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Case Report

A Case Report on Tuberculous Myelitis with Tuberculous Meningitis

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Abstract:

Transverse myelitis is an extremely rare manifestation of central nervous system tuberculosis, involving 1 or more vertebral segments of the spinal cord. However, it may extend to involve 3 or more segments of the cord, which would then be designated as longitudinally extensive transverse myelitis (LETM). Tuberculous transverse myelitis may occur in isolation or in association with adjacent meningitis. Here, we report a 15-year-old girl who was referred with rapid onset of quadriparesis with altered level of consciousness and showed LETM on spinal MRI. This report indicates that spinal tuberculosis should be considered as a differential diagnosis of LETM, especially in endemic areas.

Keywords: *Longitudinally extensive transverse myelitis, Obstructive hydrocephalus, Tuberculous myelitis, Tuberculous meningitis.*

Introduction

Tuberculosis (TB) is a commonly encountered disease in South-East Asian countries specially in Bangladesh. Being a multisystem affecting disease, central nervous system (CNS) tuberculosis (TB) is the deadliest. Clinical CNS TB is seen approximately 1-2% of patients with active TB & constitutes 8% of extrapulmonary tuberculosis cases in immunocompetent individuals[1]. It is broadly classified as intracranial and spinal tuberculosis. The intracranial form comprises tuberculous meningitis, tuberculous encephalitis/encephalopathy, tuberculoma and tuberculous abscess.

Spinal tuberculosis is again divided into osseous and non-osseous tuberculosis. Non-osseous forms include spinal arachnoiditis, spinal tuberculoma, thrombosis of anterior spinal artery, and tuberculous myelitis [2]. Transverse myelitis (TM) is a rare disorder causing inflammation of the spinal cord. It is presented with an acute onset of weakness having a sensory level along with bowel/bladder involvement. Being an extremely rare manifestation itself, tuberculous myelitis occasionally met up the criteria for longitudinally extensive transverse myelitis (LETM)[3]

LETM is defined by a spinal magnetic resonance imaging (MRI) indicating a lesion extending over 3 or more contiguous segments of the spinal cord[4]. Our experience with a rare case of CNS tuberculosis with tuberculous meningoencephalitis and LETM is presented here. This case highlights that tuberculosis should always be considered in differential diagnosis for any pathology with extensive involvement of the meninges, brain, and spinal cord, especially in regions with a high prevalence.

Case report

A 15-year-old young girl admitted on 04 February 2025 with altered level of consciousness for 1 day, weakness of all four limbs for 7 days. Initially she was suffering from low grade continued fever for 3 months with weight loss, anorexia. 7 days prior admission she developed weakness of lower limbs. Weakness was progressive, started from lower limbs. It gradually affected both upper limbs 01 day before admission that made her bed bound associated with bowel & bladder incontinence. She had history of global headache, blurring of vision & double vision developed 2 days before hospital admission. But at that time she had no history of seizure, dysphagia, pain in eye movement, low back pain, any local pain or paresthesia, nausea, vomiting or hiccups

On examination she was mildly anemic, underweight with a GCS 10/15. Respiratory rate was 45/min with SpO_2 65% in room air with cyanosis, and positive signs for CO_2 retention-warm periphery, & bounding pulse. Initial ABG revealed acute respiratory acidosis due to type 2 respiratory failure. She had neck rigidity but absent Kernig's & Brudzinski's sign. There was flaccid type of quadriplegia with a muscle power 0/5, no muscle wasting or fasciculation, absent deep tendon reflexes in all four limbs, absent plantar response with a sensory level at C4 accompanied with bowel bladder incontinence requiring urinary catheterization. On cranial nerve examination, she had bilateral 6th cranial nerve palsy with horizontal diplopia but no ophthalmoplegia. Fundoscopic examination revealed bilateral papilloedema. A detailed optic nerve examination could not be done due to her debilitated condition. Other cranial nerves were intact.

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Routine investigations were normal except hyponatremia due to Syndrome of Inappropriate ADH Secretion (SIADH). SIADH was confirmed following evaluation of serum & urine osmolality, spot urine electrolytes; simultaneously considering other diagnostic criteria[5]. Contrast enhanced MRI of spine (Fig. 1) showed T2 hyperintensity in the spinal cord from C3-D12. MRI of brain (Fig. 2) revealed dilated ventricles causing obstructed hydrocephalus. CSF shows 650 cells with 90% lymphocytes, ADA 36 U/L, CSF glucose 41.04 mg/dl, CSF protein 133 mg/dl. CSF Gene Xpert was positive for *Mycobacterium tuberculosis (MTB)*. A work up was done to exclude pulmonary tuberculosis. But MTB was not detected in sputum by acid fast staining & Gene Xpert. Chest X-ray was also unremarkable.

