CASE REPORT

Intradural Tuberculer Abscess in Lumbosacral Region in Infant

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Introduction:

Central nervous system (CNS) disease caused by *Mycobacterium tuberculosis* is an uncommon yet highly devastating manifestation of tuberculosis, which was universally fatal in the era before antituberculer therapy. CNS tuberculosis accounts for approximately 1% of all cases of tuberculosis, carries a high mortality and a distressing level of neurological morbidity, and disproportionately afflicts children and human immunodeficiency virus (HIV)-infected individuals. Due to its relative rarity and the protean nature of the symptoms, tuberculosis of the CNS remains a formidable diagnostic challenge. Because the burden of CNS tuberculosis lies largely in resource-starved regions of the world, additional challenges in implementing practical and usable methods to diagnose and treat this disease remain largely unmet. While other clinical manifestations of tuberculosis have received considerable research attention, fundamental questions regarding the pathogenesis, diagnosis, treatment, and management of CNS tuberculosis remain unanswered. What is the best way to diagnose CNS tuberculosis? What is the optimal treatment for this disease? How can we mitigate the significant neurological morbidity among survivors? How can we more rapidly diagnose CNS tuberculosis? These questions still remain open. The purpose of this review is to highlight the current understanding of the neuropathogenesis of *M. tuberculosis* and to discuss certain clinical, diagnostic, and therapeutic aspects of CNS tuberculosis.

Case report:

Puja Sarker, 20 month old child reasonably well 11 months back. Then she developed fever and was treated as out patient. As there was no improvement of her condition she was admitted in Tangial Sador Hospital. Unfortunately she had no marked improvement despite hospital stay for several days. She was referred to Dhaka for better management and undergone several investigations under the supervision of a child specialist & treated as a case of pyogenic meningitis. Again the patient failed to show any satisfactory improvement. At this stage she was also treated with antimalarial drugs. After few days she was discharged as there was some improvement of her condition. Then again she was backed to Dhaka as she developed some swelling over the medial aspect of her right thigh. It was associated fever and neck rigidity according to the statement of her mother.

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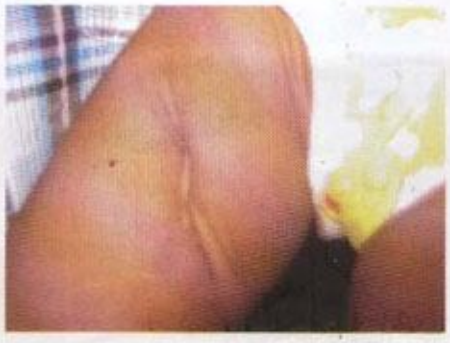

Figure-1: Site of abscess at the thigh

She had some improvement after the drainage of pus but not cured totally. Hence the child was taken to another Paediatric Hospital. But her temperature did not subside totally. Neck rigidity was present. CT scan of brain & CSF study was done again but it did not provide with no definite clue of her illness. She was treated with parenteral antibiotic. She was improved a little & was discharged. The patient was anaemic (Hb 5gm %) which was corrected by blood transfusion. As the fever continued with neck rigidity the child also treated for meningitis. The child condition improved a bit. But still there was no remission of fever. In the mean time her mother had a new complain about a swelling over her lumbosacral region which was overlooked by her previously. Hence she was referred to a Neurosurgeon. The history revealed that the patient was having muscle wasting over both the lower limbs more marked in the left gluteal region.

MRI of the lumbosacral spine was advised. MRI revealed a hypointense lesion at L4, L5 & S1 level in T1 weighted film & hyperintense in T2 weighted film. The patient was advised to contact after the improvement of her present condition. After 1 month when the patient was admitted and advised for surgery. As pre-operative procedure routine examination of blood revealed marked neutrophilic leucocytosis (T.C- 25,000/mm^3 of blood) but the patient was afebrile !

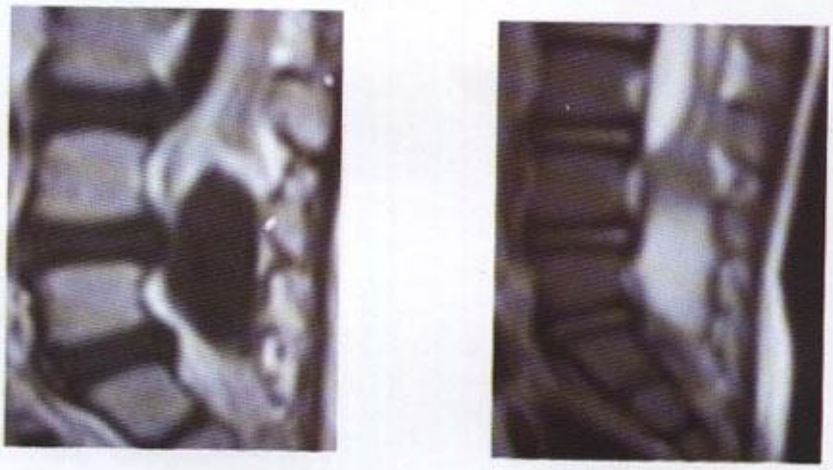

Figure-2: T1 and T2 weighted MRI showed a hypo and hyperintense lesion at L4, L5 & upper border of S1

