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CNS Tuberculosis with Stormy Presentation – treatment dilemmas in clinical practice – A Case Series

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Abstract

Tuberculosis of the nervous system has heterogenous manifestations depending on the structure of the nervous system involved – brain, meninges, spinal cord, nerve roots or peripeheral nerves. Diagnosis of this can be challenging or confusing in view of its numerous differential diagnosis and slow response to treatment. Diagnostic dilemmas could lead to delay in initiating appropriate therapy which can lead to fatal outcomes.

Herein we discuss four patients with distinct presenta tions who were eventually diagnosed with tuberculosis and initiated on anti-tuberculous therapy (ATT). The first patient presented with fever, headache followed by ataxia, the second with psychiatric symptoms, the third with neurogenic claudication and the fourth with rapidly progressive quadriparesis. All patients improved after initiation of appropriate ATT following various diag nostic and therapeutic hurdles. The first patient had resistant tuberculosis which responded to a modified ATT regimen.

We discuss a few of the commonly encountered diag nostic and therapeutic decision-making conundrums, in the setting of tuberculoma and spinal tuberculosis.

Keywords: Central Nervous System Tuberculosis, Tuberculoma, spondylodiscitis, PCR.

Introduction

The incidence of TB in India in 2016 was 212/ 100, 000, of which approximately 10% is TB of the Central Nervous System (CNS). Despite adequate ATT therapy, mortality continues to hover around 20% with disabling neurological sequelae in a third of patients who survive. [1-3] There is no clear-cut guideline regarding the optimal ATT regimen and duration of therapy for CNS TB and hence therapy in many clinical situations remains a 'Pandora's box'. In this article, we intend to highlight the varied clinical presentation of CNS TB and the

therapeutic dilemmas encountered during the course of management.

Case 1

An 18-year-old female student was referred to our institute with a history of fever and headache for 15 days, diplopia and ataxia for 2 days. She had received a course of anti-biotics including ceftriaxone and acyclovir for 7 days following an LP-CSF which was acellular with a mild elevation of CSF Protein. An MRI done prior to admission showed multiple subcortical T2 hyperintensities without diffusion restriction. (Fig 1)

On examination, she had bilateral Lateral Rectus (LR) Palsy, Cerebellar Signs and Neck stiffness. A repeat CSF analysis was performed which showed 1300 Cells, Predominantly Lymphocytes, Glucose-20mg/dl, Protein-95mg/dl and globulin-Positive. CSF CBNAAT was negative. ANA and ENA Profile were negative. RT-PCR for COVID-19 was negative. She was started on dexame thasone, Ceftriaxone, ATT (Isoniazid (H), Rifampicin (R), Pyrazinamide (Z) and ethambutol (E)) with parent eral Steptomycin and Levofloxacin in consultation with Chest Physician. As patient persisted to have symptoms even after 2 weeks an MRI was repeated which showed Increase in the number of Lesions with Intense Leptomeningeal Enhancement. (Fig 2)

Patient was continued on 4 drugs daily ATT (HRZE) for 3 months. Patient did not show any new clinical symptoms and was switched over to continuation phase with 3 drugs daily (HRE) which was given for the next 7 months. An MRI done after completion of 10 months showed multiple ring enhancing lesions in Left Hemi pons, Middle cerebellar peduncle, Right Temporal, Right Insula and Left precentral gyrus with the largest measuring 1.65 x 1.24cm in Left Hemi pons with significant edema. (Fig 3)

