Case 1

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NEW!
Knowledge is power!
Shift

How do the eccrine sweat glands and apocrine sweat glands differ?

Embryologically, eccrine glands derive from the epidermis and are not part of the pilosebaceous unit.

The eccrine sweat glands function in temperature regulation via secretion of sweat, a combination of mostly water and electrolytes, which evaporates and cools the skin.

Their ducts pass through the dermis and epidermis to empty directly onto the skin surface.

Eccrine glands are located everywhere on the skin surface except on modified skin areas, such as the lips, nail beds, external auditory canal, and glans penis. Eccrine sweat glands are found only in higher primates and horses.

……

Apocrine glands originate from the same hair germ that gives rise to the hair follicle and sebaceous gland.

The apocrine duct empties into the follicle above the sebaceous gland. Their anachronistic function is to produce scent.

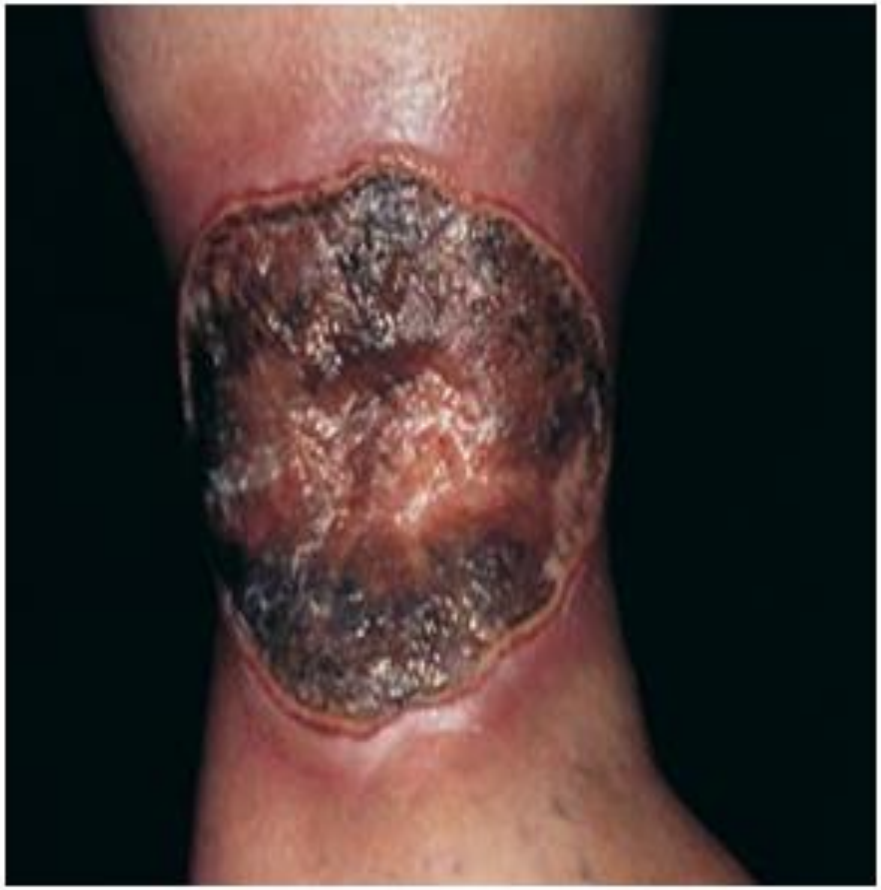
They are located primarily in the axillae and perineum, and their activity is sex hormone–dependent. The breast and cerumen glands are both modified apocrine sweat glands.

Case 1

A 27-year-old man is evaluated in the emergency department for a rapidly progressive ulcer on his leg. The ulcer is extremely tender and has been expanding over the past week. It started approximately 10 to 14 days ago. He describes the initial lesion as a "pimple." He also notes 2 months of abdominal pain and more frequent bowel movements and watery stools, occasionally blood tinged.
On physical examination, he is afebrile and other vital signs are normal. There is no streaking erythema, fluctuance, purulent discharge, expressible pus, or sinus tracts appreciable on clinical examination. The ulcer is tender to palpation. Skin findings are shown.

Which of the following is the most likely diagnosis?

