A RARE CASE OF GENERALIZED TUBERCULOSIS IN 29yr OLD FEMALE INCLUDING MULTIPLE TUBERCULOMA WITH TUBERCULOUS ARTERITIS WITH TUBERCULAR MENINGITIS ASSOCIATED WITH ACUTE CEREBELLAR INFARCTION AND HYPERSENSITIVITY REACTION TO THE FIRST LINE ANTI TUBERCULAR TREATMENT IN POST PARTUM PERIOD

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INTRODUCTION

TB of the central nervous system (CNS) accounts for 5.9% of extra-pulmonary TB most often seen in young children but also develops in adults, especially those infected with HIV[1]. Tuberculous meningitis results from the hematogenous spread of primary or post primary pulmonary TB or from the rupture of a subependymal tubercle into the subarachnoid space. In more than half of cases, evidence of old pulmonary lesions or a miliary pattern is found on CXR. But in this case we found no evidence of Pulmonary lesions in CXRAY.

The disease often presents subtly as headache and slight mental changes after a prodrome of weeks of low-grade fever, malaise, anorexia, and irritability. If not recognized, tuberculous meningitis may evolve acutely with severe headache, confusion, lethargy, altered sensorium, and neck rigidity. The disease evolves over 1–2 weeks. Because meningeal involvement is pronounced at the base of the brain, paresis of cranial nerves (ocular nerves in particular) is a frequent f inding, and the involvement of cerebral arteries may produce focal ischemia. The ultimate evolution is toward coma, with hydrocephalus and intracranial hypertension.

Lumbar puncture is the cornerstone of diagnosis. Examination of cerebrospinal fluid (CSF) reveals a high leukocyte count (up to 1000/μL), usually with a predominance of lymphocytes but some- times with a predominance of neutrophils in the early stage; a protein content of 1–8 g/L (100–800 mg/dL); and a low glucose concentration[2]. Culture of CSF is diagnostic in up to 80% of cases and remains the gold standard. Real- time automated nucleic acid amplification (the Xpert MTB/RIF assay) has a sensitivity of up to 80% and is the preferred initial diagnostic option. Treatment should be initiated immediately upon a positive Xpert MTB/RIF result. A negative result does not exclude a diagnosis of TB and requires further diagnostic workup.

Imaging studies (CT and MRI) may show hydrocephalus and abnormal enhancement of basal cisterns or ependyma. If unrecognized, tuberculous meningitis is uniformly fatal. Clinical trials have demon- strated that patients given adjunctive glucocorticoids may experience faster resolution of CSF abnormalities and elevated CSF pressure, resulting inlower rates of death or severe disability and relapse. In one study, adjunctive dexamethasone significantly enhanced the chances of survival among persons >14 years of age but did not reduce the frequency of neurologic sequelae. The dexamethasone schedule was (1) 0.4 mg/kg per day given IV with tapering by 0.1 mg/kg per week until the fourth week, when 0.1 mg/kg per day was administered; fol- lowed by (2) 4 mg/d given by mouth with tapering by 1 mg per week until the fourth week, when 1 mg/d was administered. The WHO now recommends that adjuvant glucocorticoid therapy with either dexamethasone or prednisolone, tapered over 6–8 weeks, should be used in CNS TB.

Tuberculoma, an uncommon manifestation of TB of the CNS, presents as one or more spaceoccupying lesions and usually causes seizures and focal signs. CT or MRI reveals contrastenhanced ring lesions, but biopsy is necessary to establish the diagnosis.
[3]

CASE REPORT
A 29 yr old post partum day 8 female presented at Emergency dept of C.U. Shah Medical College on 11pm 17-2-24 with history of fever since 1 week, which was low grade and intermittent with chills and rigors ; c/o severe pulsatile generalised headache since 1 week; c/o left sided upper and lower limb weakness since 3 AM on 18-2-24 n/a/w slurring of speech of speech.