To exclude the differentials viral encephalitis panel, TPHA, VDRL, ANA, ENA profile, Aquaporin 4- IgG or Anti MOG IgG were done & all came negative. CSF for oligoclonal band was also tested but came negative.

Since CSF study biochemically & microbiologically confirmed tuberculosis, we planned for starting anti tuberculous therapy (ATT) labelling it as a case of Tuberculous meningitis with tuberculous myelitis complicated by obstructive hydrocephalus with type 2 respiratory failure with hyponatremia due to Syndrome of Inappropriate ADH Secretion (SIADH).

After admission to Intensive Care Unit (ICU) at first our patient was mechanically ventilated for the treatment of type 2 respiratory failure. Adequate fluid, nutrition, prophylactic antibiotic, anti thrombotic prophylaxis with low molecular weight heparin were given as well as regular posture change was maintained. Measures for prevention of aspiration were also taken. Oral hygiene, bowel-bladder care were maintained. After proper evaluation she was started with antituberculosis drugs as per the recommendations of the World Health Organization for the treatment of central nervous system tuberculosis. A total 12 months of ATT has been started including intensive therapy with 4FDC drugs for 2 months followed by maintenance therapy with 2FDC for another 10 months. Adjuvant glucocorticoids has been given along with ATT. She received 8 weeks of dexamethasone, which included 4 weeks of intravenous dexamethasone starting at a dose of 0.4 mg/kg for 1 week and gradually tapering it off by 0.1 mg/kg/week over the next 3 weeks, followed by 4 weeks of oral steroids starting at a dose of 4 mg/d for 1 week and gradually tapering it off by 1 mg/week over the next 3 weeks. The other drugs used were intravenous 3% NaCl, acetazolamide, and diuretics for raised intracranial pressure or hydrocephalus; antiepileptic drugs for seizures; and pyridoxine for prophylaxis against isoniazid-induced peripheral neuropathy.

To treat hydrocephalus, she had undergone burr hole surgery & placement of Ommaya reservoir of Kocher's point. 30 ml of CSF has been daily drawn through Ommaya Reservoir in aseptic manner. Hyponatremia had been gradually corrected by meticulous fluid restriction as well as concurrent correction of underlying cause - CNS TB. Our patient needed persistent support of mechanical ventilation beyond 14 days. So tracheostomy was done at 14th day, then patient was shifted to ward. Afterwards, the tracheostomy tube was safely removed following successful trials.

At follow-up after 5 months, all of her symptoms including the level of consciousness & limb weakness were significantly improved. GCS becomes 15/15. Follow up neurological examination of upper limbs reveals wasting of muscles without any fasciculation, hypotonia with muscle power 4/5 proximally & distally. Jerks are diminished. All types of sensory modalities are intact.

In the lower limbs there is also muscle wasting without any fasciculation, hypotonia with muscle power 2/5 in both proximal & distal group of muscles. Jerks are brisk with bilateral plantar extensor. Clonus is absent. There is a sensory level up to L2. Bilateral 6th nerve palsy, horizontal diplopia, bilateral papilloedema – all got resolved. But the bowel & bladder incontinence still persists.

To evaluate treatment response we repeated MRI of dorsal spine with contrast (Fig. 6a & 6b). It reveals spinal arachnoid cyst with myelomalacic change evidenced by a well defined extramedullary cystic area at right side of the spinal cord which is T1WI- hypointense, T2WI- hyperintense, after intravenous contrast no enhancement is seen. This lesion compress & displace the spinal cord towards contralateral side extending from the visualised part of the cervical segment to D10 vertebral level.

There is also (Fig. 7) long segment of T2 hyperintense area involving D2 – D10 segments of spinal cord. Repeat MRI of brain (Fig. 8) shows resolving hydrocephalus.

