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Paraplegia in Pott's Spine: Evidence-based Management Strategy

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Abstract

Study Design: Review article

Objective: To present a review of literature on the epidemiology, pathophysiology, diagnosis and outline evidence-based management strategy for paraplegia in Pott's spine

Methods: A systematic literature search was performed using PubMed systematic Reviews. Studies published over the last 20 years were analysed and conclusions were drawn. The search was conducted using key words "Tuberculosis of Spine", "Paraplegia" and "Management".

Results: Early diagnosis and timely initiation of treatment is the key to successful treatment of Spinal Tuberculosis (STB) with neurological deficit. Surgery along with appropriate chemotherapy is the mainstay of treatment when STB is complicated by neurological deficits. In cases of late onset neurological deficit, the decision regarding the surgical approach is based on the severity and location of the deformity, and the nature of pathology causing the neurological deficits. Surgery is warranted in case of the early-onset spinal TB complicated by neurological deficits, disease compromising the spinal stability, spinal deformity and in patients presenting with progressive neurological deficits. Surgery for late onset neurological deficits and deformity are associated with complications like worsening of the neurological deficits, persistence of the deformity and suboptimal outcome.

Conclusions: Tuberculosis is a medical disease and chemotherapy is the cornerstone of treatment. Surgery in early disease for neurological deficits/spine instability is associated with good neurological recovery. Surgery in late onset disease is necessary to correct deformity and arrest progression of neurological deficits, However is associated with inferior outcomes

Keywords: Spinal tuberculosis, Pott's paraplegia, Neurological deficits, chemotherapy, Late onset paraplegia, kyphosis, Surgical approach.

Introduction

Neurological deficit (ND) is considered the most feared complication of Spine tuberculosis (STB). The references towards neurological deficit in STB have been noted in ancient writings of 'Rig Veda' and 'Atharva Veda' and date as far back as 3500- 1800 BC. In 1799, Sir Percival Potts described STB as "That kind of palsy of lower limbs which is frequently found to accompany a curvature of the spine", hence was coined the term 'Pott's Spine'[1].

The incidence of ND in STB varies from 10-20% in developed countries, to as high as 20-40% in underdeveloped countries [2]. The occurrence varies with the region of spine involved. The incidence of ND is the highest in the thoracic region, because of the narrow spinal canal and physiological thoracic kyphosis [3]. Conversely, in the lumbar spine it is infrequent due to the capacious spinal canal.

ND can be considered broadly under two scenarios (a) ND in early-onset disease and (b) ND in late-onset disease [4]. The approach to management of ND in STB can vary from conservative approach with Antitubercular (ATT) drugs alone; to radical debridement with instrumented stabilisation. ND in STB not only alters the morbidity, and mortality related to the disease process but also adds to the financial burden in the management of such cases. This literature review will discuss the path of physiology, staging, treatment strategies and management of ND in STB.

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Pathophysiology

ND with early-onset disease typically occurs early in the course of the disease (<2 years). However, ND with late-onset disease usually occurs many years after the disease has healed, quite often with an accompanying residual spinal deformity. ND in late-onset disease may on occasion occur with apparent dormancy of the disease process or rarely with reactivation of disease activity in the previous healed focus of the disease.

ND in early-onset disease

The compromise of the neural structures in early-onset disease is due to the following factors

A. Mechanical compression by granulation tissue, caseous tuberculous debris and abscess [5].

B. Spinal instability due to significant anterior column destruction and/or pathological subluxation/dislocation of

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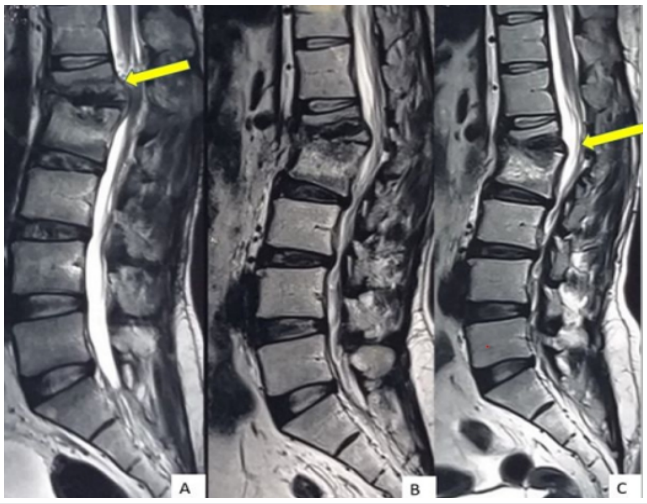

