Derma Team 436

Drug eruption

Objectives:

Not provided

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Template by group B

Before you start.. CHECK THE EDITING FILE

Sources: doctor's slides and notes + 436 group B team
[Color index: Slides| Slides| Important|doctor notes|Extra]

Introduction

- The skin is one of the most common targets for adverse drug reactions.
- 1-5% of patients receiving antibiotics and anticonvulsants may develop a drug eruption.
- ~2% of all drug-induced skin reactions are considered ''serious''.
- Skin reactions to drugs are responsible for ~3% of all disabling injuries that occur during hospitalizations.
- **Either due to:**
  - Immunologic response.
  - Non-immunologic (Overdose, side effect, drug-drug interaction, metabolism…etc).
- **Detailed drug history is very important is diagnosing drug reactions:** What is the drug? The dose? How much did you take? For how long have you been taking it? Have you ever took it before or not?

Diagnostic approach for drug eruptions:

Clinical characteristics

- Type of primary lesion.
- Distribution and number of lesions.
- Mucous membrane involvement, facial edema (Because some reactions occur in face).
- Associated signs and symptoms (Because sometine the things that happens to skin might occur in other organs).

Chronological factors

- Document all drugs to which the patient has been exposed
- Date of eruption, time interval.

It is very important to know when the drug was given and when the euption started because it will give you a clue.

Literature search (Everyday there's a new case report about a drug eruption)

Major drug-induced eruptions

Exanthematous eruptions	Urticaria	Anaphylaxis	Fixed drug eruption
Acute generalized exanthematous pustulosis (AGEP)	Drug reaction with eosinophilia and systemic symptoms (DRESS)	Stevens-Johnson syndrome (SJS)	Toxic epidermal necrolysis (TEN)

1) Exanthematous Drug Eruptions

- The most common drug reaction affecting the skin.
- Classically begins 7 to 14 days after the start of a new medication.
- Begins as erythematous macules (symmetric) that sometimes becomes palpable. (maculopapular)
- Begins on the trunk and upper extremities and progressively becomes confluent. (grouped)
- Mucous membranes are usually spared. **Mucos membreane involvement:** SJS, TEN, EM due to meds
- Pruritis (low grade) and low-grade fever and often present.
  - Fever is not always due to infection, could be immunological because of cytokines ,TH-1 mediator or IL-1
- The eruption disappears spontaneously after 1-2 weeks without complications.
  - If we stop the medication it will resolve within 1-2 weeks. Sometime even when the drug isn't stopped it resolves, the body's immune system gets used to it
- The following classes of drugs have a significantly higher incidence: Aminopenicillins, Sulfonamides, Cephalosporins and anticonvulsants. (Anticonvulsants could cause any skin eruption)

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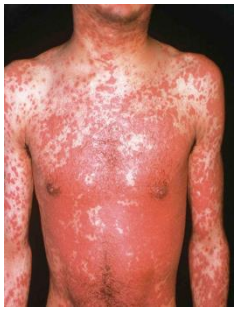
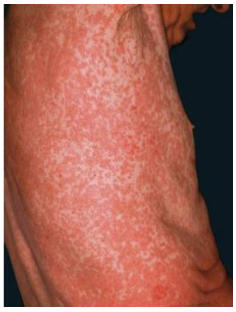
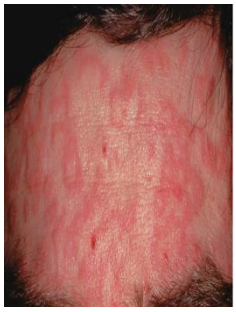
• Very clear confluent distribution, you can't see clear solitary macules or papules.
• If you press on it it's blanchable
• Started at trunk then extremities
• **Look different from TEN's features**: Nikolsky, detachment, blistering, target-like

Urticariated: annular and edematous

Numerous pink papules on the trunk due to a cephalosporin (A). Confluence of lesions on the trunk (B) and annular plaques on the forehead (C) secondary to phenobarbital.

