UPPER MOTOR NEURON PARAPARESIS AND CHRONIC LOW BACK PAIN ECAUSA SPINAL TUBERCULOSIS

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INTRODUCTION
Spinal involvement occurs in less than 1% of patients with tuberculosis (TB). The incidence of spinal tuberculosis is increasing in developed nations. Spinal TB (Pott’s disease) is the most common as well as one of the most dangerous forms of skeletal TB and accounts for 50% of all 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. The name traces back its origin from the description of tuberculosis infection of the spine by Sir Percival Pott in his monograph in 1779. The classic destruction of the disk space and the adjacent vertebral bodies, destruction of other spinal elements, severe and progressive kyphosis subsequently became known as Pott’s disease. Common clinical manifestations include constitutional symptoms, back pain, spinal tenderness, paraplegia, and spinal deformity. 1. Magnetic resonance imaging frequently demonstrates involvement of the vertebral bodies on either side of the disk, disk destruction, cold abscess, vertebral collapse, and presence of vertebral column deformities. 1. Antituberculous treatment remains the cornerstone of treatment. Surgery may be required in selected cases.

Epidemiology
The incidence of extra pulmonary TB (EPTB) is low at 3%, but there has been no significant reduction in incidence of EPTB when compared to pulmonary TB (PTB). 4. Skeletal TB (STB) contributes to around 10% of EPTB, and spinal TB has been the most common site of STB, amounting to around half of skeletal EPTB. Thoracolumbar junction remains to be the most affected region of the spinal column followed by lumbar spine and the cervical spine.

Path physiology
TB is caused by Mycobacterium tuberculosis. The primary site of infections can be in the lungs, lymph nodes of the media stinum, mesentery, gastrointestinal tract, genitourinary system, or any other viscera. The bacilli tend to remain dormant for prolonged periods. Spinal infection is always secondary and is caused by hematogenous dissemination of the bacillus from a primary focus. Anatomically the intervertebral disc is an avascular structure and the piratical arteries split on either side of the disc and reach the subchondral region of the upper and lower endplates of each disc. This arterial supply of the vertebra favors subchondral bone involvement on either side of the disc, “piratical,” which is the most common type observed. The other
patterns of involvement are “central,” resulting in vertebral body loss; “posterior,” when posterior appendicular structures are involved; and “no osseous abscess” formation. TB results in granulomatous inflammation characterized by lymphocytic infiltration and epithelioid cells, which may merge to form the classical Langhans-type giant cells and end up in caseating necrosis of affected tissues forming cold abscess. With progressive destruction of the vertebral body, deformation of spine causes kyphosis4.

Clinical Manifestations

Spinal TB usually is insidious in onset and the disease progresses at a slow pace.4 The diagnostic period, since onset of symptoms, may vary from 2 weeks to several years. Backache is the most common of all symptoms. During the active stage, it is primarily due to inflammation of the bone and rarely can be ridiculous in nature. Rest pain at the involved level is pathognomonic and the intensity is proportional to the amount of bone destruction and instability. The classical constitutional features of tuberculosis indicating presence of an active disease are malaise, loss of weight and appetite, night sweats, evening rise in temperature, generalized body aches, and fatigue. Back pain is the most frequent symptom of spinal tuberculosis. The intensity of pain varies from constant mild dull aching to severe disabling. Pain is typically localized to the site of involvement and is most common in the thoracic region. The pain may be aggravated by spinal motion, coughing, and weight bearing, because of advanced disk disruption and spinal instability, nerve root compression, or pathological fracture. The level of spinal cord involvement determines the extent of neurological manifestations. In cervical spinal tuberculosis, patients manifest with symptoms of cord or root compression. The earliest signs are pain, weakness, and numbness of the upper and lower extremities, eventually progressing to tetraplegia. If the thoracic or lumbar spine is involved, upper extremity function remains normal while lower-extremity symptoms progress over time eventually leading to paraplegia. Formation of a cold abscess around the vertebral lesion is another characteristic feature of spinal tuberculosis. Abscess formation is common and can grow to a very large size. The site of cold abscess depends on the region of the vertebral column affected. Spinal deformity is a hallmark feature of spinal tuberculosis. Type of spinal deformity depends on the location of the tuberculosis vertebral lesion. Hypnosis, the most common spinal deformity, occurs with lesions involving thoracic vertebrae. The severity of the hypnosis depends on the number of vertebrae involved.

