

Prenatal and Neonatal Infections

DR Mohamed Masood



Objectives

- **PART ONE : natal and postnatal infection**
- To briefly review neonatal immunology and why neonates are so susceptible to infections
- To review the epidemiology, clinical presentation, diagnosis and treatment of the most acquired bacterial ,Viral and fungal neonatal infections.
- To review modes of infection prevention.
- **PART TWO: prenatal (intrauterine) infections**
- To review epidemiology and clinical presentation of most common congenital infections
- Antenatal and postnatal diagnosis and treatments
- Prevention of intrauterine infections

NEONATAL INFECTIONS & SEPSIS



Infection in the Newborn

- Overall < 1%
- NICU 16%
- VLBW (<2500 g) 30%
- Mortality 30%



Preventing infection

- Handwashing
 - before and after touching any baby
 - no watches, bracelets, nail varnish
 - sleeves rolled up
 - full scrub on arrival in NICU
 - 10 second wash in between
 - alcohol solutions

Why are infants, especially premature more susceptible to infections?

Neonatal Immune System

- All neonates relatively immunocompromised
- Immature and Ineffective:
 - Antibodies
 - Complement
 - Neutrophils
 - Skin / mucosal barriers

Small/premature =

- Poor antibody response
- Poor neutrophil response
- Poor complement activation
- Impaired macrophage activity
- Poor T cell function
- Reduced placental IgG

What makes a neonate's immune system immature?

Pathogen enters body

Neutrophils move in

Chemotaxis occurs

Opsonization causes phagocytosis

Monocytes kill pathogen

Normally an immune system responds to a pathogen in a specific manner, but if there are problems with any element the immune system is unable to function properly [3&6].



Antibody

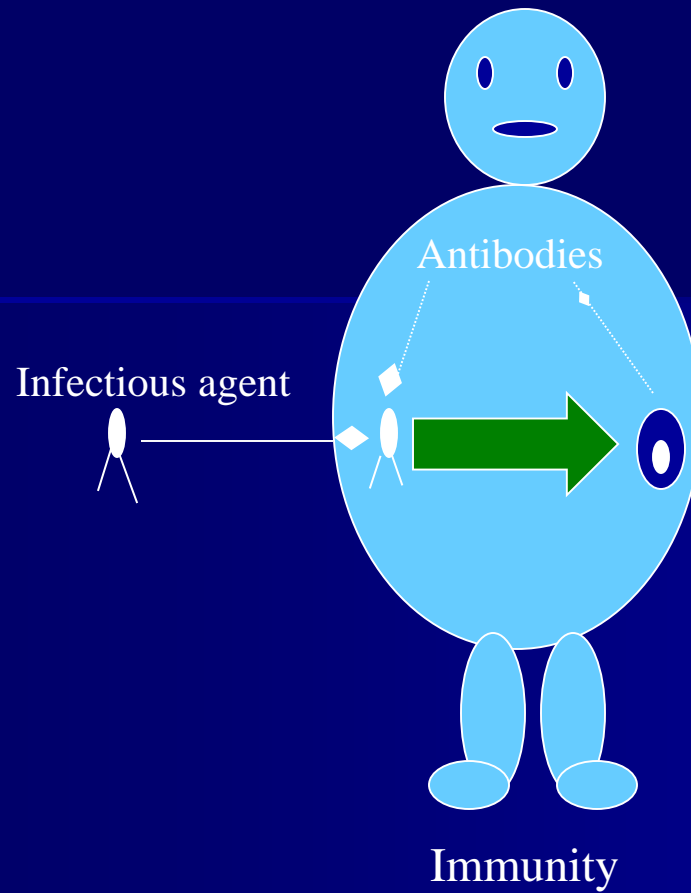
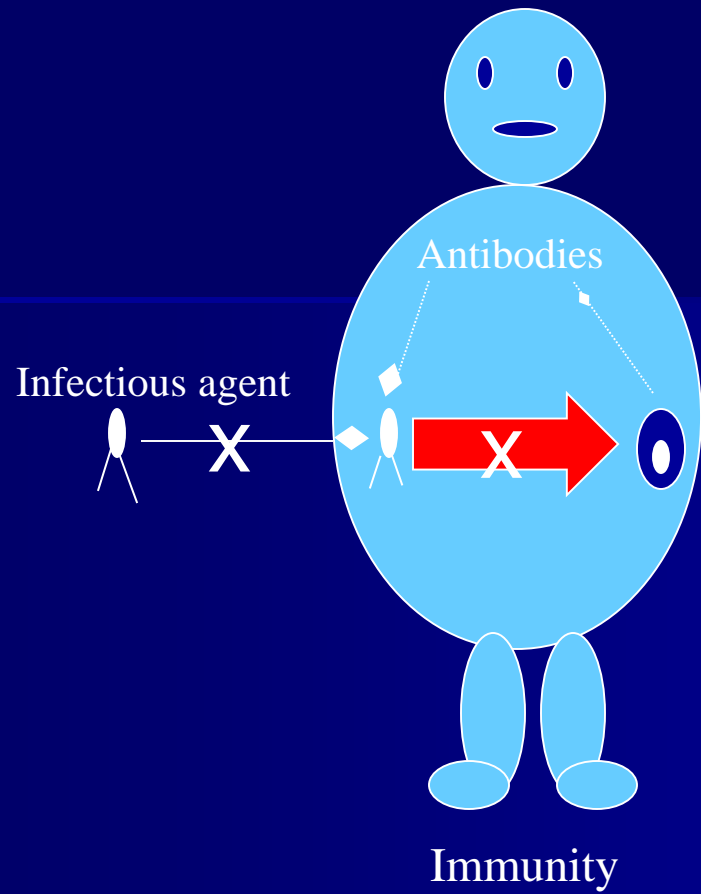


Figure 1.1 Antibodies (*anti-foreign bodies*) are produced by host while cells on contact with the invading micro-organism which is acting as an antigen (e.g. *generates antibodies*). The individual may then be immune to further attacks.

(Modified From: Roitt, I: *Essential Immunology*, 4th edition, Blackwell Scientific Publications, 1980)



No contact with infectious agents = no antibody production

Maternal Transfer of Antibodies

- Antibody transfer increases with GA
- Most during 3rd trimester
- No guarantee maternal antibodies present to the infecting organism

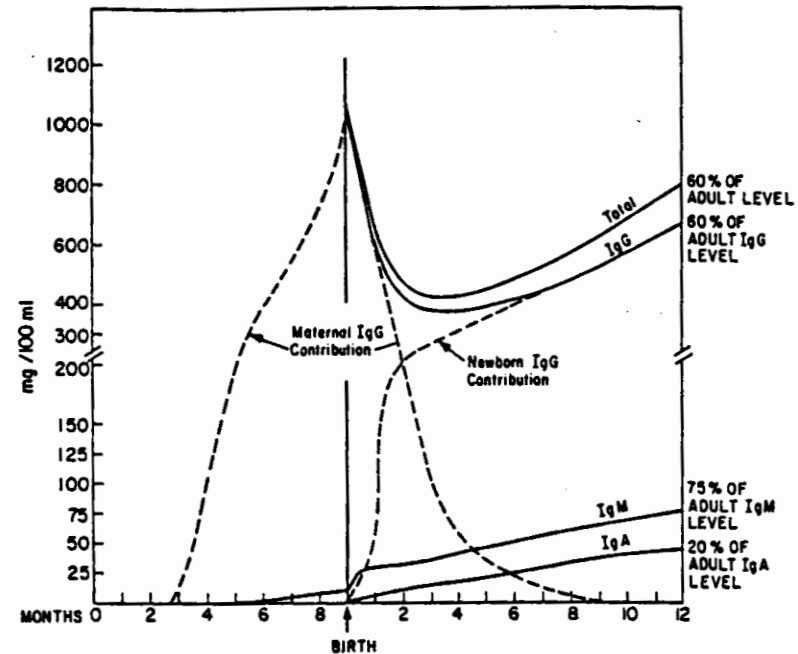
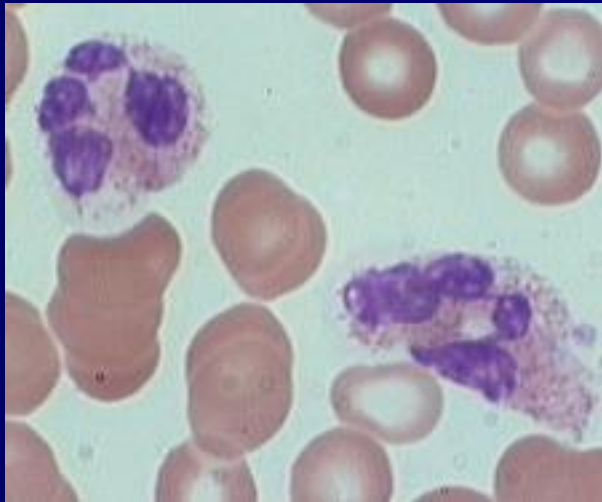


Figure 2-5. Immunoglobulin (IgG, IgM, and IgA) levels in the fetus and infant in the first year of life. The IgG of the fetus and newborn infant is solely of maternal origin. The maternal IgG disappears by the age of nine months, by which time endogenous synthesis of IgG by the infant is well established. The IgM and IgA of the neonate are entirely endogenously synthesized, since maternal IgM and IgA do not cross the placenta.

Neutrophils

Neonatal Neutrophils



■ Immature

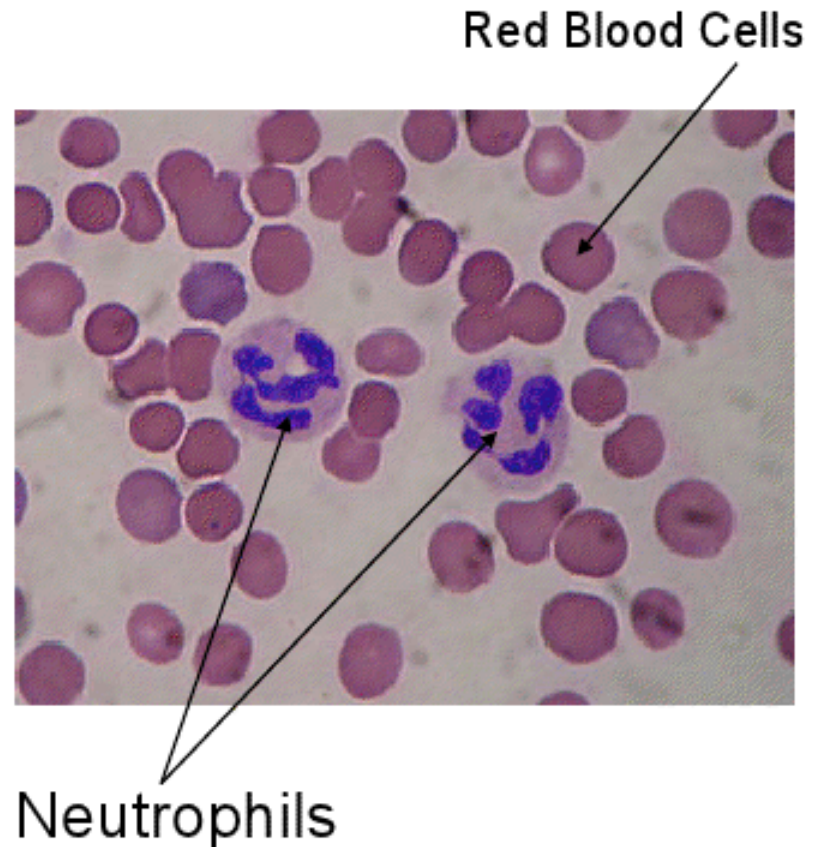
- ↓ Chemotaxis
- ↓ Deformability
- ↓ Phagocytosis
- ↓ Storage pool
 - Adults 14-fold > circulating pool
 - Neonates only 2-fold

Neutrophils: An important cell in immunity against pathogens

Neonatal neutrophils are deficient in their ability to adhere to vessel walls at site of infection [2&6].

Further release of neutrophils depletes a neonatal storage pool because the bone marrow storage of a neonate is only 20-30% of the pool in an adult [2&6].

Neonatal neutrophils have a decreased ability to “deform” & migrate into tissues [2&6].



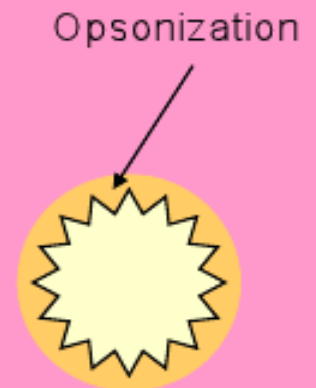
Opsonization

Opsonization is the coating of a pathogen with antibodies that makes it susceptible to phagocytosis [2&6].



Phagocytosis is the process of cells (phagocytes) engulfing, ingesting, & destroying pathogens [2&6].

Neonates have a decreased amount of opsonins (antibodies that promote opsonization) [2&6].



Neonatal Anatomic Barriers

- **Immature skin and mucosal surfaces**
 - ↓layers
 - ↓junctions between cells
 - ↓secretory IgA
- **Umbilical cord**
- **catheters, tape**

SEPSIS



Epidemiology

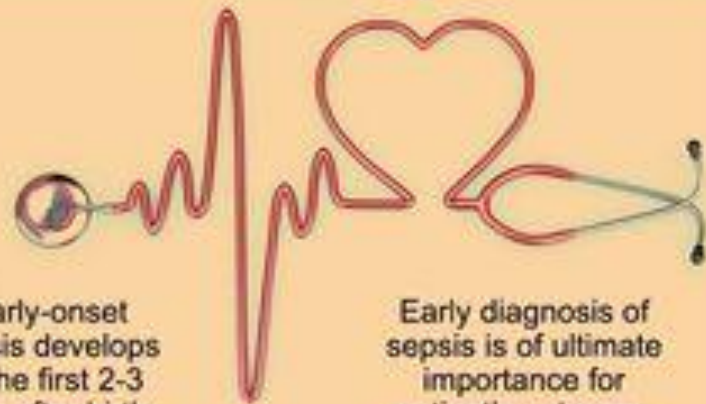
Introduction

- Neonatal sepsis is a common cause of morbidity and mortality
- Neonatal sepsis is a clinical syndrome of systemic illness accompanied by bacteremia in the first month of life

What is Neonatal Sepsis?



Neonatal sepsis is a bacterial infection in the blood.



Early-onset sepsis develops in the first 2-3 days after birth.

Early diagnosis of sepsis is of ultimate importance for patient's outcome.



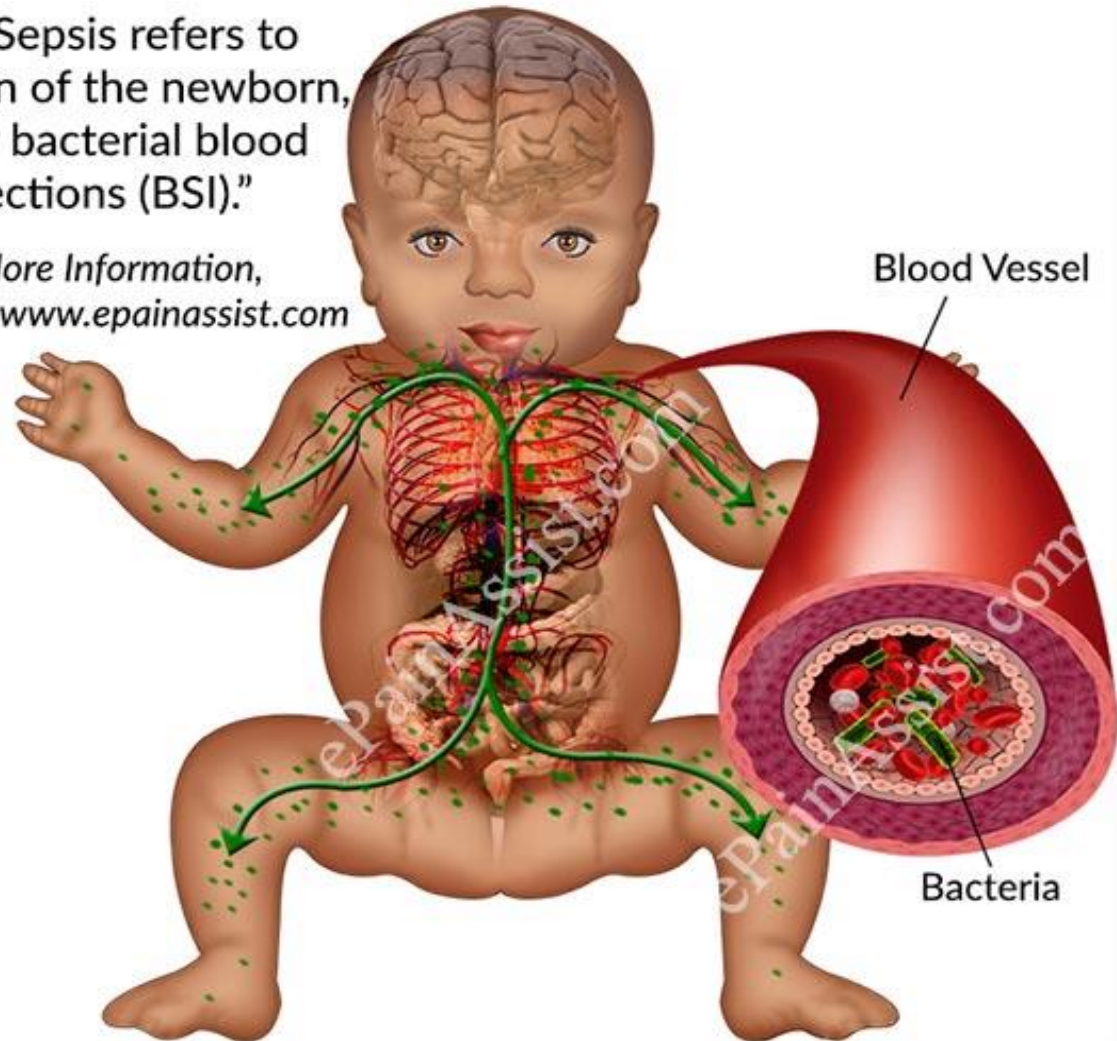
It is found in infants during the first month of life.

Late-onset sepsis develops within 3-7 days after birth.

What is Neonatal Sepsis?

“Neonatal Sepsis refers to an infection of the newborn, specifically bacterial blood stream infections (BSI).”

