Health Organization (WHO, 2016) reports a 22% drop in global TB mortality from 2000 through 2015.[1] The incidence of extrapulmonary TB is low at 3%, but there has been no significant reduction in incidence of extrapulmonary TB when compared to pulmonary TB. Skeletal TB contributes to around 10% of extrapulmonary TB, and spinal TB has been the most common site of skeletal TB, amounting to around half of skeletal extrapulmonary TB. Thoracolumbar junction remains to be the most affected region of the spinal column followed by lumbar spine and the cervical spine.[3]

Tuberculous spondylitis most often involves thoracal and lumbosacral spine. Inferior thoracal spine is the most common region involved (40-50%), followed by lumbar spine on the second place (35-45%), and the rest 10% involves cervical spine.[9] Adolescence represents another uniquely susceptible time for TB progression. Disease presentation can digress from typical childhood manifestations to include aspects of adult-type TB, including cavitary pulmonary TB or extra-thoracic manifestations that are associated with longer incubation periods.[10] The patient in our case is 3 years and 2 months old girl, with a focus of tuberculous spondylitis on the thoracal spine.

M. tuberculosis is an alcohol and acid-fast bacillus. It is part of a group of organisms classified as the *M. tuberculosis* complex. *M. tuberculosis* is a non-spore-forming, non-motile, obligate-aerobic, facultative, catalase-negative, intracellular bacteria. The organism is neither gram-positive nor gram-negative because of a very poor reaction with the Gram stain. Weakly positive cells can sometimes be demonstrated on Gram stain, a phenomenon known as "ghost cells."[1]

The first contact of the *Mycobacterium* organism with a host leads to manifestations known as primary tuberculosis. This primary TB is usually localized to the middle portion of the lungs, and this is known as the Ghon focus of primary TB. This state is known as latent tuberculosis. Latent tuberculosis is capable of being reactivated after immunosuppression in the host. A small proportion of people would develop an active disease following first exposure. Such cases are referred to as primary progressive tuberculosis. Primary progressive tuberculosis is seen in children, malnourished people, people with immunosuppression, and individuals on long-term steroid use. Most people who develop tuberculosis do so after a long period of latency (usually several years after the initial primary infection). This is known as secondary tuberculosis. Secondary tuberculosis usually occurs because of the reactivation of latent tuberculosis infection. The lesions of secondary tuberculosis are in the lung apices. A smaller proportion of people who develop secondary tuberculosis do so after getting infected a second time (re-infection). The lesions of secondary tuberculosis are similar for both reactivation and reinfection in terms of location (at the lung apices), and the presence of cavitation enables a distinction from primary progressive tuberculosis which tends to be in the middle lung zones and lacks marked tissue damage or cavitation.[1]

Tuberculosis is a classic example of a cell-mediated delayed type IV hypersensitivity reaction. Mycobacterium tuberculosis induces the recruitment and activation of tissue macrophages. This process is enhanced and sustained by the production of cytokines, especially interferon-gamma. Two main changes involving macrophages occur during this process, namely, the formation of multinucleated giant cells and the formation of epithelioid cells. Giant cells are aggregates of macrophages that are fused together and function to optimize phagocytosis. The aggregation of giant cells surrounding the Mycobacterium particle and the surrounding lymphocytes and other cells is known as a granuloma. The appearance of the granuloma in tuberculosis has been described as caseous or cheese-like on gross examination. This is principally explained by the rich mycolic acid content of the mycobacterium cell wall. Because of this unique quality, the term caseous or caseating necrosis has been used to describe granulomatous necrosis caused by mycobacteria tuberculosis.[1]

After ingestion, macrophages can undergo apoptosis or necrosis. After necrosis, bacterial spread may ensue. Surviving macrophages assist in early granuloma formation, either leading to elimination or clinical latency. B The mycobacteria can evade the immune response by inhibiting phagolysosome formation and apoptosis, as well as blocking the response of macrophages to IFNγ. C Resident dendritic cells of the lung can

travel to regional lymph nodes, presenting live mycobacteria and mycobacterial antigen, activating naïve T-cells, B-cells and regulatory T-cells. D In the lung, activated T-cells and B-cells (attracted to the lung by chemokines) control bacterial growth by production of cytokines and antibodies. Regulatory T-cells control the inflammation through the production of IL-10 and TGF-β.[11]

