CLINICAL ORTHOPAEDICS AND RELATED RESEARCH
Number 398, pp. 75–84

Treatment of Tuberculosis of the Spine With Neurologic Complications

Anil K. Jain, MS

Neurologic complications are the most dreaded complication of spinal tuberculosis. The patients who have paraplegia develop in the active stage of tuberculosis of the spine require active treatment for spinal tuberculosis and have a better prognosis than the patients who have paraplegia develop many years after the initial disease has healed. Neurologic dysfunctions in association with active tuberculosis of the spine can be prevented by early diagnosis and prompt treatment. Prompt treatment can reverse paralysis and minimize the potential disability resulting from Pott's paraplegia. When needed, a combination of conservative therapy and surgical decompression yields successful results in most patients with tuberculosis of the spine who have neurologic complications. The vertebral body primarily is affected in tuberculosis; therefore, decompression has to be anterior. Laminectomy is advocated in patients with posterior complex disease and spinal tumor syndrome. Late onset paraplegia is best avoided by prevention of the development of severe kyphosis. Patients with tuberculosis of the spine who are likely to have severe kyphosis develop (< 60°) on completion of treatment should have surgery in the active stage of disease to improve kyphus.

The incidence of neurologic involvement in tuberculosis of the spine is reported to be between 10% and 46%[3,4] and observed mostly in patients with dorsal spine affection.[14,17]

From the Department of Orthopedics, University College of Medical Sciences And G.T.B. Hospital, and University of Delhi, Delhi, India.

Reprint requests to Anil K. Jain, MS, University College of Medical Sciences & G.T.B. Hospital, Delhi-95, India.

Types and Causes of Neurologic Deficit

There are two types of neurologic deficit in tuberculosis of the spine.[3,5] Paraplegia in patients with active disease, found in the early stage of the disease (early onset), and paraplegia in patients with healed disease, which usually develops many years after the initial disease has healed (late onset).

Paraplegia in patients with active disease may be caused by mechanical pressure on the spinal cord by tubercular abscess, granulation tissue, tubercular debris, and caseous tissue. The localized pressure caused by internal gibbus on the spinal cord or mechanical instability caused by pathologic subluxation or dislocation of vertebrae also may contribute to neurologic complications. Certain intrinsic changes in the spinal cord such as inflammatory edema or direct affection of meninges and spinal cord by tuberculous infection or inflammation also may lead to paraplegia. Infective thrombosis or endarteritis of spinal vessels leading to infarction of the spinal cord also may produce neurologic complications.

Paraplegia in patients with healed disease may be produced by localized pressure on the spinal cord by a transverse ridge of bone anterior to the spinal cord or constricting scarring of and around the dura. Stretching of the spinal cord over an anterior internal salient (internal

gibbus) may lead to interstitial gliosis to produce neurologic complications. More than one cause may produce neurologic complications at the same time.[5,20]

The spinal cord seems to have enough physiologic reserve to withstand considerable pressure particularly when pressure is slow to build up, as in tuberculosis. Jain et al[7] calculated canal encroachment on computed tomography (CT) scans in 15 patients with tuberculosis of the spine without neurologic complications from C3-dorsal12 and found that as much as 76% of spinal canal encroachment is compatible with an intact neural status. However, when vascular catastrophe or mechanical instability is present, paraplegia can be produced at lesser compromise of the spinal canal.[7]

Staging of Neurologic Deficit

The classification of Frankel et al[2] and the American Spinal Injury Association score as reported by Mermelstein et al,[12] which are widely used to classify neural deficit with spinal disorders, do not differentiate all combinations of neural deficit observed in patients with spinal tuberculosis. Based on the observations of the sequence of the neurologic deficit in tuberculosis of the spine, the classification modified by Tuli[17] seems most rational. The severity of neurologic deficit should be expressed in stages. In Stage I, the patient does not have weakness but there is clumsiness of gait and signs suggestive of an upper motor neuron lesion (extensor plantar response and ankle clonus); in Stage II, the patient has motor weakness, signs of an upper motor neuron lesion but power is sufficient that the patient is able to walk. These patients usually have motor power Grade 3 or more; in Stage III, the patient is bedridden (severe motor weakness) with signs of upper motor neuron paraplegia. The patient also has sensory loss less than 50%; and in Stage IV, patients have complete motor weakness with loss of sensation more than 50% and/or bladder bowel involvement and/or flaccid paraplegia and/or paraplegia with flexor spasm.

