PEDIATRICS

ROUTINE CHECK UPS & ANTICIPATORY GUIDANCE

- Weight check @ 3-4 days of life
- 2wks, 1mo, 2mo, 4mo, 6mo, 9mo, 1yr, 15mo, 18mo, 2yrs, 2.5yrs, 3 → annually
- Lead level check @ 9mo and 2yo, + 3yo if not low risk
- Rear-facing car seat until 2yrs if possible
- If breastfeeding, start Vit D ~2-4wks
- Wean off formula and start whole milk (16-20oz/d) at 1yr
- Start introducing solid foods ~6mo
- Start toilet training ~2yrs
- Target Height (most kids achieve height within 4in of target) measured in inches
 - Boys: (dad height + mom height + 5) / 2
 - Girls: (mom height + dad height 5) / 2

DOSING

- Ibuprofen: 4-10mg/kg//dose 3-4x/d (q6-8hrs)
 - Max single dose = 400mg/dose
 - Max daily dose = 40mg/kg/d up to 1200mg/day
- Tylenol 10-15mg/kg/dose 4x/d (q4-6hrs)
 - Max daily dose = 2.6g (do not exceed 5 doses)
- Amox 80-90mg/kg divided into 2 doses per day
- Tamiflu BID x5d for treatment vs. once daily x 10d for prophylaxis

VACCINES

- Kinrex = Dtap + polio (1 shot)
- Proguad = MMR + varicella
- Prevnar = pneumococcal conjugate
- Pediarix (3 dose series) = diphtheria, tetanus, pertussis, infection caused by all known subtypes of hepatitis B virus, and poliomyelitis
- Rotavirus (liquid) 1st dose must be given by 15wks, all doses must be given by 8months

NEWBORNS

- Newborn cord typically comes out ~1wk to 10d
- Strabismus should resolve by 4mo of age → ophtho referral if not
- Newborn feeding always ask about amount of formula/milk! ~100cc/kilo/day
- Newborn weight
 - expected gain: 1oz/day
 - expected loss: 5% of birthweight → >10% is concerning
 - Expect 4-6 lbs per year
- Latching → cross cradle vs. football (cross cradle hold breast with C and baby head with C)
- Newborn pooping Ok for some babies to poop q2-3 days if soft
- GERD → Zantac

SICK VISIT

- Strep pharyngitis always tx for 10 days proven duration to prevent rheumatic fever!
 - PCN is first line, but liquid PCN tastes gross → Amoxicillin x10d (rash w/ mono!)
 - PCN VK 500mg BID x10d (if pt can swallow pills)
 - New toothbrush in 24hrs after starting abx
 - Can return to school after being on abx for 24hrs (no longer contagious)
 - Many kids have stomach aches with strep
- Fusobacterium necrophorum pharyngitis (high CRP & WBC) → PCN + clindamycin
- **PNA** → typically treat with amox and azithro
 - Atypicals = mycoplasma, chlamydia, viral
 - Sx: insidious onset, dry cough, N/V/D, HA, myalgias, sore throat
 - tx: macrolides (azithro) or levofloxacin
- Otitis Media → Amox 90mg/kg/day divided into 2 doses x 10 days for younger kids
 - Agents: S . Pneumonia, H. influenza, M. catarrhalis, viral
 - 1st line = Amoxicillin (500-875mg BID x5-10d depending on severity)
 - 2nd line (if amox fails) → Augmentin x10d
 - PCN allergy → ceftriaxone (can give IM but kids don't like shots)
 - Beta-lactam allergy → macrolide (azithromycin, erythro)
 - Serous otitis media = OME = fluid behind TM without presence of infx
 - Usually result of previous AOM, barotrauma, chronic eustachian tube dysfxn
 - Common to have pharyngitis with OM
 - Recurrent OM = 3+ episodes of AOM in 6mo, or 4+ in 1yr
 - Perforated TM d/t infx → amoxicillin PO + Floxin otic drops
 - Mastoiditis = suppurative infx of mastoid air cells

- Otitis Externa

- Etiology: **bacterial (pseudo, strep, staph), fungal, eczema
- Sx: tragal pain, hearing loss, otorrhea, fullness, itching, recent exposure to water
- Tx: neo/poly/HC if TM intact, FQ (cipro/ofloxacin)
 - FQ will cover pseudomonas
- Malignant OE = osteomyelitis of temporal bone as a result of chronic infection in DM, not cancerous! → emergent ENT referral