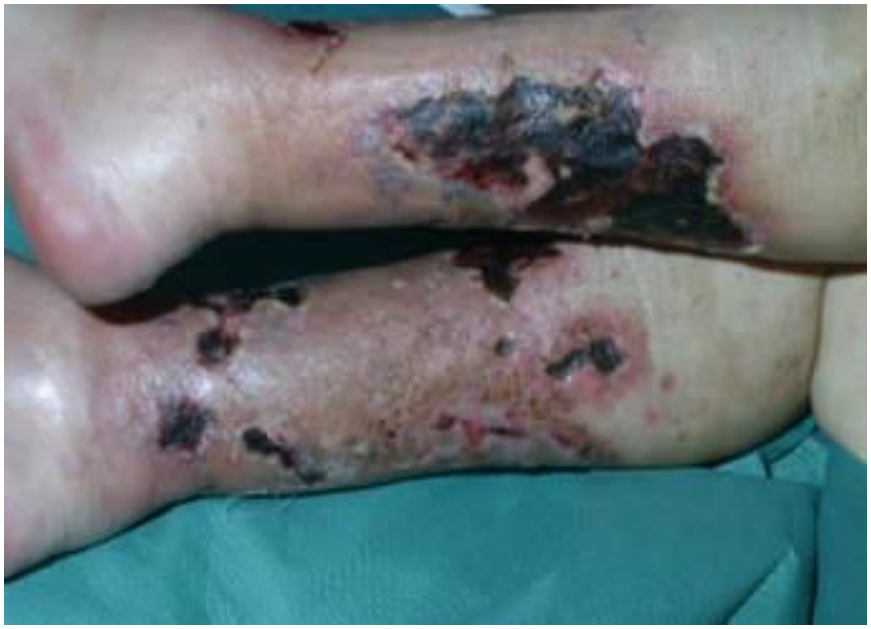
1.Calciphylaxis
2. Ecthyma gangrenosum
3. Necrotizing fasciitis
4.Spider bite
5.Vasculitis
6.induced
7. Pyoderma gangrenosum



Calciphylaxis

Calciphylaxis is a painful ulcerative process due to ectopic calcification of the arteries feeding the skin.

Calciphylaxis nearly always occurs in patients with end-stage kidney disease in the setting of very high calcium-phosphorus products and manifests with reticulated dusky erythema that then ulcerates due to cutaneous ischemia.



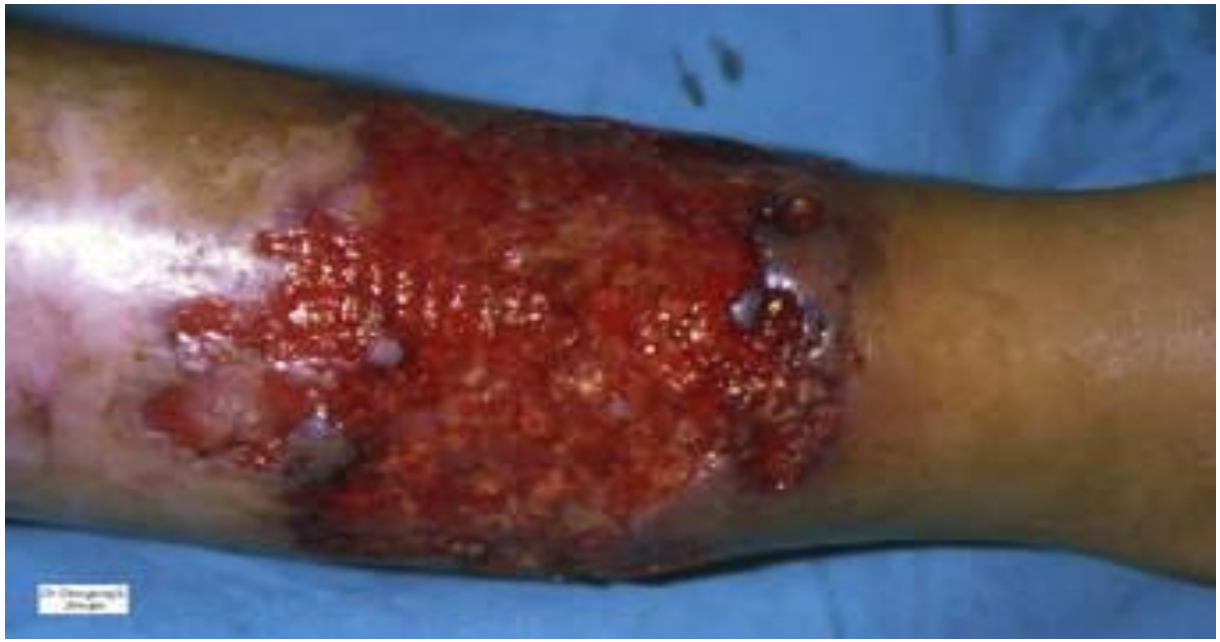
The first skin changes in calciphylaxis lesions are mottling of the skin and induration in a livedo reticularis pattern

