Microbiology Lectures 1 and 2- Summary

Disease	Etiology	Presentation (Development +signs)	Dx	Rx	Notes
Surgical Site Infections	*The most common cause is **S.aureus** and **MRSA**, in addition to: **1)** CoNS **2)**Corynebacteria(Diptheriods-G(+) aerobic rods) **3)** Bacillus spp.(G+rods), produce spores **4)** G- aerobic Bacilli (pseudomonas/ enterobactericiae ,usually hospital-acquired)	The signs require from 5 days up to 2 weeks to appear. *local signs: swelling, erythema, pain (inflammation) (fever may not be present until a few days later).	Usually clinical (Clinical signs are enough but we take a sample from body tissue or fluid for gram stain or culturing to identify the causative agent as well as its antibiotic susceptibility profile)	-The primary therapy is 1)open the incision 2)debride the infected material 3)continue dressing changes. -Local or systemic antibiotics can be given but there is little evidence supporting this practice.	1) can be prevented by using meticulously aseptic techniques. 2)External signs are delayed in case of morbidly obese patients or patients with multilayer wounds. 3)Higher risk of infections with mixed flora in GI and female genital tract surgeries. 4) Prosthetic material greatly reduces the number of organisms that are required to initiate infection, like in joint prosthesis or cardiac valve replacement.
Erysipelas	Streptococcus pyogenes				1)It spreads through lymphatics (that's why it shows blisters). 2)The lesions are raised, well-demarcated, erythematous, tender. Accompanied with fever and chills.
Pharyngitis and gingivostomatitis:	The commonest presentation of primary **HSV1 infections.**	Fever, malaise, difficulty in chewing, cervical lymphadenopathy and ulcers or exudative lesions on tongue, posterior pharynx, gums, buccal mucosa.			
Eczema Herpeticum:	caused by viral infection, usually HSV that arises from pre-existing		1)Direct fluorescent antibody testing:	WHENEVER Kaposi varecliform eruptions are	*Definition: one form of Kaposi Varcelliform Eruption; extensive cutaneous vesicular eruptions that arise

	skin disease.		rapid(several hours), accurate, distinguish btw HSV1/HSV2/ Varicella. 2)PCR: for old or atypical lesions or when there is a few number virus particles. 3)pathology lab 4)viral culturing: slow(2 days), accurate and reliable, distinguish btw different viruses. 5)Immunohistochemistry (staining with antibodies). 6)Tczank preparation: Involves taking scrapings>Wright or Giemsa stain> Microscope (multinucleate keratinocytes), requires skilled observer but it shows the results immediately.	suspected Start with Systemic Antiviral Agents. 1)Acyclovir (Oral):preferred as first line treatment in immunocompetent patients(healthy) but requires high doses (5 times per day for 7-10 days). 2)Acyclovir(IV): if there is systemic involvement and in immunocompromised patients. 3)Valacyclovir: given in small doses(twice daily) but it's cost-prohibitive. 4)Foscarnet: given in cases of Acyclovir-Resistant Infections (an antiviral medication that works on HSV but causes nephrotoxicity in 50% of patients). 5)Trifluridine or Vidarabine: topical treatment if there is ophthalmic involvement.	from pre-existing skin disease usually Atopic Dermatitis. 1)Children with AD are at high risk of developing it, in which HSV-1 is most common) 2)can cause different complications such as bacterial superinfection or bacteremia> 3)can disseminate to different organs and may lead to death if untreated.

