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Posterior Spinal Tuberculosis: A Review

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Abstract

Natural history of posterior spinal tuberculosis has been described. Classifications of the posterior spinal tuberculosis disease process and principles of management based upon the clinical behavior of the disease has been highlighted and emphasized. A thorough review of literature was conducted with the aim to provide the clinico-radiological correlation of the natural history of posterior spinal tuberculosis in described. Management strategy is developed based upon the severity of the clinical behavior of the disease. In anterior spinal tuberculosis, motor fibers are compressed first as they are placed anterior to the sensory tracts in the spinal cord. The sensory fibers are therefore involved in late stages. Ironically, in posterior spinal tuberculosis when compression is predominantly from the posterior aspect of the cord, we again find that motor fibers are involved prior to the sensory fibers. This is in contradiction to the general belief. It is difficult to offer any simple explanation to this apparent paradox. In general, motor fibers are considered more susceptible to pressure effect, whereas sensory fibers are more susceptible to ischemia.

That is why in compression paraplegia, signs and symptoms of motor loss appear prior to the sensory loss, as collaterals prevent ischemia for quite some times. In posterior spinal tuberculosis when compression is from the posterior aspect of the cord, at first pressure is exerted on the column of cerebrospinal fluid (CSF) surrounding the cord and gets transmitted to the ligamentum denticulatum. Motor fibers in the close vicinity, get pulled and show early involvement. Secondly, in compression from the posterior aspect of the cord, the cord is displaced anteriorly and anteriorly placed motor fibers are compressed against the anterior wall of the bony spinal canal causing early motor fiber functional loss. Therefore similar classification of paraplegia predominantly based upon the progressive motor weakness is valid for paraplegia noted following posterior spinal tuberculosis. Neurological deficit grading based management is developed. Grade 1 and 2, conservative treatment, grade 3, gray zone and grade 4, operative treatment is emphasized. The five stages of natural history of tuberculosis of spine have been developed from the clinician's point of view. However, indications of surgery are different than what are described for the anterior spinal tuberculosis. Principles of management with role of rest, braces, chemotherapy and surgery are discussed. Management of posterior spinal tuberculosis of spine, in general, it is no different than management of soft tissue tuberculosis, in HIV negative or positive patients. Role of surgery is very different than anterior spinal tubercolosis. Management of posterior spinal tubercular paraplegia, is simple, logical, efficient and easy to understand and remember by any orthopedic/treating surgeon.

Keywords: Spinal tuberculosis; Clinical behavior; Ischemia

Introduction

Posterior spinal tuberculosis or tuberculosis of the neural arch is caused by the same type of Mycobacterium tuberculosis which produces classical Pott's spine or anterior spinal tuberculosis. However, posterior spinal tuberculosis is rather uncommon with sporadic publications from time to time appear form different parts of the world. Maximum number of cases with most vivid description has been from India [1,2]. Better imaging techniques like computerized tomography (CT scan), magnetic resonance imaging (MRI) and bone scan/positron emission tomography (PET-CT scan) have increased the detection. High incidence of tuberculosis in association with HIV infection has added another dimension to this problem [3,4]. Emergence of HIV and increase in international human migration has again increased the interest in the developed countries, from where it was once considered eradicated [5].

Epidemiology

No preponderance of age, sex or race has been attributed. Exact incidence of posterior spinal tuberculosis is not known. Incidence of less than 2% in non-endemic areas and between 5% to 10% in endemic areas has been reported [1,2,6]. Scott et al. [7] reported 35.5% incidence of posterior spinal tuberculosis or tuberculosis which is very high and appears to be unrealistic, because they have also included tuberculosis of the posterior part of the vertebral body along with the tuberculosis of neural arch.

The lesion commonly arises in the thoracic, thoraco-lumbar and lumbar vertebral arch in that order. Cervical lesions are extremely rare. Tuberculosis of the posterior arch of atlas [8] is included under craniovertebral tuberculosis. Unlike classical pott's spine, posterior spinal tuberculosis commonly involves one vertebral arch only.

Pathology

Like any other osteoarticular tuberculosis, posterior spinal tuberculosis is always secondary to primary tubercular lesion existing

somewhere else in the body. Implantation of the mycobacterium tuberculosis is through the hematogenous route. The venous plexus surrounding the spinous, transvers and articular processes and on the posterior aspect of lamina anastomose freely with the other neighboring venous plexuses help infection to reach and lodge in the neural arch [9,10]. However, in over two third of cases, primary lesion may remain obscure. Lesions may involve any part of the neural arch viz. lamina, pedicle, spinous or transverse processes. It appears that pedicle is the most common site of involvement [1,11] where lamina as the most common site of involvement has also been reported [2,11]. Facet joints may also be involved. The involvement may be in isolation or in combination with anterior spinal tuberculosis or classical Pott's spine. When posterior spinal tuberculosis exists in association with anterior spinal tuberculosis at the same level, it is called composite lesion or pan-vertebral involvement. At times it is difficult to determine the initial site of involvement and lodgment of mycobacterium tuberculosis as the disease from anterior part can spread posteriorly and involve the neural arch and vice-versa. To avoid the confusion some authors conceived the concept of tuberculosis involving the vertebral ring, combining the tuberculosis of the posterior part of the vertebral body along with the tuberculosis of neural arch [7].

