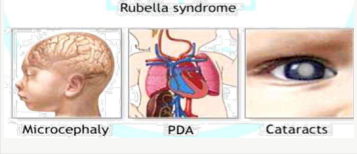
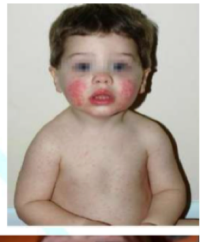
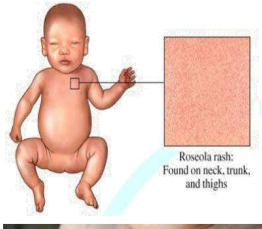
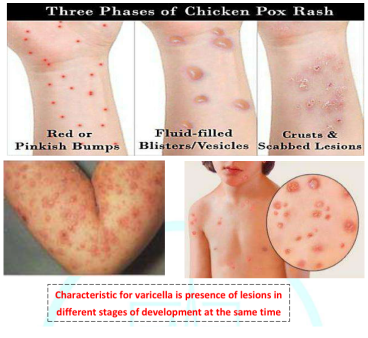
	Measles (rubeola)	German measles (Rubella)	
	Four Stages (IPER): **1. Incubation stage:** • usually lasts from (8-10) days the patient will be asymptomatic in this phase. **2. Prodromal Stage** • Duration: 4–7 days • Constitutional symptoms: fever, malaise, anorexia • The Three Cs: cough, conjunctivitis, coryza (appear ~3 days before rash) • Koplik spots: whitish spots on buccal mucosa, appear 48 hours before rash ◦ Pathognomonic for measles • Coryza: "head cold" – nasal congestion, rhinorrhea, sore throat **3. Exanthem Stage** • Rash: erythematous macules and papules start on the face → spread cephalocaudally and centrifugally ◦ By day 3, the whole body is usually involved • Fever: high (40–40.5°C) occurs 2–3 days after rash onset, then regresses • Clinical improvement: usually within 48 hours after rash appears ◦ Persistent fever beyond 48 hours → risk of complications **4. Recovery Stage** • Duration: ~2 weeks • Cough may persist 1–2 weeks • Persistent fever beyond 48 hours → risk of complications	**2. Postnatal Rubella** Clinical Presentation: • Duration: 1–5 days • ~50% asymptomatic • Young children: mild course • Older children/adults: prodromal symptoms, systemic complaints (e.g., arthritis), longer illness • Incubation: 2–3 weeks Findings: • Lymphadenopathy: retroauricular, posterior cervical, posterior occipital (up to 1 week) • Mild symptoms: low-grade fever, sore throat, conjunctivitis, headache, arthralgia • Forchheimer sign: small rose spots on soft palate (20% of patients; also seen in measles/scarlet fever) Exanthem Phase: • Duration: 2–3 days • Fine, nonconfluent pink maculopapular rash • Begins behind ears → trunk → extremities; palms/soles spared • Rash may be itchy in adults • Rash resolves from face by the time it reaches legs • Teen/adult females: 70% develop polyarthritis (usually hands, resolves without sequelae) • Paresthesias/tendinitis may occur	**1. Congenital Rubella Syndrome (CRS)** Epidemiology: • Rare in countries with widespread vaccination • Risk of fetal infection: ◦ Highest in first trimester ◦ Decreases in second trimester ◦ Slightly increases in third trimester • Infants may shed virus in nasopharyngeal secretions and urine for >12 months → can transmit to susceptible contacts Clinical Features: • Classic Triad: 1. Cardiac defect: most common (PDA, pulmonary artery stenosis) 2. Cataracts (other eye issues may appear later: salt-and-pepper retinopathy, glaucoma) 3. Cochlear defect: bilateral sensorineural hearing loss Early Features: • Hepatosplenomegaly, jaundice • Hemolytic anemia, thrombocytopenia • Petechiae/purpura ("blueberry muffin rash" due to extramedullary hematopoiesis) • Transient meningitis or encephalitis • Pneumonia Late Features: • CNS defects: microcephaly, intellectual disability, panencephalitis • Skeletal abnormalities • Endocrine disorders (diabetes, thyroid dysfunction) • Vascular disease • Immune defects Diagnosis: • Newborn & mother: ◦ PCR for rubella RNA ◦ Serology: persistent/high IgM and/or IgG • Fetus: ◦ IgM antibody detectable at ~3 months ◦ PCR for rubella RNA
Complications	High-risk groups: immunocompromised, pregnant women, malnourished, extremes of age 1. Subacute sclerosing panencephalitis (SSPE): late complication (9–10 years), behavioral, psychological, neurological symptoms (seizures, coma) 2. Pneumonia (most common fatal complication in children; most common overall in adults) 3. Bacterial superinfection 4. Gastroenteritis 5. Acute encephalitis 6. Giant cell pneumonia	• Chronic arthritis (especially in women) • Thrombocytopenic purpura • Rubella during pregnancy (TORCH infection): congenital rubella syndrome risk of IDDM. • Rare: rubella encephalitis, myocarditis, bronchitis, pericarditis, otitis • Deaths are rare (mainly in rubella encephalitis)	
