LIFE THREATENING SKIN RASHES

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I. INTRODUCTION:

The area of medicine that deals with the skin is called dermatology. It is a specialty having elements of both medicine and surgery. A dermatologist is a type of medical professional who treats conditions of the skin, hair, nails, and some cosmetic issues. The largest organ in the body, the skin acts as a barrier to guard the inside organs from damage and germs. Additionally, it is a reliable indicator of the body's general health, making the study of dermatology important to the detection and management of numerous medical disorders.

Some of the most common dermatologic conditions include:

- **Acne:** pimples on the skin due to inflammation of the sebaceous glands
- **Dermatitis:** red, swollen and sore skin caused by irritation or allergy
- **Eczema**: rough and inflamed skin that is itchy and may bleed
- **Psoriasis:** itchy, red, scaly patches on the ski
- **Fungal infections:** infection of the skin or nails caused by a fungus
- **Warts**: small hard growth on the skin caused by a virus
- **Cold sore:** inflamed blister near the mouth caused by herpes simplex virus
- **Skin cancer:** uncontrolled growth of skin cells

Dermatological emergencies are rare, but if they are not identified and treated right away, they might result in fatal complications.

II. SKIN DISEASES:

An intensive care unit is necessary for the treatment of several skin disorders. The most dangerous skin problems are listed here.

A. Necrotising fasciitis:

The aggressive skin and soft tissue infections (SSTIs) that induce necrosis of the muscular fascia and subcutaneous tissues include necrotizing fasciitis as a subset. The fascial plane, where this illness often spreads, has a weak blood supply. As a result, the underlying tissues are initially untouched, which may delay detection and surgical treatment. The infectious process can progress quickly, infecting the fascia and peri-fascial planes as well as the skin, soft tissue, and muscle directly above and below [1,2,3].

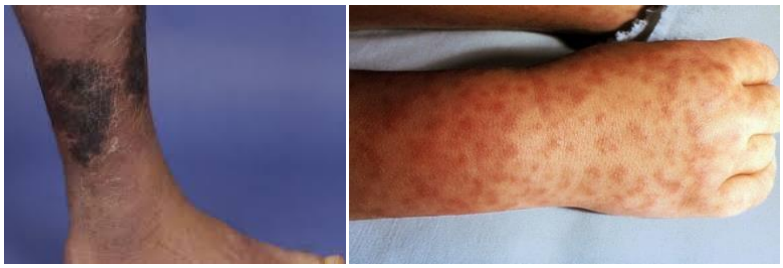


Figure 1: Necrotising fasciitis

Etiology:

Typically, necrotizing fasciitis is an acute disease that spread rapidly over several days. In about 80% of all instances, it is a direct consequence of bacterial infection introduced through a breach in the skin's integrity. The majority of these single-site source infections are caused by gram-positive cocci, specifically strains of Staphylococcus aureus and Streptococci. Additionally, gram-negative and anaerobic participation result in polymicrobial illnesses [4,5].

