VIRAL EXANTHEMS

Presented by:
Waleed AlSatari
Batool Samara
Rami Riad
Muhammad Al-Adwan

LECTURE CONTENTS

1

EXANTHEMS

What are Exanthems?
Skin rashes types

2

Common Exanthems

1-Erythema infectiosum (fifth disease)
2-Roseola infantum (sixth disease)
3-Chicken Pox
4-Hand-foot-and-mouth disease
5-Measles (Rubeola)
6-Rubella (German Measles)
7-Scarlet fever
8-Kwasaki disease

1

EXANTHEMS

What are Exanthems?

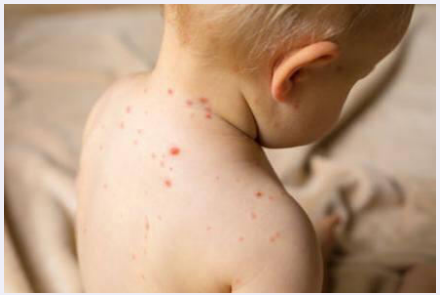
VIRAL EXANTHEMS (Rashes)

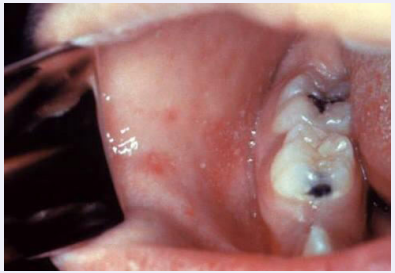
- It is an **eruptive skin rash** that may be associated with fever or other systemic symptoms, and often related to a viral infection.

- **Causes could be due to;**

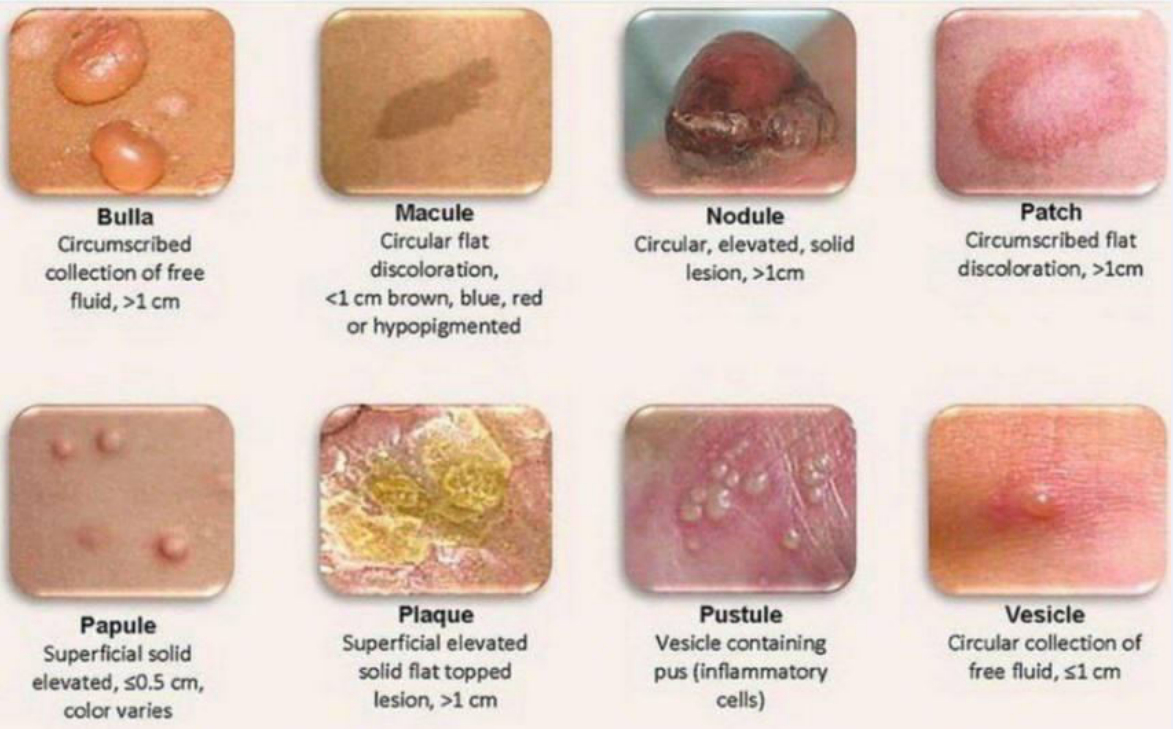
1- infectious pathogens which is the most common cause in children
2- medication reactions

- Enanthems: It is mucous membrane eruption

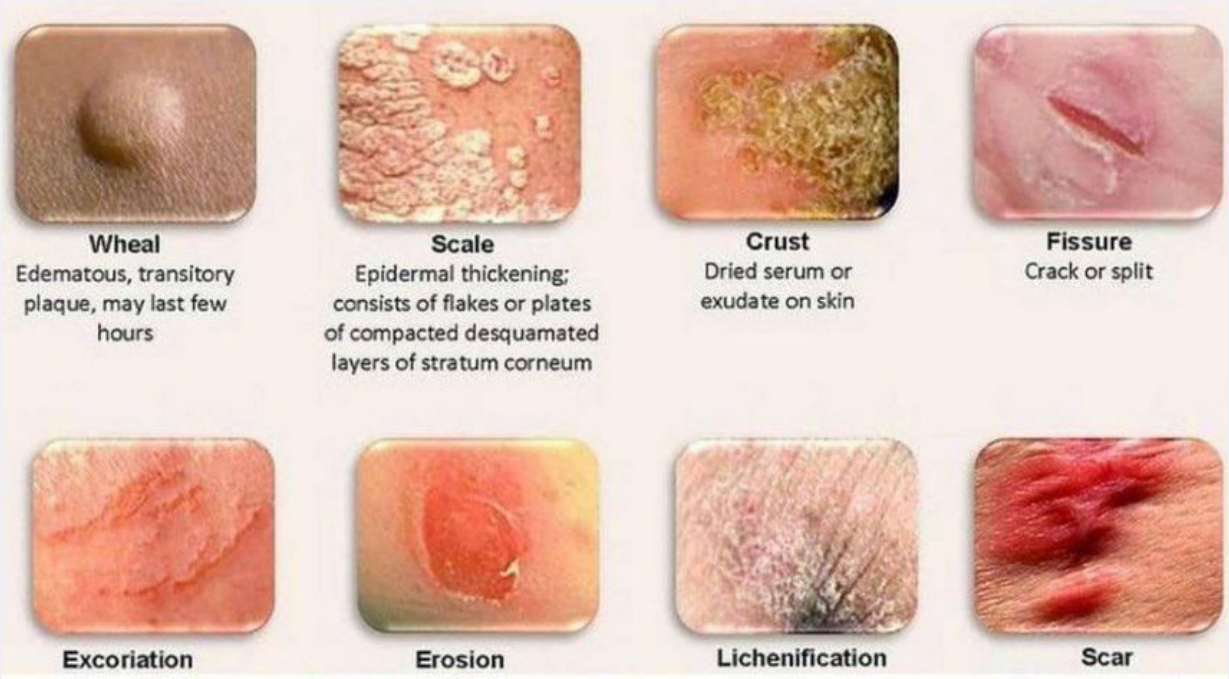




VIRAL EXANTHEMS (Rashes)



VIRAL EXANTHEMS (Rashes)



2

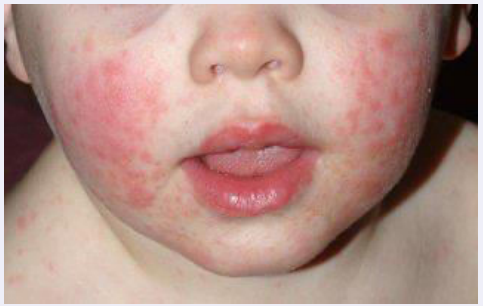
Common Exanthems

1-Erythema infectiosum (fifth disease)
2-Roseola infantum (sixth disease)
3-Chicken Pox
4-Hand-foot-and-mouth disease
5-Measles (Rubeola)
6-Rubella (German Measles)
7-Scarlet fever
8-Kwasaki disease

1-Erythema Infectiosum (Fifth Disease)

Etiology:

- **human parvovirus B19,** a single-stranded DNA virus producing a benign viral exanthem in healthy children
- The viral affinity for red blood cell progenitor cells makes it an important cause of **aplastic crisis in patients with hemolytic anemias**
- Parvovirus B19 also causes **fetal anemia** and **hydrops fetalis** after primary infection during pregnancy
- The cell receptor for parvovirus B19 is the **erythrocyte P antigen,** a glycolipid present on erythroid cells



Erythema Infectiosum (Fifth Disease)

Epidemiology:

- Parvovirus B19 seroprevalence is only 2-9% in children younger than 5 years
- Increases to 15-35% in children 5-18 years and 30-60% in adults
- The virus is transmitted by:

1. respiratory secretions
2. blood product transfusions

Erythema Infectiosum (Fifth Disease)

Clinical Manifestations:

- The **incubation period** is typically 4-14 days
- Parvovirus B19 infections usually begin with a mild, nonspecific illness characterized by:

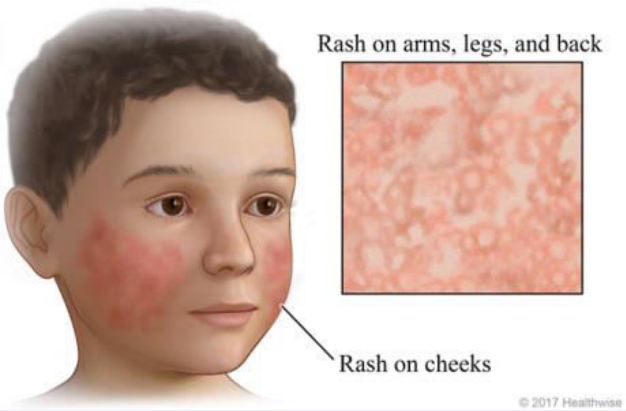
1. Fever
2. Malaise
3. Myalgias
4. Headache
5. In some cases, the characteristic rash appears 7-10 days later
6. Occasionally pharyngitis and mild conjunctivitis

