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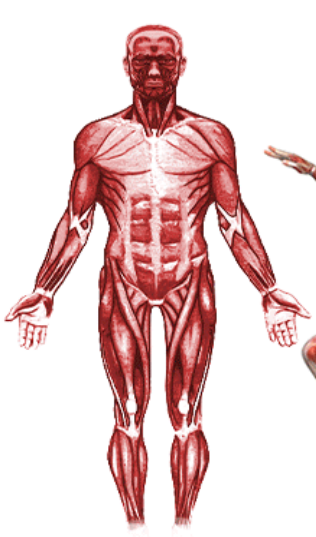

MSS

Musculoskeletal System

Microbiology

Doctor 2018 | Medicine | JU

Done by

Batool Bdour

Contributed In The Scientific Correction

Sarah Qudah

Contributed In The Grammatical Correction

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Doctor

Dr.Madadha

Hi there again, I hope you're all doing great with your system studying, it is exciting that we're a little closer to being doctors, isn't it?

In this sheet we'll continue talking about vesicular rashes, crusted lesions and start talking about bullous formation.

Vesicular rashes (remember, mostly are viral)

Hand foot and mouth disease

General/common case profile

An outbreak in a KG center (or elementary school), children with flu (URTI) like symptoms, constitutional symptoms of fever malaise, and no rash (if in early stages).

Let's break down the case:

It occurs in outbreaks because it requires presence of optimal conditions to take place like winter and presence of many not very hygienic people in small space.

- Children are probably never exposed to the virus before, the illness easily spreads between them. It's caused by an enteric virus, so it's transmitted feco-orally, very typical to occur with children (<10 even more so <5 agers) who aren't yet strong on hygienic acts (e.g their hand washing is BAD).

Causative agents:

- *Coxsackie virus A16 (an enterovirus: transmitted by direct infection)*
- *Enterovirus 71*

Why are we giving it attention?

- Enterovirus 71 has been associated with ENCEPHALITIS and MYOCARDITIS.

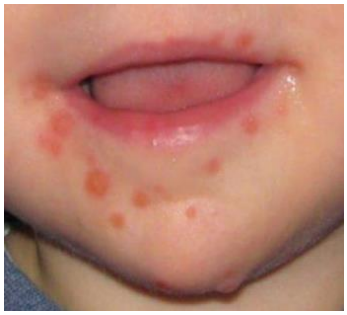
So, once we see it we have to inform the parents to watch out for any advanced symptoms so they would come back as soon as they appear to spare their children further pain. They also have to isolate their child at home away from school/ kindergarten until they get recovered.

- The atypical form of the disease infects higher age groups, with coxackie A6 virus being the dominant causative agent.
  Here it most likely would progress to encephalitis.
- Can have severe presentation due to the aggressiveness of the adult immune response.
  These include fever, arthralgia and flu like symptoms with the rash as vesicles that affect the nose, cheeks, extensor arms, elbows, thighs, buttocks, groin. These are ATYPICAL RASHES.

The **encephalitis** associated with HFMD for individuals of all age groups is **more likely to be severe**. So, we don't want it to get advanced now do we?

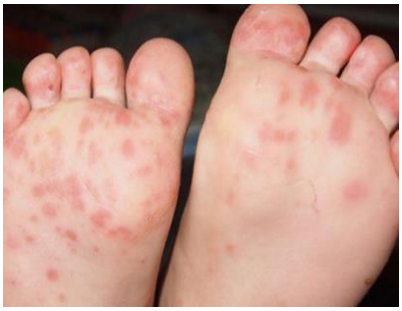
Progression:

It starts with URTI symptoms with fever, malaise and pharyngitis, then comes the rash. The symptom we depend on to decide whether to release the patient is fever, since it denotes systemic involvement. (fever= no release)



The rash is typically on the soles, palms and buttocks, and has this appearance:

- ORAL: football shaped (eye shaped) painful vesicles, involves the buccal mucosa and tongue. (spares posterior pharynx as opposed to herpangina that spares anterior pharynx)
- SKIN: red papules that progress to gray vesicles.



^Although it's vesicular it can commonly have a macular appearance we call it a maculovesicular rash.

