

Practice Dilemmas: Conditions That Mimic Pressure Ulcers/Injuries—To Be or Not To Be?

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ABSTRACT

BACKGROUND: Pressure ulcers/injuries (PU/Is) negatively affect patients by causing pain and increasing morbidity and mortality risks. Care teams have a heightened sense of awareness of the condition and may feel confident in their ability to appropriately identify and manage PU/Is, but the potential for, and consequences of, a misdiagnosis always should be considered. **PURPOSE:** The purpose of this compendium is to describe and illustrate conditions that may mimic PU/Is. **METHODS:** Advanced practice wound care nurses were asked to identify and describe conditions that may mimic PU/Is. Permission was obtained from all patients to use their cases and photos in this article. **RESULTS:** Sixteen (16) different skin and wound presentations resulting from vascular diseases, systemic infections, trauma, cancer, autoimmune disorders, coagulopathies, and multisystem organ dysfunction were identified and described. **CONCLUSION:** A complete patient history and assessment will help prevent misidentification of the etiology of a skin lesion or wound and misdiagnosis of these lesions as PU/Is.

KEYWORDS: pressure ulcer, diagnostic errors, vascular diseases, autoimmune disease, trauma

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Practitioners in various settings encounter wounds of many etiologies and stages of healing. This collection of brief articles focuses on the misidentification of pressure ulcers/injuries (PU/Is). In the United States, an estimated 2.5 million hospitalized patients develop PIs, and

60 000 die annually.^{1,2} In 2016, full-thickness (stages 3 and 4) hospital-acquired PIs cost the US health care system \$26.8 billion.^{1–3} This expenditure breaks down to approximately \$10,708 per patient.³ Factors contributing to these expenses include increased length of stay, care re-

lated to the hospital-acquired PI, and reduced reimbursement by the Centers for Medicare & Medicaid Services (CMS).^{1–4}

Due to financial and potential legal ramifications associated with PU/I recognition and management, care teams have a heightened awareness of the

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condition. Although this knowledge is instrumental to the prompt identification of PU/I, a potential pitfall is the missed opportunity to distinguish PU/I from other insidious conditions.

The true incidence of PU/I misdiagnosis is unknown. Unfortunately, once a clinician documents a diagnosis in the medical record, that diagnosis is often sustained throughout the chart, regardless of its accuracy. Therefore, correctly identifying wounds and ascribing an accurate underlying etiology is imperative. Clinicians need to be thoughtful before designating a wound as a PU/I, especially when the clinical picture does not coincide with pressure as the cause of the wound. Familiarity with common and rare conditions that can mimic PU/I will reduce the risk of inaccurate diagnosis, resulting in improved patient outcomes and reduced costs.³

The list of all conditions that could potentially mimic PU/I is exhaustive and beyond the scope of this body of work.⁵ However, this collection of articles describes skin conditions that may be encountered in routine clinical practice but may be misdiagnosed and inappropriately managed as PU/Is (Table). Contributors present wounds that mimic PU/I through the lens of etiology, such as circulatory, infectious, autoimmune, traumatic, coagulopathic, malignant, and multisystem organ dysfunction. It is hoped that this compendium will be a timely, relevant, and useful guide to differentiate PU/Is from other conditions.

EVALUATING WOUNDS

As detailed in this compendium, a wide variety of etiologies can produce wounds in the vicinity of pressure- or shearing-prone anatomical locations. Upon receiving a request to evaluate a PU/I, clinicians should start with a detailed chart review to identify all potential causes. Imaging studies and laboratory test results can provide valuable information. Patients should be asked open-ended questions such as the following:

- How did this start?
- How long has this been a problem?

Table. Conditions That May Mimic Pressure Ulcers/Injuries

Conditions	Distinguishing features	Features mimicking PU/Is	Diagnostics
Vasculitic conditions and vascular insufficiency disorders			
Vascular ulcer on the lateral malleolus	Poorly healing wound with signs and symptoms of localized hypoperfusion Ulceration with delayed healing	Lateral malleolus wound that may appear to be secondary to minor PU/I from ill-fitting footwear or orthopedic devices May be mistaken for stage 3, 4, unstageable, or DTI	Duplex ultrasonography of the peroneal and other arteries Catheter angiography Ankle-brachial index Radiographs to rule out osteomyelitis older than 21 days MRI or bone scans will reveal acute osteomyelitis
Lower extremity arterial disease	Painful, intact areas of shiny, red, or purple discoloration that progress to ulceration with slough and necrosis Lower back, hip, buttock, or thigh pain	In the initial presentation, areas may appear purple or red over intact skin, similar to DTI As the condition progresses, may mimic unstageable PU/I	Vascular diagnostic imaging including computerized tomodensitometry angiography and magnetic resonance angiography History and physical examination
Calciophylaxis	Indurated, painful, firm plaques or ulcers that may have a purpuric appearance and evolve into necrotic lesions with eschar Commonly found on the back, abdomen, and upper legs Superimposed infection may also be present	Initial presentation may be confused for a DTI Necrotic lesions and regions of black eschar may present similarly to infected PIs or DTIs, particularly when calciophylaxis nodules arise in areas exposed to pressure	Detailed history, including review for end-stage renal disease and renal replacement therapy, as well as physical examination Punch biopsy from the lesion margins

Table continues

Abbreviations: PU/I, pressure ulcer/injury; DTI, deep tissue injury; MRI, magnetic resonance imaging; COVID-19, coronavirus disease-19; SIRS, systemic inflammatory response syndrome; HIV, human immunodeficiency virus; CT, computed tomography; VZV, varicella zoster virus; CEA, cultured epithelial autographs; TBSA, total body surface area.

Table. Conditions That May Mimic Pressure Ulcers/Injuries (Continued)

Conditions	Distinguishing features	Features mimicking PU/Is	Diagnostics
Autoimmune diseases			
Bullous pemphigoid	<p>Affects persons age 70 years and older</p> <p>Pruritus and blister formation are hallmarks</p> <p>Ecematous plaques, papular, or cutaneous lesions may also develop</p> <p>Lesions usually have symmetrical distribution</p>	<p>Bullae with serous or hemorrhagic drainage may mimic a stage 2 PU/I or DTI</p>	<p>Detailed history and physical examination</p> <p>Punch biopsy</p>
Infections			
Rectocutaneous fistula	<p>An atypical tract between the rectum and the skin within the rectal vault</p> <p>The condition arises as a complication of disease, direct or indirect trauma, or a surgical procedure</p> <p>Skin is initially intact, although the patient may complain of pain relieved by defecation</p> <p>As the condition progresses, the area will present with a full-thickness wound or painful perirectal abscess</p>	<p>Perirectal abscess often presents with intact skin and redness similar to stage 1 PU/I</p> <p>Later manifestations may appear with yellow sloughy necrosis mimicking unstageable PU/I</p>	<p>Review of relevant history and clinical examination</p> <p>Computed tomography</p> <p>Enema studies</p>

Table continues

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- What treatments have you received to address the issue? Did they work?
- From your perspective, does anything help or harm the wound?

As the wound is examined, the condition of the dressing and any odors should be noted. During the physical examination, clinicians should try to connect the information gleaned from the chart and reported by the patient to what they are seeing. Characteristics such as location, size, level of tissue damage, and drainage should be noted, and close attention paid to the wound margins and surrounding skin. The remainder of the extremity, its contralateral counterpart, the torso, and the trunk should also be examined. Imaging studies, laboratory test results, and other diagnoses/medical conditions will help to confirm suspected diagnoses and guide treatment. Through a systematic evaluative process, the puzzle pieces will fit together to form a complete picture to answer the question, “To Be or Not To Be?” ■

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Table. Conditions That May Mimic Pressure Ulcers/Injuries (Continued)

Conditions	Distinguishing features	Features mimicking PU/Is	Diagnostics
Infections			
Cutaneous manifestations of COVID-19	Varying appearances ranging from clear to hemorrhagic blisters, erythematous lesions, and purple discolorations at any anatomical location Areas most prone to developing the lesions include acral locations and extremities, although they may also appear in the vicinity of pressure-prone regions	COVID-19–associated lesions may mimic stage 1 or 2 PU/Is or DTIs	Detailed patient history and assessment with high index of suspicion for onset, evolution, and resolution, usually without scar formation Evaluation for COVID-19 diagnosis via laboratory testing
Pyomyositis	The condition results from the infection of skeletal muscle tissue Progresses in 3 distinct phases (invasive, seeding of infection within skeletal muscle groups, and SIRS) Initial presenting symptoms are vague and easily missed The latter phase carries a significant risk for sepsis and multisystem organ failure	Earlier symptoms of a woody edema with erythema can be mistaken for stage 1 PU/I Later manifestations, including necrosis, can lead to a diagnosis of an unstageable PU/I	A detailed history and physical examination, with particular attention to reports of vigorous exercise, intravenous drug use, blunt force trauma, HIV, immunodeficiency, malnutrition, and diabetes MRI is the gold standard, but CT or ultrasound may also be considered Wound cultures obtained intraoperatively to identify causative agent
Necrotizing soft tissue infections	Underlying soft tissue infection with gangrenous necrosis resulting in subcutaneous emphysema and frothy purulence	Black eschar with underlying necrosis may present similarly to an unstageable PU/I	History and physical examination Presence of fat-stranding on CT
Herpes zoster	Vesicular lesions or ulcerations associated with herpes zoster May display extensive blistering and necrosis	Lesions on the buttocks or perineal area may be mistaken for PU/I associated with prolonged exposure to moisture	Physical assessment with particular attention paid to lesions distributed along dermatomes, although disseminated herpes zoster should also be considered Serum VZV immunoglobulin titers Histological analysis with direct fluorescence antigen staining

Table continues

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Table. Conditions That May Mimic Pressure Ulcers/Injuries (Continued)

