

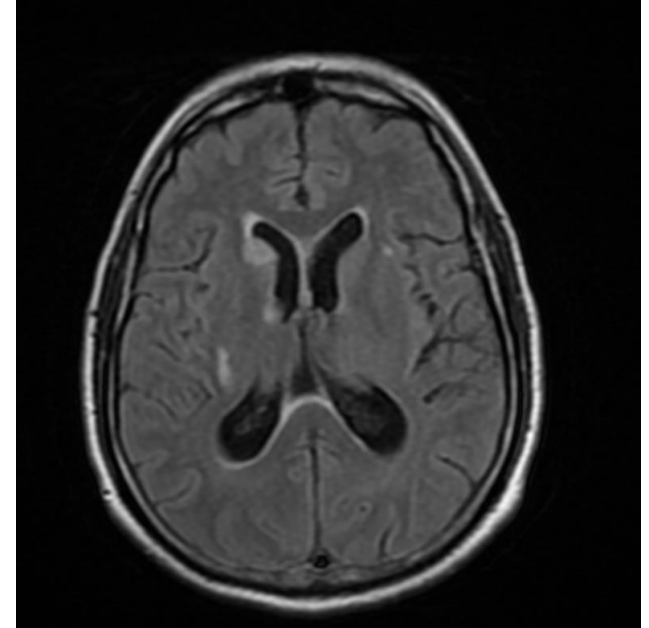
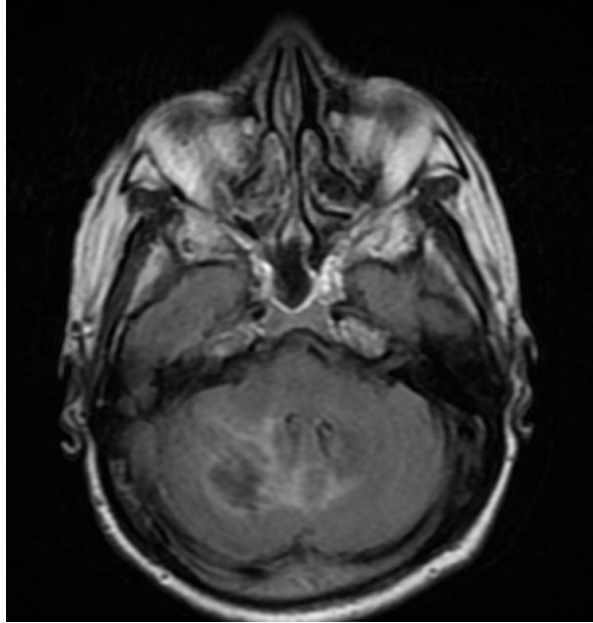
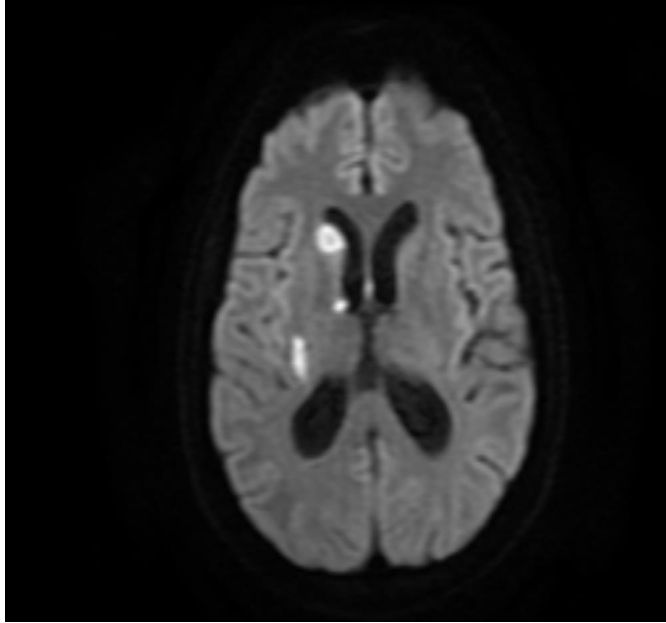


2025

KARNATAKA RADIOLOGY EDUCATION PROGRAM

Case 2: (MRN 17540000279015)

- 58 y old Male came with a history of weakness, weight loss and headache.
- Vitals were normal.
- No significant past medication.
- Outside CT reported right cerebellar space occupying lesion under evaluation.
- MR Brain was advised.