Recurrent herpes labials:	HSV1 > HSV2	After 2-20 days signs will appear, but shedding occurs within the first 2-4 days. Then after the first episode, the virus will remain dormant in the trigeminal ganglion, and it will be reactivated by stressors such as: sun exposure,stress, fever,trauma, hormonal changes, fatigue,immunosupression.		(Treatment is only to reduce the symptom duration but can't remove the virus completely): **1)First episode:** Acyclovir to reduce the lesion time to 4 days VS 10. ALSO, TO REDUCE the shedding time to 1 day VS 5 days. **2)Recurrence: a- ORAL:** Acyclovir(reduce the eruption healing time by 2 days),Famciclovir, Valacyclovir. **b-TOPICAL:** reduces healing time by less than 1 day.	1)The most frequent manifestation of HSV-2 reactivation. 2)Asymptomatic or present with symptoms that are milder and shorter than the primary infection(cold sores in about 1/4 of college students). 3)The first episode is severe(fever, LAP, mouth or gum ulcers),BUT the second episode is characterized by lesions(itchy, burn, tingling in the first 12-36 hours, then vesicles erupt.... heals within 7-14 days) 4)Immunocompromised patients may suffer from severe mucositis with spread to skin surrounding the mouth. 5)More common btw college students. 6)Transmission: It can be transmitted through mucous membranes(kissing),direct contact with open skin or sharing fomites like towels
Erythema Multiforme:	1)Idiopathic 50% 2)HSV >50% 3)other Pathogens (mycoplasma pneumoniae,HIV, VZV,HCV,CMV) 4)drugs(antibiotics, antiepiliptics,sulfa drugs,penicillin,ciprofloxacin and others).			1)Remove precipitating factor 2)supportive care(pain killers,antihistamines,wound dressing) 3)antiviral therapy for HSV(oral Acyclovir) 4)Must tell the patient if bullae erupt, systemic symptoms recur, must return (SJS/TEN emergency)	-)Def.: an acute,self-limiting, and at times recurring skin condition that is thought to be caused by type 4 hypersensitivity rxn(against certain infections like HSV1 or medications like antibiotics). 1)It forms target lesions(circular lesions with central blisters with symmetrically peripheral distribution usually on limbs). 2)Frequently seen in adults between the ages of 20-40 years, with rash occurring 5-10 days after the onset of

					viral illness, happen over 3-5 days and persist for 1-2 weeks (urticarial is the major differential here which resolves in hours). Minimal mucous membrane association and <10% epidermal detachment.
#SJS --- TEN:(acute to life threatning):		It starts with prodromal symptoms that progress to skin ulcerations that begin in the trunk and face (involving the mouth,eyes, with genital sores).	Clinical, biopsy confirms.	supportive-ICU admission(burn unit)some immune based therapy are used.	1)Mortality rate: SJS: 3% / TEN: 30%. (up to 50%) 2)toxic looking patient with positive nikolsky sign (bullous formation).
Genital Herpes:	90% by HSV2 and 10% by HSV1. (although HSV-1 is on the rise)	1)primary infection is usually associated with fever, malaise, and adenopathy. 2)The HSV DNA migrates up the infected axon and remains dormant(LIFE LONG) in the spinal cord sensory Ganglion. 3)when there is reactivation the DNA migrates down the Axon and erupts again. *First infection is the worst, then the subsequent Outbreaks are less severe. *Vesicular eruptions are	Clinical picture is enough and we can do PCR.	Antiviral agents for primary infections and recurrence which will not cure but only reduce the duration of illness. *IN PREGNANCY: the virus can be transmitted vertically causing neonatal HSV infections(encephalitis or disseminated HSV infections in newborns, or congenital HSV infections which can cause microcephalus, hydrocephalus,chorioritinitis)--- in this case we give Vaccine(experimental) +antivirals, abstinence.	1)MCC of genital ulcers (forms 60%-70% of STD ulcers). 2)Sheds asymptomatically(so without outbreaks the virus can shed 10%-20% of days). 3)Asymptomatic in most of patients(2/3); which increases the spread. 4)High prevalence in Western countries(roughly 12%) or 10%-30% of sexually active people(300K yearly in USA). 5)HSV-1 genital herpes is milder and causes fewer outbreaks than HSV-2.