Transmission:

- Two routes of transmission feco-oral and direct contact in the rash phase.
- Shedding occurs (through feces since it's feco-oral) before the URTI symptoms even appear.

We can, however, limit the bad outcomes of shedding after seeing the symptoms by giving the patient an extra day or two away from contact even after the symptoms resolve to make sure they won't be sharing their little virus friend.

We actually worry about sequestering the patient in this disease more than in common cold because of the possible progressions mentioned before, so it's important to differentiate.

Treatment: symptomatic (treat fever, pain)
we MUST maintain hydration; it is essential as in all fevers. However; a severe form of it would be combined with neurological symptoms or endocarditis.

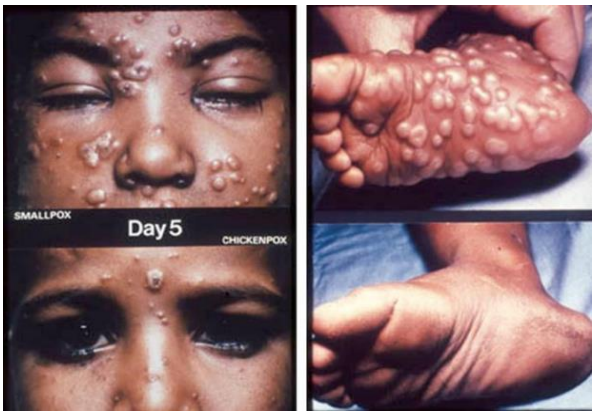
- We consider hospital admission in severe illness (enterovirus A71) which has high morbidity and mortality, as it carries risk of Encephalitis!

Small pox (briefly, because we hope to NEVER SEE IT AGAIN)

- We still mention it even though it was declared eradicated by WHO in 1980 after the last case reported in Somalia (1977), because we have no immunity against it for about 2 generations, and humans can be monsters who'd dig anything back out to life, or use anything to win, even ghost viruses!
- variola virus, and orthopoxvirus are the causative agents.
    - Two strains: variola major (mortality 20–50%) and variola minor.

Progression:

- The incubation period is 10–12 days and is followed by a prodromal period of 1–2 days. The centrifugal rash is initially maculopapular and progresses to vesicles, pustules, and scabs over 1–2 weeks.
- Death may occur with fulminant disease.
- Diagnosis may be confirmed by EM or PCR (to differentiate it from other poxviruses) and there is no specific treatment (supportive).



(comparison between smallpox and chickenpox)

Orf

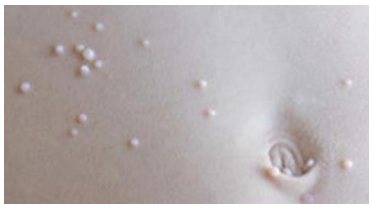
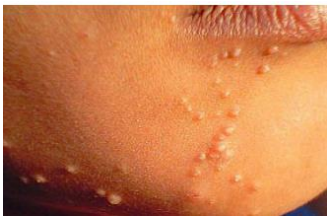
- Sore mouth disease of goat and sheep
    - humans get it in the form of finger lesions due to handling these sheep (feeding, harnessing and petting)
    - Causative agent is Parapox virus (DNA virus related to smallpox virus)

transmission: animal to human and human to human.

How to pick it up in your differential?
Hear the story of the patient! If it says that there was a scratch and then it slowly formed an abscess you suspect bacterial, if they said it started out as a small papule then a small vesicle which then quickly morphed into a nodule, which may have ulcerated or crusted then suspect Orf.

- There is no treatment. However, the papules may become **infected by bacteria**, or in case of immune compromise → **treat with antibacterials**
- Can be infected multiple times through out life, no acquired full immunity. However, each time is less severe.
  Progression: progresses in Six stages: each about a week.
  <Small papule→ nodules → ulcerate and crust>
  It can infect the same patient over & over again with the continuous exposure.

