Tuberculosis of brain,meninges

Pathogenesis

- 1.hematogenous seeding of meninges
- 2.quiscent phase of week,months until stimulus releases organisms & antigens in tubercles into meningeal spaces.

PATHOLOGY-damage to brain parenchyma(ichaemia & infarction because vascular insufficiency)

Atrophy of grey & white matter due to chronic hydrocephalus

Clinical features

- Headache,fever,meningismus,nausea,behavioural abnormality-lethargy
- Neurological signs
- Stage I-no alteration in consciousness
- No focal/generalised neurologic signs
- Stage II-altered consciousness
- 6^{th} nerve most often affected,then 3^{rd},4^{th} & 7th

- Stage III major neurologic deficits eg coma,seizures,paresis or paralysis of one or more extremities
- Ophthalamologic signs
- Choroid tubercles
- Papilledema by fundoscopy

Diagnostic tests

- Wbc –increase,decrease or normal
- Esr –increase or normal
- cxr –active or healed lesions
- CEREBROSPINAL FLUID
- Lymphocytic pleocytosis
- Spinal fluid protein increase
- Moderate hypoglycorrachia

- Protein 150-200 mg% is most reliable sign
- Glucose never falls to undetecable level as in bacterial meningitis
- MICROBIOLOGY
- Smears of spinal fluid are positive in < 10% of patients
- Culturing CSF for mycobacteria is also unreliable technique.

Radiology

- Separation of cranial sutures in infants in hydocephalus
- Meningeal calcification
- Tuberculoma-uniformly enhancing mass lesions

Other diagnostic techniques

- Despite its deficiencies,a positive culture remains by default the gold standard of diagnosis in TBM
- Adenosine deaminase ,an enzyme elaborated by activated T lymphocytes, is significantly elevated in CSF
- Tuberculostearic acid , a component of cell wall of M.tuberculosis detected in CSF.

- Newest & most powerful technique of molecular biology,polymerase chain reaction(PCR) used to assay CSF for mycobacterial DNA
- ELISA-antibodies against BCG,against PPD identified in spinal fluid by ELISA.
- CSF IgG antibody to PPD

Tuberculoma

- Form when intracranial tubercle enlarge without rupturing into subarachnoid space
- They have thick fibrous capsule.
- Occur simultaneously with TBM or independently
- Also occur or enlarge during a course of therapy
- Most common intracranial mass lesion in developing countries.

- Sumptoms often limited to seizures
- CT scan-uniform contrast enhancement or thick rind of enhancement surrounding a central clearing ie caseous center of lesion
- MRI more sensitive than CT
- Diagnosis confirmed by biopsy.

Treatment

- Pyrazinamide ,like INH, has excellent pharmokinetics for CNS,penetrating meninges well at all stages of inflammation
- Rifampicin achieves lower levels in CSF
- Penetration of ethambutol across BBB is substantial if meninges are inflammed

Steroids

- In some subsets,steroids are helpful but in others they may be unnecessary.
- Steroids in stage II & stage III as well as spinal cord disease
- Prednisolone 60 mg/day or dexamethasone 0.2mg/kg/day for several weeks ,tapered over an equal period of time.

Surgery

- Neurosurgical intervention to relieve hydrocephalus that complicates most cases of TBM.

Duration of treatment

- Several recent studies suggest 6-12 months may suffice
- Stage I/II-9-12 months
- Stage III-12-18 months
- Tuberculomas 18-24 months

- All the best..