Formation of cold abscess is an important feature of bony tuberculosis and is seen in over two third of cases. It may present in different locations, viz. an abscess posteriorly in the mid line, near the mid line or bilateral abscesses on both sides of the mid line in the back. In composite lesion or pan-vertebral involvement, abscess may be located in the paravertebral gutter, psoas abscess, anterior trunk wall abscess or abscess in the Petit's triangle. Sometimes these abscesses may burst through the skin and form chronically discharging sinuses. These sinuses may be found in different stages of healing. They have classical ring of pigmentation at their mouth, may be fixed to the underlying bone and sometimes may appear healed with unstable scar. Midline sinuses are adherent to the underlying neural arch.

Posterior spinal tuberculosis may also be seen in association with extradural granuloma or a layer of thick granular tissue surrounding the dural sac circumferentially within the spinal canal [1,2] presenting as spinal tumor syndrome [12]. Such lesions are not seen on plane radiographs but picked up on CT scan or MRI. In pre CT and MRI days, such lesions were often missed and picked up during surgery. Myelogram would reveal dural indentation with filling defect or complete block. Spinal cord may be compressed from posterior aspect [13-15] or from anterior aspect [16].

Rarely composite lesion or panvertebral involvement may exist in association with the tubercular destruction of the facet joints, rendering the spine unstable. Clinically it manifests as kyphoscoliosis and radiological as lateral shift phenomena, noted on plane radiograph. It is most commonly noted at the thoracolumbar junction [1].

Classification of posterior spinal tuberculosis

Kumar [1] proposed a four-point classification of posterior spinal tuberculosis according to site of legion, stage of lesion, associated involvement and functional loss.

Site of lesion: The lesions may be classified anatomically on the basis of the part of the neural arch involved viz. involving pedicle, lamina, transverse process, spinous process or facet joints. These lesions may be in isolation or in combination with the anterior spinal lesion called composite lesions or pan vertebral lesion. Involvement of the vertebral ring i.e. involvement of the neural arch with involvement of the posterior vertebral body has also been described [7]. Adjoining rib may also be involved.

Stage of lesion: Depending upon the activity of the lesion, it can be grouped as:

- An active or florid lesion which is not under control.
- A healing lesion which is regressing or lesion under control.
- A healed or cured lesion having undergone fibrous or fibro-osseous healing.
- A reactivated lesion.

Associated lesions: It is not uncommon to find associated granulomatous lesions within the spinal canal. Such lesions may be in for of extradural or subdural granuloma or spinal tuberculoma.

Functional loss: Posterior spinal tuberculosis is notorious for causing high incidence of neurological deficit [1,17]. It is primarily because of the fact that the tuberculous debris is mechanically pushed into the spinal canal during recumbence. On the basis of neurological manifestations, the lesion can be divided into three groups;

- Those without neurological signs and symptoms.
- Those threatening to produce neurological signs/symptoms like composite lesions and lesions associated with intra-spinal granulomatous lesions.
- Those with established neurological signs/symptoms of varying degrees. These neurological manifestations may range from irritation of the nerve root producing radicular pain to the compression of the cord producing paraplegia of varying grades.

Paraplegia: When compression is from the anterior side of the cord as seen in the classical Pott's spine or anterior spinal tuberculosis, motor fibers are compressed first as they are placed anterior to the sensory tracts in the spinal cord. The sensory fibers are therefore involved in late stages. Ironically, in posterior spinal tuberculosis when compression is predominantly from the posterior aspect of the cord, we again find that motor fibers are involved prior to the sensory fibers [1,18,19].

This is in contradiction to the general belief [20]. It is difficult to offer any simple explanation to this apparent paradox. In general, motor fibers are considered more susceptible to pressure effect, whereas sensory fibers are more susceptible to ischemia. That is why in compression paraplegia, signs and symptoms of motor loss appear prior to the sensory loss, as collaterals prevent ischemia for quite some times. In posterior spinal tuberculosis when compression is from the posterior aspect of the cord, at first pressure is exerted on the column of cerebrospinal fluid (CSF) surrounding the cord and gets transmitted to the ligamentum denticulatum. Motor fibers in the close vicinity, get pulled and show early involvement. Secondly, in compression from the posterior aspect of the cord, the cord is displaced anteriorly and anteriorly placed motor fibers are compressed against the anterior wall of the bony spinal canal causing early motor fiber functional loss. Therefore similar classification of paraplegia predominantly based upon the progressive motor weakness is valid for paraplegia noted following posterior spinal tuberculosis (Table 1).