For More Information,
Visit: www.epainassist.com



Neonatal Sepsis: Incidence

- **2/1000 live births with culture proven sepsis**
 - Bacterial / Viral / Fungal
 - 80% infants develop bacterial sepsis
 - 20% infants perinatal acquired viral infections
 - ~ 25% of infected infants have meningitis
- **Higher rate with preterm birth**
 - 15-23/1000 preterm infants with BW < 1000g
 - 8-9/1000 preterm infants with BW 1000-2000g

Causes of 4 million newborn deaths each year

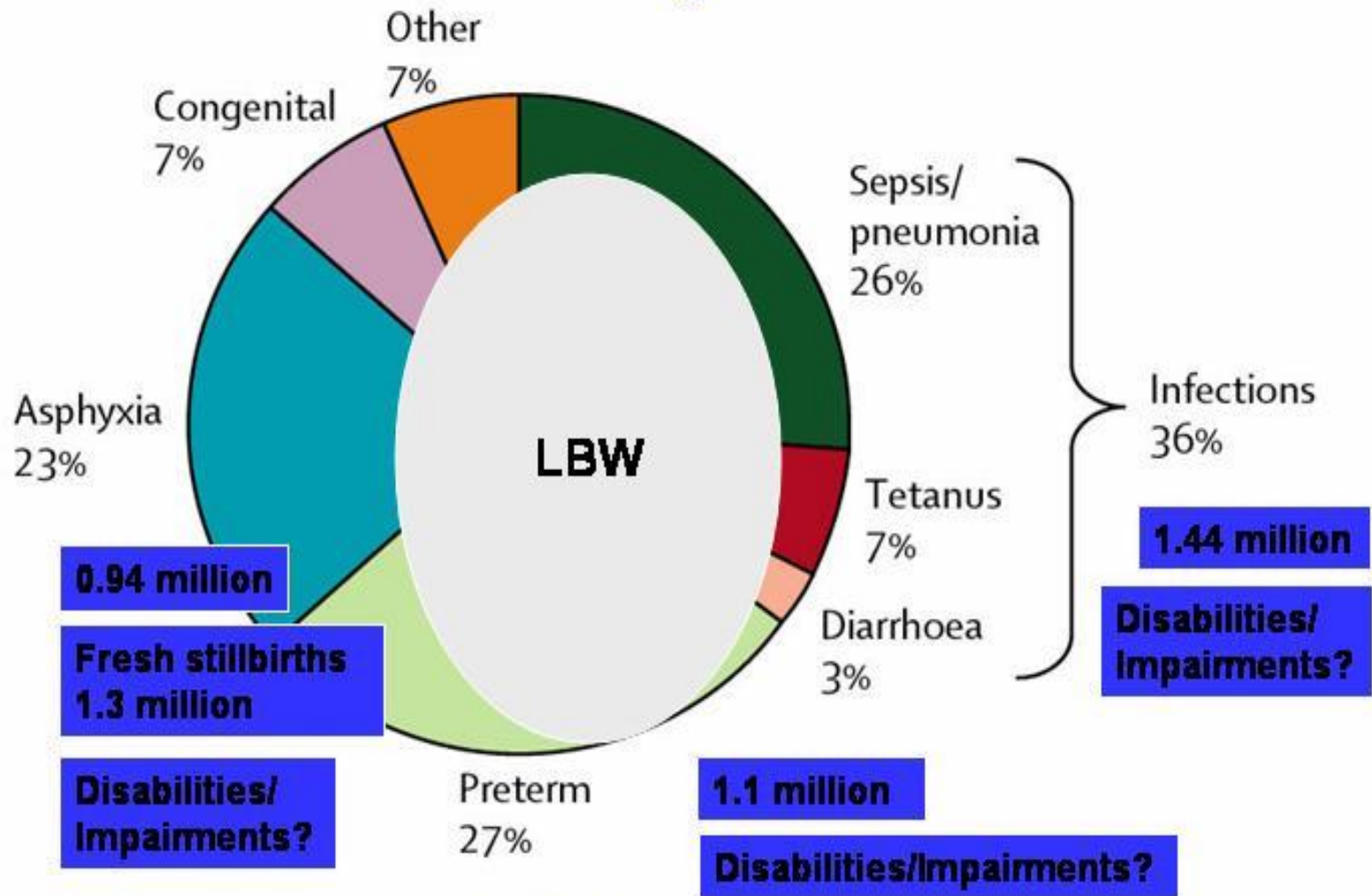
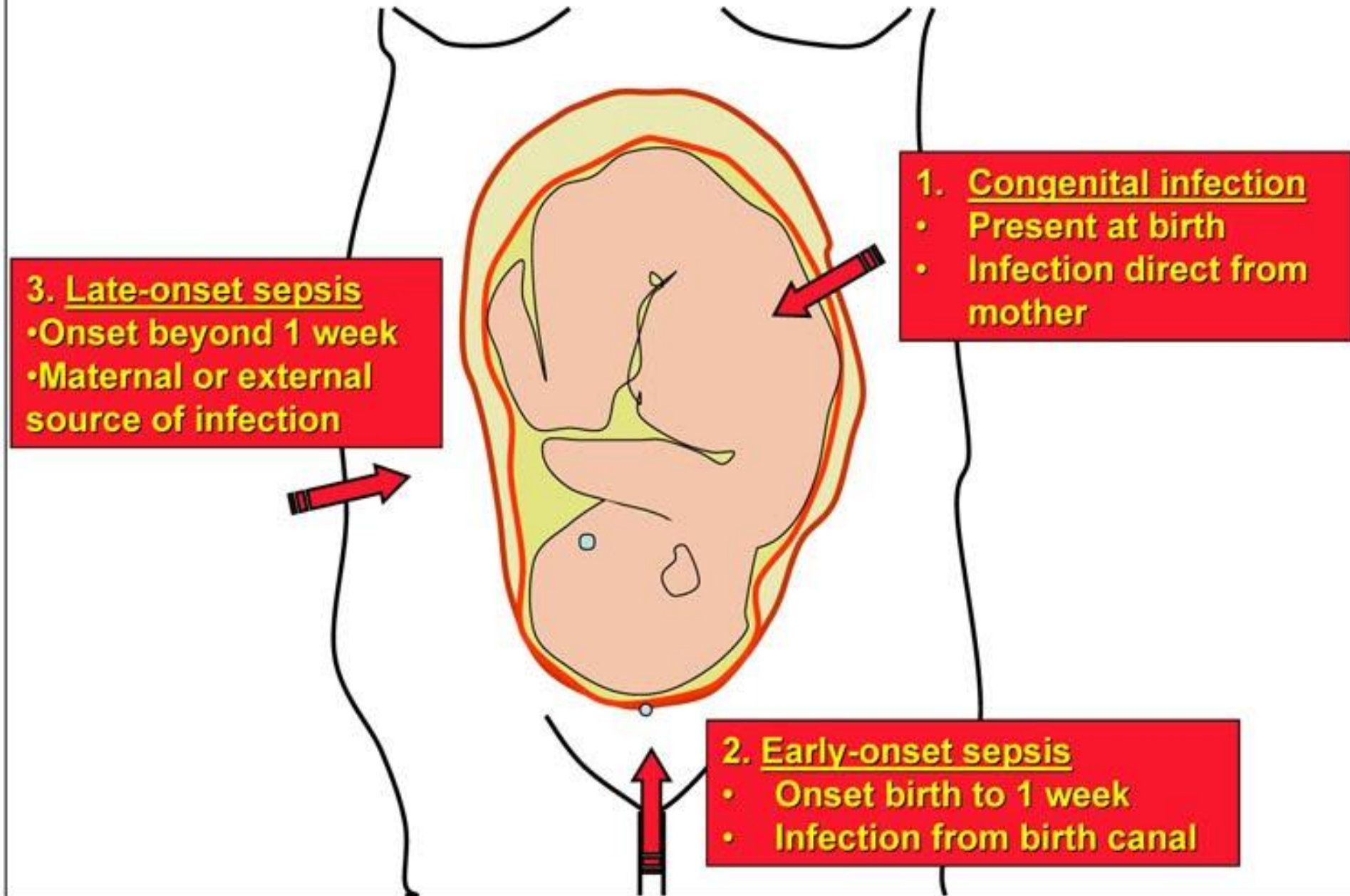


Figure 6.1

Figure 1. Causes of neonatal sepsis



pathogens can enter a neonate's body in many ways !

Pathogens can enter through the prenatal, perinatal, and postnatal periods [6].

Prenatal	Maternal Substance Abuse Premature Rupture of Membranes (>18 Hours) Maternal Infection
Perinatal	Microbial Colonization at Birth Maternal Infection Vaginal Exam of Mother
Postnatal	Invasive Catheters Endotracheal Intubation Exposure to Nosocomial Microorganisms



COMMON SITES OF INFECTION

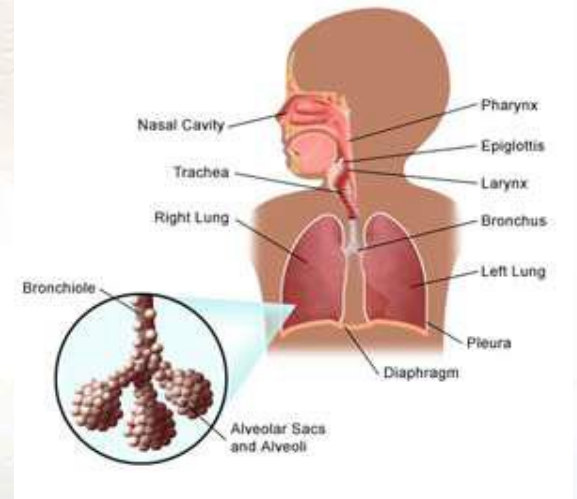


Trivial but may be serious :

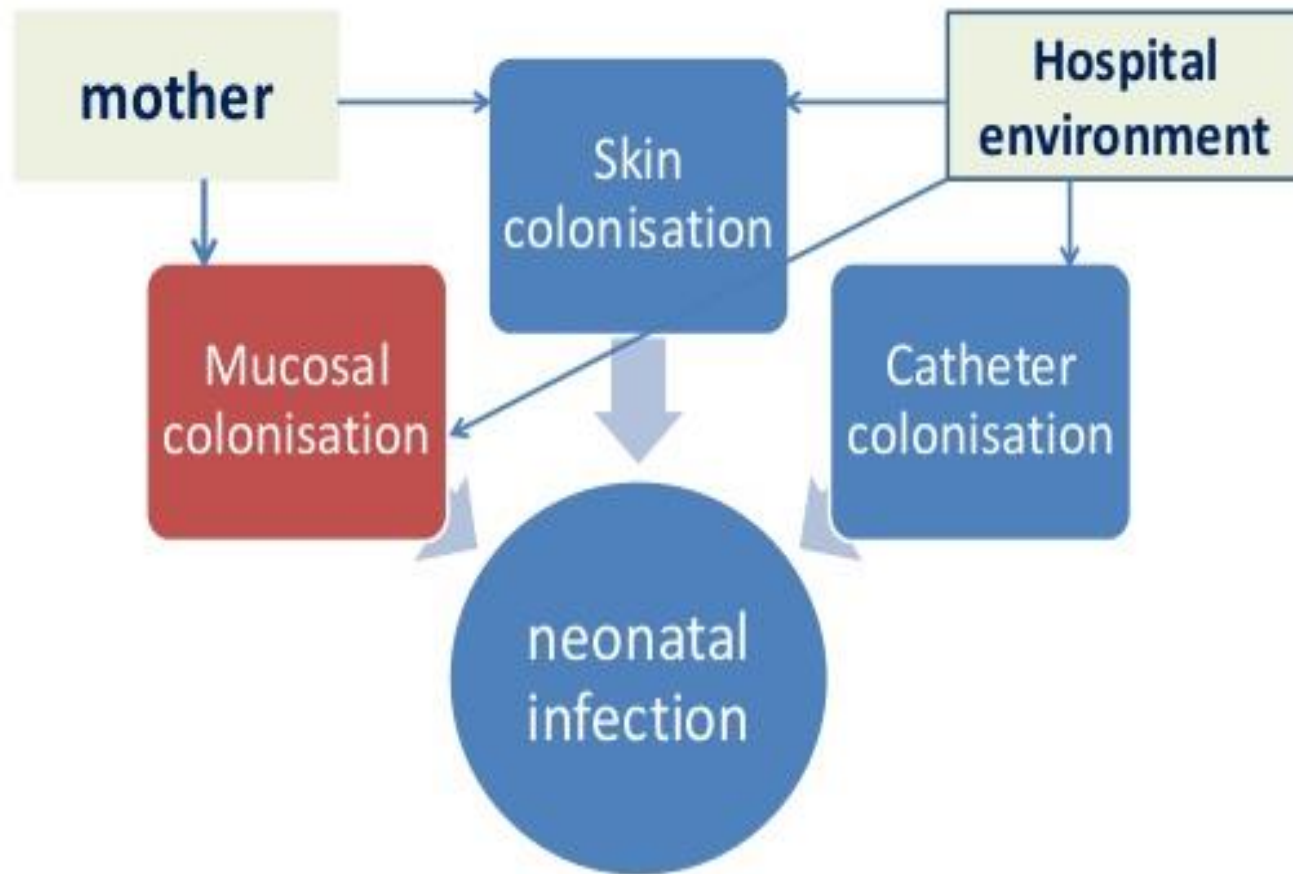
- Eyes – ophthalmia neonatorum
- Skin
- Umbilicus
- Oral thrush

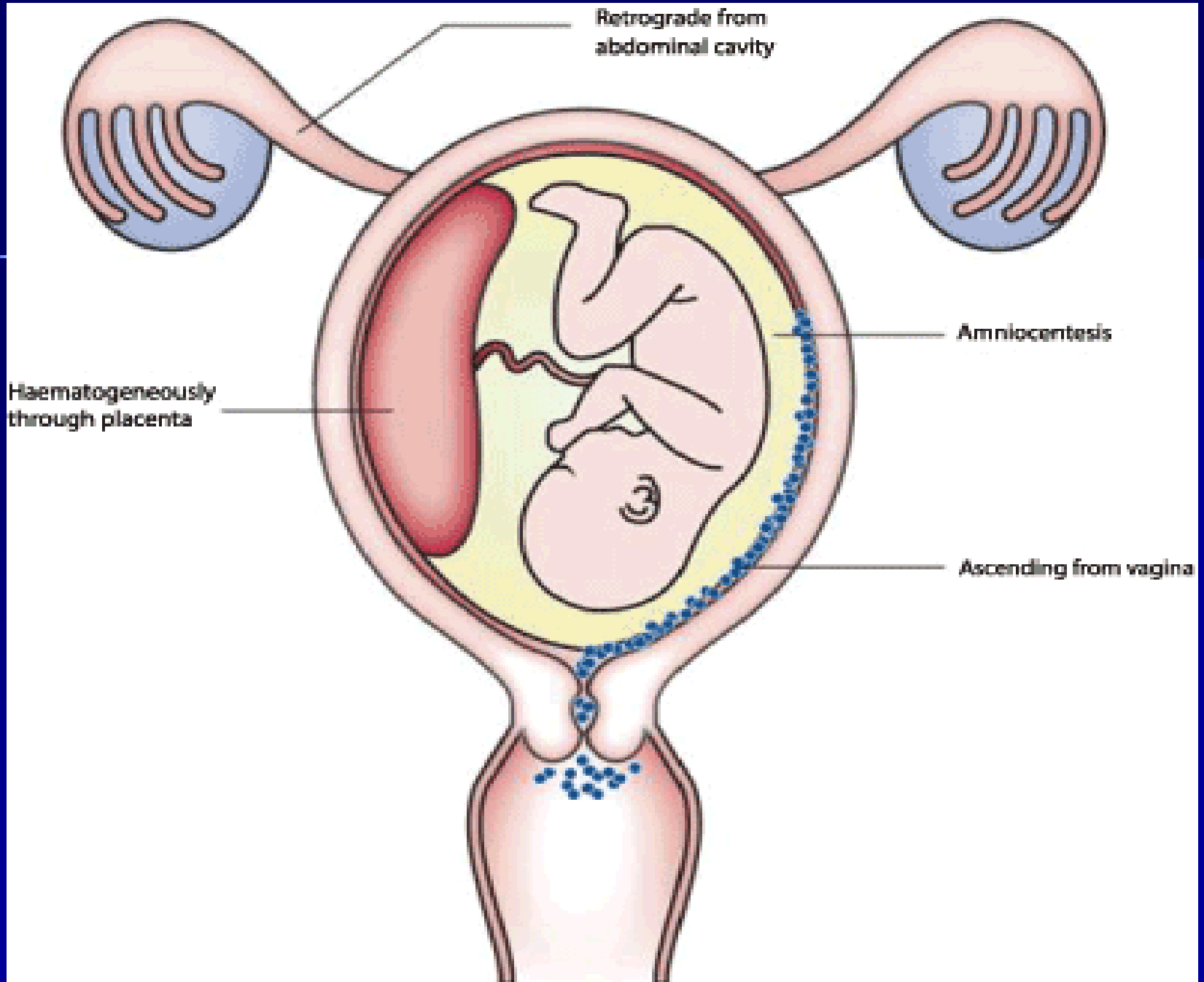
Severe systematic :

- Respiratory tract
- Septicemia
- Meningitis
- Intra – abdominal infections



Origin of sepsis in neonates





Retrograde from abdominal cavity

Amniocentesis

Haematogeneously through placenta

Ascending from vagina

More facts about Neonatal Sepsis

Neonatal Sepsis affects approximately 2 infants per 1000 births with a higher incidence in premature & low birth weight infants [2].



There are two types of Neonatal Sepsis:

Early Onset

Late Onset



* This tutorial will focus primarily on Early Onset Sepsis



Neonatal Bacterial Sepsis: Disease Patterns

- **Early Onset Neonatal Sepsis (EONS)**
 - Fulminant , multi-system illness
 - 3 days old (72 hrs)
 - Obstetrical complications
 - Prematurity
 - High mortality, 5-50%
- **Late Onset Neonatal Sepsis (LONS)**
 - Sepsis or meningitis
 - >3 days(>72hrs) to 3 months old
 - Lower mortality, 2-6%

Etiology: EOS

- **Early Onset Sepsis (EOS):**
 - Group B Streptococcus (GBS)
 - E. Coli
 - Listeria monocytogenes
 - Streptococcus species ie. Viridans
- Due to maternal or perinatal factors

Risk Factors

- Prematurity
- Low birthweight
- ROM \geq 18 hours
- Maternal peripartum fever or infection
- Resuscitation at birth
- Multiple gestation
- Male sex

Neonatal Sepsis

Major Risk Factors

- Ruptured membranes > 24 hrs.
- Maternal Fever (100.4°F(38°C))
- Chorionamnionitis
- Sustained fetal heart rate >160/min
- Multiple obstetric procedures

Minor Risk Factors

- Ruptured membranes > 12 hrs.
- Foul smelling liquor
- Maternal Fever > 99.5°F (37.5°C)
- Low APGAR < 5 at 1 min,
< 7 at 5 min
- Prematurity
- Multiple gestation

**Presence of 1 major or 2 minor risk factors ->
High Risk of Sepsis**

Etiology: Viral Sepsis

■ Congenital

- Enteroviruses (ie. Coxsackievirus A & B)
- Herpes Simplex Virus
- TORCH infections ie. CMV, Toxoplasmosis

■ Acquired

- HIV
- Varicella
- Respiratory syncytial virus

- Can be either early or late onset sepsis

Clinical Signs and Symptoms

- Lethargy
- Hypo/hyperthermia
- Feeding intolerance
- Jaundice
- Abdominal distention
- Vomiting
- Apnea

1) Not breathing well

2) Not feeding well

3) Not looking well

Symptoms of Neonatal Sepsis

The symptoms of neonatal sepsis are not concrete and vary widely [9].

Tachypnea



Heart Rate Changes



Feeding difficulties



Difficulty Breathing

Temperature Instability

Irritability



Jaundice

Why are symptoms so broad?