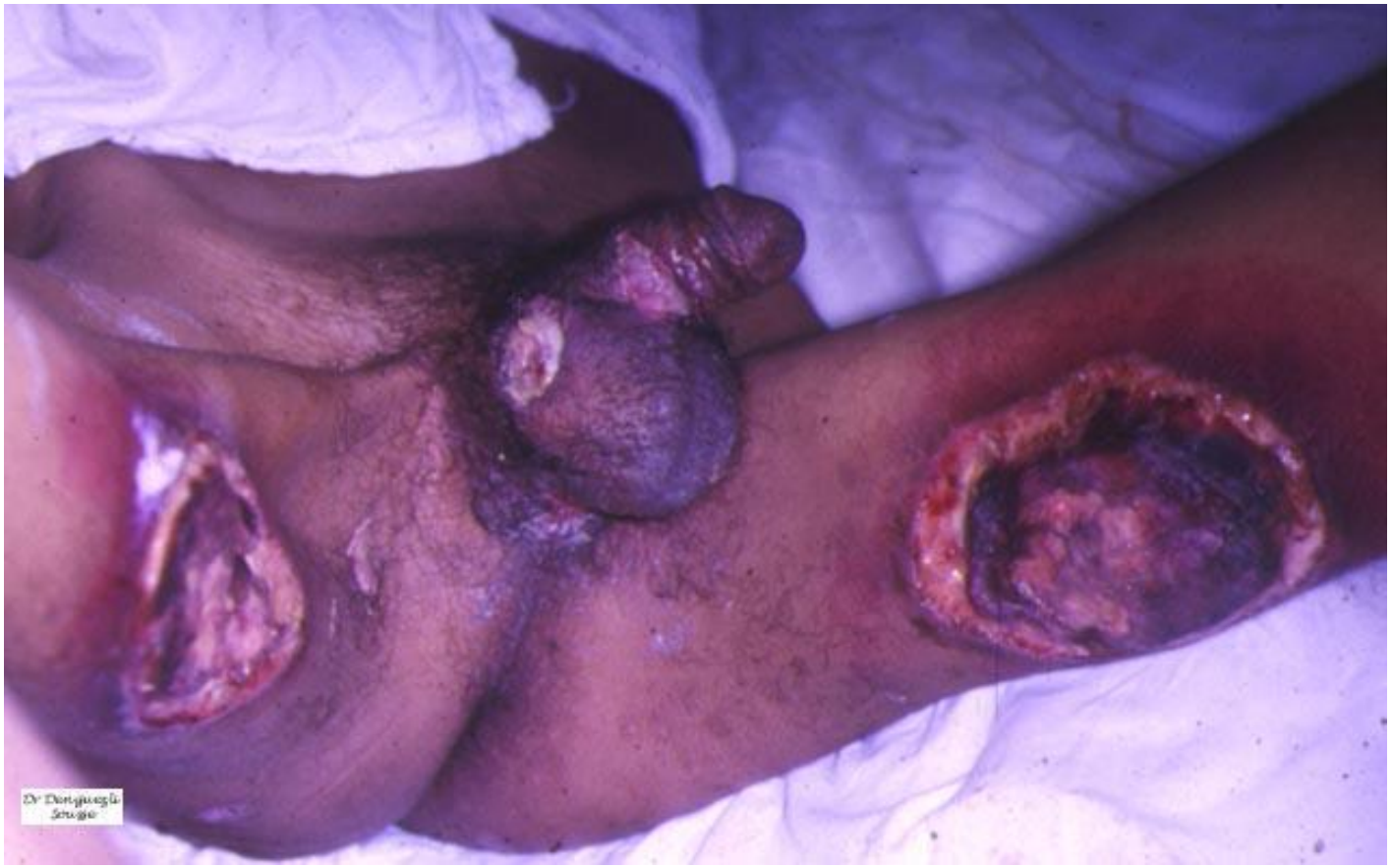
The prognosis is generally not good, with a mortality rate as high as 60-80% in patients with ulcerative disease.