Paraplegia With Active Disease

Universal surgical extirpation was advocated in all patients with tuberculous paraplegia[4] with the reported advantages being that the quality and speed of neural recovery is better, surgical decompression removes the fibrous barrier to drugs, and the diagnosis is established. An absolute nonoperative approach to Pott's paraplegia is unjustifiable because valuable time may be lost while irreparable damage may progress to complete loss of motor function.[17] At the same time, universal surgical extirpation also seems to be unnecessary in every patient. A judicious combination of conservative therapy and operative decompression when needed should form a comprehensive integrated course of treatment for patients with tuberculosis of the spine who have neurologic complications.

The middle path regimen is based on the fact that most of the patients are from a low socioeconomic state, have poor general health, anemia with or without pulmonary tuberculosis, and are not candidates for major surgery.[17] While waiting for surgery, 30% to 35% of patients have neural recovery in 3 to 4 weeks with bedrest, antitubercular chemotherapy, and a nutritious diet[9,17] Jain et al[9] reported that 26 of 64 patients had neurologic improvement in 4 weeks when given multidrug therapy.

It has been established that isoniazid, ethambutol, rifampicin, and para-amino salicylic acid reach tuberculous foci in an adequate concentration. Therefore, tubercular liquid pus, granulation tissue, inflammatory edema, and caseous tissue causing compression are amenable to nonoperative treatment. A delay of 3 to 4 weeks gives the patients with the aforementioned disorders a chance to respond to chemotherapy. Neurologic recovery may be observed during this period, thereby avoiding surgical decompression (Figs 1, 2).

Forty-three patients who received nonoperative and operative treatment were sequentially followed up with magnetic resonance imaging (MRI). The patients with a relatively preserved spinal cord with evidence of edema

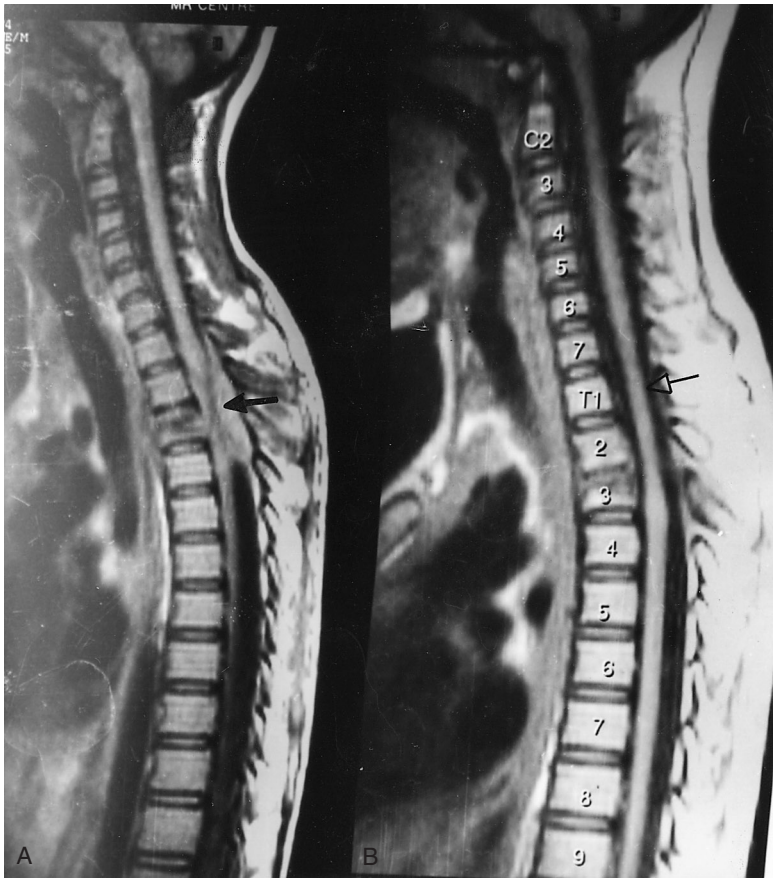

Fig 1A–B. Midsagittal T1-weighted MRI scans of a 14-year-old patient with tuberculosis of T2–T3 with no neurologic deficit show (A) cord compression (solid arrow) and evidence of vertebral destruction and (B) compression relieved (open arrow) after nonoperative treatment. There is evidence of healing of the vertebrae.

or myelitis with a predominantly fluid collection in the extradural space responded well to nonoperative treatment.[8] This observation supports the philosophy of the middle path regimen.[8] Compression in a tuberculous spine causing a neurologic complication is a slowly developing process (exceptions are vascular catastrophe and pathologic subluxation or dislocation). A short delay in surgical decompression does not significantly alter the long-term recovery of neurologic function. The algorithm of the treatment of a patient with tuberculosis of the spine with neurologic complications is shown in Figure 3.