Appearance

Lethargic

Mottled skin

Poor perfusion

**Temperature instability (not necessarily
high fever, but fever is more specific)**

Jaundice

;- Ominous late signs

Apnea

Seizures

Hypotension/ shock

Differential Diagnosis

- Respiratory
- Cardiac
- CNS
- GI
- Inborn errors of metabolism
- Hematologic

Risk Factors

Chorioamnionitis^a or
PROM ≥ 18 h or IAP
indicated but Inadequate

Diagnostic Tests

Blood culture at birth
WBC/Diff \pm CRP
at age 6-12 h

Antibiotics

Treat with broad-
spectrum antibiotics

Management

Blood culture positive

Blood culture negative
Infant remains well
Lab data abnormal

Blood culture negative
Infant remains well
Lab data normal

Continue antibiotics
Lumbar puncture^b

Continue antibiotics if mother
received antibiotics during
labor and delivery

Discontinue
antibiotics

Some specific sites of bacterial infection (Fig. 42.2)



Neonatal meningitis

Most common organism – group B streptococcus; then gram-negative organisms
Rare – *Listeria monocytogenes*
High mortality and morbidity (hearing loss, hydrocephalus and developmental delay)

Conjunctivitis

- Sticky eyes
- Purulent conjunctivitis with swelling of eyelids

Pneumonia

Presents with respiratory distress
Diagnosed on CXR and evidence of infection

Umbilical infection

Slight redness around umbilicus is common. Red flare around umbilicus needs antibiotic therapy

Urinary tract infection

Non-specific presentation. Can only be diagnosed if satisfactory urine sample has been obtained

Osteomyelitis and septic arthritis

See Chapter 60

Abscess

Localized swelling – red, warm, often fluctuant
May be at site of intravenous infusion/
extravasation

Skin – generalized

- Bullous impetigo
- Staphylococcal scalded skin syndrome (SSSS)

Paronychia

Systemic antibiotics are given

Fig. 42.2 Some specific sites of bacterial infection.

Late Onset Neonatal Sepsis

Neonatal Sepsis

Risk factors for Late onset sepsis (LOS)

- Prolonged hospitalization
- Prematurity
- LBW
- Previous antibiotic use
- Invasive procedures
- Presence of foreign material (ET Tubes/
catheters)
- Lack of disposables
- Over crowding / understaffing



Septic workup

- Goals of workup
 - Recover organism
 - Determine specific antibiotics
 - Determine antibiotic doses
 - Determine length of therapy

Septic workup

- **CBC with PBS**
 - **WBC up, down , or normal**
 - **Left shift helpful but may be delayed**
 - **Unexplained thrombocytopenia**
- **PT/PTT abnormalities**
- **Blood sugar may be high or low-
change in pattern**
- **ESR and CRP.**
- **Gastric aspirate or ET aspirate.**
- **CXR**

Sepsis Work-Up

- Blood cultures (x 2 due to low sensitivity)
- Urine cultures
- Lumbar puncture
- Tracheal aspirates
- CBC with differential

treatment

- **Selection of antibiotics based on:**
 - **Age of onset**
 - **Location (home vs hospital)**
 - **Maternal history**
 - **Known colonization**
 - **Epidemic situations**
 - **Etc.**

Empiric Antibiotic Therapy

■ EOS

- Penicillin and Aminoglycoside
- Ampicillin and Gentamicin

■ LOS

- Vancomycin and Aminoglycoside
- Vancomycin and Gentamicin

treatment

- **General supportive measures**
 - **Assisted ventilation and /or oxygen as needed**
 - **IV and possibly arterial access**
 - **NPO, NG suction if needed**
 - **Volume support, pressure**
 - **Transfuse if indicated**
 - **Fresh F.Plasma if clotting disorders**
 - **Thermal regulation / support**

Neonatal Nosocomial Infections

Skin infections

- The new born may develop a variety of rashes associated with both systemic and focal bacterial disease.
- Responsible organisms include all of the usual causes of early neonatal sepsis.
- Localized infections can arise in any site of traumatized skin or blood extraction sites

cellulitis

- Usually occurs at sites of trauma or IV lines
- clinically there is localized erythema ,swelling and/or drainage.
- In healthy term babies can be treated with careful washing and local antiseptic with antibiotic ointment.
- Cellulitis at sites of IV access or venipuncture in premature infants must be treated more aggressive due to the risk of systemic spread ,blood culture should be taken and iv antibiotics should be started

pustulosis

- Infectious pustules is usually caused by *S. aureus*
- Should be distinguished from the benign neonatal rash ;erythema toxicum and transient pustular melanosis
- Most commonly in the axellae,groin and periumbilical area
- A few lesions in healthy infants can be treated topical antibiotics







Cont.

- more extensive lesions , systemic illness or pustulosis in premature babies requires IV therapy with nafcillin or cloxacillin
- Some strains of *S. aureus* produce toxins that can cause **bullous lesions** or **scalded skin syndrome**

OPHTHALMIA NEONATORUM

- Neonatal conjunctivitis in the first month of life

PREDISPOSING FACTORS

- Organisms in vagina shed during delivery
- Premature rupture of membranes
- Long delivery
- Few tears and low levels of IgA
- Trauma to epithelial barrier
- Prophylaxis (antibiotics, silver nitrate)

OPHTHALMIA NEONATORUM



National Medical Slide/Courtesy Medical Stock Photo, Inc.

OPHTHALMIA NEONATORUM/ CONJUNCTIVITIS

Ophthalmia neonatorum is defined as inflammation of conjunctiva during first month of life.

Causes

- Chlamydia trachomatis (oculogenitalis)
- Other bacterial causes : gonococcus (rare), staphylococcus, pseudomonas, etc.
- Chemical – silver nitrate
- Viral : herpes simplex (type II)



MODE OF INFECTION

- Infection occurs mostly during delivery by contaminated vaginal discharge.
- It is more likely on face or breech delivery.
- During neonatal period, there may be direct contamination from other sites of infection or by chemical.



CLINICAL FEATURES

- It varies
- the discharge may be watery, mucopurulent to frank purulent in one or both eyes.
- The eyelids may be sticky or markedly swollen.
- Cornea may be involved in severe cases.



INVESTIGATIONS

The discharge is taken for –

(a) gram stain smear

(b) culture and sensitivity

(c) scraping material from lower conjunctiva for

Giemsa staining and also culture in suspected chlamydial infection (d) culture in special viral media for suspected herpes simplex infection.

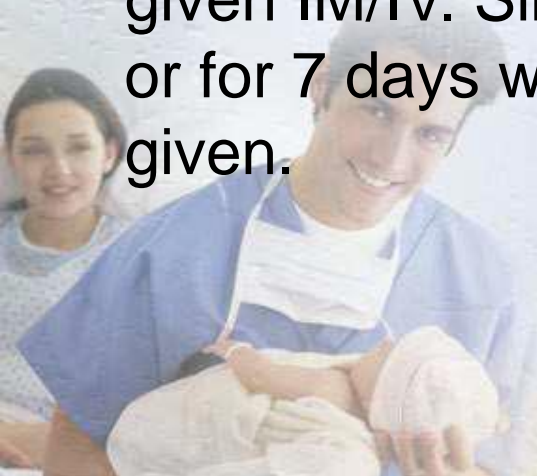


TREATMENT

Prophylaxis : 1% silver nitrate solution (1-2 drops to each eye), 0.5% erythromycin ophthalmic ointment, 2.5% povidone iodine solution is administered within 1 hour of birth and is continued for few days.

Treatment depend upon the specific aetiology.

- Gonococcal – infant is isolated during the first 24 hours of treatment. Eyes are irrigated with sterile isotonic saline every 1-2 hours until clear. In severe and culture positive cases systemic ceftriaxone 50 mg/kg/q 12 h is given IM/IV. Single dose in infant without dissemination or for 7 days when there is dissemination, is usually given.



- Chlamydia – erythromycin suspension 40 mg/kg daily orally divided into 4 doses for 14 days is given to prevent systemic infection. Topical treatment alone is ineffective.

- Herpes simplex – the infant is isolated. Systemic therapy with acyclovir 20 mg/ kg every 8 hours for 2 weeks is given IV. Topical use of 0.1% iododoxuridine ointment 5 times a day for 10 days is used.



PROGNOSIS & PREVENTION

Prognosis

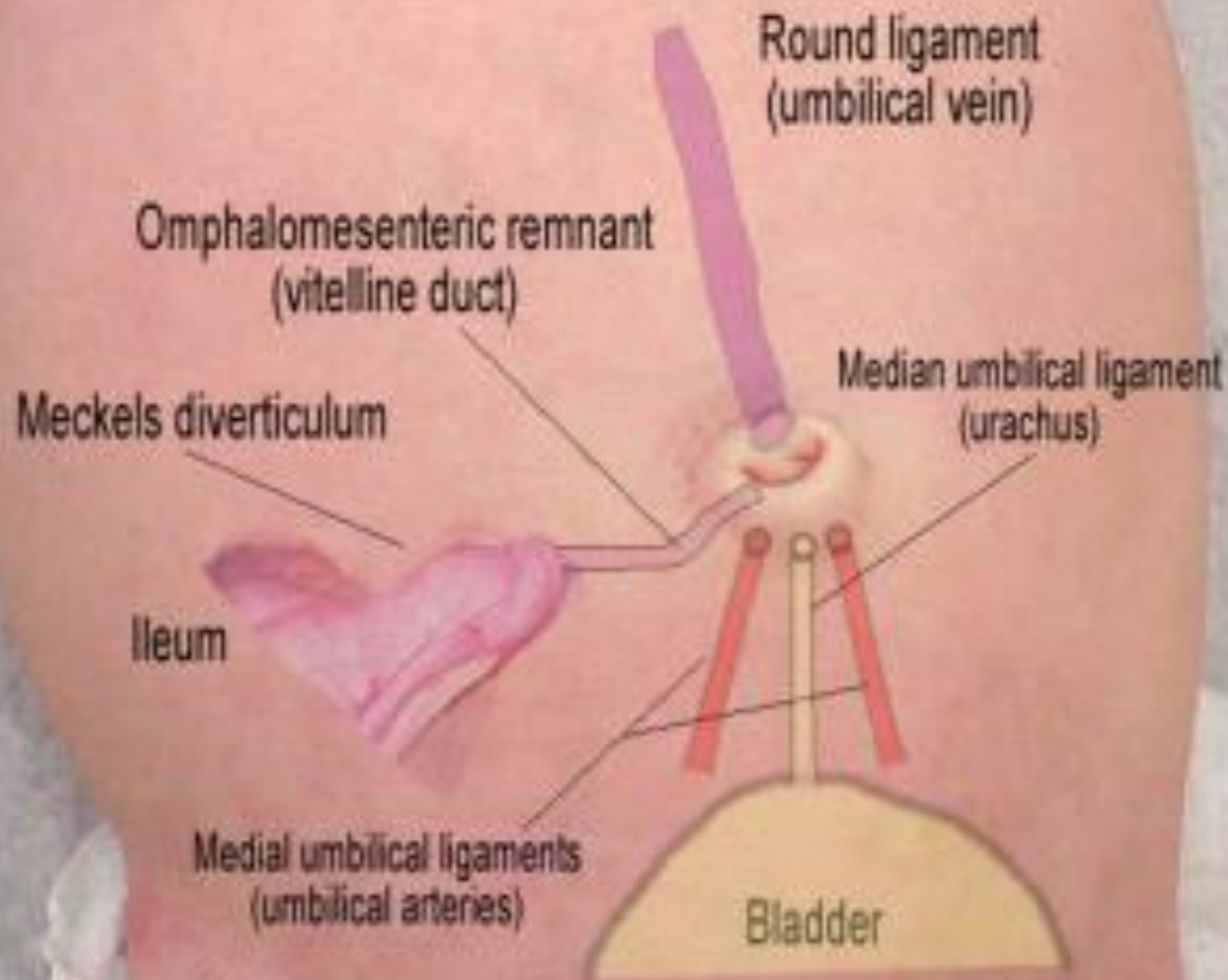
- It is favorable to most cases except in neglected cases with rare gonococcal infection. Fortunately, effective methods of prophylaxis and treatment have almost eliminated the risk of blindness.

Prevention

- Any suspicious vaginal discharge during the antenatal period should be treated and the most meticulous obstetric asepsis is maintained at birth. The newborn baby's closed lids should be thoroughly cleansed and dried.

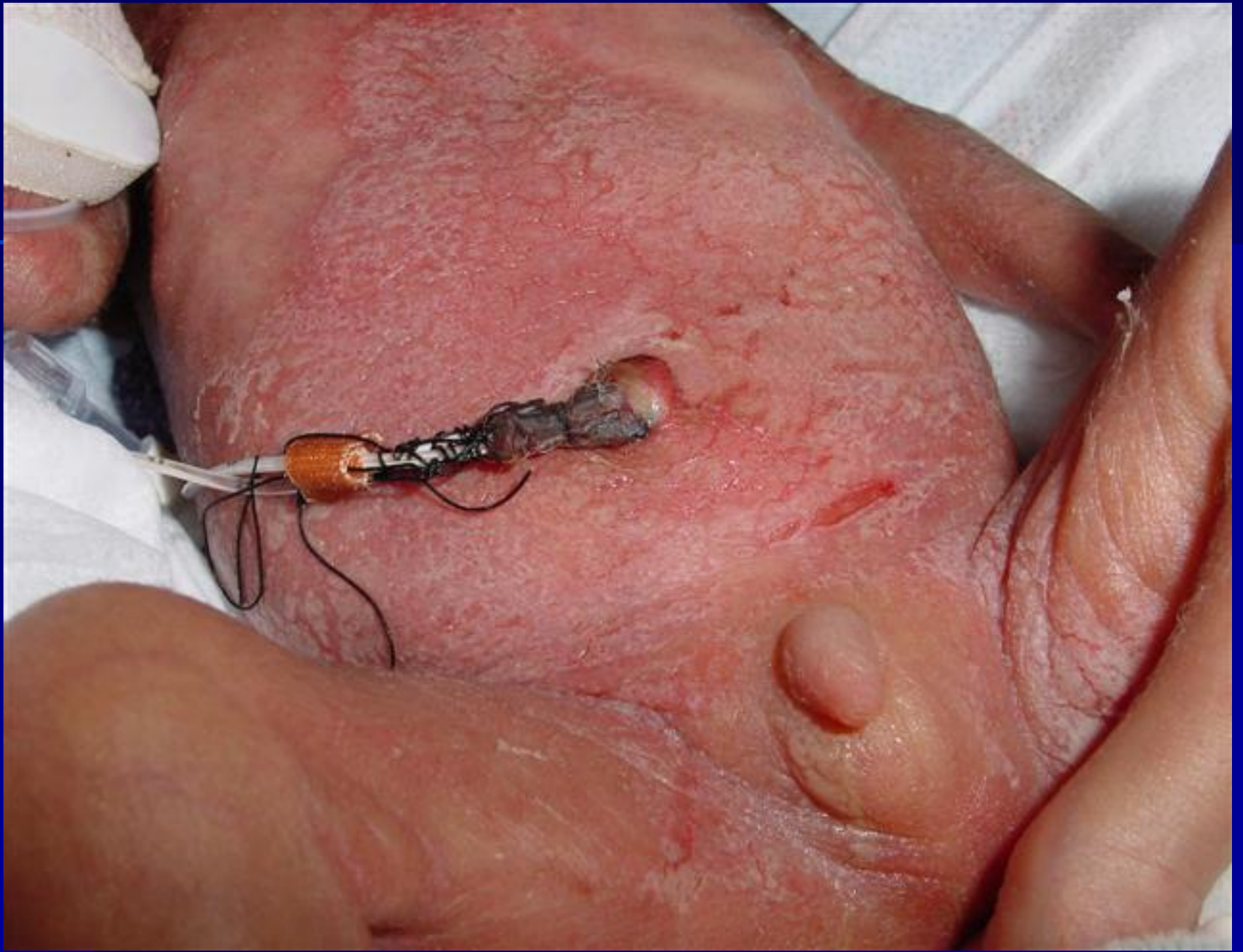


OMPHALITIS



R. Mankov 2002





Introduction

- Omphalitis is an infection of the umbilical stump.
- It typically present as a superficial cellulitis i.e. as a red 'flare' in the periumbilical skin.
- The cellulitis may progress rapidly with potentially serious consequences including systemic disease etc..
- Omphalitis is predominantly a disease of the Neonates.

- **Omphalitis** occasionally manifests from an underlying Immunologic disorder. These infants are subsequently diagnosed with **Leukocyte adhesion deficiency**, a rare disorder with AR pattern of inheritance. These infants present with the following; Leukocytosis, Delayed separation of the umbilical cord and recurrent infections.
- **Omphalitis** may also be the initial manifestation of Neutropenia in neonates i.e. NN Alloimmune Neutropenia. Affected infants present with cutaneous infections, sepsis, pneumonia e.t.c.
- Rarely, an anatomic abnormality may be present such as a patent urachus or patent omphalomesenteric duct.



Clinical Features

- In term infant the, mean age at onset is 5-9 days.
- In preterm infants, the mean age at onset is 3-5 days.
- Patient present with redness and swelling (cellulitis) around the umbilicus.
- Purulent or mal odorous discharge from the umbilicus.
- Baby is highly irritable.
- The cellulitis is rapidly progressive and may lead to necrotizing fasciitis.
- Necrotizing fasciitis is characterized by abdominal distension, fever and tachycardia.
- Despite the illness, most of the neonates at presentation have good appetite and continue to suck.



Baby O.T.with extensive local disease & systemic disease



❖ Antimicrobial therapy

- As with anti microbial therapy, local antibiotic sensitivity patterns is considered.
- **CLOXACILLIN + GENTAMICIN + FLAGYL**
OR
CEPHALOSPORIN + GENTAMICIN + FLAGYL
forms the usual antimicrobial combination.
- **Surgical care**

Prevention

- Good Ante natal care.
- Supervised aseptic delivery.
- Cord care with antimicrobial agents to decrease bacterial colonization. E.g. Methylated spirit (an alcohol) applied several times a day until cord separation. Others agents are povidone iodine, silver sulfadiazine & bacitracin ointment.
- Discourage harmful practices e.g. **application of cow dung & hot water fermentation to the cord.**

Neonatal Tetanus

- Is caused by the effect of a neurotoxin produced by anaerobic bacterium *Clostridium tetani*.
- Infection can occur by invasion of the umbilical cord due to unsanitary birth or cord care practices
- WHO estimates 59,000 deaths worldwide from tetanus neonatorum in 2008







- Infected infants develop hypertonia, convulsions and muscle spasms, include trismus and inability to feed.

- **Treatment include**

- 1-tetanus toxoid (500u IM)

- 2-Penicillin G iv for 14days

- 3-Sedation and muscle relaxants

- 4-Ventilation supports

candidial inection

DEFINITION:

It is a fungus infection of the skin and mucous membrane of the mouth characterized by white patches, resembling milk curds.

ETIOLOGY:

Candida albicans infection is due to:

- inadequate sterilization of teats and bottles.
- from mothers' breast or the attendant's hand.
- Newborns are infected during passage in birth canal.
- The long use of antibiotic therapy.
- Infant's auto-infection when he has candida diaper dermatitis and he touches the diaper area then puts his hands into his mouth.



NURSING MANAGEMENT:

- 👄 Apply the ordered medication (nystatin) after feedings. Distribute it over the surface of the oral mucosa and tongue with syringe or an applicator. The remainder of the dose is deposited in the mouth to be swallowed by the infant to treat any lesions in pharynx and esophagus.
- 👄 Use disposable applicator or sterilize it if not disposable.
- 👄 Absolute cleanliness of all articles, which enter the infant's mouth, such as, rubber nipple, pacifier, teats, mother's nipple and infant toys.



