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Original Article

TUBERCULOUS MENINGITIS IN CHILDREN

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Abstract:

Introduction: Tuberculosis Meningitis (TBM) is a dreaded complication of primary tuberculosis (Mycobacterium tuberculosis M tuberculosis). Age Commonest age is between 1 and 4 years. It is rare before 6 months of age. Pathogenesis Most common mode of spread is hematogenous.

MethodologyTuberculosis meningitis occurs during hemitog nous dissemination of M tuberculosis. It usually occurs 1 3 months after the primary infection. Small tuberculosis form near meanings the Rich foci.

Results: From these organisms are carried into meanings. Small tubercles are often best seen on ependymal lining of lateral ventricles, and on cortex and walls of ventricles. Gelatinous exudates fill spaces at base of brain basal meningitis. Hydrocephalus may develop. Areas of brain softening from vascular obstruction develop.

Conclusion: Clinical Manifestations of Tuberculosis Meningitis must be considered in any child who had measles followed by persistent temperature and CNS manifestations.

Key words: patient. Hospitals.Lungs.Disease. Death

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Introduction

Tuberculosis Meningitis (TBM) is a dreaded complication of primary tuberculosis (Mycobacterium tuberculosis M tuberculosis). Age Commonest age is between 1 and 4 years. It is rare before 6 months of age. Pathogenesis Most common mode of spread is hematogenous. Tuberculous meningitis occurs during hematogenous dissemination of M tuberculosis. It usually occurs 1 3 months after the primary infection. Small tuberculomas form near meanings the Rich foci.

From these organisms are carried into meanings. Small tubercles are often best seen on ependymal lining of lateral ventricles, and on cortex and walls of ventricles. Gelatinous exudates fill spaces at base of brain basal meningitis. Hydrocephalus may develop.Areas of brain softening from vascular obstruction develop.

Clinical Manifestations

Tuberculosis Meningitis must be considered in any child who had measles followed by persistent temperature and CNS manifestations. Insidious onset. Preceding history of measles is verysignificant.

Early diagnosis is important as prognosis of TBM is poor with late diagnosis even with treatment. The clinical manifestations are divided in three stages. Prodromal stage (1" Stage): Symptoms are
vague. The child's mentality appears to alter; he/she becomes irritable, sleeps frequently, becomes restless, and has unaccountable vomiting, constipation and headache. Neurological signs are absent however there may be slight neck rigidity and 'boggy' anterior fontanel in an infant.

Stage of irritation (2 Stage): Child becomes drowsy, turns away from light, appears dehydrated and tends to pick skin and grind teeth. Pulse is slow. Signs of meningeal irritation as neck stiffness etc. appear. Neurological signs appear as cranial nerve palsies (ophthalmoplegia, facial nerve palsy etc.), hemiparesis and papilledema. The child develops fits. Hydrocephalus may appear. There may be 'cracked pot sound' on percussion of skull.

Terminal Stage (3" stage): Child becomes comatose. He is extremely wasted. He develops hypertonia and may assume opisthotonus, decrebrate or decorticate postures. Untreated this stage leads to death,

Investigations

1. Complete blood count (Leukocytosis and lymphocytosis may be present. ESR is raised.

2. Monteux Test Positive during initial stages. However it may be negative due to energy because of TBM.

3. Chest X-ray (primary focus),
4. Blood glucose (for comparison with glucose in cerebrospinal fluid CSF)
5. Serum sodium and potassium
(Syndrome of inappropriate ADH
Secretion SIADH).
6. Lumbar puncture for CSF:SAME PARFP

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Injection Streptomycin 40 mg/kg/d 1/M OD (first 2 months) Duration of therapy 12 months at least.
Prevention
BCG vaccination has protective role against TBM.

Prognosis and Follow-up

The prognosis of meningitis depends on the stage of the disease. With appropriate treatment there is 100% cure rate with low Incidence of sequelae in stage 1. In stage 2 there is 85% cure rate and 50% survivors develop neurological defects. In the 3 stage even with best treatment the mortality is > 50% and most of the survivors have permanent handicaps. Sequlae of TBM includes blindness, deafness, palaplegia/hemiplegia/ monoplegia, cranial nerve palsies, seizures, hydrocephalus, diabetes insipidus, obesity, and mental retardation.

Count	wac/me L

Differential count	50
Normal	60

Sugar mp/al	00 (67 of blood sugar) <45

TBHPredominant LymphocytesProteins mg/d, 100Staining of sugar) sample OtherElevated (100- 1000)(<50 of blood centrifuged

7. Gastric lavage for culture for tubercle bacilli may be positive.

8. Fundi examination may show choroid tubercles.
9. Neuroimaging (cranial ultrasound, CT Scan of brain, MRI). Management ABC Assurance of adequate airway, ventilation and cardiac function. Stabilization of hemodynamic status by achieving venous access and treatment of shock if present.Control of seizures intravenous diazepam, phenobarbitoneetc.Control of feverparacetamol, ibuprofen.

Maintenance of fluid and electrolyte balance
I/V fluids 70% of the daily requirement, monitoring of sodium concentration (SIADH), correction of hypoglycemia (I/V glucose) & acidosis (sodium bicarbonate) and nutritional support. Management of raised intracranial pressure.

Mannitol infusion

Steroids Dexamethasone 0.2 mg/Kg/d 1/V later orally in 2 3 divided doses for 3 4 weeks.
Drugs Isoniazid 10 mg/Kg/d orally in morning Rifampicin 15 mg/kg/d orally in morning empty stomach.
Pyrizinamide 30 mg/kg/d in 2 dd orally (first 2 months).

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