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 1 month to 5 yrs

• Incidence:
  – 5.4 to 7.3/1,000,000 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.menigitidis
- 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
RISK FACTORS

- CSF leak:
  - fracture skull, fracture of cribiform plate
  - cranial midline defects
- VP shunt
- Meningocele
- Lateral sinus thrombus
### SPECIFIC PREDISPOSING FACTORS AND ORGANISM

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

- Around the cerebral vessels & venous sinuses
- Base and surface of the brain
- Sylvian fissures, cerebellum &
- Subdural space
• Leads to
  – Hydrocephalus
  – Signs of meningeal irritation
  – Signs of raised intra cranial tension
  – Cerebral ischemia resulting in neurodeficits.
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
KERNIGS 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

- Sub-arachnoid hemorrhage
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/mm$^3$
CSF

- Leucocyte >1000/mm³ (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 2 days
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
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