Hepatitis B

- Transmission is vertical specially in the third trimester in acute infection

HBsAg positive indicate chronic disease and risk of transmission to the fetus

HBeAg indicate high infectivity

**** The baby should be given Hepatitis B immunoglobulin at birth and an active immunization and repeated at 3,6 months.**

Cesarean section or breast feeding is unlikely to alter the incidence of neonatal infection

Human immunodeficiency virus

25% of infants born to HIV infected mothers will become infected with HIV

**Vertical transmission is 13-30% and the rest is through the birth canal

Cesarean section lower the transmission rate by two third in patients with no therapy

**If ROM cesarean section within 4 hours is advised to protect the fetus

AZT (Zidovudine) that decrease the viral load during ante partum, intrapartum, and neonatal period can reduce the risk of fetal infection by two thirds in mildly symptomatic ladies

HIV

Avoidance of breast feeding reduce the risk of transmission

Special care during labour and in the operating room should be taken and needle brick prophylaxis when handling the infected patient

Newborn is given I.V or oral AZT

ZDV Dosing in the Perinatally HIV-Exposed Infant

- Administration of neonatal ZDV
 - Oral: 2mg/kg/dose every 6 hours for 6 weeks
 - Give first dose as soon as possible after delivery: within 6–12 hours
 - IV dose for full-term infant is 1.5 mg/kg every 6 hours
 - Dose is adjusted for preterm infants
- Consult a pediatric HIV specialist
 - For ZDV dosing for premature infants
 - For additional ARV drugs for prophylaxis in infants





INFECTIONS DURING PREGNANCY

DR Mohamed Masood

Case scenario

- You are taking care of a term newborn male with **birth weight/length <10th %ile**. Physical exam is normal except for a slightly **enlarged liver span**. A CBC is significant for **low platelets**.
- What, if anything, do you worry about?

INTRODUCTION

Intrauterine infections:-

- An important cause of still births and neonatal morbidity
- Many diseases go undiagnosed
- Appropriate treatment can prevent morbidity and mortality
- 1971 Andres Nahmias proposed acronym TORCH
- 1975 Harold Fuerst add Syphilis to the acronym

- mostly viral
- previously known as (TORCH) infections:
 - Toxoplasmosis,
 - Other(syphilis)
 - Rubella,
 - CMV
 - Herpes(and Hepatitis)
- the first four are acquired antenatal, herpes and hepatitis usually perinatal.
- The term TORCH is now obsolete as other agents are important (e.g. HIV).
- Most fetuses if infected during the first trimester will suffer from a syndrome of congenital malformation.

Infections in the mother

- ▶ “CHEAP TORCHES” is an acronym for a special group of infections that can affect the developing baby during pregnancy. CHEAP TORCHES stands for the following:
 - ▶ C: Chickenpox and shingles
 - H: Hepatitis B, C, D, E
 - E: Enteroviruses, a group of viruses including poliovirus
 - A: AIDS
 - P: Parvovirus B19, also known as fifth disease
- ▶ T: Toxoplasmosis
- O: Other infections such as group B streptococcus, listeria, candida
- R: Rubella
- C: Cytomegalovirus
- H: Herpes simplex virus
- E: Everything else sexually transmitted such as gonorrhea and chlamydia
- S: Syphilis



- **ACRONYM: TORCHES CLAP**

- T**oxoplasmosis

- R**ubella

- C**MV

- H**erpes simplex

- E**nterovirus

- S**yphilis

- C**hickenpox

- L**yme disease

- A**IDS

- P**arvovirus B19

- Latest addition: Zika virus

Congenital, Perinatal, and Neonatal Viral Infections

TORCHS

Intrauterine Viral Infections

Rubella

Cytomegalovirus (CMV)

Parvovirus B19

Varicella-Zoster (VZV)

Enteroviruses

HIV

HTLV-1

Hepatitis C

Hepatitis B

Lassa Fever

Japanese Encephalitis

Perinatal and Neonatal Infections

Human Herpes Simplex

VZV

Enteroviruses

HIV

Hepatitis B

Hepatitis C

HTLV-1

CONGENITAL INFECTION

Manifestations

- Growth retardation
- Congenital malformation
- Fetal loss



Rubella
CMV
HIV
Toxoplasma
T. pallidum
Parvovirus
VZV

PERINATAL INFECTION

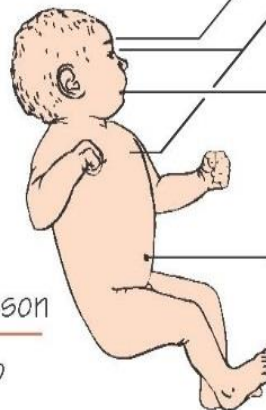
Manifestations

- Meningitis
- Septicaemia
- Pneumonia
- Preterm labour

POSTNATAL INFECTION

Manifestations

- Meningitis
- Septicaemia
- Conjunctivitis
- Pneumonitis



N. gonorrhoeae

Chlamydia

Breast milk

HIV

CMV

Person-to-person

Group B strep

Listeria

E. coli

Umbilicus

Staphylococci

Tetanus

Gonococcus

Chlamydia

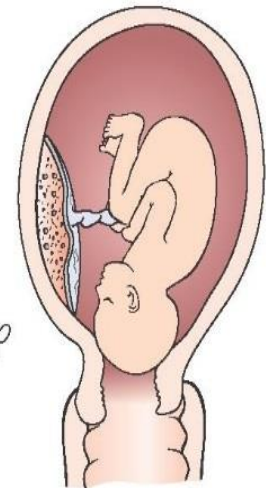
HSV

VZV

Group B strep

E. coli

Listeria



Index of Suspicion

- When do you think of congenital infections?
 - IUGR infants
 - HSM
 - Thrombocytopenia
 - Unusual rash
 - Concerning maternal history
 - Microcephaly
 - “Classic” findings of any specific infection

TOXOPLASMOSIS



What causes Toxoplasmosis?

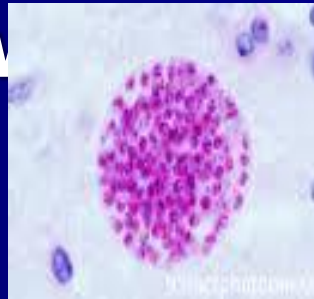
- The protozoan *Toxoplasma gondii*, is a coccidian, obligate, intracellular parasite responsible for zoonotic infections in man and other mammals

TOXOPLASMOSIS

- **Causative agent:** by protozoan toxoplasma gondi.
- **Risk of fetal infection:**
 - First Trimester - 15 % (less incidence of fetal infection but serious disease is most common).
 - Second Trimester - 25 % .Third Trimester - 65 % (but almost 90% of newborns are without clinical signs of disease).
- 40% of fetuses are affected if the mother has the illness.
- the earlier in pregnancy the more damage.
- **Maternal symptoms:** usually asymptomatic, fever, rash & eosinophilia If symptomatic (the CNS prognosis is poor).

Congenital Toxoplasmosis

- Congenital transmission happens when the mother is exposed to infection by **Toxoplasma gondii** for first time while she is pregnant.



- The effect on the foetus depends on the time of infection during

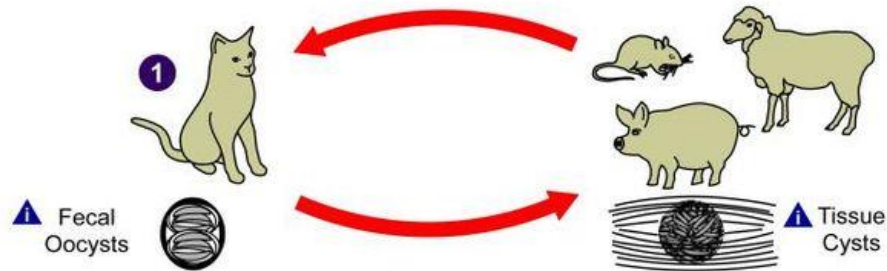
Host

- **Definitive host** (final host):-
Only Cat
- **Accidental host** (Non specific host) :-All mammals including **Man**, Farm animals ,Rat , Mice

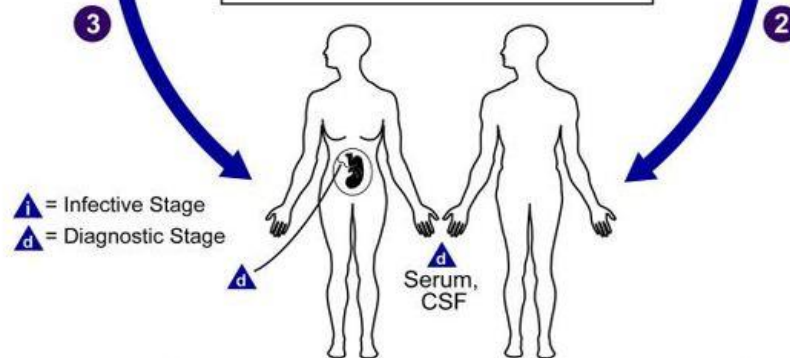
■

Toxoplasmosis

(*Toxoplasma gondii*)



Both oocysts and tissue cysts transform into tachyzoites shortly after ingestion. Tachyzoites localize in neural and muscle tissue and develop into tissue cyst bradyzoites. If a pregnant woman becomes infected, tachyzoites can infect the fetus via the bloodstream.



Diagnostic Stage

- 1) Serological diagnosis.
- or
- 2) Direct identification of the parasite from peripheral blood, amniotic fluid, or in tissue sections.

Transmitted by 3 C's:

Pregnant women
should avoid CATS.



Tachyzoites
transmitted
through
placenta



Infection of fetus

3. Crosses Placenta

Contamination
of
food and
water

DEFINITIVE
HOST
Cat



1. Cysts in Cat feces

Unsporulated
oocysts
passed in feces

Toxoplasma gondii

© medXclusive

Diseases:

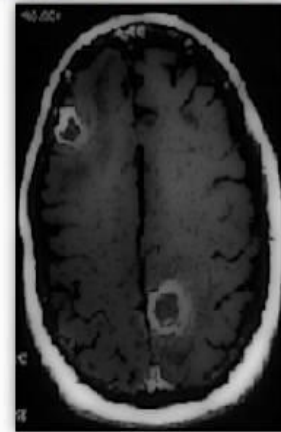
Congenital toxoplasmosis →

Classic triad:

1. *Chorioretinitis*
2. *HydroCephalus*
3. *IntraCranial Calcifications*

CNS infections in HIV patients

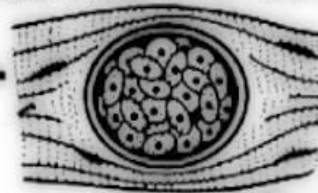
(Seen as Ring-enhancing brain
lesions on CT/MRI) →



Cysts ingested
by cat

Cysts in tissues of Intermediate Host

ingests
cysts in
infected
meat

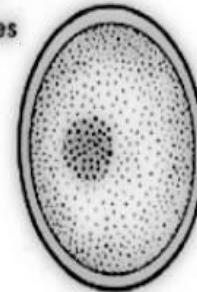


2. Cysts in meat

INTERMEDIATE HOSTS

Intermediate
host
ingests
oocysts

in feed
or
water
or
soil

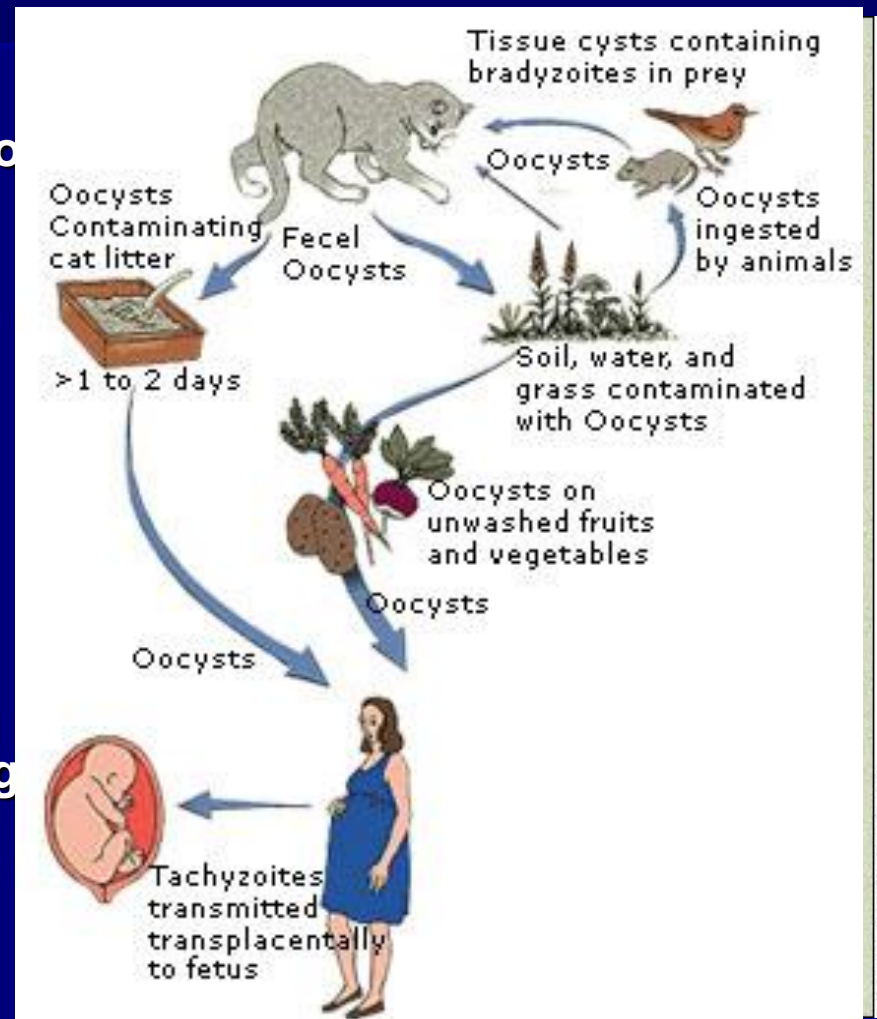


Sporulated oocyst

Ways of Infection

Oral intake of raw or rare ("under-cooked") **meat** or contamination with **cats feces** or consumption of contaminated **vegetables, fruits, and salad, ...**

- A fresh maternal infection during pregnancy can lead to an infection of the placenta.
- **Congenital Toxoplasmosis** results from transplacental infection of the fetus during pregnancy.



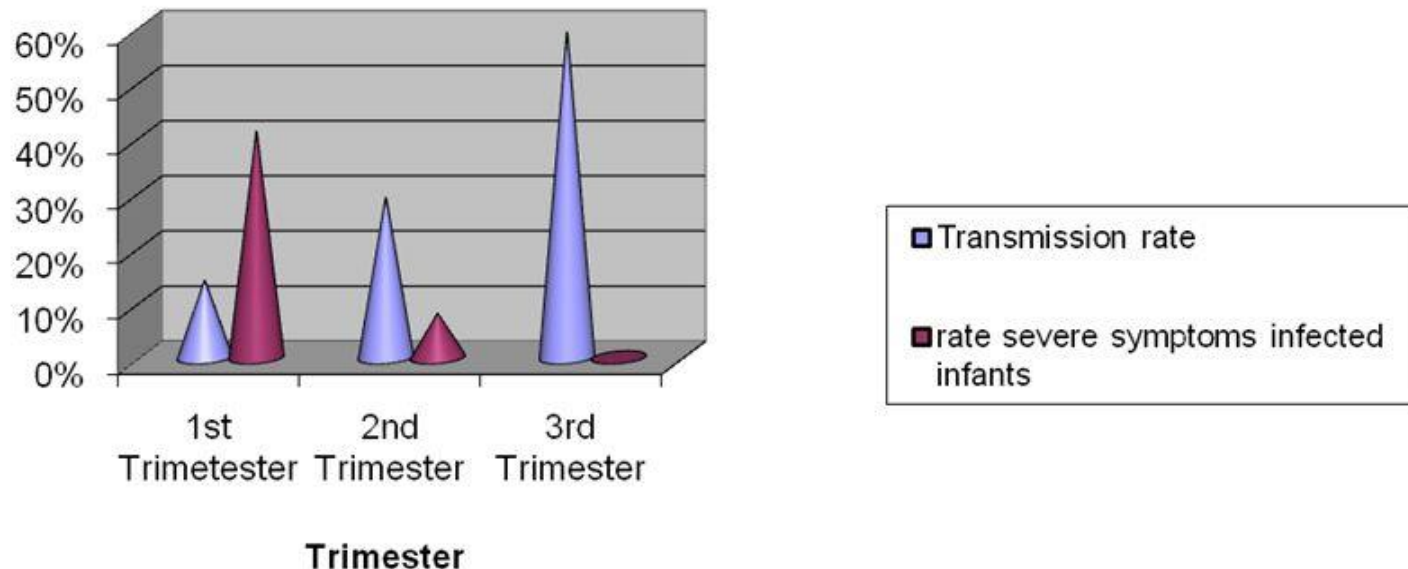
Congenital Toxoplasmosis

- Fetal infection varies
 - 15% in the 1st trimester
 - 30% in 2nd trimester
 - 60% in 3rd trimester
- Highest risk of severe congenital toxoplasmosis associated with primary maternal infection (weeks 10 - 24 of preg)
- Spiramycin administered to expectant mothers with documented primary infection during pregnancy can reduce transmission to the fetus up to 60%

Congenital infection;

- Most cases, due to 1^o maternal inf.
- Rarely, reactivation of a latent inf.

Transplacental Toxoplasma and Congenital Infection



Clinical presentation of congenital toxoplasmosis

- Chorioretinitis, blindness, seizures, hydrocephalus, microcephaly, intracranial calcifications, encephalitis, mental retardation, lymphadenopathy, hepatosplenomegaly, anemia and rash.
- Congenitally infected infants may be asymptomatic at birth but then develop symptoms during childhood.(chorioretinitis, developmental delays)
- PCR may help to confirm diagnosis in neonate.
- Toxoplasma IgG negativity or disappearance on serial testing is only way to exclude congenital infection in neonates.



