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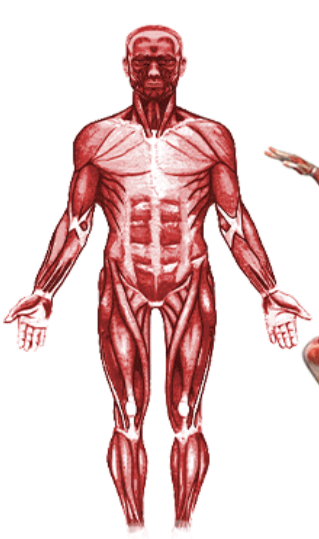

MSS

Musculoskeletal System

Microbiology

Doctor 2018 | Medicine | JU

Done by

Dana Alnasra

Contributed In The Scientific Correction

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Contributed In The Grammatical Correction

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Doctor

Mohammad Madadha

Hello there future doctor! this is our first microbiology sheet this semester, so show me that big bright smile and let the journey begin:

It's a brief introduction to the musculoskeletal infections.

➢ Parts of the musculoskeletal system that are prone to infection:

1. skin, it provides protection by acting as a barrier to external environment
2. fascia
3. muscles
4. bones

How do these structures get infected?

a. Infections from external environment

These mainly infect the skin because it's the uppermost layer.

b. Infections from internal environment

A pathogen in your system. Mainly infect the deeper organs; bones and muscles.

But that doesn't mean that external pathogens cannot infect deeper organs.
For example, joints, ears, jaw and the temple are areas where skin forms a thin layer, which means it's very close to the bone and it might get infected.

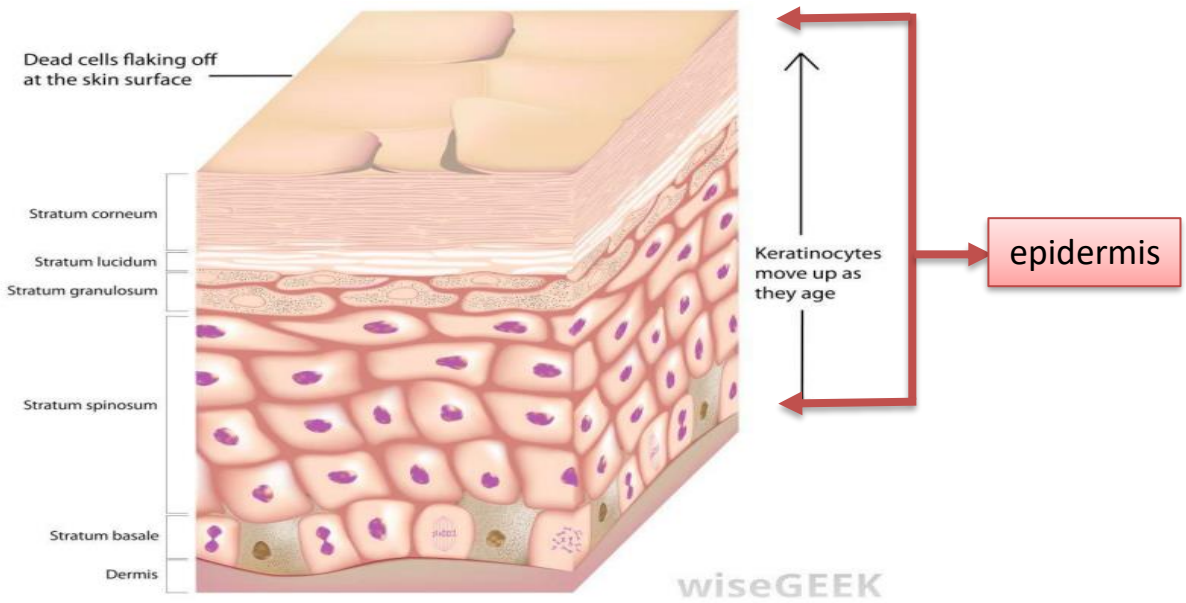
**It's important to know the anatomy of the infection site because it might give us hints about the diagnosis; type of pathogen causing the infection and its pathology.

Between 2000 and 2004 hospital admissions for these illnesses rose by 27%. Why?

- Emergence of antibiotic resistant bacteria like MRSA (methicillin resistant staph. Aureus) and other species.
- Increase in the number of procedures and operations done in hospitals.
- Pollution may also contribute to infections.

➢ Infection routes:

The mechanical barrier provided by the stratum corneum provides protection against infection, note that the epidermis itself is devoid of blood vessels.



Mechanical disruption of the stratum corneum layer which is the first line of defense by:

1. Burns, which are a concern because of:
   a. Loss of a large surface area of skin, thus losing the protective barrier.
   b. Dehydration; Loss of blood, fluid, along with proteins and antibodies which are needed for the function of the immune system against the infection.
2. Procedures
3. abrasion
4. Bites, lead to infection through introduction of pathogens in the saliva.
5. Foreign bodies, e.g. catheters, tubes, central lines, etc..
   a. They reduce the bioburden* needed for infection; because they're sites for biofilm formation. This is especially seen in joint prosthesis and cardiac valve replacement (both Surgical site infection and prosthesis infection).
   b. They protect the bacteria from the immune system.
6. primary dermatologic disorders (such as herpes simplex, varicella, ecthyma gangrenosum), surgery, or vascular or pressure ulcer.