Figure 1: A 35 years old male patient presented with mild neurological deficits and Frankel D neurology. Patient was diagnosed as L1-2 tubercular discitis and Started on ATT. Patient underwent MRI at 6monthly intervals which are shown in the figure
A.L1-2 destruction of the L1 vertebral body with epidural collection compressing the cord B. Repeat MRI after 6 months of ATT shows signs of healing with resolution of the epidural abscess.
C. MRI at 12 months shows complete resolution of the disease with reconstitution of the canal and restitution of the normal bone marrow signal in the vertebral bodies at 1 year follow up.

the posterior facet joints. Rarely posterior appendiceal tuberculosis can cause instability due to direct involvement of the facet joints [6]. The spinal instability can be assessed based on the 'spine at risk signs' on radiological examination as defined by Rajasekaran et al. as retropulsion, subluxation, lateral translation and toppling [7].

C. Rarely, infective thrombosis/endarteritis of the spinal vessels can cause vascular infarcts and result in ND [5].

D. Hodgson et al. demonstrated histologically that TB infection does cross the meningeal barrier and thus could be a contributing factor for Pott's paraplegia [5]. Primary spinal cord tuberculoma have also been reported which may present with neurological dysfunction.

STB is a chronic infection and as the pathological lesion is slow growing; the neural elements tolerate the insidiously developing mechanical compression longer before any overt

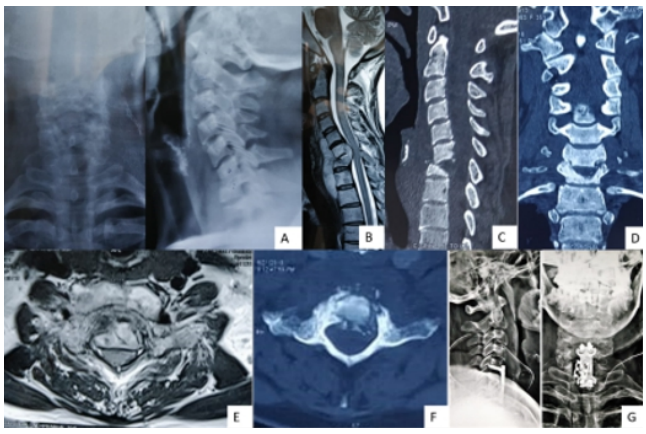

Figure 3: 35 Years old female presenting with neck pain and weakness in both upper and lower limbs for 4 months
A. X-ray showing destruction of C7 with subluxation of C6
B, E. Sagittal and axial sections of cervical spine MRI demonstrating epidural abscess at C6-7 level and extensive prevertebral abscess from C5 to D1
C, D. CT scan of cervical spine sagittal coronal and axial sections showing destruction of C7 vertebral body with subluxation of C6 vertebra
F. shows the axial CT scan image showing sequestered bone debris into the spinal canal G. Postoperative X-ray of the patient showing corpectomy of C6 and C7 and C5-7