Patient was restarted on first line daily ATT(HRZE) and steroids. One month later patient presented with numb ness along ophthalmic division of trigeminal nerve. A repeat MRI was taken which showed significant increase in size of lesions largest measuring 4.7 x 3.2 x 2.8cm in the Left Hemi pons extending into the Lt Cerebellum. In view of significant increase in size of lesions, patient was advised to undergo a stereotactic biopsy at a referral Centre for a pathological diagnosis, but patient refused. Possibility of MDR-TB was considered and hence patient was started on modified ATT (HRZE, Levo floxacin 1gm, Strep to mycin 750mg, Linezolid 600mg). Patient was also given Albendazole for 3 weeks. Two months later she presented with weakness of right upper and lower limb of 1 week duration. MRI showed decrease in the size of lesions with the largest in Left Hemi pons measuring 1.7 x 1.5cm with significant Vaso genic edema in Left Basal ganglia and Thalamus. Patient was given parenteral Dexamethasone for 1 week, Pyrazinamide and ethambutol were stopped, and patient was discharged on Rifampicin 600 mg OD, Isoniazid 300 mg, Linezolid 600mg and Levofloxacin 1gm. Opinion was sought from an expert on infectious disease, and patient was advised to continue on the same anti-biotics. Steroids were slowly tapered over the next 3 months. Patient continued to maintain clinical improve Ment and MRI done 6 months later showed further reduction in size of lesions. (Largest measuring 1.2 x 1.2 x 1.3cm). Patient is on regular follow-up and has been counselled regarding the possible need for anti-tuberculous drugs for 18-24months.

Case 2

A 48-year-old male, diabetic, on treatment for pulmonary tuber culosis, was referred from a psychiatrist for evaluation of delirium and formed visual halluci

nations since 4 days. On examination he was not oriented to time, place or person, was agitated and had poor attention span. He had bilateral 6th Cranial Nerve Palsy and bilateral cerebellar signs. Terminal Neck stiffness was present.

His MRI (Figure 4) showed Multiple FLAIR hyper intense contrast enhancing foci noted in the subcortical regions of bilateral frontal, parietal, temporal, occipital lobes, pons and bilateral cerebellar hemi spheres with Intense Lepto meningeal enhancement. CSF analysis showed elevated Protein (132mg/ dl), no inflammatory cells and CSF GeneXpert PCR positive for M. tb with indeterminate Rifampicin resistance. Patient was started on 4drug daily regimen of standard weight-based ATT (HRZE). He had a paradoxical reaction 2 months after starting ATT which was managed with steroids. Patient was treated with 4 drugs(HRZE) for 2 months followed by 3 drugs (HRE) for 9 months following which he had clinical and radiological resolution.

Case 3

A 42-year-old Male, on irregular treatment for Diabetes Mellitus since 4 years presented with 2 months history of progressively worsening low back ache with neuro genic claudication of right lower limb. On examination, he had normal power in both his lower limbs with preserved deep tendon reflexes. CT of lumbar spine was done which showed altered density involving L4 & L5 vertebral bodies with reduced disc height and end plate irregularity at L4-L5 disc level suggestive of infective spondylodiscitis. A hypodense collection was seen along posterior aspect of right psoas muscle, extending from L2 up to ilio-psoas level with surrounding fat stranding suggestive of abscess. MRI confirmed the diagnosis of iliopsoas abscess extending from L1 vertebra to the right inguinal ligament (Figure 5)

Patient was treated as a case of Clinically diagnosed TB and was started on 4 drug daily regimen with HRZE with adequate analgesics and strict control of blood sugars with Insulin. Patient showed a steady and gradual improvement one month after starting ATT and was continued on HRZE for 2 months followed by 3 drugs (HRE) for the next 7 months. Patient showed complete clinical and radiological recovery.

Case 4

A 40-year-old lady presented with history of neck pain with shock like sensation running down the spine for 1 month, followed by rapidly progressive quadriparesis for 5 days. She was bed bound for 1 day. MRI of the Cervical spine was suggestive of infective spondylitis with epidural and perivertenral collection extending from CV Junction to mid C3 levels with significant cord compression (Figure 6).

HRCT thorax did not show any significant abnormality. A tuberculin skin test was strongly positive. With a clinical diagnosis of spinal tuberculosis, patient was initiated on 4 drug daily regimen (HRZE) and steroids. Patient showed rapid clinical improvement within 2 weeks of initiating ATT and steroids. A spine surgery consult was obtained and patient was continued on ATT. Patient is currently able to walk without support and able to perform her activities of daily living independently and is on close follow-up, having completed 2 months of HRZE, and currently on HRE.

