



ACUTE BACTERIAL MENINGITIS

INTRODUCTION



- Inflammation of the meninges by bacterial infection
- Involvement of brain parenchyma meningo-encephalitis
- One of the most potentially seriously infection
- High rate of acute complications and chronic morbidity



AGE AND INCIDENCE

- Age:
 - 90% between 1month to 5 yrs
- Incidence:
 - 5.4 to 7.3/1,00000 population
 - Decreased after use of conjugate H. Influenza type b vaccine



ETIOLOGY

Depends on :

- Age
- Immune Status of the host
- Environmental factors



NEONATAL PERIOD

- Group B streptococcus.
- Gram negative enteric organisms.
- *Listeria monocytogenes*.
- *H. influenza*.



2MONTHS -12YRS:

- H. influenza(<5yrs)
- Streptococcus pneumoniae
- N.meningitidis
- Rarely : Staph.aureus, S.typhi, Pseudomonas

RISK FACTORS



- Lack of immunity
- Close contact with invasive disease caused by H.influenza, N.meningitidis.
- Overcrowding
- Occult bacteraemia in infants



RISK FACTORS

- Specific host defense deficits leads to recurrent & severe meningitis
 - Hypo/agammaglobulinemia
 - Defects in the complement & properidin systems
 - Splenic dysfunction
 - T cell defects, HIV infection

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RISK FACTORS

- CSF leak :
 - fracture skull, fracture of cribriform plate
 - cranial midline defects
- VP shunt
- Meningocele
- Lateral sinus thrombus

SPECIFIC PREDISPOSING FACTORS AND ORGANISM



Predisposing factors	Organism
Defects of the complement, properdin system	Meningococcus
Splenic dysfunction	Pneumococcus, H. Influenza, Meningococcus
Meningomyelocele, CSF shunt inf, cranial trauma	Staphylococcus
CSF leakage, basal skull fracture, ear inf, cochlear implant, cranial defect	Pneumococcus
T cell defect(AIDS, malignancy, chemotherapy)	<i>Listeria monocytogenes</i>



ROUTES OF INFECTION

- Blood stream
- Direct Invasion: Osteomyelitis, Sinusitis
- Cochlear implant
- Head trauma
- Cong. defects: myelomeningocele,
- Neurosurgical procedure



PATHOLOGY AND PATHOPHYSIOLOGY

Pathological changes:

- Exudates.
- Subpial toxemia leading to parenchymal disease.
- Vasculitis & Thrombosis of veins.

PATHOLOGY AND PATHOPHYSIOLOGY



- Exudates accumulate
 - Around the cerebral vessels & venous sinuses
 - Base and surface of the brain
 - Sylvian fissures, cerebellum &
 - Subdural space

PATHOLOGY AND PATHOPHYSIOLOGY



- Leads to
 - Hydrocephalus
 - Signs of meningeal irritation
 - Signs of raised intra cranial tension
 - Cerebral ischemia resulting in neurodeficits.

PATHOLOGY AND PATHOPHYSIOLOGY



Vascular changes:

- Subintimal changes in the small vessels & arteries.
- Thrombosis of small cortical veins
- Subarachnoid hemorrhage
- Hemiparesis, cranial nerve palsies, convulsions



PATHOLOGY AND PATHOPHYSIOLOGY

- Subpial toxemia:
 - Leads to cerebral edema.
 - Cytokine induces
 - Increased vascular permeability &
 - Increased hydrostatic pressure
 - Signs of ICP, altered mental status

CLINICAL FEATURES



- Hyperacute :1-2 days
- Acute presentation : 2-7 days

Symptoms:

- Fever
- Headache
- Nausea, projectile vomiting
- Irritability, confusion
- Seizures



SYMPTOMS IN NEWBORNS

- Irritability
- Hypothermia, Lethargy
- High pitched cry, refusal of feeds
- Altered mental status (60%)
- Bulging AF (30%) & seizures (40%)



SIGNS OF MENINGEAL IRRITATION

DEMONSTRATION OF NUCHAL RIGIDITY



NUCHAL RIGIDITY IN SITTING POSITION



KERNIG'S SIGN



BRUDZINSKI'S SIGN





FALSE POSITIVE NECK SIGN

Extracranial causes

- Upper lobe pneumonia
- Typhoid fever
- Cervical spine diseases
- Retro pharyngeal abscess
- Myalgia
- Tender Lymphadenitis
- Tonsillitis

Intracranial causes



NECK SIGNS MAY ABSENT IN..