		similar to oral herpes preceded by itch, tingling and burning. *Primary lesions remain up to 2 weeks but in reactivation 6-12 days.		C/S in patients with active lesions. Loose clothing, ice pack or baking soda compression. Topical antivirals and low dose anesthetic can be given.	
Herpetic Whitlow	HSV infections of the finger either by auto-inoculation(from oral or genital infection) or by direct external inoculation from the environment.	1)presents with vesicles ±regional lymphadenopathy.	Tzanck smear	1)prevent transmission (bandage) 2)Antivirals in case of recurrent infections or immunocompromised patients.	**1)Herpes gladiatorum:** mucocutanous infections in surfaces like ears,face,chest seen in rugby players or wrestlers (long intimate contact).
Paronychia:	Due to penetrating trauma (manicure, finger biting) infected by bacteria from the skin (staph. aureus)or from the mouth(streptococcus pyogenes, bacteroides).	Infection appears 2-5 days after trauma,characterised by pain and local inflammation signs(red,hot,tender). May progress to form abscess or nail bed infections.		Soak in warm water or acetic acid for 15min. I and D (if abscess) -For mild cases: Topical antibiotics, topical bacitracin ,gentamicin,fluroquionolone. -For prolonged infections: Systemic antibiotics (suspect cellulitis or ingrown nail)must do I&D then as a first line treatment we use cephalexin or dicloxacillin. Second line therapy: TMP-SFX or doxycycline ,especially when suspecting MRSA.	-Differentiating features from herpetic whitlow: pain and local signs of inflammation (redness, swelling, tenderness..etc) -MRSA colonizes the nose, so biting can bring it easily to the nail.

Chickenpox:	Varicella-zoster virus (human herpes 3)	The virus requires an incubation period of 10-14 days, after this period, a 1-to 2-day febrile prodrome follows before the onset of constitutional signs(malaise,anorexia,itch) and then the papulovesicular rash(<5mm) which is Itchy (pruritic red papules and vesicles). Appears in successive crops (2-4 days). Patient will have different stages of vesicles all over		Usually no antivirals (some guidelines depict oral antivirals in households that are large and in immunocompromised patients) but we can give BACITRACIN to treat the bacterial superinfection (Impetigo). *just reduce itch by giving(calamine lotion, oatmeal bath) and antihistamines at bed time.	*Complications: 1)Bacterial superinfection, encephalitis, pneumonitis). 2)The involvement of mucosa of oropharynx and Vagina (RARE condition) 3)Disease may be severe In pregnancy(it can cause congenital varicella infections or pneumonia for the mother)or in immunocompromised patients. *90% in cases are for children under 13 years old (peak btw 5-9). *Peak outbreak time (January to May-cold season-fall). *Transmission: respiratory droplets, transplacental (vertical transmission),through direct contact(in 90% of cases in household (pox parties)).
Shingles:	Herpes zoster, localized recurrence of varicella virus that causes a unilateral Vesicular eruption in a dermatomal distribution-mostly thoracic and lumbar dermatomes).	1)Most commonly preceded by rash,fever,headache,numbling along the sensory nerve, pain. 2)The rash starts erythmatous, maculopapular, later clear distinct vesicle erupts. 3)Vesicles turn cloudy in 3-5 days and crust by 10 days, may leave residual scar. *Resolution may take 2-4 weeks.	*The best and MOST SENSITIVE is PCR. rashes develop 2-3 days of first symptoms and lasts for 2-4 weeks (followed by a dermatomal distribution: proximal to distal and most commonly on the back T1-T2, also on the face).	1)Antivirals (within 3 days of onset is the best). 2)Pain management NSAID-opiods-refractory pain amitriptline or gabapentine. 3) Steroids. 4)Vitamine B to promote healing.	*Variants of shingles: **1)Zoster Sine Herpete**(Zoster without a rash):uncommon, the pain, prodrome and fever are all present with no or little rash seen.(rare but on the rise). **2)Ramsay hunt syndrome** (VZV of facial nerve):rapid onset with facial pain, Tinnitus and vertigo if cranial nerve 8 is involved. -seen as unilateral herpetic rash of ear pinna, hearing loss may occur. -peripheral facial paralysis. - Management (similar to bell's palsy = facial nerve paralysis due to VZV Reactivation): antivirals+corticosteroids+