Always look for the following:

- Edema of the face (because it might be a different diagnosis) + blood eosinophilia (DRESS).
- Mucous membrane lesions or painful dusky skin (SJS, TEN).
  - It could be the beginning (no nikolsky or blistering yet) of SJS or TEN
  - **The clue is:** mucocutanous involvment

Summary:
- **Morphology:** maculopapular
- Generalized
- Blanchable
- Starts at trunk
- Itchty
- Possible fever
- 1-2 weeks to appear
- 1-2 week to resolve

Histology:

- Nonspecific changes, eosinophils may be present.
  - We do histology when were'e in doubt

The major DDx

- Viral exanthem (often indistinguishable).
  - Drug etiology favored in adults, viral favored in pediatric patients.
  - The presence of peripheral blood eosinophilia favors a drug reaction.
  - If viral you'll find low lymphocytes
  - In peds, there's always a confusion if it's viral or drug because most viruses present as maculopapular. Even biopsy sometimes wouldn't help. They build the decision on other symptoms and what it most appropriate with the presentation.

Treatment:

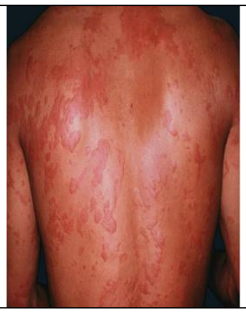
- Supportive (symptomatic: antihistamine)
- Discontinue the offending drug (risk vs benefit).
- Topical antipruritics and corticosteroids may help to alleviate pruritis. Or systemic antihistamine
- So continuing the drug is not life threatening and discounting it sometimes is not in the favor of the patient (pneumonia , UTI).
- We tell the primary physician that we will observe, meaning daily derma checksups, if it is still exanthmatous we're good. If it started to become SJS,TEN or vasculitis STOP the drug.
- Vancomycin is one of the most common causes of exanthmatous.

2) Urticaria, Angioedema and Anaphylaxis

Urticaria:

- Urticaria is either acute or chronic
- Here we will discuss acute
- Urticaria is edematous, red, blanchable plaque
- Example: After a new hair dye
- 90% of people will have an attack of aute urticaria once in their lives
- **Chronic urticaria:** pink spots appear then elevate and disappear in less than an hour. Then others appear.
- **Acute urticaria:** After an antibiotic for example, within half hour they appear. GP can treat this
- **Transient** erythematous and edematous papules and plaques that are usually associated with pruritis.
- They can appear anywhere in the body including palms, soles and scalp.
- Duration is usually a few hours to 24 hours.

We must look at the face to know if there's angioedema



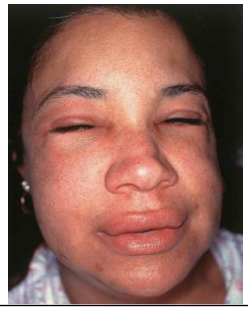
Urticaria secondary to penicillin. Several of the lesions have a figurate appearance.

- Skin is normal after they resolve. Without complications
- **Acute:** Less than 6 weeks.
- **Chronic**: Persist longer.
- Drugs associated with <10% of all cases of urticaria (acute > chronic)
- Mostly antibiotics (Penicillins, cephalosporins).
- **Tx:**
  - Discontinue drug. (but if necessary, we can continue, harm vs benefit)
  - Antihistamines. Why do we give antihistamine? Because it is type-1 immunity, IgE mediated there's mast cells and histamine

• If it is acute, I will always think of drugs but more common is infections
• If chronic think of: food, infection, autoimmun (SLE), endorcrine (hypo/hyper thyroid)

Angioedema:

- Involvment of mucous membrane, edema of lips and eyes, SOB because the muscosa of respiratory system is involved
- Not all urticaria angioedema and not all angioedema urticaria. But the can occur both at the same patietn, so there are 3 catogries.
- **There are many causes:** inherited (due to some missing enzymes in the complement system), drugs, etc..
- **Transient** edema of the dermal, subcutaneous and submucosal tissue.
- Associated with urticaria in 50% of cases.
- May be complicated by life-threatening anaphylaxis. If not managed immediately and properly
- ACE inhibitors (1 day to several years after starting). (due to immune system changes)
- Usually on the face (eyelids, lips), less often on genitals and extremities.
- Unilateral or asymmetric. جهة اكثر من جهة
- Can involve the larynx, epiglottis, oropharynx and intestinal wall.
- **Drug induced angioedema causes**: ACE inhibitors, Penicillins and NSAIDs.
- Life threatning go to ER because the repiratory mucous membrane might close the whole tract and there will be no oxygen then
- Some patient think that taking antihistamine is enough, NO
- **Anaphylaxis**: Patient is unstable with systemic invilvemnt (hypotension)



• Be careful this is not nephrotic syndrome because it would be periorbital edema only.
• Here you can see the lips are edematous too

Anaphylaxis:

- An acute life-threatening reaction that can results from exposure to a number of drugs.
- Penicillin (1 per 5000).
- Combines skin with systemic manifestations (hypotension, tachycardia).
- Serious cases tend to appear within minutes and more common with parenteral administration as compared to oral ingestion.

Treatment: of angioedema and anaphylaxis caused by drugs

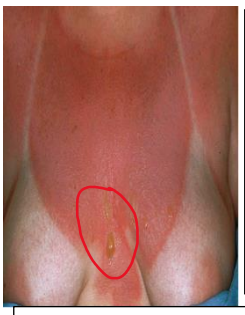
- Discontinue drug and strict avoidance in the future.
- Systemic steroids.
- SubQ epinephrine in cases of life-threatening angioedema or anaphylaxis. (Never in urticaria)
- So antihistamine pill is not enough, patient must come to the ER to measure oxygen level in blood and check if they need an oxygen mask, steroid injection and epinephrine injection.
- Sometimes we give angioedema patients the epinephrine injection to keep it at home with them to they gain more time if the reaction occurs.
- Drug induced anaphylaxis and angioedema are managed similarly

3) Photosensitivity

- **Cutaneous photosensitivity may be:**
  - Idiopathic.
  - Due to endogenous photosensitizers (Porphyrins). Rare
  - Due to Exogenous photosensitizers (Medications). This is what we'll discuss
- **The photosensitivity drug reactions are classically divided into 2 major types:**
  1- Phototoxic (more common).
  2- Photoallergic.

1) Phototoxicity

- Fairly common and predictable.
- Can occur in any person who receives a sufficient amount of a drug together with sufficient exposure to UVR.
- **Clinically**: an exaggerated sunburn in a shorter than expected time.
- Limited to sun-exposed areas and followed by hyperpigmentation.
- Most common drugs: Tetracyclines (doxycycline keep on mind that we give it in acne), NSAIDs, Fluoroquinolones (for UTIs).
- Administering a short half-life drug in the evening decreases the risk.
- **Phototoxic:** we are all at risk, either increased appliance or whatever.

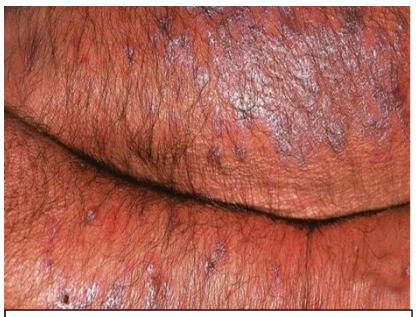


- **How did we know that this is phototoxic not urticaria or exanthmatous?** Because of photosensitive distribution
- Bullea is a sign of inflammation
- Look slike sun burn

Phototoxic reaction in a patient receiving methotrexate.
The erythema and bullae are obviously limited to sun-exposed sites and resemble an exaggerated sunburn. Patients on methotrexate can also experience a "sunburn-recall" phenomenon.

