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Authors

Howell, Melania
Loera, Salomé
Kirkland-Kyhn, Holly

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Cutaneous Anomalies of the Critically Ill Patient

Melania Howell, DNP, RN, AGCNS-BC, CWOCN, DAPWCA
Salomé Loera, DNP, ACCNS-AG, PCCN, CCRN-CMC
Holly Kirkland-Kyhn, PhD, FNP-C, GNP-C, CWCN, FAANP

ABSTRACT

Critically ill patients are at high risk for organ failure, including that of the integumentary system. Nurses working in intensive care are adept at performing comprehensive assessments that include the skin. Although pressure injury is a well-known complication associated with critical illness, patients may also have debilitating and life-threatening dermatoses. Conditions such as skin failure and medical adhesive–related skin damage are commonly seen in the critically ill. Infectious processes, such as Fournier gangrene, invasive candidiasis, mucormycosis, and herpetic lesions, can result in severe or superimposed critical illness and elude detection. Similarly, cutaneous manifestations of COVID-19 may develop prior to commonly recognized symptoms of infection. Nurses and providers caring for critically ill patients should be aware of common, but less widely known, skin conditions to facilitate early detection and treatment.
Key words: COVID-19, critical care, mucormycosis, skin, skin failure

Maintaining healthy, intact skin depends on controllable and uncontrollable factors, including age, illness, and medications. Critically ill patients may have numerous comorbidities or receive treatments that place them at high risk for skin injury and breakdown.[1] Underlying conditions commonly seen in intensive care, such as obesity, diabetes mellitus, and immunosuppression, may result in secondary skin infections or injury. Furthermore, hemodynamic instability, thrombotic processes, and vasopressor therapy can negatively affect macrocirculation and microcirculation, resulting in tissue and skin hypoperfusion.[2] The complex interactions among the comorbid conditions, current medical status, hypoxia, and vasopressor therapy may contribute to skin failure.[3] Prompt recognition of infectious processes such as Fournier gangrene, invasive candidiasis, and herpetic conditions can help providers avert adverse sequelae such as septic shock and multiorgan dysfunction.[4-6] Medical adhesive–related skin damage (MARSI) may cause skin reactions and wounds, resulting in patient discomfort and negatively affecting the patient's quality of life.[7] Cutaneous manifestations of COVID-19 may suggest the presence of the disease before overt symptoms occur or provide clues related to the illness's severity.[8] Nurses' recognition and understanding of skin conditions are

Melania Howell is Wound Ostomy Continence Specialist, Emanuel Medical Center, 825 Delbon Avenue, Turlock, CA 95382 (mhowell4@alumni.jh.edu).

Salomé Loera is Clinical Nurse Specialist, Adult Critical Care Services, Inova Fairfax Medical Campus, Falls Church, Virginia.

Holly Kirkland-Kyhn is Director of Wound Care, University of California, Davis Medical Center, Sacramento, California.

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essential to timely identification and treatment, thus reducing mortality and morbidity risks.[9]

Skin Failure

The skin is the body's largest organ and receives one-third of the circulating blood volume.[10] Disease processes and conditions that negatively affect organ perfusion can result in organ failure, including the skin. Skin failure is the presence of skin or tissue damage at areas that are not subjected to pressure and shearing, resulting in pressure injuries.[11] This condition has several names, including Kennedy terminal ulcers, skin changes at life's end, and Trombly-Brennan terminal tissue injuries.[12] Furthermore, reports in the literature suggest skin failure may occur on a spectrum that includes acute, chronic, and end-of-life skin.[11] Acute skin failure appears during a critical illness such as shock.[11] Chronic skin failure results from a chronic disease that contributes to hypoperfusion.[11] End-stage skin failure typically occurs during the dying process.