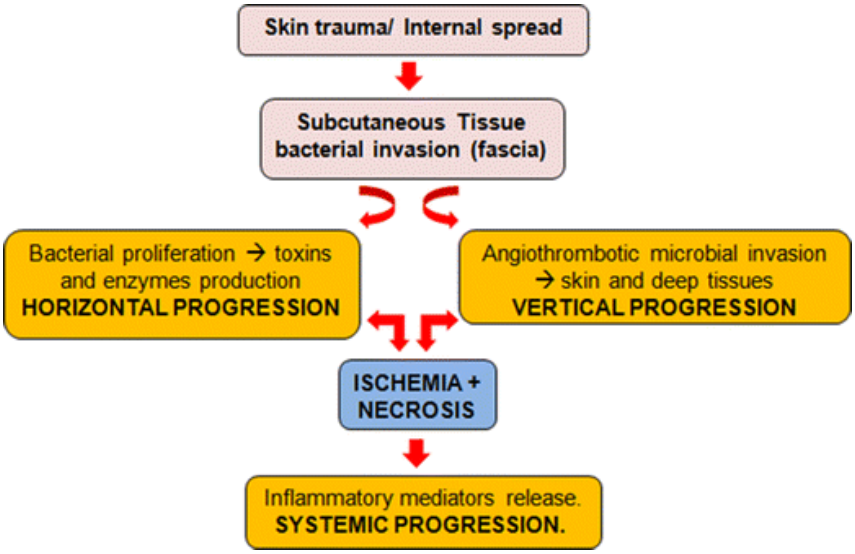


Figure 2: Pathophysiology of Necrotizing fasciitis

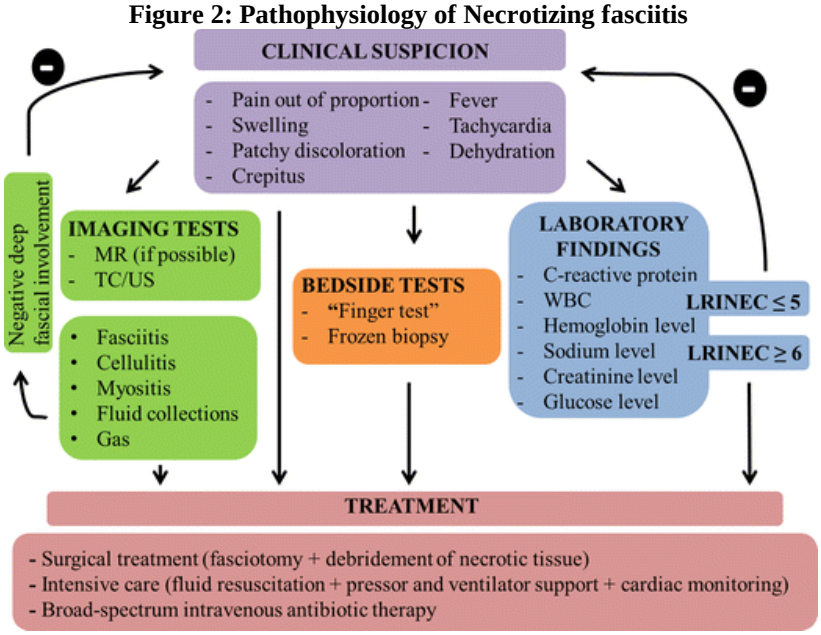


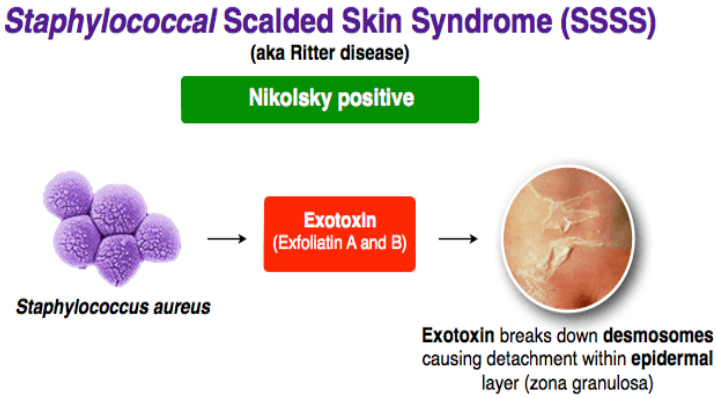
Figure 3: Diagnosis of Necrotizing fasciitis

Outcomes:

A dangerous condition with a fatality rate ranging from 30 to 90% is necrotizing fasciitis. Patient age, organism type, quickness of diagnosis and therapy, and patient comorbidities all play a role in mortality. Patients with certain streptococcal strains have the poorest prognosis. Renal failure, respiratory distress, ARDs, and loss of consciousness are other factors that have a negative impact on prognosis. Patients who receive immediate aggressive debridement, hydration, and broad-spectrum antibiotics have the best chance of surviving. Even following therapy, those with the condition typically have shorter lives than age-matched controls [6].

B. Staphylococcal Scalded skin syndrome:

The skin infection staphylococcal scalded skin syndrome (SSSS) is caused on by the bacterium staphylococcus aureus. The skin's outer layers blister and peel as a result of the bacterium's exfoliative toxin.



