POTT'S SPINE AND PARAPLEGIA

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ABSTRACT

Spinal tuberculosis is usually secondary to lung or abdominal involvement and may also be the first manifestation of tuberculosis. Spinal tuberculosis (often called Pott's disease) is by definition, an advanced disease, requiring meticulous assessment and aggressive systemic therapy. Physicians should keep the diagnosis in mind, especially in a patient from a group with a high rate of tuberculosis infection. This review aims on updating the knowledge on spinal tuberculosis and its management. Skeletal involvement has been reported to occur in approximately 10% of all patients with extrapulmonary tuberculosis, and half of these patients develop infection within the spinal column. Symptoms of spinal tuberculosis are back pain, weakness, weight loss, fever, fatigue, and malaise. It is much more prone to develop neurological manifestation, paraplegia of varying degree. The palpation of spinous process in routine clinical examination is the most rewarding clinical method and is an invaluable measure for early recognition. Diagnosis of spinal tuberculosis is made on the basis of typical clinical presentation along with systemic constitutional manifestation and the evidence of past exposure to tuberculosis or concomitant visceral tuberculosis. Magnetic resonance imaging can define the extent of abscess formation and spinal cord compression. The diagnosis is confirmed through percutaneous or open biopsy of the spinal lesion. Surgery is necessary as an adjunct to antibiotic therapy if the vertebral infection produces an abscess, vertebral collapse, or neurologic compression. Some patients need aggressive supportive care owing to tuberculous meningitis or encephalopathy. Moreover, the importance of immediate commencement of appropriate treatment and its continuation for adequate duration along with the proper counseling of the patient and family members should not be underestimated for successful and desired outcome.

Key Words: Tuberculosis, Pott's spine, Paraplegia.

INTRODUCTION

In April 1993, World Health Organization (WHO) has declared Tuberculosis as a global emergency because it was out of control in many parts of the world.[1] More than 3.8 million new cases of all forms of tuberculosis, 90 % of them from developing countries, were reported to the WHO in 2001.[2] However, because of low level of case detection and incomplete notification, reported cases represent only the tip of the iceberg and it is estimated that 8.5 million new cases of tuberculosis occurred world wide in 2001, 95 % of them in developing countries. It is also estimated that 1.8 million deaths from tuberculosis occurred in 2000, 98 % of them in developing countries.[2]

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South Asian Association for Regional cooperation (SAARC) region bears 22% of the global population and 29% of the global burden of tuberculosis. It is estimated that SAARC region has 2.5 million new cases and 0.6 million death per year.[3] It is the leading cause of death in adults of productive age in developing countries including Nepal.[4] At present there are 80,000 tuberculoses patients including 44,000 new tuberculosis cases in Nepal and 9,000 cases die of tuberculosis each year.[4] Skeletal involvement has been reported to occur in approximately 10% of all patients with extrapulmonary tuberculosis, and half of these patients develop infection within the spinal column.[5,6] Up to 45% of patients with spinal tuberculosis develop corresponding neurologic deficits.[7]

It is a preventable disease to some extent and totally curable if the treatment is started in early stage. For successful management of tuberculosis the patient and his family should understand the importance of early recognition of disease and adherence to the prescribed regimen of antitubercular treatment.[8] There are a lot of undesirable consequences of tuberculosis due to late presentation with advanced sequel or due to non-compliance to the prescribed regime. Among the cases of tuberculosis, extrapulmonary cases remain neglected from the National Tuberculosis Program because they are pauci-bacillary and mostly non- contagious and therefore, do not constitute a public health problem. In addition to this, extra-pulmonary tuberculosis is difficult to diagnose and convince patients about its presence and need on treatment due to lack of definite evidence, leading to late presentation and initiation of treatment. This is more applicable in the case of spinal tuberculosis where outcome depends on the early recognition of this illness and immediate commencement to the appropriate treatment.[9]

The aims and objectives of this review article is to emphasize on the importance of Pott's spine, its early recognition and early initiation of treatment by reviewing the past and present work till date and to outline the method of early diagnosis, early treatment and to save from its disability and sequel.