The course of spinal infection follows 2 main routes, namely arteries and veins, as well as additional routes. The main route runs systemically along the arteries to the periphery to the vertebral bodies, originating from the segmental lumbar arteries that provide blood to half of the adjacent bodies, where each is supplied by 4 arteries. It ends as an end-artery therefore the expansion of the vertebral body infection often begins in the paradiscus area.[9]

Tuberculous osteomyelitis and arthritis are believed to arise from foci of *M. tuberculosis* bacilli lodged in bone during mycobacteriemia in primary infection. The primary focus may be active or quiescent, overt or latent, either in the lungs, mediastinal lymph nodes, mesentery, kidney or other internal organs. *M. tuberculosis* bacilli can travel from the lungs to the spine via Batson's paravertebral venous plexus, via lymphatic drainage to the paraaortic nodes. In healthy individuals, the cellular immune response already contains these bacilli.[9]

The spread of tuberculosis infection will cause paradiscus inflammation, hyperemia, spinal cord edema and osteoporosis. Bone damage occurs progressively, due to lysis of bone tissue in the anterior part, as well as secondary ischemia, periarthritis and endarteritis, which will cause collapse in the respective part. This will result in a loss of the mechanical strength of the bones to support the load so that vertebral collapse will occur with the intervertebral joints with the posterior neural arch remaining intact, resulting in a progressive deformity in the form of kyphosis (posterior angulation) depending on the level of damage, the level of the lesion and the number of vertebrae involved which is often referred to as gibbus. When these abnormalities have appeared, it suggests that the disease has spread.[9]

Neurological deficits by extradural compression of the spinal cord occur as a result of many processes, namely: 1) narrowing of the spinal canal by paravertebral abscess, 2) pathological joint subluxation, 3) granulation tissue, 4) vasculitis, spinal arterial or venous thrombosis, 5) collapse vertebra, 6) epidural abscess or 7) direct dura mater invasion. In addition, spinal cord invasion can also occur intradurally via meningitis and tuberculosis as space-occupying lesions. The healing process then occurs gradually with the emergence of fibrosis and calcification of tuberculous granulomatous tissue. Sometimes the fibrous tissue hardens, resulting in ankylosis of the collapsed vertebra.[9]

The increased predisposition of the nutrient-deficient host to infection is presumed to be largely due to impaired immune function. Most of what is reported relating to the impact of malnutrition on host defense involves children or animal models that are broadly described as suffering from protein-energy malnutrition, but this is often poorly defined. Studies of children are limited mostly to the descriptive quantitation of specific cells or factors, often without an assessment of function or consequence. Little is known about the impact of malnutrition on mucosal and skin defense, leukocyte trafficking, leukocyte effector function, and inflammatory mediator activity in an in vivo context.[12]

After primary infection, the majority of patients remain asymptomatic. Most of these asymptomatic individuals clear the infection. However, a portion enters a "latent" phase with the potential "reactivation" in the future. Symptomatic individuals (around 10 percent) develop primary lung infection with some suffering spread to distant organs, particularly immune-compromised patients (e.g., HIV patients). Prolonged fever is the most commonly reported symptom as only one-third of patients with pulmonary involvement develop respiratory symptoms. This fever usually follows a diurnal pattern. It increased as the day goes and subsides at night, although sometimes it is associated with night sweat. Pulmonary symptoms include chest pain, shortness of breath, and cough. Cough is often mild and non-productive. However, in disease progression, it might produce green or blood-tinged sputum. Other nonpulmonary symptoms may occur, such as lymphadenopathy, fatigue, and pharyngitis. Anorexia, weight loss, and loss of muscle mass could happen in advanced cases.[13]

The clinical presentation of spinal tuberculosis is variable. The manifestations depend upon the duration of illness, severity of the disease, site of the lesion, and presence of associated complications including deformity and neurological deficit. In uncomplicated disease, the patient typically presents with back pain; while the presentation associated with complicated tubercular spine disease involves deformity, instability, and neuro deficit. Back pain in tuberculosis can be related to the active disease itself (secondary to inflammation),

bone destruction and instability. Rest pain is pathognomonic, and rarely, radicular pain can be the main presenting symptom. Constitutional symptoms including weight or appetite loss, fever, and malaise/ fatigue are less commonly associated with extrapulmonary tuberculosis than pulmonary disease. [4]

