CLINICAL MANAGEMENT

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Common Nail Disorders and Fungal Infections

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Afsaneh Alavi, MD • Clinical Fellow • Sunnybrook Health Sciences Centre • Toronto, Ontario, Canada
Kevin Woo, MSc, PhD(C), RN, ACNP, GNC(C) • Lecturer of Public Health Sciences and Medicine, University of Toronto • Clinical Scientist/Nurse Practitioner • Wound Healing Clinic, The New Women's College Hospital • Toronto, Ontario, Canada
R. Gary Sibbald, BSc, MD, FRCPC (Med) (Derm), FAPWCA, MEd • Professor of Public Health Sciences and Medicine • University of Toronto • Toronto, Ontario, Canada • Director • Wound Healing Clinic, The New Women's College Hospital • Toronto • Clinical Associate Editor • *Advances in Skin & Wound Care* • Ambler, PA

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PURPOSE

To provide the practitioner with current information on the most common nail disorders.

TARGET AUDIENCE

This continuing education activity is intended for physicians and nurses with an interest in wound care and related disorders.

OBJECTIVES

After reading this article and taking this test, the reader should be able to:

1. **Describe the structures that compose the nail apparatus.**
2. **Identify the most common nail disorders, including etiology and treatment.**

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Nail disorders make up about 10% of dermatologic conditions and are more prevalent in the elderly.[1] This prevalence can be related to several factors, including impaired circulation, concurrent chronic systemic disease (eg, diabetes mellitus), neoplasm, changes in foot biomechanics, and a deficient immune system.[2,3] It is increasingly acknowledged that nails and systemic diseases are interconnected and that nails can provide valuable diagnostic clues to the underlying pathologic conditions.[4]

Nail disorders can impact a patient's quality of life. Pain, difficulty walking, difficulty wearing shoes, fear of spreading

disease, and social embarrassment are common concerns. Awareness of normal and pathologic changes in the nail permits more efficient treatment and management of these common problems. In this article, the authors will review the most common nail disorders in the differential diagnosis of fungal infection (onychomycosis) and cutaneous clues to systemic disease.

NORMAL NAILS

The origin of a nail is the same as that of the skin, hair, and teeth, and these structures may have combined involvement in genetic disorders. The nail apparatus consists of several components, including the nail matrix, nailbed, nail plate, the hyponychium, and the surrounding proximal and lateral nail folds. Although the nail plate is the largest and most visible part of the nail, one-fourth of the nail plate is actually concealed by skin known as the proximal nail fold. The proximal nail fold covers the proximal aspect of the matrix or nail-forming organelle, extending distally to the end of the lunula (half moon).[5] The lunula has a whitish discoloration due to a thin nail plate and immature keratin with retained epidermal cell nuclei. Where the lunula ends, the mature nail begins. The nailbed is highly vascular and it extends distally to the hyponychium, where normal keratinizing cells lift the nail off the nailbed and allow the free edge of the nail to extend beyond the distal finger or toe. The proximal and lateral edges of the nail are bordered by nail folds. The cuticle is the keratinized structure at the proximal nail fold that protects the space from water and yeast invasion.[5–7]

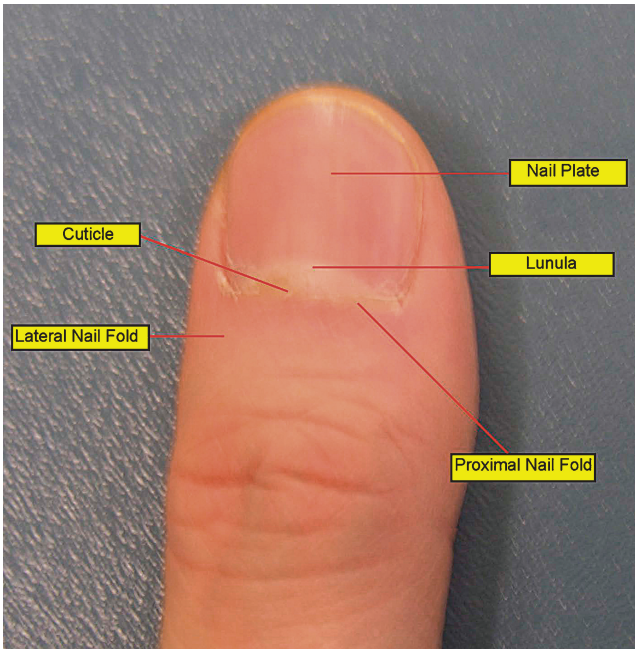
The nail grows along the nailbed at a rate of 0.1 mm per day or 3 mm per month. Finger nails take 6 months to grow out completely, and toenails take 12 to 18 months and longer with aging.[8] Nail growth can be variable because of certain physiologic and pathologic conditions. Because half of the matrix is hidden between the distal interphalangeal joint and the proximal nail fold, visible changes are delayed by 6 to 12 weeks. Given this slow growth rate, diseases involving the nail matrix often present after coexisting skin involvement, and nail changes require a long time to become normal after treatment. A normal nail plate is a hard keratin structure that covers the dorsal portion of the distal phalanx (Figure 1).