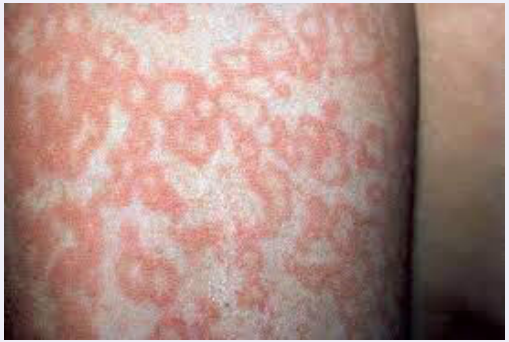
Erythema Infectiosum (Fifth Disease)

- **The rash appears in three stages:**

1. Initial stage is **“slapped cheek”** rash with circumoral pallor
2. An erythematous symmetric, maculopapular, truncal rash appears 1-4 days later
3. Distinctive lacy, reticulated rash that lasts 2-40 days (mean: 11 days)

- This rash may be pruritic but does not desequamate
- Adolescents and adults may experience myalgia, significant arthralgias or arthritis, headache, pharyngitis, and gastrointestinal upset





Erythema Infectiosum (Fifth Disease)

Laboratory and Imaging Studies:

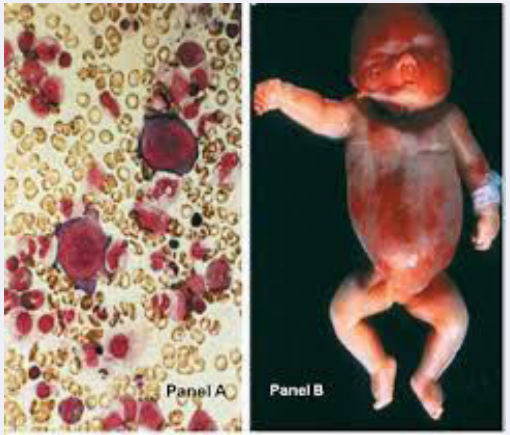
- Hematological abnormalities occur with parvovirus infection:
1. Reticulocytopenia lasting 7-10 days
2. Mild anemia
3. Thrombocytopenia
4. Lymphopenia
5. Neutropenia

- Parvovirus B19 can be detected by PCR
- Serological tests showing specific IgM antibody to parvovirus are diagnostic, demonstrating an infection that probably occurred in the prior 2-4 months

Erythema Infectiosum (Fifth Disease)

Treatment:

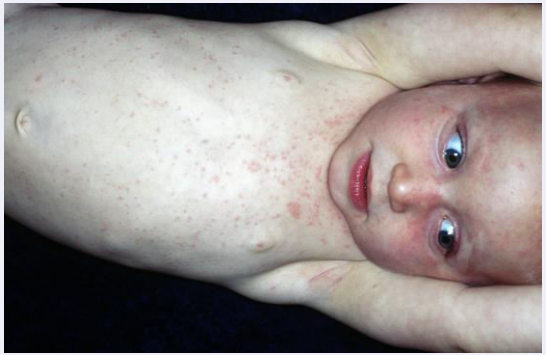
- supportive care
- Transfusions may be required for transient aplastic crisis
- Intrauterine transfusion has been performed for hydrops fetalis associated with fetal parvovirus B19 infection
- Intravenous immunoglobulin may be used for immunocompromised persons with severe anemia or chronic infection



2-Roseola Infantum (Exanthem Subitum)

Etiology:

- **Roseola infantum** (exanthem subitum, **sixth disease**)
- caused primarily by human herpesvirus type 6 (HHV-6), and by HHV-7 in 10-30% of cases.
- HHV-6 and HHV-7 are large, enveloped double-stranded DNA viruses
- infect mature mononuclear cells and cause a relatively prolonged (3-5 days) viremia
- They can be detected in the saliva of healthy adults, which suggests, as with other herpesviruses, the development of lifelong latent infection and intermittent viral shedding



Roseola Infantum (Exanthem Subitum)

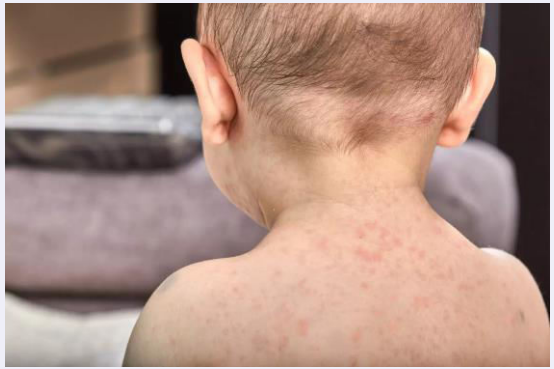
Epidemiology:

- Transplacentally acquired antibody protects most infants until 6 months of age
- The incidence of infection increases as maternally derived antibody levels decline
- By 12 months of age, approximately 60-90% of children have antibodies to HHV-6, and essentially all children are seropositive by 2-3 years of age

Roseola Infantum (Exanthem Subitum)z

Clinical Manifestations:

1. high fever (often >40°C) with an abrupt onset that lasts 3-5 days
2. A maculopapular, rose-colored rash erupts coincident with disappearance of fever
3. Upper respiratory symptoms, nasal congestion, erythematous tympanic membranes, and cough may occur
4. Gastrointestinal symptoms are described
5. Associated with approximately one third of febrile seizures
6. Rash starts on trunk then spreads to the extremities



Roseola Infantum (Exanthem Subitum)

Laboratory and Imaging Studies:

- Routine laboratory findings are nonspecific
- Encephalitis with roseola is characterized by pleocytosis (30-200 cells/mm3) with mononuclear cell predominance
- elevated protein concentration
- Serological testing showing a fourfold rise in acute and convalescent sera or documentation of HHV-6 DNA by PCR in the cerebrospinal fluid is diagnostic
- PCR has also been used to detect HHV-6 in blood but may not be sensitive in primary infection

Roseola Infantum (Exanthem Subitum)

Treatment:

- Routine supportive care includes maintaining adequate hydration and antipyretics
- In immunocompromised hosts, use of ganciclovir or foscarnet can be considered

Roseola Infantum (Exanthem Subitum)

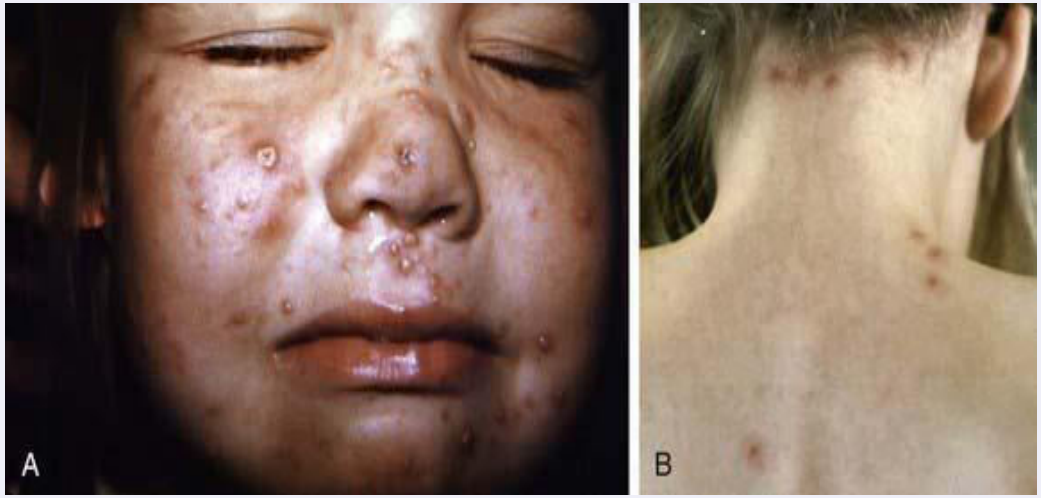
Complications and Prognosis:

- The prognosis for roseola is excellent
- A few deaths have been attributed to HHV-6, usually in cases complicated by encephalitis or virus-associated hemophagocytosis syndrome

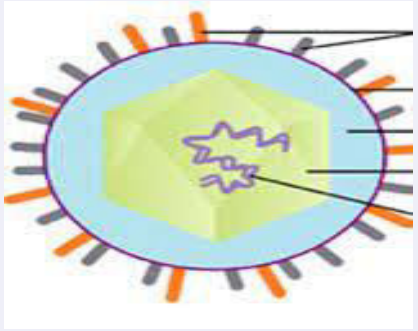
Prevention

- No preventative measures are available

“CHICKENPOX”



Etiology ?



- Chickenpox are caused by **varicella-zoster virus (VZV),**

- Varicella-zoster virus (VZV) causes primary(manifested as chickenpox) , latent, and reactivation(manifested as herpes zoster) infections.

Epidemiology ?

- In the prevaccine era,
  - the peak age of occurrence was 5-10 years,
  - with peak seasonal infection in late winter and spring.
- In the postvaccine era:
  - the incidence of varicella has declined in all age groups, with the peak incidence now in 10-14-year- olds.

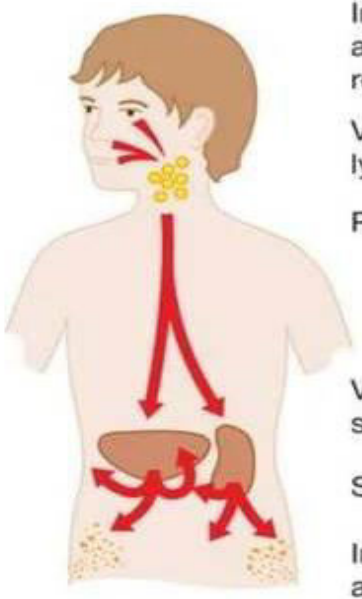
Epidemiology ?

- Varicella is a more serious disease in :
  - *young infants*,
  - *adults*,
  - *immunocompromised persons.*

- Transmission :
  - by direct contact, droplet, and air.
- Chickenpox is highly contagious.
  - Persons with varicella may be contagious 24-48 hr. before the rash is evident and until vesicles are crusted, usually 3-7 days after onset of rash.

Pathogenesis ?