Discussion

The pattern of neuroimaging abnormalities along with the clinical setting gives a fair idea to the physician with regards to the diagnosis. At times, the lack of a definitive microbiological evidence remains to be an Achilles heel, and leads to delay in initiation of treatment. This also causes problems in early identi fication of resistant

organisms and thus leading onto further diagnostic and the rapeutic dilemmas.

Clinical features of Tuberculomas could vary based on the size and location of the lesions. In the first case, the patient presented with bilateral LR Palsy (a sign of raised Intra-cranial Pressure (ICP), a false localizing sign) and Cerebellar signs. Later on, she developed Trigeminal sensory loss followed hemiparesis. In Case 2, the patient presented to a psychiatric Centre with delirium and visual hallucinations. These manifestations highlight the spectrum of clinical manifestations of CNS tuberculomas ranging from seizures, features of raised ICP and focal deficits to a purely psychiatric mani festation.

The first case is a classic example of the difficulties faced by the clinician during diagnosis and the vagaries during treatment. Tuber culomas are 'encased granu Lomas' arising due to Hema to genous spread of tuber culous bacilli, because of which detection of the Myco bacterial Antigens in Serum or CSF has very low sensitivity. [7] This is unlike in TBM, wherein rupture of the Rich focus into the subarachnoid space leads to symptoms of meningitis. Thus in tuberculomas, when associated with TBM, CSF leucocytosis and lympho cytosis are still the best predictors of CNS TB with a low CSF glucose and elevated CSF Protein being supportive. [8] Specific tests such as polymerase chain reaction (PCR), [9] QuantiFERON [10] and Gene Xpert [11], though being rapid and able to detect drug resistance, are sensitive only in situations in which the Serum/CSF would otherwise show a culture positivity, as seen in the second case. The difference in patho physiology of tuberculomas versus TBM also explains the lower PCR positivity rates in Tuberculomas. (Sensitivity of PCR in tuber culomas– 30%, Specificity – 100%, Sensitivity of PCR in TBM – 32 to 40%, Specificity – 92%, Positive Predictive Value – 85%) [12,13].

Certain neuroimaging features of Space Occupying Lesions (SOL) are said to favour a diagnosis of Tuberculoma versus Neuro cysticercosis (NCC), which is the commonest cause of a solitary ring enhancing lesion in patients with focal seizures in developing countries like India.[14-16]Size more than 20mm, irregular margins and presence of midline shift are said to favour a tuber culoma, [17-18] whereas, presence of eccentric calcification, size less than 20mm with mild to moderate peri lesional edema which usually does not result in mid line shift, with an enhancement pattern which differs with the stage of cysticerci favour a diagnosis of NCC.(Table 1)[19-22] These features, though suggestive, are not diagnostic. Magnetic Resonance Spectroscopy (MRS) in these situations, is an invaluable tool can distinguish between the two. The presence of a lipid peak on MRS in a patient with a ring-enhancing lesion, is virtually diagnostic of tuberculoma, as opposed to an elevated lactate, alanine, succinate, glutamate and glycine with a reduction in NAA in NCC. [16,20, 23-27]

Though MRS provides indirect evidence in the diagnosis of tuber culoma, a thorough search for extra neural tuber culosis has to be done. Sputum examination, Mantoux test, Ultrasonography of the abdomen, Whole body CT followed by CT guided aspiration/biopsy may be required for the diagnosis. [7] CT/ MRI guided Stereo tactic biopsy with his to patho logical confir mation remains the gold standard for the diagnosis of tuber culoma.

The diagnosis of multidrug-resistant TB (MDR-TB)/ extensively drug-resistant TB (XDR-TB) /Totally drug-resistant TB is extremely difficult in the clinical setting of tuber culoma with a negative Serum / CSF Culture /

PCR. This has numerous clinical implications as current Indian guidelines for treatment of drug-resistant TB, do not allow starting MDR-TB/XDR-TB regimens without definite microbiological evidence of drug resistance. It is in these cases that a stereotactic biopsy is extremely useful especially if the lesions are superficial and easily amenable for biopsy.