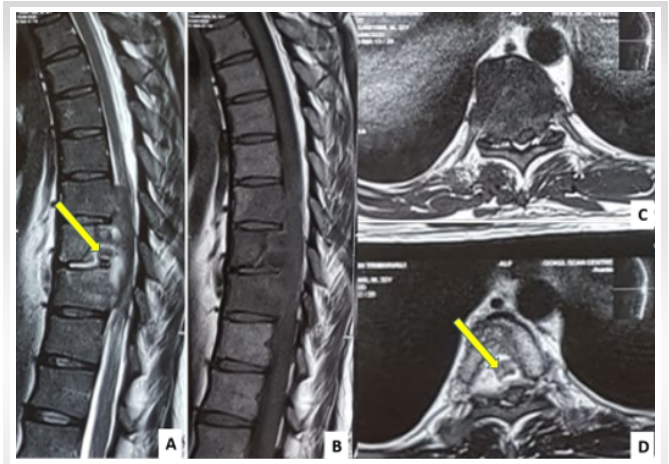

Figure 2: Shows MRI images of a 27 years old male patient who presented with worsening neurological deficit, progressing from Frankel D to Frankel B despite being on ATT. MRI images shows sequestered posterior vertebral body fragment retropulsed into the spinal canal
A.T2 weighted sagittal MRI image of D9-10 tubercular spondylodiscitis with sequestrum (arrow) and epidural abscess
B.T1 weighted sagittal MRI image of D9-10 tubercular spondylodiscitis with epidural abscess
C.T2 weighted axial image demonstrating the compression of the spinal cord by tubercular debris (granulation tissue, pus and sequestered bony fragment)
D. T1 weighted axial image demonstrating the extent of disease

ND develops [3].

ND in late-onset disease

The pathophysiology of ND in late-onset disease is quite

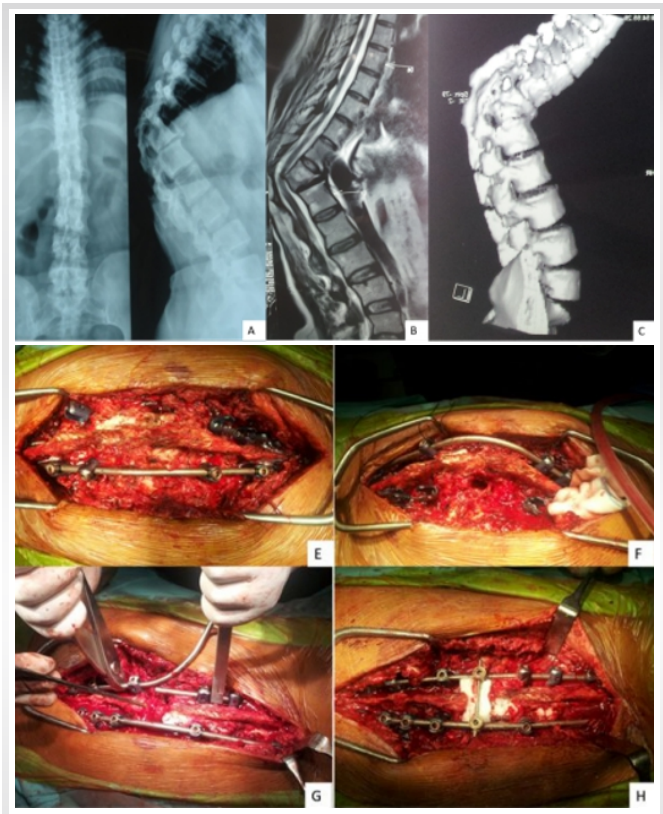

Figure 4: 26 years old male patient with post tubercular kyphotic deformity presenting 10 years later with neurological deficit.
A. Shows the X-ray of the Patient with sharp angular kyphosis at dorsolumbar junction.
B. Showing T2 weighted sagittal MRI image showing the stretching of the thecal sac over the internal salient
C. Shows 3D reformatted CT scan with gibbus at the dorsolumbar junction. Patient underwent deformity correction through posterior column osteotomy.
D. Shows the intraoperative picture with screws and rods fixed. E shows the prominence of the gibbus and the initial rod contoured according to the deformity. G demonstrating the correction manoeuvres
for deformity correction after performing the posterior osteotomy. H showing the correction of the deformity when compared to E