Indications of Surgery in Tuberculosis of the Spine With Neurologic Complications

The indications for surgery[20] are categorized as described below.

Clinical Factors

Patients with paraplegia of rapid onset have severe paralysis from rapidly accumulating mechanical compression or from a mechanical accident such as pathologic subluxation or dislocation. It also may result from vascular thrombosis or endarteritis, which is difficult to prove or disprove. It is an explanation for non-

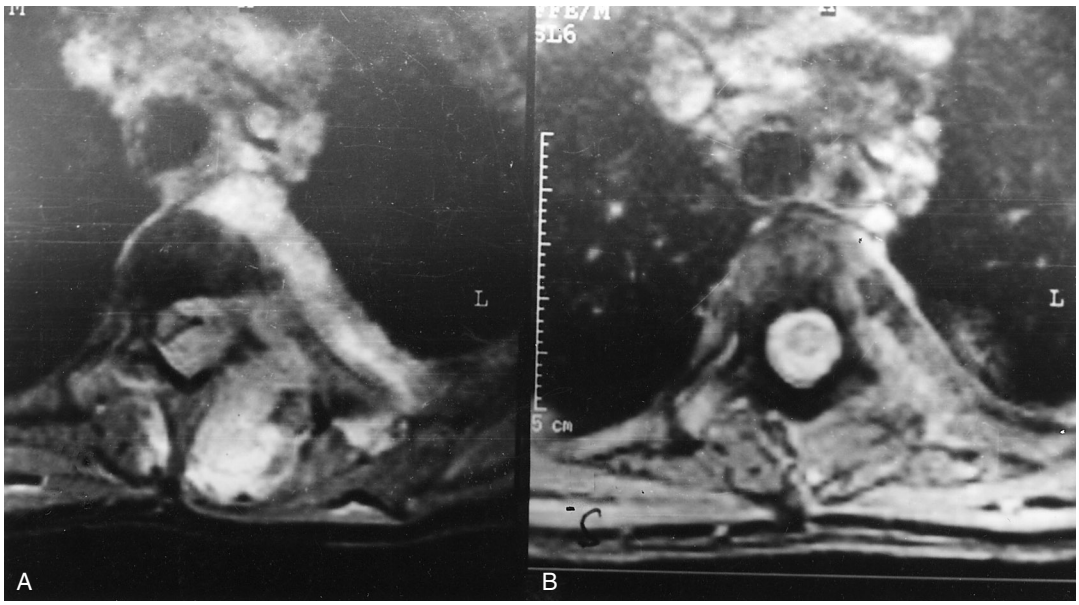

Fig 2A–B. Axial T2-weighted MRI scans of the same patient as shown in Figure 1 show (A) prevertebral, left paravertebral collection, and intraspinal compression predominantly fluid in nature and (B) prevertebral, paravertebral collection, and intraspinal compression that resolved with nonoperative treatment.

recovery of neural deficit despite evident adequate surgical decompression

Surgical decompression is indicated in patients with paraplegia with neural arch affection, recurrent paraplegia, and patients with massive retropharyngeal abscess causing difficulty of deglutition or respiration. Patients with tuberculosis of the spine with uncontrolled spasticity require surgery because reasonable rest and immobilization are impossible. Surgical decompression, although uncommon, is indicated for patients with spinal tumor syndrome to establish the diagnosis. Patients with severe paraplegia such as those with flaccid paraplegia, paraplegia in flexion, complete sensory loss, and complete loss of motor power, also may have surgery.

Treatment Factors

A patient with tuberculosis of the spine who has neurologic complications develop, whose condition remains stationary, or whose condition becomes worse during conservative treatment with rest and antitubercular drugs should have surgical decompression.