REVIEW

Extra-pulmonary tuberculosis, especially spinal tuberculosis is still prevalent in developing countries and it is most common during first three decades of life.[7] Tuberculosis of the spinal column was first described by Percival Pott in 1779. The classic destruction of the disc space and the adjacent vertebral bod

Fig.1 : Dorso-lumbar spine – AP view of a patient with Pott's spine with paraparesis showing regional osteoporosis of the bodies of T9 and T10 with radio lucent shadow in the body of T10 and reduced intervertebral disc space between T_9 and T_{10}. The margins of the adjacent vertebral bodies are normal, and there is no paravertebral soft tissue shadow

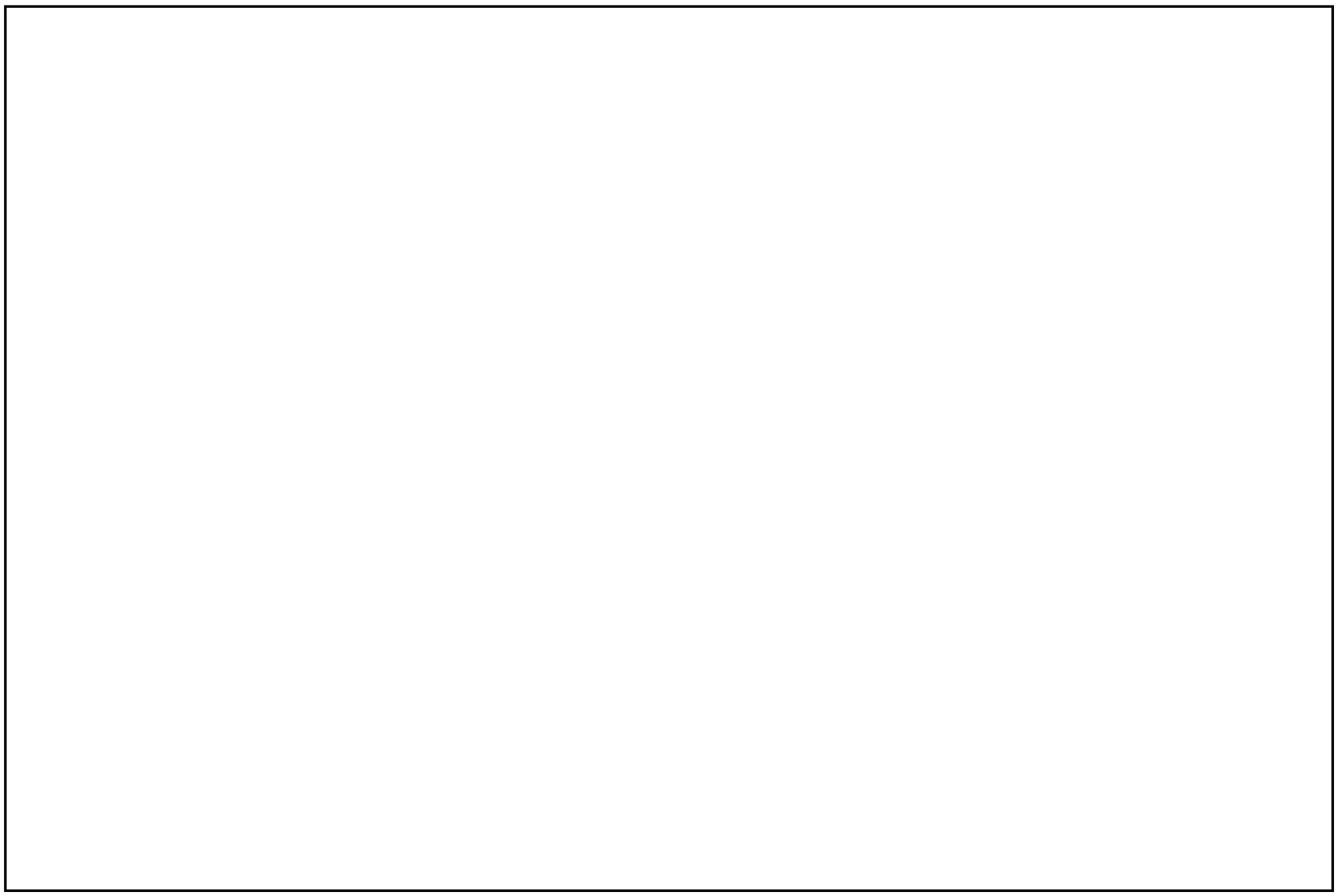

Fig.2 : Tuberculous bacilli spread to the disc space from surrounding tissues (contiguous spread) or through the vascular supply (hematogenous spread).

ies, collapse of the spinal elements, and severe and progressive kyphosis (Figure 2) subsequently became known as Pott's disease.