Conditions	Distinguishing features	Features mimicking PU/Is	Diagnostics
Cancer			
Marjolin’s ulcer	<p>Aggressive skin neoplasm arising from scar tissue</p> <p>The area is recalcitrant with reopening weeks, months, or years after healing from the initial insult</p> <p>May manifest as chronic ulcerations with a cauliflower-like friable surface with epibole at the margins</p>	Ulceration with incomplete healing may appear to be associated with PU/I, especially when occurring in a region subjected to friction and shearing forces	<p>Biopsy</p> <p>Positron emission tomography scan to evaluate for metastasis</p>
Coagulopathies			
Cold agglutinin cutaneous manifestation	Signs and symptoms consistent with cutaneous ischemia associated with cold exposure, including acrocyanosis	Purpura and ecchymosis without coinciding trauma or hematologic abnormalities or dry necrosis, including black eschar as seen with severe frostbite, may resemble a DTI or unstageable PU/I despite lack of pressure to the area	<p>Laboratory tests including cold agglutinin test</p> <p>Vascular Doppler studies including flowmetry and ultrasound</p> <p>Capillary microscopy</p>
Warfarin-induced skin necrosis	<p>Erythematous lesion with progression to a painful, full-thickness wound and skin necrosis</p> <p>Commonly occurs in areas with significant amounts of adipose tissue</p> <p>History of recent or past warfarin therapy</p> <p>More likely in patients given high doses of warfarin initially</p>	<p>Initial lesions present similarly to stage 1 PU/I</p> <p>As the condition progresses to purple discoloration, may mimic a DTI</p> <p>Evolution may resemble stage 3 PU/I with necrosis; this misdiagnosis is more likely if there is a risk of prolonged pressure in an area, such as the buttocks</p>	<p>History and clinical presentation</p> <p>Skin biopsy</p>
Thrombocytopenic purpura	Hemorrhagic lesions ranging from pinpoint petechiae to widespread nonblanchable ecchymosis	Purpuric lesions may appear to be consistent with DTI due to their propensity to situate in areas of dependency, such as the buttocks	<p>A thorough history, including investigation for hematological conditions</p> <p>Physical assessment and evaluation of the pattern of distribution of purpura</p> <p>Complete blood count</p>

Table continues

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Table. Conditions That May Mimic Pressure Ulcers/Injuries (Continued)

Conditions	Distinguishing features	Features mimicking PU/Is	Diagnostics
Traumas			
Morel-Lavallée lesions	<p>Closed degloving injury resulting from blunt force trauma with shearing</p> <p>Commonly appears in the greater trochanteric region</p> <p>Immediate or delayed pain and swelling of the affected area</p>	<p>Purpuric discoloration with evidence of compromised underlying tissue closely mimics a DTI</p>	<p>MRI</p>
Burn treated with cultured epithelial autograft	<p>Described by depths of tissue damage ranging from partial-thickness to full-thickness tissue damage</p> <p>Presentation ranges in color from bright to deep red and black; may also manifest as blisters or bullae</p> <p>CEA used for full-thickness burns covering ≥ 50% TBSA may fail, especially over pressure-prone areas</p>	<p>Failure of CEA used to treat full-thickness burns may mimic a stage 3 or 4 PU/I</p>	<p>A detailed history and physical examination</p> <p>Serial wound photography of all areas affected by the burn, starting on admission</p>
Multisystem organ failure			
Skin changes at life's end	<p>A complication of prolonged hypoperfusion resulting in skin changes</p> <p>Usually seen in patients with multisystem organ dysfunction or failure</p> <p>May occur at any anatomical location but commonly present in acral regions or sacrococcygeal area</p>	<p>Early manifestations may mimic stage 1 PU/I</p> <p>Progression of the condition closely mimics a DTI</p>	<p>History of or current multisystem organ dysfunction</p> <p>Hemodynamic instability with hypoxia</p> <p>Evaluation of clinical progression, including sudden onset and rapid deterioration of areas of concern</p>

Abbreviations: PU/I, pressure ulcer/injury; DTI, deep tissue injury; MRI, magnetic resonance imaging; COVID-19, coronavirus disease-19; SIRS, systemic inflammatory response syndrome; HIV, human immunodeficiency virus; CT, computed tomography; VZV, varicella zoster virus; CEA, cultured epithelial autographs; TBSA, total body surface area.

Vasculitic Conditions and Vascular Insufficiency Disorders: Vascular Ulcer on the Lateral Malleolus

Anthony Tickner, DPM, FACCWS, FRCPS

Arterial ulcers are a result of inadequate tissue perfusion, which leads to ischemia and tissue necrosis. Peripheral arterial disease (PAD) affects 10% to 20% of the adult population in the United States.¹ Approximately 1% of the adult population and 3.6% of people older than age 65 will develop an arterial ulcer.² There is an increased incidence of arterial ulcers in patients who smoke, are obese, have diabetes, have hypertension, or are sedentary.³ Arterial ulcers often have a “punched out” appearance with defined edges and tend to be painful. Patients exhibiting wounds with these characteristics who also have shiny, hairless skin; reduced capillary refill; and loss of pedal pulses must be screened for PAD.⁴ With increased age, lower extremity circulation may be decreased due to arterial insufficiency, resulting in arterial ulceration. The soft tissue around the ankle is thin and may break down more easily with a minor injury, increasing the risk of arterial ulcers within this region. Soft tissue around the lateral malleolus is weaker because of the high pressures generated by the external rotation of the hip.² When an open wound develops in the lateral malleolus, it can be difficult to heal.

Assessment. A foot and ankle examination begins with a general inspection. Patients should remove shoes and socks completely so that the practitioner can examine the bilateral feet thoroughly. The practitioner should inspect the feet for signs of ischemia or wounds at the tips of the toes¹ and then assess the temperature and pulses of the feet bilaterally. Pallor upon elevation of the extremity and dependent rubor should raise the index of suspicion for PAD.¹ Posterior tibial and dorsalis pedis pulses should be evaluated

by palpation and Doppler ultrasound as well as by capillary refill time. The practitioner should assess for any points of tenderness over the lateral malleolus.^{1,3}

Diagnosis. Clinical evaluation can begin with noninvasive techniques in outpatient settings. The ankle-brachial index (ABI) is the most widely used noninvasive test. The test is performed by dividing the ankle pressure by the brachial systolic pressure. One of the drawbacks of using an ABI test is that the results may be falsely elevated due to calcified leg arteries in patients with diabetes or on dialysis. The toe-brachial index is obtained similarly to the ABI, except the blood pressure of the great toe is used versus the ankle pressure. However, this test lacks specificity, and a low toe-brachial index vaguely indicates ischemia somewhere within the leg arteries.⁵ Arterial duplex ultrasound assesses the velocity of blood flow in healthy and diseased vessels. A normal peripheral arterial waveform is triphasic; mild disease results in biphasic waveforms, and monophasic waveforms indicate increasing severity of the narrowing of the vessel.⁶ Transcutaneous oxygen measurement can assess the microcirculation of the skin. However, this method cannot detect calcified arteries.

Catheter angiography is the gold standard for diagnosing PAD and is frequently performed before endovascular intervention due to the invasive nature.⁷ The angiosomes theory can help diagnose lateral malleolar ulcers. The theory described by Taylor and Pan⁸ suggested that angiosomes are 3-dimensional blocks of tissue fed by source arteries. The peroneal artery angiosomes supply the lateral ankle and lateral heel area. Therefore, abnormal results of vascular studies of the peroneal artery may suggest PAD as the underlying etiology of a lateral malleo-



FIGURE 1. Vascular ulcer at the malleolus

lus ulcer. Finally, radiographs of the area should be taken to assess for any underlying osteomyelitis or bony changes.

Case study. An 86-year-old woman presented with an ulceration on her right lateral malleolus. The patient reported that she was diagnosed with a stage 3 PU/I. The wound was circular, measuring approximately 3 × 3 × 0.5 cm, with a pink and yellow, minimally moist base (Figure 1). The patient stated that it had been there for about 2 months but denied any pressure to the area. The patient was ambulatory and had a history of anxiety, type 1 diabetes, hypertension, osteoporosis, and depression. The most recent A1C value was 6.3%. Right lower extremity ABI was 0.97, and the results of an arterial duplex ultrasound of the right lower extremity showed occlusion of the posterior tibial artery, biphasic flow through the peroneal artery, and biphasic anterior tibial artery with 3% to 50% stenosis. A diagnosis of an arterial ulcer was made, and a comprehensive treatment plan was enacted.

Topical treatments included collagen and Hydrofiber dressing (ConvaTec, Bridgewater Township, NJ). Practitioners can also use a variety of foams as either primary or secondary dressings. The patient was instructed to avoid any

pressure on the area, and pressure-reducing devices, including foam donuts and offloading boots, were used whenever the patient was in bed. After 3 months of comprehensive treatment, including glycemic control, nutritional optimization, and surgical placement of a stent, complete epithelialization of the wound was noted.