Imaging Factors

The cause of paraplegia in patients with tuberculosis of the spine who have panvertebral involvement is instability associated with mechanical compression and inflammation. The panvertebral involvement is suggested on plain radiographs by associated scoliosis, severe kyphosis, or both. The CT and MRI scans show global destruction of the vertebral body. In these patients, decompression and spinal stabilization also are indicated. In patients in whom extradural compression, as seen on MRI scans, consists of granulation or caseous tissue with little fluid component, compressing the spinal cord circumferentially and constricting the cord with the features suggestive of cord edema, myelitis, or myelomalacia, should be done for early surgical decompression.[8]

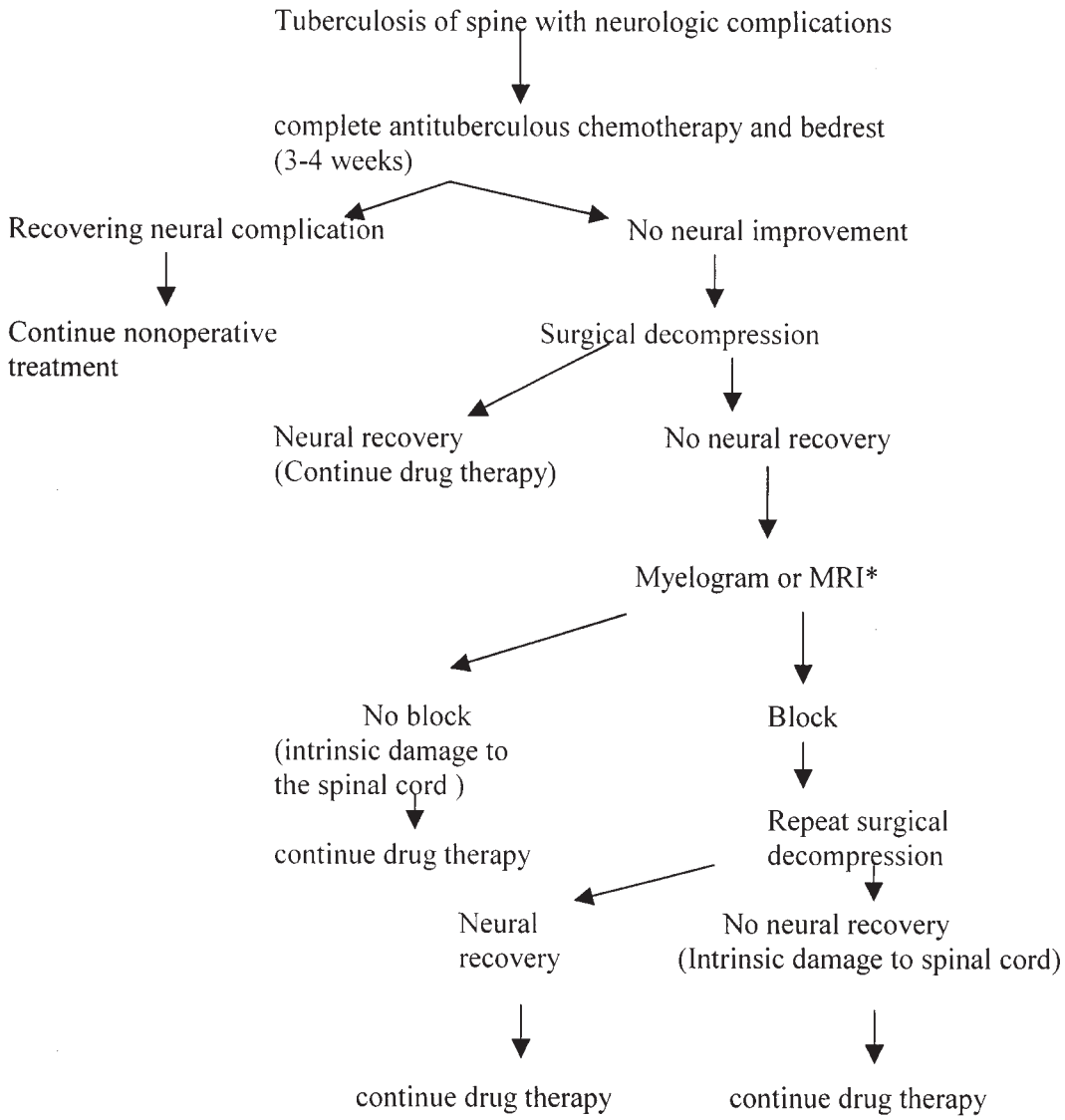

Fig 3. An algorithm for the treatment of patients with tuberculosis of the spine who have neurologic complications is shown. *A myelogram is the best modality to assess adequacy of surgical decompression. The author recently has treated patients in whom the MRI scan suggested inadequate surgical decompression, whereas the myelogram taken at the same time showed a clear flow of dye across the disease area.

Patient Factors

Patients with tuberculosis of the spine sometimes have painful paraplegia resulting from severe spasm or root compression, which is a relative indication of surgery. Older patients with Pott's paraplegia require surgical decompression to avoid the hazards of prolonged immobilization.