Diagnosis	• Clinical diagnosis is primary • All suspected cases: serologically confirmed and reported immediately • Testing: ◦ Serology: Anti-measles IgM & IgG, measles virus isolation, RNA identification ◦ CBC: leukocytosis with lymphocytosis ◦ ELISA, PCR	• Clinical suspicion is common, but lab confirmation is needed in: pregnant women, encephalitis cases, or CRS risk assessment • Lab tests: ◦ Routine labs nonspecific ◦ CBC: normal or low WBC, relative lymphocytosis, increased plasma cells; thrombocytopenia rare • Confirmatory test: ◦ IgM antibodies (positive ~5 days after onset) ◦ ≥4-fold increase in IgG titer	
Treatment	• Uncomplicated measles: self-limiting (10–12 days), mainly supportive care 1. Supportive care: fluids, antipyretics, antibiotics • Malnutrition, immunosuppression, poor health, inadequate care → worse prognosis 2. Vitamin A supplementation: protective against respiratory and gastrointestinal complications (enhances mucosal immunity)	• No specific antiviral therapy • Supportive care: ◦ Severe pruritis → antihistamines ◦ Severe polyarthritis → rest, NSAIDs ◦ Fever → antipyretics ◦ Adequate hydration	
Prevention and Vaccination	• MMR vaccine: ◦ 1st dose: 12 months ◦ 2nd dose: 18 months • Measles-only vaccine: 9 months • Infected individuals: limit contact with others • Post-exposure prophylaxis: ◦ Within 3 days: vaccine ◦ Within 6 days: immunoglobulin	**Prognosis** • Usually benign • Rash resolves quickly • Arthralgia may persist several weeks; joint pain up to 1 month in adults **Vaccination** • Live rubella vaccine recommended • Usually as MMR: 12–15 months & 4–6 years • Contraindications: ◦ Immunocompromised ◦ High-dose corticosteroids (>2 mg/kg/day for >14 days) ◦ Pregnancy	
			

	Erythema Infectiosum (Fifth Disease)	Roseola Infantum (Exanthem Subitum / Sixth Disease)
	• Common childhood viral infection causing "slapped cheek" appearance. • Also called fifth disease or human erythrovirus infection.	
Etiology	• Caused by parvovirus B19. • Infects erythroid progenitor cells via P antigen, leading to erythroid aplasia.	• HHV-6 (primary), HHV-7 (10–30%) • Large, double-stranded DNA herpesviruses • Incubation: 5–15 days (avg 9 days)
Epidemiology:	• Common, with spring epidemics • Transmission: respiratory secretions, blood products, transplacental	• Children 6–15 months, mostly <5 years • Transmission likely via nasopharyngeal secretions • Life-long latent infection possible
	Clinical Scenarios: 1. Healthy child → benign viral exanthem 2. Children with hemolytic anemia (sickle cell disease, thalassemia, spherocytosis) → may develop transient aplastic crisis 3. Pregnancy: fetal infection can cause hydrops fetalis and fetal anemia Clinical Manifestations: • Incubation: 4–14 days (rarely 21 days) • Prodrome: mild flu-like illness (fever, malaise, myalgia, headache) → infectious stage • Rash (3 stages, non-infectious): 1. Stage 1: "slapped cheek" with circumoral pallor 2. Stage 2: symmetric erythematous maculopapular truncal rash → lacy/reticulated pattern (2–40 days, mean 11 days) 3. Stage 3: may recur with exercise, rubbing, bathing, or stress; pruritus may occur	Clinical Presentation: Prodrome: • High fever (up to 40–41°C) for 3–7 days • Mild URI symptoms, palpebral edema, cervical lymphadenopathy • Child often appears well Exanthem: • Appears as fever resolves (1–2 days) • Pink maculopapular rash: starts on trunk → neck → face & extremities • Rapidly fades in 1–3 days, non-pruritic, blanches on pressure • Enanthem (Nagayama spots): erythematous papules on soft palate/uvula (present in ~2/3 of patients)
Complications in Hemolytic Disorders:	• Transient aplastic crisis (7–10 days) → fever, lethargy, pallor, headache, malaise, GI/respiratory symptoms • Lab: reticulocytopenia, low Hb, possible neutropenia/thrombocytopenia • Persistent infection → pure red cell aplasia in immunocompromised patients	• Febrile seizures (6–15%) → main morbidity • Rare: encephalitis, fulminant hepatitis, disseminated infection • Prognosis: excellent; self-limited