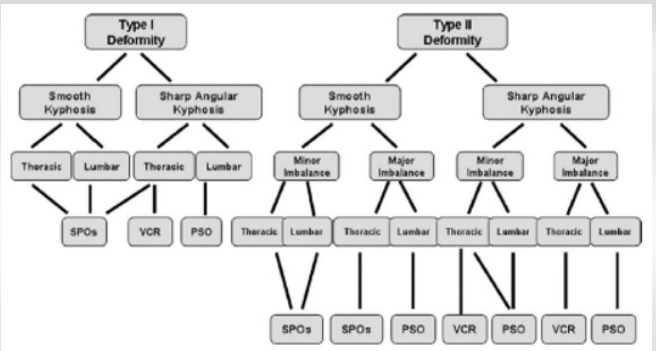

Figure 5: Algorithm by Bridwell et al. for selection of appropriate osteotomy type based on the character of the sagittal deformity. PSO = Pedicle Subtraction Osteotomy, SPO = Smith Peterson Osteotomy VCR = vertebral column resection

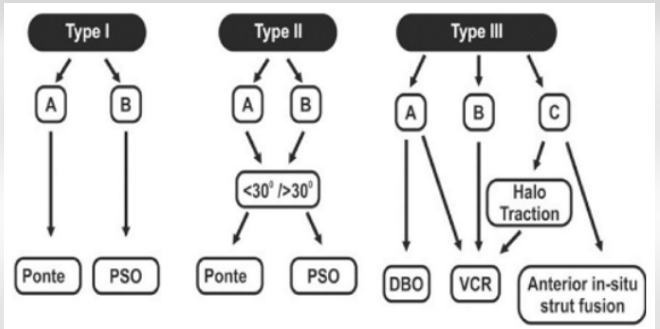

Figure 6: A treatment algorithm by Rajasekaran et al. to guide selection of appropriate posterior approach osteotomies based on the classification presented in the study. PSO = pedicle subtraction osteotomy, DBO = disc bone osteotomy, and VCR = vertebral column resection.

different and can be considered due to the following factors

A. Recrudescence / Reactivation of the healed disease [8].

B. Spinal kyphotic deformity with the neural elements being stretched over the internal gibbus causes localised pressure and ND [6].

C. Rarely late-onset STB forms a fibrous union. These paradiscal fibrous unions may allow micromovement resulting in spinal cord dysfunction due to instability.

More than one of the above causes may coexist and contribute to the ultimate development of ND in late onset disease. Often ND in late-onset disease can be accompanied by the findings on MRI of spinal cord oedema, atrophy, interstitial gliosis and/or myelomalacia changes [9].

Correlation of spinal canal dimensions and spinal cord signal changes with ND

Studies in the past have correlated the amount of canal compromise with the ND. Canal occupancy of up to 60 to 76% on mid sagittal section of MRI was found compatible with intact neurological status [10]. However, in presence of mechanical instability, deformity or vascular insult; lower degrees of canal compromise may lead to ND [11].

Correlation of MRI evidence of spinal cord signal changes with the presence of ND suggested that patients showing predominantly extradural soft compression due to collection of pus, relatively preserved cord size, presence of isolated T2 hyperintense changes suggesting oedema, had negligible ND and also good prognosis for neurological recovery with treatment [11].

The spinal cord calibre did not always correlate with severity of ND, however, thinning of cord in association with myelomalacia, syrinx formation and features of arachnoiditis carried a poorer prognosis [3].

Table 1 : Staging of neurological deficits in Pott's paraplegia

Stage I	Patient do not complain of any neurological symptoms but clinician can elicit exaggerated reflexes and plantar extensor
Stage II	Patient is able to walk but has spasticity and weakness. Pain and temperature sense are affected
Stage III	Patient is a non-walker and sensory symptoms same as in Stage II
Stage IV	Stage IV Patient is a non-walker and pain, touch temperature, vibration and joint position sense are affected
Stage V	Stage IV along with bowel and bladder involvement. Patients may have flexor spasms or flaccid paralysis.