2) Photoallergy

- Occur as a result of cell-mediated hypersensitivity (to an allergen activated or produced by the effect of light on a drug).
- UVR is required to convert the drug into an immunologically active compound (Photo-allergen) that induces the immune response.
- More chronic than phototoxic.
- **Clinically:** Pruritic and resemble dermatitis or lichen planus but primarily in sun-exposed sites.
- **Most common drugs:** Thiazide diuretics, Sulfonamides antibiotics (MC), Sulfonylureas and phenothiazines (all contain sulfur).
- This looks like lichen planus (violaceous, flat topped papules, shiny). The difference is that these aren't on flexure, they are on photosensitive areas.
- Could be lichenoid or drug induced lichen planus that is related to UVR

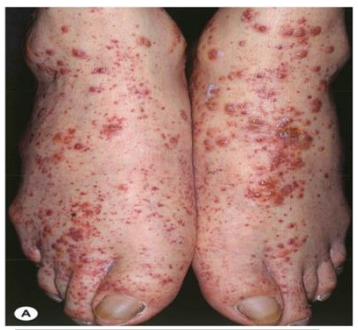


Photolichenoid drug eruption due to hydrochlorothiazide.
The lesions favored the extensor surfaces of the forearms.

Treatment of phototoxicity and photoallergy: Drug withdrawal, Topical steroids, physical barriers, reduce sun exposure + broad-spectrum sunscreens.

4) Vasculitis

- **Causes of vasculitis:** infection, drugs, autoimmune (SLE, CT disease), hepatitis.
- ~10% of the cases are due to drugs. In exthanthematous, SJS or TEN we would say that the most likely cause is drugs. But here it is only 10% of the causes.
- **Clinically:** Purpuric papules on the lower extremities. (non-blanchable)
- Systemic involvement is very unusual.
- Occurs 7-21 days after drug administration and less than 3 days following re-challenge.
- **Most common drugs:** Penicillins, NSAIDs, Sulfonamides and cephalosporins.



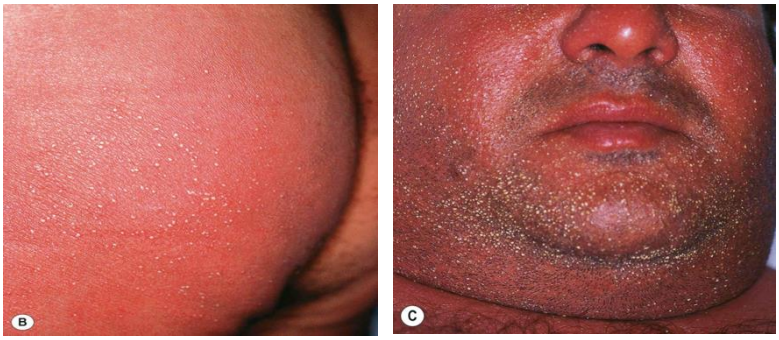
Lower limb purpura

5) Neutrophilic drug eruptions

Meaning that there are neutrophils under the skin, the others were eosinophils

A) Acute generalized exanthematous pustulosis (AGEP)

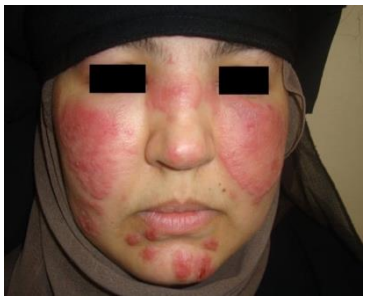
- Acute febrile drug eruption.
- Numerous small, non-follicular, sterile pustules, arising within large areas of edematous erythema.
- More than 90% of cases are drug-induced.
- The onset is usually within 2 days of starting the medication.
- Lesions begin on the face or intertriginous zones (groin, axillae) and then disseminate within a few hours.
- The lesions last for 1 to 2 weeks and are followed by a superficial desquamation.
- **DDx:** Acute pustular psoriasis. (by history and biopsy)
- **Drugs:** Antibiotics (Beta-lactam and macrolides), CCB and Antimalarials (for SLE).



• Numerous non follicular pustules in an ill looking patient.

Acute generalized exanthematous pustulosis (AGEP).
A A positive patch test result 4 days following the application of 0.75% metronidazole in a patient with a previous pustular drug eruption to that medication. Diffuse erythema of the buttock (due to cephalosporin, B) and face (due to metronidazole, C) studded with sterile pustules. Spongiform pustules are seen within the epidermis of lesional skin (D).