- Primary infection (varicella) results from -



Infection of conjunctivae and/or mucosa of upper respiratory tract — Day 0

Viral replication in regional lymph nodes

Primary viremia — Day 4–6

Viral replication in liver, spleen and (?) other organs

Secondary viremia

Infection of skin and appearance of vesicular rash — Day 14

Incubation period

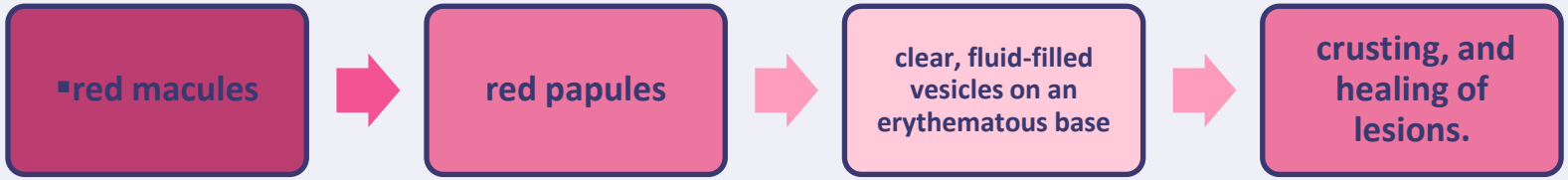
Clinical Manifestations ?

- The incubation period of varicella is 10-21 days after exposure.
- Prodromal symptoms:
  - fever.
  - Malaise.
  - Anorexia
  - Headache.
  - Mild abdominal pain.
  - ✓ these symptoms precede the rash by 1-2 days& resolve within 2-4 days after the onset of the rash.

Clinical Manifestations ?

- Varicella lesions:

- lesion stages:

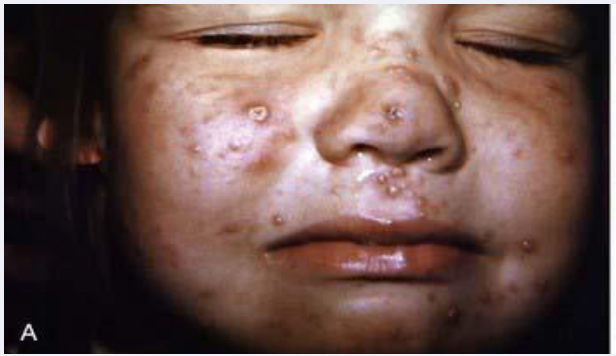


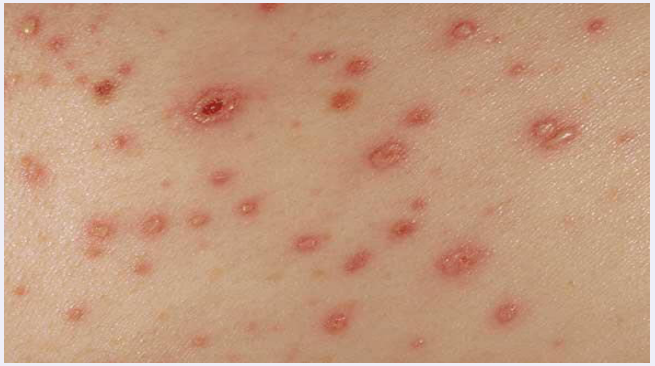
  - the presence of lesions in various stages of evolution at the same time is characteristic of varicella.
  - intensely pruritic.

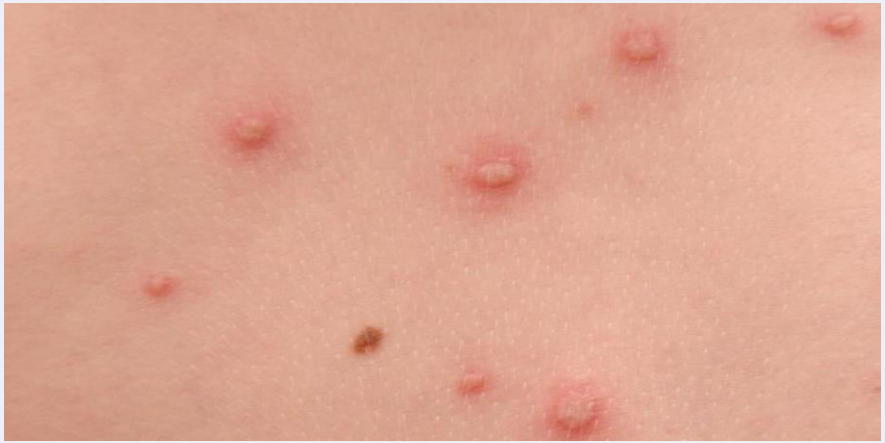
Clinical Manifestations ?

- "Varicella lesions" cont..:
  - **Distribution**: most on the trunk ,head, and the face, and, less commonly, the extremities.
  - Lesions may be present on mucous membranes.
  - new crops of lesions may continue to develop for more than 7 days.

Clinical Manifestations ?







Diagnosis ?

- Clinical diagnosis.
- PCR(diagnostic method of choice).
- Direct immunofluorescence.
- Rapid culture with specific immunofluorescence staining(shell vial technique).
- Tzanck smear.
- Serology(VZV IgG antibody).

Complications ?

- Secondary Bacterial Infection (caused by S.aureus or group A streptococcus)
- Encephalitis and cerebellar ataxia
- Pneumonia
- Herpes zoster
- Myocarditis, peri- carditis, orchitis, hepatitis, ulcerative gastritis, glomerulonephritis, and arthritis
- Reye syndrome

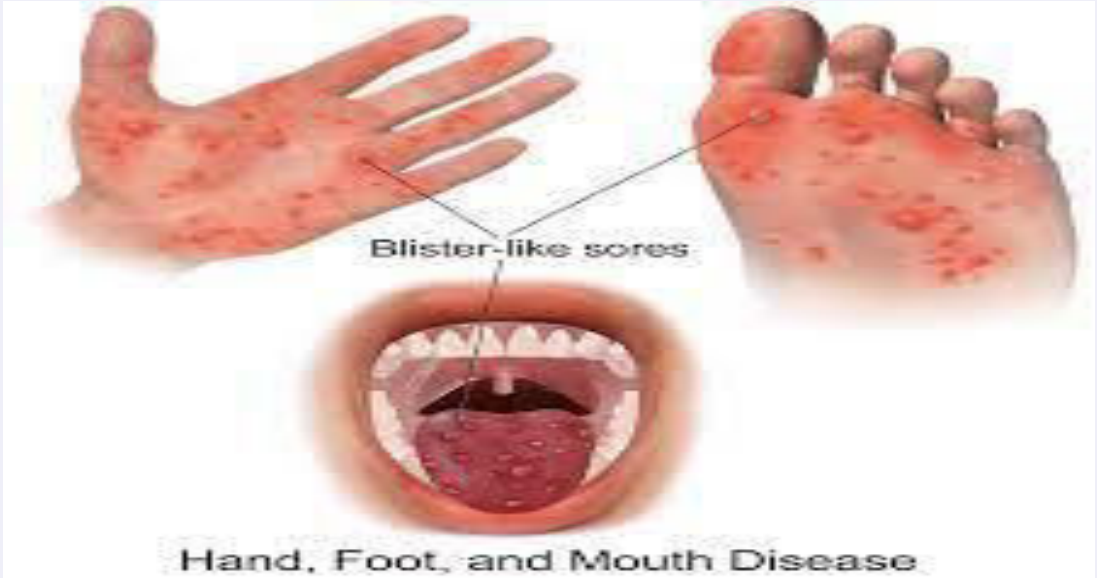
TREATMENT ?

- Symptomatic therapy of varicella includes:
  - nonaspirin antipyretics.
  - cool baths.
  - careful hygiene.
- Antiviral therapy:
  - Acyclovir is not recommended routinely for treatment of healthy children with varicella.
  - Acyclovir may be considered in those at risk of severe varicella.

Vaccine ?

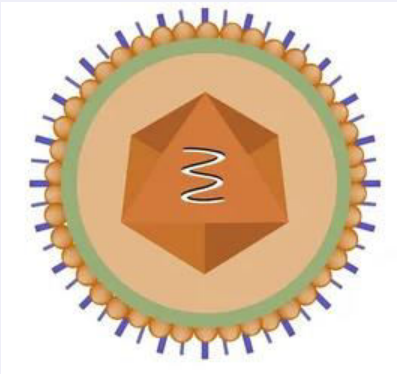
Chicken Pox Vaccine Dose	Chicken Pox Vaccine Age
1st Dose	12–15 months of age
2nd Dose	4–6 years of age (may be given earlier, min gap of 3 months after the 1st dose)
People not been vaccinated earlier or Chicken Pox Vaccine for adults	People 13 years of age and older should get two doses at least 28 days apart.

“Hand-Foot-and-Mouth Disease(HFMD)”



Etiology ?

- HFMD is most frequently caused by **coxsackievirus A16.**
- It can also be caused by enterovirus A71(more severe); coxsackie A viruses 5, 6, 7, 9, and 10; coxsackie B viruses 2 and 5; and some echoviruses



Clinical Manifestations ?

- Prodromal symptoms:
  - low-grade fever.
  - Malaise.
  - Reduced appetite.
  - Nausea & Vomiting.

Clinical Manifestations ?

- Eruption of vesicular rash:
  - scattered, painful vesicles that may ulcerate, leaving shallow lesions with surrounding erythema on the tongue, buccal mucosa, posterior pharynx, palate, gingiva, and/or lips.
  - Maculopapular, vesicular, and/or pustular lesions may occur on the hands and fingers, feet, and buttocks and groin.
    - ✓ more commonly on the hands than feet
    - ✓ and are more common on dorsal surfaces, but frequently also affect palms and soles.
    - ✓ Hand and feet lesions are usually tender.
    - ✓ vesicles that resolve in about 1 wk.
    - ✓ Buttock lesions do not usually progress to vesiculation.

Clinical Manifestations ?

- **Atypical hand-foot-and-mouth disease** (Coxsackievirus A6):
  - Relatively severe.
  - Affecting adults and children
  - Characterized by:
    - fever, generalized rash (face, proximal extremities, and trunk, in addition to hands, feet, and buttocks), pain, dehydration, and desquamation of palms and soles.
- **Onychomadesis (nail shedding)** has been observed following coxsackievirus A6 and other coxsackievirus infections

Clinical Manifestations ?

