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Review Article

Spinal Tuberculosis - Current Management Approach

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Abstract

Broad narrative review. To review and summarise the current literature on the management of tuberculosis (TB) spine. A thorough review of literature was performed on the epidemiology, aetiology, pathophysiology, pathology, clinical features and management of TB spine. Spinal TB accounts for half of skeletal TB and remains a common cause of public health concern in the developing world. The thoracic spine segment is the most affected. Patient commonly present with back pain and gibbus. The diagnosis involves demonstrating Mycobacterium tuberculosis on microscopy or culture as well as characteristic histology findings. Magnetic resonance imaging is the gold standard imaging modality. Medical therapy with anti-TB agents is the mainstay of treatment. Surgery is supplementary and indicated in selected cases. Spinal TB carries a good prognosis when detected and treated early. Delays can be associated with the development of complications including difficult-to-manage deformities. Multi-drug anti TB chemotherapy remains the bedrock of treatment. Surgery is supplementary and when indicated, takes the form of abscess drainage, debridement and fusion with or without instrumentation.

Keywords: Anti-tuberculosis drugs, kyphosis, Pott's disease, spondylodiscitis, surgery, tuberculosis

Introduction

Tuberculosis (TB) is an infectious disease caused by *Mycobacterium tuberculosis* (MTB). It is one of the oldest diseases in human history. Documentation has been made of this disease as far back as the fourth millennium BC in Egyptian mummies[1] and till date remains a disease of significant public health importance. TB is a global epidemic with over 2 billion people (a third of the world population) infected. The third world account for a majority of TB cases.[2-4] The incidence of TB is increasing worldwide and this resurgence can be attributed to the human immunodeficiency virus (HIV) epidemic and increase in multi-drug resistant strains.[3,5,6] With 8.8 million new TB cases worldwide and 1.4 million deaths annually, TB is one of the commonest infectious causes of mortality.[3,5] TB commonly affects the lungs but can affect any other extra-pulmonary organ or region in the body. Extrapulmonary tuberculosis (PTB) accounts for 10%–15% of TB and the skeletal system is second most affected after the lymph nodes.

The spine is affected in about 50% of skeletal TB.[3,7] Spinal TB is a serious form of TB affecting the vertebrae. It can be associated with severe complications unless diagnosed and treated early. This disease was first described by pervical Pott over 200 years ago, he described a case of spinal TB with drainage procedure for associated abscess.[1] Hence, spinal TB subsequently referred to as Pott's disease. The clinical presentation is variable with back pain being the commonest presentation[1,5] but patients can present for the first time with complications such as abscess, deformity or neurological deficits including urinary and faecal incontinence.[1]

Several modalities for investigating spinal TB exist including laboratory, immunological, microscopy, culture and imaging. Culture remains the gold standard for isolating the organism and magnetic resonance imaging (MRI) scan is the most sensitive imaging modality.[1,8] Computed tomography (CT) scan gives excellent details of the bone abnormalities such

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as the collapse and deformity associated with this disease. Treatment of uncomplicated cases is completely medical with the use of multiple anti-TB drugs. Surgery is indicated for complicated cases such as deformity, collapse and compressive abscess. This is usually augmented by medications.

Epidemiology

Spinal TB is a destructive 'cosmopolitan' disease. The highest incidence occurs in the developing world of Sub-Saharan African and South-East Asia due to high prevalence of poverty, ignorance, malnutrition, overcrowding and HIV. The highest prevalence is in the Indian subcontinent.[3,5] It affects all ages but commonly occurs in the young and middle-aged adults. In Nigeria and other developing nations, spinal TB is a common cause of morbidity in children and young adults, presenting most times with thoracic and thoracolumbar kyphosis with or without neurological deficits. In contrast, it is more common in elderly people in the developed world.[1] In fact, spinal TB is the most common cause of non-traumatic paraplegia in the developing countries.[9] There is no difference in occurrence between males and females.[3,8]

About 10% of TB occurs in the musculoskeletal system[1,3] and spinal TB accounts for about 50% of cases. This also translates to about 15% of all cases of extra-PTB that are Pott's disease, and about 1%–2% of all cases of TB.[1] The incidence of musculoskeletal TB is about 3%–5%. This incidence is increasing[3,10] and noted to be higher (60%) in association with HIV infection.[3] Risk factors for TB include low socioeconomic status (Crowded living conditions), Immunosuppressive drugs and diseases, significant exposure to active TB patients, living, or travel to an endemic area and a high risk occupation including laboratory and health care jobs.[6,11]

Aetiology

TB is caused by a slow-growing bacterium called MTB.[1,10,11] MTB belong to the MTB complex, a group of seven closely related mycobacterial species (*Mycobacterium bovis*, *Mycobacterium africanum*, *Mycobacterium microti*, *Mycobacterium caprae*, *Mycobacterium pinnipedii*, *Mycobacterium canetti* and *Mycobacterium mungi*). The majority of TB cases are caused by MTB. These organisms are also called tubercle bacilli.[6,12] Spinal TB, like other extra PTB, occurs as a secondary form of TB and usually follows haematogenous spread from a primary focus.[12] The primary site is usually pulmonary but can also be in the gastrointestinal tract, mesenteric and mediastinal lymph nodes, urinary, genital or any other viscera.[12,13] TB also reaches the spine from lymphatic spread from nearby para-aortic lymph nodes.[5] Mycobacteria are non-motile, non-spore-forming, slender, aerobic rods. They are slow growing acid and alcohol fast bacilli with generation times of 8–24 h. The cell wall of mycobacteria contains long chain fatty acid, particularly mycolic acid, which form a complex with peptides and polysaccharides creating a waxy cell surface that is responsible for the acid and alcohol fast staining properties seen during laboratory test. Mycobacteria are also resistant to drying, but not to heat or ultraviolet irradiation. Unlike other bacteria, mycobacteria do not produce toxins nor form biofilms.[11,14]

Pathophysiology

There are two forms of the disease at site of lesion: the intradural involvement which is rare and the extradural involvement which comprises four types - paradiscal, central, anterior and posterior lesions.