Diagnosis

Diagnosis of spinal tuberculosis depends on presence of characteristic clinical and neuroimaging findings. Etiological confirmation requires the demonstration of acid-fast bacilli on microscopy or culture of material obtained following biopsy the lesion. Polymerase chain reaction is also an effective method for bacteriological diagnosis of tuberculosis. Screening of the whole spine should be done to look for noncontiguous vertebral lesions. Culturing M. tuberculosis is time consuming, taking 6–8 weeks for the growth to appear. Erythrocyte sedimentation rate (ESR) is generally raised many folds in the majority of patients with spinal tuberculosis. Culture is not the gold standard for diagnosing spinal tuberculosis because mycobacterium bacilli are not readily detected from extra pulmonary sites. False-negative results of biopsy are common and, therefore, diagnosis of spinal tuberculosis must be made on ground of clinical manifestations and radiology when bacteriology proves negative.

Management

In patients with spinal tuberculosis, antituberculous treatment should be started as early as possible. Antituberculous treatment often needs to be instituted empirically, much before an etiological diagnosis is established. Various studies have shown that the majority (82–95%) patients of spinal tuberculosis respond very well to medical treatment. The treatment response is apparent in form of pain relief, decrease in neurological deficit, and even correction of spinal deformity. The total duration of treatment and numbers of drugs needed for adequate treatment have always been subject to controversy.84 World Health Organization (WHO) recommends 9 months of treatment for tuberculosis of bones or joints, where 4 drugs—isoniazidrifampicin, pyrazinamide, ethambutol, or streptomycin—are administered in the “initiation” phase for 2 months, followed by ionized and rifampicin for 7 months in the “continuation” phase. In some circumstances, however, surgery appears to be beneficial and may be indicated. The advantages of surgical treatment include adequate sampling for histological confirmation of the diagnosis, early and better healing by removal of the disease focus, correction and prevention of the spinal deformity, reducing the rates of recurrence, and promoting early neurological recovery. Anterior procedure is recommended when posterior structures are intact, and are best avoided in pan vertebral disease. Posterior approach is most commonly performed because the ability to achieve adequate exposure for circumferential spinal cord decompression, better deformity control through pedicle screws. Combined approach play a vital role especially in osteoporotic bones, multiple vertebral body involvement, and in severe hypertonic deformities.

Case Report

A 22 years old male patient was referred to the Emergency Department with chief complaints of progressive par paresis since January 2019. Before the par paresis has began, he complained of low back pain since November 2017. It became aggravated and then followed by neurological deficits. The neurological deficits were par paresis for 6 month and hypoesthesia with sensory level. His autonomy function is normal. There is a lump in his lower back and it was suspected as a gibbous in the lower thoraces region. The back pain has began for 1 years that means there is chronic pathology process in the vertebra so it can exclude vascular process (infarction of the spinal cord) or trauma. For the differential diagnosis of chronic back pain and par paresis in this case is suspected spinal tuberculosis, extramural mass (abscess or tumor) or phylogenic spondy litis. The characteristic clinical features of spinal tuberculosis include local pain, local tenderness, stiffness and spasm of the muscles, a cold abscess, gibbus, and a prominent spinal deformity. In this case, the symptoms and sign that appeared are chronic low back pain in thoraces region, gibes, neurological deficit (par paresis and abnormal sensory level) because the spinal deformity that
compress spinal cord. The symptoms and signs were progressed slowly that support the onset of spinal tuberculosis. For spinal tuberculosis, the pain is typically localized to the site of involvement and is most common in the thoracic region. The pain may be aggravated by spinal motion, coughing, and weight bearing, because of advanced disk disruption and spinal instability, nerve root compression, or pathological fracture. The total duration of the spinal tuberculosis varies from few months to few years, with average disease duration ranging from 4 to 11 months. In this case, the constitutional features of tuberculosis has denied by the patient. The risk factors of tuberculosis in this patient are malnutrition (his body mass index is categorized as underweight) and Indonesia is endemic for tuberculosis. The clinical manifestation for extramural mass are local pain, early onset of upper motor neuron signs, late onset of autonom function, and ascending sensory involvement. Pyogenic spondylitis takes a shorter duration for the clinical signs compare to spinal tuberculosis. For the clinical signs and symptoms of phylogenic spondylitis, include non-specific pain, high fever and neurological manifestation from the compression on spinal cord and nerve root.