Surgical Decompression

The typical lesion in tuberculosis of the spine affects the vertebral body, so surgical decom-

pression in patients with anterior disease must be anterior. Laminectomy for decompression[3] removes the only healthy component of the vertebral column in patients with anterior disease, rendering the spine unstable and resulting in pathologic dislocation, increase in kyphosis, and deterioration of neural deficit. Surgical decompression by laminectomy is indicated only in patients with isolated neural arch affection[11,19] and in patients with compressive myelopathy who have spinal tumor syndrome.

The areas of bone, which are infiltrated but not necrosed with tubercular disease, recover and reconstitute with drug treatment. The ischemic and infarcted bone also will recover and reconstitute as the disease subsides and the circulation of the lesion improves with chemotherapy alone. Surgery, in addition to chemotherapy, is essential for areas of necrosis that are past recovery and that harbor tubercular bacilli. While doing surgical decompression, one should remove only that part of viable bone (debridement surgery) that allows removal of pus, caseous tissue, and sequestra to decompress the spinal cord. The gap created should be bridged by bone grafts to achieve the maximum possible correction of kyphosis. Excision of bone up to healthy bleeding bone (radical surgery) will leave a large gap to be bridged by a long graft. Longer grafts are associated with more complications such as slippage and breakage, which lead to an unstable spine. However, debridement in which the entire vertebral body is not excised, leaves a relatively more stable spine.

The results of radical and debridement surgery were compared in a long-term followup in patients with tuberculosis of the spine with or without paraplegia, where two vertebral bodies were diseased. Healing of the lesions and neural recovery were similar in both groups with marginally better correction of deformity in patients who had radical surgery.[18] Deterioration of kyphus is observed in patients with extensive disease in whom radical surgery has left a large defect after debridement (two vertebral body heights or more) to be bridged by rib graft.[15] Tuli[17] followed up 104 patients after debridement surgery in whom 2.5 vertebral bodies were affected on average. In 80% of the patients, kyphus decreased or remained static or increased by only 10°.[17] Only 20% of patients had an increase in kyphus more than 10° and 4% had more than 30° increase in kyphus. The current author observed 40 such cases of behavior of kyphus after debridement surgery. In 60% of the patients, kyphus remained static or improved. The kyphus increased less than 10° in 25% of patients and 11° to 20° in 10% of patients. The increase was more than 20° in only 5% of patients.[10] There is no advantage of radical surgery over debridement surgery for correction of kyphotic deformity, particularly when an extensive spinal lesion is confronted.

The anterior approach is indicated for cervical and lumbar spine decompression. In the dorsal spine there are two approaches: the thoracotomy approach and the extrapleural approach (anterolateral approach). The thoracotomy approach as advocated by Hodgson and Stock[4] is an operation of severe magnitude, particularly in patients with extensive disease, and should not be considered lightly even where good surgical facilities exist. It should not be done where surgical facilities are poor as suggested by the originator.[4] In an excellent set-up, 6% of patients with moderate paraplegia and 11% of patients with severe paraplegia die after surgery.

Approximately 50% of the patients with spinal tuberculosis have anemia and evidence of healing or active pulmonary tuberculosis. In a patient with paraplegia in whom the intercostal muscles are paralyzed (paretic) with a compromised pulmonary function, thoracotomy will increase the risk of postoperative complications. In such patients with compromised pulmonary reserve, a lateral extrapleural (anterolateral) approach can be done. It allows an easier and better exposure of the spinal cord in a severely kyphotic spine, which is technically difficult to expose by a transthoracic approach. The determining factors for which a particular approach to use should be preference and technical skills of surgeon, availabil-

ity of surgical facilities, and general and pulmonary reserve of the patient.

Paraplegia With Healed Disease

Patients with paraplegia with healed disease usually present with a history of treatment for extensive spinal tuberculosis and remain symptom-free for a long period with severe kyphosis. This type of paraplegia usually is seen in patients with severe kyphosis in the dorsal and dorsolumbar spine because kyphosis does not progress so severely in the cervical and the lumbar spine.[3] The causes of late onset paraplegia in patients with Pott's disease have been enumerated previously. Reactivation of quiescent disease at the apex or proximal or distal to kyphosis also is a possibility in such patients.