The location and appearance of the wound may lead to a misdiagnosis of a pressure ulcer/injury. Examining the bilateral lower extremities as well as evaluating the medical and wound history can help clinicians include PAD in the list of differential diagnoses. ■

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Lower Extremity Arterial Disease

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Peripheral arterial disease (PAD) is present in up to 10% of the US population and results in chronic ulcers with tissue loss in approximately 100 000 individuals annually.^{1,2} In the presence of severe arterial disease, care teams may misdiagnose advanced skin ulcers as pressure ulcers/injuries (PU/Is).³⁻⁵ A form of PAD is known as lower extremity arterial disease (LEAD), which is a prevalent, systemic disease affecting the normal structure and function of blood vessels and mainly caused by atherosclerosis.¹ Clinicians may suspect a distal or proximal lesion causing LEAD, depending on where symptoms of claudication exist. Lesions of the common and external arteries, as well as the femoropopliteal artery, result in distal LEAD, which is commonly exhibited by calf pain.⁶ Common iliac or isolated internal iliac arterial lesions cause proximal

LEAD, and symptoms include lower back, hip, buttock, or thigh pain.⁶ Nonmodifiable risk factors for LEAD include age, sex, and genetic predisposition, whereas modifiable risk factors include smoking, hypertension, type 2 diabetes mellitus, and dyslipidemia.⁶

Assessment. Patients with LEAD usually present with an area of injury that is painful.¹ The skin is intact, but the site may appear shiny, deep red, or purple.¹⁻³ Eventually, ulceration develops over the purplish discoloration. The wound may appear with ischemic tissue, slough, and necrosis.⁵ In its initial phases, the ulcer may mimic a deep tissue injury.⁷

Diagnosis. Diagnosing LEAD is accomplished by considering the patient's history and performing a physical examination and vascular diagnostic imaging. Patients often report symptoms of claudication, defined as pain with am-



FIGURE 2. Vascular compromise of the iliac arteries manifesting as full-thickness lesions on the buttock

bulation relieved after 10 minutes of rest.⁶ The physical examination should focus on eliminating hip osteoarthritis and spinal stenosis as the cause of the lower extremity discomfort.⁶ Ankle-brachial indices can help but are not definitive for identifying the lesion's location.⁶ Computerized tomodensitometry angiography and magnetic resonance angiography are more definitive for diagnosing the condition and detecting the location of the arterial occlusion.¹

Case study. A 61-year-old man was admitted with chronic wounds and diagnosed with stage 4 PU/I on the left buttock. Medical history included type 2 diabetes mellitus, smoking, hypertension, and dyslipidemia. Surgical history included aortoiliac bypass surgery and bilateral above-knee amputations. The wound was present for more than 1 year. The left buttock ulcer measured 5 × 5 × 1 cm and was light red with nongranular tissue exuding a moderate amount of serous drainage (Figure 2). Wound assessment revealed epibole at the wound margin.

Unlike patients with PU/I who may report no to minimal pain, this patient reported pain to the left buttock as 8 on

a scale of 10. The wound did not display evidence of infection. Despite PU/I prevention and topical therapy, the wound failed to heal. The wound did not meet the classic criteria of PU/I location over a bony prominence. Further assessment was unable to reveal pressure or shearing forces contributing to the ulcerations.

Finally, the complicated healing trajectory prompted the clinicians to search for other underlying etiologies. A vascular surgery consultation was obtained for evaluation, differential diagnosis, and treatment options. A diagnosis of occlusion of the internal iliac arteries was established, and the diagnosis of PU/I was changed to reflect the true underlying etiology. Successful revascularization surgery with topical care resulted in the eventual healing of the wound. ■

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Calciphylaxis

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Calciphylaxis is a cutaneous ischemic infarct caused by occlusion of blood vessels in the subcutaneous fat and dermis. Persons with this condition report severe pain and may be more susceptible to infection at the wound site. Although rare, calciphylaxis is typically devastating and debilitating, with an annual mortality rate of 40% to 80%.^{1,2} Calciphylaxis that develops in patients with end-stage renal disease is classified as uremic calciphylaxis. Estimates for the annual incidence of uremic calciphylaxis in the United States is 0.35%.^{1,2} The actual incidence is probably higher than reported because this condition is often misdiagnosed. Calciphylaxis in patients with preserved renal function is known as nonuremic calciphylaxis. Its

incidence is unknown but likely much lower than that of uremic calciphylaxis. As of 2016, only 116 cases were reported in the literature.^{1,2}

Calciphylaxis is an occlusive disease affecting the cutaneous blood vessels. Typical disease progression involves narrowing of the lumens by calcification within the media layer of vessel walls, proliferation of endothelial cells, and fibrosis underneath the intima.¹ Late effects of the condition are exhibited as ischemic injuries that develop after thrombosis development in the vessel lumen.¹

Assessment. Identifying calciphylaxis requires a detailed history and a physical examination. Calciphylaxis manifests as painful, firm nodules that may initially

appear purple and are often pruritic.² Physical examination in the initial stages of the disease may result in a diagnosis of suspected deep tissue injury.³ As the condition evolves, the lesions may become necrotic with extensive regions of black, leathery eschars.² A superimposed infection may develop in vulnerable individuals. The necrotic appearance may lead to the misdiagnosis of an unstageable pressure ulcer/injury. These conditions can be differentiated based on patient history, which often includes a long history of renal failure with renal replacement therapy.³ In addition, during the physical examination, clinicians must consider the lesions' anatomical location and whether the areas were



FIGURE 3. Calciphylaxis lesions on posterior torso

subjected to pressure or shearing forces.

Diagnosis. A diagnosis of calciphylaxis can be made on clinical grounds when indurated tender plaques or ulcers develop on the abdomen and/or legs of patients with end-stage renal disease. It is recommended that a skin biopsy be performed, however, because calciphylaxis

can mimic other diseases, especially in its early stages.^{1,2,4} Skin biopsy is also recommended to assist in the diagnosis of nonuremic calciphylaxis. A single 6-mm punch tool is inserted 8 mm into the defect to obtain the deep subcutaneous fat. The biopsy specimen should be obtained at the lesion's margin. Care must be taken to avoid the center of the lesion or necrotic areas where nonspecific necrotic tissue is more likely to be found. Excisional biopsy should be avoided due to the potential for ulceration, necrosis, and bleeding.¹

Case study. The patient was a 60-year-old man with end-stage liver disease, monoclonal gammopathy of undetermined significance, sarcoidosis with multiorgan involvement, stage 3 chronic kidney disease, benign prostatic hyperplasia, deep vein thrombosis, hypothyroidism, and lymph node granulomas. He had recently undergone a transjugular intrahepatic portosystemic shunt procedure at another institution, and his hospital course was complicated by cirrhotic portal hypertension, persistent ascites, recurrent *Clostridium difficile* infection, and non-uremic calciphylaxis. Following admission to the author's institution, necrotic wounds were noted on the back

and thighs (Figure 3). Key assessment findings included exquisitely painful necrotic wounds surrounded by palpable subcutaneous nodules. The wounds were not located in areas subject to pressure, friction, or shearing forces, but were initially identified as unstageable pressure injuries. However, after a thorough chart review, patient interview, and examination, the documentation was updated to reflect that these wounds were non-uremic calciphylaxis lesions. ■

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Autoimmune Diseases: Bullous Pemphigoid

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Bullous pemphigoid (BP) is the most common subepidermal blistering autoimmune disease, and it commonly affects individuals age 70 years and older.¹⁻³ Annual occurrence is 6 to 13 new cases per million older individuals in the United States and 12 to 13 per million senior persons in Central Europe.¹ BP is a severe skin disorder with very high morbidity and mortality rates.¹

In addition to advanced age, systemic drugs such as diuretics, nonsteroidal anti-inflammatory drugs, some antibiotics, and tumor necrosis factor- α inhibitors may predispose some

individuals to have BP-like skin eruptions.^{1,4,5} Pruritus and blister formation are significant hallmarks of this disorder.^{1,6}

BP has 2 primary pathophysiologies, immunologic and inflammatory. Blisters develop when immunoglobulin G antibodies bind to the skin's basement membrane and activate complement and inflammatory components, leading to proteolytic enzyme release. This process destroys hemidesmosome proteins and forms blisters.^{1,4,7}

Assessment. BP may present initially as a nonbullous, pruritic rash.

The disorder may also manifest as eczematous plaques, papular, or urticarial cutaneous lesions. As a result, rendering an accurate diagnosis may be a challenge to clinicians.^{4,6} As the condition progresses to the bullous phase, patients exhibit tense bullae measuring from 1 to 4 cm in diameter that may appear serous or hemorrhagic, mimicking a stage 2 or deep tissue injury. Surrounding tissues may appear normal or erythematous. Bullae or blisters can last several days before evolving into erosions and crusts that heal without scarring. Lesions usually

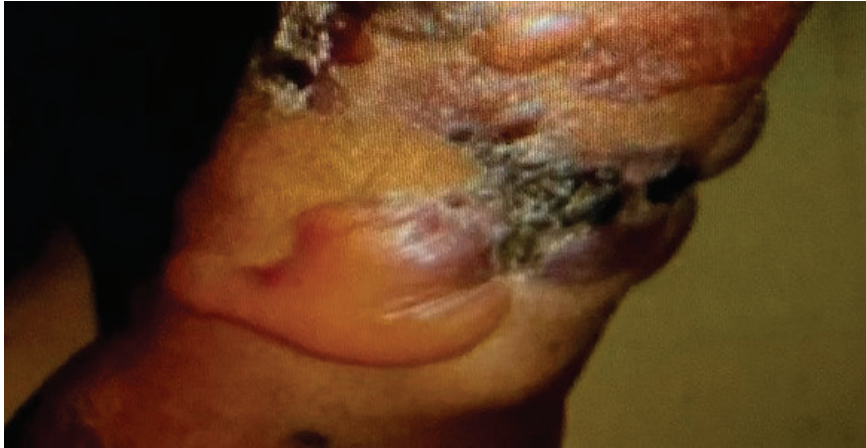


FIGURE 4. Bullous pemphigoid on lower extremity

have symmetric distribution and commonly appear on the trunk, abdomen, axillae, groin, extremities, and flexor surfaces. BP rarely involves oral, ocular, or other mucosae.^{1,4,6}

Diagnosis. Practitioners often arrive at a diagnosis of BP through evaluating the clinical manifestations of the disorder and taking a detailed patient history.⁶ A definitive diagnosis may be made by obtaining a 24- to 48-hour-old blister that is shaved in its entirety or a 3- to 4-mm punch biopsy at the edge of the lesion.⁷

Case study. A medically complex 90-year-old woman presented for vomiting caused by a small bowel obstruction. She was recently diagnosed with BP of

the upper and lower extremities, treated with 2 differing topical corticosteroids before admission. Skin assessment revealed scattered dry, stable, and scabbed lesions on the upper and lower extremities in the vicinity of pressure-prone areas (Figure 4).