To support the working diagnosis for this patient, we need to perform several supporting examination, like routine blood test, plain chest X-ray, thoraces X-ray, lumbosacral X ray dan MRI based on the result of thermoregulatory sweat test and sensory finding. The WBC count is usually normal and the differential count may show a lymphocytosis in spinal tuberculosis. It is appropriate in this case. The plain chest Xray shows no abnormality in radiology. The plain thoracal and lumbosacral X-ray shows deformity in the vertebra (burst fracture CV Th 12 with angulations to posterior and compression fracture CV L1). The result of thermoregulatory sweat test is an abnormality in sensory level at L3. The result of MRI thoraces spine non contrast is antelorlipsis CV T12 toward CLV1 with angulations to posterior, compression fracture CV L1 that compress spinal cord, and inflammation CV T11, T12, L1, and L2. The type of spinal tuberculosis deformity depends on the location of the tuberculous vertebral lesion. Kyphosis, the most common spinal deformity, occurs with lesions involving thoracic vertebrae. Based on the symptoms, signs, supporting examination (plain thoracal, lumbosacral, thermoregulatory sweat test, MRI thoracal) the final diagnosis for this patient is spinal tuberculosis. The patient was consulted to orthopedic and planned to undergo decompression and posterior stabilization operation. When operation was performed, there was a kind of caseative necrosis that found in the spinal deformity. The result for tissue microbiology didn’t found any aerobe bacteria. The patient then consulted to pulmonologist after operation for treatment tuberculosis extrapulmonal. The result for Ziehl–Neelsen staining for acid-fast bacilli is negative. The result for gene pert from sputum is negative (Mycobacterium tuberculosis is not detected). The result of culture, staining for acid fast bacilli and geneXpert is negative that means the process is extrapulmonal. Conventional microbiological methods like Ziehl–Neelsen staining for acid-fast bacilli and culture of M. tuberculosis on Lowenstein Jensen media have low sensitivity and specificity. False-negative results of biopsy are common and, therefore, diagnosis of spinal tuberculosis must be made on ground of clinical manifestations and radiology when bacteriology proves negative. The limitation for this case is the pathology anatomy for the tissue is not done and the specimen for gene pert examination is sputum not the sampling from operation so Mycobacterium tuberculosis not detected. But it didn’t exclude spinal tuberculosis as a diagnosis. The treatment for spinal tuberculosis is conservative and surgical management. 1. 2. Conservative management consists of antimicrobial therapy and non-pharmacological treatments, including physiotherapy and immobilization. Immobilization through bed rest is for pain control and prevention of deformity or neurologic deterioration. In patients with spinal tuberculosis, antituberculous treatment (ATT) should be started as early as possible. Multidrug ant tubercular treatment is the mainstay of treatment in both complicated and uncomplicated TB.
Antituberculous treatment often needs to be instituted empirically, much before an etiological diagnosis is established. Various studies have shown that the majority (82–95%) patients of spinal tuberculosis respond very well to medical treatment. The treatment response is apparent in form of pain relief, decrease in neurological deficit, and even correction of spinal deformity. World Health Organization (WHO) recommends a category-based treatment for tuberculosis. The duration of chemotherapy for spinal TB has been long debated, and the WHO recommends 9 months of treatment where 4 drugs—isoniazid, rifampicin, pyrazinamide, ethambutol, or streptomycin—are administered in the “initiation” phase for 2 months, followed by isoniazid and rifampicin for 7 months in the “continuation” phase. There is no definite role for corticosteroids in spinal tuberculosis except in cases of spinal arachnoiditis or no osseous spinal tuberculosis. For this patient, ATT was given 10 months from pulmonologist. The initiation phase for 2 months (4 FDC-isoniazid and rifampicin) and the continuation phase for 7 months (2FDC-isoniazid and rifampicin). Certain situations necessitating surgical management are as follows: lack of response to chemotherapy or recurrence, severe weakness at presentation, and static or worsening neural deficit even after initiating chemotherapy, instability, incapacitating pain, and deformities. The advantages of surgical treatment include adequate sampling for histological confirmation of the diagnosis, early and better healing by removal of the disease focus, correction and prevention of the spinal deformity, reducing the rates of recurrence, and promoting early neurological recovery. Nowadays posterior procedures are the most commonly performed, due to ease, familiarity of approach, and the lesser learning curve involved. For this case, surgical management was performed based on neurological deficit, instability posture, incapacitating pain, and deformities in the vertebra. After the operation, the patient was consulted to Physical medicine and rehabilitation for physiotherapy. 1 week after the operation, there is an improvement from the numeric pain rating scale and motor strength. So, the prognosis for this patient is good. 1 month after the operation, patient can stand independently and still learn to walk with walking stick. Based on the literature, prognosis for spinal tuberculosis is generally good in patients without neurological deficit and deformity. Various studies show that 82–95% cases respond to medical treatment alone in the form of pain relief, improving neurological deficit, and correction of spinal deformity.

Younger age and radical surgery in conjunction with antituberculosis chemotherapy have been suggested as favorable prognostic factors.

REFERENCES


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