In developing countries, spinal tuberculosis affects mostly children and in some instances elderly persons. In contrast to this, adult infection has become more common in North America, the Middle East, and Europe, places where the overall incidence of the disease is lower.[11,12]

PATHOGENESIS AND CLINICAL MANIFESTATION

Spinal tuberculosis can occur:

1. Usually secondary to tuberculosis elsewhere by hematogenous or lymphatic spread, most commonly from a pulmonary lesion or from an infection of the genitourinary system.[9,13]
2. By contiguous extension from a pulmonary abscess, commonly leading to thoracic spondylitis.
3. As primary infection. This is being increasingly reported, possibly by ingested bacteria reaching there by heamatogenous route from gastro-intestinal tract.

The detection of associated visceral tuberculous lesion varied from 40 to 50 percent in different studies.[14] Majority of patients have typical paradiscal lesions characterized by destruction of adjacent bone end plates of the bodies and diminution of the intervening disc, most common site being the lower thoracic and thoraco-lumbar region in adults.[15] The involvement of large number of adjacent vertebrae produces a severe kyphotic deformity.[16] Spinal tuberculosis above L_1 lumbar vertebra is associated with neurological manifestation in 10 to 45 percent of cases, the higher prevalence being in patients with thoracic spinal tuberculosis.[9]

The progression of spinal tuberculosis is usually slow and insidious, and its main symptom; backache - is nonspecific. Therefore, spinal tuberculosis is more difficult to recognize than other acute infections (e.g. staphyloccal or streptococcal osteomyelitis). Considerable delay in diagnosis may occur before an infectious process is even considered. Even when spinal tuberculosis is considered, it may be difficult to confirm. Before the diagnosis is established, the vertebral bone and disc material may sustain extensive destruction. Progressive vertebral collapse and fracture can lead to spinal deformity and a classic Pott's kyphosis. Neurologic deficits are common with long-standing thoracic and cervical involvement, and if untreated, neurologic involvement may progress to complete paraplegia.

In developing countries, spinal tuberculosis and its complica-

tion Pott's paraplegia is still common. Many patients reach hospital with advanced neural deficits. The duration of symptoms of the illness at the time of presentation in the hospital varies from few months to few years, seeking advice only when there is severe pain, marked deformity or neurological complications.

Tubercular spondylitis, uncomplicated by neural deficit, is classified into the following varities according to the radiological involvement of the vertebra:

1. Paradiscal: This is the commonest variety and involves the adjacent margins of two consecutive vertebrae. The intervening disc space is reduced and the vertebral margins appear fuzzy. The infection is believed to be via the arterial blood supply, which is segmental and follows embryonic pattern that supplies inferior half of superior vertebra and superior half of inferior vertebra.
2. Central: This involves the central portion of a single vertebra, keeping the proximal and distal disc spaces intact. The possible lodgment of the infection comes via the venous route in this variety.
3. Anterior marginal: The lesion begins as a destructive lesion in one of the anterior margins of the body of a vertebra, minimally involving the disc space but not involving part of the vertebra on either sides.
4. Posterior: The disease localizes itself in the posterior elements i.e. the lamina, pedicle or the spinous process. The infection is said to be coming via the arterial supply to these structures. There is no involvement of the body of the vertebra. This variety many a times may present primarily as neural deficit without any lesion in the body. The clinical differentiation comes by the presence of an acute posterior midline spinal tenderness over the involved vertebra, much more than the vertebral body lesions.
5. Synovial: The disease involves synovium of atlanto-axial and atlanto-occipital joints.

All the above varieties can progress to other parts of the same vertebra or to the adjoining vertebrae if left untreated. All these varieties can ultimately lead to a panvertebral disease and pathological dislocation in advanced cases, in addition to the neural consequences of the disease.

The clinical presentation of spinal tuberculosis is extremely protean. The type and severity of symptoms vary depending on the level of involvement, the severity of the disease, and the duration of the infection. Patients usually present with a combination of constitutional manifestations such as weight loss, fever, fatigue, and malaise, as well as focal back pain. Most of the patients present with relatively moderate and chronic symptoms despite severe vertebral destruction. The pain varies from mild and constant to severe and activity-related. Pain is typically localized to the site of involvement and is most common in the thoracic spine. It can be constant and indolent, reflecting the progressive destruction of the involved disc space and vertebral elements, or it can be intense and directly linked to spinal motion, coughing and weight bearing, which is caused by more advanced disc disruption and spinal instability, nerve root compression, or pathological fracture.[17,18] Patients with lumbar disease may develop an anterior abscess, which may track into the psoas muscle. Patients with a “psoas sign” tend to lie with the leg drawn up in a flexed position, and they experience exquisite pain when the hip is extended to a neutral position. An abscess within the spinal canal may compress the cord or cauda equina, and neurologic symptoms may develop rapidly. Depending on the level of involvement, a spinal abscess may cause nerve root symptoms mimicking a herniated disc, or it may produce acute and progressive spinal cord compression, resulting in paraplegia or quadriplegia if untreated.[19-21] In rare cases, meningitis develops in association with spinal disease, but most experts feel that meningeal involvement represents hematogenous spread rather than direct infection. Meningitis is suggested by headache, nausea, vomiting, photophobia, and changes in level of consciousness. More severe symptoms, including seizure, acute hydrocephalus, and cranial nerve palsy, and in rare instances cavernous sinus syndrome, are associated with a higher mortality rate and frequent neurologic sequelae.