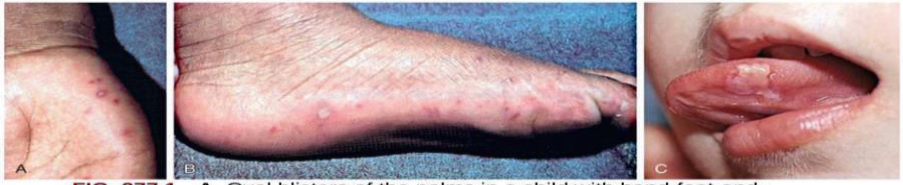


FIG. 277.1 **A,** Oval blisters of the palms in a child with hand-foot-and-mouth disease (coxsackievirus A16 infection). **B,** Oval blisters on the feet of a child with hand-foot-and-mouth disease. **C,** Erosion of the tongue in a child with hand-foot-and-mouth disease. (From Weston WL, Lane AT, Morelli JG: *Color textbook of pediatric dermatology* , ed 3, St. Louis, 2002, Mosby, p. 109.)

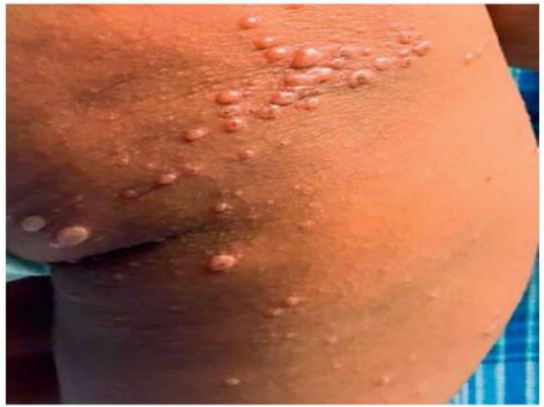
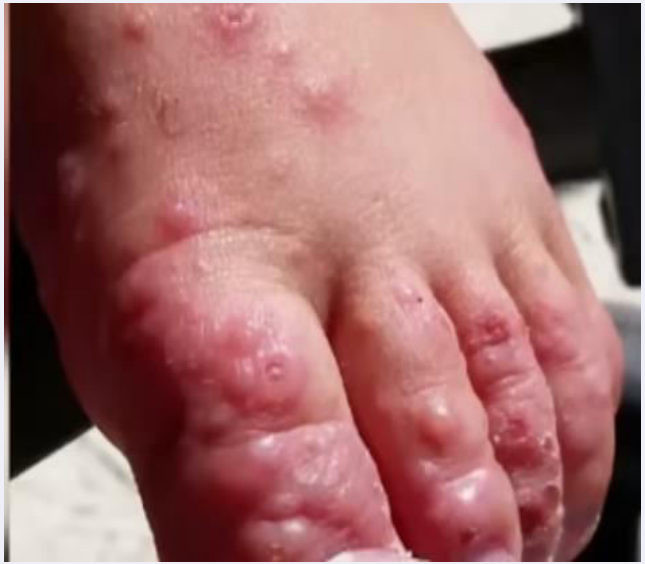
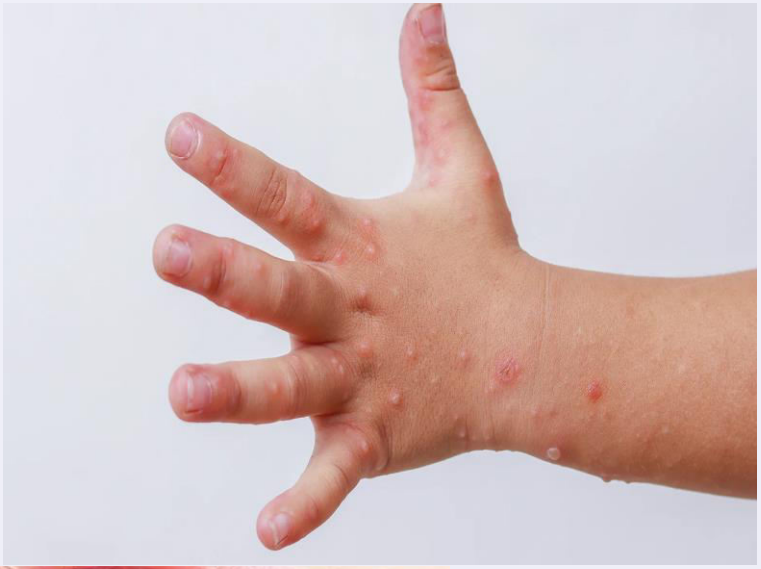
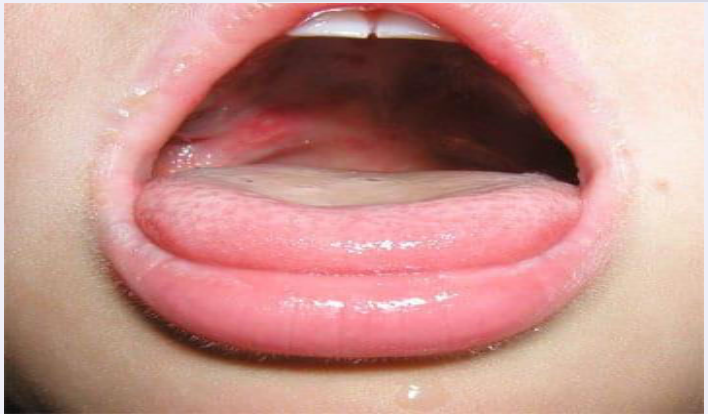


FIG. 277.2 **Atypical hand-foot-and-mouth disease. Vesiculobullous rash on the right buttock and posterior thigh. (From Waldman A, Thomas L, Thacker S, et al: Vesiculobullous eruption as an atypical hand, foot, and mouth presentation. *J Pediatr* 179:273, 2016, Fig. B.)**

Clinical Manifestations ?







Clinical Manifestations ?

Onychomadesis

Complications ?

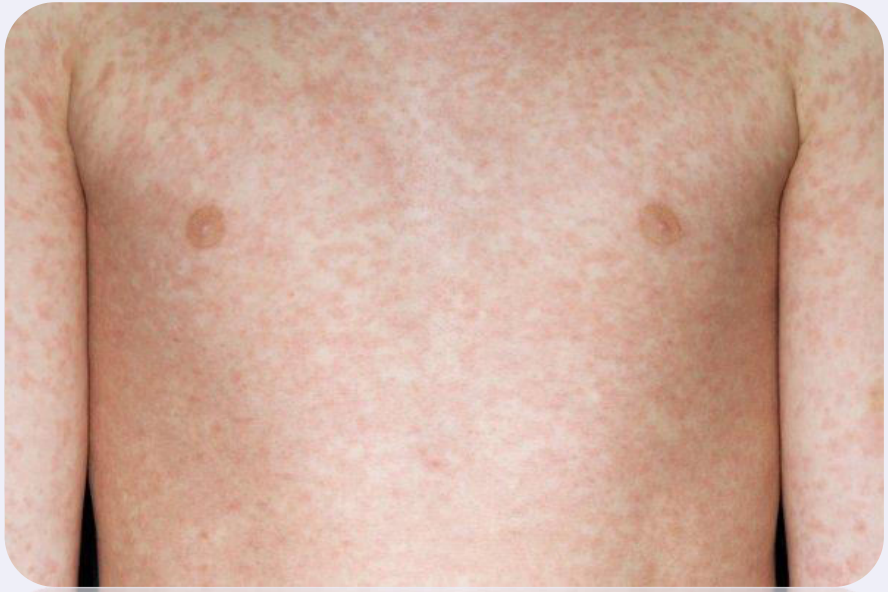
- **coxsackievirus A16** also can occasionally be associated with complications such as :
  - encephalitis, acute flaccid paralysis, myocarditis, pericarditis, and shock .

- **Enterovirus A71** can be associated with:
  - neurologic and cardiopulmonary involvement, especially in young children

Diagnosis & Treatment ?

- **Diagnosis**:
  - Clinical diagnosis.
  - RT-PCR.
- **Treatment:**
  - supportive care is the mainstay of treatment .
  - No antiviral use.

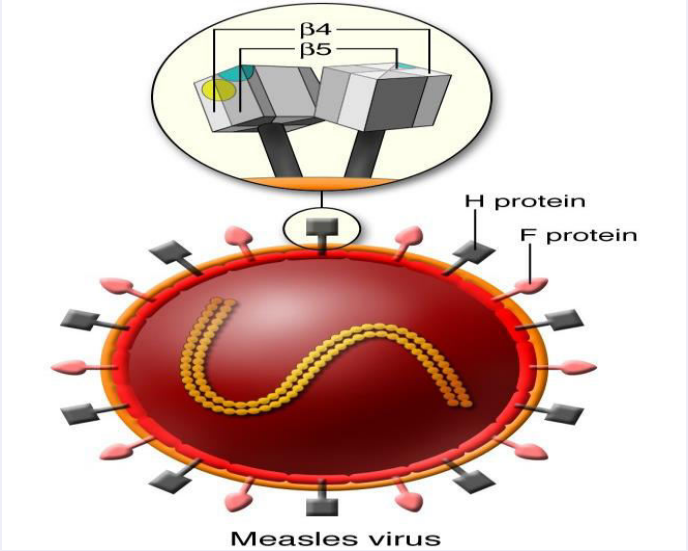
“Measles virus”



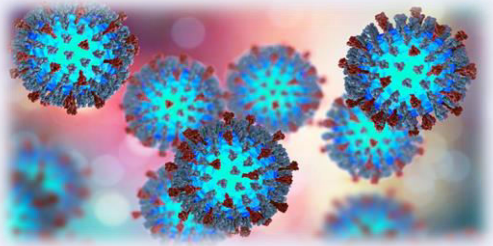
Etiology ?

- Measles is highly contagious, **single-stranded**, lipid-enveloped RNA virus in the family Paramyxoviridae.
- The World Health Organization recognizes **8 clades, A-H**, and 23 genotypes.
- Humans are the only host of measles virus.

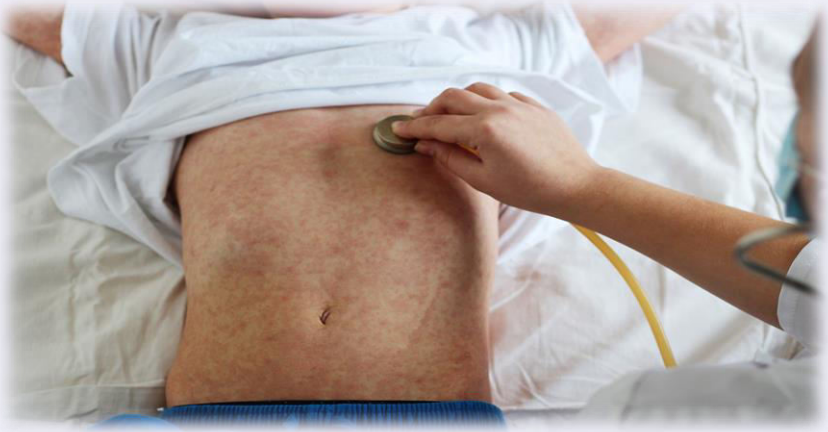
- Of the 6 major structural proteins of measles virus, the 2 most important in terms of induction of immunity are the **hemagglutinin (H) protein** and the **fusion (F) protein**.