Treatment

Anterior decompression with removal of internal gibbus is the treatment of choice for patients with paraplegia with healed disease, although it seldom produces complete neural recovery and is fraught with complications including deterioration of the neural deficit.[3,6] These patients have severe kyphosis, costovertebral impingement, and poor pulmonary reserve. Therefore, these patients are poor risk for major surgery such as anterior decompression, and they have a higher risk for neural deterioration. The correction of a severely deformed spine in a patient without neural deficit should not be done for cosmetic reasons only. Tuli[16] suggested removal of the internal gibbus only in patients who have moderate to severe paraplegia; however, patients with mild paraplegia should not have decompression.[16] Anterior decompression with removal of an internal gibbus can be done by a thoracotomy approach but because kyphosis is severe, the anterolateral approach is easier and provides direct access to the apex of the kyphosis.[16]

Prevention of development of severe kyphosis is desirable to avoid late onset paraplegia.[16] In growing children in whom there are more than three vertebra affected in the dorsal spine, posterior spinal fusion should be done to stop growth of the posterior element of the spine and eventual progression of kyphosis. Adults who have loss of anterior vertical height of more than two vertebral body height, are likely to have severe kyphus develop on completion of treatment. These patients should have surgery during the active stage of the disease to improve the kyphus.

Paraplegia secondary to reactivation or recrudescence usually is severe and relatively rapidly developing but these patients respond early and better to treatment as compared with patients with late onset paraplegia with healed lesions.[14] Some of these patients have good neural recovery with antitubercular chemotherapy only.

Prognosis in Patients With Tuberculosis Paraplegia and Quadriplegia

Numerous factors have been identified that determine the neural recovery in patients with Pott's paraplegia. Younger age and good nutritional status are associated with better neural recovery. The cervicodorsal junction and upper dorsal spine affection show poor neural recovery because the spinal canal is narrow. The patients with paraplegia with active disease have a better chance of neural recovery as compared with patients with paraplegia with healed disease. Patients with severe kyphosis have poor neural recovery. Patients with neurologic complications of gradual onset and shorter duration have better neurologic recovery than patients with neurologic complications of a longer duration and rapid onset. Rapidly progressive paraplegia signifies a mechanical insult because of pressure from disc or sequestrae, pathologic subluxation or dislocation, or vascular catastrophe, and shows the worst prognosis. In long-standing compression, some permanent changes in the cord may be responsible for nonrecovery or poor recovery. Patients with severe paraplegia (Stage IV paraplegia) have poor neural recovery. Patients with typical anterior vertebral body disease have a better neural recovery as compared with patients with grossly unstable panvertebral lesions.

Jain et al[8] studied MRI observations in 43 patients with tuberculosis of the spine with neurologic complications sequentially and correlated them with the clinical behavior of the disease. Extradural compression attributable to fluid on MRI scans resolves well with treatment and patients have a good neural recovery in comparison with extradural compression of mixed or granulomatous (dry) nature showing constriction of the cord. Patients with preserved cord volume with edema or myelitis of the cord on MRI scans have a good neural recovery. Myelomalacia of the spinal cord was found to be a poor prognostic sign of neural recovery.[8] The magnitude of thinning of the spinal cord did not always correlate with severity of neural deficit; however, thinning of the cord in association with myelomalacia, syrinx, or both carries a bad prognosis.[8] The patient is likely to have better neural recovery if, on surgical decompression, pus and granulation tissue are drained (wet lesion) in comparison with thick inspissated pus, caseous tissue, fibrous tissue, bony sequestrae or bony salient and disc (dry lesion).

Evoked potential studies are helpful in objectively documenting the respective sensory and motor deficit in patients with Pott's paraplegia. The motor evoked potential is more frequently abnormal as compared with sensory evoked potential and correlates with respective clinical improvement.[13]

Craniovertebral Tuberculosis

Tuberculosis of the upper cervical spine seems to begin either in the retropharyngeal space with secondary involvement of bone or rarely in the bone itself.[10] With progression, increasing ligamentous involvement and osteolytic erosions of the odontoid or into C1 allow anterior subluxation of C1 on C2 and proximal translocation of the odontoid.[10] In the most severely affected patients, there is complete loss of the odontoid and anterior arch of C1 with a grossly unstable articulation between the occiput and C2. The subluxation or dislocation of C1 on C2 is reported in 56% to 75% patients at the time of presentation.[10]

The spinal cord at the medullary cervical junction is threatened by atlantoaxial subluxation and upward translation of the dens, compression by tubercular abscess, inflammatory edema of the spinal cord, and direct tubercular invasion of the cord.[1] Not all patients with subluxation or dislocation of C1 on C2 have a neural deficit although some patients have neural recovery despite persistence of dislocation of C1 on C2, which suggests that dislocation of C1 on C2 is not the prime cause for deficit.[9,17] These patients present with severe neck pain, limitation of movement, local tenderness, tilt of the neck, tendency to support the neck, difficulty in swallowing, hoarseness of voice, stridor, or even lateral nystagmus.