Investigations	• Serology: IgM (acute), IgG (past infection) • PCR: blood, CSF, or bone marrow if severe or immunocompromised • Hematology: reticulocytopenia, mild anemia, lymphopenia, neutropenia, thrombocytopenia	• Routine labs nonspecific • Encephalitis: CSF pleocytosis (30–200 cells/mm³, mononuclear), ↑ protein, normal glucose • PCR for HHV-6 DNA in blood/CSF for severe cases
	• Supportive care • Transfusions for transient aplastic crisis • IVIG in immunocompromised patients • No specific antiviral therapy	• Supportive care (hydration, antipyretics) • Immunocompromised: consider ganciclovir or foscarnet
	Prevention: • Greatest risk: pregnant women • Hand hygiene reduces transmission • Exclusion from school not necessary (children not infectious once rash appears) Prognosis: • Excellent; fatalities are rare • In utero infection → hydrops fetalis, fetal death (≈10% of infected fetuses)	Prevention: • No vaccine or specific preventive measures
	   	    Rash fade rapidly without desquamation

	Chicken pox		Scarlet fever
Etiology:	• Caused by varicella-zoster virus (VZV), a double-stranded DNA herpesvirus. Transmission: • Respiratory secretions • Direct contact with skin lesions Pathogenesis: 1. Respiratory entry → replication in nasopharynx and lymph nodes 2. Viremia → multiple tissues infected, including sensory ganglia 3. Latent infection in dorsal root ganglia → reactivation causes herpes zoster (shingles)		• Group A Streptococcus (GAS) exotoxin-mediated disease • Usually follows strep throat; rarely impetigo or other infections • Not the same as rheumatic fever Epidemiology: • Children 5–15 years old
Clinical Features:	• Age: 5–9 years most affected • Incubation: 10–21 days • Prodrome: 1–2 days; low-grade fever, malaise, anorexia • Exanthem: ◦ Small red papules → vesicles → crusts ◦ Pruritus universal ◦ Centrifugal spread: chest/trunk/scalp → arms/legs ◦ Lesions may appear on mucous membranes (mouth, conjunctiva) ◦ New lesions appear daily for 5–7 days ◦ Total course ~10 days Characteristic: • Lesions in different stages of development simultaneously	Neonatal Chickenpox: • Occurs if maternal varicella 5 days before → 2 days after delivery • Risks: eye, brain, skin (cutaneous scars, microcephaly, chorioretinitis, cortical atrophy) • Management: 1. Varicella-zoster immune globulin (VZIG) for at-risk newborns 2. IV acyclovir for severe cases or immunocompromised infants	• Incubation: 1–4 days • Onset: Abrupt, fever, sore throat, headache, nausea, vomiting, myalgia, malaise • Rash: ◦ Appears 12–48 hours after fever ◦ Fine red sandpaper-like rash ◦ Starts on chest, axilla, behind ears; worse in skin folds → Pastia lines • Tongue: ◦ Day 1–2: white coating with red papillae → white strawberry tongue ◦ Day 4–5: coating disappears → red strawberry tongue • Other signs: circumoral pallor, follicular tonsillitis, lymphadenopathy Desquamation: • Peeling on groin, axilla, fingertips, toes • Begins 7–10 days after rash, may last up to 6 weeks Complications: • Usually benign • Rare complications from untreated GAS: ◦ Suppurative: peritonsillar abscess, sinusitis, bronchopneumonia, meningitis ◦ Immune-mediated: rheumatic fever, post-streptococcal glomerulonephritis
Diagnosis:	• Clinical • Confirmation: 1. Varicella-specific antigen in vesicle fluid (immunofluorescence) 2. Fourfold rise in acute and convalescent sera		• Throat culture: gold standard for GAS • ASO titer: evidence of recent GAS infection
Treatment:			• Penicillin: ◦ <12 years: 25–50 mg/kg/day PO divided TID/QID (max 3 g/day) ◦ ≥12 years: adult dosing • Penicillin allergy: erythromycin • No resistance to penicillin
	 Characteristic for varicella is presence of lesions in different stages of development at the same time	 	 Pastia Lines  Folicular Tonsilitis  Circumoral Pallor  Strawberry Tongue • Cc • Strawberry tongue • Circumoral pallor • High Fever (40c) • Folicular tonsilitis • Pastia line • Rash appear 12-48 h after fever onset   **Desquamation and peeling** on (groin axilla tips of fingers and toes), one of the most distinctive features of scarlet fever, begins **7-10 days** after resolution of the rash and may continue up to 6 weeks. (occur also in kawasaki)