(Table 2, Figure 7) In Case 1, a modified regimen with First line ATT drugs (Rifam picin, pyrazinamide, isoniazid and ethambutol) along with Linezolid, Streptomycin and Levofloxacin was initiated after consultation with the Chairperson of the District Resistant TB Committee, as patient had rapidly worsening symptoms as per current guide lines for management of drug resistant tuberculosis. [28]

Other rare presentations of CNS tuberculosis include tubercular spondylodisctis (TS) (Case 3) and Spinal epidural abscess (Case 4). TSis thought to result from hematogenous spread from a primary lung focus (arterial seeding) or from lymphatic spread from contiguous Pleura. [29]

The former is the more likely mechanism in Lumbar TS, whereas, the latter, is the more likely mechanism in thoracic TS. Thoracic TS is more common and accounts for approximately 45-57% of cases with TS, whereas Lumbar TS is rare and accounts for around 20-32% of cases. [30,31] They are frequently complicated by paraspinal abscess in 20 to 90% of cases, [32-35] and can include local soft tissue infection, as seen as a Psoas Abscess in Case 3.

These commonly present with symptoms ranging from neuro genic claudication or radiculopathy to paraplegia/ quadriplegia secondary to compressive myelo pathy. ATT has to be initiated early with or without steroids. Surgery may be required in the presence of neuro logical deficits, deformity of the spine, instability of the spine, spinal cord compression and when there is little or no response to ATT.TS involving the Cervical Spine complicated by an epidural abscess (Case 4) is even rare and is seen in less than 5% of cases. [32] Early identification and immediate institution of ap propriate therapy including ATT, steroids and Surgery is needed to prevent major disability.

Conclusion

Clinical presentation of tuberculosis of the nervous system, particularly tuberculomas is varied and covers a wide spectrum ranging from an apparently innocuous chronic headache, psychiatric mani festations to seizures, focal neuro logical deficits and features of raised Intracranial pressure.

The diagnosis and treatment of these can be challenging owing to difficulties in obtaining micro bio logical evidence and in demonstration of drug resistance.

The decision of performing a stereo tactic biopsy in cases which are suspicious of drug-resistant TB has to be done without delay to prevent disastrous consequences. Other rare presentations like spondylo discitis with abscesses in volving unusual locations should prompt the physician to consider TB as a part of differential diagnosis and surgery/ ATT should be instituted without undue delay.

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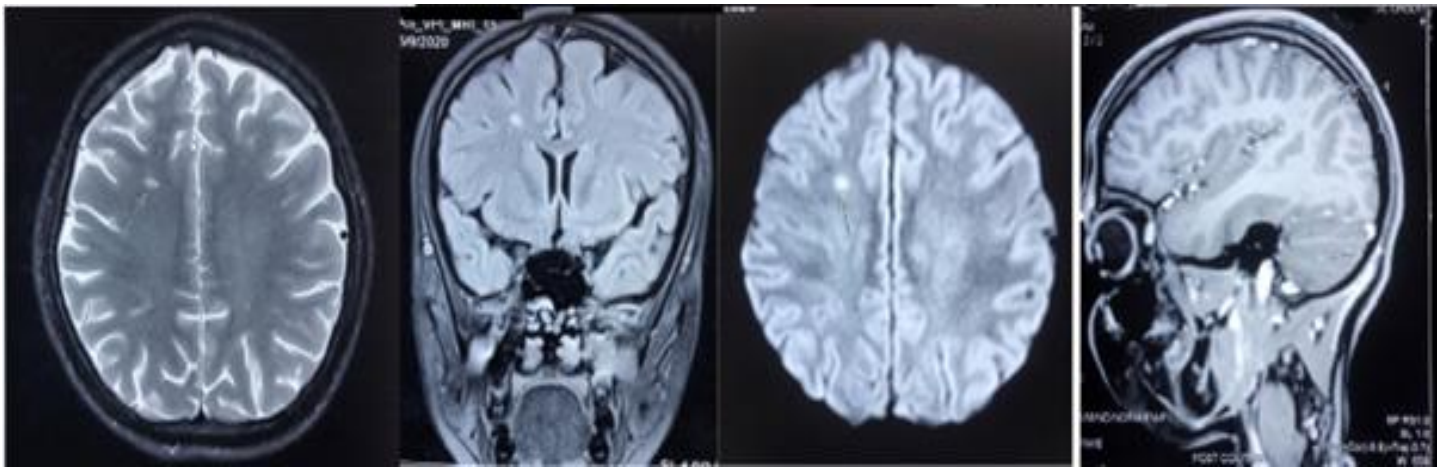

