3-Meningitis

1-Meningitis is an inflammation of the membranes (the meninges), whereas **encephalitis** is an inflammation of the brain tissue. **75% of cases of meningitis are believed to occur in those <15yrs of age**.

2-**Three organisms** (***Streptococcus*** *pneumoniae*, ***Neisseria*** *meningitides* and ***Haemophilus*** *influenzae* type b) account for 80% of the cases. [In newborns, *Group B streptococcus* , *E. coli*, and *Listeria monocytogenes* are the most common pathogens].

Clinical Manifestations

1- In **young infants symptoms may be non-specific** including fever, poor feeding, lethargy.

2-In older children clinical features include:

- ***General****:* fever, with headache.
- ***Central****:* irritability, disorientation, altered mental state.
- ***Seizures****:* occur in 30%.
- ***Neck stiffness****:* more common in older children.
- **Kernig** and **Brudzinski** signs of meningeal irritation are often positive in children older than 12 months.

Diagnosis

1-If bacterial meningitis is suspected, a **lumbar puncture** should be performed . Routine CSF examination includes a white blood cell count, differential, protein and glucose levels, and Gram stain.

Treatment

1-Treatment of bacterial meningitis focuses on **sterilization of the CSF by antibiotics**.

2-Duration of treatment is 5 to 7 days for *N. meningitidis,* 7 to 10 days for *H. influenzae,* and 10 to 14 days for *S. pneumoniae*.

3-Steroids In bacterial meningitis:

• Do not use corticosteroids in children younger than 3mths.

• There is benefit from the use of dexamethasone and the dosing schedule is 0.15mg/kg qds for 4 days **to reduce the severity of neurological sequelae**, **particularly deafness**, after bacterial meningitis).

• If dexamethasone was not given before the first dose of antibiotics, but was indicated, try to give the first dose within 4hr of starting antibiotics,

but do not start dexamethasone more than 12 hours after starting antibiotics [b].

Table 1: Initial Antimicrobial Therapy by Age for Presumed Bacterial Meningitis

AGE	RECOMMENDED TREATMENT	ALTERNATIVE TREATMENTS
Newborns (0–28 days)	Cefotaxime or ceftriaxone plus ampicillin with or without gentamicin	Ampicillin plus gentamicin Ceftazidime plus ampicillin
Infants and toddlers (1 mo–4 yr)	Ceftriaxone or cefotaxime plus vancomycin	Cefotaxime or ceftriaxone plus rifampin
Children and adolescents (5–13 yr) and adults	Ceftriaxone or cefotaxime plus vancomycin	Cefepime or ceftazidime plus vancomycin

4-Encephalitis

1-Encephalitis is an inflammation **of the brain tissue** . **Viruses are the principal causes** of acute infectious encephalitis [(1)].

Clinical Manifestations

1-Acute infectious encephalitis usually is preceded by a prodrome of several days of nonspecific symptoms such as sore throat, fever, and headache followed by the **characteristic symptoms** of progressive **lethargy**, **behavioral changes**, and **neurologic deficits**. **Seizures are common** at presentation.

Diagnosis

The diagnosis of viral encephalitis is supported by **examination of the CSF**.

Treatment

1-With the exception of HSV, varicella-zoster virus, cytomegalovirus, and HIV, there is no specific therapy for viral encephalitis. **Management is supportive**.

2-Intravenous **acyclovir is the treatment of choice for HSV** and **varicella-zoster virus** infections. **Cytomegalovirus** infection is treated with **ganciclovir**. HIV infections may be treated with a combination of antiretroviral agents.

5-Visceral Leishmaniasis (Kala-azar) (Black fever)

- Visceral Leishmaniasis (VL) is caused by the protozoon **Leishmania donovani**.
- Infection are introduced by the feeding female **sand fly**.
- The great majority of people infected remain asymptomatic. In visceral diseases the spleen, liver, bone marrow and lymph nodes are primarily involved.