Toxoplasmosis in Pregnancy

- Abortion
- Overt disease. The symptoms vary widely, the **classical triad** of Congenital Toxoplasmosis is
 - Hydrocephalus
 - Intracranial calcification
 - Chorioretinitis
- Subclinical infection: no symptoms at birth
- late onset symptoms (most common in the eyes: Chorioretinitis)
- no symptoms at all
-

Congenital Toxoplasmosis

- Hydrocephalus



Toxoplasma in retina

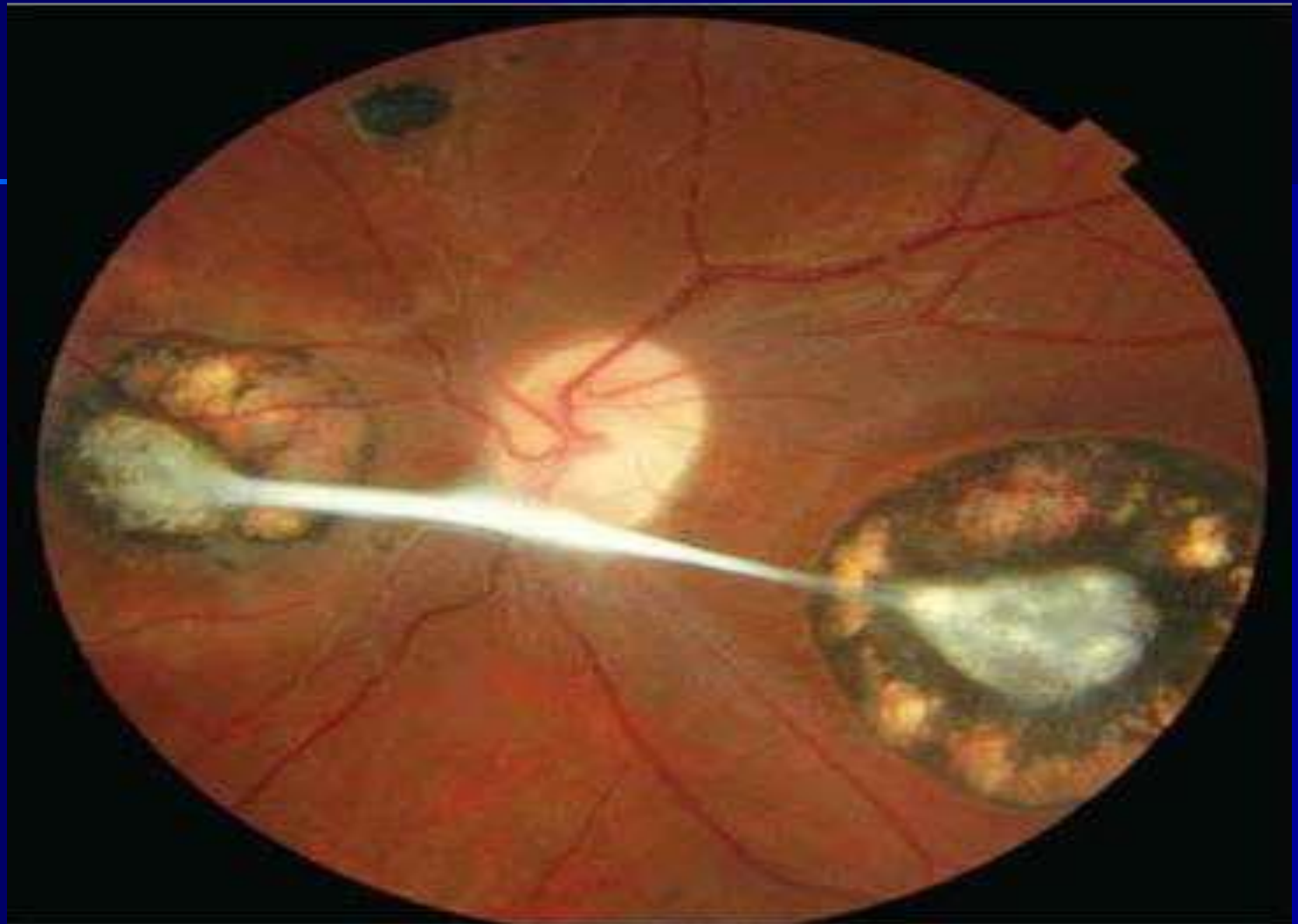
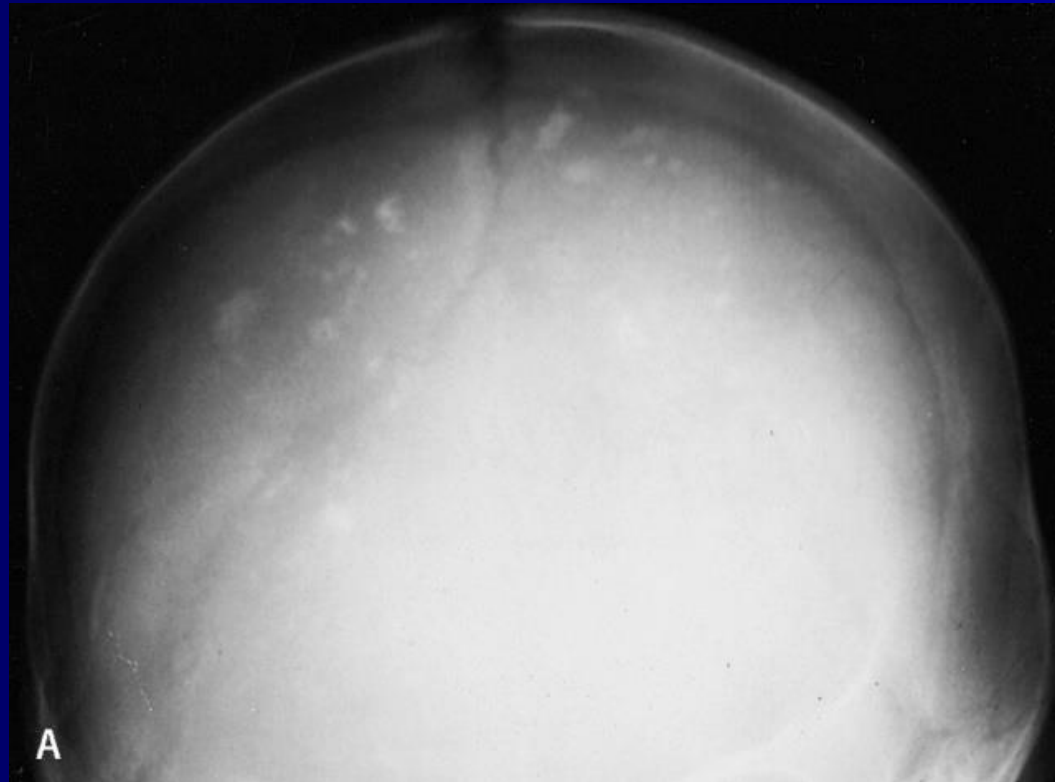


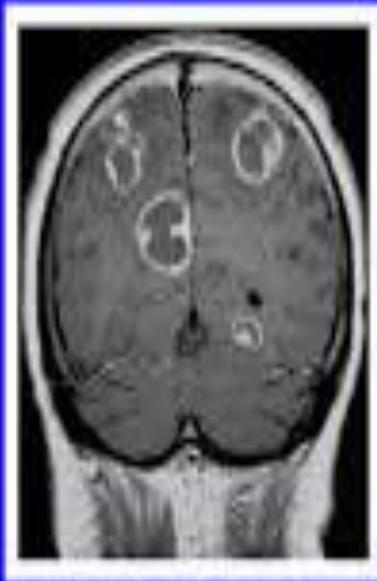
Fig. 7: retinal scars linked by vitreous strand in congenital toxoplasmosis

Congenital Toxoplasma

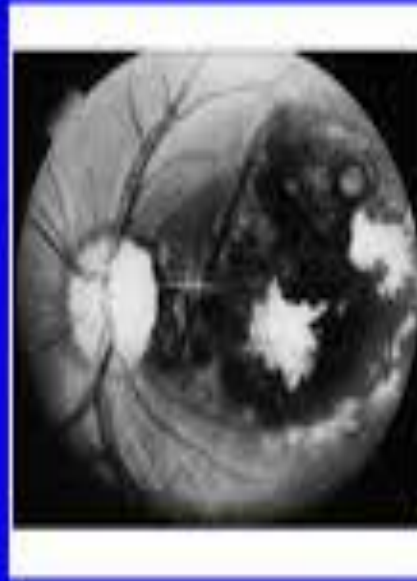
- Intracranial calcification



Encephalitis



Chorioretinitis

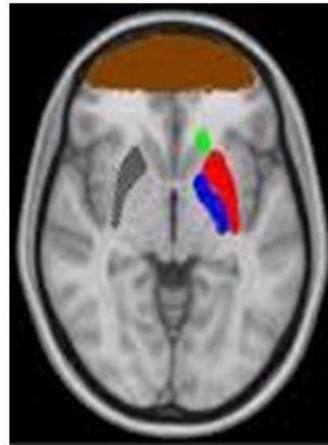


Hydrocephalus



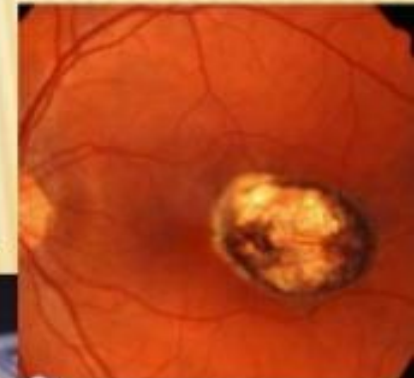
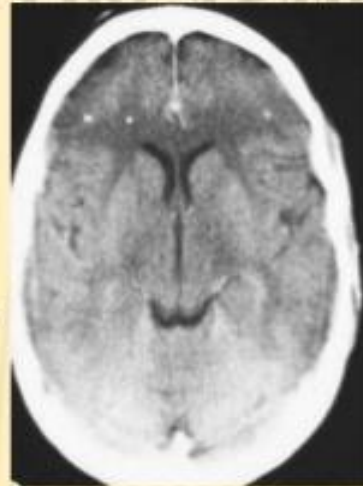
Congenital Toxoplasmosis

- The consequences of the infection of the fetus can be very different: between subclinical and very serious.
- **Abortion** Overt disease. The symptoms vary widely, the **classical triad** of Congenital Toxoplasmosis is **Hydrocephalus**
 - **Intracranial calcification**
 - **Chorioretinitis**



CONGENITAL TOXOPLASMOSIS

- ❖ Cerebral calcification leading to convulsion
- ❖ Bilateral healed chorioretinal scars: central vision jeopardized
- ❖ Hydrocephalus
- ❖ Microcephaly
- ❖ Psychomotor retardation
- ❖ Organomegaly
- ❖ Jaundice
- ❖ Rashes and fever



CONFIRMED CONGENITAL TOXOPLASMA INFECTION

Any of the following:

1. Detection of toxoplasma **specific IgM** (after 5 days of life) or **IgA titres** (after 10 days of life) is considered diagnostic of congenital toxoplasmosis in infants with a positive Toxoplasma IgG titre
2. Positive for Toxoplasma **IgG** beyond 12months of age
3. Positive **CSF PCR**
4. Increase in anti-Toxoplasma IgG titer during the first year of life or increasing IgG titer compared with the mother's



- **Toxoplasma:**

- **Diagnosis :**

- IgG, IgM, IgA (Serum/CSF)
- PCR
- Ophthalmologic, auditory, and neurologic examinations
- CT Brain

*Redbook American Academy of Pediatrics.
2012. p. 720–8.*

Treatment of pregnant women

- Before 16th weeks' gestation
- 4 weeks Spiramycine [Rovamycine©]
- After 16th weeks' gestation , if fetus is infected:
 - alternating to birth 4 weeks combination of:Pyrimethamin [Daraprim©], Sulfadiazin, Folinic Acid 4 weeks Spiramycine [Rovamycine©]
- After 16th weeks' gestation, if fetus is not infected:
 - Spiramycine [Rovamycine©] to birth
 - The combination of:Pyrimethamin [Daraprim©], Sulfadiazin, can pass through placenta and treat the fetus. But it is not allowed to give before about 16th weeks' gestation.

Treatment of prenataly infected children

- Treatment of prenataly infected children with (symptomatic) disease
- 6 months: Combination of:
Pyrimethamin [Daraprim©) Sulfadiazin
Folinic Acid
- 6 months: alternating to the first birthday
- 4 weeks Spiramycine [Rovamycine©]
- 4 weeks Pyrimethamin [Daraprim©],
Sulfadiazin, Folinic Acid .

Treatment of prenataly infected children with subclinical infection (no symptoms)

- 6 weeks: A) Combination of: Pyrimethamin [**Daraprim**©] Sulfadiazin **Folinic Acid** 6 weeks: B) Spiramycine [**Rovamycine**©] alternating A and B to the first birthday 4 weeks Pyrimethamin [**Daraprim**©], Sulfadiazin, Folinic Acid 6 weeks Spiramycine [**Rovamycine**©]

Treatment of Infected Newborns

- Infected babies should be treated as soon as possible after birth with pyrimethamine and sulfadiazine which, as mentioned earlier, can help prevent or reduce the disabilities associated with toxoplasmosis.



Prevention of Congenital Toxoplasmosis.

- **Primary** prevention is an information about the ways of infection (cats, raw meat) to avoid ingestion or inhalation. This is important for all pregnant women who are "seronegative"
- **Secondary** prevention is the detection of infected women during pregnancy to start treatment before the fetus gets infected.
- **Tertiary** Prevention is the treatment of infected children to reduce or avoid symptoms.

RUBELLA

Rubella

- Rubella (**German measles** or **3-day measles**)
- Single-stranded RNA virus
- Vaccine-preventable disease
- mild, self-limiting often exanthematous disease of infants and children.
- In the pre vaccine era, rubella appeared to occur in major epidemics every 6-9 yr,
- with smaller peaks interspersed every 3-4 yr,
- was most common in preschool and school-aged children.

Rubella

Mainly first trimester infection can lead to **congenital Rubella(deafness, cardiac abnormality, cataract, microcephaly, mental retardation)**

** No treatment

Prevention is by vaccination (childhood or post-natal)

*Vaccine is live attenuated so, 3 months contraception is advised after vaccination.

Risk of Fetal infection %

Gestational age in weeks	Fetal infection %
< 11	90 %
11-12	30 %
13-14	20 %
15-16	10 %
> 16	5 %

- Despite of availability of effective vaccines up to 20% of women of child bearing age don't possess rubella antibody.

Rubella

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> 16	5 %

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A woman **infected with rubella** during the first 3 months of pregnancy has up to a

90%
chance



of giving birth to a baby with **congenital rubella syndrome**

Or her baby may not survive.

- **The congenital rubella syndrome includes:**

- a-Neuropathic changes:**

1. microcephaly.
2. mental & motor retardation.
3. meningoencephalitis
4. cerebral palsy.
5. cerebral calcification

- b-Cardiovascular lesions:**

1. persistent ductus arteriosus
2. pulmonary artery stenosis
3. atrioventricular septal defects

- c-Ocular defects:**

1. cataract
2. microphthalmia
3. retinal changes, retinitis
4. blindness

Microcephaly

Heart disease

Petechiae and
purpura



Eye anomalies may include cataracts, glaucoma, strabismus, nystagmus, microphthalmia, and iris dysplasia.

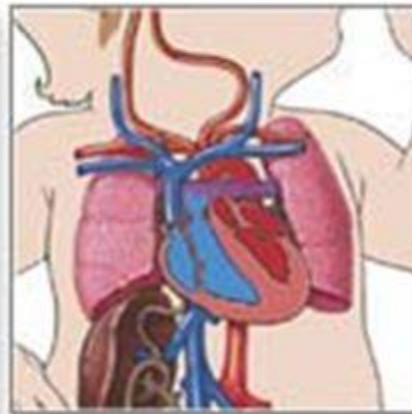
Rubella

- Rubella virus - transmitted by a **respiratory route**
- congenital rubella **triad** of **deafness, congenital cataracts & heart defects**

Rubella syndrome



Microcephaly



PDA



Cataracts

Normal head size



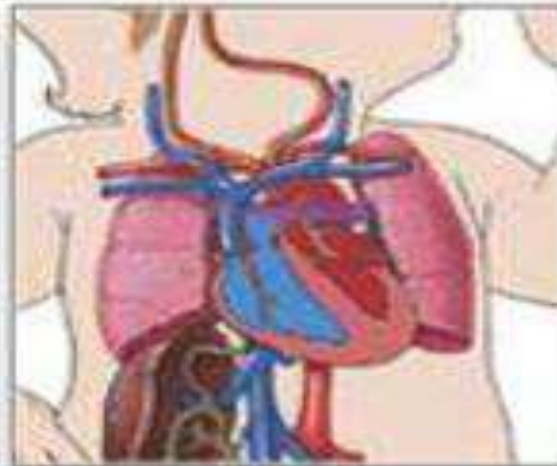
Microcephaly



Rubella syndrome



Microcephaly



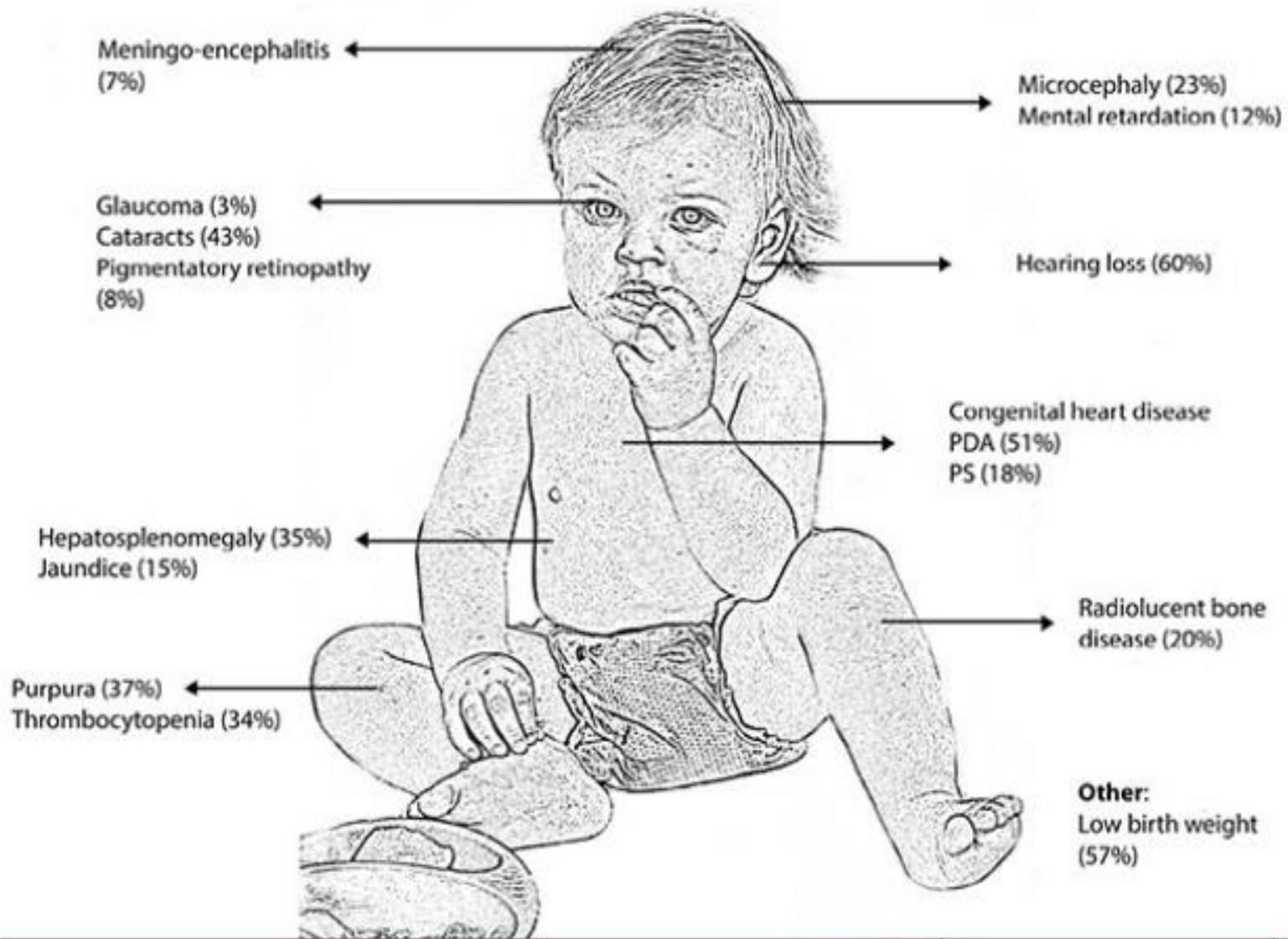
PDA



Cataracts



Frequency and clinical course of selected clinical findings following intrauterine rubella infection



Congenital Rubella Syndrome

- Diagnosis:
 - Rubella-specific IgM antibody in the neonatal serum, or by culturing rubella virus from the infant (nasopharynx, urine, or tissues)
 - Isolating the virus from amniotic fluid or by identification of rubella-specific IgM in cord blood.