Tuberculosis

- Either hematogenous spread from distant systemic infection (e.g. PTB) or direct extension from local infection.
- extra-axial
 - tuberculous meningitis (leptomeningitis): most common
 - tuberculous pachymeningitis: rare
- intra-axial
 - intracranial tuberculous granuloma (tuberculoma)
 - focal tuberculous cerebritis
 - intracranial tuberculous abscess
 - tuberculous rhombencephalitis
 - tuberculous encephalopathy
- Complications: hydrocephalus (communicating or non communicating), ventriculitis, choroid plexitis, arterial vasculitis and dural sinus thrombosis, cranial nerve involvement (mainly second, third, fourth, and seventh cranial nerves.)

Epidemiology and Clinical features

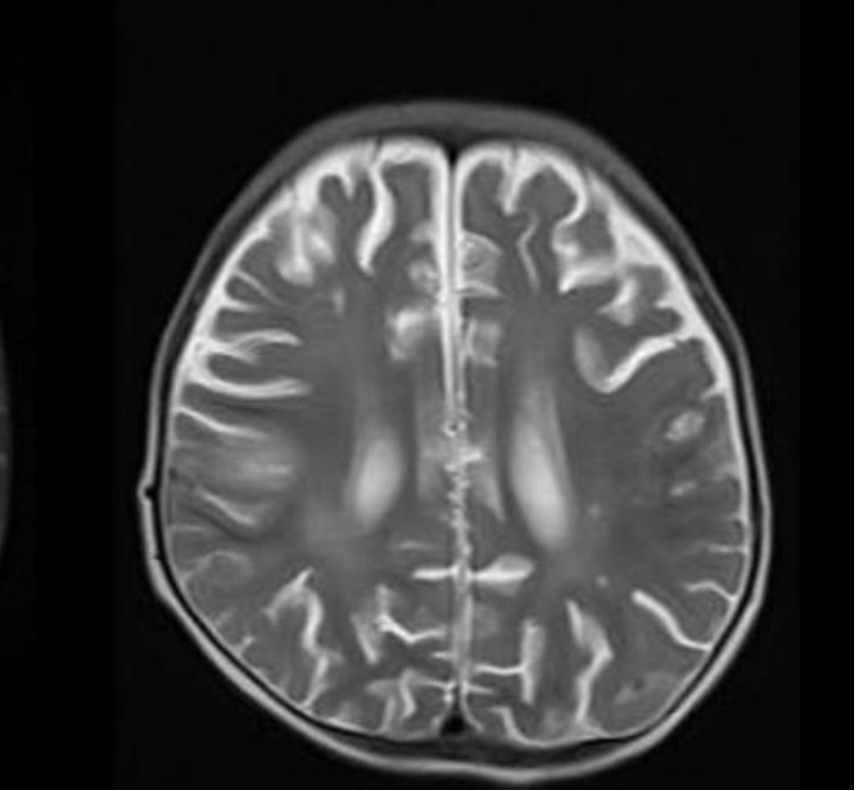
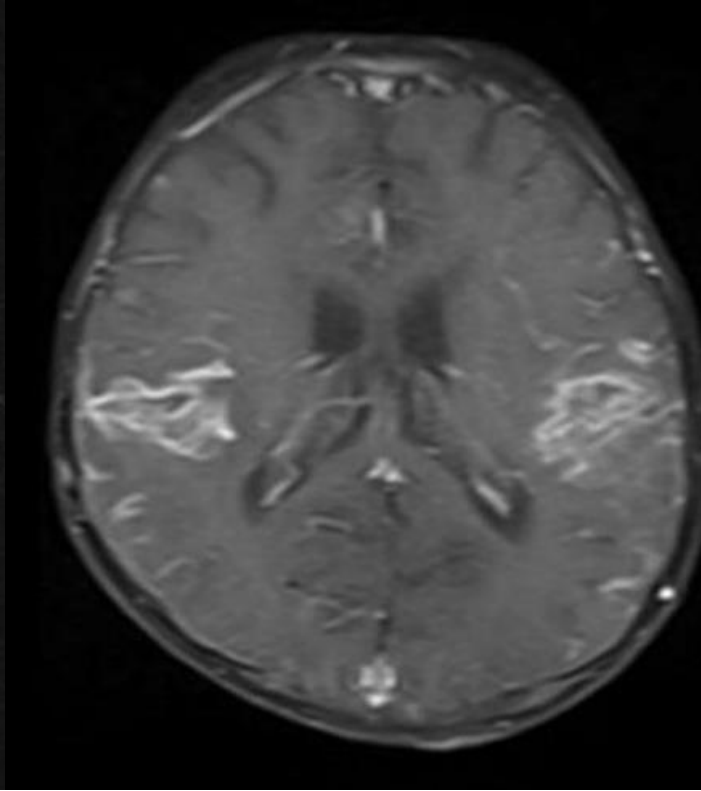
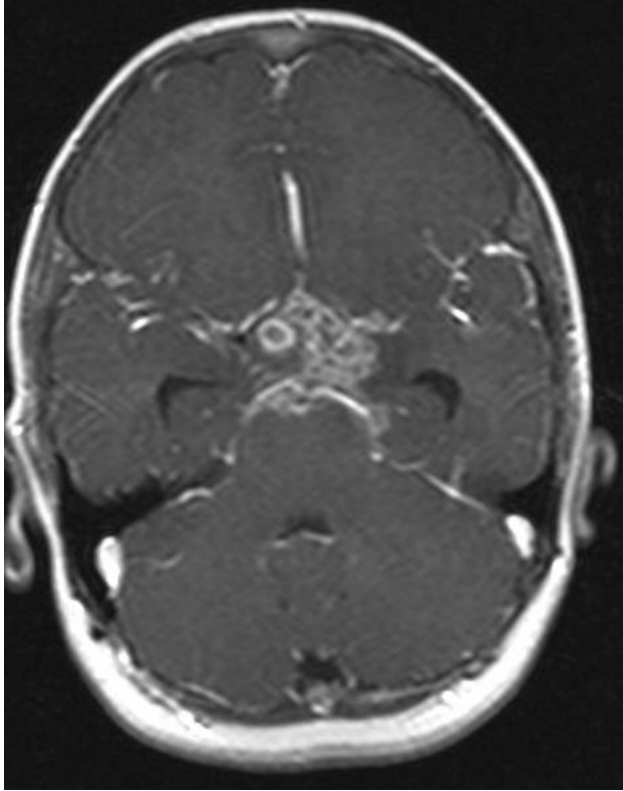
- CNS involvement is thought to occur in 2-5% of patients with tuberculosis and up to 15% of those with AIDS-related tuberculosis.
- Predilection for younger patients, with 60-70% of cases occurring in patients younger than 20 years of age.
- Tuberculomas account for as many as 50% of all intracranial masses in endemic regions.
- Non-specific clinical features include fever, seizures, meningism and focal neurological deficits (e.g. altered sensorium, hemiparesis)