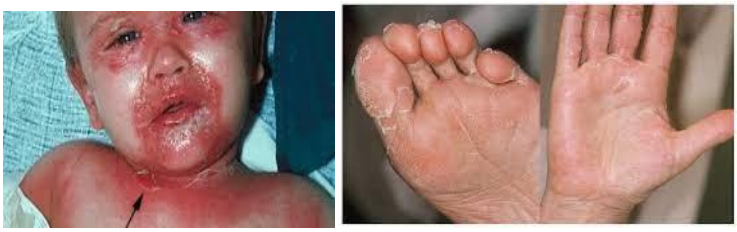


Figure 4: Scalded skin syndrome

When bacteria get into the body through a skin crack. The bacterium's toxin weakens the skin's ability to remain together. The characteristic peeling of SSSS is then set on by the removal of the upper layer of skin from the deeper layers.A reaction might occur all over the skin when the poison enters the bloodstream. Young children, especially newborns, are most susceptible because they have developed immune systems and kidneys that can eliminate toxins from the body.

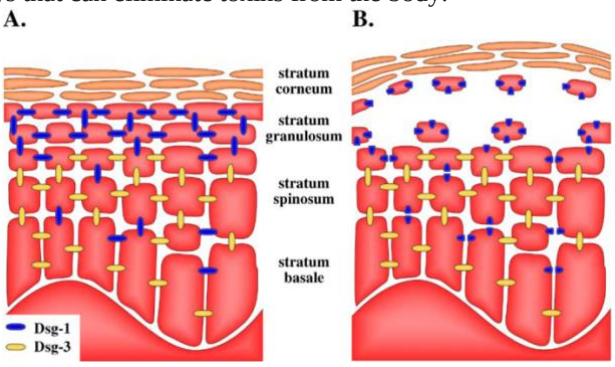


Figure 4: Scalded skin syndrome pathophysiology

The exfoliative toxin-induced splitting at the stratum granulosum is explained by the differential distribution of desmoglein isoforms in the epidermis (8). Desmoglein distribution in (A) healthy skin and (B) skin that has been subjected to an exfoliative toxin is shown schematically. Desmoglein 3 compensates for the exfoliative toxin-mediated hydrolysis of desmoglein 1 (Dsg-1) in all layers other than the stratum granulosum (Dsg-3). The absence of Dsg-3 in the stratum granulosum explains why cells detached and the epidermal layers separated when Dsg-1 was hydrolyzed.

Management of SSSS: As a dermatological emergency, SSSS calls for immediate hospitalisation and care. This typically entails:
Intravenous antibiotics:
First-line: a penicillinase-resistant, anti-staphylococcal antibiotic such as flucloxacillin.
Other options include: ceftriaxone, clarithromycin (for penicillin-allergy), cefazolin, nafcillin, or oxacillin.
Methicillin resistance (MRSA) infection: vancomycin.

C. DRESS Syndrome:

A specific, severe, idiosyncratic reaction to a medicine is known as the "drug rash with eosinophilia and systemic symptoms" (DRESS) syndrome. It is marked by a protracted latency phase. It is followed by a wide range of mild-to-severe systemic presentations and a variety of clinical symptoms, which typically include fever, rash, lymphadenopathy, eosinophilia, and lymphadenopathy. It is a phrase for an unpleasant reaction that is currently used to denote an allergic reaction with a 10% mortality rate.

Early in the course of the condition, patients frequently experience fever, which is then followed by rashes. A pruritic, macular erythema that may involve papules, pustules, or vesicles is more common than a moderate exanthem or significant blistering and skin loss. Lymphadenopathy, hepatitis, pericarditis, interstitial nephritis, or pneumonitis are common manifestations of systemic involvement. Auto-immunity could arise as a result of DRESS [8].