Rubella-Treatment

- **No specific treatment available for either acquired rubella or CRS**
- Supportive treatment- antipyretics and analgesics
- Intravenous **immunoglobulin or corticosteroids**-for severe, nonremitting thrombocytopenia
- Hearing screening- important, **early intervention** improve outcomes

Outcome Congenital Rubella

- 1/3 rd will lead normal independent lives
- 1/3 rd will live with parents
- 1/3rd will be institutionalised



Prevention

Antenatal screening

- Non-immune women vaccinated post partum
- Effective live attenuated vaccine (95% efficacy)
- Universal vaccination (MMR)
- Selectively vaccination of schoolgirls

Cytomegalovirus

- member of the herpesvirus
- primary infection usually asymptomatic. Virus then becomes latent and is reactivated from time to time.
- transmitted by infected saliva, breast milk, sexually and through infected blood
- 60% of the population eventually become infected. In some developing countries, the figure is up to 95%.

What is CMV?

CYTOMEGALOVIRUS (CMV) IS A COMMON VIRUS THAT INFECTS ONE IN EVERY TWO PEOPLE IN MANY DEVELOPED COUNTRIES

- Most CMV infections are “silent” – most people who are infected with CMV exhibit no signs or symptoms
- However, CMV can cause serious disease in newborns when a woman is infected during pregnancy, including:
 - Deafness
 - Blindness
 - Physical birthmarks
 - Developmental delays
- This is known as **CONGENITAL CMV**



CMV

- The most common congenital infection affecting 1% of all live births
- 10% of infected neonates demonstrate clinical manifestations that potentially could be identified by prenatal sonography
- Ventriculomegaly, FGR, Intracranial calcifications and oligohydramnios are the most frequently reported findings

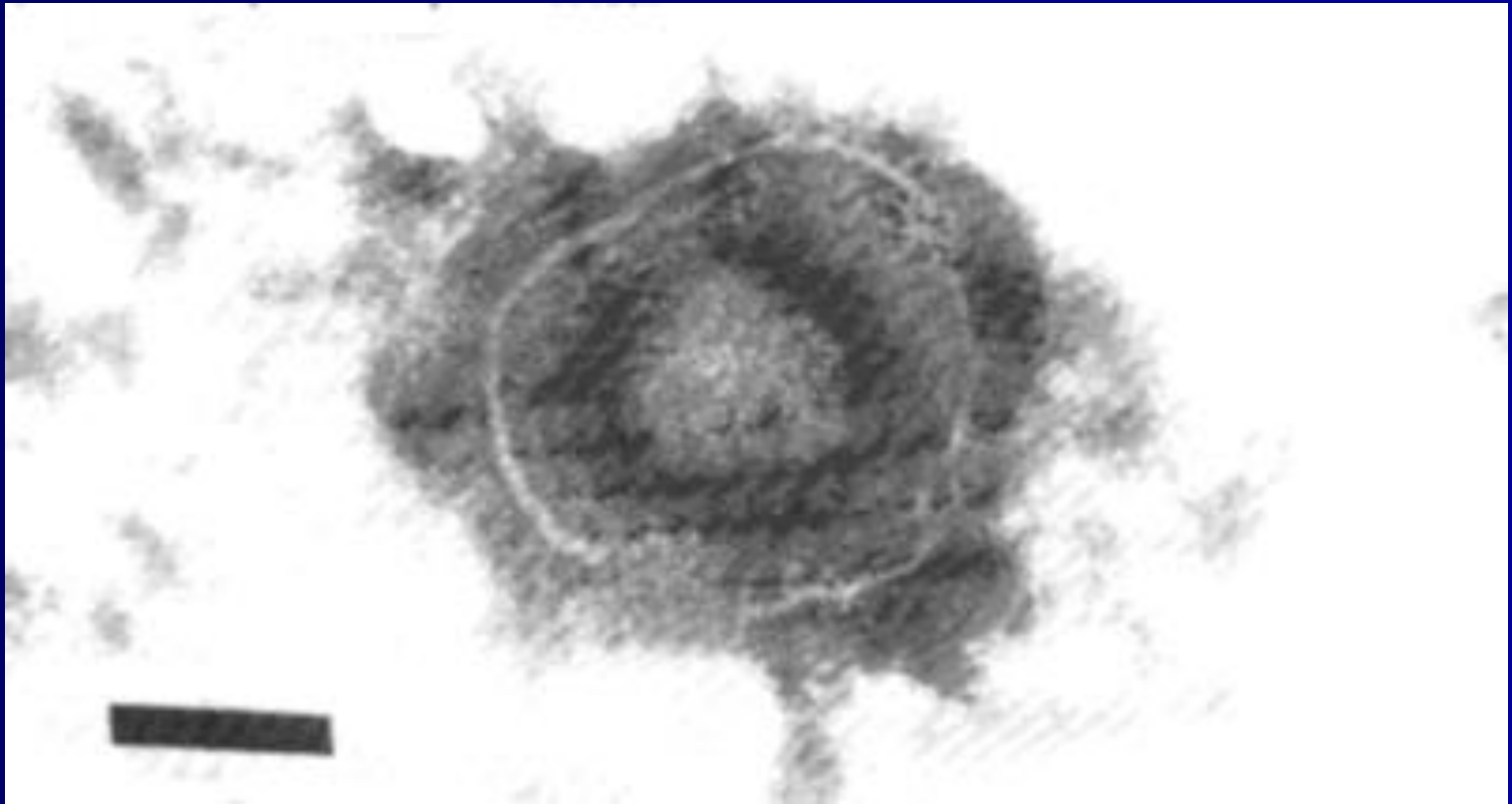
CYTOMEGALOVIRUS (CMV)

- in UK CMV is commoner cause of cong. Retardations than rubella.
- ✱ Infects 50%to 60% of women of childbearing age.
- **Causative agent:** CMV (Herpes virus DNA).
- **Maternal symptoms :** usually mild or asymptomatic, fever with/without lymphadenopathy, sore throat.

Transmission:

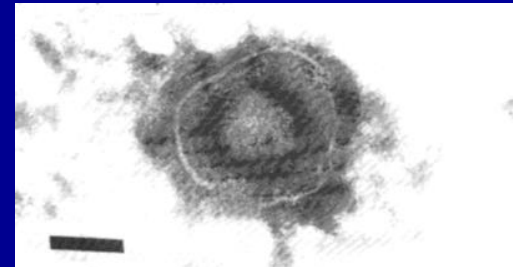
- direct: person to person contact.
- indirect: contaminated fomites.
- in primary CMV infection , 30-40% of fetuses will be infected, 2-4% of them will develop severe malformations at birth.
- in recurrent CMV infections (about 1% of fetuses will be infected and the rest will appear normal at birth, but later in life, they may suffer from delayed speech and learning difficulties due to cerebral calcification and sensorineural hearing loss. And small group will have chorioretinitis.

Cytomegalovirus CMV



Epidemiology of CMV

- Shed
 - body secretions, urine
- Transfer
 - Intimate contact
 - transplacental
 - during birth
 - breast feeding
 - blood products
 - organ transplantation



CMV in the newborn

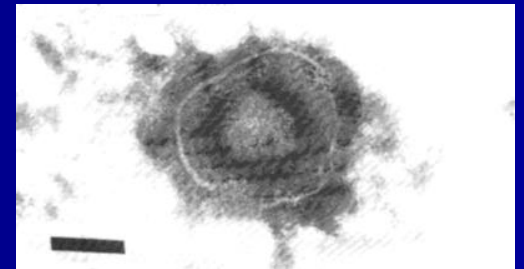
- 1% newborns infected (0.2% - 2.5%)
- Higher infection rate with primary maternal (50%) than after recurrent maternal (1%)
- 50% of babies fed CMV infected breast milk get infection

CMV in Pregnancy

- 90% of women asymptomatic
- Primary infection 1 - 4% of pregnancies
- Foetal transmission 25 - 75% (~ 40%)
- More neuro sequelae in infants of primary infection
- Maternal antibody does not protect against infection but may be associated with less sequelae

Diagnosis in Pregnancy

- Usually asymptomatic
- Sometimes 'flu-like illness (like EBV)
- Conversion to IgG positive
- Rising titres not helpful



CMV and foetus

- Transmission can occur in all trimesters
- Adverse neuro outcome more likely in 1st trimester infection

Oligohydramnios

Microcephaly

polyhydramnios

dilated ventricles

IUGR

calcification

*non-immune
hydrops*

*pseudomeconium
ileus*

ascites

effusions

DIAGNOSIS OF CONGENITAL CMV INFECTION IN FOETUS

Amniocentesis - viral culture and PCR

Ultrasound

Cerebral
calcification



CMV and newborn

- 90% newborn asymptomatic
- 10% have clinical signs

SGA

microcephaly

calcification

chorioretinitis

hearing loss

hepatosplenomegaly

jaundice

Petechiae

thrombocytopenia

meningitis

Complications of fetal CMV infection include:

1. micro-& hydrocephaly
2. chorioretinitis
3. cerebral calcification
4. mental retardation
5. heart block
6. petechial rash

Maternal screening: not recommended.

Prevention: hand washing(especially after changing diapers).

Cytomegalic Inclusion Disease

- **CNS** abnormalities
 - - microcephaly, mental retardation, spasticity, epilepsy,
 - periventricular calcification.
- **Eye** - choroidoretinitis and optic atrophy
- **Ear** - sensorineural deafness
- **Liver** - hepatosplenomegaly and jaundice which is due to hepatitis.
- **Lung** - pneumonitis
- **Heart** - myocarditis
- Thrombocytopenic purpura, Haemolytic anaemia
- **Late sequelae** in individuals asymptomatic at birth
 - hearing defects and reduced intelligence.

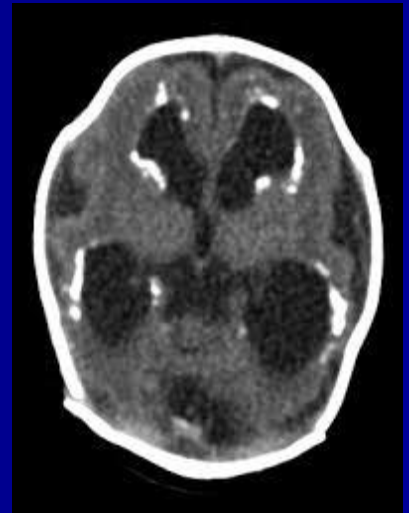
358



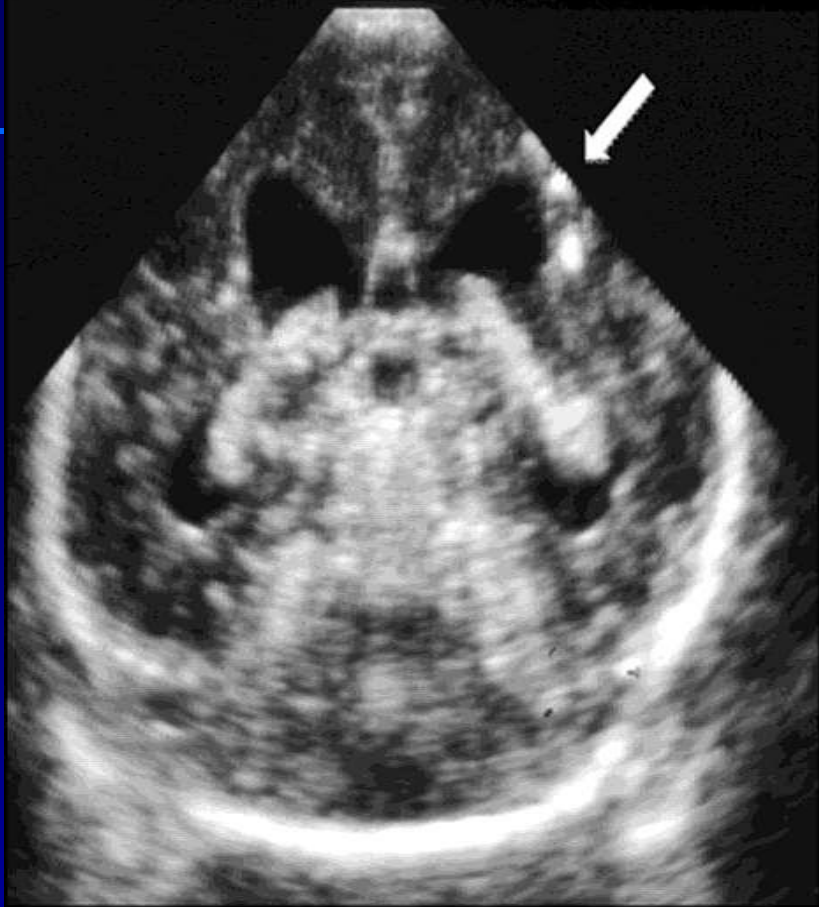
358 Blueberry muffin' rash compatible with congenital infection with CMV or rubella.



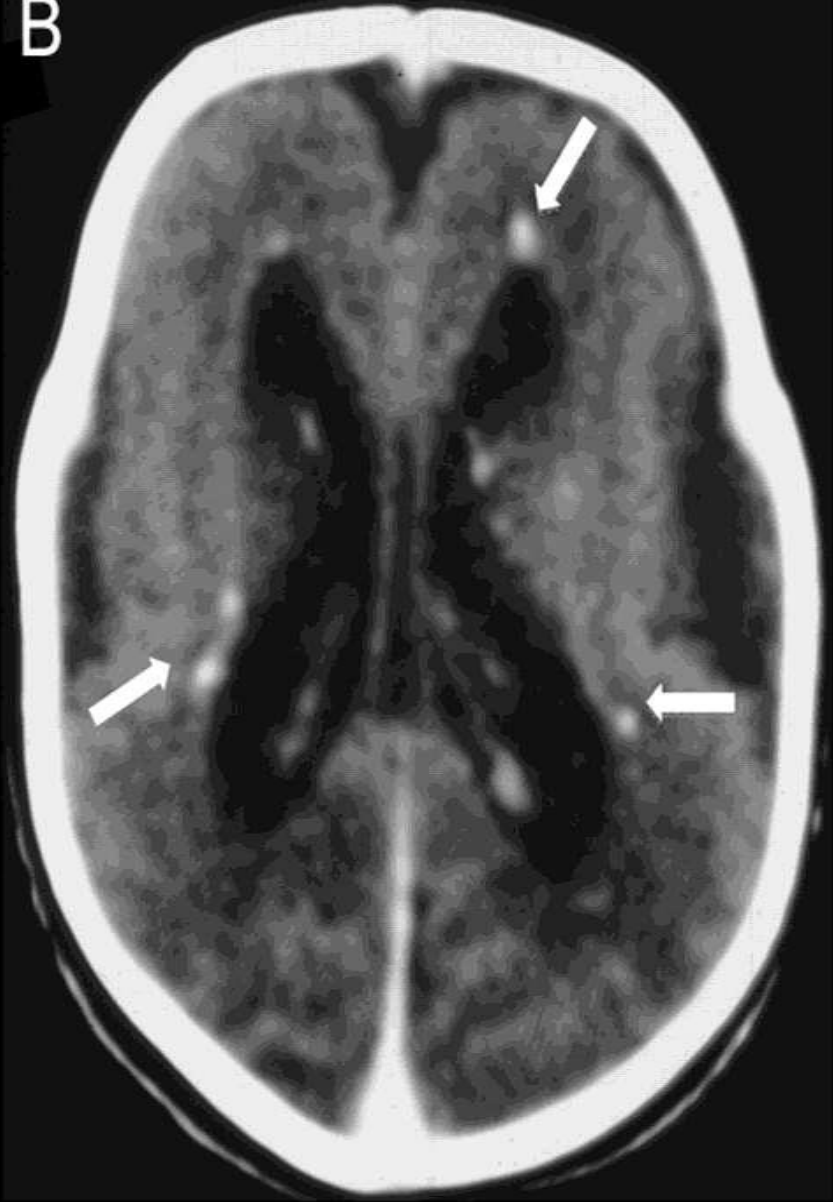


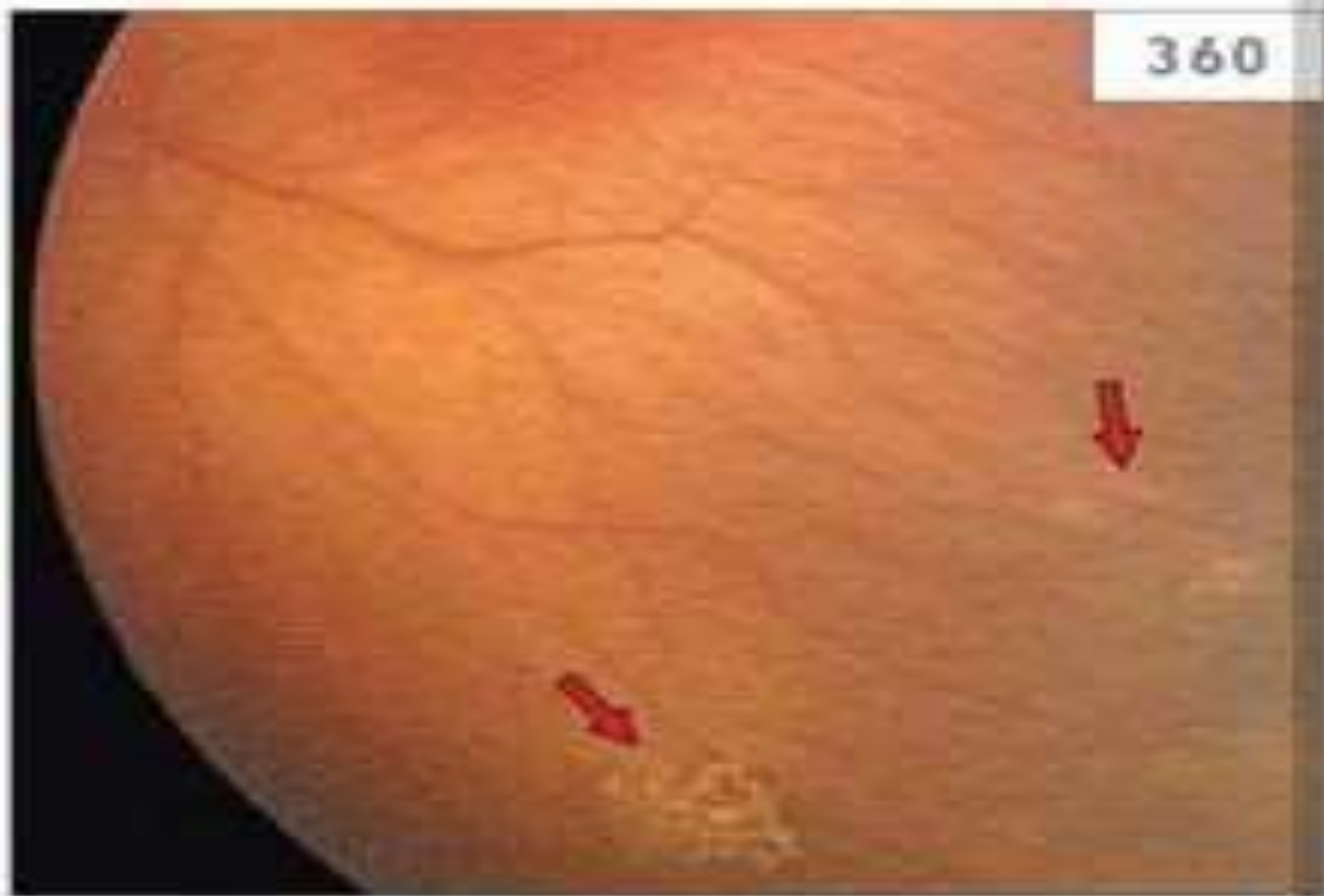


A



B





360 Chorioretinitis (arrows) in an infant with congenital CMV infection.

Lab diagnosis in newborn

- Urine culture (gold standard)
 - positive in first 3 weeks suggests congenital
- CMV IgM (sensitivity = 75%)
 - *negative result does not exclude infection*
- CMV PCR (urine)
- LFT's, thrombocytopenia, anaemia, leucopenia