Figure 1: T2, FLAIR, diffusion and T1-C images showing T2 and FLAIR hypertintense foci with no diffusion restriction noted in frontal subcortical region and bilateral centrum semiovale with contrast enhancement of the cerebellar lesion

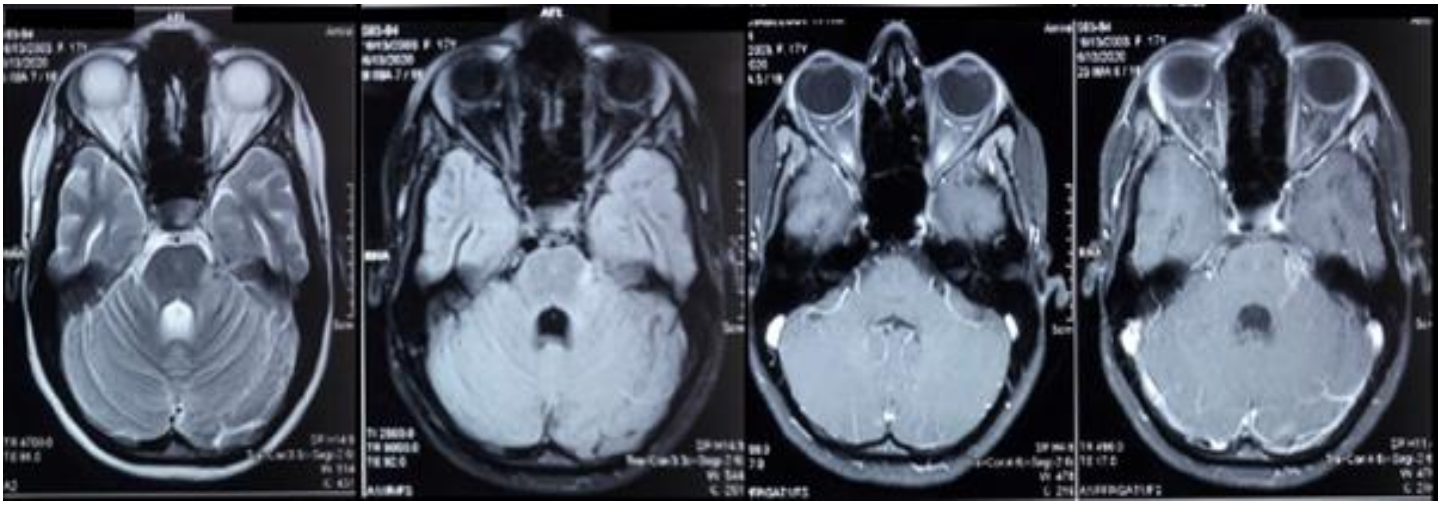

Figure 2: T2 and Flair hyperintensity seen in left ambient cistern and adjoining pons with diffusion restriction. Contrast Enhancement seen in left ambient cistern and left inferior cerebellar hemisphere and leptomeningeal enhancement