Table 2 : Indications for surgery in Pott's spine

	Indications for surgery in Pott's spine
1	In case of severe neurological deficits at presentation i.e. flaccid paraplegia, paraplegia in flexion
2	New onset deficits in a patient on ATT
3	No signs of improvement even after 6 weeks of ATT
4	Recurrence of disease with ATT
5	Worsening of deficits while on ATT
6	Spinal tumour syndrome
7	Posterior disease with pedicle and neural arch destruction.
8	Children with "spine at risk signs"
9	Kyphosis more than 30 degrees in single vertebral involvement
10	Large paraspinal abscess causing respiratory distress or deglutition problems due to prevertebral abscess
11	Impending spinal instability suggested by loss of 1 vertebra in thoracic spine or 1.5 vertebra in lumbar spine
12	Uncertain diagnosis

Neurological deficit at clinical presentation

Neurological involvement at presentation is being widely reported with varying incidence in different studies. Pertuiset et al reported neurological deficits in 50% of their patients with significant neurological manifestations seen in the cervical and thoracic involvement. Patients with lumbar disease had mainly radicular symptoms as a part of the neurological dysfunction [12]. Jain et al reported 57% incidence of neurological involvement at presentation [13].

Patients may present to the clinician at various stages of neurological deficits and if left untreated, early neurologic

Table 3 : Pathogenesis of Restabilization in Pott's disease

Type of restabilization	Pathogenesis of restabilization
Type A	Aligned facets and less than 0.75 of the vertebral body destruction stabilize with maximum area of contact and progress to uneventful healing.
Type B	Destruction of 0.75 - 1.5 vertebral body causes subluxation of facets at one or more levels and restabilization occurs by descent and sagittal rotation of the superior normal vertebra so that its anteroinferior margin touches the superior surface of the inferior normal vertebra. Deformity is usually < 6o degrees.
Type C or Buckling collapse	with >1.5 vertebral body loss and age < 7 years facet joints at two or more levels may dislocate and cause the rotation of superior normal vertebra by more than 90 degrees. Stabilization occurs by anterior portion of the superior normal vertebra coming into contact with superior part of inferior normal vertebra.

involvement may progress to complete paraplegia or tetraplegia. In a case series of 100 patients with Pott's paraplegia reported by Tuli et al., 33% reported within 4 weeks,40% within 1 to 3 months and 27% presented after 3 months of onset of deficits [14].

The most common symptom if STB is pain over the region of involvement cervical, thoracic or lumbar. The extent of ND can vary depending on the level of spinal cord involvement. In cervical spinal tuberculosis, patients manifest with symptoms of weakness in the myotomal distribution of the upper limb in case of cervical root compression or more commonly with weakness in all four limbs with upper motor neuron (UMN) signs due to cervical spinal cord compression.

In thoracic STB, upper extremity function remains normal while lower-extremity symptoms progress over time eventually leading to paraparesis/paraplegia with UMN signs. A lower motor neuron (LMN) type of ND is seen with the involvement of the lumbosacral levels and they can occasionally present with significant cauda equina deficit with involvement of bowel and bladder symptoms. Posterior element and appendiceal STB is rare however, they often present with neurological complications due to early compromise of the spinal canal [6].

Staging of neurological deficits

Symptomatic ND in STB can be preceded by a period of elicitable signs of UMN involvement such as exaggerated deep tendon reflexes and plantar extensor response. Over a period of time the patient experiences motor weakness in the limbs which may be incomplete to begin with and may progress to complete motor paralysis. The motor weakness may be accompanied by varying degrees of sensory neurological dysfunction. Bowel, bladder deficits are seen late in the course of the disease quite often after established motor and sensory deficits have occurred. In long standing ND the initial UMN signs of spasticity are gradually replaced by flaccidity and flexor spasm. This was highlighted by Tuli et al. and Jain et al in their extensive work on STB. Tuli et al has suggested a classification to grade the ND and this was later modified by Jain et al. Neurological deficits in STB spine are classified into five stages [15]. Table 1

Treatment of Pott's spine with neurological deficits

Early diagnosis, timely initiation of Anti tubercular treatment drugs (ATT), and proper supportive care will prevent the occurrence of neurological deficits in the vast majority of patients. Once the neurological deficit has occurred early diagnosis of the cause of paraplegia, investigating the extent of the disease by MRI and CT scans, and instituting early and effective management can reverse and/or minimize the effect of this devastating complication [6].