Mycobacteria reach the spine via haematogenous dissemination from other organs (commonly the lungs) or lymphatics during the mycobacteraemia stage of primary infection. TB spine may manifest when the primary focus is active or quiescent, apparent or latent depending on the host cellular immune response. The arterial supply of the vertebra favours the involvement of bones on each side of the disc with relative disc sparing. The arteries to the spine split on either side of the disc and reach the subchondral region of the superior and inferior endplates. Hence the 'paradiscal' pattern is the most common type with relative disc sparing in the early stage of the disease in adults. In contrast, children have relatively vascular disc, as such, it may not be spared as in adults.[13] The other patterns of involvement are 'central', resulting in vertebral body loss but the disc is preserved in early lesion mimicking malignancy but as the disease progresses the disc is involved; 'anterior' with scalloping lesions and abscess formation; 'posterior', or appendicular, when posterior elements are affected; and 'non-osseous abscess' formation.[2,13]

Pathology

When the MTB reaches the bone, a granulomatous inflammatory process is triggered which set a cascade in motion for caseous tissue formation. Ultimately a tubercle is formed consisting a central area of large, multinucleate giant cells (Langhan's cells) containing tubercle bacilli. This central area of caseation is surrounded by a zone of pale epithelioid cells, and a peripheral rim of fibroblasts and lymphocytes; these are the findings commonly seen in histopathological analysis. The tubercles grow by expansion and colescence. Caseation occurs in the centre of the tubercle by coagulation necrosis (soft tubercle) caused by the protein fraction of the tubercles bacilli. Presence of caseation necrosis is almost diagnostic of tuberculous pathology. A tubercle may however not show central caseation (hard tubercle) under the influence of treatment. Cold abscess is formed by a collection of products of liquefaction and reactive exudation. The cold abscess is mostly composed of serum, leucocytes, caseous materials, bone debris and tubercle bacilli. Osseous destruction takes place by lysis of hyperaemic and osteoporotic bone, which is thus softened and easily yields under gravity and muscle action, leading to compression, collapse or kyphosis. Ischaemic necrosis is also a contributory factor responsible for vertebral collapse and disc degeneration.[7] The characteristic pathology is spondylodiscitis, a combination of spondylitis, vertebral

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osteomyelitis and discitis with destruction of two or more consecutive spinal segments.[9] Skip lesions and vertebral collapse are common.

With destruction of the bone (single or multiple), there is collapse of the vertebral column with varying degrees of deformity. An abscess can form and track down planes of least resistance commonly stripping the anterior longitudinal ligament. The lack of proteolytic enzymes in mycobacterial infections may be responsible for the subligamentous spread of infection. Compression of the spinal cord or nerves can occur from granulation tissue, abscess, sequestrum, prolapsed disc as well as extradural or intradural tuberculoma. Furthermore, spine deformity and dislocation can result in neurological deficits as can non-mechanical lesions such as pachymeningitis, thrombophlebitis and end arteritis.[2,7,9] Spinal TB commonly affects the thoracic in about 42% cases, lumbar in 26%, thoracolumbar junction in 12%, cervical spines in 12%, cervicodorsal in 5% and lumbosacral in 3% of cases.[7,13] TB of the lumbosacral has been described though very uncommon.[15] Interestingly, it is commoner at spine segments forming junctions of mobile and immobile parts of the column. Thus the thoracolumbar and lower cervical segments are commonly affected in spite of the statistics. Other reasons for preference for lumbar segments include the proximity to cisterna chili and other lymphatics, large composition of cancellous bone, large nucleus populsus, prominent veins (valveless vein of Batson's) and relative avascularity.[7] The complications of the TB spine are related to the site of lesions, these include neurological deficits as in Pott paraplegia, cold abscess, spinal deformity, secondary infections, fatality and amyloid disease.

Clinical Features

Spinal TB is an insidious but devastating disease. Patients are usually without symptoms for long period sometimes presently as complications. The diagnostic period can range from a few weeks to several months and years.[1,13] The clinical manifestation of spinal TB depends on the location, severity of the disease and the presence of complications such as neurological deficit, abscess, sinuses, collapse and angular kyphosis[9,13] as shown in Figure 1.