Before discussing nail disorders, the authors will introduce 2 case scenarios. In each case, consider the differential diagnosis, approach to investigations, and treatment.

CASE 1

John, aged 49 years, is a bartender who lives with his wife and 3 children. He has been diagnosed with type 2 diabetes mellitus that is controlled by oral hypoglycemic agents. The rest of his medical history is unremarkable. He has a 10-year smoking history of 1 pack a day. The nail of his large toe (hallux) is discolored and thickened (Figure 2).

Figure 1.
NORMAL NAIL



CASE 2

David, aged 65 years, is a businessman who presents with a loss of luster and a thickening of his toenails that have recently spread to his fingernails. David has a 10-year history of skin rash that is controlled with topical steroids. He complains of occasional pain and swelling of interdigital joints involving both hands and feet. He has had triple-bypass surgery. He is otherwise healthy (Figure 3).

ABNORMALITIES OF THE NAIL

Clinically, the most common nail disorders can be classified into 5 groups based on the most salient presenting features: nail discoloration, nail thickening, nail surface abnormalities, inflammation of the nail fold, and nail fungal infection.

NAIL DISCOLORATION

Brown-black nail: Longitudinal pigmented bands can be found in up to 77% of people with darkly pigmented skin.[9] The bands are regular and extend from the proximal nail fold to the distal tip of the nail (Figure 4.) *Subungual hematoma* is a common manifestation of nail trauma that must be distinguished from subungual melanoma. Subungual hematoma is commonly found under toe nail plates in joggers and people with ill-fitting footwear.[7] Acute subungual hematomas are red and painful, and become dark and nontender when mature.

Figure 2.
CASE 1

Sequential clipping or removal of the overlying nail plate usually reveals subungual hemorrhage to confirm the diagnosis. *Splinter hemorrhages* are thin, red or brown longitudinal lines that occur beneath the nail plate. They usually arise from minor trauma, psoriasis, or fungal infection, but they are also associated with subacute bacterial endocarditis, systemic lupus erythematous, rheumatoid arthritis, and other less common disorders.[3] Although darkly pigmented lesions are common, they must be differentiated from subungual melanomas. A sudden change in the appearance of the bands, including darkening pigmentation, additional bands, feathering (irregular lateral extension of the pigmentation), band width of more than 3 mm, or involvement of the pigment in the proximal nail fold, cuticle, or surrounding skin (Hutchinson's sign) should raise the suspicion of subungual lentiginous melanoma (Figure 5).[5] Any concern about malignant transformation warrants a skin biopsy; the specimen should be taken from the most proximal pigment segment through the nail plate and include nailbed skin.

Green discoloration: Green discoloration is most commonly caused by *Pseudomonas* excreting a pyocyanin pigment (Figure 6). *Pseudomonas* prefers a wet and alkaline environment, so it is common in persons frequently exposed to water. The organism often coexists with *Candida* and *Aspergillus*, leading to inflammation of the nail fold (chronic paronychia),

Figure 3.
CASE 2

Figure 4.
BROWN BLACK NAIL

Figure 5.
SUBUNGAL LENTIGINOUS MELANOMA

especially in persons with diabetes.[3] Treatment includes cutting the nails short, clipping abnormal areas, and decreasing water exposure by using cotton-lined gloves. An agent such as acetic acid (1%) compress, silver sulfadiazine, or ciclopirox olamine may be applied topically.[10] In the presence of severe inflammation, oral antibiotics, such as quinolones including moxifloxacin and ciprofloxacin, may be needed.