Risk factors associated with increased risk of skin failure development include mechanical ventilatory support lasting longer than 72 hours, peripheral arterial disease, liver failure, severe sepsis resulting in septic shock, as well as end-stage renal disease.[3,11]

Pathophysiology

Myriad conditions can lead to skin failure among critically ill patients. It is crucial to note that a mean arterial pressure less than 60 mm Hg is not the sole cause of skin failure.[3] The risk of skin failure for the patient in the intensive care unit increases exponentially when 2 or more vasopressors, usually norepinephrine and vasopressin, are administered.[3] Patients with a history of hemodynamic instability requiring vasopressor support may exhibit skin failure resembling a pressure injury, resulting in misdiagnosis.[11] The concept of angiosomes and their complex relationship with perfusion and underlying comorbid conditions may explain the development of acute skin failure.[3] Angiosomes are regions of tissue perfused by specific arteries and their branches.[3] Hypoperfusion associated with cardiogenic or septic shock may reduce blood flow to angiosomes, causing acute skin failure.[3] Using 2 or more vasopressors, especially those with α-agonist effects, may substantially reduce angiosome perfusion.[3] As the perfusion to an angiosome decreases, the arterial perforators and associated choke vessels cannot compensate, and the overlying tissues become ischemic and eventually die.[3] Death of the tissues, particularly the choke vessels, impairs the body's ability to mount an inflammatory response with its corresponding clinical indicators of leukocytosis and increased levels of inflammatory markers.[3]

Diagnosis

There are no current laboratory tests or imaging techniques to help clinicians diagnose skin failure. Examining the patient's comorbidities, current clinical condition, and history of the appearance and progression of the skin damage can suggest the diagnosis. Initial stages of skin failure may present as mottling, blisters, ulceration, and even gangrene.[12] Skin failure should be considered if skin damage appears suddenly and rapidly deteriorates to necrosis or full-thickness tissue loss.[13] Patients with skin failure may be misdiagnosed as having a deep tissue injury, or unstageable or full-thickness pressure ulcer or injury. The difference, however, may be elucidated if the area of concern does not occur over an anatomic location subjected to pressure or shearing forces. Alternatively, the sudden appearance with rapid deterioration of the skin damage in critically ill patients receiving 2 or more vasopressors and with numerous comorbidities heightens the index of suspicion for skin failure.[10,12]

Fournier Gangrene

Fournier gangrene is necrotizing fasciitis of the perineum, genitalia, and perianal region.[14] Fournier gangrene is rare, progresses rapidly, and is usually caused by a member of the β-hemolytic *Streptococcus* genus and, less frequently, by *Staphylococcus aureus*.[15] This necrotizing soft-tissue infection has a mortality rate of 40% and occurs more frequently in men, at a rate of 1.6 cases per 100 000 men.[14] Risk factors for Fournier gangrene include immunosuppression, diabetes mellitus, or localized trauma.[4] Additionally, patients with liver or renal diseases or alcohol use disorder are also susceptible to this condition.[4]

Prompt recognition of Fournier gangrene is vital to reducing the risk of complications such as septic shock. Delayed treatment of the condition can also place patients at risk

for stroke and kidney and cardiac failure, as well as lower-extremity thrombosis.[4]

Pathophysiology

The infectious process associated with Fournier gangrene begins in the hypodermis. As bacterial toxins are released, they cause tissue necrosis at levels deeper than the hypodermis until the fascial plane is penetrated.[16] Along the way, arteries and veins in the region thrombose, leading to tissue hypoxia and death with deep gangrene. As the infection progresses, nerves become hypoxic and infarct, resulting in severe pain. Despite this excruciating pain, initial physical examination findings are unremarkable.[16] As the nerve endings die, pain sensation may be reduced. However, this should not be interpreted as clinical improvement; rather, it is a progression of tissue death, and increased risk of sepsis should be suspected.[16]

Diagnosis

A patient with Fournier gangrene may present with complaints of intense pain to the genitalia, perianal area, and perineum followed by hypoesthesia.[14,16] Initially, examination of the overlying tissue is benign, but as the infection advances, overlying tissues may appear erythematous or purpuric and, at later stages, gangrenous.[14] Anaerobic pathogens can cause the affected areas to exude malodorous "dishwater" purulence.[14] Additional examination findings may include crepitus and fluctuance.[4] Imaging tools, such as magnetic resonance imaging, ultrasound, and computed tomography may reveal the presence of air in the soft tissues. A magnetic resonance image can help determine the extent of the tissue damage and is more specific to diagnosing Fournier gangrene than ultrasound or computed tomography.[4,15]

