A Review of Nail Dystrophies for the Practitioner

Andrew Bloom, BS, Medical Student, School of Podiatric Medicine, Temple University, Philadelphia, Pennsylvania;
Brandon Blanken, BS, Medical Student, School of Podiatric Medicine, Temple University, Philadelphia, Pennsylvania;
Brandon Schlakman, BS, Medical Student, School of Podiatric Medicine, Temple University, Philadelphia, Pennsylvania;
Tom Arena, BS, Medical Student, School of Podiatric Medicine, Temple University, Philadelphia, Pennsylvania;
Zachary Mironov, BS, Medical Student, School of Podiatric Medicine, Temple University, Philadelphia, Pennsylvania;
Tracey C. Vlahovic, DPM, Clinical Professor, Temple University School of Medicine, Philadelphia, Pennsylvania

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GENERAL PURPOSE: To provide information about nail pathology from its clinical presentation, pathophysiologic origin, clinical diagnosis, diagnostic testing, and treatment.

TARGET AUDIENCE: This continuing education activity is intended for physicians, physician assistants, NPs, and nurses with an interest in skin and wound care.

LEARNING OBJECTIVES/OUTCOMES: After participating in this educational activity, the participant should be better able to:

1. Review the etiology of and risk factors for the various types of nail pathology.
2. Describe the clinical manifestations, diagnosis, and treatment of the various types of nail pathology.

ABSTRACT Nail pathology has a range of etiologies, from biomechanical trauma to systemic associations. Within this review, nail pathology is examined from a clinical presentation, pathophysiologic origin, clinical diagnosis, diagnostic testing, and treatment standpoint. Nail dystrophy reveals both systemic and exogenous pathology, reinforcing the value of assessing nails during the medical examination.

KEYWORDS: koilonychia, leukonychia, longitudinal melanonychia, onychorrhexis, nail dystrophy

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INTRODUCTION

Nail pathology is often overlooked or poorly understood; however, a patient's fingernails and toenails can provide great insight into their overall health. Specifically, nail pathology can be an indication of systemic underlying disease, but also have etiologies ranging from biomechanical abnormalities, fungal infections, physiologic disturbances, vitamin and mineral deficiencies, or secondary bacterial infections. This article will review both common and uncommon nononychomycosis toenail pathologies that practitioners may encounter.

LEUKONYCHIA

Leukonychia describes the white discoloration of the nail plate, which can be attributed to several different etiologies. It can present clinically in various forms including leukonychia totalis, leukonychia partialis, leukonychia striata, and leukonychia punctata.[1] Leukonychia totalis is an autosomal dominant hereditary condition that presents as whiteness of the entire nail.[2] Leukonychia partialis, on the other hand, involves only a portion of the nail and is more commonly associated with a systemic disease or trauma. However, both conditions can be acquired or inherited.[3] Although the exact pathophysiologic mechanism of leukonychia totalis is unclear, it is thought

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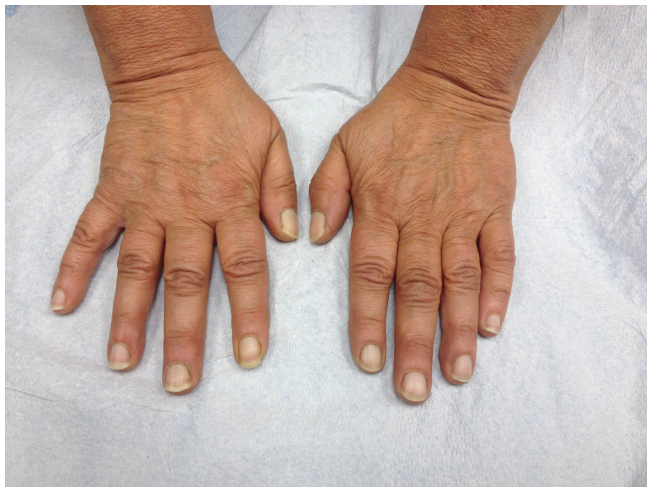
Figure 1. TERRY'S FINGERNAILS



to be attributable to abnormal keratinization of the nail plate.[4]