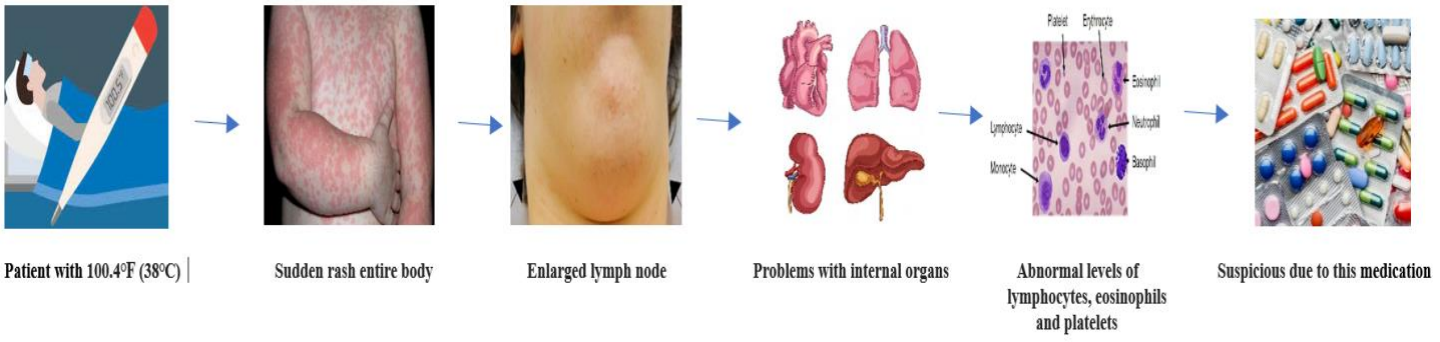


Figure 5: Pathophysiology of DRESS

Genetic factors are also important. The risk of DRESS may be as high as 25% for individuals who have a first degree relative who has experienced this syndrome.

Drugs that lead to dress syndrome

- Anti-seizure drugs, such as lamotrigine (Lamictal), carbamazepine (Tegretol), and phenobarbital
- Allopurinol (Zyloprim)
- Antibiotics, are minocycline (Minocin), vancomycin (Vancocin), and those that are sulfa-based

- Sulfasalazine (Azulfidine), drugs used to treat autoimmune diseases like rheumatoid arthritis and ulcerative colitis
- Nonsteroidal anti-inflammatory drugs (NSAIDs), are ibuprofen (Advil, Motrin) and celecoxib (Celebrex)
- HIV medications, such as nevirapine (Viramune) [9].

Management of DRESS: In order to treat DRESS, systemic corticosteroids have been used.

D. Rockey mountain spotted fever:

The obligate intracellular coccobacillus Rickettsia rickettsii is the source of Rocky Mountain Spotted Fever (RMSF). The disease is spread by ticks, either Dermacentor andersoni (Rocky Mountain wood tick) in the west or Dermacentor variabilis (American dog tick) in the east. Although the disease was first discovered in Idaho and Montana, the majority of cases are concentrated in the south central and southern regions of the United States, making the label "Rocky Mountain spotted fever" misleading from an epidemiologic perspective. Almost all states have received reports of the illness [10].

Since RMSF is a seasonal illness, the majority of cases take place in the spring and summer, when tick activity is at its peak and human-tick contact is most common. The most commonly impacted groups include farmers, kids, and outdoor recreationists. A 5 to 25% mortality rate is possible. The prognosis is greatly influenced by early diagnosis of the illness and the beginning of the proper antibiotic therapy.

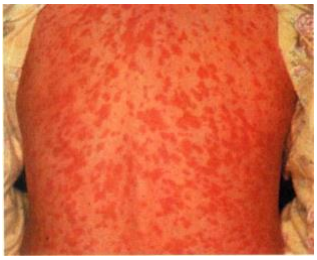


Figure 6: Symptoms of RMSF

Laboratory tests won't help with the diagnosis of RMSF during the acute stage. Recognition is solely dependent on comprehension of the clinical and epidemiologic symptoms, including fever, headache, and rash in people who have had or may have had contact with ticks. It is best to look for signs of recent outdoor activity, travel to an endemic region, or a history of tick bites. Unfortunately, only 60 to 70% of patients initially examined have the characteristic triad of fever, rash, and history of a tick bite.

Management of RMSF:

The cornerstone of treatment for RMSF is doxycycline (administrated intravenously or orally). A pregnant woman or a small child may use chloramphenicol. For the seriously ill patient, supportive care may be required. Importantly, RMSF cannot be treated with normal broad-spectrum antibiotics [11].