Advanced cases can be diagnosed on plain radiographs by regional osteoporosis, increased prevertebral soft tissue shadow in front of the anterior arch of C1, and subluxation or dislocation of C1 on C2. In a patient without dislocation of C1 on C2, plain radiographs only may show an increased prevertebral soft tissue shadow, more than 7 mm in front of the anterior arch of C1. The alteration of bony texture cannot be appreciated on plain radiographs; therefore, on strong radiologic suspicion one has to resort to CT and MRI scans to see the destruction of bone.[9]

Treatment

Crutchfield tong traction or a halo vest seem to be effective methods to immobilize and achieve reduction in a subluxated or dislocated upper cervical spine.[1] Transoral debridement is a simple procedure for decompression and obtaining a biopsy specimen. However, transoral debridement for reduction of subluxation or dislocation is a major procedure and has a 50% failure rate.[9] Fine needle aspiration cytology can be done for a histologic diagnosis either from a palpable cold abscess or by a transoral route.

Patients without subluxation or dislocation should be treated with a cervicothoracic orthosis (9 to 12 months) and bedrest (4 to 6 weeks) followed by mobilization while receiving antitubercular chemotherapy. Patients with subluxation or dislocation (Fig 4) are

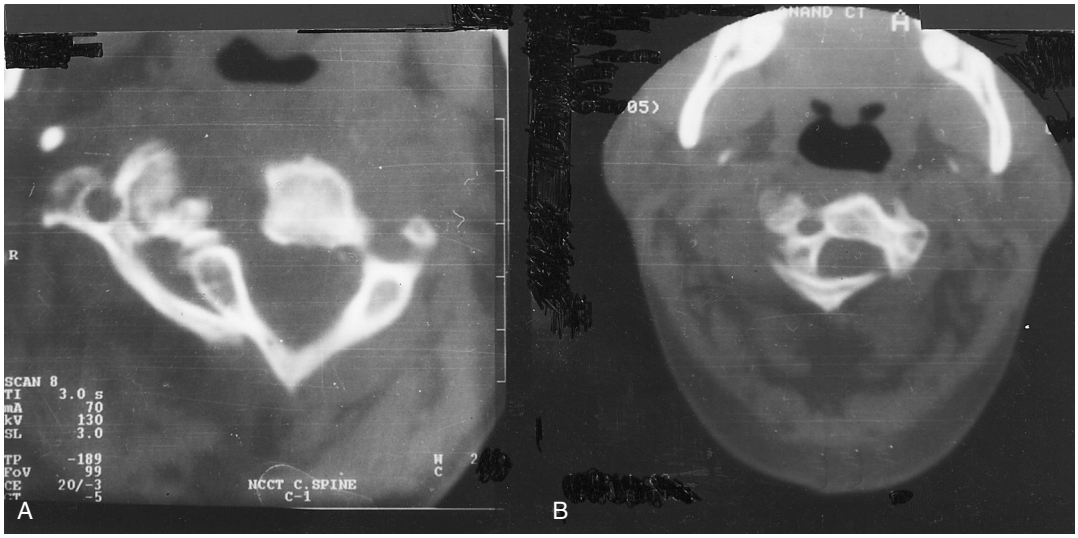

Fig 4A–B. Axial CT scans were obtained of a 23-year-old patient with quadriplegia who has tuberculosis of the C1–C2 spine. (A) A CT scan taken before treatment shows displacement of one vertebrae over the other. (B) Realignment of the spine on Crutchfield tong traction is shown.

treated best by Crutchfield tong traction or a halo vest to achieve reduction. At 3 months, the stability of spine can be assessed by a stress film (lateral view of the spine in flexion and extension). If the distance between the dens and anterior arch of the atlas increases more than 3 mm in two views, the spine can be considered unstable and posterior spinal fusion should be contemplated on Crutchfield traction. The spine in these patients usually remains stable in a subluxated or reduced position at 3 months. Jain et al[8] reported 11 such cases as Stage I (six patients), Stage II (two patients), and Stage III (three patients) on the same protocol.

Surgical decompression is indicated when a retropharyngeal abscess produces dysphagia or hoarseness of the voice or if the patient does not have an adequate clinical response and neural recovery with nonoperative treatment.