B) Sweet's syndrome (Acute febrile neutrophilic dermatosis)

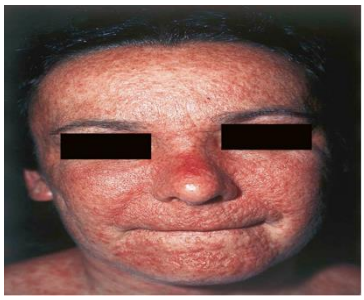


- This syndrome is characterized by fever, peripheral blood neutrophilia, and painful erythematous plaques that favor the face and upper extremities.
- Drugs represent <5% of all cases and starts about a week after the onset of drug administration.
- Involving subcutaneous fat
- The skin presentation might be a plaque or a tumor (lymphoma)
- The body's immune reaction to the tumor
- Most likely it caused by malignancy
- **Scenario:** a patient came in with pancytopenia, and they were confused at the time wondering what could it be. After a while an eruption appeared, and they thought it was cellulitis because it was painful, red and on her face. So, they gave her antibiotics and it didn't improve then shifted to antifungal because she's immunocompromised but still no response. It started to deteriorate and spread all over her body and in general looking ill. After calling the dermatology for consultation they told them this is sweet syndrome this is acute lymphocytic leukemia, go for bone marrow and peripheral blood stem.

6) Drug reaction with eosinophilia and systemic symptoms (DRESS) / drug hypersensitivity syndrome

- If you see exanthematous on the body and edema of the face think of DRESS
- AKA Drug-induced hypersensitivity syndrome (DIHS).
- Due to alteration in the metabolism of drugs + immune mechanisms.
- Possible role for viruses HHV-6 and HHV-7?
- **Drugs:** Anticonvulsants (phenobarbital, Carbamazepine and phenytoin) and sulfonamides.

- **Clinically:**
  - Starts 2-6 weeks after drug initiation.
  - Fever (85%) and a cutaneous eruption (75%) are the most common symptoms.
  - Begins as a morbilliform (maculopapular) eruption, which later becomes edematous, with follicular accentuation.
  - The face, upper trunk and extremities are the initial sites of involvement.
  - Edema of the face is a hallmark of DRESS.
- Lymph nodes are often enlarged.
- The most common and most severe site of visceral involvement is the liver (majority of deaths associated with this syndrome.
- **Other organs involved:** Heart, lungs, kidneys and thyroid.
- Prominent eosinophilia is a very characteristic feature.
- If seen by medicine, they will think it is an infection or TB
- **Treatment:** very serious life threatening
  - Early withdrawal the offending drug. (may not be sufficient for obtaining a full recovery)
  - Topical steroids for mild cases.
  - Systemic steroids for life-threatening heart and lung involvement.
- **The criteria:** facial edema, high liver enzymes and lymph nodes involvement



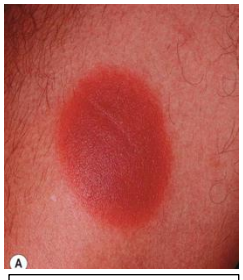
Drug reaction with eosinophilia and systemic symptoms (DRESS)/drug-induced hypersensitivity syndrome (DIHS).
Multiple edematous papules are present.

7) Bullous Eruptions

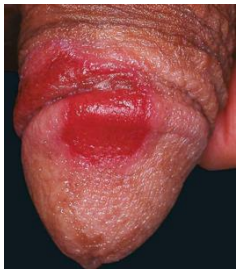
- Fixed drug eruption.
- Linear IgA bullous dermatosis.
- Drug-induced bullous pemphigoid.
- Drug-induced pemphigus.
- Steven-Johnson syndrome and TEN.