The patient's previous diagnosis of BP raised the index of suspicion for BP being the etiology of the current lesions rather than a pressure ulcer/injury. Physical examination revealed similar lesions on the bilateral upper and lower extremities with symmetrical distribution, which provided further confirmation. In this patient, the irregular shape of the resolving blister was different than the expected ap-

pearance of a partial-thickness pressure ulcer/injury, which typically has a more circular appearance. Results of a punch biopsy confirmed the diagnosis of BP. ■

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Infections: Rectocutaneous Fistula

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A fistula is an atypical tract between 2 epithelial surfaces. The leading cause of fistula formation is the loss of organ wall integrity due to underlying disease, surgery, or trauma.¹ This usually leads to penetration to an adjacent organ or surface.¹ Therefore, fistula is considered a disease complication resulting from surgery or trauma, rather than a disease itself.¹

Rectocutaneous fistula is a passage between the rectum and skin.¹ Given its location and presentation as a full-thickness wound, clinicians may confuse a rectocutaneous fistula with a pressure ulcer/injury (PU/I).² As a result, it may not be treated correctly. The incidence of fistulas depends on many factors, including underlying disease prevalence, quality of health care

and surgical practices, trauma events, and radiation therapy.^{1,3} The incidence of rectocutaneous fistula from a rectal abscess is about 26% to 38%.³ Rectal fistulas are most commonly related to underlying conditions, including anal abscess, trauma, cancer, radiation therapy, surgical procedures, autoimmune disease, congenital defect, or iatrogenic cause.^{1,4}



FIGURE 5. Rectocutaneous fistula with abscess

Assessment. Initial presentation of a perirectal abscess secondary to rectocutaneous fistula is typically painful. Patients describe a pulsatile, achy, dull pain around the anus.² Exacerbating factors include sitting and needing to defecate. Once defecation occurs, the patient may describe a reduction in pain and discomfort.² The overlying skin is often intact, but the area of concern may appear raised and erythematous. The wound can quickly become necrotic, usually presenting with a yellow slough mimicking an unstageable PU/I.¹⁻³

Diagnosis. Diagnosis of rectocutaneous fistula is primarily through clinical

examination, usually a rectal examination.² However, further investigation using computed tomography or enema studies may be employed to determine the extent of the fistula.⁵

Case study. A 59-year-old man with type 2 diabetes mellitus and hypertension was admitted to a level 1 trauma center following an auto versus pedestrian accident with loss of consciousness. The patient presented with a right gluteal hematoma, pelvic fracture, and left femoral neck fracture. Subsequently, the patient underwent multiple surgical procedures. After his condition was stabilized, he was able to ambulate with a

walker. However, after 2 weeks of hospitalization, a painful full-thickness coccyx wound, measuring 2 × 2 cm with moist, yellow slough in the wound bed and periwound erythema, developed (Figure 5). The wound was initially classified as an unstageable PU/I and reported as an adverse event. Despite an extensive wound treatment regimen, the patient reported increased pain and tenderness in the buttocks. Examination and imaging showed that the coccyx wound had internal tracking distal to the rectum. The diagnosis of the wound was changed to a rectal abscess with an underlying etiology of rectocutaneous fistula. ■

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Herpes Zoster

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Clinicians often misdiagnose perianal lesions, perirectal lesions, or lesions appearing on the fleshy buttocks as moisture-associated skin damage, incontinence-associated dermatitis, or pressure ulcers/injuries. When evaluating a patient with vesicular conditions or ulceration, the potential of a viral cause such as herpes zoster (shingles) should be considered. The incidence of

varicella zoster viral (VZV) ulceration is unknown because of poor recognition and confusion with other conditions that result in blisters and superficial tissue ulceration.^{1,2}

Herpes zoster is the causative agent for chickenpox and shingles, and the viral infection is located in the epidermis.¹⁻³ The condition typically appears unilaterally along a dermatome

or may involve numerous adjacent dermatomes.⁴ Herpes zoster may appear more disseminated, indicating a spread to a nonadjacent dermatome and is referred to as zoster duplex, unilateralis, or bilateralis.³ In some patients, eruptions may be extensive, with an inflammatory response displaying extensive blistering, necrosis, and even secondary infections.⁵



FIGURE 6. Herpes zoster lesions at sacrococcygeal region

Primary VZV, usually exhibited as chickenpox in children, starts when a patient transmits the virus, through respiratory secretions, to a vulnerable patient.^{2,5} The individual acquires the disease via the oropharynx, which provides direct access to the tonsils, where the virus replicates.¹ VZV targets T cells, which distribute it relatively efficiently to the skin and, in some cases, other organ systems.¹ Patients exhibiting the primary form of the disease present with pruritus, vesicular lesions, or eruptions.^{2,5} Admittedly distressing, the primary infection is self-limiting. Despite the resolution of the blisters, the virus becomes quiescent in the sensory neurons. Over time, however, the host's immunity fades, allowing the virus to reemerge as zoster, commonly known as shingles.^{2,5} Compromised immunity caused by aging, cancer therapies, human immunodeficiency virus, fatigue, stress, and emotional trauma can individually or synergistically trigger viral reactivation.² Medications increasing the risk of zoster include those impacting the host's immunity, such as disease-modifying antirheumatic drugs, tofacitinib, and oral corticosteroids. The prodromal phase

includes complaints of a burning pain, followed by an outbreak of blisters that may be unilateral, within one dermatome, or lesions along adjacent dermatomes.^{2,4}

Assessment. Clinicians may diagnose VZV either by clinical symptoms, objective testing, or both.³ The clinical appearance of VZV includes vesicles, pustules on an erythematous base, and pain. The history reveals rapidly occurring lesions. The host's immunity and comorbidities can cause an atypical appearance, including extensive ulcerations with inflammation, blisters, necrosis, and secondary infections.^{3,5}

Diagnosis. Wound culture results are often false-negatives. A viral polymerase chain reaction with the isolation of DNA may be ordered.^{3,5} Serologic testing may reveal elevated serum VZV immunoglobulin titers, often seen with herpes zoster. The primary form of the infection will exhibit high immunoglobulin M levels. Histologically there is acute inflammation with necrosis, while direct fluorescence antigen staining will be positive.^{3,5} Prompt recognition, diagnosis, and management can improve patient outcomes by reducing postherpetic neuralgia and healing time.

Case study. A healthy 55-year-old woman with Crohn's disease resulting in a long-standing ileostomy presented to the hospital with a diagnosis of pneumonia. The patient reported that her primary care team treated a labial blister 2 weeks prior. The labial blister had now progressed to ulceration. The patient also "noticed a couple of blisters under pouch adhesive one day," and the next day the patient exhibited extensive ulcerations at the peristomal plane. Lesions were also discovered at the sacrococcygeum. The region was purpuric with several raised areas that were painful, with yellow moist bases.

The area was initially diagnosed as a suspected deep tissue injury in evolution (Figure 6). These ulcerations were differentiated from moisture-associated skin damage or pressure ulcer/injury due to the patient's history. The lesions at the peristomal plane were distinguished from contact dermatitis mainly due to their severity. Also, the area was uncharacteristically painful, a feature not observed with an allergic reaction. The distribution of the lesions at the sacrococcygeal and other anatomical locations provided additional clues of a zoster etiology. The patient received 1 week of intravenous antiviral therapy. Healing quickly progressed, and there was a prompt decrease in erythema, pain, and necrosis. Finally, the ulcerations exhibited epithelialization. ■

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Cutaneous Manifestations of Coronavirus Disease-19

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Severe acute respiratory syndrome coronavirus 2 causes coronavirus disease-19 (COVID-19) and is associated with cutaneous manifestations.¹ Case reports describing complications due to hypercoagulation and microvascular occlusion are increasing.^{1,2} The accelerated clotting process also appears to affect the skin. Lesion descriptions vary. Some clinicians report lace-like purple rashes called livedo reticularis. Some patients may exhibit a measles- or morbilliform-like rash.² Reports of ischemia of the acral areas of fingers and toes also abound in the literature.¹⁻⁵ Practitioners also describe the presence of retiform purpura, which has a deep red to purple presentation.² Yet other case descriptions include erythematous macules and urticarial papules.^{2,3} The association between these lesions and COVID-19 is unclear at this time.¹⁻⁴

Prevalence and incidence are unknown due to the evolving worldwide public health crisis.¹⁻⁴ Also, because of this unique time, practitioners may misdiagnose the skin lesions due to the different dermatological manifestations of COVID-19.^{2,4}

The pathophysiology is poorly understood. Theories include a combination of viral and host factors. Some researchers propose that maladjusted immune responses or immune insufficiency may increase viral replication, causing tissue damage.² Other hypotheses include overactive immune responses driving immunopathological conditions known as cytokine storm, leading to macrophage activation syndrome.² Immune system cells secrete proteins called cytokines. These substances aid in the mediation and regulation of immunity, inflammation, and hematopoiesis.¹⁻⁴

Assessment. There are several recognized clinical patterns as a result of prospective consensus. Practitioners report pseudo-chilblains or erythema observations, with vesicles or pustules and purple discoloration affecting the acral areas of the hands and feet.^{1,4} Another pattern includes vesicular eruptions. These wounds

manifest as small monomorphic hemorrhagic lesions and commonly affect limbs, but may also appear on the trunk.¹ Of particular interest to this body of work is the appearance of livedo or necrosis at anatomical locations in the vicinity of pressure-sensitive anatomical areas.^{1,4} Theories exist that the lesions' varying appearance suggests an occlusive vascular etiology, especially when ischemia is also noted at the truncal or acral locations.¹⁻⁴

Clinicians may mistake purple or hemorrhagic vesicular eruptions on the buttocks for deep tissue injuries. The location of the lesion on the fleshy buttocks, its irregular shape, and lack of a pressure or shearing etiology should raise the index of suspicion for a COVID-19 lesion.⁵ Another distinguishing feature of the condition is its rapid resolution with less scarring than a deep tissue injury, even in the setting of a severe illness.⁵