- Conjunctivitis

- Bacterial typically unilateral, viral typically affects 2nd eye 24-48hrs later, allergic = itchy
- If infant, think chlamydia or gonorrhea
 - Recommended standard prophylaxis given immediately after birth includes erythromycin ointment, topical tetracycline, silver nitrate, or povidone-iodine
 - IM ceftriaxone needed once infection has occurred
- 50% of peds conjunctivitis is bacterial, so tx w/ abx even if suspect viral
- *Ocuflox drops 1 drop 4xd until clear for 2 days (use for one day past when it looks better)
- Polytrim (polymyxin B sulfate and trimethoprim ophthalmic solution, USP) x5d
- Bronchiolitis → inflammation of bronchioles causing wheezing & airway obstruction
 - **RSV, rhinovirus, adenovirus, influenza, parainfluenza
 - CXR → hyperinflation, interstitial pneumonitis, infiltrates
 - prednisone 20mg tablet TID x5d
 - albuterol sulfate HFA 90 mcg/actuation aerosol inhaler 2 puffs q4hrs prn
- $Croup \rightarrow cool mist humidifier, steroids, epi at hospital if severe$
 - **parainfluenza virus, RSV

- Stridor, hoarseness, barking cough, lowgrade fever, rales, ronchi, wheezing (worse in pm)

- Allergic Rhinitis

- Samter's triad = syndrome of aspirin sensitivity, nasal polyposis, and asthma often seen with allergic rhinitis, frequently leading to severe pansinusitis
- URI → dexamethasone*, neb tx
- Pertussis → macrolide is DOC (azithro), bacrim as alternative
- Orbital Cellulitis typically d/t rhinosinusitis (m/c ethmoid sinusitis) → vanc + zosyn/unasyn/ceftriaxone

COMMON COMPLAINTS

- Bedwetting → 11pm wake up, bedwetting alarm (pottypager), DDAVP
- Risk factors for oral candidiasis → ICS, abx use, AIDS → KOH prep
 - Tx: oral nystatin rinses (or swabs if infant) or systemic fluconazole if severe
- ASTHMA Spo2 may go decrease slightly after neb tx → okay! d/t V/Q mismatch

TORCH INFX

- Toxoplasmosis
- Other Syphilis
- Rubella
- CMV
- Herpes HSV 1 (predominantly oral-labial) & HSV 2 (predominantly genital)
 - o If mom has active vaginal infx, child has 50% of transmission
 - Does NOT increase risk of congenital malformations
 - o complication vesicles, resp distress, seizure, meningoencephalitis, impaired neuro devel
 - HSV 1 more common in infancy → widespread, severe herpetic gingivostomatitis with oral erosions
 - eczema herpeticum occurs when infant w/ preexisting eczema develops a disseminated HSV infx; can be life-threatening, requires immediate IV acyclovir
 - \circ Dx \rightarrow tzank smear ("multinucleated giant cells"), viral cx, antibody staining

ACUTE RHEUMATIC FEVER

- Sequela that happens within weeks (~2-4wks) after GAS tonsillopharyngitis
- Jones criteria → 2 major, or 2 minor + 1 major, manifestations w/ evidence of preceding GAS infx
- 5 major manifestations: **carditis & valvulitis**, **arthritis** (usually migratory polyarthritis of large joints), CNS involvement (**chorea**), **subcutaneous nodules**, **erythema marginatum**
- 4 minor manifestations: arthralgia, fever, elevated ESR/CRP, prolonged PR interval
- Acute febrile illness (m/c) vs. neurologic illness (less common, slower onset)
- Carditis: pancarditis that can involve the pericardium, epicardium, myocardium, and endocardium; the predominant manifestation of carditis is involvement of the endocardium presenting as a valvulitis, especially of the mitral and aortic valves; usually presents within 3wks of GAS infection. The presence of valvulitis is established by auscultatory findings together with echocardiographic evidence of mitral or aortic regurgitation
- Late sequela = RHD and Jaccoud arthropathy
 - RHD usually occurs 10 to 20 years after the original illness, although it may present earlier after a severe or recurrent episode of ARF. It is the most common cause of acquired valvular disease in the world; MV>AV, mitral regurg ism/c finding; may progress to mitral stenosis