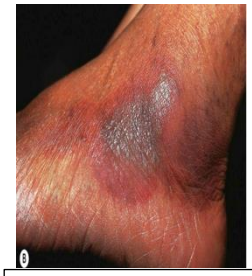
Fixed drug eruption

- Lesions develop 1-2 weeks after a first exposure and within 24 hours within subsequent exposures.
- One or a few round, sharply demarcated, erythematous and edematous plaques are seen.
- Sometimes a dusky, violaceous hue and a central blister may be seen
- Favors the lips, face, hands, feet and genitalia.
- The lesions progressively fade over several days (leaving PIH behind). (brown discolouration)
- Lesions **recur** at exactly the same sites upon re-administration of the drug. (but different morphology)
- There is a generalized form of FDE (similar clinically to EM/SJS).
- A non-pigmenting variant of FDE occurs mainly with pseudoephedrine.
- **Drugs:** Sulfonamides (in burn creams), NSAIDs, Barbiturates, Tetracyclines and Carbamazepine.



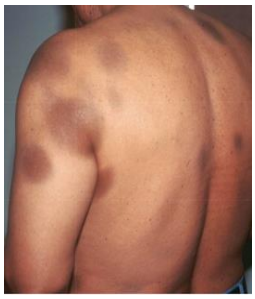
Very round well-defined erythematous plaque

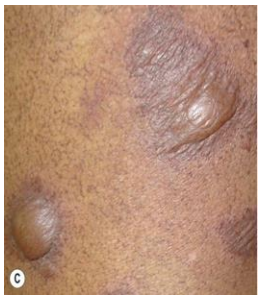




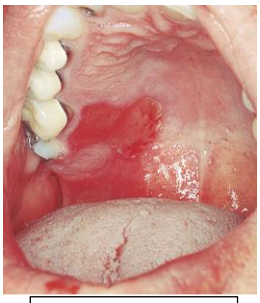
Brown plaque like LP

Fixed drug eruptions.
Well-demarcated erythematous (A) to violet–brown plaques that can develop a detached epidermis (B), bulla (C) or erosion (D,E) centrally. As lesions heal, circular or oval areas of hyperpigmentation are commonly seen (F). Histologically, serous crust, a few necrotic keratinocytes, mild vacuolar changes at the dermal–epidermal junction, papillary dermal edema and melanophages can be seen as well as a mononuclear infiltrate (G). Responsible drugs were phenolphthalein (A), naproxen (B), ciprofloxacin (D), allopurinol (E) and trimethoprim–sulfamethoxazole (F).





Brown plaque with a bullous in the centre



Could affect the mouths mucosa

Summary:
- **Location**: extremities and gentalia
- **Morphology**: different
- Duration: 1-2 weeks
- Leaves pigmentation (so if a patient come with pigmentation you MUST ask "was it red?" and "what are the drug that you take?"

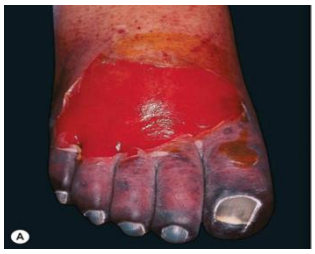
8) Others

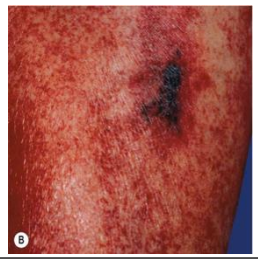
- Anticoagulant-induced skin necrosis.
- Serum sickness-like eruption.
- Drug-induced lupus.
- Drug-induced psoriasis.
- Acneiform eruptions.
- Pigmentary changes.
- Pseudolymphoma.
- Chemotherapy reactions.

A) Anticoagulant-induced skin necrosis

- Sudden red spots > necrosis > ulcer
- Rare, sometimes life-threatening.
- Induced by Warfarin or Heparin.
- Begins 2 to 5 days after therapy.
- Erythematous, painful plaques evolve into hemorrhagic blisters and necrotic ulcers.
- Mainly over the breasts, thighs and buttocks. (fatty areas)
- Patients with hereditary deficiency of protein C are at highest risk.
- So, if you're patient is taking an anticoagulant, order protein C.
- **Tx:** Discontinue warfarin & start Vitamin K + I.V infusion of protein C.

You might think it is bleeding, but it is actually necrosis.