White nail (leukonychia): Leukonychia is a white discoloration of the nail plate that can be total, subtotal, transverse, punctuate, or longitudinal, depending on the presenting feature and nail plate involvement. White discoloration may be from incomplete keratinization or abnormal vasculature of the nailbed. The punctuate form, more common in children, presents as small white opaque spots and is usually trauma related or idiopathic. Multiple horizontal parallel lines may appear in fingernails after manicure (Figure 7) and in toenails after trauma from tight-fitting shoes. In some patients with hepatic failure, chronic heart failure, hyperthyroidism, diabetes mellitus, or malnutrition, leukonychia is characterized by proximal white discoloration and a distal normal pink band 0.5-3 mm wide (Terry's nails).[3]

Yellow nails: Yellow discoloration of the nail has several causes, including external staining from nicotine or formaldehyde resins from nail polish remover. Photosensitivity caused by drugs, such as tetracyclines, can also produce yellow discoloration; the drug binds to keratin and is photo activated as a free radical, leading to local nail destruction.[3] Yellow nails can also result from slow nail growth caused by a disruption of local vascular supply. A very rare syndrome, yellow nail syndrome, may be associated with lymphatic abnormalities (including lymph edema), pulmonary effusion, recurrent pulmonary infections with destruction and widening of the large airways (bronchiectasis), and potential associated immune system abnormalities. The exact mechanism is not known but may be related to protein leakage from increased microvascular permeability that is common in the associated conditions.[11]

Figure 6.
PSEUDOMONAS INFECTION

Figure 7.
WHITE NAIL

THICKENED NAILS

Nail plate overgrowth (onychogryphosis): Onychogryphosis is the enlargement and thickening of the nail plate (Figure 8). Infrequent cutting with decreased arterial blood supply or recurrent trauma allows the nails to become thick and lose their surface luster. The nail keratin distally bends around and eventually curves under the toe. This appearance is eloquently described as Ram's horn or oyster like. The danger of excessive pressure is subungual hemorrhage, especially in the presence of diabetes mellitus or peripheral vascular disease. Other types of thickened nails may not have the dramatic changes of onychogryphosis. Psoriasis causes thickened nails because of abnormal retained hard keratin; other characteristics include pits and small irregular depressions in the nails, distal onycholysis (abnormal thick and separated distal nail plate), and whole-nail dystrophy similar to tinea. A fungal culture may be needed to rule out tinea. Psoriatic nails may also have an adjacent proximal, yellow-brown, regular band of discoloration of the nailbed (oil on water sign) not seen with tinea. This pigment is caused by immature keratin in the nailbed that can be seen through the nail plate.

Figure 8.
YELLOW THICKENED NAIL

Nail plate thickening: Thick nails may be the result of genetic abnormalities, trauma, poor circulation, fungal infection, or inflammatory skin condition such as psoriasis. Thick nails may cause pain and are associated with distal separation, secondary fungal invasion, subungual hemorrhage, and skin breakdown. Patients with these conditions may benefit from regular podiatric foot care, including periodic reduction of the thickened nail with electric drills and burrs.[12]

Ingrown nails: This condition is most common with the large toenails when part of the nail plate pierces the lateral nail fold. The exaggerated curvature may gradually grow into the lateral nail fold and produce pain. Eventually, granuloma formation may exacerbate swelling and pain. The 3 major types are pincer nail (overcurvature of the nail plate that may be genetic with an adult onset), subcutaneous ingrown toenail, and hypertrophy of the lateral nail fold. Pressure from ill-fitting shoes and improper cutting of the lateral edge of the nail are the usual predisposing factors. Proper shoes with an ample toe box should be worn to remove pressure from lateral nail folds. The patient should be instructed to cut the nails straight across and to not cut them too short. To prevent spicule formation and skin trauma, the patient should not cut below the distal lateral nail fold.[2,5] Clipping either the lateral edge or a central inverted V niche out of the nail plate even down to the lunula may mitigate pain from compression of the tented toenail while wearing shoes.[5] To help the nail grow up and over the soft tissue and prevent future ingrowth, a small piece of cotton wool can be inserted under the lateral nail plate. Granulation tissue can be destroyed by silver nitrate stick or surgical excision. Alternatively, the condition can be treated with aggressive surgery to remove the entire nail and destroy the nail forming matrix with phenol or laser.

SURFACE CHANGES

Pitting: Nail pitting appears as pinpointed pitted spots or defects in the keratin on the nail plate. It is caused by defective superficial layering and incomplete clumps of keratinized cells falling out of the nail plate. Pits can be scattered in patients with psoriasis, alopecia areata, or trauma. A linear pattern is most commonly seen in eczematous patients (Figure 9).