The mortality rate is higher in patients with proximal disease than in those with only distal or acral disease. Patients who do not die of sepsis or organ failure frequently undergo amputation of an involved limb.

Calciphylaxis is one type of extraskeletal calcification

Ecthyma gangrenosum




Dr Dangwala
Senegal

Pathophysiology

Impaired humoral or cellular immunity leads to increased susceptibility to infections with P aeruginosa or other pathogens. In addition, breakdown of mechanical defensive barriers, such as the skin and mucosa, may allow infectious organisms to disseminate. The lesions of ecthyma gangrenosum (EG) result from perivascular bacterial invasion of arteries and veins in the dermis and subcutaneous tissues, producing a necrotizing vasculitis.

Perivascular involvement can occur by hematogenous seeding of the skin in bacteremic patients or by direct inoculation through the skin in non-bacteremic patients.

Extravasation of blood, edema, and necrosis around the vessel interrupt the blood supply to these tissues, resulting in secondary ischemic necrosis of the epidermis and dermis, which manifests as nodular lesions that rapidly evolve through stages of central hemorrhage, ulceration, and necrosis.

Ecthyma gangrenosum results from perivascular bacterial invasion of blood vessel walls with secondary ischemic necrosis.

Multiple lesions may be present at different stages of development.

The infecting agent is usually Pseudomonas aeruginosa and almost always occurs in a significantly immunocompromised patient who is clinically ill.

The primary skin lesion usually starts with a macule that is painless, round and erythematous. Then, it develops into a pustule, and then a bulla with central hemorrhagic focus. The bulla progresses into an ulcer which extends laterally. Finally it becomes a gangrenous ulcer with a central black eschar surrounded by an erythematous halo.

The lesions may be single or multiple. They are most commonly seen in perineum and under arm pit. However, they can occur in any part of the body.

Ecthyma gangrenosum (EG) is a well-recognized but uncommon cutaneous infection classically associated with Pseudomonas aeruginosa bacteremia.

EG usually occurs in patients who are critically ill and immunocompromised; it is almost always a sign of pseudomonal sepsis.

The characteristic lesions of EG are hemorrhagic vesicles or pustules that evolve into necrotic ulcers with a tender erythematous border.

Not all cases have been associated with sepsis.

Necrotizing fasciitis is a rapidly progressive infection of the subcutis, often from a streptococcal infection or polymicrobial flora. Patients are critically ill, and the disease progresses over hours, not days or weeks. Patients typically have extreme pain, dull or dusky skin, potentially with crepitus, and a clinical picture of sepsis.

Necrotizing fasciitis (NF), commonly known as flesh-eating disease, is an infection that results in the death of parts of the body's soft tissue. It is a severe disease of sudden onset that spreads rapidly.Symptoms include red or purple skin in the affected area, severe pain, fever, and vomiting.The most commonly affected areas are the limbs and perineum.

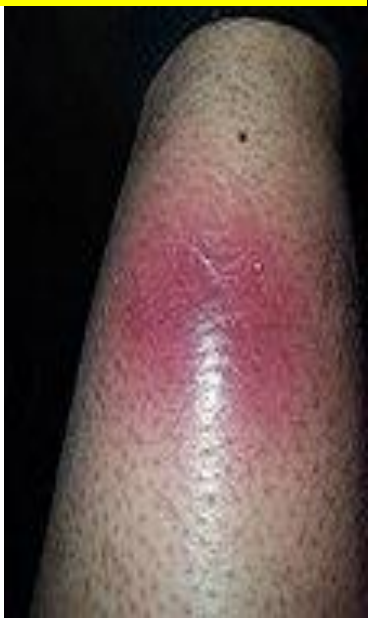
Typically, the infection enters the body through a break in the skin such as a cut or burn.Risk factors include poor immune function such as from diabetes or cancer, obesity, alcoholism, intravenous drug use, and peripheral artery disease.

disease is classified into four types, depending on the infecting organism.