Prevention of CMV

- Vaccine
- Focus on high-risk (childbearing)
- Education
- CMV negative blood

Diagnosis

- Isolation of CMV from the urine or saliva of the neonate.
- Presence of CMV IgM from the blood of the neonate.
- Detection of Cytomegalic Inclusion Bodies from affected tissue (rarely used)

• CMV: Treatment :

Life Threatening
infection

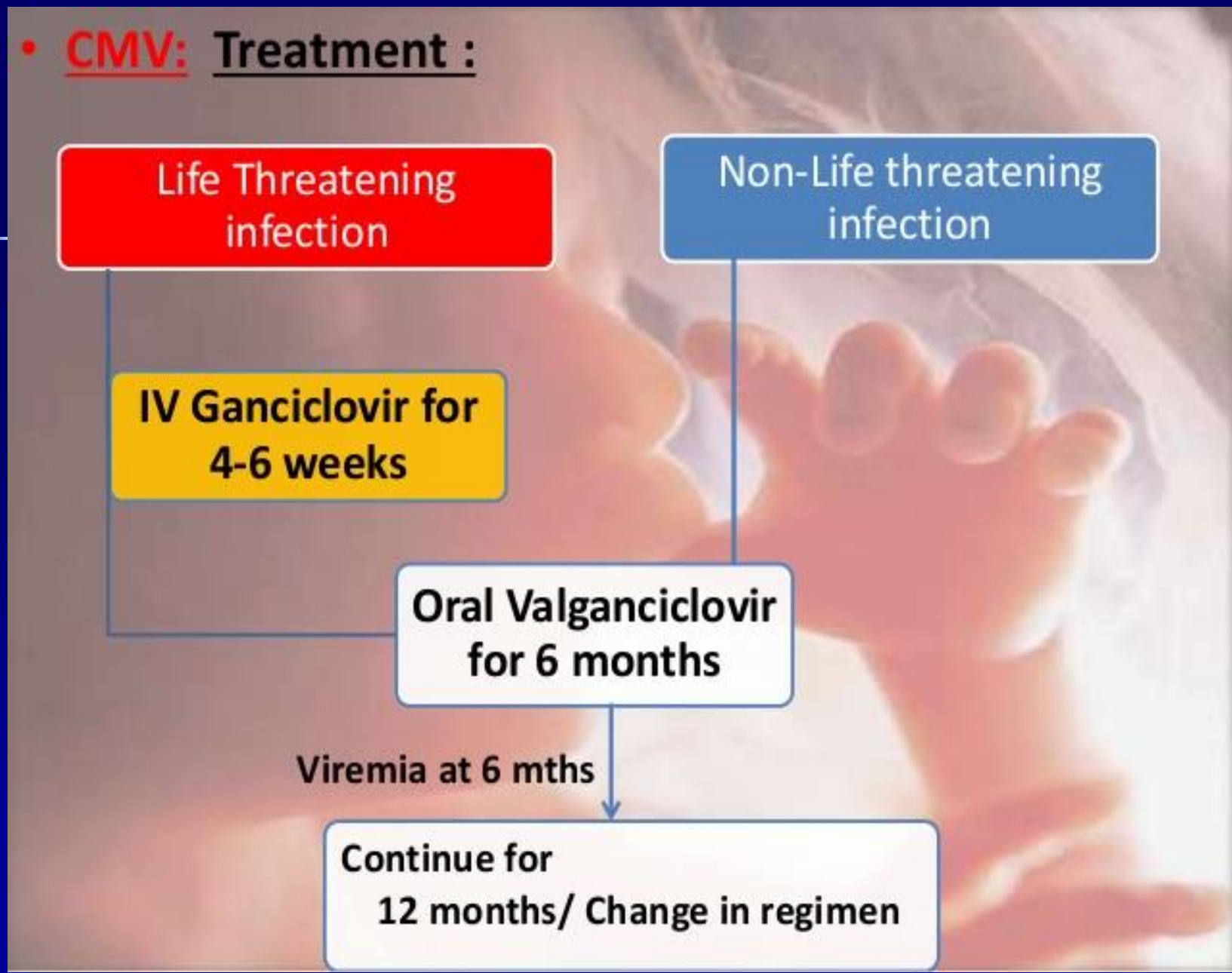
Non-Life threatening
infection

IV Ganciclovir for
4-6 weeks

Oral Valganciclovir
for 6 months

Viremia at 6 mths

Continue for
12 months/ Change in regimen



Follow up

- Neuro-developmental
- Ophthalmology
- Hearing (BAER)
- Educational
 - dyspraxia
 - learning difficulties

Better outcome

- Reticulo-endothelial involvement
- When CNS not involved

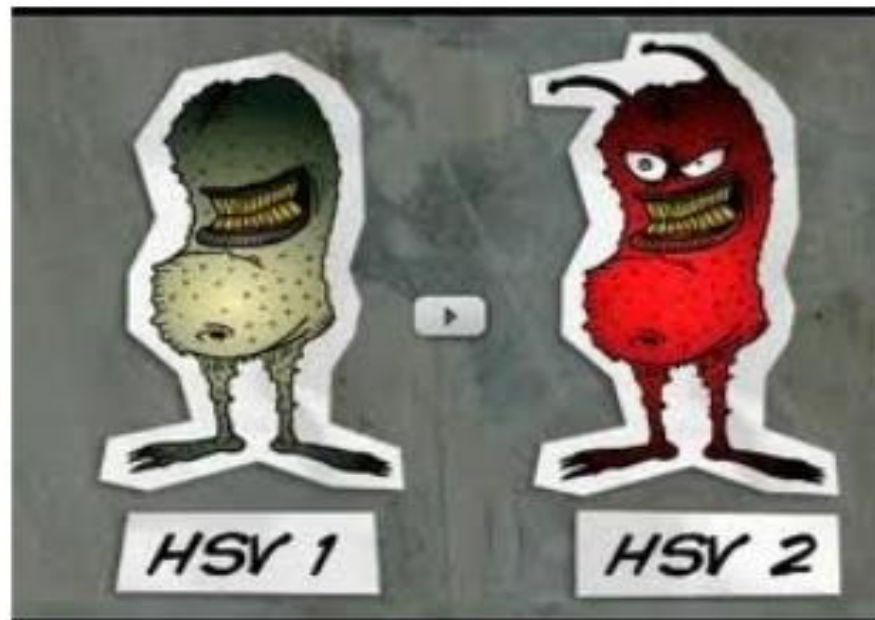
Worse outcome

- Chorio-Retinitis
- Microcephaly
- Early neurological findings

'Silent' CMV outcome

- Hearing loss 15%
- mostly normal neurologically

HERPES SIMPLEX



Genital Herpes Simplex Virus

Herpes Simplex Type II

- *Risk of vertical transmission & though the birth canal
 - ***If lesion are present, cesarean section is the optimal mode of delivery**
 - * Patients with outbreak during pregnancy should take acyclovir prophylaxis from 36 weeks until delivery
 - ***Primary infection make more damage than secondary attack**
 - * Primary Herpes infection in the late third trimester is far more dangerous than earlier infection

Neonatal HSV

- 1 in 2,500-5,000 deliveries / 500-1500 per yr.
- Birth to 7 weeks of life
- HSV2 = 70-75%, HSV1 = 25-30%
- **3 Main Types**
 - Skin, Eye, Mouth (SEM)
 - CNS
 - Disseminated Disease (DISSEM)
- At Risk: Premature, ROM >6hr, Fetal scalp monitoring
- Can be acquired **congenitally**, **during the birth process**, and in the **post-partum period**

What is the risk?

- Vaginal delivery when mom has presence of **first symptomatic lesions** – 50%
- Vaginal delivery when mom is **asymptomatic**, but is newly infected – 33%
- Vaginal delivery when mom has **recurrent lesions** – 4%
- Vaginal delivery when mom has a history of herpes lesions in **past**, none presently – 0.04%

Genital Herpes

Infection can cause **neonatal viral sepsis, herpetic lesions on skin, eyes, pneumonia, herpes encephalitis** which can lead to **neurological abnormality and death.**

HSV from skin lesion ,PCR blood and CSF

Infected infants should be treated with
I.V. acyclovir

CLINICAL FEATURES

Inutero Infection:

Skin: Scarring, vesicles, hypo/hyperpigmentation

Eyes: Microphthalmia, retinal dysplasia

CNS: Microcephaly, encephalomalacia, hydranencephaly

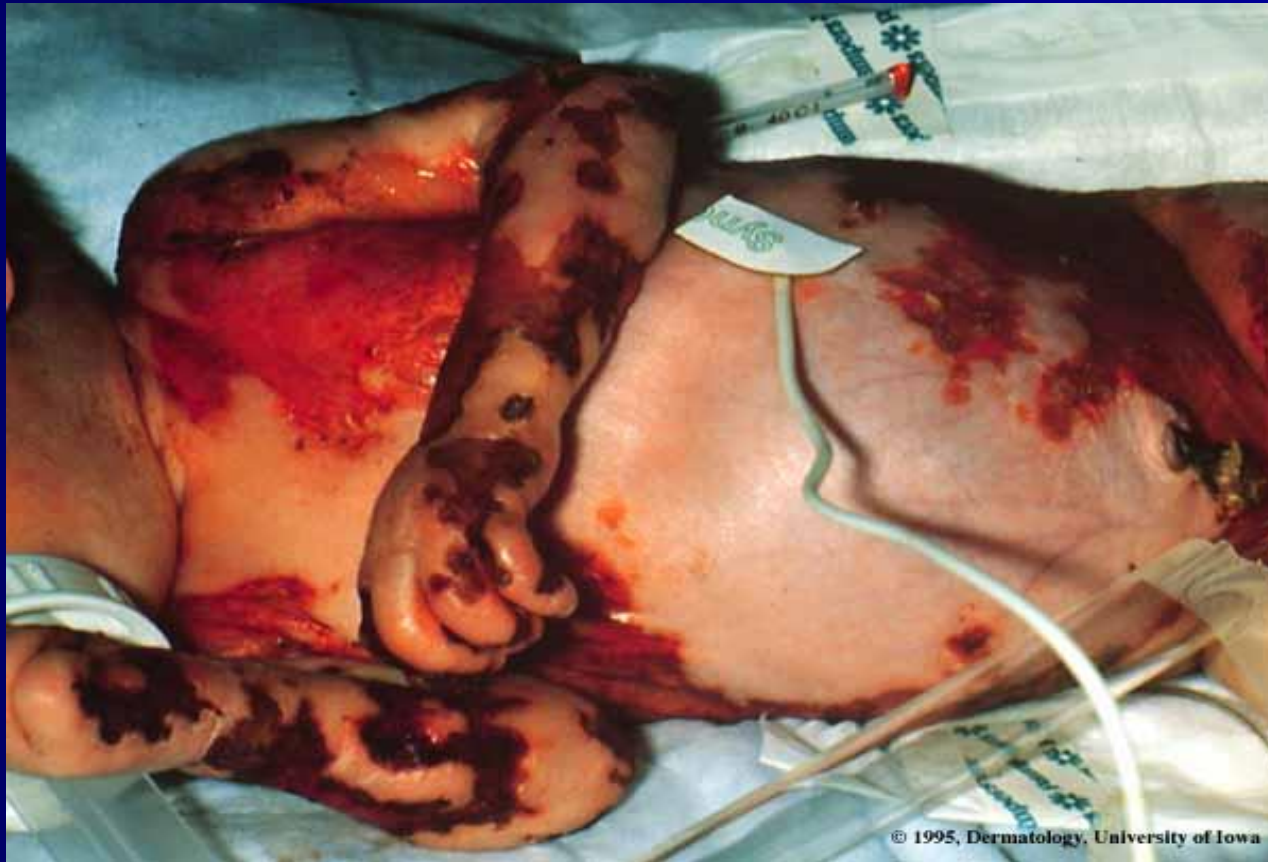
Intrapartam/Postpartam Infection:

SEM disease : Vesicular lesions, Conjunctivitis, excessive tearing, Ulcerative lesions of the mouth, palate & tongue

Disseminated disease : Sepsis, Fever, Respiratory distress, DIC, Skin lesions, CNS involvement(60 to 75%)

CNS disease +/- Skin : Seizures, Lethargy, Irritability, Tremors, Poor feeding, Skin lesions(60 to 70%)

Congenital herpes



Skin lesions of a newborn with HSV-2 infection



LESIONS OF NEONATE WITH SEM DISEASE

EYE VESICLES



HYPOPIGMENTED, SCALING, AND CRUSTED EROSIONS OF THE TRUNK AND EXTREMITIES



DIAGNOSIS

- For SEM disease - Viral Culture by Isolation – Newer vesicular fluid, Urine & conjunctival smears.
- For Non SEM disease– PCR of CSF
- EEG and Imaging studies of brain also aids in the diagnosis of HSV encephalitis
- Cytology of vesicular fluid – Presence of Tzanck cells

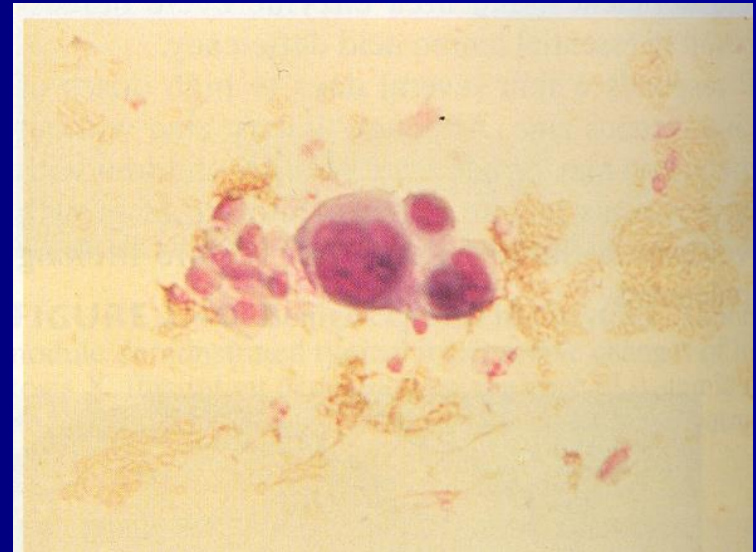
PCR Testing

- Detects minute amounts of DNA, RNA
 - DISSEM – 93%
 - CNS – 76%
 - SEM – 24%
- False negative may occur if CSF is obtained “too early”
- Order through IVF!



Diagnostics (cont)

- Surface cultures
 - Mouth (40-50%)
 - Eyes (25%)
 - Rectum
 - Skin
- Cultures
 - Stool
 - Urine
 - CSF >100 WBC/Inc. Pro
- Tzanck – neither sensitive nor specific



Treatment - Acyclovir

- SEM infections
 - 60mg/kg/day divided q8h for 14 days
 - May be lengthened to 21 days in the near future
 - Oral Acyclovir needed later in life?
- DISSEM and CNS HSV infections
 - 60mg/kg/day divided q8h for 21 days
 - Re-tap if CNS disease exists prior to d/c
- Watch for neutropenia – 2x week

Varicella-Zoster Virus

- 90% of pregnant women already immune, therefore primary infection is rare during pregnancy
- Primary infection during pregnancy carries a greater risk of severe disease, in particular pneumonia

First 20 weeks of Pregnancy

up to 3% chance of transmission to the fetus ,
recognised congenital varicella syndrome;

- Scarring of skin
- Hypoplasia of limbs
- CNS and eye defects
- Death in infancy normal



Congenital Varicella Syndrome



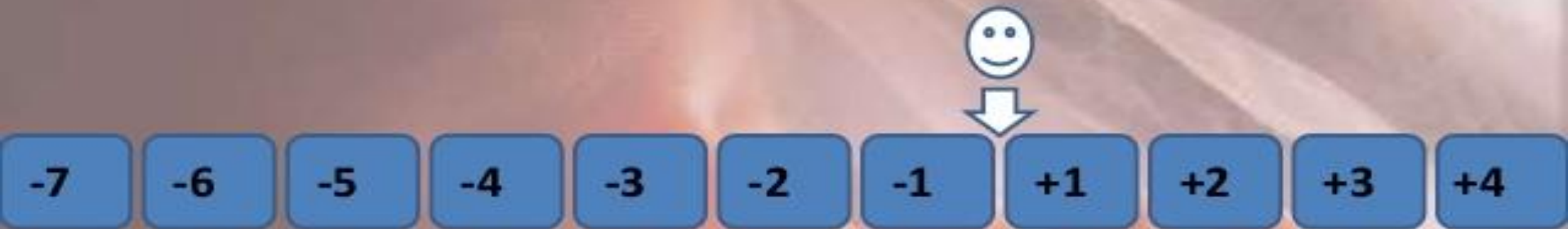
Neonatal Varicella

- VZV can cross the placenta in the late stages of pregnancy to infect the fetus congenitally.
- Neonatal varicella may vary from a mild disease to a fatal disseminated infection.
- If rash in mother occurs more than 1 week before delivery, then sufficient immunity would have been transferred to the fetus.
- Zoster immunoglobulin should be given to susceptible pregnant women who had contact with suspected cases of varicella.
- Zoster immunoglobulin should also be given to infants whose mothers develop varicella during the last 7 days of pregnancy or the first 14 days after delivery.



- **Serology** (IgG and IgM).
- **Screening:** Routine screening generally not recommended.
- **Prevention:** If pregnant woman (with no history of previous chickenpox) is exposed, perform STAT Varicella IgG.
- Exposed neonate should receive VZIG prophylaxis.