At first, neurologic symptoms of spinal tuberculosis may be subtle, but will progress over time. Compressive myelopathy is the most common neurological manifestation of Pott’s spine. Numbness and tingling in the lower extremities, numbness or paresthesia in a belt-like distribution around the chest wall, or a subjective sense of weakness with activity are the earliest features. With the progression of disease, symptoms become more prominent. Neurological complication may appear due to recrudescence of the disease or mechanical pressure on the cord because of tubercular caseous tissue, tubercular debris, and sequesters from vertebral body and disc, internal gibbus, stenosis of the vertebral column or severe deformity. As a rule, more than one cause may be acting in the same case. Motor functions are always affected before and to a greater extent than the sensory functions because the diseased area in the spine lies anterior to the cord thus being nearer to the motor tracts along with high sensitivity of motor tracts to the compression of cord.[9]

The level of spinal cord involvement determines the level of impairment. When cervical tuberculosis progresses and causes cord or root compression, the earliest signs are weakness, pain, and numbness of the upper and lower extremities. Progressive deformity or abscess gradually increases pressure on the cord, and symptoms eventually progress to full-blown quadriplegia. If the thoracic or lumbar spine is involved, upper-extremity function remains normal while lower-extremity symptoms

progress over time. As cord compression becomes more severe, subjective symptoms give way to objective findings of motor weakness, hyperreflexia, and reflex abnormalities, including an extensor plantar response and sustained clonus. If a lumbar abscess or vertebral collapse results in compression of a single nerve root, symptoms may mimic those of a herniated disc. These patients experience radicular pain radiating over the distribution of the affected nerve root, and they may experience weakness in a specific motor distribution. Unlike a true herniated nucleus pulposus, in which symptoms typically increase with activity and decrease with rest, the radicular pain secondary to tuberculosis infection tends to be intractable and constant. Patients with cauda equina compression due to lumbar infection have weakness, numbness, and pain, but have decreased or absent reflexes among the affected muscle groups, in contrast to the hyperreflexia seen with spinal cord compression.

Paralysis may pass through the stages of spastic motor paraparesis, spastic paraplegia in extension and spastic paraplegia in flexion. In advance cases there may be involvement of bladder and anal sphincters along with varying degree of sensory deficit, areflexic paraplegia with anaesthesia and loss of sphincter control being the extreme manifestations.

True synovial tuberculosis in the spine is seen in the atlantoaxial area, which has a relatively large synovium in the facet joints. This may present as rotary subluxation of the C_1-C_2 area. In uncommon situations, there may be no bony disease and the patient presents with neural signs only. Such a compressive myelopathy can be due to a tubercular granuloma in the extradural space. This variety has been labeled as the 'spinal tumour syndrome' type of spinal tuberculosis. Sudden and acute onset paraplegia or rapidly progressing paraplegia in either a preexisting disease or without a preexisting disease can be due to an acute infective thrombosis of the anterior spinal artery and has the poorest of prognosis as far as neural recovery is concerned. Another uncommon variety called tubercular myelitis is also described which involves the cord alone, without any of the above-described entities. The presentation of this variety is that of paraplegia and the diagnosis confirmed on MRI only.