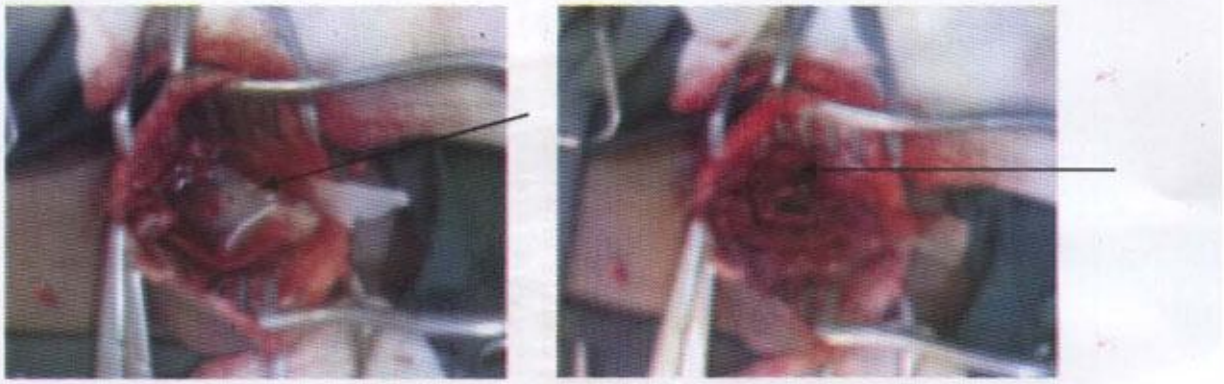

Figure -3: Per-operative picture of the lesion

By posterior midline incision laminectomy performed at L4 & L5. Dura opened by posterior midline incision and an intradural lesion was found. After freeing the overlying nerves. Huge amount pus was came out from the lesion and was aspirated. Through irrigation was done with normal salaine & the adhesion was made free. Pus and part of the wall of the lesion was sent for culture, cytology, gram & AFB staining, also for AFB culture & histopathology. AFB stain showed multiple AFB and anti tubercular therapy was started. Patient condition gradually improved. The patient develop no fever afterward. Also there was all over marked improvement. Culture report after six months revealed tuberculous bacilli.

Discussion:

Infection of the CNS is one of the most devastating clinical manifestations of tuberculosis. In a large-scale epidemiological study of extrapulmonary tuberculosis in the United States, CNS involvement was noted in 5 to 10% of cases[1], with more recent CDC data in 2005 indicating that 6.3% of extrapulmonary cases (1.3% of total tuberculosis cases) involve the CNS[2]. In the largest prospective epidemiological study on CNS tuberculosis, the chance of developing CNS tuberculosis was 1.0% among 82,764 tuberculosis cases from 1970 to 2001 in a Canadian cohort[3]. Several risk factors for CNS tuberculosis have been identified. Both children[4] and HIV-co-infected patients[5] are at high risk for developing CNS tuberculosis. Other risk factors include malnutrition and recent measles in children[6] and alcoholism, malignancies, and the use of immunosuppressive agents in adults[7]. Studies conducted in developed countries have also identified that foreign-born individuals (individuals born outside of developed countries) are overrepresented among CNS tuberculosis cases[8]. In a large study in Taiwan, Lu et al. reported that 1.5% of tuberculosis deaths between 1997 and 2001 were attributable to CNS disease, a percentage that had increased from previous years[9]. *M. tuberculosis* is an aerobic, nonmotile, non-spore-forming, acid-fast bacillus (AFB) that infects primarily humans. Its doubling time is quite slow (15 to 20 h) and requires several weeks to grow on conventional Löwenstein-Jensen medium, where it tends to grow in parallel groups, producing the colonial characteristic of serpentine cording. Biochemical as well as RNA/DNA-based methods can identify *M. tuberculosis* from other AFB. This hematogenous seeding occurs

most frequently in regions of the body that are highly oxygenated, including the brain. A complex interplay of host immune factors and *M. tuberculosis* virulence factors in the end determines whether or not the infection is contained and whether, or to what extent, the dissemination of the bacilli leads to clinical disease[10].

For CNS tuberculosis, the disease begins with the development of small tuberculous foci (Rich foci) in the brain, spinal cord, or meninges. The location of these foci and the capacity to control them ultimately determine which form of CNS tuberculosis occurs. CNS tuberculosis manifests itself primarily as tuberculous meningitis (TBM) and less commonly as tubercular encephalitis, intracranial tuberculoma, or a tuberculous abscess.

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