Molluscum contagiosum


- Clusters (<30) of virus induced flaccid, Skin colored (non-erythematous) and umbilicated vesicular rash.
  - Can infect the healthy AND the immune compromised.
  - Appears on the skin. (On face and trunk, pubis and rarely mucosa)
- It's innocuous, **spots resolve** on their own and may be present for YEARS.
  - Cryotherapy or other dermatologic treatments can be done when the site of this rash is sensitive or bothersome.
- The severe form accompanies HIV. This skin colored, localized, innocent rash becomes widespread, erythematous, broken with the permeation of super infections (in immune compromised patients they can become very severe)
  Complications:
  1. Scarring, especially if manipulated. 2. Bacterial secondary infections
  3. conjunctivitis for those that are near the eyes.

Crusted lesions (Crusted lesions are lesions LARGER than vesicles that crust over)

- In general, caused by anything but viruses:

Bullous impetigo/ecthyma	*S. aureus*
Impetigo contagiosa	*S. pyogenes*
Ringworm	Superficial dermatophyte fungi
Sporotrichosis	*Sporothrix schenckii*
Histoplasmosis	*Histoplasma capsulatum*
Coccidioidomycosis	*Coccidioides immitis*
Blastomycosis	*Blastomyces dermatitidis*
Cutaneous leishmaniasis	*Leishmania* spp.
Cutaneous tuberculosis	*Mycobacterium tuberculosis*
Nocardiosis	*Nocardia asteroides*

Impetigo

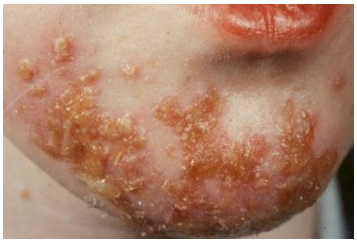
Common case profile

In the **summer**, or in a country of **high temperatures** all around the year, a **child (2-5 yrs mostly)** playing outside, not taking care of his hygiene and getting **very dirty** gets **skin abrasions**, scratches. Further **scratching** of these areas gets his **own skin flora** (GAS or staph.) inside and over growing. Lymph infected leaves this area, in the **upper layer** of the skin, and form these honey crusted lesions (yellow discharge formed by lymph and dead cells)

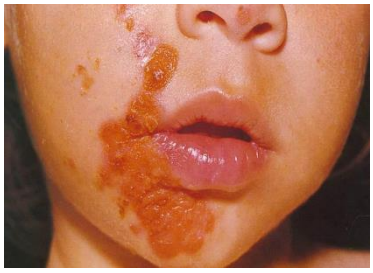
- It is the most common bacterial skin infection in children

  Why do we care more?

  It is common and it's highly contagious (scratching, towels, clothing, autoinfection and spread in daycares). In short, poor hygiene is the problem.



- Clinical features—occurs on the face and extremities.
  - Lesions start as small macules or papules → small vesicles (with erythema) →develop into flaccid bullae → rupture, releasing a yellow discharge which forms thick crusts. [70% are non-bullous and appear as Crusted lesions]
  - Usually seen with regional Lymph adenopathy (there's invasion of the lymph)



Scary scenarios: something that's very common, with high quantity is highly possible to present with the **rare complications**. see next

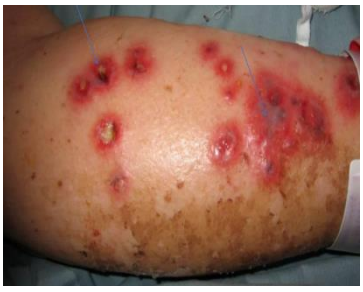
- Impetigo can infect deeper than the epidermis
  - Can cause cellulitis which is more worrisome than impetigo (deeper infection) and it can spread quicker, cause damage and reach the blood, and cause bacteremia.
- PSGN (post strep GN), M proteins of GAS are cross antigenic with other normal tissue antigens, and other strains may cause rheumatic fever.

  Management:
- Most important part is making it prompt. Tell the parents that the child needs to learn better hygiene manners, give topical treatment to avoid complications→ mupirocin is the best topical agent.

  Here you throw the safety net, you tell them that if the lesions progressed, increased, if swelling, erythema showed they SHOULD come back, or pre prescribe antimicrobials to take if progression appeared.
- Patients who have numerous lesions or who do not respond to topical treatment should receive oral antibiotics

  (flucloxacillin, go to drug for skin infections caused by normal flora, or cefalexin).
  - If MRSA is suspected/isolated, you give doxycycline, clindamycin, or co-trimoxazole with hospitalization.