Along with the improvements of cognitive function & limb weakness, unfortunately she has to bear complications of involvement of brain & spinal cord. Multiple bed sores had been developed in lower back, and buttocks. She also developed mild dementia with a Mini Mental State score 21 out of 30. From history of her parents & also from our observation there are certain changes in her behaviour that developed through the course of her illness. She has grown emotionally labile, excessively fearful to seemingly normal situations but does not give any history of hallucination. For last 3 months she has been experiencing neuropathic pain in the form of moderate to severe episodic shooting pain in the left chest wall appearing in almost every night which often wakes her from sleep. Whereas examination of her chest reveals normal findings.

At the same follow up visit, she has been seen by neurologist, neurosurgeon, physiatrist, and psychiatrist of Dhaka Medical College Hospital. We have reorganized her treatment according to multidisciplinary approach. In addition to anti tuberculous therapy, she has been added Gabapentin for neuropathic pain, Selective Serotonin Reuptake Inhibitor (SSRI) for emotional lability & also for soothing neuropathic pain along with reinforcement of regular physiotherapy (as patient was somewhat non compliant before & was not on regular physiotherapy), maintenance of adequate nutrition & proper counselling. According to the suggestions of Dept. of Neurosurgery, the arachnoid cyst will be considered for

excision in a hope for full neurological recovery, if the patient develops persistent or residual limb weakness after full completion of anti TB chemotherapy (after 12 months) provided regular proper physiotherapy is given. Ommaya reservoir of Kocher's point is planned to remove after full resolution of obstructive hydrocephalus. The patient is also advised for regular follow up.

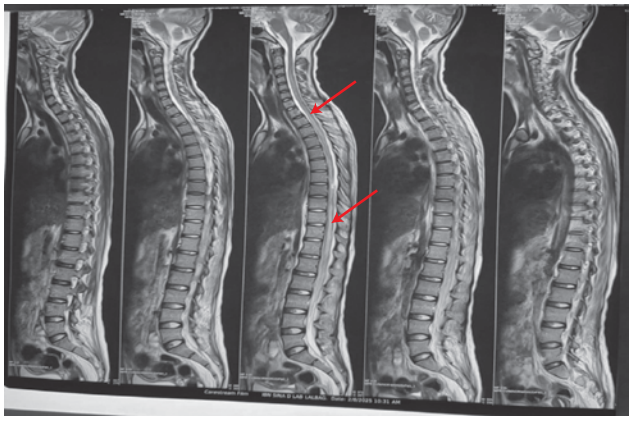

Fig.1 : MRI of Spine with contrast shows T2 hyperintensity (red arrow) in the spinal cord from C3-D12 level

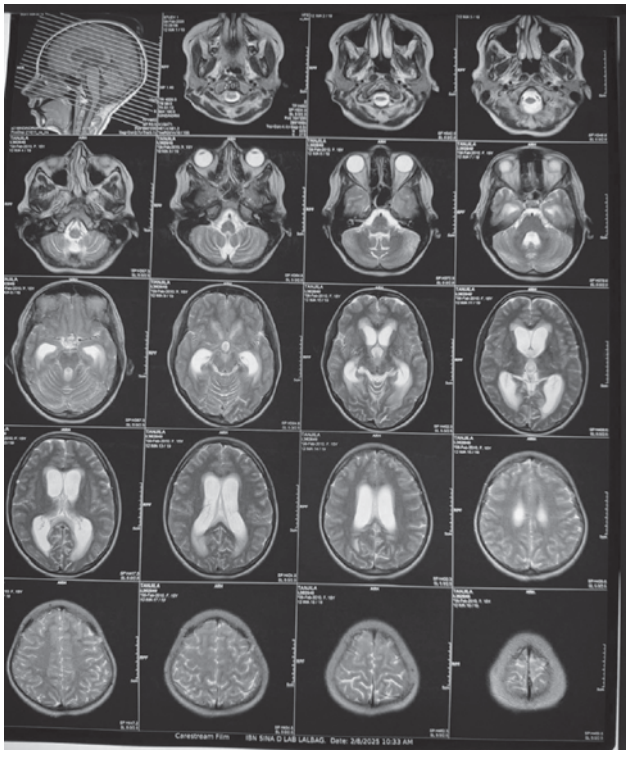

Fig.2 : T2 sequence of MRI Brain shows lateral & third ventricles are mildly dilated, 4th ventricle appears normal

