Tubercular prevertebral and epidural abscess is rare when compared to pyogenic abscess in pediatric age group. These are usually secondary to tuberculosis of spine which is the commonest form of skeletal tuberculosis. Mostly dorsolumbar spine is involved and the involvement of cervical spine is rare (7%) [1]. Cervical spine tuberculosis is the rarest and the most dangerous form because of the diagnostic difficulties and serious residual disabilities [2]. We report a case of cervical spine tuberculosis with prevertebral and epidural abscess in a five month old child which is extremely rare.

**CASE REPORT**

A five month old Indian girl child presented with fever for 2 months, dysphagia and stridor for 1 month. Child began to have fever 2 month back which was usually low grade, followed by development of stridor at 4 month of age which was progressive, prominent at the time of feeding and crying and associated with difficulty in breathing. It was associated with dysphagia which was progressive; swallowing was followed by choking, cough and rapid breathing, resulting in regurgitation of feed, excessive cry and stiffening of body. There was significant history of not gaining weight during the course of illness. Also, history of birth asphyxia requiring mechanical ventilation for 12 days was present. There was no history of any penetrating trauma, ear discharge, convulsions or significant weakness in any limbs. Parents were healthy. Mother had a history of primary infertility for 4 years for which she took some herbal medications. The cause of infertility couldn’t be determined as she declined consent for gynaecological examination. Child had no contact with TB patients.

On examination, child was sick looking and thin built. She was pale, febrile, anicteric with no significant lymphadenopathy. Neck was kept hyperextended, spine was normal on palpation and there was no paraspinal fullness. She had respiratory distress in the form of tachypnea, suprasternal, subcostal and intercostal retractions with a high pitched stridor which increased on crying and swallowing. She had increased tone in lower limbs more than upper limbs with brisk deep tendon reflexes. Throat examination under general anesthesia...
revealed a bulge in posterior pharyngeal wall. The mucosa was normal with no erythema, edema or pus point over it.

Figure 1 – X-ray Chest, AP view showed mild perihilar infiltrates in the lung parenchyma with normal bone and soft tissue shadow

Chest X-ray was normal (Fig. 1) and mantoux was non-reactive. Her ESR was 52 mm/hr (Westergreen method) and USG abdomen showed multiple small (2-3 mm) calcific foci throughout the liver and spleen parenchyma (few calcific foci showed active halo around) which were suggestive of tubercular origin. MRI of cervical spine revealed C2-C3 spondylodiscitis with prevertebral abscess compressing the airway with epidural abscess causing compressive myelopathy, possibly tubercular in etiology (Fig. 2).

Figure 2 - MRI T1 weighted image cervical spine revealed C2-C3 spondylodiscitis with prevertebral abscess compressing the airway with epidural abscess causing compressive myelopathy, possibly tubercular in etiology

Keeping the clinical picture in view, child was started on broad spectrum antibiotics and abscess was drained by ENT surgeon. About 20 ml of pus was aspirated but culture showed no growth. Within 10 days of aspiration, child again became symptomatic and repeated MRI showed recollection. On repeat aspiration, cheesy material was obtained. It was sent for mycobacterial culture but showed no growth. In view of no response to antibiotics, aspiration of cheesy material, and the incidental finding of granulomas in liver and spleen, quadruple regimen of antitubercular medications (Isoniazid, Rifampicin, Pyrazinamide and Ethambutol) was started.

Figure 3 - MRI T1W image of cervical spine suggestive of osteolytic lesion involving C2-C3 vertebra with prevertebral and retropharyngeal collection. As compared to previous scan a decrease in size of collection was noted

The child responded and there was significant improvement in stridor and dysphagia. At the time of discharge, child was stridor free, accepting orally and was sent home on ATT. The child was doing fine at 2 month follow up with adequate weight gain and repeated MRI showed resolution of abscess and healing (Fig. 3).

DISCUSSION

Tuberculosis is highly prevalent in developing countries like India. It is estimated that 1 to 6% of children with primary infection may develop bone and joint tuberculosis in 1 to 3 years if left untreated (4). The most common bone to be involved in tuberculosis is spine and it accounts for nearly 50% of osteoarticular tuberculosis (3). Skeletal involvement is usually secondary with the primary lesion usually in chest and the infection spreads hematogeneously
via batson’s plexus. The mycobacterium which is carried hematogenously or lymphatically invades the anterior and subchondral portions of the body of vertebrae, leading to development of osteomyelitis. As the infection progresses, the periosteum and longitudinal ligament gets lifted off the surface of the vertebral bodies followed by caseation necrosis, abscess formation and destruction of vertebrae. Initially the abscess is close to the vertebrae, which presents as prevertebral or paravertebral abscess on plain radiograph. The abscess may bulge into the mouth or the pharynx resulting in dysphagia, pharyngeal discomfort and rarely airway obstruction and respiratory distress which was the initial presentation in our case.

Any abscess involving the vertebrae may progress and involve the epidural space. The cervical epidural abscess usually presents as nuchal stiffness or rigidity with paraspinal muscle spasm. The propensity of cervical lesions to cause neurologic deficit may be explained by the fact that the spinal canal in this region is small relative to the diameter of the cervical cord. The sequence of pathophysiological processes in cervical spine tuberculosis include: local inflammatory response, tuberculous vasculitis and ischemia, subluxation of the vertebrae, abscess on the spinal cord or nerve root and impringement of the discs. The degree of spinal cord compression depends on the size of epidural abscess. Usually the neurological deficit clinches the clinical diagnosis and site of abscess. Specific investigations include plain radiograph of the cervical spine, examination of abscess aspirate for acid fast bacilli and magnetic resonance imaging of the cervical spine for confirmation of the diagnosis.

The mainstay of treatment for tubercular epidural abscess is combined medico-surgical approach with surgical drainage under ATT cover. Securing the airway (either by intubation or tracheostomy) should take precedence in any case with features of airway compromise leading to impending failure. Although this was not required in our case and the abscess was drained by the ENT surgeon by transoral approach and it responded well on ATT.

CONCLUSION

The primary diagnosis of cervical spine tuberculosis in our patient was made on the basis of combination of clinical (dysphagia, stridor, prolonged fever, not gaining weight), radiological (prevertebral and epidural abscess on MRI and granulomas in liver and spleen) and non specific laboratory investigations (raised ESR) and history of primary infertility in mother. The learning point in this case report is the need for high index of suspicion, value of careful history and how combinations of non specific findings help in making the diagnosis of spinal tuberculosis.

REFERENCES