Laboratory markers can be instrumental in helping detect the disease and its severity. A complete blood cell count can reveal leukocytosis and thrombocytopenia.[17] Inflammatory markers such as C-reactive protein can help gauge the condition's severity.[17] A basic metabolic panel will help clinicians examine the patient's renal function, and blood cultures can help identify causative organisms.[17] Fournier gangrene should be considered if the patient describes pain out of proportion to clinical observations at the perineum, especially if other sources of infection have been ruled out (see Table).

Medical Adhesive–Related Skin Injury

The use of medical adhesives is pervasive in critical care to secure therapeutic devices such as enteral feeding tubes, endotracheal tubes, or vascular-access devices. Adhesive placement may cause traction on the skin, and removal of tape or securement devices may result in medical adhesive–related skin injury (MARSI).[18]

Pathophysiology

A MARSI occurs when the bond between the medical adhesive and the skin is stronger than the cellular bonds between skin layers.[26,27] As the medical adhesive is removed, the epidermis layers can be pulled apart, and the epidermis can separate from dermal layers.[28] Patients at risk for MARSI include the elderly, neonates, patients with edema, those receiving steroids, and malnourished individuals.[22,26] Medical adhesive–related skin injury falls into 3 broad categories: mechanical, dermatitis, and other.[7]

Diagnosis

A mechanical MARSI can appear as skin stripping, a skin tear, or tension-related injury such as blisters or vesicles, often mistaken as a stage 2 pressure ulcer or injury.[7,18] Dermatitis MARSI can be irritant contact dermatitis or allergic dermatitis. The allergic form can last up to 1 week. The skin will demonstrate erythema with or without vesicles and pruritus.[7] Irritant contact dermatitis typically resolves within 48 hours.[7,18] The other category is used to identify maceration and folliculitis. Macerated skin is waterlogged and may appear grayish-white and wrinkled and have a slightly boggy texture. Folliculitis occurs when the occlusive nature of the adhesive promotes moisture buildup and bacterial growth. Medical-adhesive removal can irritate the follicles of new growing hair. Bacteria can invade the region, causing inflammation, and may appear as pustular lesions at the base of the hair shaft.[7,18] Careful consideration should be given to the quantity of medical-adhesive use in at-risk populations. For example, for blood collection, teams could consider a compression wrap instead of an adhesive that could cause substantial skin injury upon its removal.