Paraplegia with early-onset disease

Tuberculosis is a medical disease and chemotherapy is the mainstay of treatment. Pathophysiology of cord compression in STB is a chronic process, and the spinal cord can adjust to this slow developing compression unless there is acute compression due to pathological process causing sudden instability due to subluxation or dislocation. Therefore, a watchful delay in surgical decompression will not significantly alter the recovery of neurology [6]. Fig 1. Duration of chemotherapy varies in different countries. American thoracic society recommends 6 months of chemotherapy in adults and 1year for children. British thoracic society recommends 6 months chemotherapy to all age groups [16]. India extrapulmonary TB guidelines (INDEX TB guidelines) which are based on WHO recommendation advises 1 year chemotherapy.

(2 months intensive phase + 10 months of continuation phase) of chemotherapy to patients [17]. Appropriate and timely ATT help reduce the oedema and relieve the compression. However, the response to conservative treatment is slow in scenarios, when compression is by large sequestrum and/or soft healing ridge [6]. Fig 2 Dobson et al in the pre antibiotic era reported 48 % improvement with conservative treatment alone. Subsequent MRC trials have concluded that patients with Pott's paraplegia can be managed with ATT alone without any surgery, although MRC trials considered patients with limited disease with small kyphotic angle and mild to moderate paraparesis resulting from abscess and inflammatory oedema [16]. Tuli has reported 38 % neurological recovery out of his 200 cases on 4–6 weeks of rest and ATT. While the remaining 62 % were surgically decompressed [14]. The heterogeneity of the study population, varying stages of neurological complications, individual physical condition, bacterial drug sensitivity, and patient selection criteria for non-operative management may be the reason for variable neurological recovery reported.

'The Middle path regimen' was advocated by Tuli et al. and this recommendation developed based on their institutional experience. The authors noted that patients reaching at paraplegic state had very poor general health, anaemia, coexisting pulmonary tuberculosis and were unfit for major surgery. While waiting for clinical optimization for subsequent surgery, many of them (38 %) started

showing neurological recovery on standalone ATT, bed rest and nutritional support over a period of 4–6 weeks. The patients who did not recover were taken up for surgical decompression. But the middle path regimen has phased out in current scenarios with better resources and improved surgical techniques.

Surgeons advocating universal disease extirpation reported recovery rates ranging from 75-84% in patients undergoing surgical treatment for ND in STB. The excellent recovery rate was postulated by the authors to be a result of direct decompression of neural elements by removing the debris. The debridement also improved antibiotic penetration in the chronic inflammatory lesions [18],[19],[20].

Current literature recommends early intervention in ND arising out of active disease. A meticulous implementation of chemotherapy regimen in addition to operative

decompression has become standard of care. Tubercular liquid pus, granulation tissue, caseous tissue causing compression and inflammatory oedema but relatively preserved clinical spinal cord function may be considered for watchful nonoperative treatment with ATT alone. The patients with extradural compression of mixed or granulomatous natures showing entrapment of the spinal cord should be undertaken for early surgical decompression [6]. The neurological recovery has been observed even where decompression was performed up to 11–12 months after developing paraplegia [3],[6],[10]. Therefore, is it recommended to offer the benefit of surgical decompression to all patients even for long standing paraplegia [6]. The indications for surgical intervention in ND in early-onset STB are listed in Table 2.

Goals of surgery in early-onset disease are to decompress the spinal cord by debriding the disease focus, debulking the disease load and to achieve a stable and well aligned spine at the end of the treatment. Factors to be considered while deciding surgical approach for STB are age, comorbidities, location of disease, associated deformity, surgeon's preference and his experience with the approach [21].