→ all of which allows penetration of bacteria to the deeper (and susceptible) structures.
Each of those mechanisms predisposes to different pathogens.

***bioburden**: the number of bacteria a person can tolerate before getting infected.

a) Burns: Most commonly associated with pseudomonas infections.

****Remember:** pseudomonas species are obligate aerobes and they love water, so they're more likely to infect burns because when a person gets burned, and they lose fluids, these

fluids contain serum, or they may apply water to the wound. Also, they may go to the hospital which is a place full of those bacteria.

Degree of burns:

Degree of burns determines how much of this mechanical barrier has been compromised.

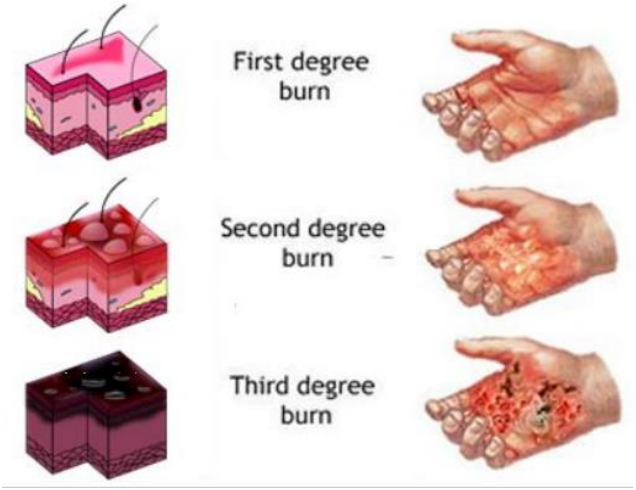
1st degree: upper layer, epidermis.

2nd degree: dermis.

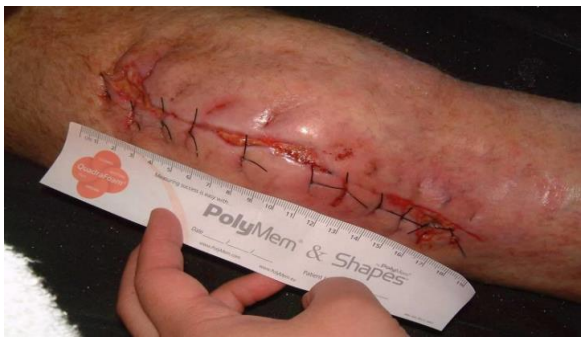
3rd degree: reaches bottom layer to the dermis and possibly to the fascia and msucles.

**Degree of burn + surface area determine bioburden; Pathogens usually infect burns that are deep and cover large areas of skin rather than small superficial burns.

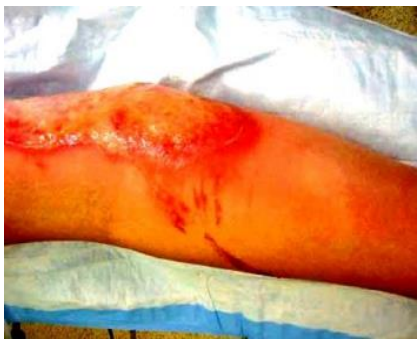
When burns cause a loss of a large area of skin, skin grafts are needed.



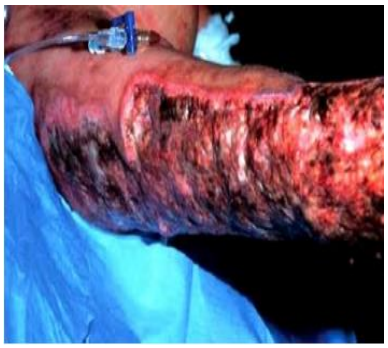
Cellulitis, and **surgical wound infections** are usually a cause of normal flora. **The hair follicle** can serve as a portal either for components of the normal flora (e.g. Staphylococcus) or for extrinsic bacteria (e.g. Pseudomonas in hot-tub folliculitis).



Surgical wounds (and common cuts) usually introduce skin flora and thus the common pathogens seen are Staphylococci and Streptococci, and pseudomonas (in hospital settings).



Cellulitis following a burn wound



Pseudomonas infection following a burn wound

b) Surgical site infections (SSI):

Infection is the biggest limit to surgeries, and it's always a complication/side effect; this increases the health burden and prolongs hospital stay, which is not good.

However, it depends on the type (category) of surgery.
For example, the cerebrospinal fluid (CSF), urine, and blood are sterile fluids; they don't contain bacteria, so they're much safer than GI and female genital surgeries (e.g. C-section), where the surgeon has to be very careful because these sites are highly loaded/contaminated with normal flora and bacteria, mixing of skin flora G+ve with anaerobes and aerobes of the GI tract is a high risk. Also, superficial surgeries have a lesser chance of infection than deep surgeries.

There are three categories of SSI: not mentioned by the doctor

I. Superficial incisional SSI—involves subcutaneous tissue, occurs within 30 days of operation.
II. Deep incisional SSI—involves muscle and fascia, occurs within 30 days of operation (up to 1 year if prosthesis inserted).
III. Organ/space SSI—involves any part of the anatomy (organs or spaces) other than the incisional site.