Table: Characteristics of Cutaneous Anomalies in Critical Illness

Condition	Characteristics
Skin failure	Initially presents as mottling, blisters, ulceration, or gangrene and rapidly necrosis or full-thickness tissue loss[12,13] Condition can mimic deep tissue injury or unstageable or full-thickness pressure ulcer or injury; however, it can be distinguished from pressure lesions by anatomical location, history of presentation, or both.[11,13]
Fournier gangrene	Intense pain to genitalia and perineal region, followed by hypoesthesia[14,16] Erythematous or purpuric tissue; eventually becoming gangrenous[14] Malodorous "dishwater" purulence[14]
Medical adhesive–related skin injury	Mechanical-type can appear as skin-stripping, a skin tear, or tension-related injury, such as blisters or vesicles.[7, 18] Allergic or dermatitis type Allergic erythema with or without vesicles and pruritus[7] Dermatitis usually resolves within 48 hours.[7,18] Other type Macerated skin may appear grayish-white and wrinkled with a slightly boggy texture. May appear as pustular lesions at the base of the hair shaft[7,18]
Fungal infections	Candidiasis Patient may exhibit various signs and symptoms, including low-grade fevers, malaise, septic shock, and multiorgan failure.[5] Acute disseminated candidiasis may present as a generalized rash with erythema and small blisters surrounded by purpuric papules.[19] Mucormycosis Initially may exhibit nonspecific findings such as induration with surrounding erythema[20] As the infection advances, the skin may necrose rapidly, with purpuric periwound lesions often present at the site of traumatic injury[21] In burn patients, mucormycosis may present with eschars associated with cellulitis[22]
Viral infections	Herpes simplex virus type 1—typically appears above waistline Herpes simplex virus type 2—typically distal to waist or on genitals Varicella zoster virus (causing herpes zoster or shingles) Emerges along a dermatome unilaterally, although dermatome is circular around the anal orifice and lesions may be single lesions versus bilateral[23] Patient may complain of prodromal symptoms such as nerve pain over the affected area.[24] Rash is maculopapular and lesions are surrounded by erythema. As disease progresses, skin eruptions appear as vesicles that become pustular and may proceed to ulcerate.
COVID-19 lesions	Skin conditions falling into 6 major patterns, including urticarial rashes, chilblain-like manifestations, livedo reticularis, maculopapular lesions, blister-like eruptions, and petechiae[8,24,25] First appearance may be generalized rash, especially if the pattern appears at the acral regions with or without urticaria or is herpetiform[8] Purpuric or petechial COVID-19 lesions are nonblanchable and may appear anywhere on the body, including the buttocks.[8]

Fungal Infections

Candidiasis

Invasive candidiasis is the leading cause of fungal-related death in the United States and has a mortality rate of 40% to 55% among critical care patients.[19,29,30] Although some species of *Candida* are a normal part of the skin and gut microbiome, specific risk factors can result in an invasive opportunistic infection.[31] Immunocompromised individuals are particularly at risk for invasive candidiasis.[30] Additional risk factors include treatment with antibiotic drug classes β-lactam, β-lactamase inhibitors, or steroids; sepsis with or without shock; total parenteral nutrition; severe burns; or having just undergone surgery.[5,31] Admission to a critical care unit increases a patient's risk for *Candida*-related hospital-acquired infection

due to mechanical ventilation, central venous catheters, and dialysis.[30,31] Another risk factor is COVID-19. Patients at risk for the most severe form of COVID-19 are also at risk for invasive candidiasis.[4,23] Patients with COVID-19 also are at higher risk for hospital-acquired invasive candidiasis as a result of these patients requiring antibiotics and use of invasive devices during their care.[32]

Pathophysiology. *Candida* must be presented with an opportunity to flourish, making it easier for the organism to translocate from its point of origin to other body areas.[29] Invasive candidiasis is deep-seated candidiasis secondary to candidemia or translocation of *Candida* to a sterile region.[29] Immunocompromised patients are particularly vulnerable to acute disseminated candidiasis with accompanying skin lesions.[32,33] As *Candida* grows unchecked by the body's natural microbiome flora, the pathogenic fungus will form biofilms on invasive devices and on organs of the body.[5] Eventually, patients will exhibit signs or symptoms of infection ranging from mild nonspecific manifestations to multiorgan dysfunction.[5]

Diagnosis. Patients with invasive candidiasis may exhibit various symptoms, including low-grade fevers, malaise, septic shock, and multiorgan failure.[5] Accurate diagnosis requires sterile blood and peritoneal or pleural fluid culture.[5] Invasive candidiasis may also present as an acute disseminated condition associated with a generalized rash, which is often mistaken for other dermatoses.[19] Acute disseminated candidiasis skin lesions appear erythematous, with small blisters surrounded by purpuric papules.[19] Invasive candidiasis should be a part of the differential diagnosis, mainly when the patient exhibits candidiasis at differing anatomic locations.