Epidemiology?



- The measles vaccine has changed the epidemiology of measles dramatically.
- The current rate is <1 case per 1,000,000 population.



Transmission?

- The portal of entry of measles virus is through the **respiratory tract** or **conjunctivae** following contact with large droplets or small-droplet aerosols in which the virus is suspended.

- Patients are infectious from 3 days before to up to 4-6 days after the onset of rash.

Pathology?

- Measles infection causes **necrosis** of the **respiratory** tract epithelium and an accompanying lymphocytic infiltrate.

- In lymphoreticular tissue, lymphoid hyperplasia is prominent. Fusion of infected cells results in multinucleated giant cells, the **Warthin-Finkeldey giant cells** that are pathognomonic for measles.

- Measles infection consists of **4 phases:**

incubation period, prodromal illness, exanthematous phase, and recovery.

Clinical Manifestations?

- After an **incubation** period of **8-12 days**, the prodromal phase begins with a mild fever followed by the onset of conjunctivitis with photophobia, coryza, a prominent cough, and increasing fever.

- Of the major symptoms of measles, the **cough** lasts the **longest**, often up to **10 days**. In more severe cases, generalized lymphadenopathy may be present, with cervical and occipital lymph nodes especially prominent.

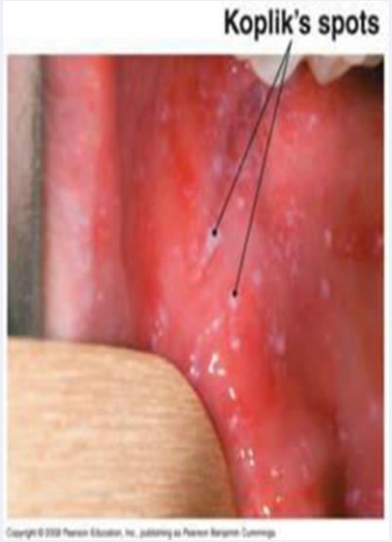

SYMPTOMS OF
MEASLES
• Dry Cough & Runny Nose
• Body Pains & Headache
• Sore Throat
• Watering & Swelling in Eyes
• Discomfort & Fatigue
• Loss of Appetite
• Diarrhea
• Light Sensitivity
• Inflammation in Lymph Nodes
• Koplik's Spots (blue & red spots in the mouth)

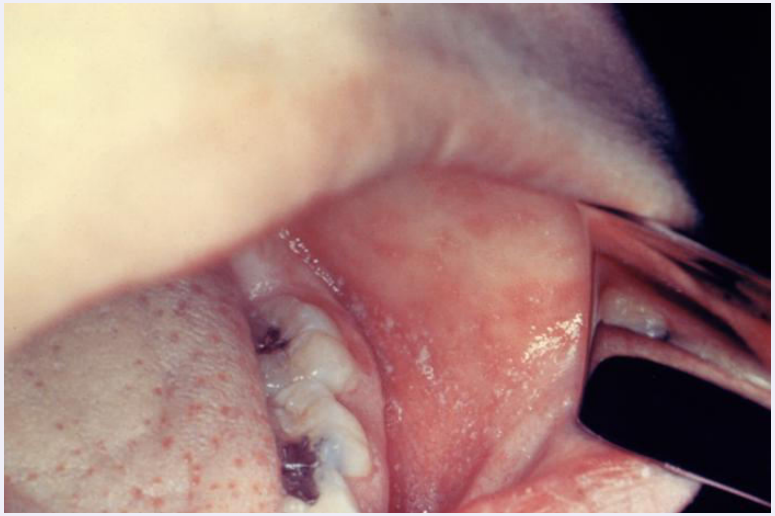
Koplik spots

- represent the enanthem and are the pathognomonic sign of measles, appearing **1-4 days prior** to the onset of the rash

- They also may occur in conjunctival folds and in the vaginal mucosa.

Koplik spots have been reported in 50–70% of measles cases but probably occur in the great majority.





Laboratory Findings?

- The diagnosis of measles is almost always based on **clinical** and epidemiologic findings.
- Laboratory findings in the acute phase include **reduction** in the total white blood cell count, with lymphocytes decreased more than neutrophils. However, absolute neutropenia has been known to occur.
- In measles not complicated by bacterial infection, **the erythrocyte sedimentation rate** and **C-reactive protein** level are usually **normal**.

Diagnosis?

- Serologic confirmation is most conveniently made by identification of immunoglobulin **(Ig) M antibody in serum**. IgM antibody appears **1-2 days** after the onset of the rash and remains detectable for about **1 month.**
- Serologic confirmation may also be made by demonstration of a **4-fold rise** in **IgG** antibodies in acute and convalescent specimens collected 2-4 wk apart.
- **Viral isolation** from blood, urine, or respiratory secretions can be accomplished by **culture** at laboratories.
- Molecular detection by polymerase chain reaction **(PCR)**

Differential Diagnosis?

- Typical measles is unlikely to be confused with other illnesses, especially if **Koplik** spots are observed.

1. Rubella
2. Adenovirus infection
3. Enterovirus infection
4. Epstein-Barr virus infection
5. Exanthem subitum (in infants)
6. Erythema infectiosum (in older children).
7. Mycoplasma pneumoniae and group A streptococcus
8. Kawasaki syndrome

Complications?

- Morbidity and mortality from measles are greatest in individuals younger than 5 yr of age (especially **<1 yr of age**) and **older than 20 yr** of age.

- **Factors** associated with higher case fatality rates:

1. crowding
2. Severe malnutrition
3. Low serum retinol levels in children with measles
4. Among patients with malignancy

Complications?

1. Pneumonia
2. Acute otitis media
3. Diarrhea and vomiting and therefore Dehydration
4. Sinusitis and mastoiditis
5. Retropharyngeal abscess
6. Viral and/or bacterial tracheitis or bronchiolitis
7. higher rate of activation of pulmonary tuberculosis
8. Appendicitis or abdominal pain
9. Encephalitis

Subacute Sclerosing Panencephalitis

- A chronic complication of measles with a delayed onset and an outcome that is nearly always fatal.

- **After 7-10 years** the virus apparently regains virulence and attacks the cells in the central nervous system leading to an inexorable **neurodegenerative process**.

- Males 2:1 Females

- Clinical manifestations of SSPE

1. Irritability
2. Reduced attention span
3. Subtle changes in behavior or school performance
4. Involuntary movements and repetitive myoclonic jerks
5. Choreoathetosis
6. Coma and death

Subacute Sclerosing Panencephalitis

- **Diagnosis of SSPE**

1. measles antibody detected in cerebrospinal fluid
2. Characteristic electroencephalographic findings
3. typical histologic findings

- Cerebrospinal fluid analysis reveals normal cells but **elevated IgG and IgM** antibody titers

- Management of SSPE
  - is primarily **supportive** and similar to the care provided to patients with other neurodegenerative diseases.
  - **Isoprinosine** with or without interferon

Treatment?

- Management of measles is supportive because there is no specific antiviral therapy approved for the treatment of measles.
- Maintenance of **hydration**, **oxygenation**, and **comfort** are goals of therapy.

- Vitamin A
  - reduced morbidity and mortality from measles. Vitamin A therapy is indicated for **all** patients with measles.
  - Vitamin A should be administered once daily for 2 days.

Prevention?

- Patients shed measles virus from 7 days after exposure to 4-6 days after the onset of the rash. **Exposure** of susceptible individuals to patients with measles should be **avoided** during this period.

- Vaccination (most effective)

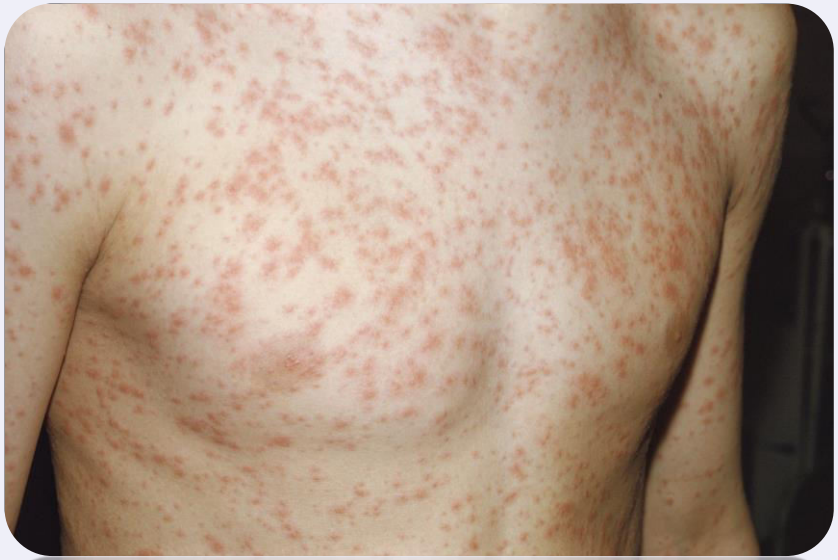
Vaccine

- Available as a combined vaccine with measles-mumps-rubella vaccine.
- The current recommendations include a 1st dose at **12-15 mo** of age, followed by a 2nd dose at **4-6 yr** of age. However, the 2nd dose can be given any time after 30 days following the 1st dose
- **Adverse events** from the measles-mumps-rubella vaccine include fever, rash, and rarely, transient thrombocytopenia.

Postexposure Prophylaxis

- Susceptible individuals exposed to measles may be protected from infection by either vaccine administration or with Ig.
- The **vaccine** is effective in the prevention or modification of measles if given within **72 hr** of **exposure**.
- **Ig** may be given **up to 6 days after exposure** to prevent or modify infection.