E. Toxic epidermal necrolysis (TEN):

Extensive exfoliating of the epidermis and mucous membrane is a feature of toxic epidermal necrolysis (TEN), a potentially fatal illness that can cause sepsis and death. Alan Lyell first referred to it as "an eruption resembling blistering of the skin" in 1956. (12). The disease process that defines Steven-Johnson Syndrome (SJS) is the same as that seen in drug-induced epidermolysis. The degree of skin separation has the greatest difference. (13)

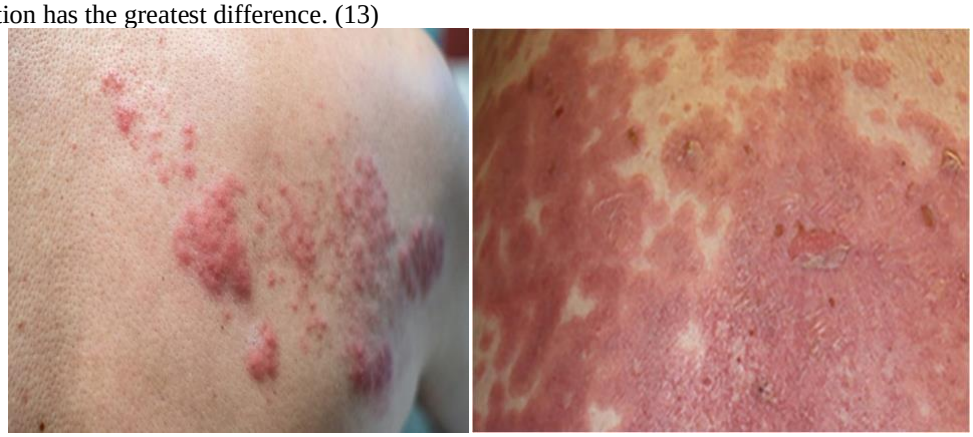


Figure 7: Toxic epidermal necrolysis

Etiology:

Lamotrigine, carbamazepine, phenytoin, nevirapine, phenobarbital, sulfonamide, sulfasalazine, allopurinol, and oxicam-NSAIDs are medication with a high risk of TEN (14,15,16).

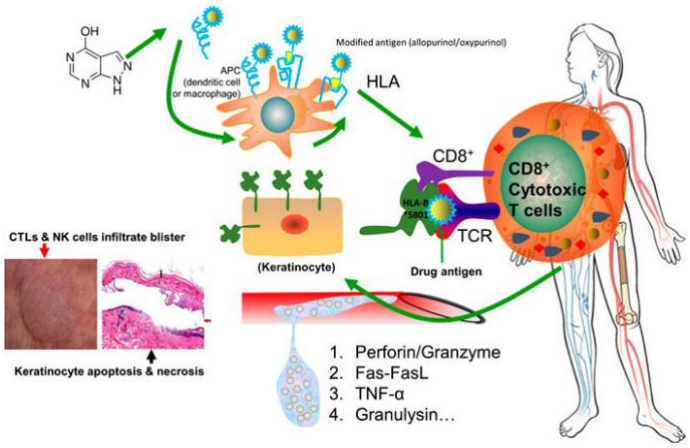


Figure 8: Toxic epidermal necrolysis Pathophysiology

Diagnosis:

Serum granulysin measurements made in the early days of a drug eruption may be able to predict the development of SJS/TEN.

a) **Skin biopsy**: usually necessary to verify the clinical diagnosis and exclude out other generalised rashes with blisters, such as staphylococcal scalded skin syndrome (SSSS), as well as other generalised rashes.

b) **The histopathology** shows keratinocyte necrosis (death of specific skin cells), full thickness epidermal/epithelial necrosis (death of an entire skin layer), and mild inflammation (very mild lymphocytic infiltrate of the superficial dermis). Since the skin biopsy's direct immunofluorescence test came back negative, it is evident that the condition is not caused by the accumulation of antibodies in the skin.

c) **Blood tests** are necessary to identify abnormalities, evaluate prognostic factors, and ensure that hydration and important nutrients have been provided but do not aid in the diagnosis. Abnormalities may include:
   - Anaemia most often occurs (reduced haemoglobin).
   - Leucopenia (low white blood cell counts), particularly lymphopenia (low lymphocyte counts), is extremely common (90%)
   - Neutropenia, or decreased neutrophils, is a symptom that the outcome is poor.
   - There is no eosinophilia (increased eosinophil count) or atypical lymphocytosis (lymphocytes with unusual appearances).
   - 30% of people have mildly elevated liver enzymes, and 10% have observable hepatitis.
   - About 50% of people have mild proteinuria, or protein leakage into the urine. The majority of people experience some changes in renal function.

Investigations are being done on in vitro diagnostic techniques for medication allergies, including SJS/TEN. Patch testing is not advised because it rarely identifies the cause in SJS/TEN.

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