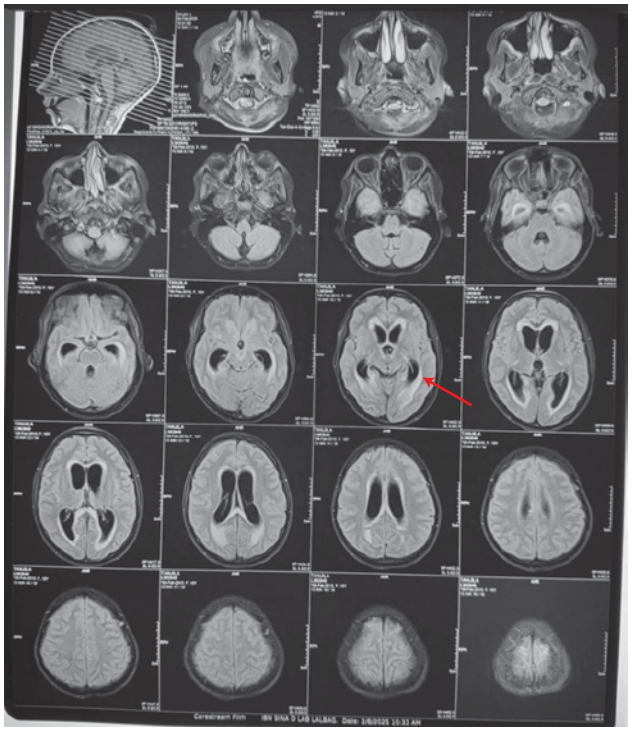

Fig.3 : Periventricular diffuse T2-FLAIR hyperintensity (red arrow) in MRI Brain suggests chronic periventricular ischemic change.