- Varicella:



ONSET OF RASH IN MOTHER

• Varicella:



Newborn will have protective antibodies
Likelihood of severe disease is low



- Do not separate baby from mother
- Continue breast feeding
- No VZIG
- Acyclovir if baby develops rash

• Varicella:

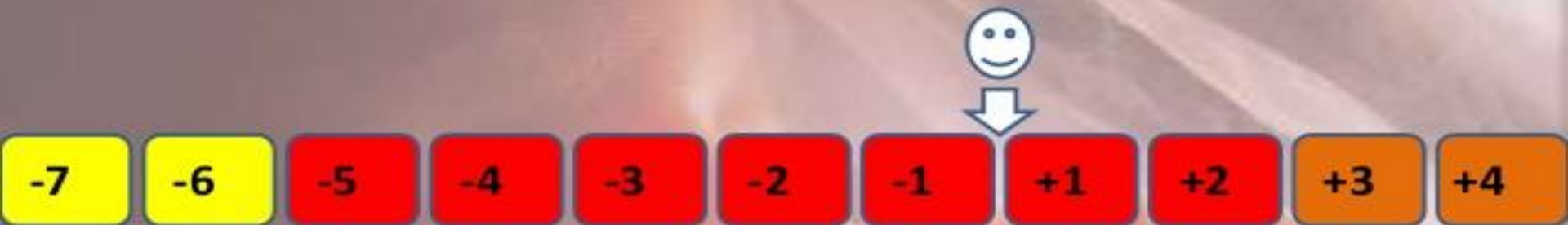


Newborn will not have protective antibodies
Likelihood of severe disease is high



- Separate baby from mother
- If baby develops rash → stay with mother
- VZIG within 72 hours
- Acyclovir

• Varicella:



Newborn will not have protective antibodies
But, likelihood of severe disease is low



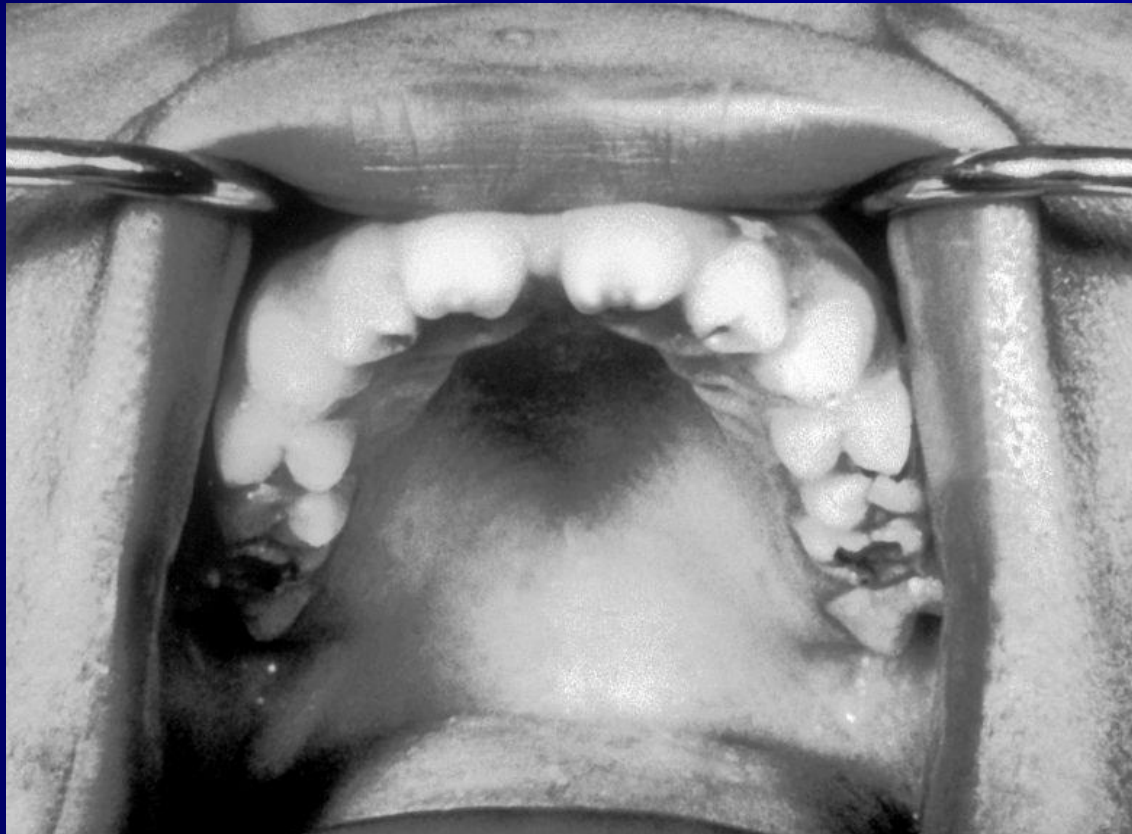
- Separate baby from mother
- If baby develops rash → stay with mother
- No VZIG
- Acyclovir if baby develops rash

Syphilis(Treponema pallidum)

- *Infection to fetus is vertical in patients with primary and secondary syphilis
- *Can lead to abortion, still birth, or congenital syphilis (maculopapular rash, hepatosplenomegaly, lymphadenopathy, jaundice, 8th nerve deafness, saber shins, Hutchinson's teeth, saddle nose)
- **Diagnosis by IGM antitreponemal antibodies.
- *Treatment is Penicillin
- **Latent Syphilis may not transmit the disease

Congenital syphilis

- Hutchinson teeth





CONGENITAL SYPHILIS





• **Syphilis:** **Treatment :**

PHYSICAL EXAM
SUGGESTIVE OF
CONGENITAL
SYPHILIS

BABY'S
VDRL/RPR
4 TIMES HIGHER
TITRE THAN
MOTHER

MOTHER NOT
TREATED OR
INADEQUATELY
TREATED

ADDL TESTS: CSF VDRL,
LONG BONE XRAY,
OPHTHAL EVALUATION,
BERA

INJ. PENICILLIN G OR PROCAINE PENICILLIN FOR 10 DAYS

PARVO VIRUSES

- Maternal infection is usually self limited
- Fetal infection occurs in 33% cases following maternal infection
- Characterized by cheek slap appearance
- It mainly affects the erythroid precursor cells → anemia, aplastic crises, congenital heart failure, hydrops
- Fetal loss is more when infection occurs early (< 10 weeks) in pregnancy.



33% hydrops- resolve spontaneously

30% mortality rate

Parvovirus B19

Causes erythema infectious

- ****Vertical transmission can lead to hydrops fetalis, hemolytic anemia, myocarditis, abortion, death**
- ***If less than 20 weeks and the fetus survive the infection, the fetus may be healthy**

Parvovirus B19

- ▶ Single-stranded DNA virus
- ▶ Usually causes “fifth disease” (“slapped cheek”), and other symptoms.
- ▶ Route of infection:
 - ▶ Respiratory tract secretions
 - ▶ Contaminated blood
 - ▶ Transplacentally
- ▶ Clinical manifestations:
 - ▶ Hydrops fetalis (due to severe fetal anemia)
 - ▶ Pleural and pericardial effusions
 - ▶ IUGR
 - ▶ death

Hydrops fetalis: a condition in the fetus characterized by an accumulation of fluid, or edema, in at least two fetal compartments.

Very high mortality rates



courtesy: Dr. Indrajit Rana

Fifth Disease



Parvovirus B 19

1/400 pregnancies

15% risk of miscarriage

3% risk of hydrops

Hydrops fetalis. Fetal edema due to heart failure in utero.
RDG Milner and SM Herber.
Color Atlas of the Newborn. 1984.

Congenital Parvovirus Infection

- Known to cause fetal loss
 - hydrops fetalis; severe anaemia, congestive heart failure, generalized oedema and foetal death
- No evidence of teratogenicity.
- Risk of foetal death highest in second trimester (12%).
- Minimal risk to the foetus in first or third trimesters
- Maternal infection during pregnancy does not warrant termination of pregnancy.
- Cases of diagnosed hydrops fetalis had been successfully treated in utero by intrauterine transfusions



ZIKA 101

Updated February 2, 2017



U.S. Department of
Health and Human Services
Centers for Disease
Control and Prevention

What is Zika?

- Zika virus is spread to people primarily through the bite of an infected *Aedes* species mosquito (*Ae. aegypti* and *Ae. albopictus*).
- Many people infected with Zika virus won't have symptoms or will only have mild symptoms.
- Zika virus infection during pregnancy can cause microcephaly and other severe brain defects.



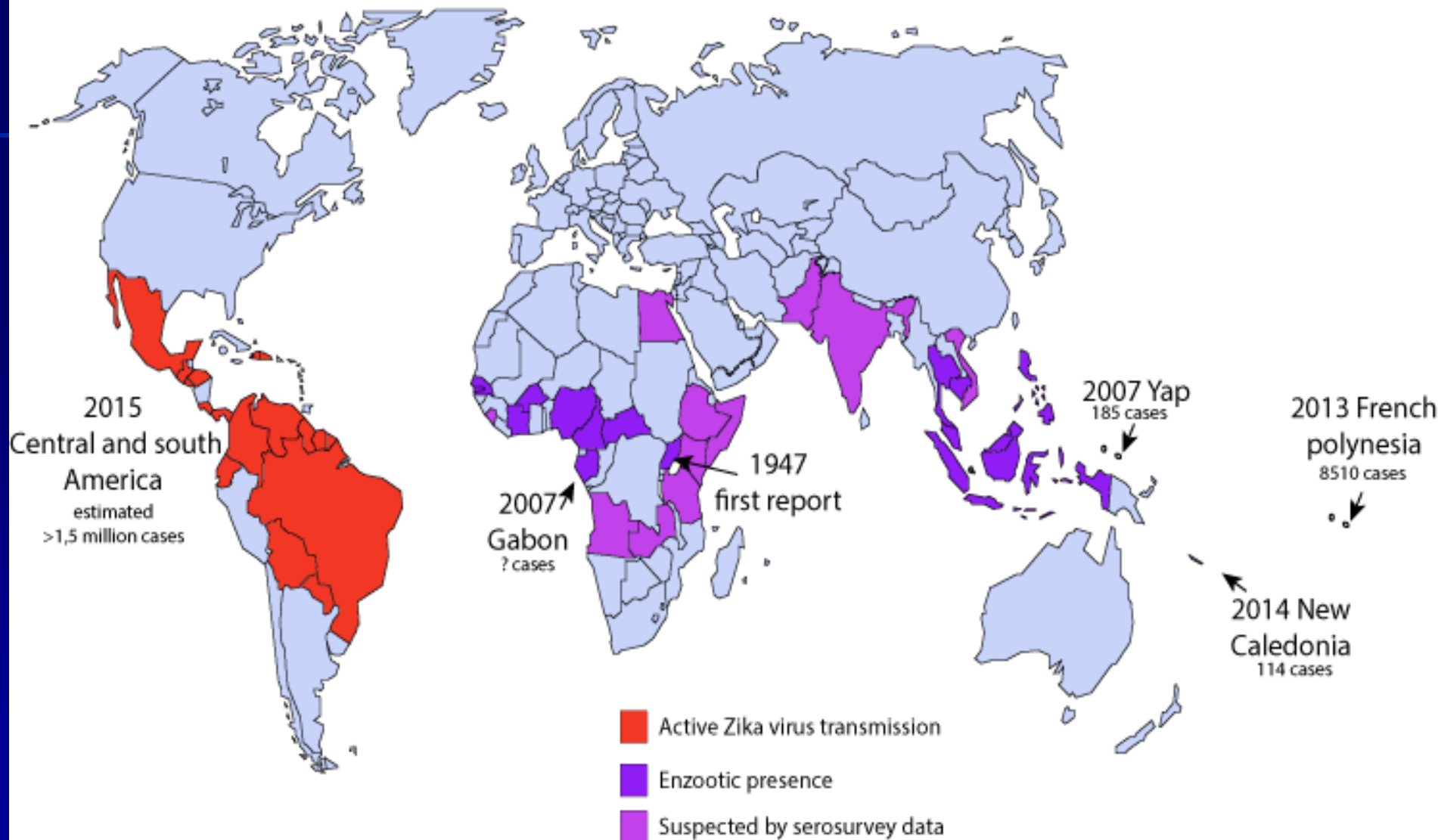
Where has Zika been found?

- Before 2015, Zika outbreaks occurred in Africa, Southeast Asia, and the Pacific Islands.
- Currently outbreaks are occurring in many countries and territories.



<http://www.cdc.gov/zika/geo/index.html>

Zika Virus Outbreaks, janvier 2016



Infants with confirmed or possible Zika infection

Doctors have found problems among fetuses and infants infected with Zika virus before birth, including

- Microcephaly
- Miscarriage
- Stillbirth
- Absent or poorly developed brain structures
- Defects of the eye
- Hearing deficits
- Impaired growth



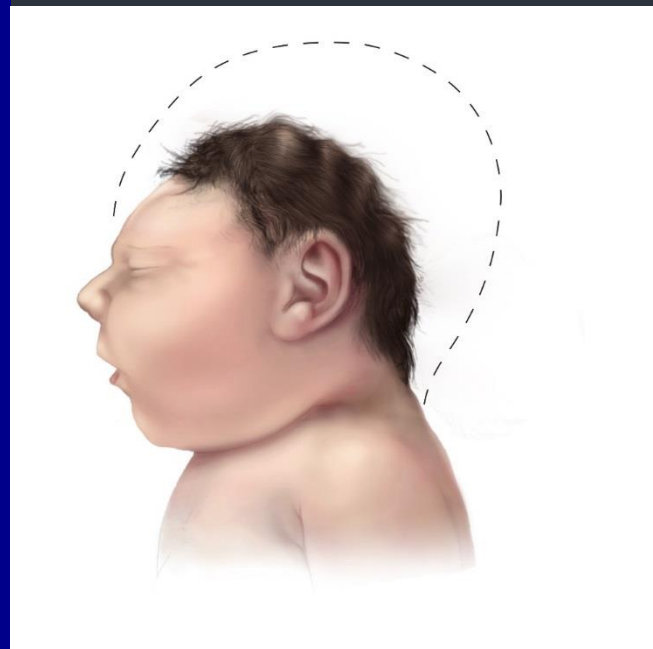
Case definition of microcephaly

Definite congenital microcephaly for live births

- Head circumference (HC) at birth is less than the 3rd percentile for gestational age and sex.
- If HC at birth is not available, HC less than the 3rd percentile for age and sex within the first 6 weeks of life

Definite congenital microcephaly for still births and early termination

- HC at delivery is less than the 3rd percentile for gestational age and sex.



How is Zika spread?

- Zika can be spread through
 - Mosquito bites
 - From a pregnant woman to her fetus
 - contact with an infected person
 - Laboratory exposure
- Zika may be spread through blood transfusion.
- No reports of infants getting Zika through breastfeeding



How can Zika affect pregnancies?

- Infection during pregnancy can cause microcephaly and other severe brain defects.
- Linked to other problems, such as miscarriage, stillbirth, and birth defects
- No evidence that past infection will affect future pregnancies once the virus has cleared the body

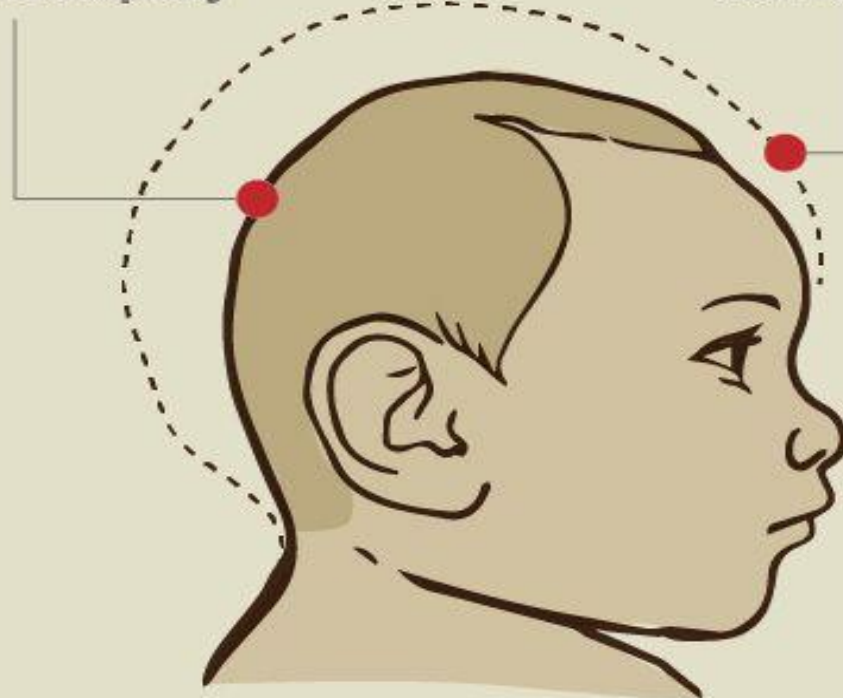


Researchers think Zika might be behind the rise of “microcephaly”

A birth defect that is associated with a small head and incomplete brain development in newborns

Microcephaly

Normal head



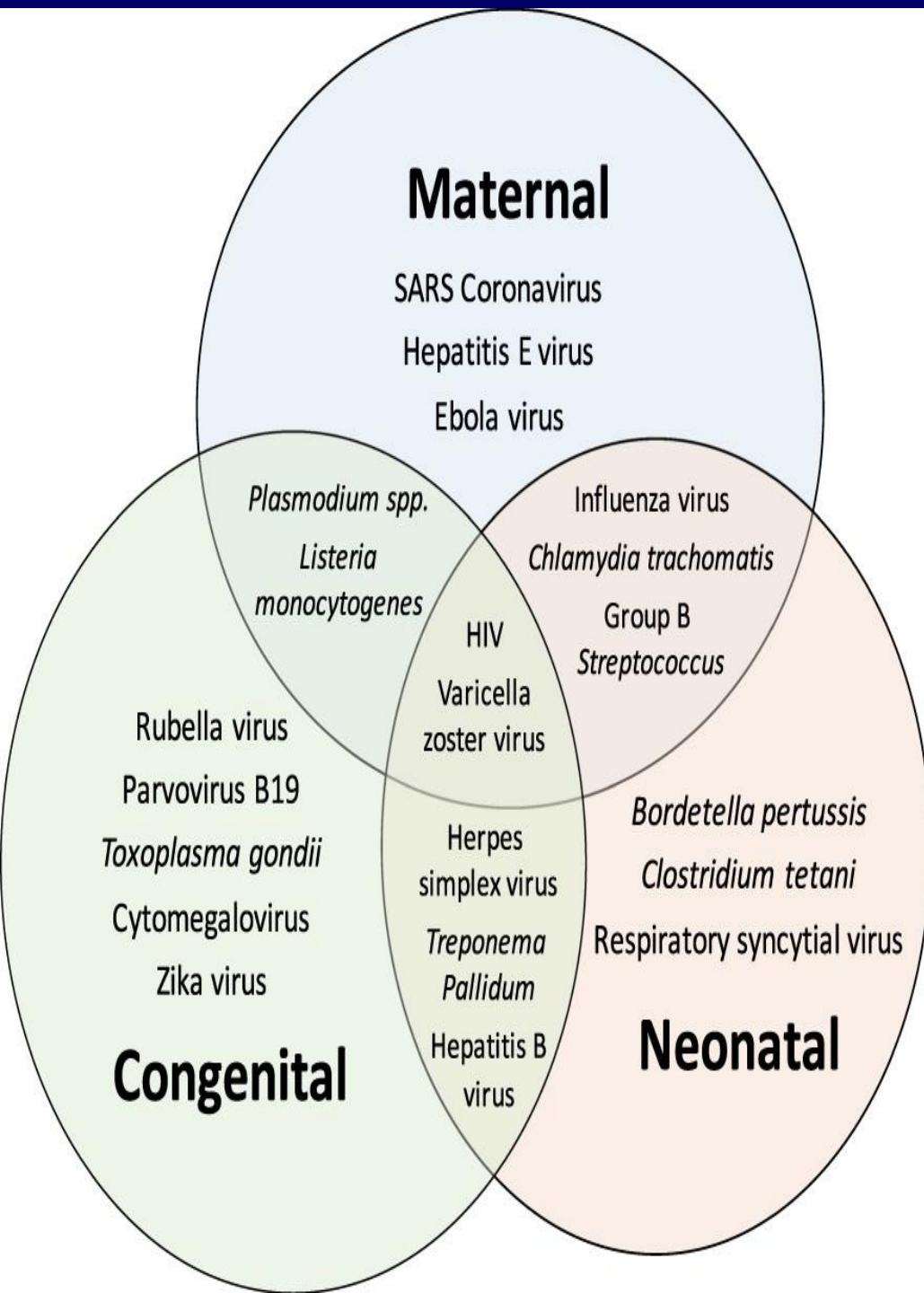
Vox

فیروس

زیکا

و خطورتہ





Pathogen Category	Goal of Vaccination	Optimal Timing of Vaccination
Maternal	Prevent maternal infection/disease	PRIOR to or DURING pregnancy
Congenital	Prevent fetal infection/disease	PRIOR to pregnancy
Neonatal	Prevent neonatal infection/disease	DURING pregnancy

Thank You!