Ecthyma



Craters (holes) in the skin, usually caused by *Staph. aureus* or GAS.

- Punched out ulcers that are deeper than the epidermis, with elevated erythematous or violet margins (moon craters appearance)

- Other similar lesions (ecthyma gangrenosum) may occur with *P. aeruginosa* in neutropenic (reduced neutrophils in blood) patients. (immune depressed)
- Can progress from impetigo[1] or start out as ecthyma[2] right away

Treatment:

Empirically we treat it with flucloxacillin or cephalexin (unless cultures yield streptococci alone, in which case penicillin is appropriate). Notice in the deeper form, topical treatment isn't used rather systemic is•

- Antipseudomonal agents, e.g. piperacillin-tazobactam, used for pseudomonas.

Dermatophytes (fungal cause of crusted lesions)

- A group of fungi 🎈 (more than one) that share two things → capable of invading [1] and feeding off of the dead keratin[2] of skin, hair, and nails (require keratin for their growth). They don't invade usually, because no keratin deeply.
  Transmission: patient to patient or environment (soil or animal) to patient.

Common case profile
A child, playing in the mud and dirt, few days later a ring worm lesion appears on his arm. Most of these would dissolve on their own, so they don't come seeking your help, those who come it didn't actually dissolve on its own. So, You're more likely to see more advanced forms. **Or a gym attendee, complaining about sudden discomfort and smelliness in their feet.**

Classification by age group:

Children:

- *Tinea capitis* (scalp hair and the commonest in children), 👤
- *Tinea corporis* (trunk and limbs) 🧍
- *Tinea faciale* (face) 😟

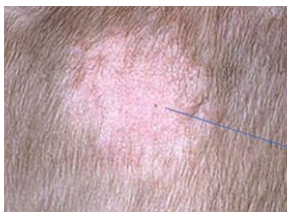
Adolescents:

- *Tinea manuum* and *pedis* (palms and soles – athletes foot- the commonest overall worldwide) 🤚👣
- *Tinea unguium* (nail—also known as onychomycosis) 🖐

Adults:

- *Tinea cruris* (groin) AKA jock itch ♂ ♀
- *Tinea barbae* (beard area and neck) 😶
- *Tinea corporis Gladiatorum* (wrestlers) 🏋

Tinea capitis is BAD, the child infected with it might transfer it to other members of the family (like siblings), which presents a problem.

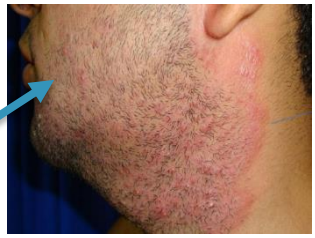


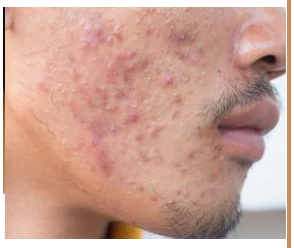
A complication of *Tinea capitis* is that if the hair follicles get affected the hair doesn't grow back, even if it grows back it grows back weaker.

- The main **challenge** in tinea infections for medicine is to differentiate it from other dermatological conditions

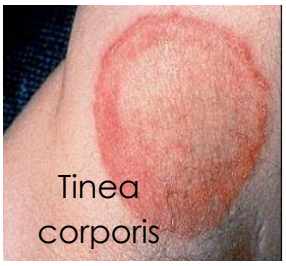
Mix up can happen between acne vulgaris and tinea barbae.

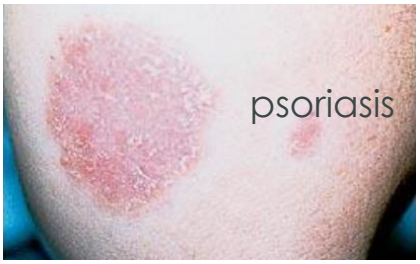
>The difference is in the presence of demarcation lines with erythema within in tinea barbae<

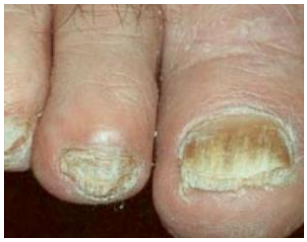


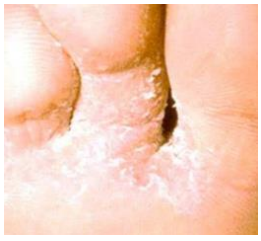


- *Tinea corporis* vs psoriasis, one is infective, the other is autoimmune. Presentations are similar, psoriasis however tends to be itchier and gives silvery white scales.



