



TUBERCULOUS MENINGITIS

An early diagnosis makes for a better outcome

What is the problem?

Tuberculous meningitis (TBM) is extremely common. It is becoming the commonest cause of meningitis in South Africa. TBM is notifiable.

TBM is a basal meningitis causing a severe vasculitis, which results in infarction, particularly of the brainstem.

Why is it important?

- Morbidity: 80% mental handicap, 25% motor handicap
- Mortality: "high"

What is the severity?

Initial severity is defined by the findings at presentation

Stage 1

- Non-specific symptoms and signs. May present with lethargy only.

Stage 2

- Meningeal signs, with neurological fallout and signs of raised intracranial pressure.

Stage 3

- Depressed level of consciousness

Children in our setting usually present with advanced disease. Sometimes this is because of the reasonable parental thought that the child with vague symptoms will get better, sometimes it is because health workers have not had a high enough index of suspicion early in the illness. Only 1/3 of TBM patients are diagnosed on their first visit!

What are the implications of severity?

All children with suspected TBM must be admitted. All children with signs of raised ICP should have a CT scan because the cause may be hydrocephalus, which is treatable. If your hospital has no scanner, refer to a hospital with one.

What is the cause and the associated problems?

The cause in children is haematogenous dissemination of AFB's. ALL CHILDREN WITH ANY OTHER SIGN OF DISSEMINATED TB - like a miliary appearance on Chest X-ray - MUST HAVE A LUMBAR PUNCTURE (unless contraindicated by raised intracranial pressure)

1) Diagnose TBM

- Find the household contact
- Skin test
- Chest X-ray: child and caregiver
- Find the AFB's: CSF, induced sputum, gastric washings, lymph node biopsy (tailor to age and presentation findings); all specimens MUST be sent for TB culture and sensitivity

A lumbar puncture should be done in all patients unless contra-indicated by signs of raised ICP. Definitive diagnosis is essential as it determines treatment, which needs to be comprehensive and sustained.

CSF findings

- The following findings support the diagnosis of TBM: pleocytosis with lymphocyte predominance, high protein, low chloride. A CSF picture that differs from this does NOT exclude TBM.
- In TBM, the protein is usually still markedly high a month after treatment onset
- Exclude other infections, especially cryptococcus (CSF)
- Exclude space taking lesions: tumour and abscess (CT Scan)

2) Evaluate Raised Intracranial Pressure

70% of hydrocephalus in TBM is communicating, 30% is non-communicating

- Do a CT Scan: look for dilated ventricles, basal enhancement, areas of infarction
- Raised ICP may be due to hydrocephalus which is easily treatable, or cerebral oedema which is not easily treatable
- DECIDE whether it is safe to do a LP
- Do opening and closing pressures on the CSF at lumbar puncture and consider doing an air encephalogram – injecting 5ml of air after LP is the easiest, quickest and cheapest way of determining whether the hydrocephalus is “communicating” or not. Do a skull X-ray within half an hour with the child sitting up

3) Evaluate and document neurological deficit

- Do a thorough neurological assessment, and do a developmental screen as soon as possible after admission. The degree of recovery is often remarkable, and we should be documenting this

4) Look for SIADH (syndrome of inappropriate antidiuretic hormone)

- Check if urine osmolality is inappropriately high for serum osmolality + hyponatraemia

What is the management?

1) Admit

There is no place for ambulatory care in the treatment of TBM. Admit all suspected/confirmed cases. Never discharge until you are sure that you have achieved a cure.

2) Kill the AFB's

- RIFAMPICIN 20mg/kg 24H PO
- ISONIAZID 20 mg/kg 24H PO
- PYRAZINAMIDE 40 mg/kg 24H PO
- ETHIONAMIDE 20 mg/kg 24H PO

Continue 4-drug treatment for six months

If transferring a child with (suspected) TBM, initiate treatment prior to transfer, AND check the child's HIV status

3) Treat hydrocephalus

- **Non-communicating:** emergency VP shunt
- **Communicating:** ACETAZOLAMIDE 10mg/kg 6H PO (max 1g/day) and FUROSEMIDE 1-2 mg/kg 24H PO for one month, +/- serial lumbar punctures

4) Dampen the vasculitis

- Steroids decrease morbidity and mortality - give PREDNISON 4mg/kg 24H PO for one month, then taper

5) Treat SIADH

- Give normal maintenance fluids for age (discuss with Paediatrician)

Fluid restriction is probably illogical, and it potentially decreases cerebral perfusion pressure, and may contribute to thrombosis and worsen overall outcome

What is the follow up?

- The long term follow up venue is determined by the degree of neurodevelopmental deficit. Optimisation of neurodevelopment by physio- and occupational therapists should begin IN HOSPITAL, involving the caregiver early.
- In any child with severe developmental delay, a Care Dependency Grant should be applied for at the earliest practical opportunity. Encourage the caregiver to start obtaining the necessary documentation from early on (see separate guideline on [Grants](#) and use the [Grant Application Letter](#)).

What are the preventive measures?

- Home-based: alleviate poverty and overcrowding
- Health service-based: BCG may offer some protection, notification, HIV testing – because co-infection is LIKELY and it causes increased morbidity and mortality - , case finding, contact tracing and prophylaxis and DOTS

What information should be given to caregivers?

Caregivers should be informed that the hospital stay will be a long one and that a neurodevelopmental deficit is possible. Assistance should be provided from very early on with learning home-based physiotherapy and occupational therapy, and with grant applications.

Your final and **MAJOR** responsibility is to make sure that the child is **NOTIFIED**