Although leukonychia can be associated with several disease states and toxicities, some common ones include renal disease, liver disease, cardiac disease, and arsenic intoxication.[5] When associated with these disease states, it will often present as one of four nail patterns: Mees lines, Terry's nails, half and half or Lindsay's nails, and Muehrcke's lines.[5] Mees lines are transverse, nonblanching, nonpalpable white bands that run parallel to the lunula.[6] These lines are associated with arsenic poisoning, certain chemotherapeutic drugs, and Hodgkin lymphoma.[6] Terry's nails involve whiteness of almost the entire nail with a sparing of the distal 0.5 to 3.0 mm; this pattern is indistinguishable from the lunula (Figures 1 and 2).[7] It can occur with heart failure, chronic kidney disease, hepatic disease, diabetes, and peripheral vascular disease.[7] In Lindsay's nails, the proximal third to half of the nail is white, and the distal aspect is reddish brown.[8] This arrangement is seen in patients with chronic kidney disease and renal failure.[8] Muehrcke's lines are multiple white parallel bands that are separated by normal pink tissue and are present on all nails bilaterally.[9] These lines are characteristic in individuals who have chronic hypoalbuminemia associated with hepatic disease and cirrhosis.[9] For these disease-associated manifestations of leukonychia, correction of the underlying disease may lead to normalization of the nails.[10]

BEAU'S LINES

Beau's lines are transverse palpable grooves that run along the nail and represent periods of arrest in growth of the nail plate (Figure 3). They may be attributable to prior severe systemic illness or trauma.[11] Each insult to the nail matrix is eventually followed by recovery and continuation of nail growth.[12] This generates the grooved appearance of the nail.[12]

Beau's lines most commonly occur bilaterally, affecting all 20 digits, as a result of systemic illnesses and drugs. In some cases, it can occur in isolation when trauma or digital inflammation is involved, specifically trauma to the hand, wrist, elbow, or foot. These traumas can include manicures, pedicures, and shoe trauma, as well as fractures to the hand, wrist, or elbow.[13] Some more common systemic conditions linked to Beau's lines are zinc deficiency, erythroderma, psoriasis, eczema, pemphigus, Raynaud phenomenon, hyperpyrexia, acute stress, rheumatic fever, malaria, myocardial infarction, oral retinoid treatment, and chemotherapy treatment.[13–15] Considering that fingernails grow at a rate of about 0.1 mm/d and toenails about 0.03 mm/d, the length of

Figure 2. TERRY'S TOENAILS

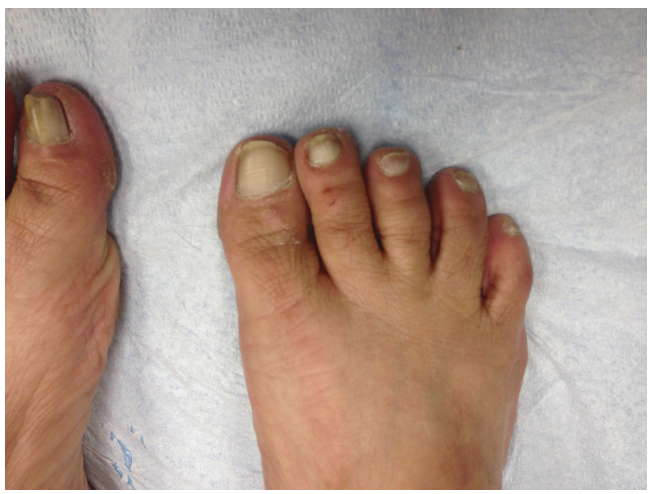
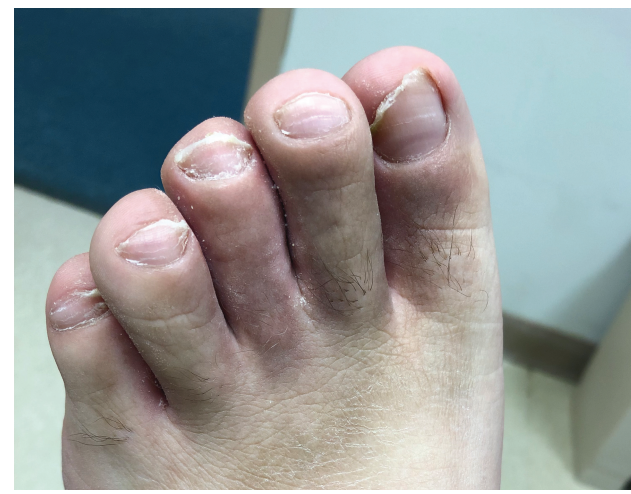