Case study. A 60-year-old man presented to the emergency department with a 3-day complaint of worsening shortness of breath. Medical history included congestive heart failure, type 2 diabetes mellitus, and chronic obstructive pulmonary disease. Initial screening rendered a diagnosis of congestive heart failure with chronic obstructive pulmonary disease exacerbation. During his hospital stay, however, the patient's condition deteriorated. Chest radiographs and COVID-19 rapid screening test confirmed a diagnosis of COVID-19. The patient required intubation, extracorporeal membrane oxygenation, numerous vasopressor medications due to hemodynamic instability, and continuous renal replacement therapy. On hospital day 10, a purple discoloration with partial-thickness tissue loss was noted on the left buttock. The entire area, encompassing the bilateral buttocks, measured 15 × 5 cm (Figure 7). Chart review revealed a rapid onset of the lesion, initially described by team members as pseudo-chilblains progressing to a deep purple discoloration. The team treated the



FIGURE 7. COVID-19 lesion

area conservatively, and it resolved before transfer from the facility 10 days later. ■

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Pyomyositis

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Pyomyositis is a rare bacterial infection of skeletal muscles classified as primary or secondary.¹ A key differentiation between the forms is the location of the patient at the time of disease development. Patients in the tropical regions of Africa and the South Pacific exhibit the primary form of the disease.¹ In these settings, the patients are relatively young, often ranging from 2 to 5 years old.¹ Pyomyositis occurring in the world's temperate regions (eg, North America) is classified as secondary, and patients are usually older.¹ The increasing incidence of pyomyositis in temperate areas is mostly associated with an underlying disease process compromising the immune system.¹ Clinicians observe secondary pyomyositis in patients with immunodeficiency, trauma, intravenous drug use, concurrent infection, diabetes, and malnutrition.² Blunt force trauma or vigorous exercise account for 20% to 50% of pyomyositis cases.² Additionally, spontaneous hematoma formation from anticoagulants for deep vein thrombosis prophylaxis may also cause the infection.² Males appear to be affected more often than females at a 3:1 ratio.³

The exact pathophysiology of pyomyositis is unknown. Researchers theorize that the condition results from the hematogenous spread of infection through the bloodstream.^{3,4} The disease behaves like a malignant metastasis in which the bacteria travel through the bloodstream and seed in the larger striated muscle groups of the lower body.³ There are 3 distinct phases of pyomyositis. During the initial invasive stage, pyomyositis signs and symptoms are vague and nonspecific.¹ Patients may exhibit fever, pain, leukocytosis, and a woody edema.^{2,3} Interestingly, due to the deep-seated location of the infection, fluctuance and erythema are absent.^{2,5} In the second stage, 10 to 21 days after the original insult, muscle pain increases, edema worsens, and fever persists.³ As the infection progresses to the suppurative third stage, the patient exhibits signs and symptoms of



FIGURE 8. Pyomyositis on buttocks

systemic inflammatory response syndrome.³ The condition becomes disseminated, resulting in septicemia, septic shock, and multisystem organ failure.^{2,3}

Globally, *Staphylococcus aureus* is the most common culprit of the disorder, although clinicians are increasingly reporting methicillin-resistant *S aureus*.² The most common organisms obtained via wound cultures during surgery are *S aureus*, β -hemolytic *Streptococcus*, and the gram-negative bacilli *Escherichia coli*.²

Assessment. Clinicians can differentiate pyomyositis from pressure ulcers/injuries (PU/Is) and necrotizing soft tissue injury (NSTI) or necrotizing fasciitis by starting with a detailed patient history.³ Practitioners should be attuned to reports of recent or remote blunt force trauma to the area of concern.^{2,3}

Patients with PU/Is exhibit tissue damage over vulnerable, high-risk, bony prominences or under medical devices.^{6,7} PU/I infection with associated bacteremia is rare.⁶

A PU/I infection is diagnosed by examining the wound bed for necrosis, large amounts of exudate level, malodor, periwound cellulitis, fluctuance, or induration.⁶ Also, patients with an infected PU/I may not exhibit sepsis, unlike the patient with pyomyositis.^{3,7} Infected PU/Is are usually full-thickness ulcerations, unlike pyomyositis, which has vague initial presenting symptoms.^{3,7}

Clinicians may confuse pyomyositis with an NSTI. Necrotizing fasciitis evolves rapidly and involves the superficial and deep fascia.³ Pyomyositis, however, affects large striated skeletal muscle groups.³ NSTIs are more apparent as the patient presents with hyperemia, blisters, and subcutaneous emphysema.³ Imaging can help to distinguish pyomyositis from NSTIs.³

Diagnosis. Magnetic resonance imaging is the best diagnostic tool to identify pyomyositis.¹⁻³ Practitioners may also use computed tomography or ultrasound.^{2,3} The patient with pyomyositis exhibits abnormal laboratory values, including leukocytosis, positive blood, or deep wound cultures.^{1,3} Latter stages of pyomyositis present with signs of sepsis.^{1,3} Immunocompromised patients may have polymicrobial pyomyositis infection.^{1,3}

Case study. A 62-year-old man jumped out of the second story of his burning apartment building. He sustained burns on his bilateral buttocks and upper extremities involving 24% body surface area. Medical history included chronic and recurrent nephrolithiasis with hydronephrosis, skin cancer

with recent radiation therapy, and type 2 diabetes. He underwent debridement and grafting of his burns 24 hours after admission and was given anabolic steroids to support muscle development. He was fully mobile but reluctant to engage in physical therapy and occupational therapy daily.

Approximately 2 weeks into the hospital stay, an abscess was found on the left medial buttock and the patient had a concurrent fever of 102°F (Figure 8). This left buttock abscess was treated with incision, drainage, and local wound care. He then underwent a uroscopy and received intravenous antimicrobial therapy for the treatment of a urinary tract infection. The following week he complained of buttock pain and a fever developed. Computed tomography and nuclear scans were performed due to persistent leukocytosis with fever. Imaging results revealed a loculated fluid collection in the right gluteal muscle, leading to diagnosis of pyomyositis. The progression of the abscess from the left gluteal area to the right corresponded with the diagnosis of pyomyositis, as opposed to a PU/I. Moreover, most often PU/Is occur over the bony prominences and not between the gluteal cleft. Initial management included needle-guided aspiration of the abscessed area. Surgical intervention was necessary due to septic shock. Intraoperative findings included gluteal muscle ischemia and abscess. Presumably, the right gluteal infection continued to progress under pressure, causing necrosis, which

was relieved with surgical debridement. Multiple surgeries were required to excise the nonviable gluteal musculature. ■

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Necrotizing Soft Tissue Infections

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A necrotizing soft tissue infection (NSTI) is a severe, rapidly progressing infection resulting in full-thickness tissue destruction and sepsis.^{1,2} The condition, also called gas gangrene and necrotizing fasciitis, is rare and accounts for 0.3 to 5 per 100 000 infections.^{1,2} Causative organisms for NSTIs are the β -hemolytic *Streptococcus* species (group A streptococcus) and

occasionally *Staphylococcus aureus*.¹⁻³ Necrotizing soft tissue infections have a 25% to 30% mortality rate.^{1,3} The condition can occur anywhere in the body, but clinicians most often observe its appearance in the extremities, perineum, and torso.⁴ The mortality rate increases if the patient has comorbid conditions such as advanced age, immunocompromise, and shock.¹ Additional conditions

that can increase the risk of NSTIs are morbid obesity, diabetes, and illicit intravenous drug use.³

NSTI pathophysiology typically starts with an initial injury that allows pathogenic access to the tissues.³ Microbial infiltration and proliferation occur simultaneously with the release of endotoxins.^{1,2} These processes trigger a systemic inflammatory cascade, culminating in el-



FIGURE 9. Necrotizing soft tissue infection

evated cytokine production. The result is potentially deadly septic shock.^{1,2,4}

Assessment. Clinicians can differentiate NSTIs from pressure injuries through the patient's history and physical examination.¹ Caregivers should be wary of reports of a sudden onset and rapid deterioration of the skin condition, as this is the hallmark of NSTI.^{1,2} Additionally, the physical examination may reveal a wound that is not over a bony area or under a medical device. Palpation of tissues revealing subcutaneous emphysema or a "snap, crackle, pop," foul-smelling tan purulence that is frothy or contains bubbles, should raise the index of suspicion for an NSTI.^{1,2} Differentiating an NSTI from pyomyositis is revealed by imaging, patient history, and clinical presentation.³

Diagnosis. Diagnosing NSTIs is primarily achieved through evaluating the clinical presentation.^{2,4} An essential feature of this infectious process is its rapid evolution. NSTI presentation can be patient-dependent and may include hyperemia, edema, blister, and bullae formation.^{1,2,4} As the infection progresses, tissues may necrose, and signs and symptoms of subcutaneous emphysema can appear.⁵ The immunocompetent patient will exhibit the usual systemic signs of infection, such as tachycardia, leuko-

cytosis, hyperthermia, and mental status alterations.^{3,4} Severe cases present with hemodynamic instability and multisystem organ dysfunction or failure.⁵ The possibility of a necrotizing soft tissue infection should be considered if the patient reports pain that is out of proportion to the clinical observations.^{2,4} The laboratory risk indicator for necrotizing fasciitis scoring is not a sensitive indicator for NSTIs.⁵ For clarification, clinicians can order a computed tomography scan. Imaging will identify fat stranding, or a less organized appearance of the adipose tissue, as well as fluid and gas transecting fascial planes and fascial thickening.¹⁻³ If the test is performed with contrast, poorly defined fascia is highly suggestive of fascial necrosis.¹⁻³ Alternatively, an ultrasound can be ordered for hemodynamically unstable individuals.⁵

Reducing risk of death from an NSTI requires early recognition and aggressive treatment.⁴ Clinicians must consider surgical debridement, appropriate antibiotic treatment, and systemic support.¹⁻⁴ Early recognition of sepsis and its management to protect major organs are crucial to improving patient outcomes.¹