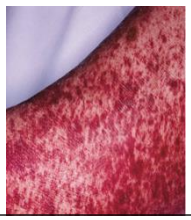




Heparin-induced thrombocytopenia with thrombosis syndrome.
A) Ischemia and necrosis of the foot.
B) Petechiae due to thrombocytopenia and an irregular area of cutaneous necrosis due to thrombosis.

B) Serum sickness-like eruption type-3 immune reaction

- Mainly in children.
- **Type-3:** antigen-antibody complement mediated. So they attach to the skin or blood vessels
- Immediately after taking oral or IV drug, they show symptoms that are not just drug eruption but deficiency of complement in blood. (like vasculitis but here there's fever and hypo-complement in blood)
- **Appears on skin as:** purpura.
- Fever, Arthralgias, arthritis, rash and lymphadenopathy.
- 1 to 3 weeks after drug exposure.
- Unlike true serum sickness, hypocomplementemia, vasculitis and renal disease are absent.



Serum sickness due to antithymocyte globulin. The purpuric lesions are due to small vessel vasculitis in this patient with aplastic anemia.

- Occurs in approximately 1 in 2000 children given cefaclor. Rare

C) Drug-induced Lupus

Drug-Induced systemic lupus:

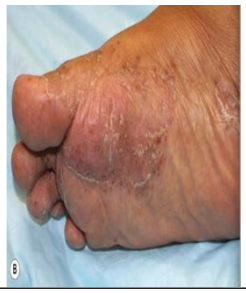
- Fever, weight loss, pericarditis and pulmonary inflammation.
- Skin involvement is rare but includes: malar-erythema, photo eruption and discoid lesions.
- Vasculitis, renal and neurologic involvement is rare.
- Starts over a year after the medication is initiated.
- +ve anti-histone Abs in 95% of cases (-ve DsDNA).
- Clinical symptoms resolve within 4 to 6 weeks.
- Procainamide, hydralazine, chlorpromazine, isoniazid, methyldopa, quinidine, D-penicillamine and Minocycline.
- Dealt by rheumatology

Drug induced subcutaneous lupus:

- Psoriasiform and annular lesions on the upper trunk and extensor arms.
- Hydrochlorothiazide, CCBs, Terbinafine, NSAIDs, Griseofulvin.
- Resolution of the rash may or may not occur after discontinuation of the responsible drug.

D) Drug-induced Psoriasis

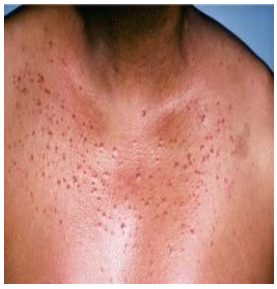
- Drugs can affect a patient in 3 different ways:
  1) Exacerbation of pre-existing psoriasis.
  2) Induction of lesions of psoriasis in clinically normal skin in a patient w psoriasis.
  3) De novo psoriasis.
- Terbinafine, NSAIDs, Antimalarials, ACE inhibitors, Lithium and B-blockers. Thiazide
- TNF-induced Psoriasis. IBD (psoriasiform) , psoriasis itself
- Lesions of drug-induced psoriasis usually regress within weeks to a few months of discontinuing the inciting drug.
- How to know that it is drug induced psoriasis not typical psoriasis? Atypical presentation in drug induced (location missing silver scales)
- First thing to do is we see psoriasis lesion is to check drugs, sometimes we treat the psoriasis and forget to stop the medication, so it won't improve.
- If we stop the drug it will get better and NEVER come back



Psoriasiform eruptions due to TNF-α inhibitors. A Widespread papulosquamous lesions in a patient being treated with infliximab for gastrointestinal GVHD. Histologically, there was no evidence of cutaneous GVHD. B Sterile pustulosis of the plantar surface developed in this patient with rheumatoid arthritis who had received infliximab for the previous 5 years. Neither patient had had a reduction in immunosuppression prior to the onset of the psoriasiform eruption.

E) Acneiform eruptions

- Represent ~1% of drug-induced skin eruptions.
- Clinically, just like acne but comedones are absent.
- Corticosteroids, Androgens, hydantoins, lithium, progestin-containing OCPs.
- Monomorphic



F) Pigmentary changes patient come complaining of facial pigmentation, so we review the drugs

- Hyperpigmentation:

Usually more pronounced in sun-exposed areas.