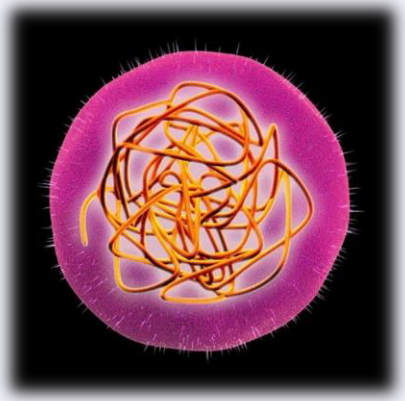
“ Rubella ”

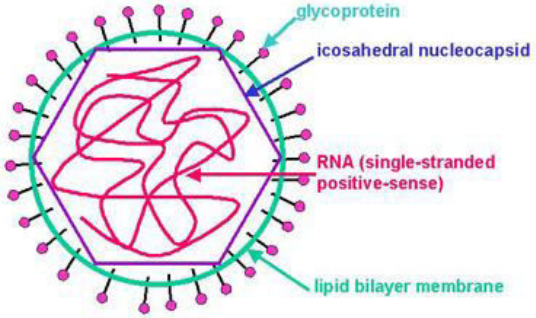


Etiology?

- Rubella (German measles or 3-day measles) is a **mild**, disease of infants and children that is typically more severe and associated with more complications in adults.

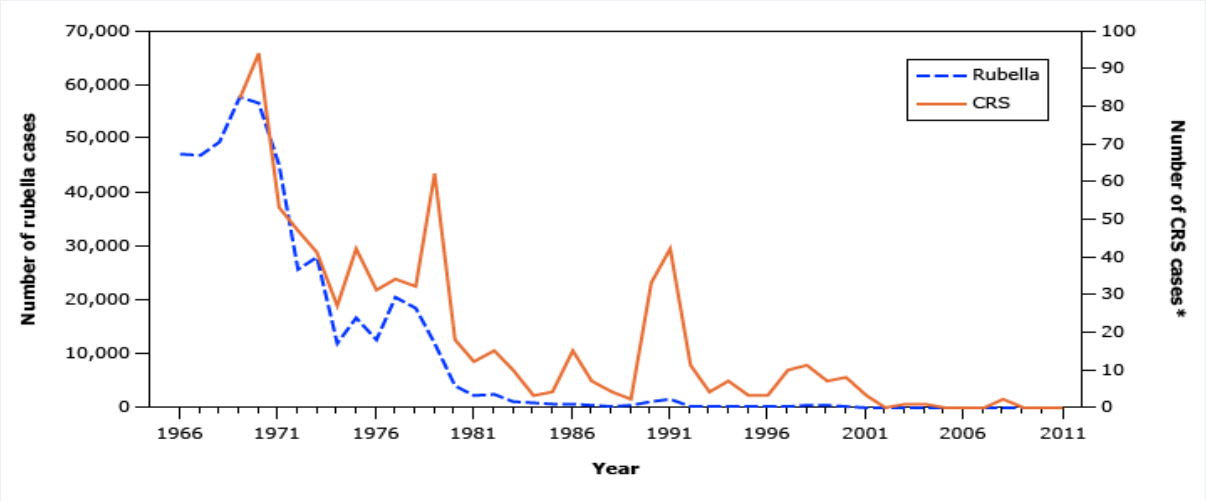
- It is a **single-stranded RNA** virus a member of the family Togaviridae and is the only species of the genus Rubivirus.





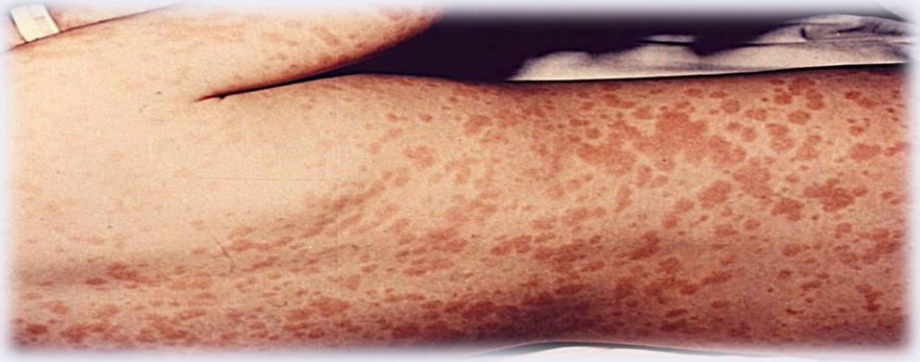
Prevalence?

- In the pre-vaccine era, rubella appeared to occur in major epidemics **every 6-9 year**, and was most common in preschool-age and school-age children.
- Now, 20 cases of rubella occur annually in the United States.



Pathogenesis?

- Following infection, the virus replicates in the **respiratory** epithelium and then spreads to regional **lymph nodes**.
- Viremia ensues and is most intense from 10 to 17 days after infection.
- The period of highest communicability 2 days before until 5 to 7 days **after rash** onset.



Clinical Manifestations?

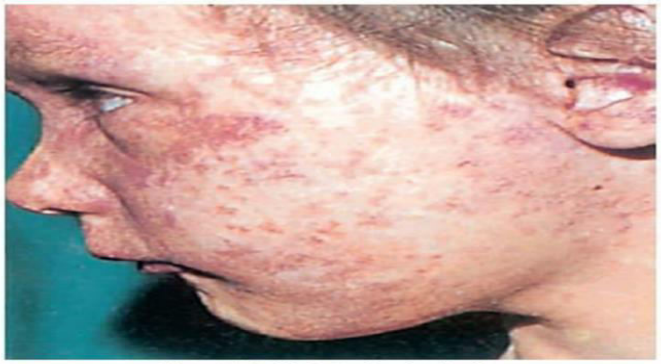


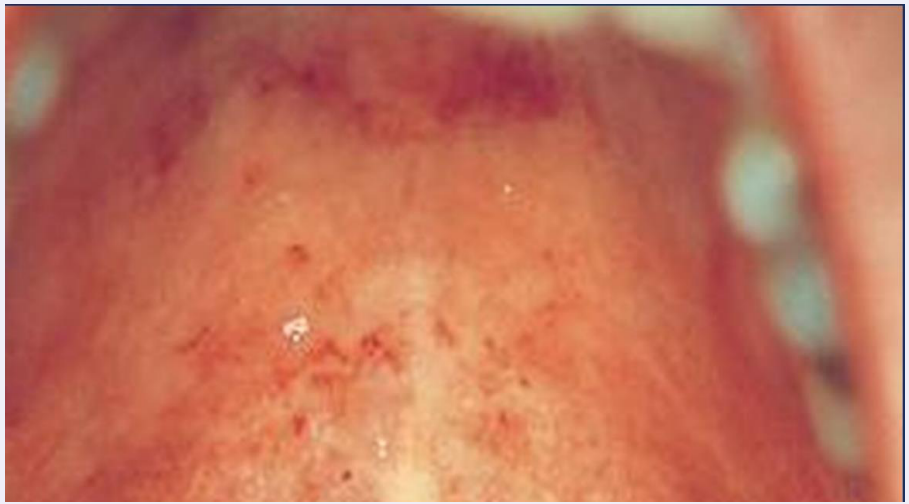
Figure 247-3 Rash of rubella.

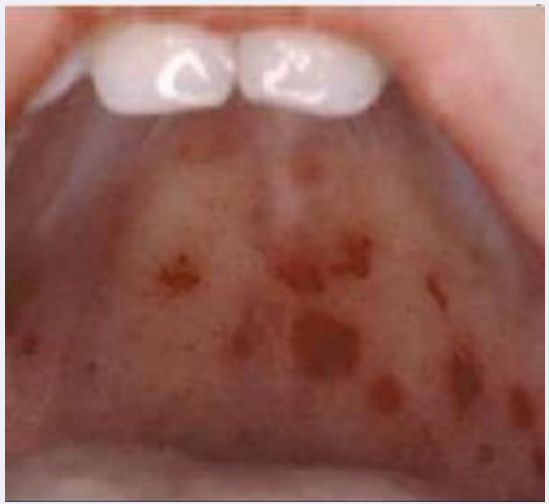
- Following an incubation period of **14-21 days**, a prodrome consisting of low-grade fever, sore throat, red eyes with or without eye pain, headache, malaise, anorexia, and lymphadenopathy begins.

- Lymph nodes that are most Prominent:
  - **Suboccipital**, **postauricular**, and **anterior cervical**

- The 1st manifestation of rubella is usually the rash, which
  - is variable and not distinctive.
  - It begins on the **face** and **neck**
  - The duration of the rash is generally **3 days**, and it usually resolves without desquamation.
  - 25- 40% of children may not have a rash in Subclinical infections

- Oropharynx may reveal tiny, rose-colored lesions (Forchheimer spots)





<u>Laboratory Findings:</u>

Leukopenia, neutropenia, and mild thrombocytopenia have been described during postnatal rubella.

Diagnoses?

- The most common diagnostic test is **rubella immunoglobulin (Ig) M enzyme immunosorbent assay**, which is typically present about 4 days after the appearance of the rash.
- **Viral isolation by culture** of nasopharyngeal secretions, urine in the newborn, or cord blood or placenta can be used to diagnose congenital infection.
- **PCR testing of amniotic fluid** during pregnancy is also an appropriate approach to diagnose congenital infection.

Differential Diagnoses?

1. Adenoviruses
2. parvovirus B19 (erythema infectiosum)
3. Epstein-Barr virus
4. Enteroviruses
5. Roseola
6. Mycoplasma pneumoniae
7. In severe cases, it may resemble measles. The absence of Koplik spots and a severe prodrome, as well as a shorter course, allow for differentiation from measles.

Complications?

- 1. **Thrombocytopenia**
  - Manifests as petechiae, epistaxis, gastrointestinal bleeding, and hematuria. It is usually self-limited.

- 2. **Arthritis**
  - classically involves the small **joints of the hands**. It is self-limited and resolves within weeks.

- 3. **Encephalitis**
  - ❑ Most serious complication of postnatal rubella.
  - ❑ Appears **within 7 days after** onset of the **rash**, consisting of headache, seizures, confusion, coma, focal neurologic signs, and ataxia.
  - ❑ Cerebrospinal fluid may be **normal**
  - ❑ Most patients recover completely, but **mortality** rates of **20%.**

Complications?

- 4. **Progressive rubella panencephalitis**
  - An extremely **rare** complication of either acquired rubella or CRS.
  - It has an onset and course similar to those of the **subacute sclerosing panencephalitis** associated with measles.
  - Unlike Encephalitis, rubella **virus** may be **isolated** from brain tissue of the patient with PRP

- Rarely, Guillain-Barré syndrome and myocarditis are reported.