Back ache is the most common of all symptoms.[5,13] It is primarily due to inflammation of the bone and sometimes radicular in nature. The severity of pain is proportional to the amount of bone destruction and instability.[13] Patients give history of back pain and stiffness at the early stage. In the cervical lesion, pain is over the occiput, ear, jaw and upper limb; in the upper thorax is intercostal neuralgia; in the thoracolumbar lesion, girdle pain or epigastric pain and in the lumbar lesion, hip and leg pains. Patients can present with constitutional symptoms such as fever, malaise, loss of weight and appetite. However, these are more frequently associated with PTB which is noted to co-exist with spinal TB in 30%–60% of cases.[3] The patient may present with typical Alderman's gait in which the patient is very cautious and avoids jarring of spine and walks with head and chest thrown backward and the legs apart and waddles in TB of lower thoracic and upper lumbar spines.

Other clinical features are due to complications which may be the first clinical feature.[1] They include radicular pain, paraesthesia, incontinence, para or quadriparesis (or–plegia) depending on the location. Furthermore, deformity such as angular kyphosis and gibbus are common. A painful angular kyphosis with or without neurological deficits in children and young adults is a probable indicator of TB spine in northern Nigeria. Hemiplegia due to cervical spine TB has also been recorded.[1]

A common complication is the formation of tuberculous abscess. There is usually absence of the characteristic features of inflammation; hence, it is called a 'cold abscess'.[13] It takes the path of least resistance usually along neurovascular or fascial planes. Therefore, it can present as a swelling in the paraspinal region, anterior or posterior triangle of the neck, axilla or as a retropharyngeal abscess with or without pressure symptoms. It could also appear as chest wall swelling along the intercostal spaces or track through the diaphragmatic openings. The lumbar cold abscess can present as abdominal mass in the iliac fossa or a swelling in the lumbar triangle or groin mimicking hernia or can track down along the psoas muscle to cause pseudo-flexion deformity of the hip[13] and point in the perinium, upper thigh or around the knee.

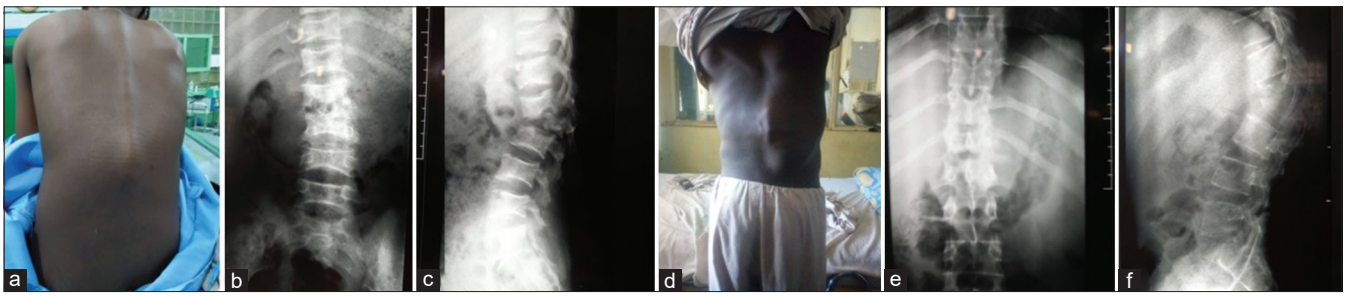

Figure 1: Common clinical features of tuberculosis spine in Nigeria: (a) A painful gibbus in a 16-year-young lady at the thoracolumbar region and (b and c) plain X-ray of the same lady showing complete collapse and disappearance of L2 vertebral body and angular kyphosis with no neurological deficit; (d) another painful thoracolumbar gibbus in a 20-year-old man with paraspinal cold abscess extending to the right lumbar triangle and paraparesis (e and f) plain X-ray of the same man showing wedge collapse fracture of L2 vertebral body and angular kyphosis

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Differential Diagnosis

Differential diagnoses of spinal TB include pyogenic spondylodiscitis or vertebral osteomylitis, atypical degenerative disk disease, neuropathic spondyloarthropaties and brucellosis. Others are granulomatous diseases such as sarcoidosis, histoplasmosis, actinomycosis, candida, blastomycosis, multiple myeloma and eosinophilic granuloma. TB spine is therefore a great mimicker[2,8,16] that must be thoroughly investigated. TB spine and metastatic diseases of the spine are both destructive lesions, but the disc is spared in neoplastic lesions while the discs are destroyed in the infective lesions. Hence the saying 'good disc, bad news; and bad disc, good news'.

Classification and Staging of Tuberculosis Spine

Spinal TB can be broadly divided into complicated and uncomplicated types based on the presence or absence of complications.[13] Complications include deformities (such as gibbus, kyphosis and dislocation), neurological deficits, abscess and collapse. They can occur in active disease or long after the disease has healed.[2,13,17] This classification gives a sense of direction on the best form of treatment to institute. Neurologic complications are seen in about 10%–43% cases.[2]

Another classification is based on the location of the lesion. The disease can be cervical, thoracic, thoracolumbar, lumbar or sacral. Thoracolumbar involvement far constitutes the majority of cases.[2,7,13]

Oguz *et al*.[18] described a new classification system of spinal TB based on the number of vertebrae involved and the presence of complications. The types are as follows:

1. Type I: Involving one disc level and soft tissue infiltration without abscess, collapse and neurologic deficit
2. Type II: Degeneration involving one or two disc levels with abscess formation and mild kyphosis. Neurological deficit may be present
3. Type III: Degeneration involving one or two disc levels with abscess formation, instability and deformity that cannot be corrected without instrumentation.