Mucormycosis

Mucormycosis, a type of opportunistic, invasive fungal infection, is caused by Mucorales fungi.[34] Portals of entry for this filamentous mold are airways and wounds.[34,35] The least common form of mucormycosis occurs in the gastrointestinal tract after ingesting contaminated food such as fermented milk and dried bread.[35] The disease is mainly found in immunocompromised patients, though it can also occur in immunocompetent people.[21,35] The exact prevalence and incidence of mucormycosis are difficult to ascertain because of the nonspecific manifestation of the infection.[35,36] In the United States, the estimated incidence is 0.43 cases per 1 million patients annually.[36] When detected in people in industrialized and economically advanced countries, mucormycosis often appears in those with hematologic cancers or autoimmune challenges, and those who have received solid-organ or stem cell transplants.[21] Other vulnerable groups include individuals with extensive burns or diabetes mellitus.[21,22,34,36] In immunocompetent, critically ill trauma patients, mucormycosis incidence is increasing because of wounds being inoculated with the fungus.[23]

Pathophysiology. The sporangiospores inoculate tissues after trauma, spreading to soft-tissue vasculature and reducing tissue perfusion, resulting in ischemia followed by tissue necrosis.[23] In other instances, the organism can overcome the body's phagocytic defenses because of immunocompromise.[22] Additionally, high blood glucose and acidotic states impair the body's immunity.[21] Primary mucormycosis may occur at the original site of injury. Alternatively, the sporangiospores may disseminate from the original infectious site, resulting in secondary mucormycosis.[20,21] The fungi can migrate into the vasculature, resulting in thrombi, tissue ischemia, and tissue death.[35]

Diagnosis. Cutaneous mucormycosis may occur acutely or gradually and is usually the result of direct inoculation.[21,35] Nonspecific findings of induration with surrounding erythema may be among the first signs of cutaneous mucormycosis.[20] As the infection advances, the skin may necrose rapidly, with purpuric periwound lesions often present at the site of traumatic injury.[21] In patients with burns, mucormycosis may present with eschars associated with cellulitis.[22] Nurses should be alert to new-onset skin inflammation and eschar, because biopsy specimens facilitate early diagnosis and treatment. Nurses working in trauma critical care should consider mucormycosis if the patient's wounds are recalcitrant or resistive to improvement despite the standard of care.[23]

Viral Infections

Herpetic Lesions

Herpes simplex virus (HSV) and varicella zoster virus, the latter of which also is known as herpes zoster or shingles, are members of the Herpesviridae.[22,23] Although the 2 viruses may initially have very similar presentations, because they both present as vesicular eruptions on the

skin and have a period of latency, they are different.[28] The HSV is either type 1 (HSV-1), a form that typically appears above the waistline, or type 2 (HSV-2), which occurs distal to the waist or on the genitals.[26] HSV-1 and HSV-2 are transmitted via oral or genital secretions.[37]

Unlike its HSV-1 and HSV-2 counterparts, varicella zoster virus is transmitted to the respiratory tract via an airborne transmission and often infects the elderly.[22,28] The incidence of herpes zoster, or shingles, in the United States is 1 million cases per year.[23] The risk of someone contracting herpes zoster over their lifetime ranges between 20% and 30%.[23] Herpes zoster's typical presentation is unilateral along a dermatome, although it can spread to adjacent dermatomal regions.[23] Because of the circular, versus dermatomal, configuration at the perianal region, the herpetic lesion may appear as a singular, circular bulla.[23]

Critically ill patients 80 years and older comprise approximately 10% to 20% of critical care admissions.[22] Elderly and immunocompromised persons are at the highest risk of herpes zoster infection.[23] Varicella zoster virus and other herpes viruses may cause infections in immunocompetent patients if the critical illness is protracted and severe, and the patient has higher blood lactate and C-reactive protein levels.[25] Also, COVID-19 compromises immunity, especially in severe cases during which a cytokine storm develops. The underlying disease and the body's exaggerated immune response may deplete certain cytokines, placing these patients at higher-than-average risk for shingles.[25]

Pathophysiology. The risk of herpes zoster developing starts years before the disease manifests. The varicella zoster virus is contracted earlier in life and lies dormant in the peripheral nervous system's dorsal root, autonomic, or cranial nerve ganglia.[23] As a person's immunity wanes or becomes challenged by myriad conditions, the virus can emerge and spread through the infected ganglions, eventually reaching the skin.