Figure 3. BEAU'S LINES ACROSS ALL NAILS



time of the pathology can be gauged by measuring the distance from the proximal nail fold to the distal groove of the Beau's line.[13,14]

ONYCHOSCHIZIA

Onychoschizia is nail plate layering.[16] The layering of the distal nail is attributable to an impairment of adhesive factors.[17] Diminished adhesive factors leading to the separation of nail in a transverse manner can potentially result in further pathology because the space of dead tissue serves as a bacterial nidus. Most notably, when onychoschizia presents concurrently with onychorrhexis, it is known as brittle nail syndrome.[17] As the pathology progresses over time, Bodman[18] noted that, in addition to transverse nail separation, the upper part of the nail often separates and flakes off. In a clinical setting, the detachment at the distal portion is visible and can be seen in the patient's fingernails.[18]

A multitude of studies and reviews have been undertaken to connect onychoschizia with greater systemic pathology, but have resulted in nonsignificant correlations. Wallis et al[19] demonstrated that repeated hydration and drying of nails showed clinical demonstration of onychoschizia. In their study, nail samples were hydrated with variable solvents; half were air dried for 4-hour periods at room temperature. Of the solvents tested, only water plus a 4-hour dry time manifested the onychoschizia pathology.

The connection between recurrent hydration and dehydration and onychoschizia can explain the increased incidence among those who are in professions requiring constant hand use, washing, and trauma. Baran and Dawber[20] deduced that there was a higher incidence of onychoschizia among bartenders, homemakers, and medical personnel. Individuals older than 60 years with onychorrhexis were the most likely to have onychoschizia.[18]

A case study by Chinazzo et al[21] noted a connection between onychoschizia and children with scabies. In the study, 47 children were included. Of the 47, three children had *Sarcoptes* mites within their nails as a result of crusted scabies. Chinazzo and colleagues[21] concluded that nails cannot be overlooked in the assessment of a patient with scabies.

Physical examination reveals the obvious clinical signs of onychoschizia, but additional diagnostic testing involves scanning electron microscopy,[22] which may reveal ragged nail cells and leaning edge dystrophy of nail cells, leading to the clinical presentation.

Both onychoschizia and brittle nail syndrome can be treated conservatively with biotin.[23] Biotin increases nail thickness and reduces splitting, yielding a significant increase in cosmesis.

HAPALONYCHIA

Hapalonychia, also referred to as onychomalacia or "soft-nail disease," is a condition characterized by a thin, malleable nail plate that may crack or split at the ends.[18] In some cases, the nails may be semitransparent with a bluish white hue ("eggshell nails").[24,25]

Hapalonychia can be congenital or caused by a deficiency in vitamins and minerals. More specifically, hapalonychia has been associated with deficiency in vitamins A, B_6, C, and D and calcium[26] Further, hapalonychia can be a clinical presentation of multiple pathologies that induce hyperhidrosis. There are multiple conditions that may present clinically with hapalonychia: cachexia, wet gangrene, leprosy, kwashiorkor, scleroderma, paronychia, and myxedema.[18]

If the cause is deficiency, the treatment regimen for hapalonychia should be vitamin or mineral supplementation to replenish adequate storages. Otherwise, hapalonychia can be managed by properly trimming the nails to reduce symptoms.

ANONYCHIA

Anonychia may present as a partial or total absence of one, several, or all nails on the extremities.[27] There are various phenotypic presentations of anonychia with a range of severity depending on the underlying etiologies and associated conditions.[27] The milder form of anonychia that results in hypoplasia of the nail is hyponychia, or incomplete anonychia. Total loss or absence of the nail is referred to as complete anonychia, and anonychia occurring in the absence of coexisting congenital mutations is isolated or simple congenital anonychia.[27] Anonychia may be genetic or acquired and can result from a multitude of etiologies within these two overarching classifications.