Case study. A 70-year-old woman with morbid obesity, chronic renal insufficiency, and type 2 diabetes presented to the emergency department with severe pain

to the buttocks of 3 days' duration. The right buttock exhibited a central area of black necrosis measuring 11 × 4 cm surrounded by a zone of hyperemia (Figure 9). The region was exquisitely tender to very mild palpation, and subcutaneous emphysema was detected upon physical examination. Initially, no drainage was observed but the area was foul-smelling. Probing of tissue yielded copious amounts of malodorous tan purulence with bubbles. Magnetic resonance imaging confirmed the presence of an NSTI resulting from a fistula-in-ano, and the patient underwent surgery. Postoperative management of the site included negative pressure wound therapy as well as negative pressure wound therapy with instillation to help solubilize necrotic tissue remnants. It is the practitioner's experience that this facilitates the easier removal of devitalized tissue during subsequent dressing changes. The patient was not a candidate for a fecal diversion due to body habitus and various social determinants of health. As a result, pharmacotherapeutics were used to liquify the fecal output to accommodate the use of an indwelling fecal management device. ■

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Cancer: Marjolin's Ulcer

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Marjolin's ulcer (MU) is a rare, aggressive form of skin cancer that develops in scar tissue, chronic skin ulcers, and areas affected by chronic inflammation. The incidence is estimated to range from 1% to 2% of all burn scars, and it most frequently takes the form of squamous cell carcinoma.¹

New ulcerations form whenever the integrity of the skin is compromised by spontaneous rupture, friction, or shearing. After a new ulceration in the area, patients may experience repeated cycles of healing and reopening. Malignant changes of chronic ulcers are closely related to the duration of the ulceration. Studies have found that in 82% of cases of MU, the latency period is greater than 10 years with an average time of 29 years after initial injury.²

Several theories have been developed as to how the chronic ulcers develop into malignancy. Suggestions have been made that the chronic irritation causes cell atypia and that continuous mitotic activity in regenerating tissue leads to DNA mutation, both of which precipitate malignant changes.³

Assessment. Wound appearance depends on various factors, including the history of the patient and the wound. Any wound can undergo a malignant transformation giving rise to an MU.³ As a result, clinicians should consider MU in the presence of any nonhealing friable wound with an atypical appearance and complicated healing trajectory.³ Full-thickness pressure injuries heal via scar formation. As a result, after ruling out osteomyelitis, clinicians must keep MU on the list of potential diagnoses.

Diagnosis. Because MU tends to be more aggressive compared with other skin neoplasms, early diagnosis and a well-designed treatment plan will increase the patient's chance of survival.³ Biopsy remains the gold standard for diagnosis and should be completed for lesions with an atypical appearance and complicated healing trajectory, especial-

ly if they have not healed in 3 months.³⁻⁵ Clinicians must be suspicious of lesions that exhibit cycles of forming scars that subsequently reopen.³ MU wounds often appear as flat ulcers with raised or rolled margins surrounded by induration.³

Case study. A 51-year old man with a history of bilateral lower extremity amputations secondary to a motor vehicle accident was readmitted with an ulceration of the right stump. The lesion presented as a significant ulceration with a cauliflower-like appearance covered by a layer of yellow fibrinous exudate. The center of the wound demonstrated hypergranulation, and epibole was present at the edges (Figure 10).

The motor vehicle accident occurred when the patient was 18 years old and resulted in his being trapped under a burning car. At that time, he incurred third-degree burns to bilateral legs. He underwent a below-the-knee amputation on the left leg along with skin grafting and an above-the-knee amputation on the right leg. The patient was active and wore bilateral prosthetics.

At the current admission, the patient stated that he had gotten a new socket for his prosthetic approximately 1 year ago and believed that the current lesion was a pressure ulcer/injury related to the new socket. At that time, an adjustment of the prosthetic had been made and a wound treatment regimen, including medical-grade honey and amniotic skin substitute wound grafting, was implemented. The patient believed that significant clinical progress had been made and had not pursued follow-up treatment until now.

A punch biopsy was performed. Pathology findings revealed atypical inflamed squamous proliferation with endophytic growth characteristic of squamous cell carcinoma. Following consultation with the surgical team, the patient decided to undergo above-the-knee amputation. Subsequent positron emission tomography scan showed no metastasis. ■



FIGURE 10. Marjolin's ulcer at right knee

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Coagulopathies: Cold Agglutinin Cutaneous Manifestation

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Cold agglutinin disease, also known as cold hemagglutinin disease, is a type of autoimmune hemolytic anemia in which autoantibodies trigger the agglutination of erythrocytes in response to cold temperatures.^{1,2} Specifically, when antibodies known as cold agglutinins are exposed to lower body temperature, they recognize antigens on erythrocytes; this results in agglutination or clumping of the erythrocytes and possible extravascular hemolysis.² Cold agglutinin disease (CAD) is a primary or idiopathic condition that is thought to arise from a monoclonal low-grade lymphoproliferative disorder.² Alternatively, secondary cold agglutinin syndrome (CAS) is a separate and distinct condition seen in the setting of an underlying disorder, such as viral infection.¹⁻³ Additionally, CAD- or CAS-associated hemolytic anemia can be triggered by cold exposure, fever, or acute illness.²

Primary cold agglutinin disease is present in 15% to 25% of persons with autoimmune hemolytic anemia, but the prevalence of secondary cold agglutinin syndrome is unclear.¹ Cutaneous manifestations of CAD or CAS are induced by cold temperatures and include acrocyanosis, Raynaud phenomenon, and skin ulceration or necrosis.²⁻⁴ These skin conditions may be found in acral areas such as the nose and fingertips, but cutaneous ischemia is not limited to these regions and clinical findings are specific to each type of condition.²⁻⁴ Tissue damage can result in permanent disfigurement, including scarring and loss of toes or other parts of the body.³

Assessment. The presence of CAD/CAS is often unknown to clinicians but should be considered whenever wounds indicating cyanosis, necrosis, or deep tissue injury (DTI) are pres-



FIGURE 11. Cold agglutinin lesion on right medial to lateral abdomen coinciding with placement of a targeted temperature management hydrogel pad

ent without a clear underlying etiology, such as evidence of vasculopathy or sustained pressure. Because even moderately cool temperatures (10°C/50°F) can trigger CAD or CAS, they should be considered when there is risk of hypothermia, such as during the immediate postoperative period, prolonged exposure to cool conditions prior to admission, or with cool-

ing therapy.⁵ Additionally, current and prior medical history is important because CAS may occur secondary to viral infection.

Assessment for cutaneous effects of cold agglutinin includes inspecting the wound and surrounding tissues for pallor, rubor, purpura, and cyanosis with or without necrotic skin breakdown consistent with dry gangrene.^{3,5-7}

The presence of cool or cold skin surrounding the wound and prolonged capillary refill time will be noted despite normal pulses and movement.⁴ The patient may or may not complain of decreased sensation and pain on palpation.³ Other signs consistent with cold agglutinin disease or syndrome may be present, including hemolytic anemia, usually without hematuria, and hepatomegaly.^{2,4,7}

Diagnosis. Thermostatic laser Doppler flowmetry may reveal decreased basal blood flow at the wound bed; however, good peripheral pulses with normal central and peripheral vascular flow will be seen on Doppler ultrasound.⁴ Capillary microscopy is useful to distinguish between Raynaud phenomenon and uncomplicated CAD/CAS, as it will be normal in the setting of cold agglutinin conditions.^{4,8} Laboratory tests to evaluate for the presence of coagulopathies, anemias, infection, and related underlying conditions should be completed; these may include a complete blood cell count, coagulation tests, differential blood smear, C-reactive protein, serum lactate, transaminases, serum creatinine, and urinalysis.⁴ Results may reveal anemia consistent with extravascular hemolysis independent of coagulopathy; however, this is not always present in CAS, and hematuria is unlikely.^{2,4} A cold agglutinin test should be conducted to detect the presence of autoagglutination of immunoglobulins, particularly immunoglobulin M, at temperatures less than 25°C.^{4,7} Increased immunoglobulin agglutination that resolves when exposed to warmer temperatures is indicative of cold agglutinin activity consistent with CAD or CAS.⁴ Serology results may reveal the presence of an underlying prior or ongoing bacterial or viral infection, which may have precipitated cold agglutinin syndrome.^{2,4}

Case study. A 40-year-old man was admitted to the intensive care unit following cardiac arrest with resuscitation and head trauma secondary to being hit in the head with a baseball

bat. Medical history included hepatitis C, hypertension, and obesity. The patient was unresponsive following return of spontaneous circulation, and targeted temperature management was initiated using an automated ice-water hydrogel pad system with a goal of lowering core body temperature to 36°C in accordance with American Heart Association guidelines and recommendations.⁹ Upon admission, the patient's skin was intact with the exception of a scalp laceration with surrounding hematoma.

Two (2) days after admission, the patient became spontaneously responsive, and targeted temperature management was discontinued. When the hydrogel pads were removed, a severe ecchymotic and purpuric rash with blistering was discovered on the patient's abdomen and flanks (Figure 11). This skin injury was initially identified as a DTI, owing to its areas of dark purple eschar and almost entirely intact skin. However, the clinical team was unable to determine how the patient could have developed a DTI in areas that did not sustain prolonged exposure to pressure. In considering underlying causes and etiologies of the lesions, it was noted that the patient had no known allergies and that the wounds included areas consistent with avascular necrosis. Pulses and results of Doppler examinations of the bilateral upper and lower extremities were within normal limits, and there were no other significant abnormal physical findings. Clinical and medical history were negative for autoimmune disorders or drug-induced acrocyanosis due to vasopressors. Routine laboratory test results were consistent with the clinical course, including mild normocytic anemia, but otherwise unremarkable. To evaluate for coagulopathies, a complete cell count and coagulation panels were drawn. Additionally, a cold agglutins test was performed, and results revealed agglutination of erythrocytes at temperatures below 20°C. It was therefore determined that cold agglutinin syndrome secondary to

hepatitis C infection in the setting of critical illness, together with cold therapy, had precipitated cutaneous necrosis similar to frostbite. ■

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Warfarin-induced Skin Necrosis