Clinical features

- VL is predominantly a disease of small children and infants.
- The **first sign of infection is high fever**, usually accompanied by rigor and chills.
- **Splenomegaly** develops quickly in the first few weeks and becomes massive as the disease progresses. Moderate hepatomegaly occurs later. Lymphadenopathy may also seen .
- Blackish discoloration of the skin, from which the disease derived its name, kala-azar (the Hindi word for 'black fever'), is a feature of advanced illness and is now rarely seen.
- **Pancytopenia** is a common feature.
- Without adequate treatment most patients with clinical VL die.

Diagnosis

- Demonstration of amastigotes in **splenic smears** is the most efficient means of diagnosis, with 98% sensitivity ; however, it carries a risk of serious haemorrhage in inexperienced hands.
- Serodiagnosis, by ELISA or indirect immunofluorescence antibody test (**IFAT**). A significant proportion of the healthy population in an endemic region will be positive for these tests due to past exposure.

Treatment

- The pentavalent antimony compound [**sodium stibogluconate** (Pentostam®)]. The daily dose is 20 mg/kg body weight, given either intravenously or intramuscularly **for 28 days.**
- Side-effects are common and include arthralgias, myalgias, raised hepatic transaminases, pancreatitis and ECG changes.

- **Amphotericin B** is very useful in the treatment of antimony-unresponsive VL

6-Cytomegalovirus

- **Cytomegalovirus** (CMV) **is the most common congenital infection** and the leading cause of hearing loss, mental retardation, retinal disease, and cerebral palsy.
- Transmission occurs **transplacentally or perinatally** through contact with cervical secretions or through breast milk. Perinatal exposure is not usually associated with disease in term infants, but preterm infants may be infected.
- When primary infection occurs in mothers during a pregnancy, the virus is transmitted to the fetus in approximately 35% of cases.
- The earlier in gestation that the primary maternal infection occurs, the more symptomatic the infant will be at birth.

Presentation

- More than 90% of infants who have congenital CMV infection **exhibit no clinical evidence of disease at birth.**
- Approximately 10% of infected infants are small for gestational age and have symptoms at birth. The characteristic signs and symptoms include:

Intrauterine growth retardation, **prematurity**, **hepatosplenomegaly** and **jaundice**, **thrombocytopenia and purpura, microcephaly (small head)and intracranial calcifications**. Other neurologic problems include **retinitis**, and **hearing abnormalities**.

- Mortality is 10% to 15% in symptomatic newborns.
- In case of perinatal CMV infection acquired during birth or from mother's milk, the majority of infants remain asymptomatic and do not exhibit sequelae.

Diagnosis.

1-Congenital CMV infection is diagnosed by detection of virus in the urine or saliva.

2- Positive CMV immunoglobulin M (IgM) serology is highly suggestive, but NOT diagnostic.

Treatment

1-There are limited options for treatment of CMV infection. Treatment is not indicated for immunocompetent persons, but is recommended for immunocompromised persons, and remains controversial for infants with symptomatic congenital infection.

2-Treatment of Congenital Infection: Trial studies in severely symptomatic newborns of the antiviral agent **ganciclovir** have shown a lack of progression of **hearing loss**.
Dose: 6 mg/kg I.V every 12 hours for 6 weeks.

3-Ganciclovir is related to aciclovir but it is more active against cytomegalovirus; it is also much more toxic (**Myelosuppression**) than aciclovir.

Febrile convulsion

1-A febrile convulsion is a fit occurring in a child (**generally between the ages of 6mths and 6yrs**), **precipitated by fever** (temp > 38 C) arising from infection outside the nervous system in a child who is otherwise neurologically normal [1-3] and in case of absence of acute electrolyte imbalance [4].

2-They occur in up to 4% of all children . The vast majority of febrile seizures are **harmless** [3].
3-Children prone to febrile seizures **are not considered to have epilepsy** (95–98% of children who have experienced febrile seizures do not go on to develop epilepsy) [3].

Etiology

1-The **etiology is unknown**. Genetic predisposition appear to be a risk factor [1,2]
2-Typically febrile seizure **occurs within the 1st 24 hour of a febrile episodes** [1] and most commonly due to acute viral respiratory infections [5].