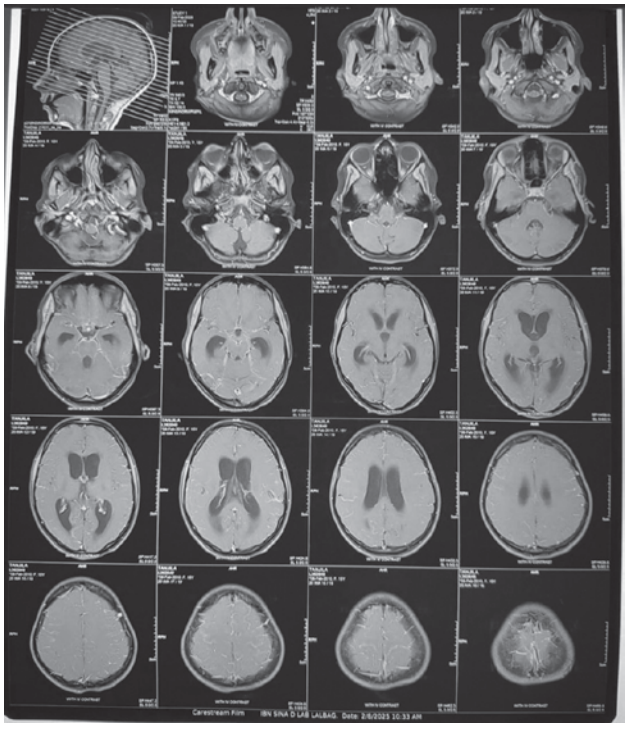

Fig.4 : T1 sequence with contrast MRI shows no focal lesion in Brain

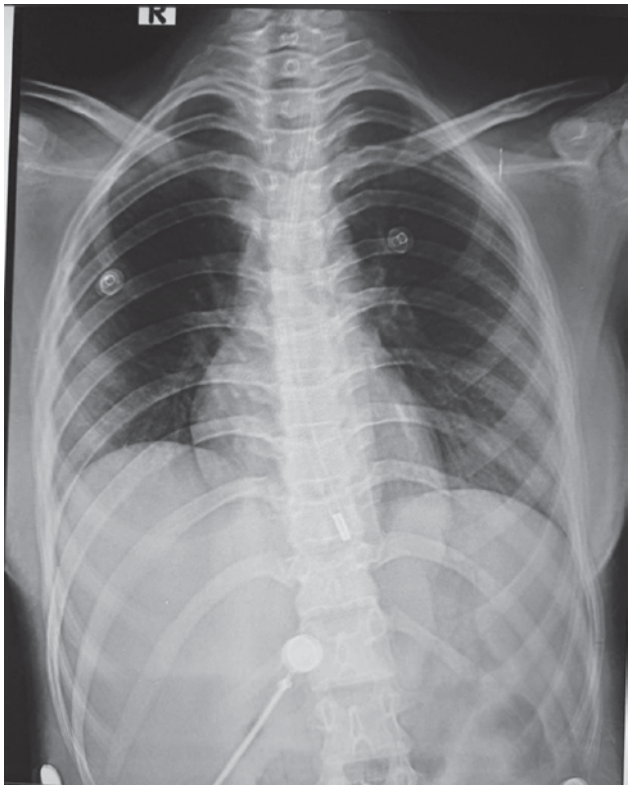

Fig. 5: Xray of chest P/A view is normal. There are shadows of multiple wires

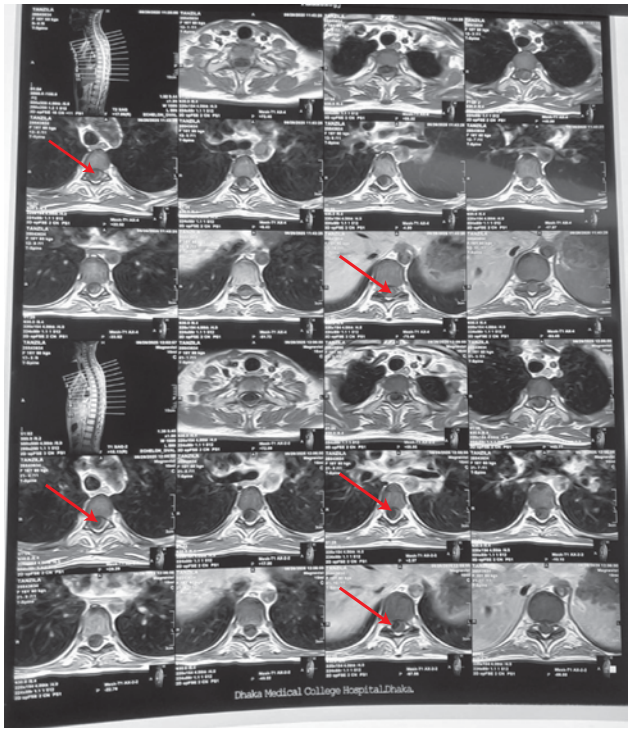

Fig. 6a

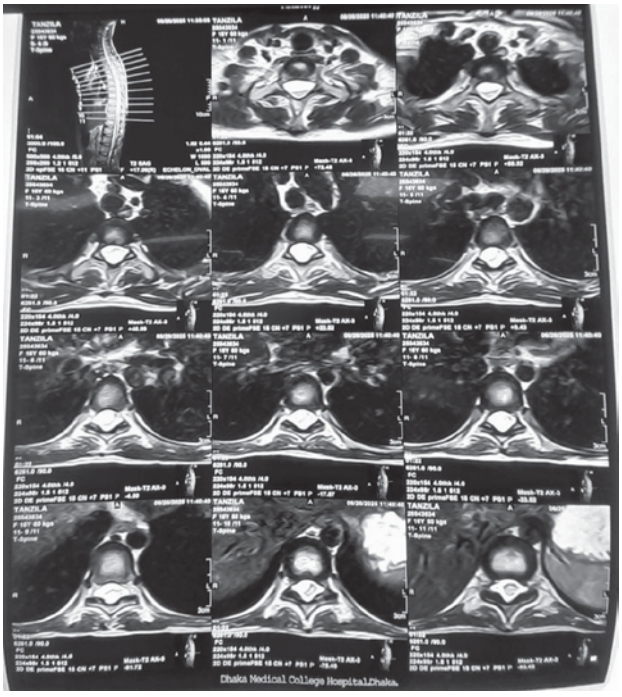

Fig. 6b

Fig. 6a & 6b : shows spinal arachnoid cyst with myelomalacic change evidenced by a well defined extramedullary cystic area (red arrow) at right side of the spinal cord which is T1WI- hypointense, T2WI- hyperintense, after intravenous contrast no enhancement is seen. This lesion compress & displace the spinal cord towards contralateral side extending from the visualised part of the cervical segment to D10 vertebral level.