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Acute cutaneous necrosis is caused by a variety of etiologies, with warfarin being the most common.¹ However, warfarin-induced skin necrosis (WISN) is relatively uncommon, and its pathophysiology is not fully understood. Anticoagulants are among the most commonly prescribed medications, and between 0.01% and 0.1% of patients receiving warfarin will develop WISN.² There are case reports of WISN occurring on limbs, which can lead to amputation.³ Clinicians frequently report observing the condition in fatty anatomical areas such as the breasts, thighs, abdomen, and buttocks.³ Early recognition can mitigate severe sequelae.³ WISN predominantly occurs in middle-aged or perimenopausal women treated for thrombotic conditions.¹ Those with obesity, thrombophilia, or hepatic disease are at higher risk of this condition.^{1,2,4} High warfarin loading doses may predispose some patients to WISN.²

The exact pathogenesis of WISN is challenging to elucidate. The leading theory includes vitamin K-dependent clotting factors (II, VII, IX, and X; proteins C and S) and their variable half-lives.^{2,3} Warfarin's short half-life may contribute to a transient protein-C deficiency, which initially disrupts the balance between anticoagulant and procoagulant pathways. This imbalance creates a paradoxical hypercoagulable state that results in microvascular thrombosis.¹ Clinical manifestation in the majority of WISN cases (90%) occur between 3 and 6 days of medication initiation and progress to full-thickness necrosis within 2 to 3 days after presentation of the original skin lesion.¹ However, WISN may appear up to 3 years after initiating warfarin therapy.¹ WISN has been associated with intermittent warfarin therapy, previously mentioned risk factors, and potential drug interactions complicating warfarin metabolism.⁴



FIGURE 12. Warfarin-induced skin necrosis on the right buttock that was mistaken for a deep tissue injury

Assessment. Generally, WISN presents as an erythematous flush that is poorly demarcated and sometimes associated with concurrent edema or paresthesias.^{1,2} As the lesion evolves, it typically becomes painful and begins to demarcate. As fluid collects in the dermis and subcutaneous layers, a peau d'orange effect may occur. Surrounding petechiae and hemorrhagic bullae follow. As a result of occlusive thrombi in the skin and subcutaneous vessels, this eventually leads to painful, full-thickness tissue damage and skin necrosis.^{1,2} Rapid progression from onset to necrosis is associated with higher morbidity rates.⁵

Clinicians may encounter the disease at any point during its evolution. Evaluation for WISN should include a detailed history, including medications, their doses, and date of therapy initiation. WISN's color, purpura and necrosis, as well as location of the lesions can lead to a misdiagnosis of a pressure ulcer/injury.

Diagnosis. WISN may be confused with a variety of conditions, including pressure ulcer/injury, hematoma, necrotizing fasciitis, disseminated intravascular coagulation, calciphylaxis, purpura fulminans, embolic events, vasculitis, heparin-induced thrombocytopenia, and cellulitis.⁶ Diagnosis of WISN is usually clinical. Skin biopsy shows dermal platelet or fibrin thrombi and microthrombi, typically without inflammation.¹ Endothelial cell destruction and erythrocyte extravasation are also characteristic histological findings of WISN.²

Case study. A 65-year-old patient with a history of end-stage renal disease, end-stage liver disease, and morbid obesity was transferred from a skilled nursing facility with reports of a pressure ulcer/injury to the right buttocks that was deteriorating. Staff reported that areas initially appeared red with rapid progression to extensive purpuric areas with erythematous margins (Figure 12). Additional areas

appeared on the patient's abdomen and thighs. It was thought that these wounds were related to pressure because the patient often reclined in bed with her bedside table pressed against her abdomen and thighs. Her body habitus impaired the ability to reach the items on the table. Moreover, the patient spent extended periods sitting in bed or in a wheelchair. The patient's history and medication reconciliation included warfarin therapy for recurrent deep vein thromboses. Diagnostic workup included tissue biopsy revealing a histological pattern consistent with WISN. The diagnosis was then changed to WISN. Subsequently, the patient underwent surgical debridement. However, the wounds further deteriorated, but the patient was not a candidate for additional surgical intervention. As a result, the team initiated a series of biological debridements to achieve a clean wound base, amenable to reconstruction. ■

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Thrombocytopenic Purpura

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Severely low platelet count manifests as thrombocytopenic purpura. Reduced platelet production or increased platelet destruction causes this condition.¹ Thrombocytopenia < 20,000/ μ L is considered severe and is problematic for spontaneous bleeding.^{2,3} Endothelial cells line the walls of blood vessels and help to control permeability and maintain hemostasis. Purpura occurs when a lack of functioning platelets leads to extravasation of blood through the vessel walls. This process results in areas of hemorrhage into the skin or mucous membranes.^{1,4} Coagulation abnormalities and organ dysfunction may further potentiate bleeding risk in patients with thrombocytopenia.

The incidence of thrombocytopenic purpura varies and is dependent on etiology. Most cases of purpura are drug-induced.⁴ Common causes include chemotherapy, heparin, and certain antimicrobials.³ Other etiologies associated with thrombocytopenic purpura include viral infections, primary or secondary immune-mediated thrombocytopenic purpura (ITP), hematologic malignancies, and

thrombotic thrombocytopenic purpura.²⁻⁴ ITP is a rare condition, with an annual incidence of 0.01%.³ In children, a febrile illness may precede the development of ITP.^{3,4} Thrombotic thrombocytopenic purpura is a rare, potentially life-threatening condition characterized by hemolysis and microvascular thrombosis.¹

Assessment. The appearance of thrombocytopenic purpura ranges from a scattering of light red, pinpoint lesions to large areas of deep purple, bruised-looking skin.² These lesions are nonblanchable and nonpalpable. The patient may exhibit petechiae, small, diffuse purpuric lesions < 3 mm, which manifest in a rash-like fashion.^{2,4} The confluence of petechiae can create larger lesions, termed ecchymoses or purpuric lesions, > 3 mm. Any area of the body may be affected by mucocutaneous bleeding. Thrombocytopenic purpura, however, may be more pronounced in dependency areas, such as the back, buttocks, and lower extremities.⁴

Thrombocytopenic purpura can be differentiated from deep tissue injury



FIGURE 13. Thrombocytopenic purpura

(DTI) based on patient assessment and the pattern of distribution of the purpura. A high index of suspicion for thrombocytopenic purpura exists when atypically shaped purpuric lesions appear in regions not subjected to prolonged pressure or shear or in locations over a bony prominence or under a medical device.⁵ Risk factors for DTI and purpura may overlap, particularly in patients who are seriously ill. Due to the complexity and intersection of risk factors, the diagnosis of thrombocytopenic purpura versus DTI should involve multidisciplinary assessment.

Diagnosis. Diagnosing the condition starts with acquiring a thorough patient history, including comorbid conditions, complaints of easy bruising, petechial rashes, and medications.⁶ Patients with thrombocytopenic purpura may present with reports of prolonged bleeding and mucocutaneous hemorrhage, commonly in the gingiva and nasal mucosa.^{4,7} A complete blood count will identify suboptimal platelet levels and guide the need for additional tests.⁴⁻⁶

Case study. A man in his 30s with relapsed acute myeloid leukemia was undergoing aggressive chemotherapy and was admitted with septic shock and cy-

topenia. Skin breakdown had been noted on admission due to frequent incontinent stooling. Diffusely distributed petechiae were present across the lower back and thighs (Figure 13). These areas surrounded the more prominent ecchymotic areas on the upper back, flanks, and bilateral buttocks. Based on the distribution of purpura and laboratory test results indicating coagulopathy, a diagnosis of thrombocytopenic purpura was made.

It is likely that several factors contributed to the development of thrombocytopenic purpura. The patient was severely thrombocytopenic due to bone marrow suppression. The platelet count remained less than 10,000/ μ L, despite daily platelet transfusions. Spleen enlargement further aggravated the thrombocytopenia through the sequestration and destruction of platelets.³ The patient's sepsis-induced multi-organ dysfunction included liver failure and acute kidney injury, impacting both the quantity and functionality of the platelets.⁷ The back and buttocks were the most significant dependent areas due to poor medical condition and debilitation, resulting in the purpura distributing in similar locations as pressure or sheer injuries. ■

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Traumas: Morel-Lavallée Lesions

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Morel-Lavallée (ML) lesions are closed degloving soft tissue injuries resulting from acute blunt force trauma.¹ ML injuries are also known as ML effusions, closed degloving injuries, posttraumatic pseudocysts, and posttraumatic soft tissue cysts.² The true incidence of ML is unknown due to frequent misdiagnoses. One study examined 1100 fracture cases, which revealed a rate of 1.7%.² In reality, ML incidence is likely higher because the condition is often mistaken for deep tissue injuries, subcutaneous hematomas, muscle contu-

sions, bony cysts, fat necrosis, and myositis ossificans.¹⁻³

The typical ML lesion presents after a low blunt force trauma causes shearing forces that separate subcutaneous adipose tissue and skin from the anchoring fascia.^{1,2} Lymphatic and perforating vessels, located in the underlying musculature, flood the newly formed cavern with a milieu of blood, fat, and nonviable debris.²

Assessment. ML lesions usually appear in the greater trochanteric region, in the vicinity of the bony prominence. The area has a rich vascularity.² Individuals usu-

ally present with complaints of pain and swelling of the affected area either immediately or months to years after the traumatic event.¹ Tissues may exhibit compressible fluctuance and can be purpuric, dry, cracked, or necrotic.¹ Clinicians can arrive at a diagnosis through a detailed patient history with attention to a history of trauma at the affected area. Due to the mechanism of injury typically occurring over large muscle groups and manifestations of the condition, ML lesions are often misdiagnosed as deep tissue injuries or unstageable pressure ulcers/injuries.