Anterior approach surgery

STB commonly affects the anterior elements of the spine; and anterior approach allows surgeons to access the lesion directly to thorough evacuation of the abscess, debridement of the necrotic tissue and reconstruction of the anterior column defect. Anterior debridement and fusion was popularized by Hodgson et al. and was considered the standard of STB treatment. Hodgson et al in their study of 100 patients of STB performed anterior surgery for disease clearance and fusion using only bone graft without instrumentation. They reported a fusion rate of 93% in their series.[20]. Benli et al in their retrospective study of 100 patients who underwent anterior decompression and fusion with instrumentation reported that addition of instrumentation helped to effectively correct spinal deformity which was better maintained on final follow up. They reported a loss of correction of 23 degrees without instrumentation. Application of anterior instrumentation in addition to debridement and grafting ensures effective deformity correction and stability of the spine [22].

In a study by Rajasekaran et al they observed slipping, fracture, resorption or subsidence of the graft in 59% when only debridement and grafting was done without instrumentation and was more common when the graft spanned 2 or 3 vertebral bodies. Although the anterior approach offers the advantage of short segment fusion, single approach and also eliminates the need for external immobilization especially in the Lumbar spine; there are several downsides for this approach. Anterior surgery can be complicated by cage migration, vascular injury, displacement of screws and visceral organ injury. Additionally, anterior surgery can be challenging in case of pulmonary scarring, when lesions are at T2-T4 region. In subaxial cervical and cervico dorsal STB anterior debridement and fusion with instrumentation is the gold standard till date [21]. Fig 3 Patients of TB spine with concomitant osteoporosis need posterior instrumentation [23].

Posterior approach surgery

In patients with less than 50% of the vertebral body destruction and < 30 degree of kyphosis posterior only surgery with instrumentation can be considered. Decompression in the form of posterolateral/ transpedicular / transfacetal approach can adequately evacuate pus, granulation tissue and the inflammatory debris. This approach restores stability and maintains the alignment of the spine as the disease healing occurs with ATT. Sahoo et al. in their study reported that when the disease is limited to one or two vertebrae posterior transpedicular decompression approach gives satisfactory results [24]. Lee et al in their retrospective study of with limited vertebral body destruction concluded that posterior transpedicular decompression and instrumentation gives good results in early early-onset disease [25].

Anterior and posterior surgery

Rajasekaran et al in their prospective study of 81 patients with a n 8 years follow-up reported that if the graft has to span more than two disc spaces the chances of graft failure were high. The authors concluded that the such long segment anterior grafts should be supported by posterior instrumentation [26]. Posterior instrumentation gives the benefit of better reduction of kyphosis and maintains correction better especially with the use of pedicle screws which offer three column fixation of the spine.

Though anterior approaches are ideal for evacuating the infected material; recent trend is for an "all posterior" approach with circumferential reconstruction especially in thoracic and lumbar disease [21],[27].

Paraplegia in late-onset disease

Neurological deficits in a late-onset case of TB can occur many years (4-40 years) after the disease has been cured, and can be due to recrudescence of the disease or due to stretching of the cord over the internal gibbus [3]. ND is usually incomplete but can be severe at times. The severity of neurological deficit has not been correlated to the degree of kyphosis but Tuli et al. believed that occurrence of neurological deficit is more common if the kyphosis is more than 60 degrees [28]. This type of paraplegia is commonly seen in individuals with dorsal or dorsolumbar kyphosis. The spinal cord stretches over the sharp anterior angulation at the apex of the kyphosis or there is constriction around the cord due to scarring. Fig 4. This prolonged impingement of the cord or circumferential constriction results in inflammatory oedema, cord atrophy(gliosis), myelomalacia or syringomyelia in the cord. Once these changes occur, it results in exhaustion of the physiological reserve of the cord and ND which may not resolve even after the offending elements are surgically tackled. Patients with significant spinal kyphotic deformity usually have severely compromised cardiopulmonary reserve, which

makes them high risk candidates for any major deformity correction surgery. Moreover, these patients have high risk of neurological worsening following surgical intervention on account of the ' the sick cord syndrome' which frequently accompanies late onset ND in STB [3].