Etiology and pathogenesis:

The commonest organisms are **S. aureus and MRSA** (skin flora/hospital acquired). **Others** include:

1. CoNS (coagulase negative staphs)
2. Aerobic Gram-negative bacilli (pseudomonas, Enterobacteriaceae), remember these are usually hospital acquired organisms and it means the surgery has had a breach and contaminated the wound.
3. Bacillus spp., G+ve rods, implicated in food poisoning, but also in soft tissue infections especially seen in road traffic accidents, because bacillus species are anaerobic, spore forming bacteria. And these spores are found in the soil and dirt on the roads.
4. Corynebacteria (Diphteroids; G+ve aerobic rods).

Most SSIs have no clinical manifestations for at least 5 days after the operation, and many may not become apparent for up to 2 weeks, to give the bacteria a chance to divide and grow.

Bacteremia: the presence of bacteria in blood

Toxicemia: the presence of toxins in blood

Septicemia: the presence of both the bacteria and toxins.

all these can cause systemic infection which will lead to systemic manifestations like fever.

Local signs of infection then become apparent:

a. inflammation signs; pain, swelling, hotness, and redness (erythema).

b. purulent drainage, pus formation due to infection and drainage of pus.
c. Fever may not be present until a few days later.

External signs of SSI become even more delayed in the case of morbidly obese patients or in patients with deep, multilayer wounds, such as thoracotomy.

since the surgeons are one of the main causatives of surgery site infections, how can we solve this problem?

Try to make sure your procedure is meticulously aseptic, to reduce SSI and thus reduce patients returning for prolonged wound dressings and complaints.

** the surgeon's technique is more important and more effective than using antibiotics. Actually, studies showed that using prophylactic antibiotics doesn't cause that much of a difference.

Diagnosis and treatment:

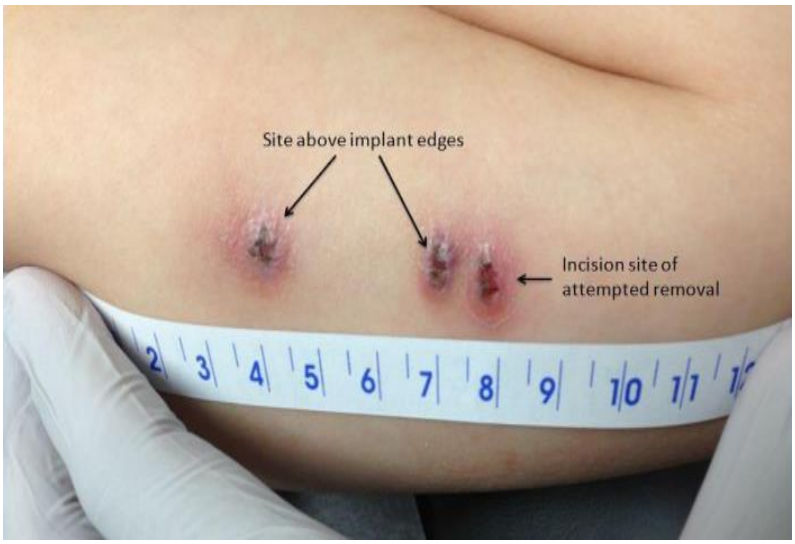
The diagnosis is usually clinical (signs and symptoms mentioned are enough), but samples of fluid or tissue should be sent to the laboratory for Gram stain and culture to confirm and identify the causative organism (as well as an antibiotics susceptibility profile).

The primary therapy for SSIs is to open the incision, debride the infected material, and continue dressing changes until the wound heals by secondary intention (the aim is to reduce bioburden, and promote healing as well as disrupt oxygen requirements).

Patients are prescribed systemic/local antibiotics for SSIs, however, there is little or no evidence supporting this practice. The common practice, endorsed by expert opinion, is to open all infected wounds (aerate the wound). If there is minimal evidence of invasive infection a short course of antibiotics (24–48h) may be indicated.

c. **Foreign bodies:** These can introduce skin flora and cause infection with Gram positives, but also can bring in spores from soil (C. tetani) Or even introduce viruses with needles (HIV, HBV..etc)

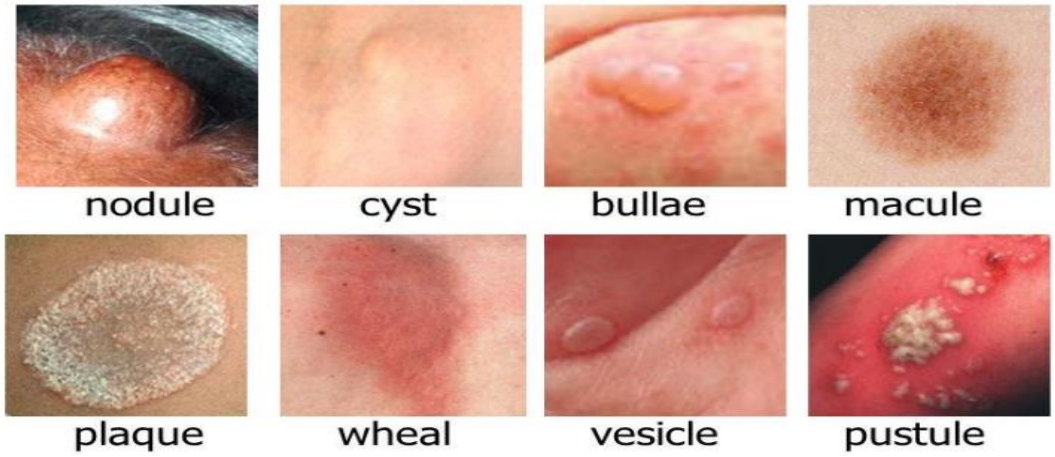
e.g. intradermal drugs, implants, tatoos, needles.