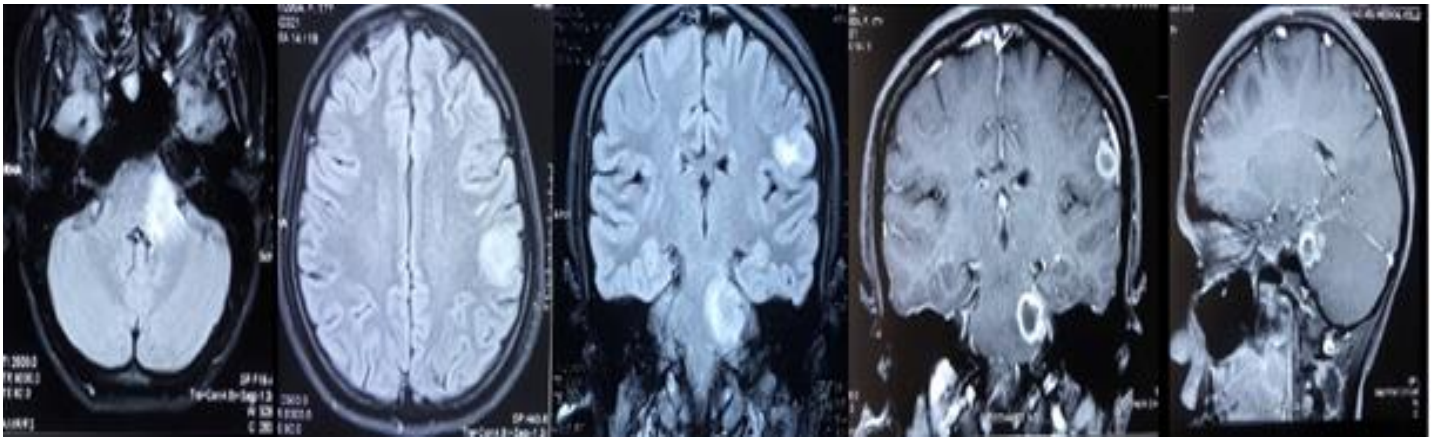

Figure 3: MRI showing multiple ring enhancing lesions in Left Hemi pons, Middle cerebellar peduncle, Rt Temporal, Rt Insula and Lt precentral gyrus with the largest in Lt Hemi pons with significant edema.

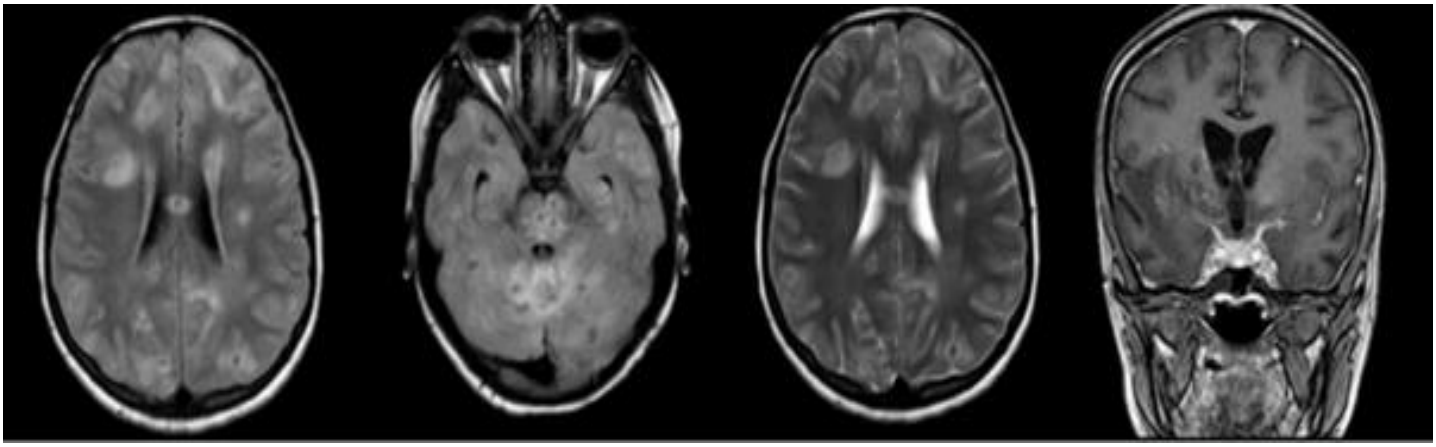

Figure 4: Multiple FLAIR hyperintense contrast enhancing foci noted in the subcortical regions of bilateral frontal, parietal, temporal, occipital lobes, pons and bilateral cerebellar hemispheres. Intense Leptomeningeal enhancement is also seen in the post contrast image.