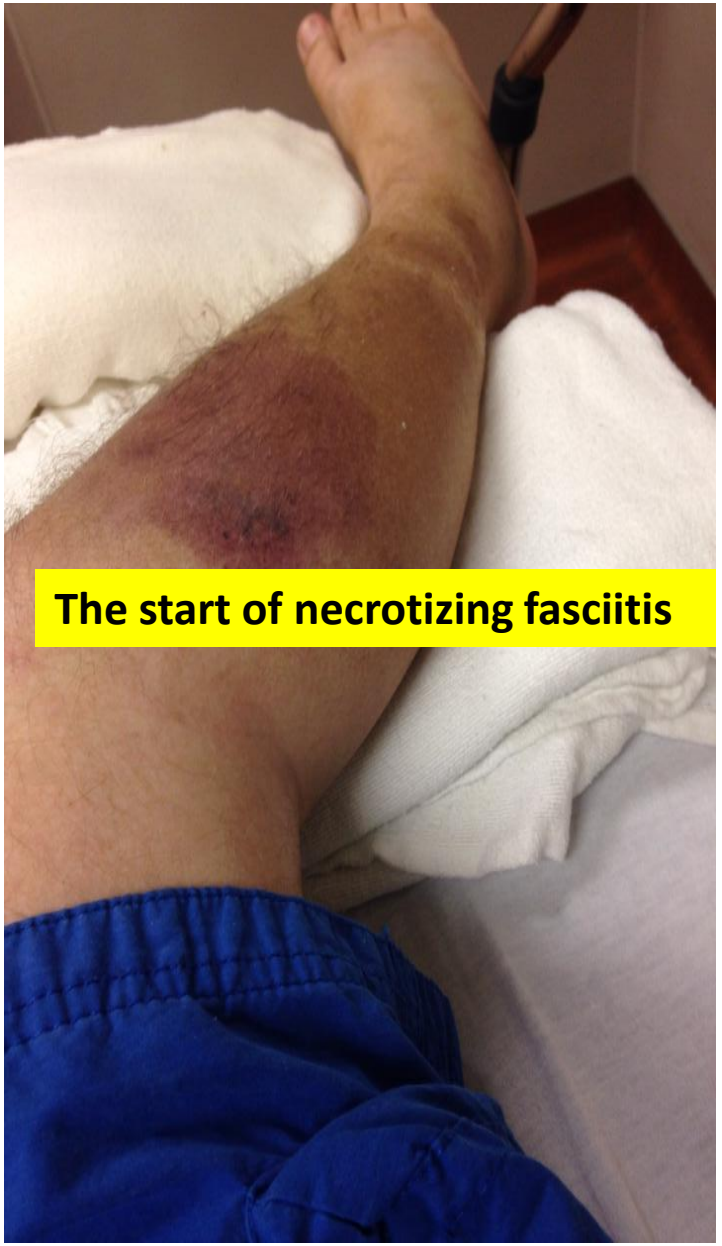
Necrotizing fasciitis may be prevented with proper wound care and handwashing.It is usually treated with surgery to remove the infected tissue, and intravenous antibiotics.Often, a combination of antibiotics is used, such as penicillin G, clindamycin, vancomycin, and gentamicin.Delays in surgery are associated with a much higher risk of death.

Even with high-quality treatment, the risk of death is between 25 and 35%

The very first symptom of NF. The center is clearly getting darker red (purple).



Early symptoms of necrotizing fasciitis. The darker red center is going black.



The start of necrotizing fasciitis

Pyoderma gangrenosum is an uncommon, ulcerative cutaneous condition of uncertain etiology. It is associated with systemic diseases in at least 50% of patients who are affected.

The diagnosis is made by excluding other causes of similar-appearing cutaneous ulcerations, including infection, malignancy, vasculitis, collagen vascular diseases, diabetes, and trauma.