PAST HISTORY
H/O IVF 9 months ago
H/O preterm delivery 3 years back
C Section done, neonate died in 12 days
H/O one unit PCV

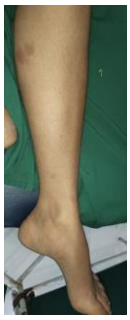
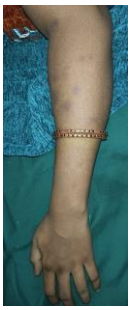
ON PRESENTING VITALS
P 114/ MIN
BP 110/70
RS BLAE CLEAR
CVS S1S2
CNS —PUPIL BL DILATED NRTL
Power. R L
5/5 0/5
5/5 0/5

TONE R. L
N. /DECREASED
N / DECREASED

PLANTAR R L
EXTENSOR/ EXTENSOR

SPO2 95% RA

INVESTIGATIONS
- ALTERED ESR 41
- LIPID PROFILE
- CHOL 297
- TG 191
- HDL 49.6
- LDL 212.4
- ALP 115.40
- TP 5.36
- ALB 1.70
- GLB 3.66
- A:G 0.46
- CSF REPORTS TOTAL. CELLS 120 POLYMORPH 5% LYMP 95% PROTEIN 239.3 GLUCOSE19 ADA 11.8

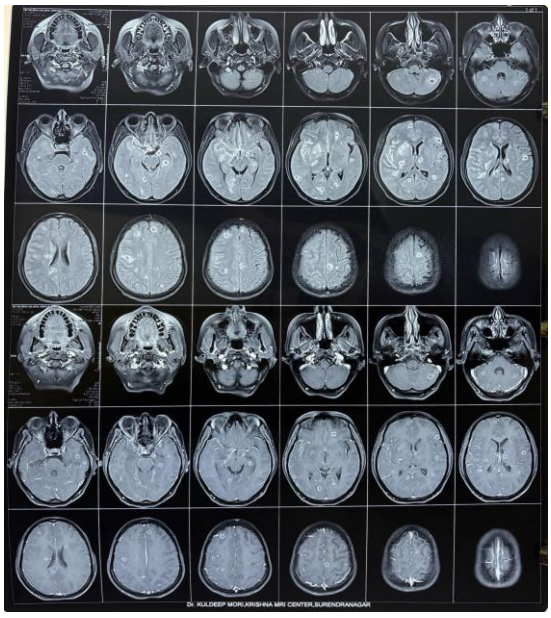
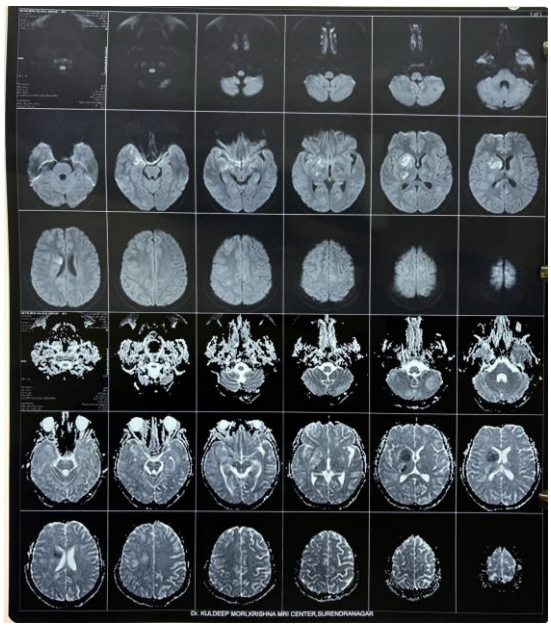



DRUG INDUCED FIXED DRUG ERUPTION by Isoniazid, Rifampicin, PAS

X-RAY CHEST

MULTIPLE TINY NODULAR RADIO OPACITIES IN BL LUNG FIELDS

MRI BRAIN

Changes of mild diffuse leptomeningitis with enchancing tuberculomas & mild perifocal edema seen involving bilateral fronto- temporal-parietal-occipital lobe, bilateral cerebellar hemisphere, bilateral thalamocapsular areas suggesting tuberculous acute infarcts involving right sided basal ganglia secondary to vasospasm.