Congenital Rubella Syndrome

- Congenital infection occurs **during maternal viremia**. After infecting the placenta, the virus spreads through the vascular system of the developing fetus and may infect any fetal organ.

- The most important risk factor for severe congenital defects is the stage of **gestation** at the time of infection:
  - The risk for congenital defects has been estimated at **90%** for maternal infection **before 11 weeks** of gestation
  - Defects occurring **after 16 weeks** of gestation are **uncommon**, even if fetal infection occurs.

CRS Clinical Manifestations?

- **Nerve deafness** is the single most common finding among infants with CRS.
- **Retinal** findings described as salt-and-pepper retinopathy
- Unilateral or bilateral **cataracts**
- **Patent ductus arteriosus** is the most frequently reported cardiac defect, followed by lesions of the pulmonary arteries and valvular disease during the first 8 weeks of gestation.
- Interstitial pneumonitis
- Meningoencephalitis
- Glaucoma
- intrauterine growth restriction

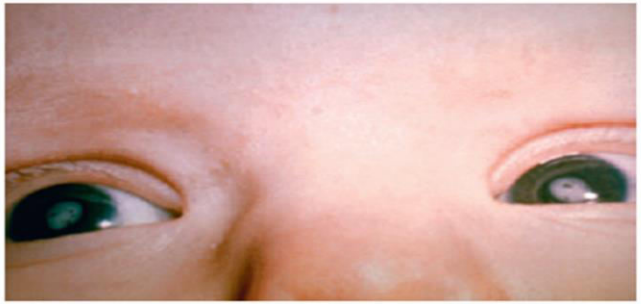


Figure 247-4 Bilateral cataracts in infant with congenital rubella syndrome.

Organ	Clinical Manifestation	Risk
Ear	Sensorineural deafness	60–75%
Eye	Cataract Glaucoma Retinopathy Microphthalmia	10–25%
Heart	Patent ductus arteriosis Ventricular septal defect Pulmonary stenosis Coarctation of the aorta	10–20%
Neurologic	Microcephaly Meningoencephalitis Mental retardation	10–25%
Other	Bone lesions Hepatosplenomegaly Thrombocytopenia with characteristic purpura (“blueberry muffin” appearance)	Variable

Treatment?

- There is no specific treatment available for either acquired rubella or CRS.

- Supportive Care
  - Requires no care beyond antipyretics and analgesics.
  - **Intravenous immunoglobulin** or **corticosteroids** can be considered for severe, non-remitting thrombocytopenia.

- Management of children with CRS is more complex.
  - Hearing screening is of special importance because early intervention may improve outcomes in children with hearing problems caused by CRS.

Prognosis?

- The prognosis of postnatal rubella is **good** with full recovery, while CRS may have a poor outcome with severe multiple-organ damage.

Prevention?

- Patients with postnatal infection should be **isolated** from susceptible individuals **for 7 days** after onset of the rash.
- Standard plus **droplet precautions** are recommended for hospitalized patients.
- Children with CRS may excrete the virus in respiratory secretions up to 1 year of age.

Vaccination?

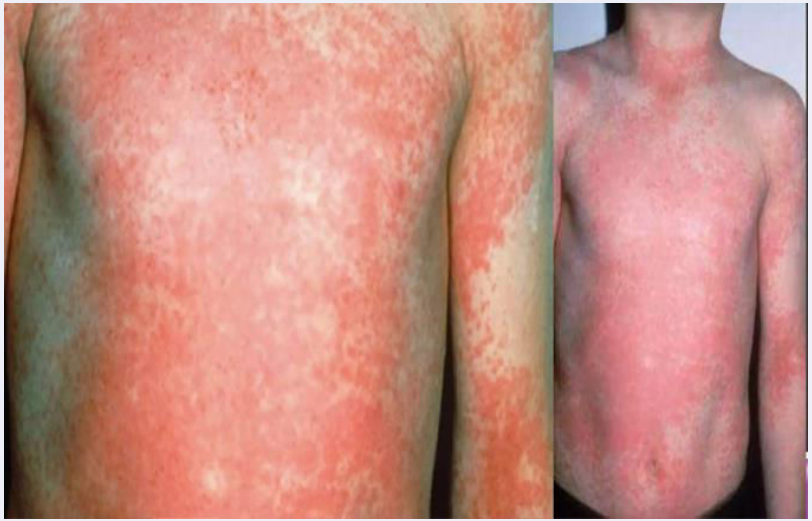
- Usually administered in **combination with measles and mumps** (MMR) or also with varicella (MMRV) in a 2-dose regimen at **12-15** month and **4-6** years of age.
- It theoretically may be effective as **post-exposure prophylaxis** if administered within **3 days** of exposure.
- Detectable **antibodies** remain for **15 years** in most individuals vaccinated following **1 dose**, and 91–100% had antibodies after 12-15 years after 2 doses.

- Vaccine should **not** be **administered** to severely immunocompromised patients (e.g., **transplant** recipients).

- Patients with **HIV infection** who are not severely immunocompromised may **benefit** from vaccination.

- Adverse reactions to rubella vaccination are uncommon in children:
  1. Fever
  2. Rash
  3. Arthralgia and arthritis
  4. Peripheral neuropathies and transient thrombocytopenia

“Scarlet Fever”



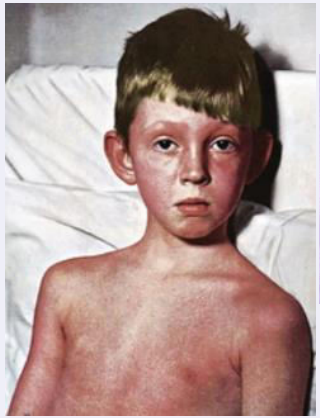
Scarlet Fever

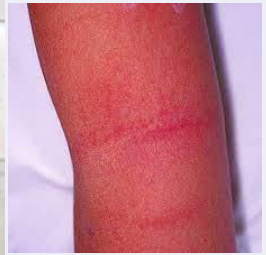
- It is GAS pharyngitis associated with a characteristic rash
- caused by an infection with pyrogenic exotoxin producing GAS in individuals who do not have antitoxin antibodies.

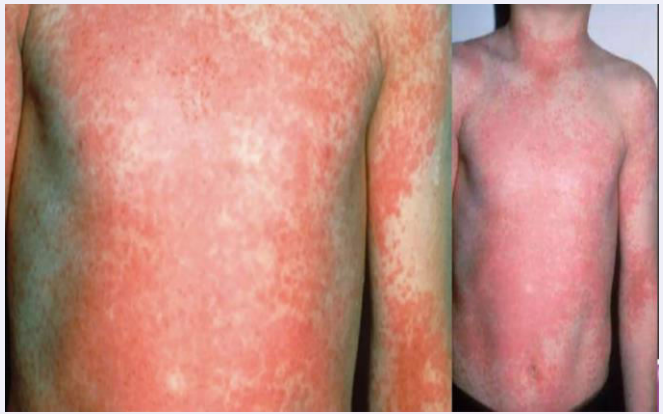
- The rash appears 1-2 days after the onset of symptoms, although it may appear with the first signs of illness

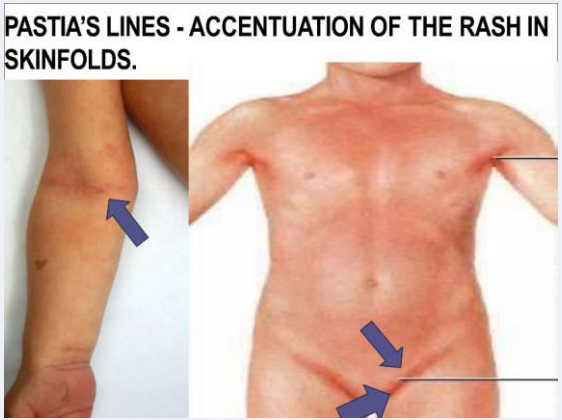
- It frequently starts at the neck and progresses to the trunk and limbs. The rash is a widespread, finely papular, erythematous eruption that causes the skin to become bright red and blanch under pressure.

- It is often accentuated in the creases of the elbows, axillae, and groin (Pastia lines).



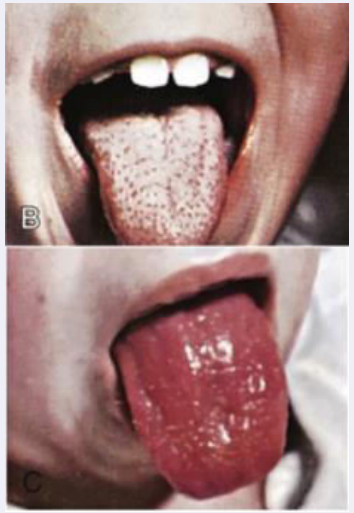





PASTIA'S LINES - ACCENTUATION OF THE RASH IN SKINFOLDS.

- After 3–4 days, the rash starts to fade and is then followed by desquamation, which usually looks like a minor sunburn and starts on the face

- the tongue is usually coated and the papillae are swollen.
- After desquamation, the reddened papillae are prominent, giving the tongue a strawberry appearance



Investigations?

- 1. Culture of a throat swab on a sheep blood agar plate:
- When performed correctly, It has a sensitivity of 90–95%.
- 2. Streptococcal rapid antigen detection
- 3. elevated or increasing streptococcal antibody titer. (anti–streptolysin O)
- 4.cbc: leukocytosis, high ESR CRP

Differential Diagnosis?

- Measles
- Rubella
- Drug induced exanthem
- Epstein-Barr virus

Treatment?

- For patients with classic scarlet fever, antibiotic therapy should be started immediately.

- Oral penicillin V (250mg/dose 2 or 3 times daily for children weighing ≤27 Kg and
- 500mg/dose 2 or 3 times daily for children >27 Kg) is recommended

- They must be taken for a full 10 days , even though there is symptomatic improvement within 3-4 days.

Complications?

1. Cervical lymphadenitis
2. Peritonsillar abscess
3. retropharyngeal abscess
4. otitis media
5. mastoiditis
6. Sinusitis
7. Acute rheumatic fever
8. acute poststreptococcal glomerulonephritis

Prognosis?