Pathology

- Hematogenous spread from the lungs or gastrointestinal tract is most common, leading to small subpial or subependymal infective foci (Rich focus) which forms a reservoir from which intracranial manifestations may arise.
- This can occur either during the primary infection (uncommon, and more frequently seen in children) or be reactivated later and cause a post-primary infection.
- These foci can rupture into the subarachnoid space, forming an exudate, primarily affecting the vicinity of basal cisterns: inferomedial surface of frontal lobe, anteromedial surface of temporal lobes, superior cerebellum and floor of the fourth ventricle.
- From here, infection spreads to interpeduncular cisterns, around optic chiasm and to pontomesencephalic, ambient and suprasellar cisterns. Although the exudate can reach the Sylvian fissures it uncommonly extends over the cerebral convexities .
- Choroid plexitis may also be a late manifestation as is mass-like regions of caseous necrosis within this exudate.

Tuberculous meningitis

- Most common presentation of intracranial tuberculosis, and usually refers to infection of the leptomeninges.
- Pachymeningitis is rare.
- CT: NCCT may be normal. CECT: basal enhancing exudates, leptomeningeal enhancement, ependymitis.
- MRI
 - T1: normal initially. After disease progression T1 shortening may be seen
 - T2: normal initially. After disease progression T2 shortening may be seen
 - T1 post contrast: diffuse basal enhancement with enhancing exudates
 - MT spin echo: significantly lower MT ratio is seen in tuberculous meningitis as compared to fungal and pyogenic meningitis



Tuberculoma

- A tuberculoma is distinct from a tuberculous abscess in that it demonstrates evidence of granulomatous reaction and caseous necrosis histologically.
- may be solitary, multiple, or miliary and may be seen anywhere within the brain parenchyma, although it most commonly occurs within the frontal and parietal lobes.
- CT: round or lobulated nodule with moderate to marked edema. Either solid or ring enhancement is typical post-contrast. A central focus of calcification with a ring of peripheral enhancement (target sign) is described but is not specific to tuberculosis.