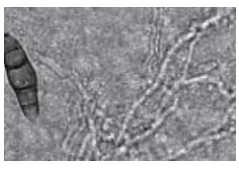






- *Tinea unguium* or athletic foot (tinea pedeis) are easy to diagnose

Diagnosis: The goal is to distinguish dermatophytoses from other causes of skin inflammation since Infections caused by bacteria, other fungi, and noninfectious disorders (psoriasis, contact dermatitis) may have similar features.



- KOH mounts of skin scrapings and infected hairs demonstrate hyphae.
- Some species fluoresce by a U.V. lamp.
- Culture is used when KOH preparations are negative.

Treatment:

"Anything that grows slowly, dies slowly. And here, tinea is the case"

- Topical use of tolnaftate, allylamines, or azoles is usually sufficient.
- Nail bed and more extensive skin infections require systemic therapy with griseofulvin or itraconazole and terbinafine combined with topical therapy. Therapy must be continued over weeks to months, and relapses may occur.
- No specific preventive measures such as vaccines exist.

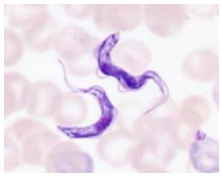
Cutaneous leishmaniasis

Common case profile
Poor person, no access to resources whether water or health care and living outside all year. Refugees for example, no resources no nothing or Bedwins that live outside in tents.

- Our region is endemic for leishmania, that's why we mention it. It's becoming an even bigger issue in surrounding countries especially Iraq and Syria (no sanitation, poor health standards due to war)

Transmission: by sandflies

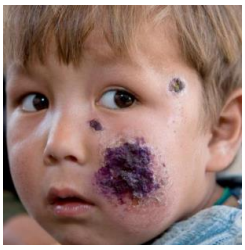
- Parasite Has two forms: disease causing (resistant) form and infectious motile form (promastigote) which it does alternate between to cause the disease.



Deemed as a **neglected** tropical illness (causes mortality with small effort to battle it) → **Disease of the poor**

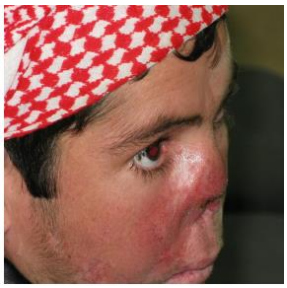
Leishmania comes in 3 forms (Visceral or Kala Azar- serious form), mucocutaneous and cutaneous. The visceral being the one with most mortalities.

- The cutaneous forms Has many types (other than visceral)
    - Old world in Asia, Africa and Europe → long incubation time (2-24 MONTHS).



Progression: Skin lesion(s) on the face or leg
papule at site of bite → small nodules→ painLESS ulcer→ crust (pigmented) → leave ugly pigmented scars after healing (social issues)

"In 2015 over two thirds of new CL cases occurred in 6 countries: Afghanistan, Algeria, Brazil, Colombia, Iran (Islamic Republic of) and the Syrian Arab Republic"



- Mucocutaneous: leads to partial or total destruction of mucous membranes of the nose, mouth and throat.
    - If not treated it could leave a hole in place of mouth and nose

"Over 90% of mucocutaneous leishmaniasis cases occur in Bolivia (the Plurinational State of), Brazil, Ethiopia and Peru"->countries with poor sanitation.

- *L. major* and *L. tropics* are **most common causes**
- would Resolve over months, but **round depressed scars remain**

May resemble other skin lesions (**nodular lymphangitis**) caused by waterborne pathogens (such as *Sporothrix schenckii*, *Nocardia brasiliensis*, *Mycobacterium marinum*, *Leishmania (Viannia)*.
Nodular lymphangitis is a granulamatous reaction to these pathogens on the path of lymphatics.