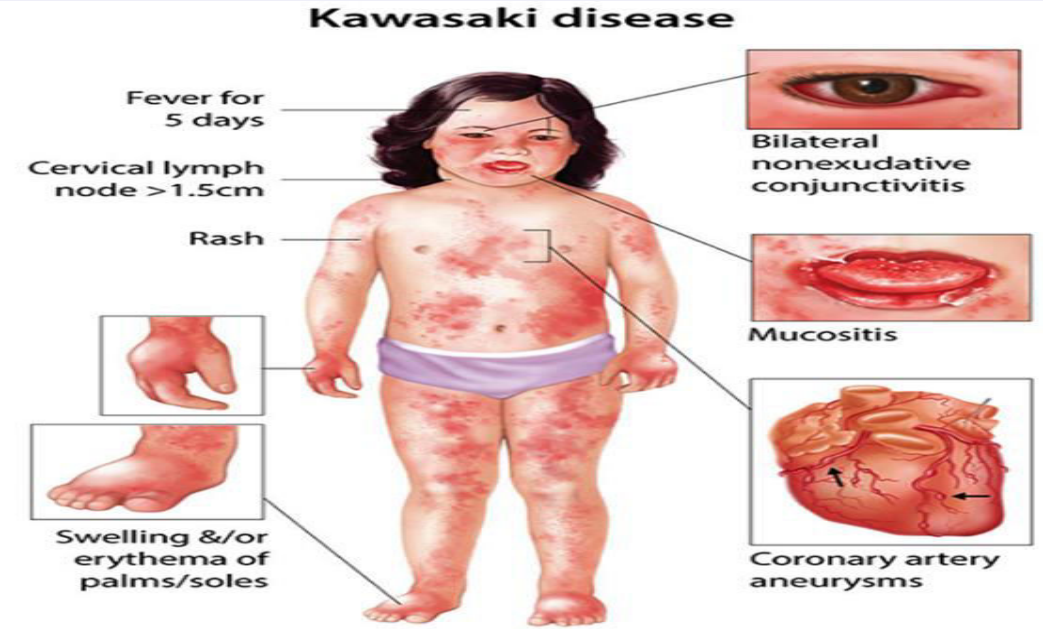
- The prognosis for appropriately treated GAS pharyngitis is excellent, and complete recovery is the rule. When therapy is instituted within 9 days of the onset of symptoms and continued for the full course, acute RF is almost always prevented.

Prevention?

- The only specific indication for long-term use of an antibiotic to prevent GAS infections is for patients with a history of acute RF and/or rheumatic heart disease.

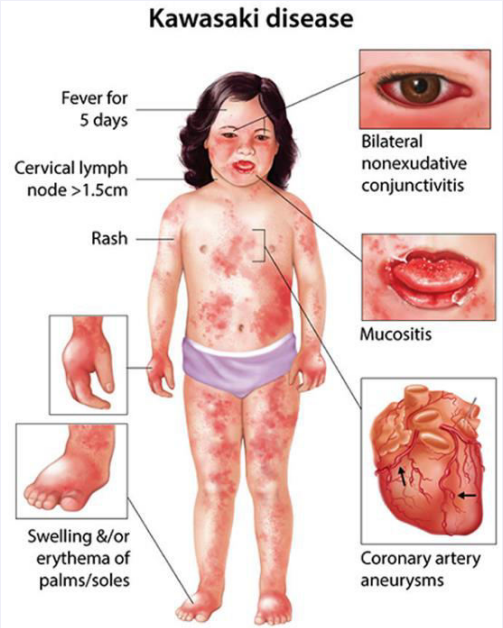
- Mass prophylaxis is generally not feasible except to reduce the number
- of infections during epidemics of impetigo and to control epidemics of
- pharyngitis in military populations and in schools.

“Kawasaki disease”



Kawasaki disease

- is an acute febrile illness of childhood seen worldwide, with the highest incidence occurring in Asian children.
- It is a systemic inflammatory disorder manifesting as a vasculitis with a predilection for the coronary arteries.

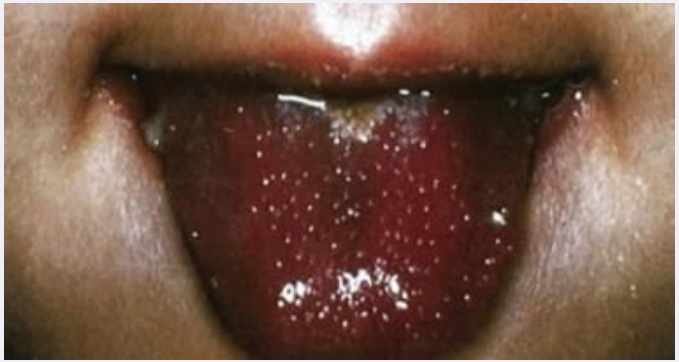


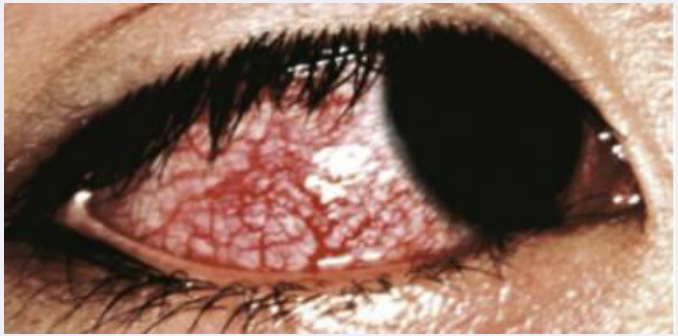
Epidemiology?

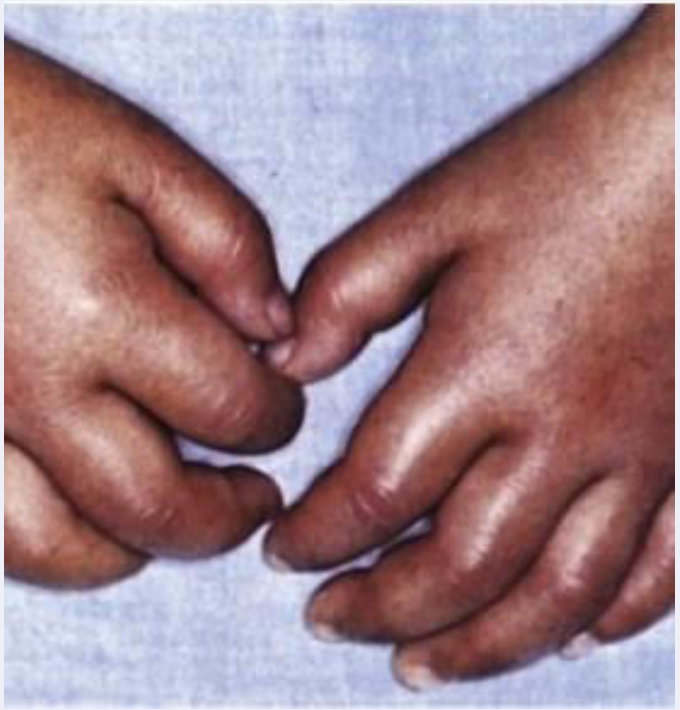
- KD is a disease of early childhood, with higher susceptibility in boys.
- Children age <5 yr had the highest annual hospitalization rates, and children of Asian ancestry had the highest rates among all racial groups.

Clinical Criteria?

- Fever ≥38.3℃ persistent for >=5 days
- Presence of at least four:
- bilateral nonexudative conjunctival injection
- Cervical lymphadenopathy
- erythema of the oral and pharyngeal mucosa with strawberry tongue and red, cracked lips
- rash
- Edema and erythema of the hands and feet;







Other clinical features

- Cardiovascular System:
- Myocarditis, pericarditis, valvular regurgitation, shock
- Coronary artery abnormalities
- Aneurysms of medium-sized noncoronary arteries
- Peripheral gangrene
- Aortic root enlargement

Other clinical features

- Respiratory System:
  - Peribronchial and interstitial infiltrates on chest radiograph
  - Pulmonary nodules
- Musculoskeletal System:
  - Arthritis, arthralgias (pleocytosis of synovial fluid)
- Gastrointestinal Tract:

Diarrhea, vomiting, abdominal pain
Hepatitis, jaundice
Hydrops of gallbladder
Pancreatitis

Other clinical features

- Central nervous system:
  - Extreme irritability
  - Aseptic meningitis (pleocytosis of cerebrospinal fluid)
  - Facial nerve palsy
  - Sensorineural hearing loss

- Genitourinary System:
  - Urethritis/meatitis, hydrocele

Differential Diagnosis?

Viral Infections	Bacterial Infections	Rheumatologic Disease	Other
Adenovirus	Scarlet fever	Systemic-onset juvenile idiopathic arthritis	Toxic shock syndromes
Enterovirus	Rocky Mountain spotted fever	Behçet disease	Staphylococcal scalded skin syndrome
Measles	Leptospirosis	Rheumatic fever	Drug hypersensitivity reactions

Laboratory findings?

- There's no diagnostic test but patients usually have characteristic laboratory findings:
- leukocytosis
- Normocytic, normochromic anemia
- The platelet count is normal in the first week and
- rapidly increases by the 2nd to 3rd week of illness
- Elevated ESR/CRP
- Elevated serum transaminases

Radiology

- Two-dimensional echocardiography is the most useful test to monitor for development of CAA.
- Echocardiography should be performed at diagnosis and again after 1-2 weeks of illness.

- If the results are normal, a repeat study should be performed 6-8 weeks after onset of illness.

Treatment?

- Acute Stage:
  - Intravenous immune globulin 2 g/kg over 10-12 hours
  - Aspirin 30-50 mg/kg/day or 80-100 mg/kg/day divided every 6 hr orally until patient is afebrile for at least 48 hours
- Convalescent Stage(when all symptoms and signs resolve):
  - Aspirin 3-5 mg/kg once daily orally until 6-8 weeks after illness onset if normal coronary findings throughout course

- Long-Term Therapy for Patients With Coronary Abnormalities:
  - Aspirin 3-5 mg/kg once daily orally
  - Clopidogrel 1 mg/kg/day
  - Warfarin or low-molecular-weight heparin for patients at high risk of thrombosis
- Acute Coronary Thrombosis:
  - fibrinolytic therapy with tissue plasminogen activator

Prognosis?

- The vast majority of patients return to normal health
- the risk of coronary aneurysms is <5% with timely treatment
- fatality rates are very low <1.0%.
- 50% of coronary artery aneurysms regress to
  - normal lumen diameter by 1-2 yr after the illness, with smaller aneurysms being more likely to regress.