KAWASAKI DZ

- Generalized vasculitic dz of medium-sized arteries, unknown etiology but thought to be infectious
- m/c in Asians, <5yo
- Criteria = Fever for 5+ days with % of the following: :
 - Edema & erythema of hands & feet w/ periungual desquamation
 - Polymorphous exanthem (widespread rash, most pronounced in perineum)
 - Conjunctivitis (limbic sparing)
 - o Oropharyngeal mucosal changes (throat erythema, strawberry tongue, red cracked lips)
 - Cervical LAD >1.5cm (usually unilateral, nontender)
- Lab findings: increased ESR/CRP, thrombocytosis, normocytic anemia
- May also see sterile pyruria, inc. ALT/AST, RUQ pain (hydrops of GB)
- Cardiac findings:
 - Coronary artery aneurysm** (most serious complication)
 - Coronary arteritis (increased size or lack of tapering)
 - Decreased LV contractility
 - Pericardial effusion
 - Mild valvular regurgitation
- Treatment
 - High dose IVIG and aspirin
 - Echocardiogram surveillance
 - \circ Without tx in first 10d \rightarrow 25% develop coronary artery aneurysm
 - \circ With trx \rightarrow 5% develop coronary artery dilation, 1% aneurysm

Diagnostic features of Kawasaki disease Fever (for more than 5 days) Red, dry, cracked lips and inflamed tongue Swollen lymph nodes Widespread rash Widespread rash

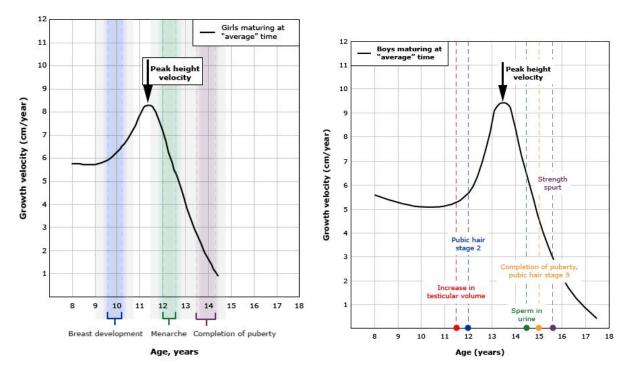
HYPERTROPHIC CARDIOMYOPATHY

- A genetic cardiomyopathy caused by mutations of the cardiac sarcomere
- characterized by left ventricular hypertrophy of various morphologies, with a wide array of clinical manifestations and hemodynamic abnormalities
- Autosomal dominant inheritance → evaluate 1st degree relatives of affected individual
- Possible abnormalities: LV outflow obstruction, diastolic dysfunction, MR, MI
 - LVOT murmur = harsh crescendo-decrescendo systolic murmur that begins slightly after
 S1 and is heard best at the apex and lower left sternal border
 - MR murmur = mid-late systolic murmur at the apex
- Sx related to HCM can be categorized as those related to HF, chest pain, or arrhythmias.
 - o fatigue, dyspnea, chest pain, palpitations, syncope/presyncope
- Patients with HCM have an increased incidence of both supraventricular and ventricular arrhythmias and are at an increased risk for sudden cardiac death (SCD).

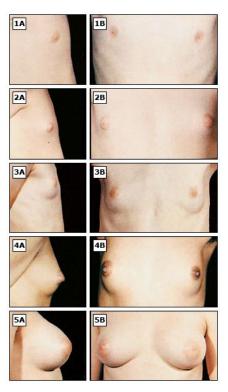
PEDIATRIC CANCERS

- Hodgkin's lymphoma m/c cancer among 15-19yo, Reed Sternberg cells
- ALL 90% survival rate
 - Lymphoblasts → no auer rods, absent granules, smooth membrane, oval nuclei, high nuclei:cytoplasm ratio, deep blue/purple cytoplasm
- AML 65% survival rate
 - Myeloblasts → auer rods, granules, irregular membrane, oval nucleus, low nuclei:cytoplasm ratio, gray/blue cytoplasm

PUBERTY



The median length of time between the onset of puberty (breast Tanner stage 2) and menarche is 2.6 years, and the 95th percentile is 4.5 years.