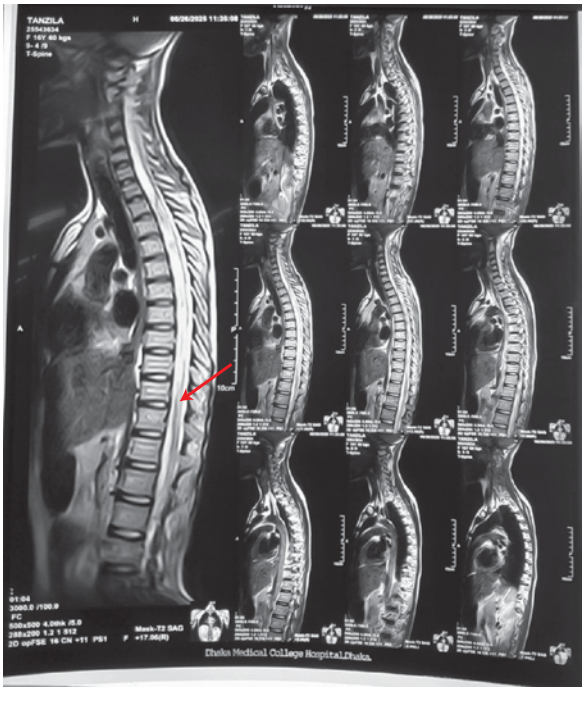

Fig. 7: Follow up MRI of spine with contrast after 5 months shows long segment T2- hyperintense area (red arrow) extending from D2 – D10 segment of spinal cord

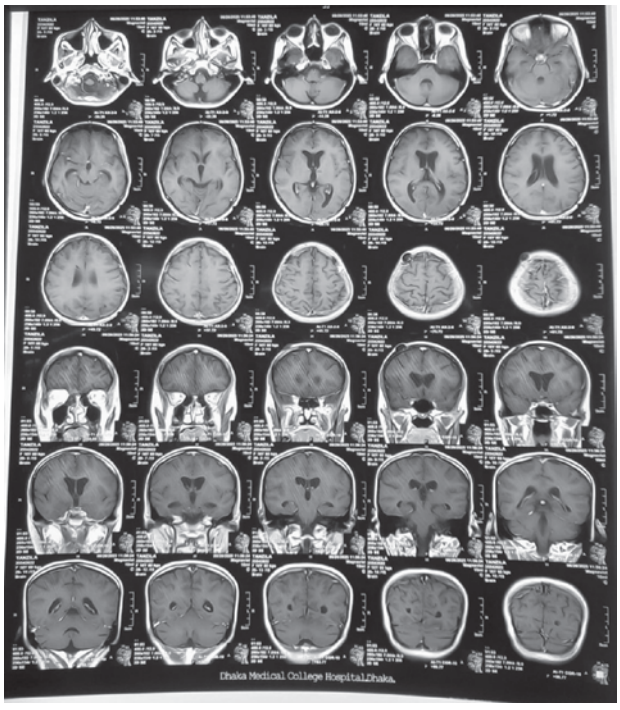

Fig 8 : Mild symmetrical enlargement of all ventricles suggests resolving hydrocephalus

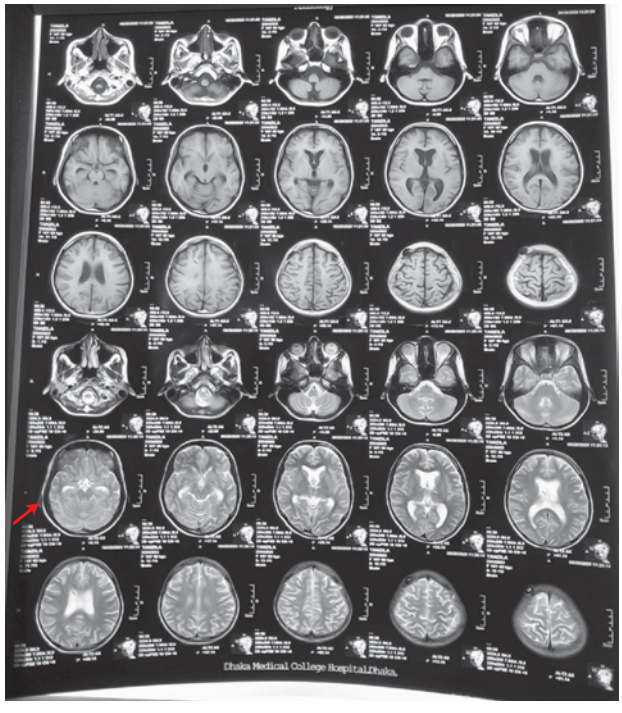

Fig.9 : T1W1- hypointense, T2W1- hyperintense area measuring about (1.7 × 0.8) cm is noted outside scalp at right sided high parietal region suggestive of Ommaya reservoir (red arrow)