Kumar described five stages of posterior spinal tuberculosis based on clinic-radiological features, site of involvement and duration of the disease:

1. Predestructive stage when patient presents in <2 months of onset of disease with the straightening of curvatures and spasm of the perivertebral muscles
2. Early destructive stage when patient presents between 2 and 4 months of onset of disease with decreased disc spaces and paradiscal erosion
3. Mild angular kyphosis stage when patient presents between 4 and 9 months of onset with two to three vertebral involvement and kyphotic angle of 10°–30°
4. Moderate angular kyphosis stage when patient presents between 6 and 24 months with more than 3 vertebral involvement and kyphotic angle of 30°–60°
5. Severe kyphosis stage when patient presents after 24 months with more than 3 vertebral involvement and kyphotic angle of more than 60°.

Pott's paraplegia which is a major complication of thoracic spine TB had been classified into two broad types, early onset and late onset TB paraplegia, Hodgson classification of Pott's paraplegia:[7]

1. Early onset, when the duration of the disease is <2 years and in active phase with oedema, abscess, caseous and granulation tissue. The prognosis of early onset Pott's paraplegia is good
2. Late onset, when the duration of the disease is more than 2 years with mechanical pressure on the cord from the TB debris, sequestra and internal gibbus resulting in canal stenosis and severe deformity. The prognosis of late onset Pott's paraplegia is poor.

The staging of neurological deficit in TB spine has been described in four stages based on degree of severity and clinical features as summarised below:[7]

- Stage I is negligible in severity and the patient is unaware of the deficit, save the ankle clonus elicited on presentation
- Stage II is mild in severity and the patient is aware of the deficit but walks with support
- Stage III is moderate in severity and the patient is non-ambulatory due to spastic paralysis in extension and sensory deficit <50%
- Stage IV is severe and the patient is non-ambulatory due to flexor spasm, paralysis in flexion or flaccid paralysis, sensory deficit more than 50% and syphincters are involved.

Modified Frankel/American Spinal Injury Association staging of neurological deficit is widely used to predict outcome from Stages A to E as summarised below (A has the worst disease while E has the least):

- A is complete motor and sensory deficit
- B is complete motor but partial sensory deficit
- C is intact sensory but useless motor function, power is <3
- D is intact sensory but useful motor function, power is more than 3
- E is normal motor and sensory functions.

This staging is useful in assessing the severity of neurological deficit and in predicting outcome of treatment.

Management

The management of spinal TB involves the making of prompt diagnosis and the institution of appropriate treatment measures. A high index of suspicion is required because delay is costly and devastating as debilitating complications can occur. It entails taking a detailed history and thorough clinical examination as well as requesting relevant investigations to reach a diagnosis. This is promptly followed by prompt appropriate treatment. It may also require resuscitation, stabilisation and long-term rehabilitation. Overall, the management approach

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is multidisciplinary, involving the orthopaedic surgeon, neurosurgeon, infectious disease physician, community health physician, orthopaedic nurses, physiotherapist, nutritionist, chiropractor and other supportive staffs.

The history will seek to establish the presence of symptoms as elucidated above. Also risk factors for TB and HIV will constitute areas of interest as will the past medical and treatment history. Further, symptoms to suggest a primary focus of TB should be searched. For example, chronic cough, weight loss, haemoptysis for PTB. Examination will aim at assessing the general clinical condition of the patient as they may be pale, wasted or malnourished. A complete examination of the spine is essential. This will include demonstration of deformities and neurological deficits such as paresis, abnormal tone and reflexes as well as lax sphincters.

Following clinical evaluation, the patient is investigated using relevant modalities. There exists a barrage of test that can be done to confirm the diagnosis of TB spine and rule out close differentials. They include laboratory, immunological and imaging studies. A tissue diagnosis is the single most important test in confirming TB.[5] Microscopic demonstration of acid and alcohol fast bacilli (AFB) with gram staining has a sensitivity 25%–75% and specificity of 99%. Also histologic evidence of characteristic granuloma with central caseous necrosis, epitheloid giant cell with surrounding lymphocytes is diagnostic of TB. This has a sensitivity of 53%–80%.[13] The tissue is harvested by image guided biopsy or at surgery.

MTB is a fastidious slow-growing organism and as such culture takes about 6–12 weeks.[11,13] It is done using the Lowenstein Johnson specialised growth medium. However, with BACTEC radiometric culture the turnover can be as low as 2 weeks.[13] AFB culture has a sensitivity and specificity of 47% and 100% respectively compared to BACTEC (56% and 100%).[13] Drug sensitivity testing has become very important with the rising incidence of drug resistant TB. Polymerase chain reaction (PCR) and Gene Xpert are newer diagnostic tests for TB. Gene Xpert MTB/rifampicin (RIF) test yields results as early as 90 min and has a sensitivity of 95.6% and specificity of 96.2%.[13,17] Other laboratory investigations include a complete blood count (relative lymphocytosis and anaemia), erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) which are non-specific marker of inflammation. And ESR of >20 mm/h has a sensitivity of 60%–90%.[13] It is also used for monitoring during therapy.