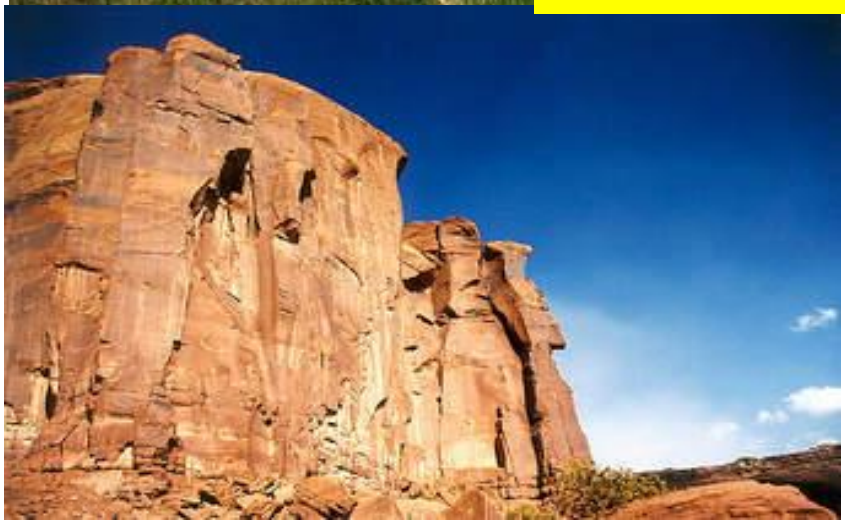
Patients with pyoderma gangrenosum may have involvement of other organ systems that manifests as sterile neutrophilic infiltrates. Culture-negative pulmonary infiltrates are the most common extracutaneous manifestation. Other organs systems that may be involved include the heart, the central nervous system, the gastrointestinal (GI) tract, the eyes, the liver, the spleen, the bones, and the lymph nodes.

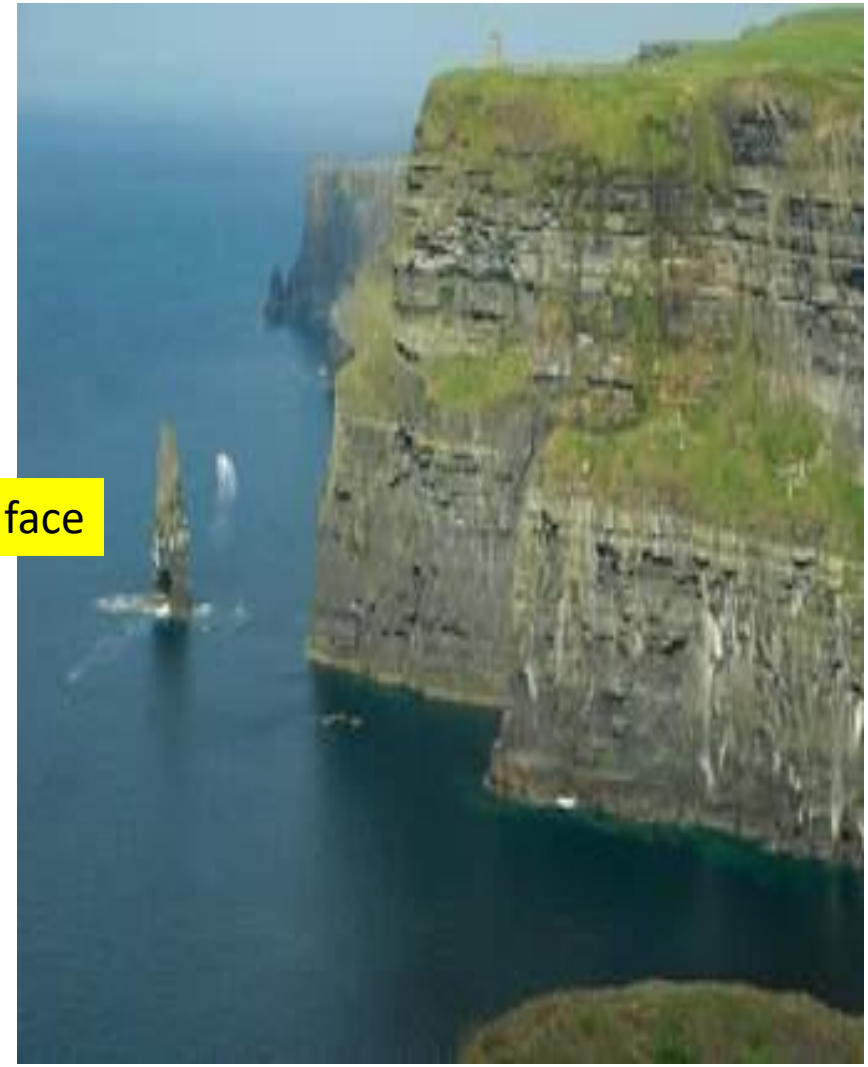
Therapy for pyoderma gangrenosum involves the use of anti-inflammatory agents, including antibiotics, corticosteroids, immunosuppressive agents, and biologic agents.