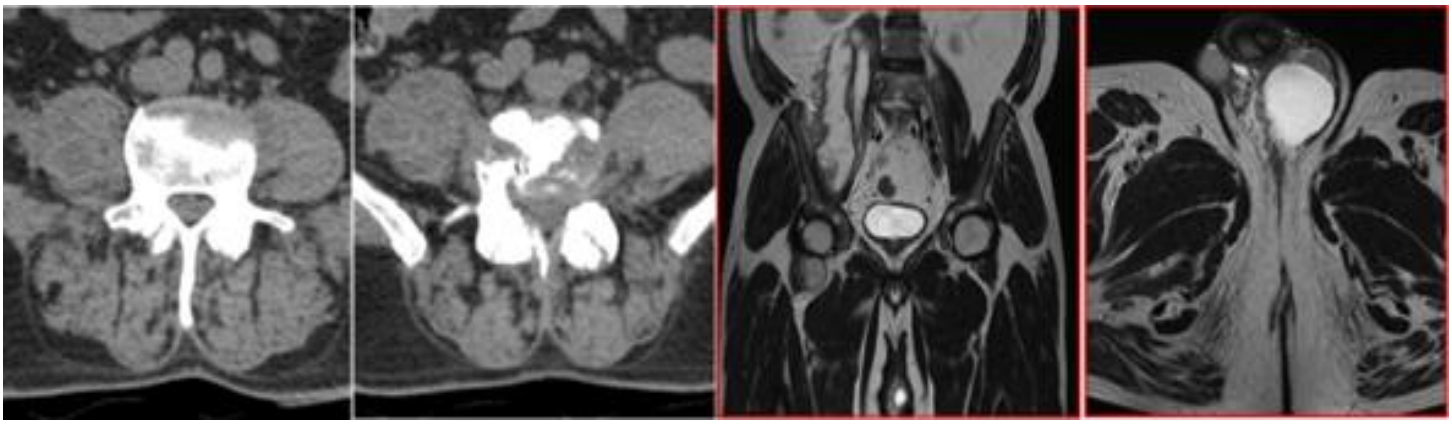

Figure 5: CT Image of lumbar spine showing altered density involving L4 & L5 vertebral bodies with reduced disc height and end plate irregularity at L4-L5 disc level. MRI of lumbosacral plexus showing Large iliopsoas abscess seen from L1 vertebra down to the lesser trochanter extending deep to the right inguinal ligament~ 5.4/6.2/26.8 cm (AP/transverse/CC). Thin internal septations noted in the abscess