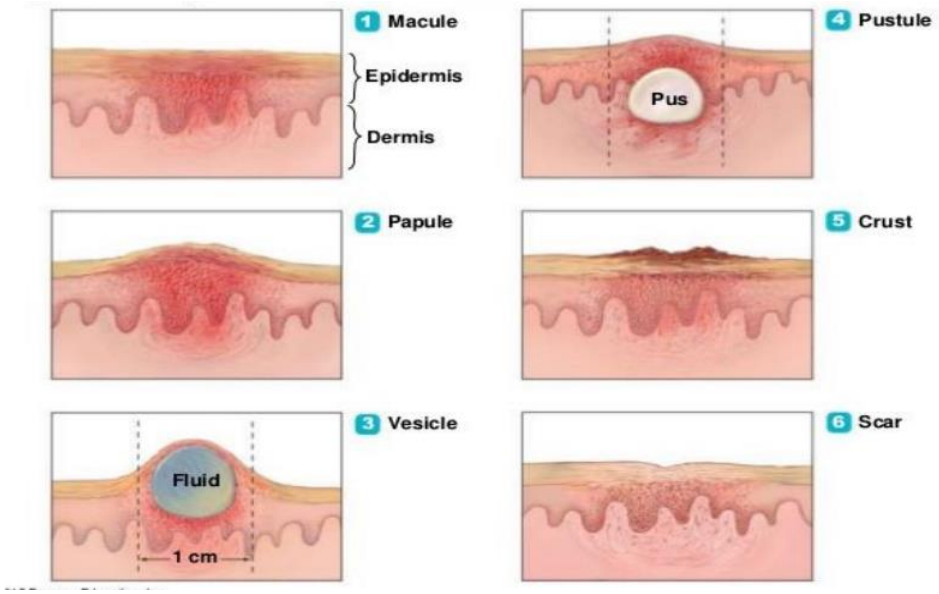


➢ Infective skin lesions:

- **Macules:** are lesions that have change in color, but not elevated or depressed from the rest of the skin surface, usually more than one. They measure less than 10mm in diameter.
- **Papules:** are elevated lesions which are less than 10mm in diameter.
- **Vesicles:** are small fluid filled lesions, typically associated with viral infections.
- **Bullae:** are large fluid filled lesions. >10 mm in diameter.
- **Crusted lesions:** thin-walled bullae that do not remain closed for long, fluid released crusts over/or lesion that crust during the course of infection.
- **Ulcers:** are a loss/depression of the layers of the skin (or mucous membranes) which fails to heal.
- **Petechiae:** a small red or purple disseminated spots caused by bleeding into the skin.
- **Purpura:** a rash of purple spots on the skin caused by internal bleeding from small blood vessels (can think of it as a collection of petechiae).
- **Eschar:** a dry, dark scab or falling away of dead skin, typically caused by a burn, an insect bite, or infection with anthrax.

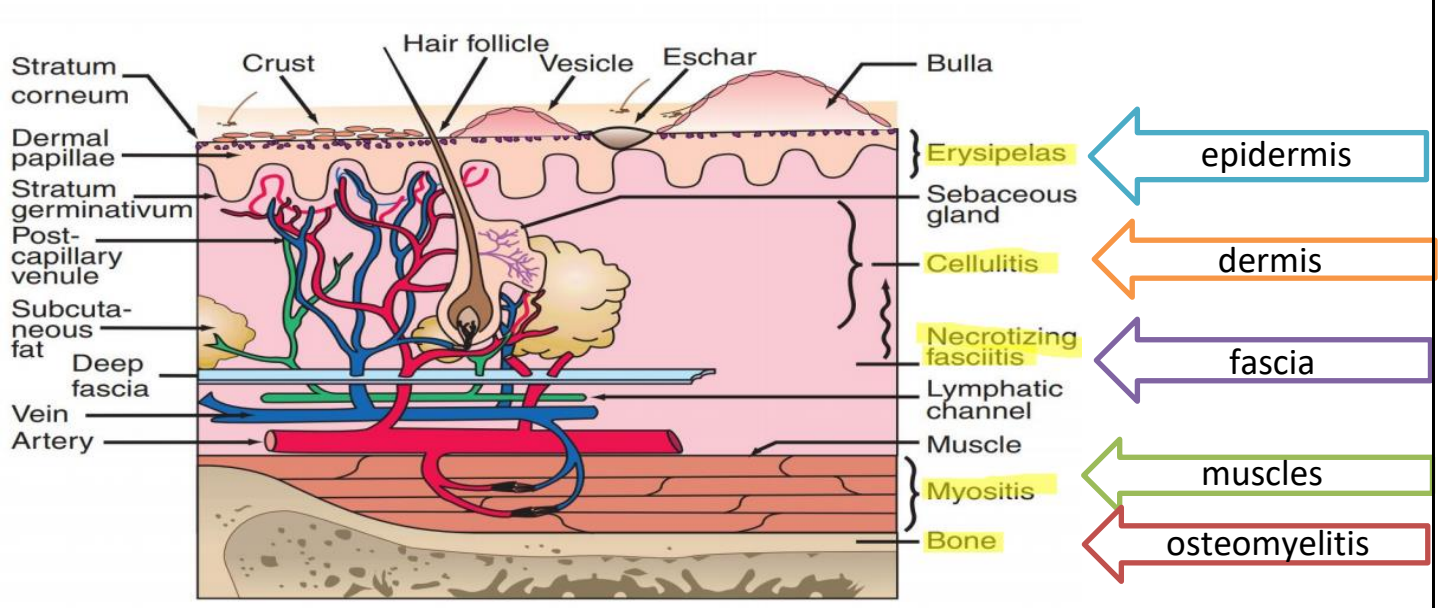
Pustule: a vesicle containing debris and pus.