The prognosis is generally good; however, the disease can recur and residual scarring is common.

People have been banned from climbing the cliff face because it is too dangerous.

وجه الهاوية cliff face



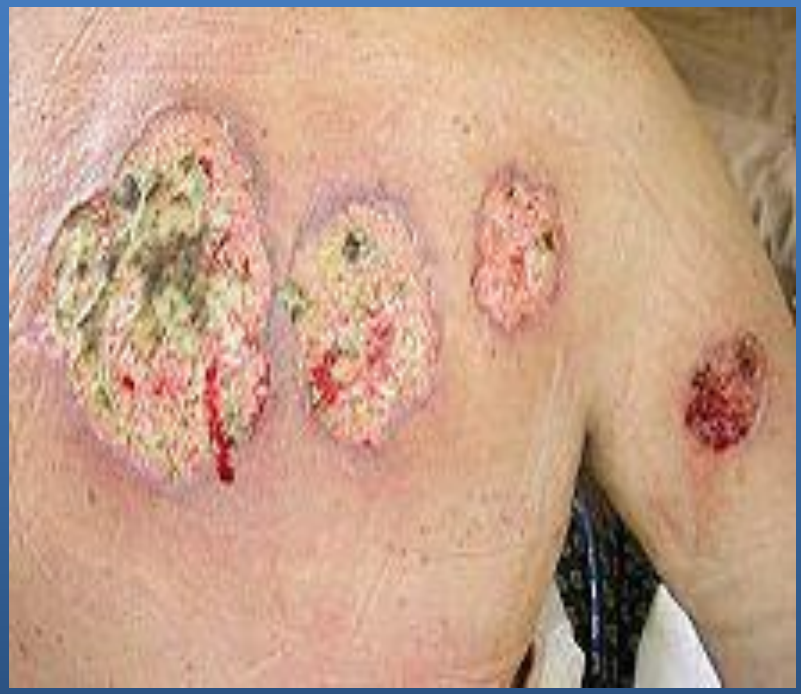


One hallmark of pyoderma gangrenosum is pathergy(30%), which is the appearance of new lesions at sites of trauma, including surgical wounds.

Diagnosis of PG is challenging owing to

its variable presentation,
clinical overlap with other conditions,
association with several systemic diseases,
and absence of defining histopathologic or laboratory findings.

Misdiagnosis and delayed diagnosis are common. It has been shown that up to 39% of patients who initially received a diagnosis of PG have an alternative diagnosis.In light of this, validated diagnostic criteria have recently been developed for ulcerative pyoderma gangrenosum.



Diagnostic criteria

In addition to a biopsy demonstrating a neutrophilic infiltrate, patients must have at least 4 minor criteria to meet diagnostic criteria.These criteria are based on histology, history, clinical examination and treatment.

1.Histology: Exclusion of infection (including histologically indicated stains and tissue cultures)

2.Pathergy (ulcer occurring at sites of trauma, with ulcer extending past area of trauma)