At its active stage, symptoms of tuberculous spondylitis are often insidious. Common symptoms are malaise, loss of appetite and weight, and night sweat. The involved spine is stiff and painful on movement with a localized humpback. Back muscle spasms are present. Occasionally, patients may have night-cries during sleep, as the relaxation of muscle spasms allows for movement between the inflamed surfaces. Cold abscess and/or sinus may be present. In the early stages of disease, some of these symptoms and signs may be absent. On palpation, a small gibbus may be detected. Rarely, neurological deficits may present as the first symptom.[14]

Our patient complained about feeling heavy on both legs causing her to limp when walking for 2 months and during the last 1 month, our patient was unable to walk. In addition, the patient also had a back pain.

A confirmatory diagnosis relies on detecting the pathogen directly; alternative approaches include detecting the histopathologic or host immune response to the pathogen. Direct pathogen-based tests include TB culture, nucleic acid amplification tests (NAAT), and smear microscopy. Mycobacterial culture is the gold-standard test for TB in solid or liquid culture media. GeneXpert MTB/RIF (Cepheid, Sunnyvale, California USA) assay is a NAAT that detects M. tuberculosis DNA. GeneXpert only has a pooled sensitivity of ~66% compared to TB culture in pediatric populations.[10]

Collecting a deep respiratory specimen for TB culture from a young child brings added challenges. Most children <7 years do not have the tussive force and/or the oromotor coordination to produce a good-quality expectorated sputum specimen on command. Semi-invasive techniques such as gastric aspiration/lavage or sputum induction with or without nasopharyngeal aspiration may be required. With procedural training, sputum induction provides at least similar microbiologic yield compared to gastric aspiration.[10]

In extrapulmonary TB, site-specific specimens for TB culture are often collected, such as cerebrospinal fluid (CSF), lymph node aspirates and other tissue specimens. However, the yield is variable. Mycobacterial blood cultures seem to be of limited yield in children compared to adults. Histopathologic diagnosis is more commonly pursued in extra-pulmonary TB. The overall yield is not well characterized, and is somewhat dependent upon the experience of the proceduralist and pathologist; sensitivity and specificity may be hindered by other granulomatous processes.[10]

The tuberculin skin test (TST) has inherent limitations of sensitivity and specificity. Even in high tuberculosis-burden areas, approximately 20% of individuals show negative to TST throughout life, despite repeated exposure to the tubercle bacilli. Additionally, the sensitivity decreases in immuno-compromised patients for whom accurate diagnosis of latent tuberculosis infection is essential. In terms of specificity, TST is influenced by Bacillus Calmette-Guérin (BCG) vaccination and non-tuberculous mycobacterial infection.[14]

Imaging techniques such as simple radiographs, bone scan, computed tomography (CT) and magnetic resonance imaging (MRI) are useful but not diagnostic. For example, when disc and/or end-plate destruction with surrounding soft tissue swelling is observed on simple radiographs.[14]

In our patient, we found a positive tuberculin test and chest x-ray showed bone destruction with bilateral pneumonia. In addition, bone CT scan showed a bone destruction caused by skeletal TB.

Children may be evaluated for TB following presentation with symptoms or signs suggestive of TB (passive case finding) or as a result of contact investigation or during routine immigrant screening (active case finding). The clinical presentation of children detected by active case finding differs from those detected by passive case finding, with the former often having M. tuberculosis infection only or disease in a very early phase. M. tuberculosis infection detected in young children or following recent TB exposure implies a higher risk of disease progression. In nonendemic areas, the pretest probability is highly dependent on the child's TB exposure status, which influences the positive predictive value of all subsequent investigations.[15]

Sputum smear microscopy provides the cornerstone of TB diagnosis in most countries, but it has limited utility in young children with paucibacillary disease who are unable to expectorate. Although sensitivity remains suboptimal in children, the Xpert-MTB/RIF assay is rapid and highly specific. Using two sputum samples. it detects three times more cases than microscopy and ~70% of the cases detected by liquid culture. Both immunological assays, the traditional TST and newer interferon-γ release assays (IGRAs), fail to

differentiate M. tuberculosis infection from TB disease. WHO recommends that IGRAs should not replace the TST for the detection of M. tuberculosis infection in low- or middle-income countries, although they may be complementary by improving sensitivity and/or specificity in specific clinical situations.[15]