Tuberculosis of the Cervical Spine and Cervicodorsal Junction With Neurologic Deficit

The overall incidence of cord compression at the cervical spine and cervicodorsal junction is slightly higher than in the dorsal spine. The cervicodorsal junction is a difficult area for an early clinicoradiologic diagnosis.[9] Disease in the cervical spine does not produce as severe a progression of the kyphosis as in the dorsal spine. These cases usually are diagnosed when the patient already has neurologic complications. Universal surgical extirpation is not warranted for fear of late onset quadriplegia because of severe kyphosis. The middle path regimen seems to be effective for patients with cervical spine tuberculosis.

The patient should be treated with Crutchfield tong traction and complete chemotherapy. If adequate neural recovery occurs, a four-post collar should be applied after 6 weeks with gradually increasing mobilization. The patients with tuberculosis of the cervicodorsal junction and upper dorsal spine should be mobilized with a four-post collar extended on a Taylor's brace or extended sternooccipitomandibular immobilizer brace. Approximately 60% to 65% of the patients have neural recovery with nonoperative treatment but the remaining patients require surgical decompression.

References

1. Fang D, Leong JCY, Fang HSY: Tuberculosis of the upper cervical spine. J Bone Joint Surg 65B:47–50, 1983.
2. Frankel HL, Hancock DO, Hyslop G, et al: The value of postural reduction in the initial management of closed injuries of the spine with paraplegia and tetraplegia. Paraplegia 7:179–192, 1969.
3. Griffiths DLL: The Treatment of Spinal Tuberculosis. In McKibbin B (ed). Recent Advances in Orthopaedics. Edinburgh, Churchill Livingstone 1–17, 1979.
4. Hodgson AR, Stock FE: Anterior spinal fusion for the treatment of tuberculosis of the spine. J Bone Joint Surg 42A:295–310, 1960.
5. Hodgson AR, Skinsnes OK, Leong JCY: The pathogenesis of Pott's paraplegia. J Bone Joint Surg 49A:1147–1156, 1967.
6. Hsu LCS, Cheng CC, Leong JCY: Pott's paraplegia of late onset: The causes of compression and results after anterior decompression. J Bone Joint Surg 70B:534–538, 1988.
7. Jain AK, Aggarwal A, Mehrotra G: Correlation of canal encroachment with neurological deficit in tuberculosis of spine. Int Orthop (SICOT) 23:85–86, 1999.
8. Jain AK, Jena A, Dhammi IK: Correlation of clinical course with magnetic resonance imaging in tuberculous myelopathy. Neurol India 48:132–139, 2000.
9. Jain AK, Kumar S, Tuli SM: Tuberculosis of spine (C1-D4). Spinal Cord 37:362–369, 1999.
10. Lifeso R: Atlantoaxial tuberculosis in adults. J Bone Joint Surg 69B:183–187, 1987.
11. Lifeso RM, Weaver P, Harder EH: Tuberculous spondylitis in adults. J Bone Joint Surg 64A:1405–1413, 1985.
12. Mermelstein LE, Keenen TL, Benson DR, et al: Initial Evaluation and Emergency Treatment of the Spine-Injured Patient In Browner BD, Levine AM, Jupiter JB, et al (eds). Skeletal Trauma. Ed 2. Philadelphia, WB Saunders Company 751, 1998.
13. Misra UK, Kalita J: Somatosensory and motor evoked potential changes in patients with Pott's paraplegia. Spinal Cord 34:272–276, 1996.
14. Moon MS, Ha KY, Sun DH, et al: Pott's paraplegia. Clin Orthop 323:122–128 1996.
15. Rajasekaran S, Soundarapandian S: Progression of kyphosis in tuberculosis of the spine treated by anterior arthrodesis. J Bone Joint Surg 71A:1314–1323, 1989.
16. Tuli SM: Current concepts: Severe kyphotic deformity in tuberculosis of the spine. Int Orthop (SICOT) 19:327–331, 1995.
17. Tuli SM: Management and Results. In Tuli SM (ed). Tuberculosis of the Skeletal System. Ed 2. New Delhi, Jaypee Brothers Medical Publishers Ltd 240–261, 1997.
18. Upadhyay SS, Seff P, Saji MJ, Seff B, Hsu LC: Surgical management of spinal tuberculosis in adults. Clin Orthop 302:173–182, 1994.
19. Watts HG, Lifeso RM: Tuberculosis of bones and joint: Current concepts review. J Bone Joint Surg 78A:288–298, 1996.
20. Wood GW: Infection of the Spine. In Crenshaw AH (ed). Campbell's Operative Orthopaedics. Ed 7. Vol 4. St Louis, CV Mosby 3326–3342, 1987.