Non-caseating granuloma

- **T1:** iso- to- hypointense
- **T2:** hyperintense
- **FLAIR:** no suppression
- **DWI/ADC:** no restricted diffusion
- **T1 C+ (Gd):** homogeneous enhancement

Caseating granuloma

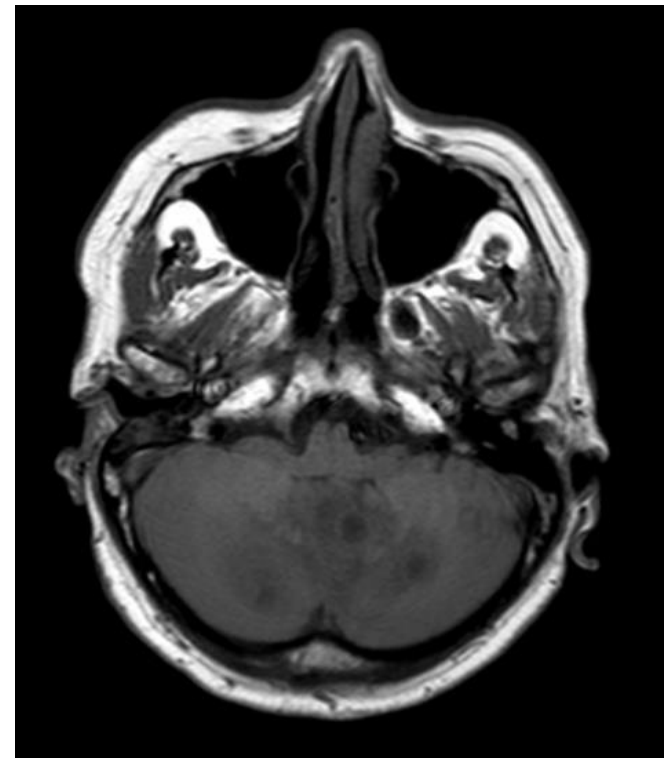
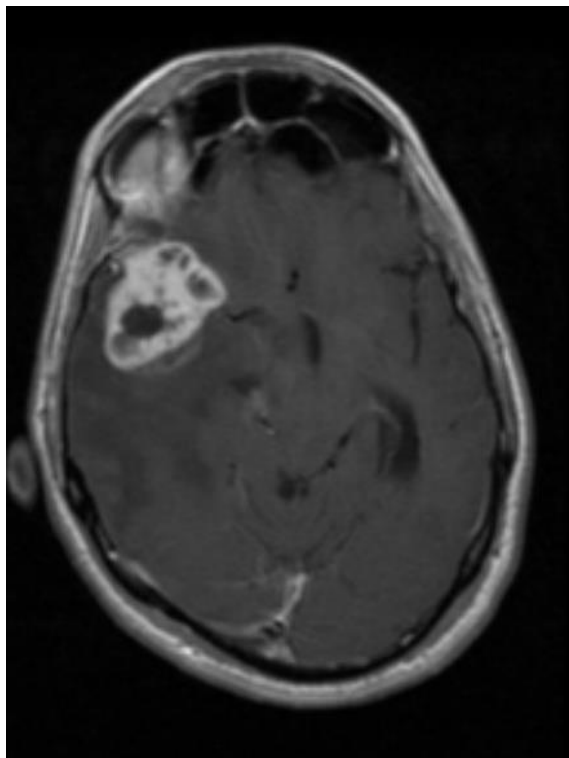
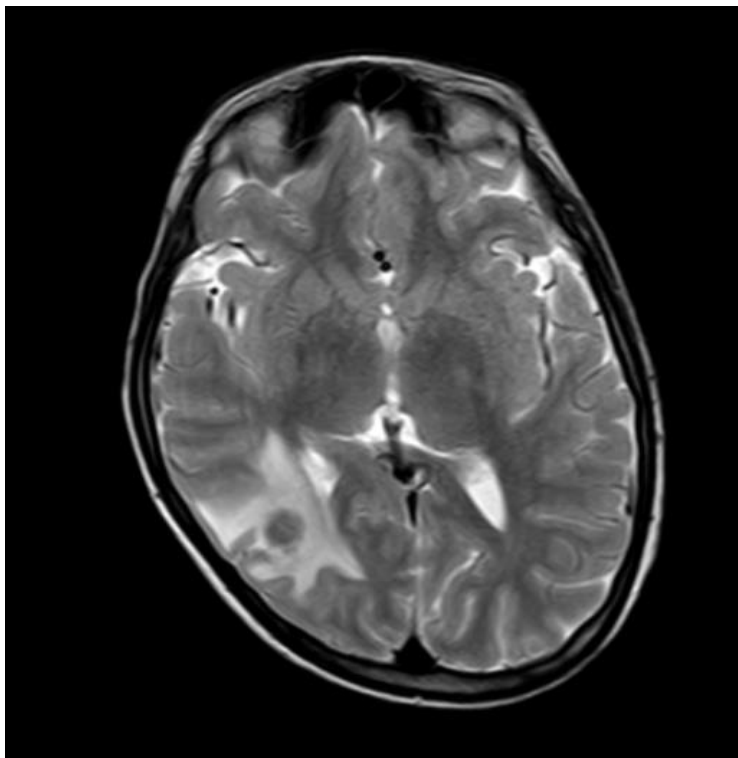
- **T1:** iso- to- hypointense with hyperintense rim
- **T2:**
 - hypointense representing gliosis and abundant monocyte infiltration ¹
 - surrounded by **vasogenic edema**
- **FLAIR:** no suppression
- **DWI/ADC:** no restricted diffusion
- **T1 C+ (Gd):** homogeneous or ring-enhancement
- **MR spectroscopy**
 - decrease in NAA/Cr
 - slight decrease in NAA/Cho
 - lipid-lactate peaks are usually elevated (86%) ²