"700 000 to 1 million new cases and 20 000 to 30 000 deaths occur annually"

Diagnosis:

• Remove crusts and take **skin scrape** for microbiology or DX by culture.
• Biopsy (punch or needle aspirate) to retrieve organism and detect under microscope.
• On **CBC shows reduced cell count** (red, white or all).

Treatment:

Local heat to area for 2-3 hours a day

- **Pentavalent antimonials** (group of chemicals given for CL)
- Others include: **Liposomal amphotericin B**, **Oral miltefosine**, **Pentamidine**
- Given for a minimum of 20 days!

Note//it will leave a scar/impression that the patient can't get rid of even after healing.

Remember that most of the time the people you're treating here live outside, they don't have a good health access so you have to take into consideration letting them know that the treatment should be continued to work.

Infections Associated with Bullae

Bullous diseases are associated with bacteria, (others like TEN and S-J syndrome are viral or drug induced). These bacteria are also mostly G+ves, which may lay the path for anaerobes and G-ves to join the party.

Bullae	
Staphylococcal scalded-skin syndrome	*S. aureus*
Necrotizing fasciitis	*S. pyogenes*, *Clostridium* spp., mixed aerobes and anaerobes
Gas gangrene	*Clostridium* spp.
Halophilic vibrio	*Vibrio vulnificus*

Staphylococcal scalded skin syndrome

It's caused by HEMATOLOGIC spread of staphylococcal particles and EXOTOXIN.

- That means there is a point of infection somewhere else in the body → (Otitis media, Respiratory tract infection)
- Staph aureus that carry exfoliative toxins A and B (only about 5% of all S. aureus) a low percent of the bacteria could get infected acquiring the ability to produce toxins by the transduction mechanisms of a phage infection.
- Breaks down desmoglein-1 resulting in Acantholysis (breakage of cell to cell adhesions)

Common case profile

A baby just born, or not over few months old, with an infection (any kind of infection. URTI, otitis media for instance) coming from staph. especially aureus with exfoliatin.

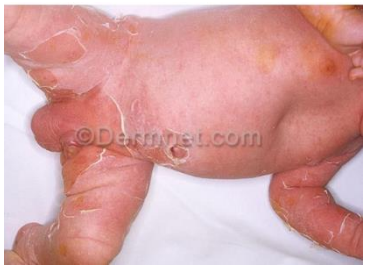
It gained access to the blood, a second fever happens, it'll affect skin EVERYWHERE.

Symptoms:

Preceded by a prodromal illness (URTI) or Otitis Media, Pharyngitis, Conjunctivitis, then the acute phase hits: fever + malaise (due to loss of fluids) and red painful skin with bullae formation.

Signs:

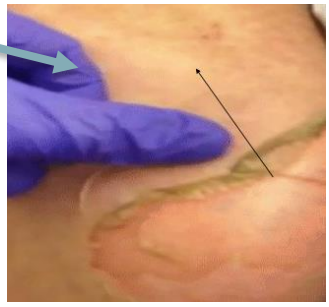
Paper thin (peeling) skin • Large flaccid BLISTERS, more in the flexor creases



Mucous membranes are SPARED! Unlike TEN. (in SJS they aren't spared either but TEN is more similar to SSSS)

Diagnosis:

- Positive Nikolsky's sign (detects acantholysis)
- In lab we can do exotoxin assay or A blood culture would be positive because such presentation is caused by bacteria gaining access to the blood



Treatment :

- MUST ADMIT THE CHILD (burn unit or ICU!)
- Systemic IV antibiotics are given (anti MRSA or anti S. aureus)
- Systemic steroids (only if patient doesn't look toxic/otherwise it is not used)
- In severe cases (IV Immunoglobulins and plasmapheresis) removal of plasma, to remove the toxins
- Cortisone addition is a susceptible point; some clinicians don't agree with & won't recommend.

Punch biopsy with frozen section is useful in making the distinction between it and TEN since the cleavage plane is the stratum corneum in SSSS and the stratum germinatum in TEN

TEN

- TEN, primarily seen in adults is potentially fatal.
- Intravenous γ-globulin is a promising treatment for TEN

Do your best