TREATMENT GIVEN

- INJ DEXONA 8 MG IV. TDS 10 DAYS F/B 8MG BD 10 DAYS

- F/B 8 MG OD 10 DAYS
- F/B 4MG OD 10 DAYS
- F/B 2 MG OD 10 DAYS
- INJ STREPTOMYCIN 750 IM OD
- INJ LEVOFLOX 500+ NS 100 IV BD
- T PYRAZINAMIDE 750 1-0-1
- T ETHAMBUTOL 1000 1–0-0
- C ECOSPRIN AV (150/20) 0-0-1
- INJ NS 500 + 1@ELDERVIT IV @ 80ML/HR
- INJ RANTAC 150 IV
- INJ PERI 4 IV
- INJ FEBRINYL 3CC IV SOS
- T FDSON MP FORTE 0-1-0
- SYP SOFTKUL 15ML HS
- WEB PRO DHA PROTIEN POWDER WITH MILK TDS

International guidelines recommend treating tuberculomas with standard ATT for CNS-TB for 9-12 months.[4][5] Clinical improvement and radiological clearance are observed in 90% and 80% of patients, respectively, by nine months. Complete resolution depends upon the initial size; those <2.5 cm resolve in 5–8 months, while 50% of >2.5 cm take over 12 months to disappear.[6]
Additionally, every patient should receive adjunctive steroids as per guidelines, especially unequivocally for those that develop paradoxical responses.Surgical intervention is indicated in patients who show no response to medical management or have raised intracranial pressure.

DIFFERENTIAL DIAGNOSIS[6]

- Partially treated bacterial meningitis
- Cryptococcal meningitis
- Viral meningoencephalitis
- Carcinomatous meningitis
- Neurobrucellosis,
- Neurosyphilis,
- Neuroborreliosis
- Neurosarcoidosis
- Parameningeal infection
- Neurosarcoidosis
- Neurosyphilis
- Nuerological Tuberculosis
- Neurocysticercosis,
- Cryptococcoma,
- CNS lymphoma

DISCUSSION

- The original description by Rich and McCordock suggested that tuberculous lesions develop in the brain during the stage of bacteremia.
- *Streptococcus pneumoniae* is the most common cause of bacterial meningitis worldwide in both adults and children, followed by *Neisseria meningitidis* [7]
- Later, one or more of these lesions rupture producing CSF tuberculosis. The initial focus may occur in the meninges, subpia or subependymal surface of the brain. The growth of these lesions is known to be immunological in nature.
- Neurological tuberculosis (TB) or central nervous system tuberculosis (CNS-TB) may take three clinic-pathological forms: a diffuse form of tubercular meningitis (TBM), a focal form as tuberculoma, and spinal arachnoiditis also referred to as TB radiculomyelitis.[8]
- Tuberculomas are firm, avascular, spherical masses with diameters varying between 2 and 10 cm. They are well circumscribed, and the compressed surrounding brain tissue shows oedema and gliosis. The inside of the masses contains areas of necrotic caseation in which tubercle bacilli may be found.[9]

- Enhancing exudates in the basal cisterns or sylvian fissure with or without presence of ventriculomegaly, infarcts in the basal ganglia, or gyral enhancement patterns.[9]
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- Deep-seated tubercular granulomatous foci acquired during initial bacteremia may coalesce and develop into conglomerated caseous masses called tuberculomas without producing meningitis. Often, they may be present as clinically silent single or multiple lesions in patients with TBM and detected on brain imaging.
- This patient developed tuberculoma involving bilateral fronto-temporo-parieto-occipital lobe. Bilateral cerebellar , b/l thalamocapsular , all vascular territories involving ACA,MCA,PCA along with tuberculous infarct and cerebral areteritis.
- It presents with symptoms and signs of focal neurological deficit without systemic disease evidence.
- The extensive exudative arachnoiditis at the base of the brain and optic chiasma encases the multiple traversing cranial nerves, most commonly the abducens, optic, oculomotor, and trochlear. Similarly, arteritis and phlebitis result in vasculitic infarcts due to arterial vasospasm, thrombosis, or hemorrhage of the vessels.The recent diagnosis of intracranial tuberculoma is mainly based on fluid-attenuated inversion recovery (FLAIR sequences).[10]
- Pt had tiny multinodular opacities in lung feild on CXRAY which is the primary focus of the neurological lesions.
- Untreated or unrecognized TBM may cause death within 5 to 8 weeks of the onset of the disease.
- This type of patient may develop obstructive hydrocephalus because of thick exudates.

COMPLICATIONS

- Raised ICT, cerebral edema, stupor
- Basal meningitis with cranial nerve palsies
- Focal neurologic deficits
- Hydrocephalus
- Tuberculoma, tuberculosis abscess
- Optico-chiasmatic pachymeningitis resulting in visual loss
- TB arteritis and stroke
- Endocrine disturbances, SIADH, Diab. Insipidus
- Internuclear ophthalmoplegia
- Hemichorea
- Spinal block, spinal arachnoiditis

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