Although the pathogenesis of congenital anonychia is not fully understood, it is generally accepted that it results from defects in the *RSPO4* gene.[28] Thus far, there are 18 confirmed mutations in the *RSPO4* gene that are implicated in isolated congenital anonychia.[28] The protein produced by this gene, R-spondin-4, is involved in the Wnt signaling pathway.[29] The Wnt pathway plays a critical role in cell division, adhesion, and migration; mutations that alter R-spondin-4 synthesis lead to pathology in nail development.[30] The inheritance of *RSPO4* gene mutations follows an autosomal recessive pattern, with parents generally lacking signs and symptoms of the condition.[30]

There are numerous coexisting genetic anomalies that are associated with congenital anonychia. Many of these genetic conditions are rare, with varying phenotypic presentation of the nail pathology. Although the associated conditions vary widely, the underlying cause of nail pathology can be traced back to *RSPO4* gene mutations or alterations in the ectoderm.[30] Consumption of

teratogenic drugs by the mother during pregnancy can also lead to hyponychia or anonychia in the neonate.[31] Incidence of either condition occurs more frequently upon consumption of the teratogens in early months of pregnancy.[31]

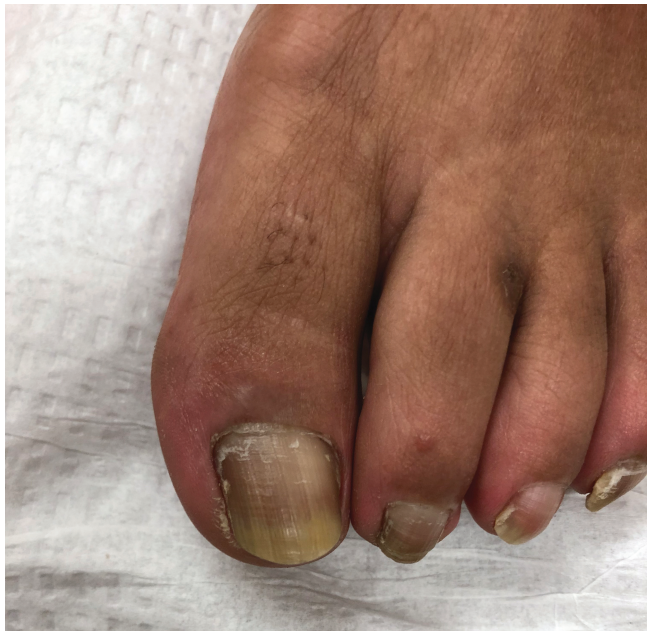
Acquired anonychia typically occurs because of trauma or disease,[31] inducing partial or total destruction of the nail matrix. Traumatic causes generally include accidents involving the digit and nail, iatrogenic injury through surgery, burns, freezing, or chemical damage.[31] In some cases, anonychia may be intentionally induced in matrixectomy procedures that are intended to prevent recurrent ingrown nails and other pathologies. Traumatic causes tend to be more common than anonychia stemming from rare inflammatory, ischemic, and bullous disorders.[31]

The most common of the inflammatory disorders resulting in anonychia is lichen planus, which can present as idiopathic atrophy of the nails.[31] Bullous disease tends to cause desquamation of the digits, resulting in anonychia vulnerability.[31] Often, ischemic diseases can result in bone destruction that contributes to ensuing anonychia.[31] Other various conditions are associated with anonychia as well and can play a role in its development.

ONYCHORRHEXIS

Onychorrhexis is described as nail thickening and ridging in a longitudinal pattern (Figure 4). Nails have a coarse appearance and can be casually described as brittle nail syndrome.[32] Features of onychorrhexis can vary from a small number of isolated ridges to deep involvement covering up to 70% of the nail. Longitudinal splitting can be superficial or deep.[17] Twenty percent of people have reported signs of brittle nails. Women are more frequently affected, and the incidence is 15% higher in patients older than 60 years.[17]

Figure 4. ONYCHORRHEXIS OF THE GREAT TOE



Onychorrhexis is most commonly caused by trauma from exogenous sources, lichen planus, and systemic pathologies. All causes are related to disorganized regulation of keratin in the nail matrix.[33] Exogenous trauma including chemical exposure, frequent hand washing, and pedicures is most common.[33] Further, onychorrhexis is a hallmark feature of lichen planus. Diagnosis of lichen planus can be inferred from a large midline indentation or ridging, but this finding can be normal in older adults.[32] Systemic findings include anemia and arteriosclerosis that decrease the oxygen content of the matrix, leading to keratin disorganization.[34] Other metabolic causes include rheumatoid arthritis, hyperthyroidism, hypothyroidism, anorexia nervosa, bulimia nervosa, and Raynaud phenomenon.[33–35] It has been theorized that sulfur content is diminished in patients with onychorrhexis. This leads to fewer disulfide bonds in proteins that form keratin fibrils.[34]