DIAGNOSIS

Diagnosis of spinal tuberculosis is made on the basis of typical clinical presentation along with systemic constitutional manifestation and the evidence of past exposure to tuberculosis or concomitant visceral tuberculosis. Confirmation of tuberculosis is done with the help of various radiological and laboratory investigations. The evidence of personal exposure to tuberculosis makes the diagnosis more obvious in presence of suggestive clinical features. Immigrants from developing countries, persons frequently visiting such region or having guest from these regions have higher possibility of contracting tuberculosis. The homeless, IV drug users, alcoholic, and chronically ill may develop active tuberculosis. These people are often immunosuppressed, suffer from malnutrition, have a higher incidence of exposure to other infected individuals, and either avoid medical care or fail to complete treatment when it is offered. Cumulatively, these factors increase the risk of chronic infection and of infection resistant to multiple drugs. Patients, who are immuno-compromised, due to either therapeutic medical suppression or HIV infection, are at high risk of developing active tuberculosis, as well as a number of other granulomatous infections otherwise rarely seen. Organ transplant recipients, patients on long-term steroid therapy, and patients undergoing chemotherapy for cancer treatment are all at increased risk of disease.

Clinical evaluation may reveal the evidence of active or latent tuberculosis in other organs. Examination of the spine may reveal focal tenderness over the spinous processes, with more diffuse back spasm in the region of pain. Fluctuation, redness, or focal heat are rare, as the spinal infection typically involves the anterior column of the spine. Range-of-motion testing, on the other hand, may produce severe pain, and the patient may guard aggressively against any twisting, bending, or extension motions. Patients are usually most comfortable lying down, and they may have moderate to severe symptoms when standing upright or walking. In advanced disease, focal kyphosis can be seen on physical examination, usually in the midthoracic to thoraco-lumbar spine. The sharp angulation results in prominence of the spinous processes at the level of the vertebral collapse, forcing the patient to stoop or lean forward. In severe kyphosis, the patient may not be able to stand without leaning with the hands on a walker or desk.

Plain anterior-posterior and lateral radiographs should be the first imaging studies ordered in any patient with chronic, progressive back pain. In patients with tuberculous spondylitis, radiographic findings depend on the extent and duration of infection. Initial radiographs may be entirely normal, but over time, disc space narrowing and end plate rarefaction both become prominent (Fig.1). A chest X-ray is mandatory as spinal infection is usually secondary to pulmonary infection. Central body type of tuberculosis preserves disc spaces entirely in up to 50% of cases unlike the pyogenic infection hence is sometimes confusing with tumor.[22] In these cases, central rarefaction of the vertebral body inevitably progresses to vertebral collapse and kyphosis.[23,24]

CT scan and MRI help in detection of lesions earlier and show the extent of lesion but lack the specificity in diagnosis. CT scan in spinal tuberculosis has its value in detecting the lesion

in areas otherwise difficult to be seen on X-rays like posterior arch, craniovertebral, cervicodorsal, lumbosacral and sacrococcygeal junctions and sacrum. It also shows bony extent of the lesion. Although Magnetic resonance imaging (MRI) with gadolinium enhancement is suggested to be the ideal modality of diagnostic study after plain radiography, this is controversial.[25] It may be sensitive since it can show disease in predestructive inflammatory stage, but is highly nonspecific. MRI demonstrates relative sparing of the disc space and, at the same time, involvement of the vertebral bodies on either side of the disc, a rare finding in malignant disease. Dissection of the anterior soft tissues, with abscess formation and collection and expansion of granulation tissue adjacent to the vertebral body, is highly suggestive of tuberculosis. MRI studies are more able to reveal epidural abscesses, compression of the nerve root, or compression of the spinal cord.[25,26] With augmentation it can differentiate between granulation tissue mass and liquid pus. Similarly it can show existence of myelitis or lepto or pachymeningitis that otherwise will compromise with expected neurological recovery following surgery for decompression.

Confirmation of diagnosis of tuberculosis is made either by demonstration of acid fast tuberculous bacilli (AFB) on pathological specimen or histological classical picture of tubercle or even mere presence of epitheloid cells on the biopsy material. The obtainment of biopsy material, however, may not be as easy as in pulmonary tuberculosis with sputum. In few cases the acid-fast bacilli staining may show acid-fast organisms in the initial clinical specimen, but these may not be present in every case. Tuberculous bacilli grow slowly in culture, and confirmation may not be available for 6 to 8 weeks.[27] However, newer methods of culture in liquid media with radiometric growth detection (BACTEC 460) and the identification of isolates by nucleic acid probes or high-pressure liquid chromatography of mycolic acid have replaced older methods of AFB detection making reports available in 2 to 3 weeks. Several test systems based on amplification of mycobacterial nucleic acid are available and reports could be available in few hours. However, their applicability is limited by low sensitivity (lower than culture but higher than microscopy) and high cost.[2] Although these tests are recommended for the rapid confirmation of tuberculosis in persons with positive sputum, they may also have utility for the diagnosis of extrapulmonary tuberculosis in selected patients.