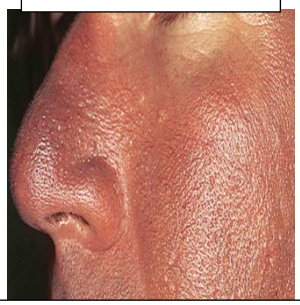
- Minocycline (brown-black photosensitive)
- Antimalarials (hydroxychloroquine = grey-blue)
- Amiodarone (blue-grey)
- Silver, gold and arsenic
- Bleomycin

- Hypopigmentation:

- Chronic use of topical steroids.

If the scenario says pigmentation on face or sun exposed area keep on mind drugs!!

Pigmentary patch



Gray–violet discoloration of the face due to amiodarone. Biopsy specimens demonstrate yellow–brown granules within dermal macrophages. Note sparing of the lower evelid.

Questions:

1) A 22-old-year female presented to dermatology clinic with an oval red patch with central blister on upper back 2 days after using NSAID. She had a similar lesion in the same location 4 months ago after using the same treatment. Which one of the following is the most likely diagnosis?

A. Pemphigus Vulgaris
B. Fixed Drug Eruption
C. Erythema Multiforme
D. Recurrent Discoid Eczema

Answer: B

2) 70 years old man developed fixed drug eruption, after stopping all his medication the lesions have gone, what drug caused this?

A. Doxycycline
B. Statin
C. ACE inhibitors

Answer: A

3) A 60-year-old female with multiple medical problems has been admitted to the hospital 7 days ago. She was started on several medications. She developed black necrotic patches over both breasts for the last 3 days. Which one of the following drugs is most likely the cause?

A. Insulin
B. Warfarin
C. Gentamicin
D. Carbamazepine

Answer: B

4) An ER doctor admits a 70 years old patient with fever, sore throat, malaise and fatigue- The patient was treated at home with an antibiotic and antipyretic- The family was alarmed when he developed acute, itchy exanthematous rash and brought him to hospital- On examination his oral mucosa was free- No past history of systemic disease. What is the most probable cause of the rash in this patient?

A. Viral infection
B. Malignant neoplasm
C. Drugs
D. Streptococcal throat infection

Answer: C

5) A known epileptic patient on phenytoin presented with a morbilliform rash that started on the face. On examination he looks sick and has periorbital edema, fever with tender right hypochondrium and lymphadenopathy. His lab showed eosinophilia, atypical lymphocytes and raised liver enzymes. What is the probable diagnosis?

a. Infectious mononucleosis
b. Dress Syndrome
c. Red man Syndrome
d. Steven Johnson syndrome

Answer:B

6) A 22 yrs old male presented to the ER with 2 weeks history of morbilliform rash that started on the face and proceeded to generalized redness and scaling. On examination he looks sick and has periorbital edema, fever, tender right hypochondrium, lymphadenitis, and muscle aches. He is known epileptic and started phenytoin 1 month ago. His lab showed eosinophilia, atypical lymphocyte, and elevated LFT. What is the most likely diagnosis ?

A. Red man syndrome
B. Steven Johnson Syndrome
C. Drug hypersensitivity syndrome
D. Infectious mononucleosis syndrome

Answer: C

7) A 25 years old male come the dermatology clinic complaining of 4 months history of itchy eruption, which waxes and wanes that is triggered by exercise he described the lesion as small punctuated and monomorphic wheels that is less than half an hour.

A. Solar urticaria
B. Cholinergic urticaria
C. Dermatographism
D. Cold urticaria

Answer: B

8) A19 year old man with acne vulgaris on Minocycline referred to venereal disease clinic with edematous erythematous sharply marginated skin eruptions with blister formation on genital mucosa for two days. He had similar lesions 2 years ago. What is the most likely diagnosis ?

A. Syphilis infection
B. Genital warts
C. Erythema multiforme
D. Fixed drug eruption

Answer: D