In cases of severely immunocompromised patients, the herpes zoster rash may provide an opportunity for a secondary streptococcal infection, resulting in necrotizing fasciitis with septic shock.[6]

Diagnosis. The classic herpes zoster infection emerges along a dermatome unilaterally, and patients may complain of prodromal symptoms such as nerve pain over the affected area.[24] It is important to bear in mind the dermatome is circular around the anal orifice and lesions may be single lesions rather than bilateral. The rash is maculopapular and lesions are surrounded by erythema. As the disease progresses, the skin eruptions appear as vesicles that become pustular and may proceed to ulcerate. Overall, the disease resolves within 4 weeks.[24] In some instances, the rash can spread to adjacent dermatomes, and in severely immunocompromised patients, opportunistic bacterial infections can complicate the clinical picture.[24]

Herpes zoster infections are diagnosed by examining the lesions, their pattern of appearance, and taking a complete history.[24] Alternatively, vesicles can be aspirated, or lesion scrapings can help confirm a diagnosis[24]; however, these options have a high rate of false-negative results.

COVID-19 Lesions

COVID-19, caused by SARS-CoV-2, is a highly transmissible respiratory virus with a constellation of systemic syndromes.[25] Increasing numbers of case reports highlight skin conditions falling into 6 major patterns: urticarial rashes, chilblain-like manifestations, livedo reticularis, maculopapular lesions, blister-like eruptions, and petechiae.[8,24,25] The lesions may appear at acral locations, palmar aspect of the hands, plantar region of the feet, and the torso.[8]

Pathophysiology. The pathophysiology of COVID-19–associated lesions may differ depending on their appearance. Chilblain-like lesions—which can be painful—are red and pruritic, may manifest in those with mild illness, and are the result of endothelial damage resulting from infection.[8] Vesicular eruptions and generalized maculopapular rashes may be observed in patients with mild to moderate COVID-19 and could be triggered by a cytokine storm.[8] In such cases, the suspected underlying pathology is endothelial damage caused by a disseminated endothelial infection.[8] Coagulopathy is a widely recognized complication of severe illness with COVID-19 and may result in stroke and limb ischemia.[38] Purpuric lesions are hypothesized to be the result of coagulopathies that result in microvascular injury.[8]

Diagnosis. In some instances, the first clue that a patient may have COVID-19 is the

appearance of a generalized rash, especially if the pattern appears at the acral regions with or without urticaria or is herpetiform.[8] Differentiating COVID-19–related lesions from other dermatoses requires close inspection of the lesions, noting the pattern of their appearance and the patient's past medical history and the history of the present illness.[8] For example, vesicular-type lesions can be misdiagnosed as a herpes infection. Unlike the latter, COVID-19 herpetiform lesions are usually located on the torso and appear similar to each other, unlike an actual herpes affliction, where the rash is polymorphic.[8] Purpuric or petechial COVID-19 lesions are nonblanchable and may appear anywhere on the body, including the buttocks.[8]

Conclusion

Cutaneous lesions of the skin of critically ill patients may occur for a variety of reasons. Early detection and assessment are pivotal to treating and mitigating the effects of these conditions. Skin examination under bright light is advised. Documentation should include the lesions' appearance, distribution, and associated symptoms, such as pain, pruritis, or other paresthesias. Furthermore, clinicians should note the medical history, including the timeline of the lesions' appearance, medications, and recent health challenges. Accurate diagnosis of skin conditions is instrumental in developing the appropriate plan of care to ensure optimal outcomes among the critically ill patient population.

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CE Evaluation Instructions

This article has been designated for CE contact hour(s). The evaluation tests your knowledge of the following objectives:

1. State at least 1 cutaneous disease or condition that may appear in the critically ill patient.
2. Identify at least 2 features of a cutaneous anomaly that may appear in the critically ill patient.
3. Describe the pathophysiologic process of at least 1 cutaneous anomaly that may appear in the critically ill patient.

Contact hour: **1.0**
Synergy CERP Category: **A**

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