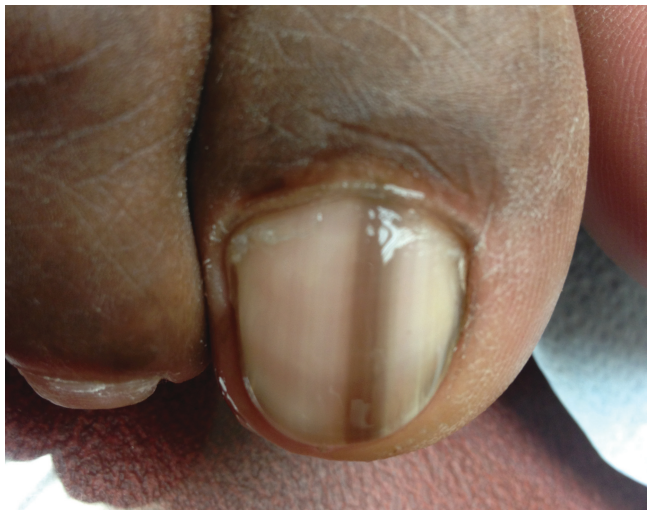
Differential diagnoses of onychorrhexis include nail bed scarring, infection, and bleeding.[17] Nail appearance can influence patient quality of life. Self-esteem may be diminished in onychorrhexis, which can negatively affect social interactions.[17] Overall, it is important to recognize and appropriately treat onychorrhexis.

Treatment can be dependent on the source of the problem. Soaks in warm water with frequent rehydration and the application of moisturizers can be used as a first-line treatment.[34] New studies have investigated the role of biotin, which has been found to increase nail plate thickness by improving the synthesis of lipids that bind the nail keratinocytes. Scanning electron microscopy determined a 25% increase in nail thickness following biotin therapy. Sixty-three percent of patients in a recent biotin clinical trial had nail improvement with almost no adverse effects.[36]

LONGITUDINAL MELANONYCHIA

Longitudinal melanonychia (LM) presents as a brown/black longitudinal band that extends from the nail matrix to the distal edge of the nail plate (Figure 5).[37] It is caused by one of two processes, either by melanocytic activation or melanocyte proliferation.[38] Most LM is caused by a benign process such as melanocytic activation or melanocytic nevi; however, another cause is malignant melanomas.[38] Nail melanomas are most commonly seen in those between ages 50 and 60 years; LM arising in patients of this age, along with factors such as location (the thumb, great toe, and index finger are most common), skin color, and a lesion larger than 6 mm, requires more in-depth examination.[37] If a patient has fair skin, LM should

Figure 5. LONGITUDINAL MELANONYCHIA



always be considered to be and treated as malignant melanoma until proven otherwise. If a darker-skinned patient presents with LM on multiple digits, it is most likely benign. However, if LM is only on a single digit, further examination should be performed because there is a higher incidence of malignancy.[37]

Performing an in-depth and thorough patient history is the first step in differentiating malignant or benign LM. Medication, social, and medical history can indicate trauma or drug use as the cause and rule out malignancy. Zidovudine, psoralens, cancer chemotherapeutic agents, and hydroxyurea are all common drugs that can cause melanonychia in patients.

A helpful tool in diagnosing whether LM is malignant or benign is the dermatoscope. Dermatoscopy allows the visualization of lesions that are not normally seen and will reduce the number of unnecessary biopsies for patients with LM. Dermatoscopy is somewhat limited because it cannot visualize the nail matrix where the disease begins.[39] For this reason, nail biopsies remain the criterion standard to differentiate benign from malignant LM. Many different techniques can be performed; however, it is important to obtain the nail matrix specimen because this is where the disease begins.

If the lesion is benign, the course of action for these patients is just reassurance. Providers should monitor the lesion as a dermatologist would monitor a mole. However, if it becomes malignant, surgical excision is indicated.[20] Conservative treatment includes the excision of 5- to 10-mm borders without bone resection and possible lymph node resection. More aggressive treatment requires amputation at either the proximal interphalangeal joint or the metacarpophalangeal joint.[37]

KOILONYCHIA

Koilonychia, also known as spoon nails, presents with concavity or flattening of the nail. This condition can affect either some or all the nails and often presents on both the hands and the feet.[41] Appearance of koilonychia at birth is a normal variant in many neonates,[42] often presenting on the nail of the hallux and persisting for several years before self-correcting as the phalanx matures.[42,43]

Causes of koilonychia include hereditary, acquired, and idiopathic etiologies. The condition is associated with a multitude of diseases and can serve as an indicator of systemic pathology in some cases.[44]

The pathogenesis is hypothetically attributable to three different mechanisms. The first mechanism proposes that koilonychia occurs "due to structural stress incorporated during the keratinization process of nail formation."[45] The second postulates that koilonychia is a result of "a lesser angulation of [the] distal part of [the] nail matrix compared to [the] proximal, altering the direction of nail growth."[45] Finally, the third theory proposes that "changes are the result of anoxia and atrophy of the distal nail matrix."[45] Although the mechanism is not fully understood, it is likely that the exact pathogenesis varies with respect to the underlying etiology.