**Infective skin lesions do not undergo all these types (phases), rather they can only progress to the following phase. e.g. a macule stays a macule and it may progress to a papule (maculo-papular rash).

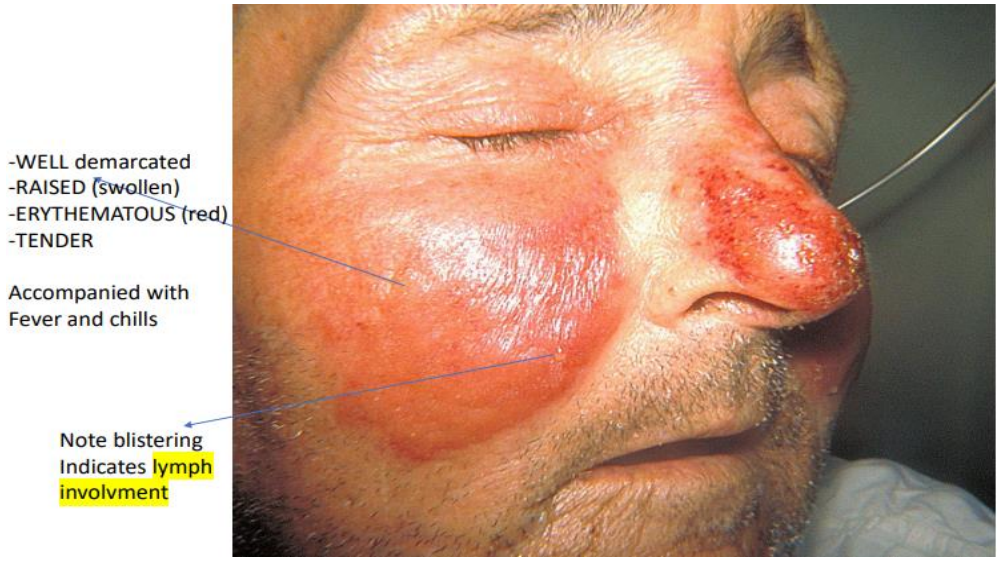
*This is a nice picture showing the different infections in different layers:



➢ Spread of pathogens through the skin and soft tissues

Bacteria also spread, infecting the epidermis by direct inoculation. However, some important bacteria such as Streptococcus pyogenes (Group A strep.) may be translocated laterally to deeper structures via **lymphatics**, this helps the rapid superficial spread of **erysipelas**.

The spread through the lymphatics later causes engorgement or obstruction of these lymphatic channels which then causes flaccid edema of the epidermis (a characteristic of erysipelas).

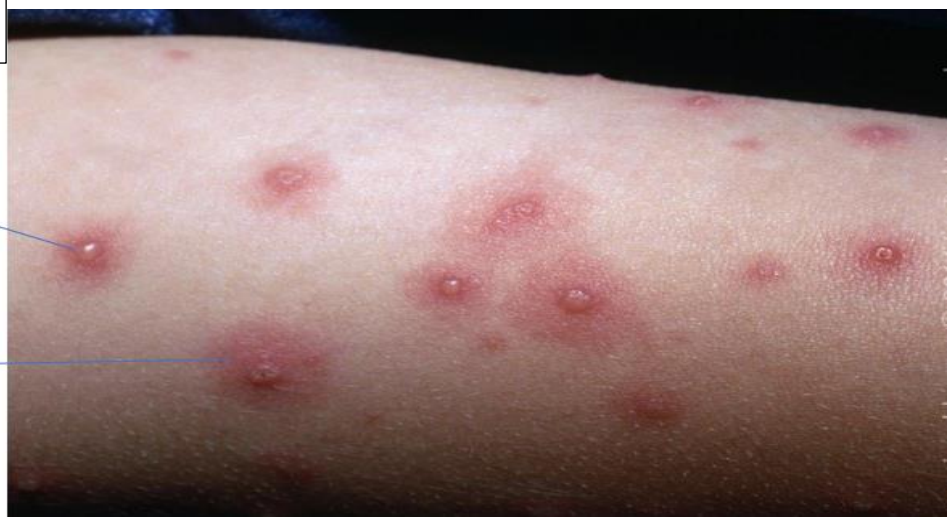


Viruses tend to cause more systemic infections, and they like to form papules and vesicles. The routes for such an event can either be due to:

a) Direct cutaneous inoculation (such as herpes simplex virus type 1).

b) Seeding indirectly from other structures (from the dermal capillary plexus, as in varicella) due to infections with other viruses associated with viremia (rashes) – from cutaneous nerve roots (herpes zoster).