3.Personal history of inflammatory bowel disease or inflammatory arthritis

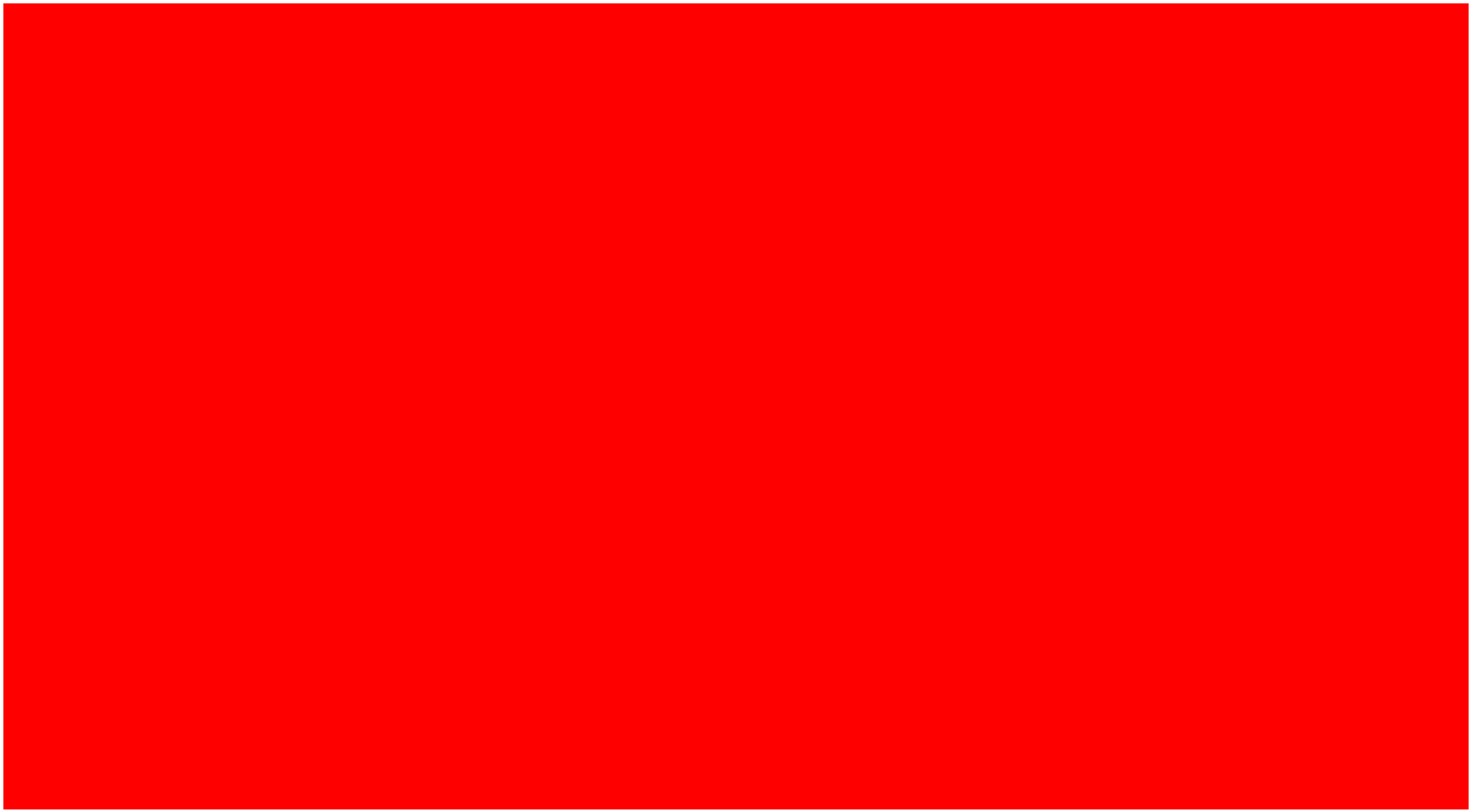
4.History of papule, pustule, or vesicle that rapidly ulcerated

5.Clinical examination (or photographic evidence) of peripheral erythema, undermining border, and tenderness at site of ulceration

6.Multiple ulcerations (at least 1 occurring on an anterior lower leg)

7.Cribriform or “wrinkled paper” scar(s) at sites of healed ulcers

8.Decrease in ulcer size within 1 mo of initiating immunosuppressive medication(s)



Associations

The following are conditions commonly associated with pyodermagangrenosum

Inflammatory bowel disease:

Ulcerative colitis

Crohn's disease

Arthritides:

Rheumatoid arthritis

Seronegative arthritis

Hematological disease:

Myelocytic leukemia

Hairy cell leukemia

Myelofibrosis

Myeloid metaplasia

Monoclonal gammopathy

Autoinflammatory disease:

Pyogenic sterile arthritis, and acne syndrome (PAPA syndrome)