Types of febrile seizure

1-**Simple febrile seizures** last **less than 15 minutes**, and occur **only once in a 24-hour** period [6]. The risk of subsequent epilepsy is not substantially greater than that for the general population [6].

2-If the seizure lasts **longer than 15 minutes** or **recurs within 24 hours** the seizure is referred to as a **complex or atypical febrile seizure** [6]. It signify a greater risk of later epilepsy [1,4].

Diagnosis:

Diagnosis is made by **exclusion of other causes** of symptomatic seizures like meningitis or metabolic abnormalities [4].

Treatment

1-**Control fever** : Measures to reduce elevated temperature should be initiated. Acetaminophen (or ibuprofen) and tepid sponge baths usually are helpful [1]. However, **administration of antipyretics during febrile illnesses does not prevent febrile seizures** [6].

2-Febrile seizures always are **outgrown** [7], so typically, **Long-term treatment or prophylaxis with antiepileptic drug (AED) for simple febrile seizures is not recommended** [1].

3-Oral diazepam (Valium) prophylaxis, **started at the onset of fever**, **prevents febrile seizure** [7] (oral diazepam, 0.3 mg/kg q8h , is administered for the duration of the illness (usually 2-3 days). This strategy may be useful when parental anxiety associated with febrile seizures is severe [8].

4-Patients who have **prolonged febrile seizures** can benefit from **rectal diazepam gel** given soon after the onset of a febrile seizure to prevent additional prolonged seizures.

Cardiovascular Disorders

1-Acute rheumatic fever

1-Acute rheumatic fever remains an important preventable cause of cardiac disease [1]. Acute rheumatic fever **usually affects children** (most commonly between 5 and 15 years) or young adults [2].

2-The condition is triggered by **an immune-mediated response to infection with specific strains of group A streptococci**, which have antigens that may cross-react with cardiac myosin and membrane protein. Antibodies produced against the streptococcal antigens cause inflammation in **the heart as well as the joints and skin** [2].

Table 1: criteria for the diagnosis of rheumatic fever

Major manifestations	
• Carditis • Polyarthritis • Chorea	• Erythema marginatum • Subcutaneous nodules
Minor manifestations	
• Fever • Arthralgia • Previous rheumatic fever	• Raised ESR or CRP • Leucocytosis • First-degree AV block
Plus	
• Supporting evidence of preceding streptococcal infection: recent scarlet fever, raised antistreptolysin O or other streptococcal antibody titre, positive throat culture	

(2).

Clinical features

1-Acute rheumatic fever is a multisystem disorder that usually presents with **fever**, and **joint pain**, **2–6 weeks after an episode of streptococcal pharyngitis** [1, 2].

2-The presence of either two **major criteria** or one major and two **minor criteria**, along with evidence of preceding **streptococcal infection**, confirm a diagnosis of acute rheumatic fever.
[Streptococcal antibody tests, such as the antistreptolysin O (**ASO**) titer, are the most reliable laboratory evidence of prior infection].

Management of the acute attack

1-A single dose of benzyl penicillin 1.2 million U i.m. or oral phenoxymethyl penicillin for 10 days should be given on diagnosis to eliminate any residual streptococcal infection. If the patient is penicillin-allergic, erythromycin or a cephalosporin can be used.
2-**Bed rest is important**, as it lessens joint pain and reduces cardiac workload.
3-**Aspirin:** This will usually relieve the symptoms of arthritis rapidly. The usual dose of aspirin is 100 mg/kg/24 hr divided qid PO for 3-5 days, followed by 75 mg/kg/24 hr divided qid PO for 4 wk.

4-Patients with carditis and cardiomegaly or congestive heart failure should receive **corticosteroids**.
The usual dose of prednisone is 2 mg/kg/24 hr in 4 divided doses for 2-3 wk followed by a tapering of the dose that reduces the dose by 5 mg/24 hr every 2-3 days.

5-Supportive therapies for patients with moderate-to-severe carditis include **digoxin**, **fluid** and **salt restriction**, **diuretics**, and **oxygen**.