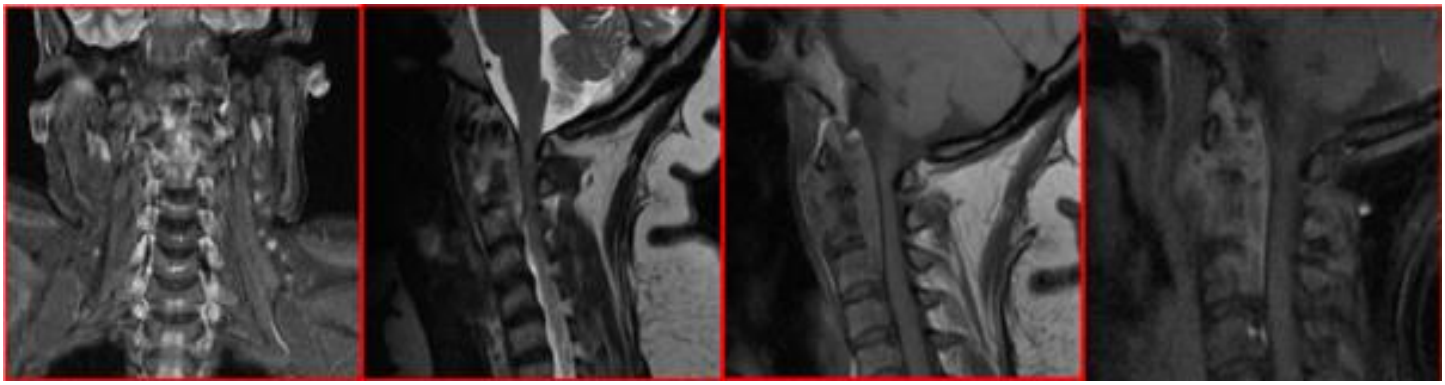

Figure 6: MRI of the Cervical spine showing Ill-defined moderately T2/T2 STIR non-homogenously hyperintense signals noted involving odontoid process and body of C2 vertebra, postero-superior aspect of C3 vertebra with adjacent epidural collection from CV junction to mid C3 level causing mild compression of the spinal cord.

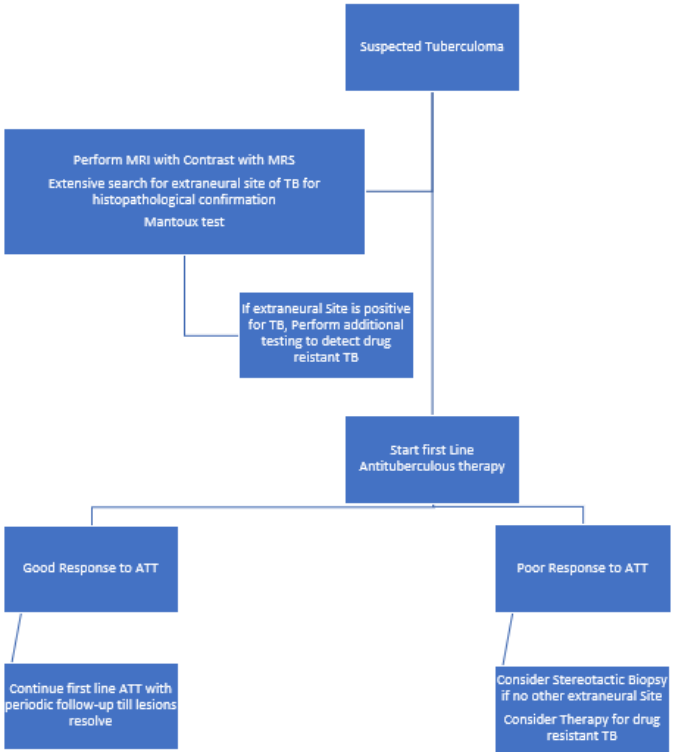

Figure 7: Management algorithm for tuberculomas.

	Tuberculoma	Neurocysticercosis
Number	Single or most often multiple, conglome ration is seen	Single or multiple
Size	>20mm	<20mm
Margins	Irregular Enhancement on contrast	Smooth enhancement on contrast
T2	Hyperintense	Hypointense scolex may be seen
Location	Posterior fossa common, grey white matter junction	Grey white matter junction
MRS	Lactate peak is seen	elevated lactate, alanine, succinate, glutamate and glycine with a reduction in NAA

Table 1: Differences between Tuber culoma and Neuro cysticercosis on neuroimaging

Indications of stereotactic biopsy in suspected cases of tuberculoma
Paradoxical increase in size of lesions in spite of adequately dosed first line Anti tuberculous therapy
Inability to demonstrate Tuberculous bacilli at extra neural sites despite extensive investigations
Suspicion of drug resistant tuberculosis
Rapidly progressive lesions with impending herniation

Table 2: Indications of stereotactic biopsy in suspected cases of tuberculoma