Stages in breast development in girls.

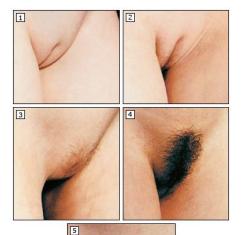
Stage 1: Prepubertal, with no palpable breast tissue.

Stage 2: Development of a breast bud, with elevation of the papilla and enlargement of the areolar diameter.

Stage 3: Enlargement of the breast, without separation of areolar contour from the breast.

Stage 4: The areola and papilla project above the breast, forming a secondary mound.

Stage 5: Recession of the areola to match the contour of the breast; the papilla projects beyond the countour of the areola and breast.



Stages of development in pubic hair in girls.

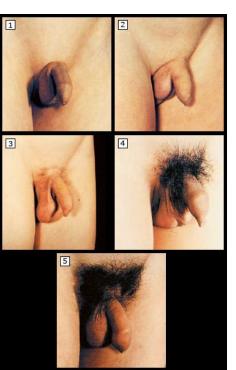
Stage 1: Prepubertal with no pubic hair.

Stage 2: Sparse, straight hair along the lateral vulva.

Stage 3: Hair is darker, coarser, and curlier, extending over the mid-pubis.

Stage 4: Hair is adult-like in appearance, but does not extend to the thighs.

Stage 5: Hair is adult in appearance, extending from thigh to thigh.



Stages of pubic hair development in boys.

Stage 1: Prepubertal, with no pubic hair.

Stage 2: Sparse, straight pubic hair along the base of the penis.

Stage 3: Hair is darker, coarser, and curlier, extending over the mid-pubis.

Stage 4: Hair is adult-like in appearance, but does not extend to thighs.

Stage 5: Hair is adult in appearance, extending from thigh to thigh.

PEDS DERMATOLOGY

ACNE VULGARIS

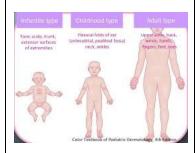


- --etiology: inflammation of pilosebaceous units, increased sebum production, follicular obstruction d/t hyperkeratinization, P. Acnes bacteria
- --causes: inflammatory response, stress, lithium, steroids, progestins, phenytoin
- --Papules, pustules, cysts, comedones (sebaceous follicles plugged with keratin & sebum)
- --Open comedone = blackheads; Closed comedones = whitehead
- --Persistent acne & hirsutism red flag for hyperandrogenism (PCOS, tumors)

Treatment

- --Retinoid: **Tretinoin**, adapalene, tazarotene, isotrentinoin
- --Benzoyl Peroxide, salicylic acid
- **--Spironolactone** (aldosterone antagonist)
- --Topical abx (erythro or clinda) vs. Oral abx (minocycline, **doxy**, erythro)

ECZEMA (DERMATITIS)



ATOPIC DERMATITIS: "the itch that rashes"

- --superficial inflammatory response of epidermis, T-cell mediated immune activation &
 †IgE production
- --altered immune rxn in genetically susceptible populations when exposed to triggers
- --triggers: heat, perspiration, allergens, contact irritants (wool, nickel, foods)
- --allergic triad: eczema, allergic rhinitis, asthma
- --tiny pruritic erythematous edematous ill-defined blisters → dries/crusts over & scales
- --m/c in extensors and face in infants (flexor surfaces i.e. antecubital folds in adults)
- --emollient ointment (glycerol, petroleum jelly) + ceramide moisturizers (Eucern)
- --topical corticosteroids (hydrocortisone, fluticasone, betamethasone) & antihistamines
- --topical immune modulators (calcineurin inhibitors i.e. Tacrolimus)

CONTACT DERMATITIS: Type IV T-cell mediated rxn

- → eczematous (irritant), vesicular (allergic)
- → poison ivy = Tecnu, calamine lotion, oatmeal baths, astringents (witch hazel)
- → topical corticosteroids, oral antihistamines

<u>ASTEATOTIC DERMATITIS:</u> very dry skin, scaling, cracking → emollients, topical steroids, antihistamines

<u>ECZEMA HERPETICUM:</u> fever & clusters of itchy blisters or umbilicated vesicles (**punched out erosions**) → oral antivirals; emergency in kids!