One of the common manifestations of koilonychia results from hereditary inheritance. Familial koilonychia is known to have an autosomal dominant inheritance pattern.[41] This disorder is known to have a high degree of penetrance, with no ectodermal, systemic, or biomechanical defects present.[45] The course of koilonychia can remain unchanged or can worsen over time.[41] In some cases, the pathology can even become debilitating (the shape of the nail can make some activities of daily living difficult) and require continuous management.[41] In rare cases, koilonychia can present as a singular disorder, but it is more often associated with several other findings, the most common being iron-deficiency anemia.[45] The presence of anemia and a thorough family history often function as identifying factors for familial koilonychia.[45]

Acquired koilonychia can be a sign of systemic disease. Again, the most common and well-known association is iron-deficiency anemia. A previous study found koilonychia appears in 5.4% of patients with iron deficiency, and signs are reversible with replenishment.[44] Other dermatologic findings include psoriasis, alopecia areata, Raynaud phenomenon, systemic lupus erythematosus, lichen planus, and onychomycosis.[44,46] Hemochromatosis has also been demonstrated as a systemic finding.[43] It is important to note that acquired koilonychia has been seen as a result of nail-irritant allergic dermatitis from chemical depilatory agents and possibly caused via the disruption of disulfide bonds.[47] Finally, acquired koilonychia can present as a result of trauma or occupational hazard.[46]

An easy way to differentiate koilonychia from other nail diseases is with the water drop test. A few drops of water can be placed on the nail. In a normal nail, fluid quickly runs off the side. With koilonychia, water pools in the central bed of the nail plate.[46] Treatment should include removal of irritants, restoration of iron levels, and treating the underlying systemic pathology, if any.

CONCLUSIONS

Common nail pathologies may be overlooked during a standard patient encounter, but it is important to recognize them because they may arise from systemic issues. Nail pathologies such as LM and leukonychia can be signs of more serious diseases such as melanoma and renal disease, respectively. Some nail disease states including anonychia and hapalonychia are congenital, whereas many others are caused by trauma or organisms (bacteria and fungi). It is vital to include nail assessments in every medical examination to assist in the diagnosis of underlying systemic disease as well as define what may be affecting the nails exogenously.

PRACTICE PEARLS

- Nail pathologies may be a sign of an underlying systemic disorder or arise from exogenous sources.
- Nail changes may be initial signs of various disorders before an illness manifests.
- Various nail issues may result from congenital abnormalities, such as anonychia and koilonychia.
- Dermatoscopy aids in diagnosis of LM, which arises from various issues: fungal pigmentation, medications, melanocyte activation (nevi), or melanocyte abnormalities (melanoma). ●

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This activity provides ANCC credit for nurses and *AMA PRA Category 1 Credit™* for MDs and DOs only. All other healthcare professionals participating in this activity will receive a certificate of participation that may be useful to your individual profession's CE requirements.

CONTINUING EDUCATION INSTRUCTIONS

- Read the article beginning on page 20. For nurses who wish to take the test for CNE contact hours, visit http://nursing.ceconnection.com. For physicians who wish to take the test for CME credit, visit http://cme.lww.com. Under the Journal option, select *Advances in Skin and Wound Care* and click on the title of the CE activity.
- You will need to register your personal CE Planner account before taking online tests. Your planner will keep track of all your Lippincott Professional Development online CE activities for you.
- There is only one correct answer for each question. A passing score for this test is 14 correct answers. If you pass, you can print your certificate of earned contact hours or credit and access the answer key. Nurses who fail have the option of taking the test again at no additional cost. Only the first entry sent by physicians will be accepted for credit.

Registration Deadline: December 31, 2021 (physicians); December 3, 2021 (nurses).

PAYMENT

- The registration fee for this CE activity is $17.95 for nurses; $22.00 for physicians.