Caseating granuloma with central liquefaction

- **T1:** iso- to- hypointense with hyperintense rim
- **T2:**
 - hypointense rim with central hyperintensity
 - surrounded by **vasogenic edema**
- **FLAIR:** partial suppression
- **DWI/ADC:** variable diffusion restriction
- **T1 C+ (Gd):** ring enhancement

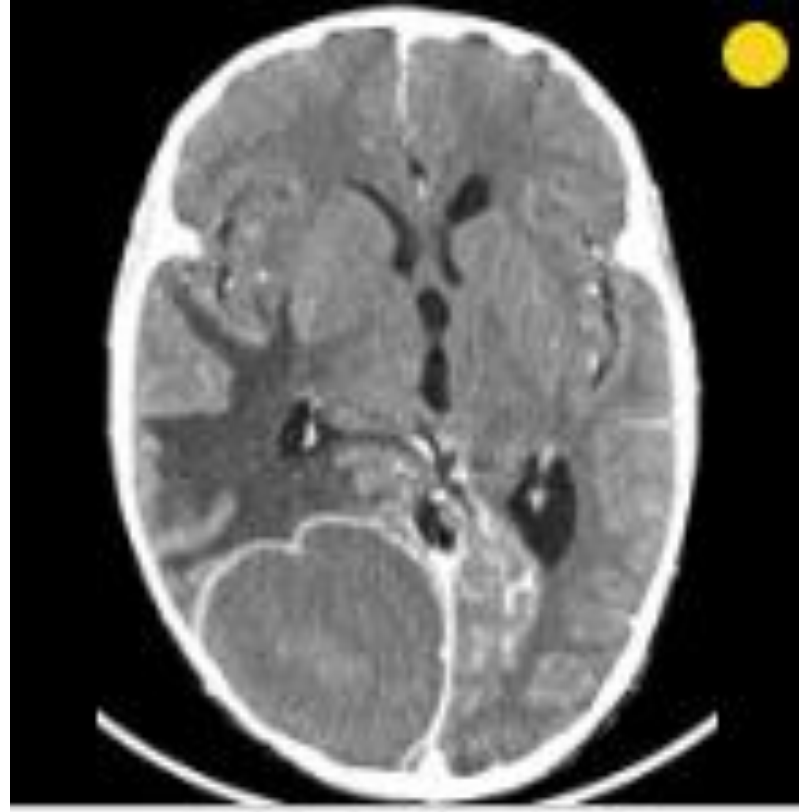
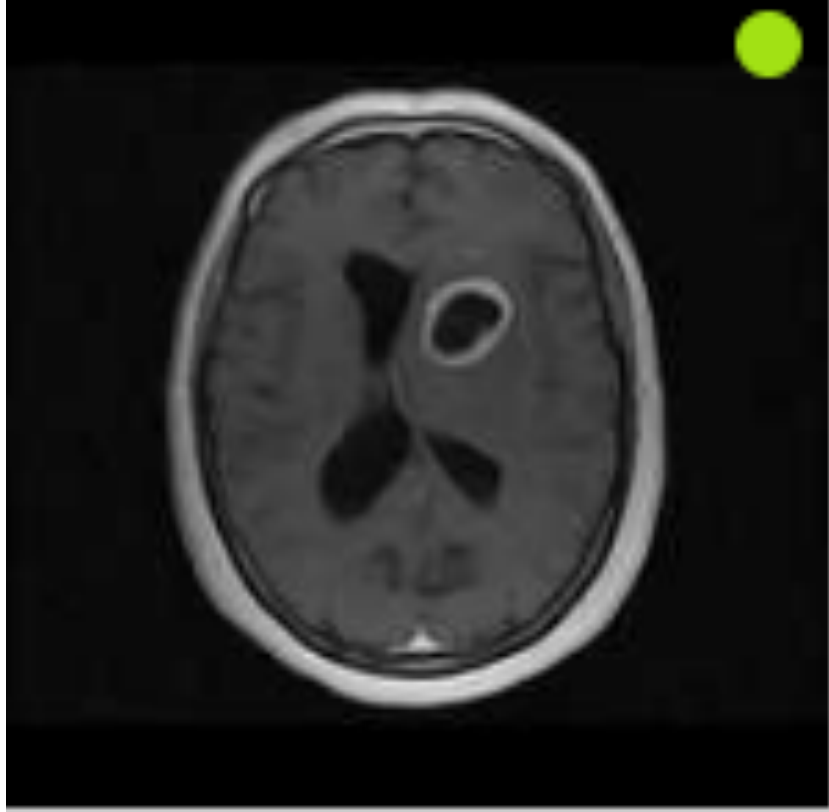
Calcified granuloma

- **T1:** iso- to- hypointense
- **T2:** hypointense
- **FLAIR:** no suppression
- **DWI/ADC:** no restriction
- **T1 C+ (Gd):** no enhancement



Intracranial tuberculous abscess

- Uncommon manifestation
- In contrast to tuberculomas, tuberculous abscesses are filled with pus
- They tend to be solitary, but can be multiloculated, and are relatively large and faster growing, compared to tuberculomas, that are usually a little smaller
- CT: peripherally-enhancing lesion with low attenuation center surrounded by vasogenic edema.
- T1: central low intensity (hyperintense to CSF), peripheral low intensity (vasogenic edema)
- T2 / FLAIR: central high intensity (hypointense to CSF, does not fully attenuate on FLAIR), peripheral high intensity (vasogenic edema), the abscess capsule may be visible as an intermediate to slightly low signal thin rim
- **T1 C+ (Gad):** ring enhancement
- **DWI/ADC:** high DWI signal is usually present centrally



Tuberculous (TB) encephalopathy

Tuberculous (TB) encephalopathy is a rare manifestation of CNS tuberculosis and is exclusively seen in children and infants with pulmonary TB.

It is characterized by cerebral edema sometimes with features similar to acute disseminated encephalomyelitis (ADEM) and may manifest with a variety of symptoms ranging from focal neurological deficits to convulsions and decreased conscious state.

It does not appear to be due to direct infection of the CNS by TB.

Miliary CNS TB

Miliary CNS tuberculosis is usually associated with tuberculous meningitis and appears at MR imaging as multiple tiny (<2-mm), hyperintense T2 foci that homogeneously enhance on contrast-enhanced T1-weighted images.

Treatment

- Anti-tubercular treatment regimen
- Multidrug-resistant tuberculosis remains a major hurdle in treatment
- Treatment of complications : drainage of hydrocephalus
- Surgical drainage in case of tuberculous abscess