Rajasekaran et al. described 3 types of restabilization during healing of STB and is illustrated in table /figure. Structural integrity of facet joints decides the type of restabilization that occurs in a healing disease. Along with facets level, age of the patient and extent of vertebral body destruction also influences the type of restabilization. Patients with aligned facets and less than ¾th of the vertebral body destruction stabilize with maximum area of contact and progress to uneventful healing. (Type A restabilization). If the destruction is between ¾ body -1.5 vertebral body there is subluxation of facets at one or more levels and restabilization occurs by descent and sagittal rotation of the superior normal vertebra so that its anteroinferior margin touches the superior surface of the inferior normal vertebra. (Type B restabilization). Growth at this level is suppressed and the deformity will progress, but not beyond 60 degrees. If the vertebral body destruction is more than 1.5 vertebra and if the age is less than 7 years facet joints at two or more levels may dislocate and cause the rotation of superior normal vertebra by more than 90 degrees. Stabilization occurs by anterior portion of the superior normal vertebra coming into contact with superior part of inferior normal vertebra. (Type C Restabilization or buckling collapse) [29]. Table 3

ND can also occur in cases of low-grade persistent infection with increasing kyphosis or reactivation of the silent disease at the apex of the kyphosis. From the management perspective it is important to differentiate the aetiology ND whether it is due to late-onset disease with increasing

kyphosis or due to recrudescence of the old disease. Clinically, ND due to disease reactivation is severe and rapidly progressive than that due to cord impingement in healed disease; it also responds well to chemotherapy without the need of additional surgery.

Assessment and management of spinal deformity in late-onset disease

Treatment of ND in a late-onset case of STB includes correction of the deformity and arresting the progression of neurologic deficits. Assessment of kyphotic spinal deformity and type of osteotomy to be considered for correction has been suggested by Bridewell et al and rajasekaran et al. Bridwell et al classified Kyphotic deformity into Type I(balanced) and Type II (unbalanced) based on whether the kyphotic deformity is compensated by adjacent segments. He proposed an algorithm for deciding the type of osteotomy needed to correct the deformity. The algorithm is given in the figure below [30].

Fig 5.

Rajasekaran et al classified kyphotic deformities into 3 types based on the extent of anterior and posterior column deficiency, flexibility of the disc spaces, curve magnitude, and correlation with the type of osteotomy typically required. He proposed an algorithm for choosing the type of osteotomy based on the type of deformity. The classification and algorithm are illustrated in the figure below [31]. Fig 6

Bridwell et al classified kyphotic deformity into two types Type I(balanced) and Type II (un balanced) based on whether the deformity is compensated by the adjacent segments or not.

Kee Yong-Ha et al in 2015 in their study of 36 patients with late onset neurologic deficits in Pott's spine reported that STB kyphotic deformity with ND is more common in patients with thoracic or thoraco-lumbar disease. They concluded that patients with cord level deficits and severe angular kyphosis with rapidly progressive deficits are high risk for ND worsening after surgery [32].

Wong et al reported a mean follow up of 34-year in 24 patients with STB and kyphotic deformity. There were 9 patients operated for late-onset disease with ND. Four patients showed improvement, 4 remained the same and 1 patient worsened. Out of 6 patients with reactivation and ND 4 recovered and 2 patients remained the same. Nine patients had restrictive lung disease and 4 patients expired at a mean age of 57 years. From their study Wong et al concluded that early correction of kyphosis and bony fusion and regular follow up will prevent late complications of the disease. Early onset kyphosis, restrictive lung disease and paraplegia were associated with higher mortality [33]. Hua et al. reported on posterior vertebral column resection surgery for kyphotic >60 degrees. The mean kyphotic angle correction was noted to be 62 degrees and the authors concluded that there is high the incidence of neurological complications associated with correction of kyphotic deformity more than 60 degrees [34].

The ideal way to treat late onset paraplegia is to prevent its occurrence, hence preventing the development of deformity by early diagnosing and treatment of STB. In patients reporting with severe kyphosis should be considered for early correction of kyphosis irrespective of their neurological status.

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

Conflict of interest: Nil **Source of support:** None

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