→ disseminated viral infection (HSV), generally occurs at sites of skin damage (eczema) <u>DYSHIDRTOTIC DERMATITIS:</u> pruritic "tapioca pudding" tense vesicles on palms, soles, fingers, webspaces → triggers are sweating, stress, warm weather, metals

→ tx w/ topical steroid ointment, cold compresses, tar soaks

<u>NUMMULAR DERMATITIS:</u> oval weeping patches → steroids, emollients

TINEA

Dermatophyte Fungus = Tinea capitis, tinea faciei, tinea barbae, tinea corporis, tinea cruris, tinea manuum, tinea pedis, tinea unguium

- --Well-demarcated scaling plaque, **KOH 10%** → **hyphae**
- **--Terbinafine (Lamisil)** x1-2wks (allymines), Ketoconazole x2wks, Griseofulvin PO for tinea capitis & unguium (avoid steroids and nystatin)

Non-dermatophyte Fungus = Tinea versicolor (M. furfur yeast)

- --Hypopigmented or erythematous macules with fine scale, no itch, predominantly on trunk
- --KOH → spaghetti & meatball pattern w/ short hyphae & yeast
- --Fluconazole (Diflucan), Ketoconazole, Selenium sulfide

LICHEN PLANUS --idiopathic cell-mediated immune response (papulosquamous) --develop on flexor surfaces of extremities, mucous membranes of skin, mouth, scalp, genitals, nails --Purple popular pruritic polygonal planar --Oral white lacy patches = Wickam striae --Koebner phenomenon, fine scales --seen more in pts with HCV infxn Treatment: typically resolves in 8-12mo --1st line = antihistamines, steroid ointment --2nd line = systemic steroids, UVB therapy Oral lichen planus → increased risk for oral cancer (SCC) PITYRIASIS ROSEA --idiopathic, likely post-viral, increased in spring/fall (paopulasquamous) --Herald patch = initial solitary salmon-colored macule on trunk → general exanthem --salmon-colored oval/round papules with white circular scaling along cleavage lines --very pruritic, christmas tree pattern - confined to trunk & proximal extrem (face spared) --Self-limited, resolves in 6-12wks --Management: none needed, but for pruritus try topical steroids, PO antihistamines. moisturizers, oatmeal baths, +/- UVB light if severe DRUG ERUPTIONS Type I = IgE mediated, immediate (i.e. urticaria, angioedema) (hypersensitivity rxn) **Type II = Ab-mediated**, cytotoxic (drugs in combo with cytotoxic antibodies) **Type III =** immune **ab-antigen complex** (i.e. drug-mediated vasculitis & serum sickness) Type IV = delayed cell-mediated (i.e. EM) Nonimmunologic = cutaneous rxns d/t genetic incapability to detoxify certain drugs (anticonvulsants, sulfonamides) --most are self-limiting if offending drug d/c --may be accompanied by fever, abd or joint pain **URTICARIA** --Type I / IgE-mediated (hypersensitivity rxn) --triggers: antigen from foods, meds, infxn, insect bites, drugs, environment --path: mast cells release histamine → vasodilation of venules → edema of dermis & subq tissue --blanchable, edematous pink papules, wheals, or plaques --Darier's sign = localized urticaria appearing where the skin is rubbed --angioedema = painless, deeper form of urticaria affecting the lips, tongue, evelids. hands, feet & genitals (anaphylaxis may occur) --tx: PO antihistamine*, H2 blockers, corticosteroids, eliminate precipitants **ERYTHEMA MULTIFORME** --acute self-limiting type VI rxn, usually evolve over 3-5d and persist ~2wks --infxn: HSV**, mycoplasma (kids), S. pneumonia (hypersensitivity rxn) --meds: sulfa drugs, B-lactams, phenytoin, phenobarbital --TARGET LESION; dull "dusty violet" red, purpuric macule/vesicle or bullae in center, surrounded by pale edematous rim & peripheral red halo --often febrile --minor → target lesions distributed acrally, no mucousal membrane lesions --major → target lesions involved 1+ mucous membranes (oral, genital, ocular mucosa) --Tx = d/c drug, antihistamines, analgesics, skin care