Polymerase chain reaction (PCR) testing is highly specific for tuberculous bacillus, and provides rapid confirmation of a positive culture. It is so specific, however, that it may overlook other species of Mycobacteria, and is only approved for use with pulmonary specimens.[28,29] In a conducted study, the sensitivity, specificity, positive predictive value and negative predictive value of PCR were shown to be 84%, 93.5%, 80.8% and 94.9% respectively.[30] It has also been concluded that there is no need for PCR test for the smear positive cases and it could be a possible diagnostic tool for the confirmation of the smear negative cases that show clinical symptoms of TB. One of the most promising uses of PCR is to detect mutations in the rpoB gene associated with resistance to rifampicin.

Skin testing with Protein Purified derivative (PPD) is most widely used in screening for M tuberculosis infection. The test has limited diagnostic value for the detection of active tuberculosis because of its low sensitivity and specificity. False-negative reactions are common in immunocompromised patients and in those with overwhelming tuberculosis. Positive reactions are observed when patients have been infected with M tuberculosis but do not have active disease and when persons have been sensitized by non-tuberculous mycobacteria or Bacilli Calmette Guerin (BCG) vaccine. In the absence of history of BCG vaccination, a positive skin test may offer additional support for the diagnosis of tuberculosis in culture-negative cases. Some experts support the use of skin testing to help guide individual decisions regarding preventive therapy, and some propose that PPD skin testing be performed for patients previously classified as anergic due to HIV infection if evidence indicates that these patients' immune systems have responded to therapy with antiretroviral drugs.

BCG test - use of BCG vaccination with an aim to detect active tuberculosis by observing early vaccine reaction, can be used as a diagnostic test for tuberculosis and has been recommended as such by the World Health Organization, especially in children.[31] Udani et. al. compared the BCG test with tuberculin testing and observed that the BCG test was positive in all children with tuberculosis, whereas 28.8% of tuberculin skin tests were negative.[32] Subsequent studies in children up to 12 years of age confirmed the greater sensitivity of the BCG test (81-92% sensitivity, 82.5-100% specificity), compared with tuberculin (sensitivities 45.5-52.3%, with specificities comparable to the BCG test).[33,34,35] The BCG test was especially helpful in children who were malnourished or who had tuberculous meningitis or miliary disease.

Other methods such as cytokine release assays and serologic tests also have been studied. At present, a commercially available whole blood cytokine assay, the QuantiFERON-TB test (Cellestis Ltd), has aided in the diagnosis of latent tuberculosis infection. Serologic tests such as ELISA (Mycodot), based on the detection of antibody to a variety of mycobacterial antigens, have low predictive value when used in a population with low probability of disease and are generally insufficient to be diagnostic.[2]

When the clinical suspicion of spinal tuberculosis is high and radiographic studies show a destructive lesion warranting surgical treatment, open debridement of the lesion may provide ample material for culture and diagnosis. However, if the process is caught earlier in its development, there may be no indication for surgical intervention. In this case, needle biopsy guided by computed tomography or MRI may provide diagnostic material. A fine needle can be introduced into the abscess cavity through the posterior muscular wall under imaging guidance and if a fluid abscess is encountered, material can be drawn through the fine needle without difficulty. When granulation tissue is encountered, a trocar may be appropriate to obtain an adequate tissue specimen. If both fine needle and trocar biopsy are unsuccessful, percutaneous biopsy can be done to obtain the tissue for histological diagnosis and culture. Imaging guided needle biopsy is a standard procedure, and an ultrasonography guided drainage of liquid cold abscess is feasible and safe.

TREATMENT

Multidrug therapy remains the cornerstone of tuberculosis treatment, irrespective of skeletal involvement. For patients with spinal infection, the goals of treatment are to eradicate the disease, to prevent the relapse and to avert or correct neurologic deficits and spinal deformities. The combination of Isoniazid (5 mg/kg, max 300 mg), Rifampicin (10 mg/kg, max 600 mg), Ethambutol (15 – 25 mg/kg), and Pyrazinamide (15 – 30 mg/kg, max 2 g) can overcome organisms resistant to Isoniazid alone or to other combinations of antibacterial drugs. Streptomycin (15 mg/kg, max 1 g) is sometimes used instead of Ethambutol. Pyridoxine (vitamin B_6) should be given concurrently to reduce the risk of peripheral neuritis, which may occur in 2% of patients taking isoniazid.[36]