Immunological test for TB include Mantoux, Heaf and Tine tests. They detect immune reaction on the skin to purified protein derivatives (PPD) of TB. A positive PPD skin test has been reported in 62%–100% of TB spine cases.[5,13] Others include nucleic acid amplification testing, interferon gamma release assay like the QuantiFERON TB gold kits. Serological testing of immunoglobulin (Ig) M and IgG against TB is generally neither specific nor sensitive and is not recommended.[13]

Imaging studies commonly deployed in the diagnosis of spinal include plain radiography, CT scan and MRI. Conventional radiographs give a good overview; CT visualises the disco-vertebral lesions and paravertebral abscesses, while MRI is useful in determining the spread of the disease to the soft tissues and spinal canal. Of these, MRI is most diagnostic with the highest sensitivity and specificity.[8,13]

Plain X-ray has no place in the diagnosis of early disease. The earliest findings are radiolucencies and the loss of definition of the plate margins.[8] The commonest findings on plain films include wedge collapse of the vertebral bodies and destruction of the intervertebral disc.[2,8,13] Other findings include erosion of end plates, vertebral bodies, bone sequestration, sclerosis and paravertebral masses as well as calcification in paraspinal masses. In advanced stages of the disease possible features include bony ankylosis, vertebral collapse and anterior wedging, progressive kyphosis and gibbus deformity. The plain radiograph describes changes consistent with TB spine in 91%–99% of cases.[8] A chest X-ray is also essential as there is a coexisting PTB in about 30% of cases.[3] The angle of kyphosis, Figure 2 and spine at risk sign in children, Figure 3 are assessed on plain radiographs. There are two methods for measuring the angle of kyphosis as shown in Figure 2.[7]

The spine at risk sign in children features are shown in Figure 3.[13]

The findings on X-ray are better elucidated with a CT. It gives finer details of bony and ligamental changes as well as paraspinal abscess. CT demonstrates vertebral destruction earlier than plain films. It clearly identifies the extent of bony destruction, joint involvement, posterior column involvement and regional stability.[13] Four types of destruction have been noticed in decreasing order of frequency: Fragmentary, osteolytic, subperiosteal and localised sclerotic lesion.[8,13] CT is also of immense value in obtaining percutaneous CT-guided biopsy for establishing a diagnosis.[8] MRI has a high sensitivity with satisfactory specificity and it is the method of choice in

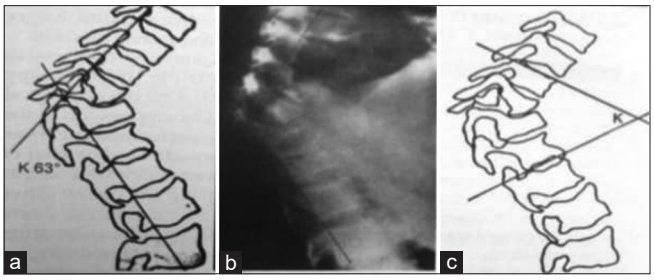

Figure 2: Measurements of Kyphotic angle: (a) Dickson method, a line is drawn along the posterior margin of the bodies of the healthy vertebral above and below the site of of disease, angle K is the angle of kyphosis; (b) Lateral spine radiography used for measuring the kyphotic angle and (c) Konstam and Blesovsky (Medical Research Council) method, is by determining the angle between the upper-end-plate of the normal vertebra proximal to the affected vertebrae and the lower-end-plate of the normal vertebra distal to the affected vertebrae. Angle K increases with increase in the angle of kyphosis. Derived from the internet, www.slideshare.net

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spinal infection. It is able to demonstrate abnormalities in early disease, when no other image modality shows lesions.[8] It is inferior in demonstrating bony changes but superior to CT in visualising extradural and subdural granuloma as well as intramedullary tuberculoma.[2] Radiological images of TB spine are shown in Figure 4.

Other imaging studies include contrast myelography and nuclear imaging like the ^{18}fludeoxyglucose positron emission tomography scan.

Therefore, making a diagnosis of spinal TB requires characteristic clinical features and classical imaging features on MRI. This is confirmed by histopathological evaluation, culture, PCR or Gene Xpert Test.[13] The presence of backache with an elevated ESR and end-plate and/or paraspinal disease on CT/MRI constitute a triad with a diagnostic sensitivity of 81.2%.[5] TB spine is a treacherous imitator. As such, further investigation may be necessary to exclude other pathologies. These include malignancy screening (e.g., prostate specific antigen), blood culture, myeloma screening, Brucella serology and bone biopsy.[5]

Treatment

Following the diagnosis of TB spine, appropriate and prompt treatment measures must be instituted. This is can be either conservative (non-surgical) or surgical treatment. Medical therapy with the use of anti-tuberculous agents is the bedrock of treatment and surgery is mainly supplementary, for complicated cases.[1,2,13] As matter of fact, uncomplicated spinal TB is considered a medical condition requiring chiefly medications.

Until recent years, spinal TB was managed by unorthodox means and then supportive care including absolute rest, decreased weight-bearing and immobilisation, and by promoting the natural processes of healing by general hygienic measures.[2] However, a lot has changed in the last 6 decades.