Discussion

TM is defined as the inflammation of the spinal cord extending over 1 or more vertebral segments in the absence of a compressive lesion[6]. It is a type of Central Nervous System Tuberculosis (CNS TB) which accounts for ~ 1% of all TB cases worldwide[7]. Tuberculous transverse myelitis is extremely rare. It may occur in isolation or in association with adjacent TB meningitis[7]. No large cohort prevalence is established for pure TB-associated TM in the literature; mostly individual case reports or small series[8]. In a prospective study of 71 patients with tuberculous meningitis (TBM), spinal cord and nerve root involvement were seen in 46.4% of subjects[9]. Myelitis specifically was documented in 16 patients (22.5%) in that cohort, although not all were classic TM but part of broader myeloradiculopathy[9]. In TM, patients present with acute onset of bilateral sensory and motor deficits and sphincter dysfunction[6]. The lesions typically occupy more than two-thirds of the cross-sectional area of the cord[10]. When it involves three or more contiguous segments of spinal cord, it is termed as longitudinally extensive transverse myelitis (LETM)[4]. TB-associated LETM is very rare; published ones are mostly case reports and small series. A systematic retrospective analysis of 147 TBM patients with spinal complications found ~8.84% with acute TM or LETM[8]. A clinicoradiological review found ~10 cases in 7 publications describing TB myelopathy with longitudinally extensive lesions[8]. A recent review identified ~14 reported cases of TB-LETM over the past decade [11].

Etiologically TM is categorized into either idiopathic or disease-associated TM. Secondary TM can be due to infection (viral, bacterial, fungal, or parasitic), systemic autoimmune diseases (eg. Systemic lupus erythematosus [SLE], Sjögren's syndrome, etc.) or CNS demyelinating disease (eg. Multiple sclerosis [MS], Neuromyelitis optica spectrum disorder [NMOSD], Myelin oligodendrocyte glycoprotein antibody disorders [MO- GAD], Acute disseminated encephalomyelitis [ADEM], etc.)[12]. LETM, although classically associated with NMOSD, can have multiple other etiologies including MS, MOGAD, ADEM, autoimmune gliofibrillary acidic protein (GFAP) astrocytopathy, systemic autoimmune illness (SLE, Sjögren's syndrome, etc.), infectious, and vascular causes[13, 14]. Initial imaging modality for suspected transverse myelitis is MRI of the spine.

MRI of brain is vital for the determination of type of TM - whether it is due to CNS demyelinating illness or is an isolated condition[15]. CSF analysis has a crucial role in differentiating the infectious causes or presence of any oligoclonal band in suspected MS[16]. Our patient did not exhibit any signs of autoimmune disease; neither did she met the Wingerchuk revised diagnostic criteria for definite NMOSD[17] which is stratified further by serologic testing (NMOSD with or without AQP4-IgG). In our patient viral, autoimmune and syphilitic causes were also ruled out during investigations.

In her case, tuberculous aetiology was confirmed in the context of classical symptoms of tuberculosis, radiological findings of hydrocephalus, LETM, and hyponatremia due to SIADH in addition to a classical CSF picture of TBM with lymphocytic pleocytosis, raised protein levels, and low glucose & most importantly positive Gene Xpert MTB report. The patient showed a good clinical and radiological response

to ATT. Follow up MRI of spine shows spinal arachnoid cyst which is also in favour of spinal TB.

Multiple mechanisms have been proposed for the development of myelitis in tuberculosis. Immune mediated processes are considered the predominant mechanism in tuberculous myelitis. Although in the present case the likely cause was the direct bacterial invasion associated with concurrent tuberculous meningitis. Other proposed mechanism is direct vascular thrombosis leading to tuberculous myelitis. Recovery from tuberculous transverse myelitis may be absent, partial or complete. It largely depends on how early treatment is initiated.

Our case had significant improvement of symptoms following treatment leaving mild residual weakness. The endpoint is that clinicians have to consider tuberculous transverse myelitis in patients with compatible clinical & imaging findings in TB-endemic regions especially when associated with meningitis.

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