Uncomplicated tubercular spondylitis alone, without neural complications is treated conservatively by antitubercular drugs and orthotic supports on an outpatient basis. In the intensive care setting, additional drugs may include Amikacin, Quinolones or newer macrolides. Supportive care is crucial in stabilizing the patient until chemotherapy provides benefit. Steroids are occasionally used in patients presenting with pericarditis or meningitis. Although comparative clinical trials of treatment for extrapulmonary tuberculosis are limited, the available evidence indicates that most forms of disease can be treated with the 6 – month regimen recommended for patients with pulmonary disease. The American Academy of Pediatrics recommends that children with bone and joint tuberculosis, tuberculous meningitis, or military tuberculosis receive a minimum of 12 months of treatment.[2] The Nepal National Tuberculosis Programme recommends 9 months duration (2HRZE +7HR) of treatment for spinal tuberculosis with neurological signs.[37] Spinal tuberculosis is a paucibacillary disease hence the insidious course of the disease. The M Tuberculosis bacilli in this case are known to be slow growers. Hence the persisters are rule rather than exception after short course of antitubercular therapy (ATT) like 9 months. Therefore ATT for spinal TB must be given for longer period, long enough not only to kill those slow growers but also to kill those persisters, to cure the disease and to prevent relapse. This period varies from 18 months to 2 years. Patients who do not complete a full course of therapy are at risk for relapse and drug-resistant infection. Newer forms of antitubercular drugs are coming up. Second-line drugs such as Cycloserine, Ethionamide, PAS, other Aminoglycosides and the Quinolones may also be used in patients who do not tolerate one of the first-line agents or have developed resistant. The selection of these drugs is based on regional patterns of sensitivity, side effect profiles, and mechanism of action. Although multidrug treatment is now the standard approach to initial care, once definitive sensitivities are established, some of the drugs may be discontinued. Medical management of tuberculosis also include immunomodulation therapy which has been shown to be effective in spinal cases.

If a patient develops fresh neural deficit while on such a treatment schedule, a urgent surgical intervention is needed. If the patient presents with primary neural deficit, the treatment depends on the severity of the neural deficit. Tuli et al [9] have classified the neural deficit due to tubercular disease of the spine based on clinical signs.

Grade I - Patient has no perception of his neural deficit. Only the clinician detects signs of early spasticity.

Grade II - Patient perceives some difficulty in walking but can manage his routine mobility.

Grade III - Patient cannot walk without support or may not be able to walk at all. Sensory deficit is present. No involvement of bladder or bowel is seen.

Garde IV - Patient has signs of grade III along with bladder and bowel involvement.

When a patient primarily presents with grade I or II paraplegia, he should be put on antitubercular drugs on an indoor basis and the neural status is monitored daily for progress. In case no improvement is seen within three weeks time, a surgical decompression should be considered. While new drugs are effective for most cases of spinal tuberculosis, surgical intervention is necessary in advanced cases with extensive bony destruction, abscess formation, or neurologic compromise.[38,39] Paraplegia of higher grade is a primary indication of decompression under the cover of ATT given for minimum 48 hours. The goal of surgery is to prevent or correct neurologic deficits and spinal deformities. Surgery also facilitates successful chemotherapy, since the abscess cavity provides an avascular environment that protects bacilli from systemic antibiotics. The

guiding investigation should be MRI as it is helpful in pinpointing the site and cause of compression. It can also differentiate between the spinal tumour syndrome and anterior spinal artery thrombosis. There is no indication of decompression in the later entity. When surgery is needed, results are best earlier in the disease process, before scarring and fibrosis develop. Later, dense scarring causes adhesions to the great vessels or vital structures, making dissection and surgical exposure dangerous. Progression of a lower grade of paraplegia in to a worse grade or involvement of sphincters is an indication of urgent surgical decompression. The clinical response to surgery is also faster and more complete in patients with active disease compared with those with chronic disease and deformity.

Every cases of neurological complication with tuberculosis of the spine warrants immediate attention and care by a team of orthopaedicians and physicians in a specialized center with all needed facilities for spinal surgery.[15] Every patient with neural complication will not be cured by antitubercular drugs and rest alone, however all patients do not need surgical decompression.[16] Early onset paraplegia with partial cord involvement in a young patient with good general condition and active disease with slowly developing or short duration of neurological complication along with kyphotic deformity of less than 60^0 are associated with better prognosis if treatment is started early.[14] No patient is considered to be too advanced for treatment because spontaneous recovery of neural complication was observed in 48 percent of cases on antitubercular drugs and bed rest alone in Tuli's series,[9] although none of the patients who had paraplegia due to severe kyphosis of more than 60^0 showed complete neural recovery, majority showed partial improvement. To prevent these complications, it is mandatory to keep a high index of suspicion for spinal tuberculosis in all possible situations such as in patients with pulmonary tuberculosis or extra pulmonary tuberculosis whether active or healed.