		*LAP with tenderness is also a common finding.			pain killers.
Herpes Ophthalmicus		Reactivation of the virus that is dormant in Trigeminal nerve ganglion. -)Reactivation occurs more in 1)Immunocompromised patients (HIV,Cancer,Chemo or Radio therapy). 2)Advanced age Patients. 3)systemic illness or stress(like typical shingles).		Consult the ophthalmologist • Antivirals (acyclovir 800 mg PO five times a day for 7-10 days), also can use Valacyclovir (1000mg 3 times a day for 1-2 weeks), famciclovir (500 mg 3 times a day for 7 days). • Antistaph antibiotics • Corticosteroids (only under the ophthalmology consultation- has a risk of corneal perforation)	*Can cause eye complications: keratitis, iritis, episcleritis, VISUAL LOSS. * **Huntchinson's sign** is typical (has a two fold risk of ocular involvement)
Kaposi Sarcoma	AKA human Herpes virus 8 (HHV-8)	*Clinically: purplish, reddish blue or dark brown/black macules, plaques, and nodules. *Nodular lesions may ulcerate and bleed.			• Form of Cancer, due to HHV8 • NOT IN A DERMATOMAL distribution • If seen must suspect immunocompromised state, (e.g HIV..) • Management is aimed at the cause of the immunocompromised state
Ecthyma Gangrenosum (Pseudomonal septiciema)		*In this case there is no pre-existing ulcer that will be infected with pseudomonas, but the pseudomonas		Appropriate antibiotics (antipseudomonal - penicillins) as well as surgical debridement of necrotic tissue.	Usually seen in immunocompromised, burn, critical patients (ICU, NICU) Risk factors include: Severe burns, malnutririon, uncontrolled diabetes, immunocompromised state (AIDS..etc)

		will cause ulcers.			
Meningococcal Rash	Is due to DIC, as well as bacterial invasion of the plexus	The Dark color (Purpura):a rash of purple spots on the skin caused by internal bleeding from small blood vessels			
Acute Foodborne Illness	Salmonella typhi or paratyphi	During active infection, patients suffer from fever, usually intermittent and sustained high fever • With the associated symptoms of headache, anorexia, vomiting and abdominal pain • Followed by change in stool consistency in less than half the patients → diarrhea in children, or constipation in adults			*__Rose spots__ can be seen in up to 30% of patients, seen as blanching macules.

- **Extra Notes (from lecture 1):**

  ✓ Infections routes:

  Mechanical disruption of stratum corneum by (burns,bites..etc).

  -Burns (usually associated with Pseudomonas). (**Degree of burn:** 1st degree: epidermis/2nd degree: dermis/3rd degree: bottom layer to dermis and possibly hypodermis)

  -Hair Follicles

- ✓ Surgical Site infections:

- Superficial incisional SSI: involves subcutaneous tissue (occurs within 30 days of operation)
- Deep incisional SSI: muscle and fascia (within 30 days, up to 1 year if prosthesis inserted)
- Organ/space SSI: any part other than incisional site

- ✓ Viruses infect squamous epithelium, forming vesicles. Routes for this event: Direct cutaneous inoculation/ Seeding indirectly from other structures (from dermal capillary plexus as in varicella/ from cutaneous nerve routes as in herpes zoster)
- ✓ Infective vasculitis of the hand with necrosis-> indicative of staph. endocarditis.
- ✓ Osler's nodes and Janeway lesions are important clues for the existence of endocarditis.
- ✓ Admissions for skin and soft tissue infections are on the rise (why? More procedures/emergence of USA300 clone of MRSA)
- ✓ Refer to slides to check the differences between different types of skin lesions (macules vs. papules vs. vesicles ...etc.)

Good luck

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