Grade of	Complaints/Symptoms		Examination /Neurological deficit		
Paraplegia	Weakness	Walking	Motor	Sensory	Autonomic
1	Negligible or weakness appearing after exercise	Able to walk without support	Extensor plantar + brisk ankle jerks, muscle power grade IV to V	Nil	Nil
2	Mild or Feels weakness	Able to walk with support	Motor weakness, brisk tendon jerks, ill sustained muscle clonus, muscle power grade III	Sensory dulling or paresthesia	Nil
3	Moderate or weakness is more marked	Not able to walk Confined to bed Can move limbs	Brisk tendon jerks, sustained muscle clonus, muscle power grade I to II	Hypoesthetic or anesthetic patches	May be present
4	Severe or Complete loss of power and control	Not able to move the limbs even in the bed	(a)Paraplegia in extension, power grade 0 (b)Paraplegia in flexion, power grade 0, flaccid paralysis	Total loss	Complete loss of bladder and bowel control and incontinence

Table 1: Classification of paraplegia in tuberculosis of spine [17,18].

Postulations of Bosworth et al. [21] suggesting the presence of some chemical substance in the tubercular pus inhibiting the conduction of the spinal cord seems only hypothetical. Functions return when pressure is relieved. Following spinal decompression, sensory recovery precedes motor recovery. Paraplegia from the extradural granuloma responds extremely well to decompression and ant tubercular drugs [2].

Clinical features

Clinical presentations of patients suffering from posterior spinal tuberculosis can be grouped under the following heads:

- Features concerning tubercular toxemia like loss of weight, loss of appetite and evening rise if temperature etc.
- Features related to the primary site of tuberculosis like tuberculosis of chest, abdomen and glands etc.
- Features related to posterior spinal tuberculosis. Localized bony pain with tenderness in the midline or diffuse chronic back pain with paraspinal muscle spasm may only be the presenting feature in the early stages. There may also be a boggy swelling in the midline or in paraspinal region a cold abscess, which may travel to distant places along the course of the nerve. There may also be formation of sinus which is fixed to the underlying bone.

Spinal curvature is not altered unless there is composite or panvertebral involvement. In such cases kyphosis or kyphoscoliosis is noted. Patients with lesions closed to the nerve root may complain of radicular pain and with displacement of tubercular debris into the spinal canal or with associated extradural granuloma may also have various grades of paraplegia.

In some of the patients, the neural arch tuberculosis may remain almost symptom free and is detected incidentally while screening the patient for vague abdominal or back problems.

Discussion and Conclusion

It is the first requirement. Thorough clinical history and examination coupled with good radiological evaluation of the spine provides the diagnosis with adequate certainty in most of the cases. Hematological, microbiological, serological, immunological and other available diagnostic tools should be used to clinch the diagnosis. Conventional radiographs are of limited help as lesions smaller than 1.5 cm are often missed due to overlapping shadows. The radiographic differentiation from neoplastic lesion may often be difficult. However certain associated radiographic features may offer certain clues. The sharp margins of the destroyed bones and presence of sclerosis and or sequestra are features suggesting a chronic inflammatory/infectious process [6].

myelography may be useful in determining the extent of intraspinal involvement and unparticular to determine the extent of extradural granuloma [22]. MRI is often positive much before plane radiographs and particularly helpful in diagnosis of extradural granuloma and extent of canal encroachment by the tuberculous debris. Delineation of primary site of tuberculosis, in approximately 20% of cases, may further support the diagnosis. Plane radiograph, CT scan and MRI are the useful radiological tools with well-established roles [4,23]. Nuclear scintigraphy by 99mTechnitium (MDP or HDP) or Gallium (67Ga) is cost effective and highly sensitive but nonspecific and may be used in localizing multifocal lesions. It is often positive much before plane radiograph, CT or MRI reveals any definitive changes. SPECT-CT increases the lesion identification accuracy. High false-negative rates of Technetium (up to 35%) and Gallium (70%) bone scans have also been reported [23].

An anti-tubercular drug (Rifampicin, Isoniazid, Pyrazinamide and Ethambutol {RIPE}) is the prime anchor in the management of posterior spinal tuberculosis. Anti-tubercular drugs reach at the site of osteoarticular tubercular lesion in adequate concentration when administered in therapeutic doses [24-27]. Posterior spinal tuberculosis is no exception. It not only cures the spinal lesions but also the primary site of tubercular disease. Posterior spinal tuberculosis is notorious for producing neurological symptoms. Due to high chances of neurological involvement excisional surgery is indicated. Laminectomy with debridement of the lesion is recommended which also helps in de-bulking the lesion but also in excision of associated lesion like extradural granuloma and pathological confirmation of the diagnosis. Prognosis of neurological recovery is extremely favorable.

Immobilization after laminectomy is not necessary if only posterior spinal elements are involved, as it does not lead any kind of significant spinal instability of the spine. In composite lesions, where associated anterior spinal tuberculosis is also present, in such patients spinal

stabilization is desired because of the fear of post-operative displacement of the spinal column.

Patients with extradural granuloma should be treated surgically by excising the granuloma: as "if treated conservatively by conservative anti-tubercular treatment, the granuloma will be replaced by the fibrous scar tissue which will maintain compression" [1]. Excisional surgery like excision of the spinous or transverse process may at times be done with the aim, not only to remove the tubercular debris and shorten the healing time but also for the confirmation of the diagnosis. Indications of surgery are different in posterior spinal tuberculosis than anterior spinal tuberculosis [28].

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