Surgical Management

The classic surgical approach to spinal tuberculosis, first described by Hodgson and Stock in 1956, is by anterior exposure of the spine.[40] The anterior approach allows the surgeon to remove all infected and devitalized bone and any material created by the infection. It is carried out either through the chest wall or via a retroperitoneal approach to the thoracolumbar or lumbar spine. Radical removal of diseased bone and disc is necessary to provide a solid substrate for reconstruction. Debridement removes the entire vertebral body, along with the posterior longitudinal ligament and any epidural abscess or granulation material compressing the neural elements. Spinal alignment is manually corrected, and a strut is placed between the remaining healthy vertebral bodies to maintain normal alignment and stability. In the past, the appropriate strut was always harvested from the patient's own iliac crest, but more recent experience has shown that allograft bone can also be used successfully. After anterior strut graft reconstruction, a second operation is usually needed to stabilize the spine and allow early ambulation and activity. Posterior instrumentation and fusion, following the anterior decompression and reconstruction, prevent recurrent kyphosis and protect the anterior construct from collapse. The staged anterior and posterior procedures consistently restore neurologic function when deficits are incomplete, and prevent or correct kyphosis in most patients.[41] Once the second procedure is completed, the patient can be up and out of bed immediately for ambulation and rehabilitation. Prolonged bed rest is strongly discouraged. Surgery alone neither eradicates local disease nor treats the systemic infection. Drug therapy remains the cornerstone of successful therapy.

Paraplegia of delayed onset or paraplegia of healed disease

Severe deformities of the spine, especially due to spinal tuberculosis of childhood can cause paraplegia due to reactivation of the old disease in adulthood or due to mechanical causes related to the deformity. The prognosis of this type of paraplegia is questionable. Antitubercular drugs may or may not help in neural recovery depending upon the cause of neural deficit. If the paraplegia is due to mechanical cause, corrective surgery may have a role in restoring functional ability.

Among other sequalae and complications of spinal tuberculosis, para vertebral abscess and deformities of spine like kyphosis are also equally common and warrants special management. They could also be associated with varying degree of neurological deficits.

The major objective of this communication is to emphasize on the prevention of paraplegia and relapse in tuberculous disease of the spine, which is of paramount importance. It can be largely achieved by early diagnosis of spinal caries and its prompt and suitable treatment for adequate duration.[16] Symptoms and signs may be minimal even in cases of active vertebral disease. A history of tuberculosis in the patient or his family should raise suspicion of tubercular nature of the spinal disease.[16] Tuberculosis as a cause of persistent backache must be remembered if we aim to diagnose the condition early.[7] If the clinician makes it a routine procedure to palpate the spinous process by moving his finger tips from the cervical spine to the sacrum he would be able to detect even a small knuckle by palpation of a step or prominence, thus diagnosing a case before gross destruction has taken place.[14] Backache in any patients should be carefully evaluated before blaming to the trivial causes or spondylotic process. In economically underdeveloped countries like ours, where tuberculosis is a public health problem with infectivity of 45% of total population, 10 % of them developing disease, high degree of suspicion should be kept on tuberculosis in all relevant situations. Since, most of the spinal and other tubercu

losis cases are lurking in remote areas rather than in cities, undiagnosed, under diagnosed or misdiagnosed, health workers working in such places should adhere to the following rules:

- Remember tuberculosis is a common disease in our region and uncommon presentation of common disease is a rule rather than uncommon presentation of uncommon disease.
- Do not neglect the backache and make every endeavor to reach to the diagnosis.
- Evaluate the patient fully with all possible and necessary investigations including chest X-ray.
- Make a routine to palpate the spinous process by moving fingertips from the cervical spine to the sacrum in any backache cases.
- Make a habit to seek help from specialist in difficult situations, remember that your patient serves for the best available management and you are supposed to guide him on this matter.
- Counsel the patients about long course of treatment with ATT and the importance of good nutritional support.

With the increase of HIV-infected cases, tuberculosis is going to be the major public health issue in the near future and difficult cases of tuberculosis like multidrug resistant tuberculosis is going to be accumulated in the community. The combination of different preventive measures, early recognition and early and adequate treatment should be the strategy for the goal of tuberculosis control.

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