In clinical practice antero-posterior and lateral view must be obtained; Spinal radiography may show a destructive process of vertebras and adjacent discs if osteomyelitis is present. These findings only appear in a late course of the disease and are less pronounced compared to pyogenic infection. The CT helps to define the extent of the disease and is the best method to detect calcified foci, characteristic in tuberculous infections and rare in pyogenic infections. The overall sensitivity and specificity of MRI for tuberculous spondylitis is 100% and 80%, respectively, which renders the MRI the best radiological method for the diagnosis of tuberculous spondylitis [8]. MRI is also helpful to clarify the need for surgical intervention, since it is the most precise radiological method to assess nervous system involvement and spine instability.[16]

Diagnosis in our case is established according to clinical manifestation and additional work-ups including Mantoux test, chest x-ray, biopsy findings, and spinal MSCT suggesting lesion on thoracal spine.

TB treatment aims to cure the individual patient, whereas the public health aim is to terminate transmission and prevent the emergence of drug resistance. Actively metabolizing bacilli are rapidly killed by bactericidal drugs, improving clinical symptoms, terminating transmission, and providing protection to companion drugs. Sterilizing drugs are required to eradicate persistent subpopulations of bacilli to establish long-term cure.[15]

The goal of tuberculous spondylitis treatment is not only to eradicate infection but also to treat and prevent neurological complications and spinal deformities. Pharmacological treatment should be initiated as soon as the diagnosis is confirmed, with 2 months of HRZE (intensive phase) followed by 4 to 7 months of HR (continuation phase). The duration of treatment remains controversial. Due to difficulties in assessing response and risk of relapse, most experts recommend 9 to 12 months of treatment, and in situations of slow radiological resolution as case 2, 12 to 24 months of treatment should be considered.[16]

The deformity of the spine in these patients will continue to increase in the active phase and even after healing of disease. The progression of deformity depends on the severity of angle before treatment, level of lesion and age of the patients. The principles of kyphotic deformity correction are anterior debridement/corpectomy, posterior column shortening, posterior instrumentation and anterior and posterior fusion performed sequentially simultaneously in a single stage.[7]

Surgery is confined to patients that present with neurological deficits caused by spinal cord compression, spinal deformity with instability, severe or progressive kyphosis, large paraspinal abscesses, and no response or failure of anti-TB therapy.[16] Our patient is treated with anti-tuberculosis of 4 regimens and was planned to undergo surgery due to neurological deficit secondary to spinal destruction.

In spinal TB, intervertebral disc is involved late in the disease process leading to disc space narrowing and constitution of spondylodiscitis. Subligamentous spread of the infection may lead to multiple levels of contiguous or skip the vertebral body involvement. Extension of the infection into the adjacent soft tissue to form paravertebral or epidural masses is commonly observed. The end may result in neurological complications such as spinal cord compression. In longstanding cases, there may be multiple levels of vertebral body collapse, resulting in a gibbous deformity.[17] In our patient, the prognosis was dubious due to delayed diagnosis, thus delayed treatment. Moreover, there are both motoric and sensory disturbance secondary to spondylitis TB.

IV. CONCLUSION

Here we report a case of 3-years old and 2 months old girl patient was referred with paraparesis secondary to extrapulmonary TB. She was presented with inability to walk for 3 months and back pain. Gibbus was found on the 9th thoracal vertebrae with tenderness. Neurological exam showed decreased motoric strength, hypotonus, and decreased physiological reflexes. Imaging studies suggested and lumbosacral x-ray suggested a grade I (Meyerding Classification) reverse spondylolisthesis with a CV T7 destruction with paravertebral soft tissue density on CV T5-T8 level concur with spondylitis TB. Our patient is treated with anti-tuberculosis of 4 regimens and was planned to undergo surgery due to neurological deficit secondary to spinal destruction. In our patient, the prognosis was dubious due to delayed diagnosis, thus delayed treatment.

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