--- steroid/lidocaine/diphenhydramine mouthwashes for oral lesions

--severe blistering mucocutaneous syn (eyes, mouth, genitals)

--infx: mycoplasma, HIV, HSV, malignancy, idiopathic

--seen esp with sulfa & anticonvulsant drugs

SJS & TEN

(hypersensitivity rxn)

IMPETIGO (bacterial, infectious)	widespread blisters begin on trunk & face → blistering & sloughing off of skin & mucous membranes, epidermal detachmenttx = treat like severe burns SJS if sloughing <10% BSA (self limited, usually resolves in 1mo) TEN if sloughing >30% (toxic epidermoid necrolysis) vesiculopustular skin infxn, highly contagiousRFs = warm, humid conditions, poor personal hygiene Non-bullous* = m/c S. aureus (2nd m/c GABHS), a/w regional lymphadenopathyvesicles, pustules → "honey-colored crusted" erosions w/ moist erythematous base Bullous = S. aureus (not common, usually seen in newborn, young children)vesicles form large bullae → rupture → thick "varnish-like crusts" Bactroban 2% ointment (Mupirocin) x10 days, Bacitracin Extensive dz → Keflex, Clinda, Dicloxacillin, Azithromycin Wash area gently w/ soap & water
MOLLUSCUM CONTAGIOSUM (viral, infectious)	benign viral infxn (poxviridae family), highly contagious (skin to skin & fomites)dome-shaped flesh-colored/pearly-white, waxy papules w/ central umbilicationcurd-like material expressed from center if squeezedusually spontaneously resolved in 3-6mo; +/- imiquimod or podophyllin, cryosurg
LICE / PEDICULOSIS	itching, papular urticaria near lice bites, visualization of live lice, visualization of nits (pearls on hair shafts) w/ eye or wood's lampTx: Permethrin 1% (shampoo & lotion), Ivermectin (Lindane but it is neurotoxic)Examine household & close contactswash bedding & clothing in hot water w/ detergent, dry in hot drier x20mintoys that can't be washed should be placed in air-tight plastic bag x14days
SCABIES (mites)	Sarcoptes scabei mites (cannot survive off human body >4d)female mites burrow into skin and lay eggs, feed, & defecate (scybala fecal particles precipitate HSN in skin)intensely pruritic, increased at nightred papules & linear burrows common in intertriginous zones i.e. webspaces, scalp (usually spare neck * face)symptoms last ~4-6wks, usually clinical dx, but can do skin scrapings for mites or eggsTx: Permethrin (Elimite), wash clothing/bedding with heat & no body contact for ~72hrs permethrin 5% cream - applied to all areas of the body from the neck down and washed off after eight to fourteen hours - 2 applications separated by 1 week Or oral ivermectin - given as a single 200 mcg/kg dose repeated after 1-2wks
VERRUCAE	CUTANEOUS HPV Wart = flesh-colored hyperkeratotic papule w/ red-brown punctations (thrombosed capillaries) Verruca vulgaris = common wart Verruca plantaris = plantar wart Verruca plana = flat wart (flat, pink/tan/flesh colored papules) Condyloma accuminata = genital warts (HPV 6 & 11) → tiny painless papules that evolve into soft, fleshy, cauliflower-like lesions, ranging from skin-color to pink-red, occuring in clusters in the genital regions & oropharynx; lesions may spontaneously resolve, remain unchanged, or grow if not treated