Chicken pox

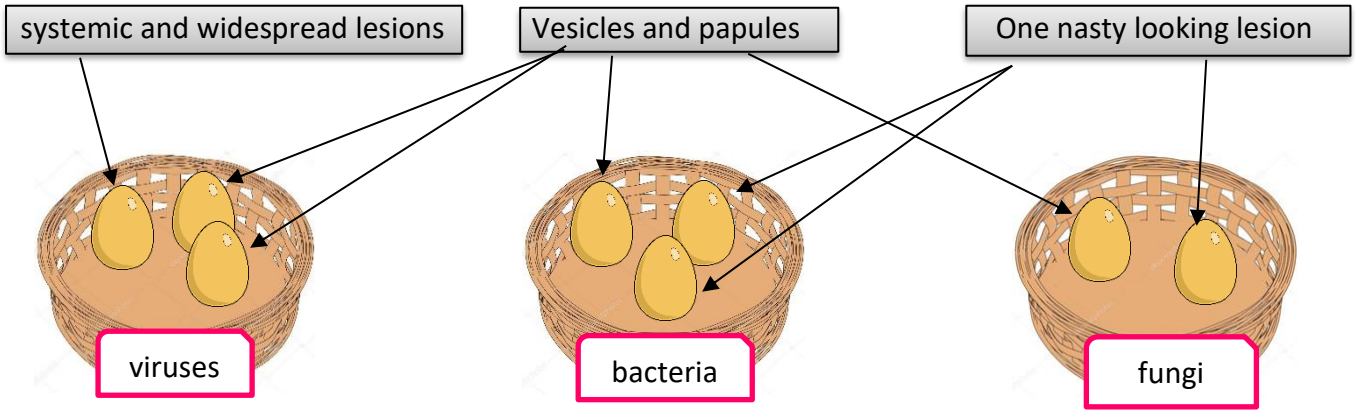


	Viruses	Bacteria	Other
Maculo/papular rash	Measles, rubella, HHV-6 EBV, HBV, HIV, enterovirus	GABHS (scarlet fever)… Salmonella, Lyme, Mycoplasma pneumoniae	Rickettsia
Vesicular, bullous	VZV, HSV, Echovirus Coxsackievirus A, B	Impetigo	
Petechial	CMV, enterovirus, EBV Hemorrhagic fever, VZV	Sepsis (N.men, S.pneu,Hib) Rat bite fever (S. minus)	Rickettsia
Diffuse erythroderma	Dengue	scarlet fever, TSS	C. albicans
Urticarial rash	EBV, HBV, HIV, Enterovirus	M. pneumoniae	

This table isn't required in this lecture, just in case you saw it in the slides.

❖ Let's talk clinical stuff:

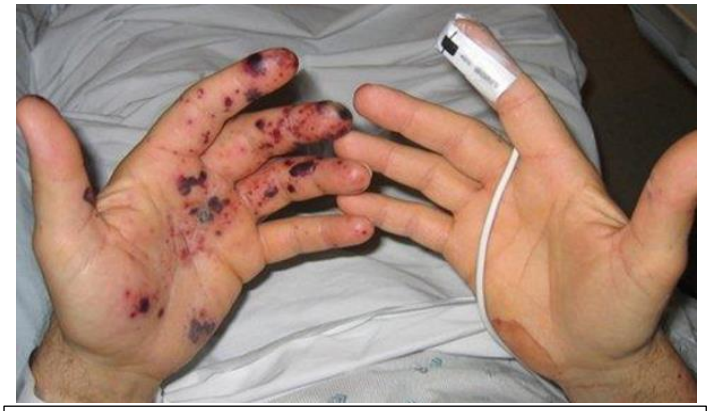
when diagnosing a case there are certain clues that will lead you to the correct diagnosis, we will treat these clues as eggs which we will put inside 3 baskets: one basket for viruses, one for bacteria, and one for fungi



Infections outside of the skin can show abnormalities on the skin; A pathogen that doesn't directly infect the skin but results in manifestations on the skin due to seeding from other organs, or the reaction of the immune system, leading to the lesions and rashes. Just like we saw in chicken pox previously.

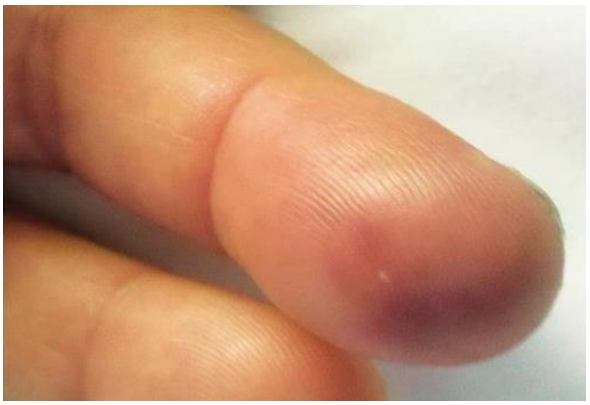
** In the body, when nutrition is compromised due to an illness or infection, many signs are seen on the skin or nail as they are the most superficial structures that can point to these abnormalities. What happens is that the rich plexus of capillaries beneath dermal papillae provides nutrition to the stratum germinativum –which is the germinal layer that maintains the epidermis. So, Physiologic responses of this plexus to stimuli elsewhere produce important clinical signs and symptoms. One example of these responses is seen in endocarditis (infective or non-infective) such as infective vasculitis seen as petechiae of the plexus results, Osler's nodes, Janeway lesions, splinter hemorrhages and palpable purpura, which are skin abnormalities, if present, are important clues to the existence of endocarditis.