In the pre-antibiotics era, only 25% of patients achieved healing when treated in a sanatorium with attendant high mortality rate.[17] With the introduction of antibiotics, better control of the disease and healing became possible. With improvements in diagnostic modalities, safer surgical methods including modern spinal instrumentation as well as better theatre facilities, healing from spinal TB has greatly improved with little or no residual deformity.[17] Modern treatment will cure up to 90% of cases.[1]

Chemotherapy

Multidrug anti-tuberculous treatment is the mainstay of treatment in both complicated and uncomplicated TB.[1,13,16] First line drugs used include RIF, isoniazid, ethambutol and pyrazinamide (PZA). In treating spinal TB, a combination of these four medications for 2 months followed by the use of RIF and isoniazid for a total period of 6, 9, 12 or 18 months is the most frequent protocol used for treatment.[2]

The appropriate duration of treatment is still a subject of debate. The WHO recommend a treatment period of 9 months.

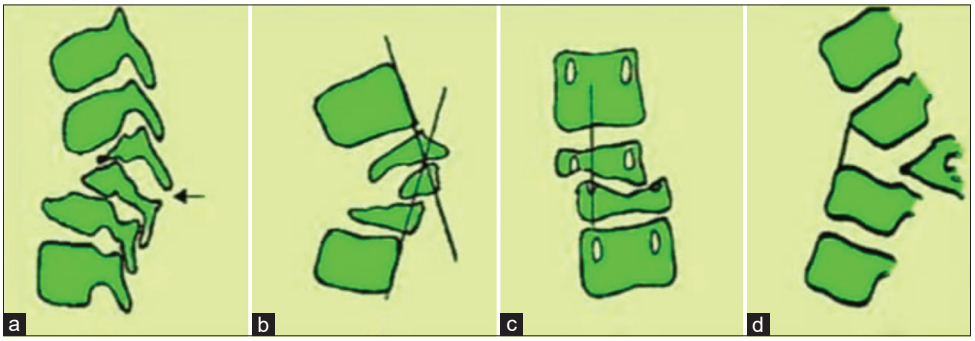

Figure 3: Spines at risk sign in children: (a) Separation of facet joint, (b) retropulsion, (c) lateral translation and (d) toppling sign. These signs of spinal instability in children are indications for surgical intervention in children with tuberculosis of the spine. Derived from internet, www.slideShare.net

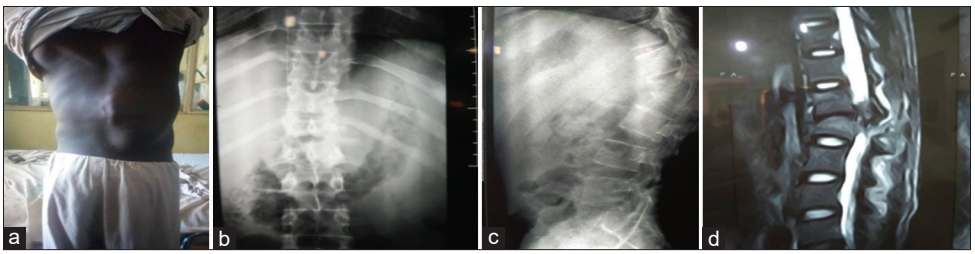

Figure 4: Radiology images of the tuberculosis spine in (a) a 20-year-old man with tuberculosis kyphosis in the thoracolumbar spine and paraspinal cold abscess extending to the right lumbar triangle and paraparesis (b and c) Plain radiography showing wedge collapse of L2 and kyphosis, (d) T2-weighted magnetic resonance imaging scan showing compression of the thecal sac, collapse of L2, degenerated disc at L1/L2 disc space and paravertebral abscess

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This divided into an 'initiation' phase of 4 drugs – isoniazid, RIF, PZA, ethambutol or streptomycin – are administered for 2 months and 'continuation' phase of 7 months using isoniazid and RIF.[13,19] The American Thoracic Society recommends 6 months in adults and 1 year in children of treatment with the same first drugs consumed for the first 2 months following by 7 months of therapy with isoniazid and RIF, while the Canadian Thoracic Society recommends a total treatment duration of 9–12 months.[2] The British Thoracic Society recommends 6 months of daily treatment (the 6-month-four-drug regimen), irrespective of age. Although 6 months of treatment is considered adequate, many experts still prefer a duration of 12–24 months or until radiological or pathological evidence of regression of disease occurs. To avoid poor compliance, direct observed treatment and short-course regimens may be administered. Albeit the majority of patients with spine tuberculosis are treated on ambulatory basis without the need for prolonged rest. The use of cast and brace which used to be popular had been abandoned. RIF and isoniazid are most effective at preventing drug resistance, hence stressing the importance of multiple drug therapy. Also, RIF and PZA are sterilising drugs as they kill extracellular intracellular, dormant and rapidly multiplying forms of the bacteria.[16]

The second-line anti-TB drugs include kanamycin, amikacin, capreomycin, levofloxacin among others. They are mainly indicated in multi-resistant TB. However, they are more expensive and have more side effects than the first-line drugs.[13] Directly observed treatments under the supervision of a health professional is recommended. It is found to have a better outcome than self-administered treatment.[16]

Specific indications for medical therapy include uncomplicated cases with minimal or no neurologic deficit, involvement of one disc space with no significant vertebral body destruction, minimal or no instability as well as the presence of medical co-morbidities such as sepsis or coagulopathy.[2] Chemotherapy has been found to be effective in treatment of spinal TB without neurological symptoms and forms the bedrock of treatment in this patient group.[1,2,16] Also, medical treatment without surgery can reverse paraparesis.[1,16] Further, when surgery is indicated, chemotherapy is also mandatory, if cure is to be achieved.[16] However, the use of chemotherapy alone has a higher occurrence of residual deformity.[2]

Other non-operative measures include use of salicylates, other analgesics (narcotics, non-steroid anti-inflammatory drugs). Correction of anaemia and malnutrition, mobilisation with a suitable orthotics as well as physiotherapy.