FIGURE 14. Morel-Lavallée lesion at the right greater trochanter

Diagnosis. Diagnosing ML is challenging and requires particular attention to the patient's history, especially for trauma reports ranging from sporting and vehicular accidents to falls. While clinicians can use a variety of imaging modalities,

magnetic resonance imaging is preferred. It is considered the gold standard for identifying ML due to its ability to provide detailed images, unlike a computed tomography scan or ultrasound.^{1,3} Magnetic resonance imaging with contrast produces high-resolution imaging that helps with a prompt, accurate diagnosis, leading to timely management and improving patient outcomes.¹

Case study. A 60-year-old woman with schizophrenia, bipolar disorder, and substance use disorders as well as liver cirrhosis presented to a tertiary level of care after being found unconscious for an unknown period. The patient sustained numerous blunt force traumas resulting in a pelvic fracture. After repair of the fracture, the orthopedic team advised for surveillance of ML development. Over the duration of her admission, the patient developed an area of deep purple discoloration over the greater right trochanteric region (Figure 14). Orthopedic follow-up included aspiration of blood and exudate from the traumatized region.

The patient was discharged to a skilled nursing facility but was promptly read-

mitted due to leukocytosis, worsening fevers, chills, generalized myalgias, and excruciating pain at the right trochanter. The patient underwent surgical debridement of the infected area, followed by negative pressure wound therapy. In this instance, ML was differentiated from a deep tissue injury based on the history of blunt force trauma to the site. Furthermore, diagnostic imaging, the patient's clinical course, and intraoperative findings confirmed an ML diagnosis. ■

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Burn Treated With Cultured Epithelial Autograft

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In the United States, every year approximately 486 000 patients require medical treatment for burn injuries, including 30 000 individuals who require admission to a burn center.¹ Fires cause 43% of the injuries, followed by scalds at 34%.^{1,2}

Thermal burns occur when a person comes in contact with a substance at either end of the temperature spectrum, hot or cold.¹ The interaction results in a transfer of heat to the patient or away from the patient, such as in frostbite.¹ The burn precipitates a systemic inflammatory response syndrome.¹ The increased proinflammatory milieu, including cytokines, triggers substantial vessel permeability, further exacerbating inflammation.¹ Shock due to fluid

shift may occur if the patient's fluid and electrolyte needs are not adequately met.¹ Patients with severe burns (ie, those with greater than 50% total body surface area [TBSA] with full-thickness tissue damage) are prone to death due to sepsis, renal failure, malnutrition, or reduced cardiac output.¹

Burn care is complicated and depends on the tissues involved and TBSA affected.³ Many centers use the "rule of nines," or the Lund and Browder chart, to estimate TBSA burned.¹ In cases in which full-thickness burns involve more than 50% TBSA and in situations in which the patient does not have sufficient tissue to donate for a split-thickness skin graft, a cultured epithelial autograft (CEA) may be used.⁴

Clinicians at major burn centers will take a piece of the patient's intact epidermis and culture stem cells to obtain the keratinocyte lineage.^{4,5} The practitioners then propagate the growth of stratified epithelium sheets.^{4,5} This technique is expensive; thus, its use is limited.⁵ CEA requires good wound bed preparation to ensure the tissue's adherence.⁵ Patients with a burn are susceptible to sepsis, and CEA infection is another concern.⁵

Assessment. Burns injuries are classified depending on the tissue layers affected.¹⁻³ Superficial burns, previously called first-degree burns, affect the epidermal layer.^{2,3} Burns limited to the epidermis appear pink to light red, are typically painful, and usually resolve



FIGURE 15. Full-thickness burn treated with cultured epithelial allograft exhibiting graft site failure at the left scapula

within 10 days.¹⁻³ Partial-thickness, or second-degree burns, affect the epidermal layers and different dermal tissue depths.¹⁻³ These injuries can appear bright red to mottled.¹⁻³ Second-degree burns may also appear as blisters, which can rupture, become unroofed, and progress to open moist lesions.¹⁻³ Partial-thickness burns often resolve within 2 weeks.¹⁻³

Deep partial-thickness burns are also called deeper second-degree injuries and reflect damage to deeper dermal layers; however, follicles and sweat glands are unharmed.¹⁻³ Clinicians can identify these wounds by their dark red or yellow/white minimally moist surfaces. Patients will complain of severe pain due to damaged sensory nerve endings, while deep pressure sensation is preserved.¹⁻³ Deeper second-degree wounds require more than 3 weeks to resolve.³ Full-thickness burns are also known as third or fourth-degree burns. They describe the destruction of the epidermis, dermis, subcutaneous layers, muscle, and bone.³ These severe burns may appear white or charred and have a leathery texture. The surrounding edematous tissues exhibit high levels of fluid loss. Due to the destruction of nerve endings, patients do not complain of pain. Full-thickness

burns require complex care, including debridements and skin grafting.³

Diagnosis. Burn assessment starts in the field by first responders. These professionals will assess the scene to determine what occurred.³ During the evaluation, the clinician must ascertain what caused the injury, if the patient was in direct contact with the offending agent, and the length of contact time.³ Burn centers receiving the patient will conduct a physical examination to determine the extent and severity of the injury.³

Partial-thickness burns over a bony prominence may appear similar to a PU/I. Both conditions are classified by depths of tissue damage and may appear similar. It is essential to obtain a full history and perform a physical assessment on admission. Additionally, it is beneficial to have photographs in the medical record from admission of all the burned areas and all areas, including regions at high risk of PU/Is (eg, heels, sacrum, and occiput). Critically ill patients with a burn may be immobile for prolonged periods, undergo multiple surgeries, experience hypotensive episodes, and have skin edema. Although these factors increase the risk of PU/I development, clinicians must consider if the area of concern was burned at the initial assessment.

Case study. A healthy 31-year-old man sustained 92.5% TBSA full-thickness burns in a structure fire. Burned areas included the head, torso, bilateral upper extremities, and bilateral lower extremities, including the feet. The genital area was not affected. Upon admission to the burn center, the team performed escharotomies at areas of full-thickness circumferential burns, including the torso as well as bilateral upper and bilateral lower extremities. Due to a lack of donor sites related to the large surface area burned, CEA was applied to the wounds approximately 3 weeks after admission. Unfortunately, some of the skin grafts failed due to an infection of the wounds. One area was located on the patient's left scapula (Figure 15), which caused some confusion among clinicians. The staff categorized the site as a full-thickness hospital-acquired PU/I. Photographs obtained on admission, as well as before and after CEA treatment, identified the scapula as a site of graft failure. As a result, the team was able to correct the documentation. ■

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Multisystem Organ Failure: Skin Changes at Life's End

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Skin changes at life's end (SCALE) is an unpreventable skin breakdown some individuals experience at the end of life. It is an insidious process affecting the skin, the body's largest organ. At life's end, the body may divert blood from the skin to save vital organs, such as the heart and lungs, thus causing skin failure despite appropriate care. SCALE may also cause minor skin trauma to evolve into significant complications, including bleeding, gangrene, infection, skin tears, unavoidable pressure ulcers/injuries, and impaired removal of metabolic wastes.^{1,2}

The SCALE phenomenon was first described in 2008 by an interprofessional panel convened to discuss the Kennedy terminal ulcer, skin failure, and other end-of-life skin breakdowns; the result was a document of 10 consensus statements regarding SCALE.¹ The SCALE panel noted previous authors' works, including Kennedy³ and her research regarding the Kennedy terminal ulcer. Also cited was Langemo and Brown's⁴ work regarding skin failure, defined as "an event in which the skin and underlying tissue die due to hypoperfusion that occurs concurrently with severe dysfunction or failure of other organ systems." They noted this could be further delineated as acute, chronic, or end-stage skin failure. The SCALE panel supported their findings and surmised that as other organs failed, SCALE could also occur with acute and chronic disease despite its end of life name.¹

The etiology of SCALE requires more research,^{1,5} but it is essential to note that the most significant risk factor is decreased perfusion.¹ Langemo and Brown⁴ determined that skin failure is a perfusion issue, manifesting itself with disease and the disease/failure of other body organs; thus, SCALE could be considered a subcategory of end-stage skin failure.^{6,7}

Assessment. Patients experiencing SCALE may have notable skin changes, including color changes, mottling, decrease in skin turgor, significant skin breakdown, necrosis, hemorrhage, gangrene, infection, odor, and increased pain. Also, some patients experience other organ failure in addition to the skin. They may also experience decreased mobility (often becoming bedridden), loss of appetite, cachexia, weight loss, dehydration, and low albumin and hemoglobin levels.^{1,2}

Hill and Petersen⁸ noted that skin failure includes Kennedy terminal ulcers, SCALE, and Trombley-Brennan terminal tissue injuries; they proposed a skin failure clinical indicator scale (SFCIS). Factors include albumin, primary diagnosis, presence of sepsis/multiple organ dysfunction syndrome, vasopressor/inotrope use, and mechanical ventilation. Also, a group of Australian researchers and clinicians are investigating strategies that can be used to better detect end-of-life ulcers.⁹

Diagnosis. The patient's medical history, response to treatment, and overall prognosis lead the clinician to a SCALE di-

agnosis. If there is skin breakdown, despite appropriate care and the patient is in the terminal phase of life, the patient may be diagnosed with SCALE.¹

Case study. A 90-year-old woman presented to the emergency department from a skilled nursing facility due to a change in her mental status. Per the staff report, the patient had stopped eating and drinking approximately 3 days ago. Medical history included advanced dementia, renal insufficiency, coronary artery disease, and osteoporosis. The patient's body habitus revealed cachexia and kyphosis. Additional findings included what appeared to be suspected deep tissue ulcers/injuries along the thoracic vertebrae (Figure 16). The primary team consulted the medical social worker to assist with filing an ombudsman report against the skilled nursing facility for potential neglect. The social worker decided to obtain input from the wound care specialist. Careful chart review, physical examination, and staff interviews revealed a patient with a slow, steady decline in the month be-



FIGURE 16. Skin changes at life's end along the thoracic vertebrae

fore hospitalization. The patient also exhibited mottling of her acral regions. Her current diagnoses included severe sepsis due to pneumonia and a urinary tract infection. The patient was also hemodynamically unstable. An interdisciplinary team meeting resulted in a diagnosis of SCALE. Based on the patient's age, comorbidities, end-of-life instructions, and family wishes, hospice was consulted. The team treated the area along the vertebrae conservatively while managing expectations that the site may deteriorate. The patient was given comfort care and died 3 days after admission to the hospital. ■

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