**In short: endocarditis; the infection is in the heart but there may be 4 manifestations on the skin. Why? Because the skin is supplied by lymph drainage, veins, arteries, and nerves. Caused by staph and strep.



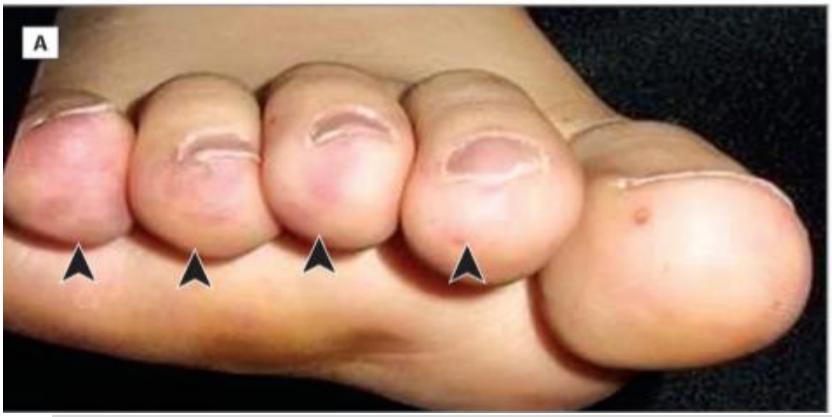
Infective vasculitis of the LEFT hand Seen with necrosis

This is indicative of staphycoccal endocarditis



Osler node- usually painful-, seen in a patient who did dental work 3 months prior. Complicated with an abscess and later on infective endocarditis

Janeway lesion

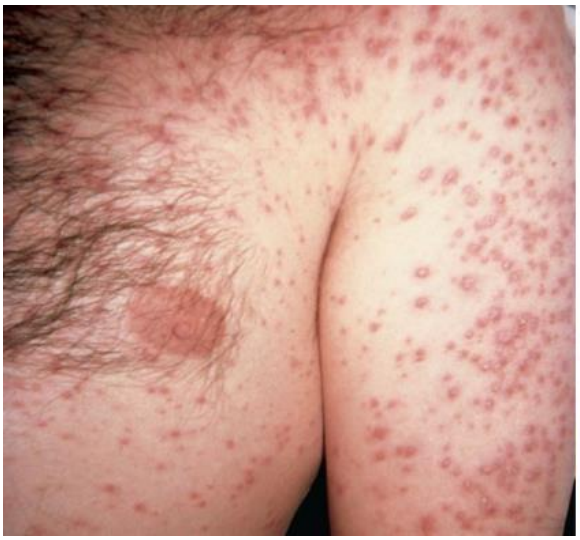


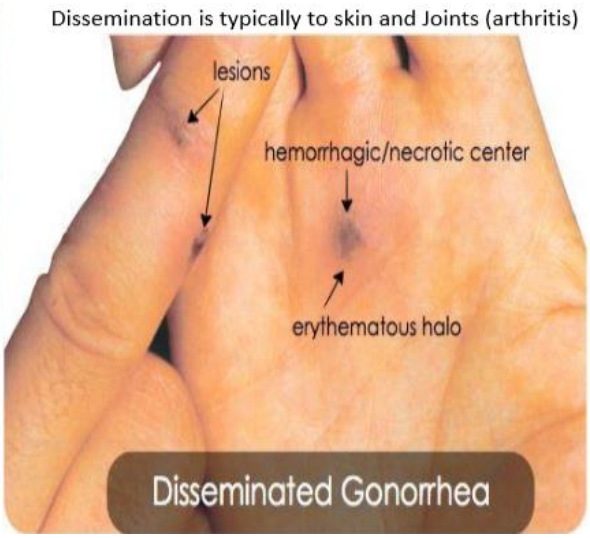
Splinter hemorrhage

Metastatic infection within this plexus can result in cutaneous manifestations such as those seen in: disseminated fungal infection, gonococcal infection, meningococcemia, Salmonella infection, staphylococcal infection.

The plexus also provides bacteria with access to the circulation, thereby facilitating local spread or bacteremia. The postcapillary venules of this plexus are a major site of polymorphonuclear leukocyte sequestration, diapedesis, and chemotaxis to the site of cutaneous infection.

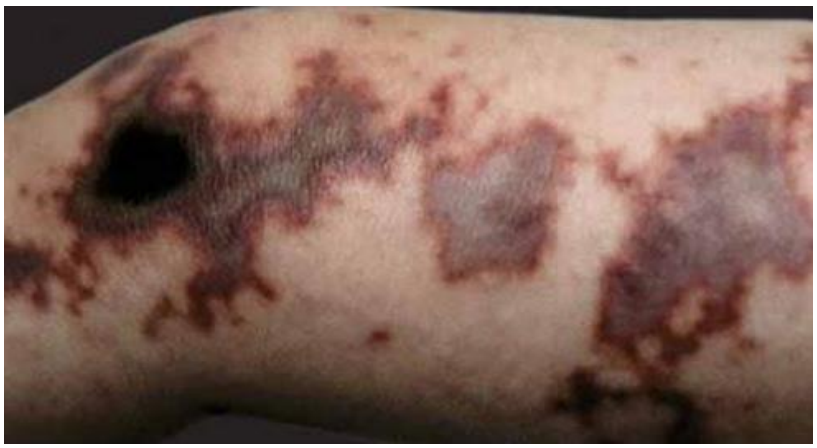
Gonorrhea → painful disseminated lesions on skin resulting from infection in the genital tract.