Surgical Treatment

In times past, surgery was the cornerstone of treatment of TB spine.[20] This has since changed as surgery is required less frequently, even in patients with neurologic deficits. This is due to the effectiveness of chemotherapy to successfully eradicate most cases of TB.[1] However, surgery is necessary in certain instances as an adjunct to medical therapy. The Indications for surgery include: Biopsy, spinal tumour syndrome (epidural spinal tuberculoma without osseous involvement) abscess causing pressure symptoms or unresponsive to 3–6 months of anti-TB medications, persistent or worsening neurologic deficits, spinal column instability and severe kyphotic deformities >60°.[2] Others include severe weakness at presentation, incapacitating pain and a pan-vertebral lesion.[2,17] The advantages of surgical intervention include early recovery, prevention and correction of deformity, lower recurrence as well as sampling for histology.[13] Early surgery in children is also associated with overall better outcome. Surgical interventions include drainage of abscess, debridement with radical clearance of a lesion, stabilisation with fusion/spinal instrumentation and correction of spine deformities. Drainage of cold abscess was initially fashionable. However, the use of multi-drugs therapy has changed that as most abscess resolves with adequate chemotherapy alone.[13,16] Indications for drainage include cervical paravertebral abscess with pressure symptoms (e.g., dysphagia, respiratory distress) and large psoas abscess with pseudo–hip flexion deformity recalcitrant to medical therapy.[13]

Debridement involves the extirpation of the affected parts of a vertebra. It takes the form of limited focal anterior surgery debridement or anterior radical excision.[20] It is usually supplemented with chemotherapy and fusion with or without instrumentation. As a stand-alone procedure, it is associated with residual deformity.[13] Debridement and fusion can be done through an anterior, posterior or combined approach.[2,13,20] Fusion is usually achieved by bone grafts in the form of iliac, rib or fibular autografts or fresh frozen allografts[2] or use of cages.

The use of spinal instrumentation in TB spine was a subject of controversy for a long time. However, recent studies have shown good outcome[2,13,20] including the use of Minimally Invasive Surgery as standalone procedure or in combination with open surgery in selected cases.[13] Instrumentation with a prosthetic is feasible because mycobacterium TB, unlike other bacteria, does not form biofilm. Furthermore, there is a low bacillary population in bones.[20] Instrumentation usually takes the form of rods, screws and plates. It commonly involves debridement, fusion and stabilisation via anterior, posterior or combined procedures. This can be done in one or multiple stages for the correction of kyphotic deformity.[2,13] However, a single stage posterior only approach is safe, effective in removing disease process, excellent in correcting and maintaining kyphosis and beneficial for patient in terms of less blood loss, less operating time and short duration of hospital stay compared to combined approach, Figure 5.

Prognosis

TB spine has a good prognosis if diagnosed early and treated appropriately. Cure is achieved with medical care plus or minus surgery in 90% of uncomplicated TB.[1] Indicators of poor prognosis include the presence of complications such as

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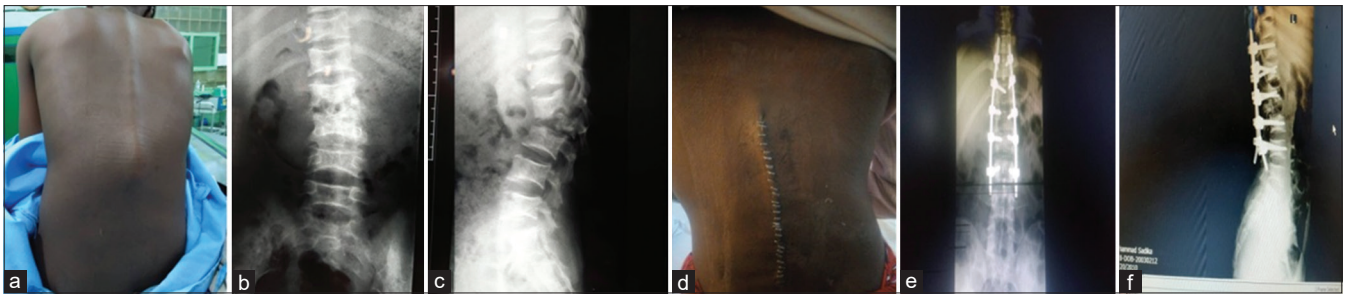