- Infants
- Partially treated meningitis
- Severe Malnutrition
- Immunocompromised
- Sick, terminal ill child



DIFFERENTIAL DIAGNOSIS

- Aseptic meningitis
- TB meningitis
- Brain abscess
- ICSOL
- Cerebral malaria



CNS COMPLICATIONS

- Short term :
 - Subdural effusion or empyema
 - Brain abscess
 - Arachnoiditis , Ventriculitis
- Long term :
 - Hemiplegia , aphasia
 - Ocular palsies , blindness
 - Auditory impairment
 - Mental retardation



SYSTEMIC COMPLICATIONS

- Shock
- Myocarditis
- Arthritis
- DIC
- SIADH
- Status epilepticus



INVESTIGATIONS

CSF analysis:

- Turbid or opalescent with elevated opening pressure.
- Normal healthy neonate may have as many as 30 leucocytes/mm³

CSF



- Leucocyte $>1000/\text{mm}^3$ (75-95% PMN)
- Absence of pleocytosis carries poor prognosis.
- CSF glucose - $<50\%$ of blood glucose
- In traumatic LP, Gram stain, culture and glucose level are not altered



CSF

- Proteins : 100- 500 mg/dl
- Grams stain: positive in 60- 90 % cases.
- Culture: Positive in 70- 85% of cases

CONTRAINDICATIONS OF LP



- Evidence of increased ICP
- Severe cardiopulmonary compromise
- Infection of the overlying skin
- Thrombocytopenia is a relative contraindication
- If an LP is delayed, empirical antibiotic therapy should be initiated



BLOOD TESTS

- Leucopenia is one of the poor prognostic signs.
- Thrombocytopenia may be seen with sepsis, meningococcal infection.
- Serum electrolytes estimation is necessary for the evaluation for SIADH.

INDICATION FOR NEUROIMAGING



- Subdural effusion.
- Hydrocephalus.
- Focal neurological signs
- Emergency CT
 - Papilloedema
 - Before LP

SPECIAL INVESTIGATIONS



- Latex agglutination techniques: to detect the antigens for H.influenza, Strep.pneumoniae, meningococcus, E.coli.
- PCR : Sensitive in the 91% of the cases.
May be useful in the partially treated meningitis.
- CT or MRI: When lumbar puncture is contraindicated or complications are expected

TREATMENT



- Initial Stabilization
- Antibiotics
- Treatment of Complications
- Supportive care
- Follow up and Rehabilitation

INITIAL STABILIZATION



- Initial treatment of ABC
- Correct shock, respiratory distress , multiple organ system failure
- Monitoring of pulse rate, BP and respiratory rate
- Frequent neurologic assessment

ANTIBIOTICS

- Prompt therapy with appropriate antibiotics

Selection depends on

- Causative pathogens.
- Ability to penetrate the blood brain barrier to achieve bactericidal concentration in CSF.
- Age of the child.
- Local incidence & susceptibility pattern



IN 0-2 MONTHS:

- Gram –ve organisms – 64%.
- Gram +ve organisms – 36%.

Ampicillin + gentamycin/ cefotaxime – Drug of choice.

Repeat CSF in 24-36hrs.

If Pseudomonas is suspected Ceftazidime should be used.



> 2MONTHS

- 3rd generation cephalosporins
- Cefotaxime / ceftriaxone



DURATION OF ANTIBIOTICS

- 10-14 days IV antibiotics
- Neonatal meningitis, staphylococcal – 21 days

ROLE OF CORTICOSTEROIDS

- Decrease ICP
- Modulate the production of cytokines
- Reduce the incidence of SN hearing loss and other neurological complications
- Improve BBB penetration of antibiotics
- Shorten duration of fever
- Dexamethasone: 0.15mg/kg/dose, 6hrly for 2days



MANAGEMENT OF COMPLICATIONS

Management of raised ICT

- Head end elevation of 30 degree
- Mannitol

Seizures:

Anticonvulsants

Fluid and electrolyte Management

Subdural empyema / hydrocephalus – Surgical intervention if needed



SUPPORTIVE CARE

- Care of oral cavity, eye, bladder, bowel, skin
- Management of constipation: Enemas
- Prevent bed sores – Frequent position change
- Regular monitoring: Vitals, Neurological status , features of raised ICT, Head circumference



FOLLOW UP AND REHABILITATION

- Assessment of IQ, Hearing, Vision
- Serial Head circumference
- Physiotherapy
- F/u of seizure treatment
- Family support

PROGNOSIS



Bad prognosis in:

- Younger patient
- Greater the antigen load
- Late treatment
- Seizure after 4th day,
- Coma or focal neurological deficit at presentation

PREVENTION



- Vaccines- for Hib, S.pneumoniae, N.meningitidis
- Antibiotic prophylaxis – For contacts
 - Rifampicin or ceftriaxone
 - In Hib and meningococcal meningitis
- Schedule depends on the specific bacteria



SUMMARY

- ABM is life threatening infection
- Early treatment with correct antibiotic is the most important prognostic factor
- Supportive treatment and treatment of complication also need attention
- There are effective vaccination, by which majority of the ABM can prevented

Thank You