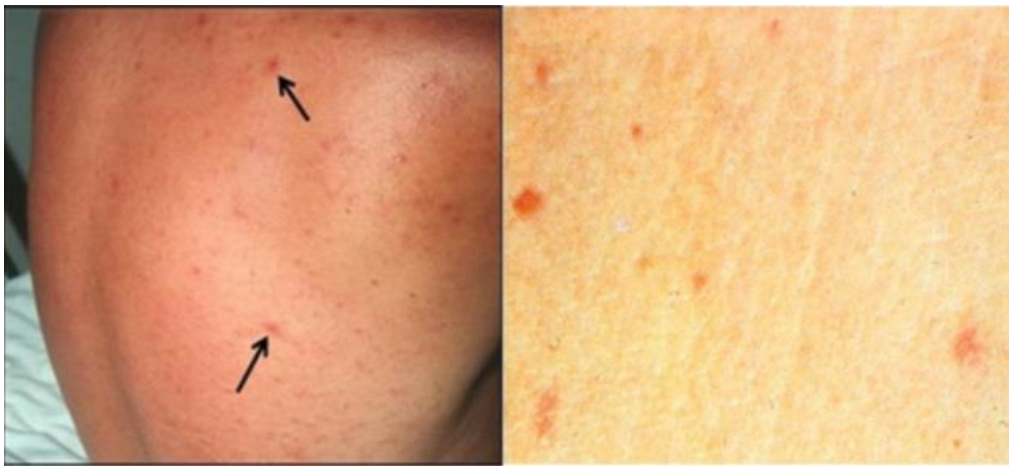




meningococcal rash

The dark color (purpura): a rash of purple spots on the skin caused by internal bleeding from small blood vessels, due to disseminated intravascular coagulation, as well as bacterial invasion of the plexus.

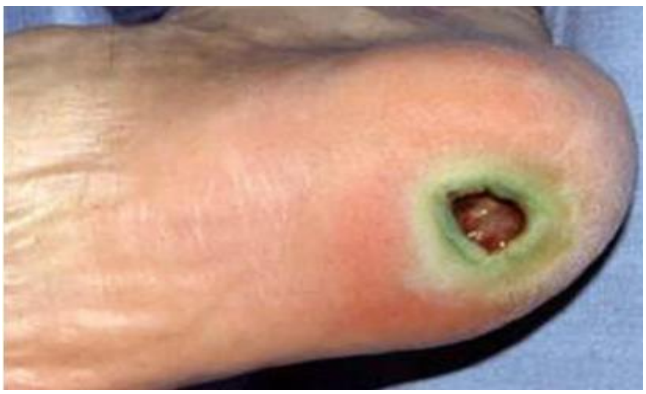


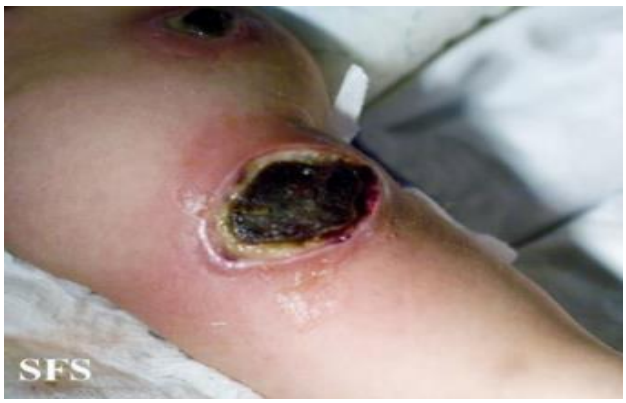


Although not diagnostic, Rose spots seen in patients with enteric fever due to Salmonella typhi or paratyphi.

Patient with enteric fever (Salmonella typhi or paratyphi) suffer from acute foodborne illness. 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. In this stage, rose spots can be seen in up to 30% of patients, seen as blanching macule.

Ecthyma gangenosum (psuedomonal speticiema): Usually seen in immunocompromises, burn, critical patients (ICU, NICU)





Pseudomonal septicemia → ulcers

He didn't have ulcers that got infected with pseudomonas, but he already had pseudomonas that caused the ulcers.

Patients with reduced or weak immunity who get exposed to pseudomonas in hospital setting are at risk. Some of the risk factors are:

- Severe and extensive burns
- Malnutrition
- Certain pre-existing conditions such as uncontrolled diabetes
- Immune compromised state: such as AIDS, organ transplantation (reduced cell immunity), chemotherapy or radiation therapy.

Blisters soon become necrotic and turn into the ulcers seen in the pictures above.

Treatment with appropriate antibiotics (antipseudomonal- penicillins) as well as surgical debridement of necrotic tissue.

A clinical case to have some fun:

A male child presented with high fever.

The first thing that would usually come to your mind is flu (upper respiratory tract infection) so the first egg would go to upper respiratory tract infection basket.

He was coughing → another egg to the upper respiratory tract infection

During Physical examination, when directing a light to the patient's eyes he squinted and looked away immediately → photophobia. This symptom completely changes the situation and the diagnosis you had in mind. So, you will have to add a whole bunch of eggs to the meningitis basket.

If no meningeal signs, you may try to look for the meningococcal rash, which you did find.

So we now have: fever, Photophobia, and meningococcal rash. And we can be sure that this case is a meningitis case.