Figure 5: Surgical treatment of tuberculosis kyphosis and instability through a single stage posterior only approach in: (a) A 16-year-old lady with tuberculosis kyphosis of the lumbar spine, (b and c) Plain X-ray of the same lady showing kyphosis due to completer destruction of second lumbar vertebral body, d) Post-operative appearance of the same lady, (e and f) Post-operative plain X-ray of the same lady following decompression, osteotomy for correction of the deformity, posterolateral fusion and stabilisation with pedicle screws and rods construct. The lady was discharged home 2 weeks post-operative period with good cosmesis and intact neurology. Majority of our young patients with tuberculosis spine kyphosis aside pain and interference with marital functions, request for cosmetic surgery in the immediate perimarital period

neurological deficits, destructive collapse of vertebra as well as severe deformities.[21] Other predictors of poor outcome include low-income due to inability to purchase drugs, poor compliance with taking anti-TB drugs, drug-resistant TB mycobacteria, poor nutritional status, other concurrent systemic diseases, compromised stability of the spine and incomplete removal of lesions at surgery.[2,20] Long-term follow-up of patients is recommended. This should include serial imaging, ESR, lymphocyte count as well as clinical evaluation for resolution of symptoms.[10,17] Generally, healing occurs before it becomes evident on radiological imaging. In the absence of reliable serological and immunological markers of healing, the patient is considered 'healed' if there is evidence of clinical and radiological healing without recurrence after 2 years.[17]

Conclusion

The rising incidence of TB, especially in the developing world, mean surgeons will have to deal with more cases of spinal TB. Acquaintance with the clinical evaluation, investigation and diagnosis is indispensable. Spinal TB carries a good prognosis when detected and treated early. Delays can be associated with the development of complications including difficult-to-manage deformities. Multi-drug anti TB chemotherapy remains the bedrock of treatment. Surgery is supplementary and when indicated, takes the form of abscess drainage, debridement and fusion with or without instrumentation.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

References

1. Mbata GC, Ofondu E, Ajuonumam B, Asodike VC, Chukwumam D. Tuberculosis of the spine (Pott's disease) presenting as hemiparesis. Afr J Res Med 2012;8:18-20.
2. Rasouli MR, Mirkoohi M, Vaccaro AR, Yarandi KK, Rahimi-Movaghar V. Spinal tuberculosis: Diagnosis and management. Asian Spine J 2012;6:294-308.
3. Iyidobi EC, Nwadinigwe CU, Ekwunife RT. Management of musculoskeletal tuberculosis in Enugu Nigeria. Tropical Med Surg 2013;1:156.
4. Knechel NA. Tuberculosis: Pathophysiology, clinical features, and diagnosis. Crit Care Nurse 2009;29:34-43.
5. Yasaratne BM, Wijesinghe SN, Madegedara MD. Spinal tuberculosis: A study of the disease pattern, diagnosis and outcome of medical management in Sri Lanka. Indian J Tuberculosis 2013;60:208-16.
6. Monica C, Masae KL. ATS patient education series: What is tuberculosis? Am J Res Critical Care Med 2017;195:7-8.
7. Agrawal V, Patgaonkar PR, Nagariya SP. Tuberculosis of spine. J Craniovertebr Junction Spine 2010;1:74-85.
8. Rivas-Garcia A, Sarria-Estrada S, Torrents-Odin C, Casas-Gomila L, Franquet E. Imaging findings of Pott's disease. Eur Spine J 2013;22 Suppl 4:567-78.
9. Ahmed EG, Elbadawi NE, Ibrahim EK, Mohammed MM. Clinical presentation of pott's disease of the spine in adult Sudanese patients. J Med Microbiol Diagnosis 2013;2:120.
10. Akinyoola A, Adegbehingbe O, Ashaleye C. Tuberculosis of the spine in Nigeria: Has anything changed? Int J Third World Med 2006;4:9.
11. Harvey R, Champe PC, Fischer B, Lippincott's Illustrated Reviews: Microbiology. 2nd ed. New Jersey: Lippincott and Wilkins; 2007.
12. Smith I. *Mycobacterium tuberculosis* pathogenesis and molecular determinants of virulence. Clin Microbiol Rev 2003;16:463-96.
13. Rajasekaran S, Soundararajan DC, Shetty AP, Kanna RM. Spinal Tuberculosis: Current Concepts. Global Spine J 2018;8:96S-108S.
14. Robert L, Wani S. Tuberculosis: Pathophysiology and microbiology of pulmonary tuberculosis. South Sudan Med J 2013;6:10-2.
15. Agrawal A, Shanthi V, Mohan KM, Reddy GV. Lumbosacral spinal tuberculosis: A case report and review of literatures. Egypt Orthop J 2015;50:73-5.
16. Dunn R. The medical management of spinal Tuberculosis. South Afr Orthop J 2009;9:37-41.
17. Jain AK. Tuberculosis of the spine: A fresh look at an old disease. J Bone Joint Surg Br 2010;92:905-13.
18. Oguz E, Sehirlioglu A, Altinmakas M, Ozturk C, Komurcu M, Solakoglu C, *et al*. A new classification and guide for surgical treatment

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of spinal tuberculosis. Int Orthop 2008;32:127-33.
19. World Health Organization. Tuberculosis guidelines for treatment of Drug-susceptible tuberculosis and patient care. WHO; 2017. Available from: http://www.who.int/tb/publication/2017/d207_guidance_2017/en/. ISBN978-92-4-155000-0 WHO/HYM/2017.05. [Last accessed on 2019 Dec 25].
20. Myung-Sang M. Development in the management of tuberculosis of the spine. Curr Orthop 2006;20:132-40.
21. Cao G, Rao J, Cai Y, Wang C, Liao W, Chen T, *et al*. Analysis of treatment and prognosis of 863 patients with spinal tuberculosis in Guizhou province. Biomed Res Int 2018;2018;10:1-8. DOI:10.1155/2018/3265735.