	Dx → mucosal HPV will whiten with acetic acid application
	Tx → most warts resolve spontaneously in 2yrs if immunocompetent ptsvulgaris & plantaris = topic OTC salicylic acid & plasters, cryotherapy, electocautery, CO2, laser, intralesional bleomycinacuminata = chemical, salicylic acid, cryotherapy, laser, podophyllinguardasil vaccine protects against 70% of HPV strains
BURNS	Parkland formula = LR @ 4ml x kg x BSA burn (half over 1st 8hrs, rest over next 16hrs) 18% BSA on head, 14% each extremity (otherwise same as adult rule of nine's)
EXANTHEMS	
ROSEOLA (6TH'S DISEASE)	HHV 6 & 7, spread by respiratory droplets, 10d incubation periodhigh fever 3-5 days → defervescence coincides with rashrose pink maculopapular blanchable rash (trunk/back → face)**only childhood viral exanthem that starts on trunk & spreads to facetx = supportive, anti-inflammatories
HAND-FOOT-MOUTH DZ	coxsackie A virus (enterovirus), spread feco-oral and oral-orallymild fever, URI sx, decreased app starting 3-5 days post-exposure → rashvesicular lesions on a reddened base in oral cavity (buccal mucosa, tongue) → lesions on distal extremities 1-2d post initial sx
MUMPS	paramyxovirus, spread by respiratory droplets, 12-14d incubation periodlow-grade fever, myalgias, headache → painful parotid gland swelling supportive, anti-inflammatories, MMR (sx usually last 7-10d)complications: orchitis , encephalitis, aseptic meningitis, deafness, pancreatitis
RUBEOLA / MEASLES	paramyxovirus, spread by respiratory droplets, 10-12d incubation periodURI prodrome: high fever, cough, coryza, conjunctivitis, koplik spots (precedes rash 24-48hr)maculopapular (mobiliform) brick-red rash on face beginning at hairline → extremities rash lasts 7 days, fevers concurrent w/ rash tx: supportive, anti-inflammatories, isolate for 1wk after rash onsetcomplications: OM* , pneumonia, diarrhea, encephalitis
RUBELLA (German Measles)	Togavirus, spread by respiratory droplets, 2-3 week incubation periodrash lasts 3 dayslow fever, cough, anorexia, LAD → rash (may see photosensitivity & joint pains)pink, light-red spotted maculopapular rash on face → extremities (blueberry muffin)tx = supportive, anti-inflammatories (generally no complications)teratogenic (esp 1st trimester), part of TORCH →congenital syn = sensorineural deafness, cataracts, TTP, mental retardation, heart
ERYTHEMA INFECTIOSUM (5TH'S DISEASE)	parvovirus B19, spread by respiratory droplets, 4-14 day incubation periodcoryza, fever → slapped cheek rash w/ circumoral pallor 2-4d → lacy reticular rash on extremities (spares palms & soles) → resolves in 2-3 wkstx: supportive, anti-inflammatoriescomplications arthropathy (in older kids and adults), aplastic crisis w/ SSD
Rashes that affect palms & soles	Coxsackie (HFM), RMSF (esp wrist), syphilis (secondary), Janeway lesions, Kawasaki, measles, TSS, reactive arthritis, meningiococcemia

DDX FOR NEONATAL JAUNDICE

- Physiologic Jaundice: increased rate of bili production (shorter RBC lifespan--> faster hgb breakdown) + decreased rate of bili elimination (immature liver); onset usually after first 24hrs of life, peaks on day 3-5 (resolves by 7-10d)
- **Breastfeeding jaundice**: dehydrational → insufficient milk intake → hypovolemia → hyperbilirubinemia; *onset is usually seen later in 1st week of life*
- **Breast milk jaundice:** factors in breast milk causing increased intestinal absorption of bili + block the proteins in the liver responsible for breaking down bilirubin; *onset is usually in 2nd week*
- **ABO incompatibility:** different blood type causes Mom's abs to attack fetus blood, causing hemolysis of fetal RBCs and increased bili production; onset within first few days
- **G6PD deficiency:** lack of G6PD enzyme makes RBCs more vulnerable to oxidative stress & lysis; requires trigger (like medication) to manifest
- **Crigler-Najjar & Gilbert syn:** inherited defects in gene for UGT1A1, which catalyzes the conjugation of bili with glucuronic acid → decreases bili conjugation
- **Hypothyroidism:** decreased ability of body to metabolize/conjugate bili (see conjugated hyperbilirubinemia)
- **Biliary atresia or intestinal obstruction:** blockage leads to inability to excrete conjugated bili (see conjugated hyperbilirubinemia)
- Congenital infx: inflammation of liver or deposition of parasites/spirochetes → hepatitis → decreased liver function
- Sepsis → causes hemolysis (one theory suggests that increased oxidative stress due to sepsis damages neonatal red blood cells that are susceptible to cell injury)
- Others: maternal diabetes, galactosemia