Transverse ridging: Surface changes on the nail plate result from abnormal keratinization in the matrix level. In elderly persons, increased longitudinal striations are not unusual. Formation of these striations is linked to an altered turnover rate of the matrix cells. Beau's line (Figure 10) is a transverse linear depression in the nail plate first described in 1846[4] and caused by a transient disruption in keratinization from severe disease, surgery, and exposure to cold temperatures in patients with Raynaud's disease. Beau's line almost always affects all nails. Multiple transverse grooves can result from several factors, including minor trauma, nail biting, chronic eczema, chronic inflammation, and paronychia.

Spoon nails (koilonychias): This condition appears as the converse of clubbing. The nailbed is concave with the edges everted similar to a spoon. The abnormality is more apparent when the nail is viewed laterally and is more obvious in the thumb. A spoon nail may be physiologic in infants and children, especially in the nails of the large toes. This nail change has classically been associated with iron deficiency anemia, but it may also be associated with other conditions, including trauma from nail biting or chemical solvents, blue-and-white fingers on cold exposure from Raynaud's disease or

Figure 9.
PITTING

Figure 10.
BEAU'S LINE

Figure 11.
SPOON NAIL

associated collagen vascular disorders such as Raynaud's phenomena, and thyroid disease.[6] Keratin is a protein composed of amino acids, and spooning has been traced to a lower level of the amino acid cysteine in the affected nails. If the underlying condition is treated, the nail changes may take up to 6 months to reverse (Figure 11).

NAIL FOLD ABNORMALITIES

Paronychia: Paronychia is inflammation of the nail fold often caused by localized, superficial infections or abscesses of the area around the nail or the epidermis bordering the nail. Acute paronychia is commonly associated with nail biting, aggressive manicuring, artificial nail placement, and trauma. The paronychial area is usually erythematous and tender, and the nail may appear discolored and distorted. Development of an abscess requires incision and drainage as well as an appropriate oral antibiotic such as amoxicillin with clavulanic acid to cover aerobes (gram positive and gram negative) and anaerobes.[13] The most common organism is *Staphylococcus aureus*, but beta-hemolytic *streptococci* or, occasionally, organisms such as *Pseudomonas* may be involved. A bacterial culture swab should be obtained to ensure that the pathogen is sensitive to empiric antibiotic therapy (Figure 12).

Chronic paronychia (Figure 13) is related to contact irritants or alkali and prolonged moisture exposure. Individuals such as cooks, bartenders, custodians, janitors, health care professionals, and patients with diabetes are at risk for chronic paronychia.[14] The affected nail fold becomes swollen and is lifted above the nail. The nail plate becomes thickened and distorted with pronounced transverse ridges. Various microbes, especially *Candida albicans*, can be found in the space between the nail plate and cuticles or nail folds. Treating chronic paronychia starts with good nail care. The cuticles

Figure 12.
ACUTE NAIL INFECTION

Figure 13.
CHRONIC PARONYCHIA WITH MUCOID CYST

should never be pushed back, and cotton gloves should be worn when handling contact irritants. The cotton gloves can be placed inside rubber or vinyl gloves when the hands are exposed to water. The treatment protocol typically involves using a combination of topical steroids and an antifungal.[14] If secondary infection is a concern, a topical antimicrobial agent or oral antibiotic is indicated.

Pterygium: Pterygium is an adhesion of the proximal nail fold to the proximal nailbed after inflammation destroys the nail matrix. When the damaged nail matrix can no longer generate the nail plate, the proximal nail fold fuses with the nailbed, and they grow out to form a permanent, wing-like deformity.[7] Pterygium usually occurs with lichen planus or impaired circulation.

Mucous cyst: Mucous or myxoid cysts arise from degeneration in the connective tissue over the matrix area of the nail and can cause nail dystrophy. Typically, they present as an asymptomatic translucent nodule on the dorsum of the digit that contains a clear gel. Transillumination and extraction of gelatinous material is diagnostic. Treatment includes cryotherapy, simple drainage, photocoagulation, repeated puncture, and surgery, but recurrence is common (Figure 13).[15]

NAIL INFECTIONS

Herpetic whitlow: Herpetic whitlow results from autoinoculation of type 1 or type 2 herpes simplex virus in the broken skin. It is occasionally seen in health care professionals who are exposed to oral secretions, if universal precautions were breached.[16] Patients with herpetic whitlow complain of pain that is out of proportion to the physical findings. Small clear vesicles present close to the nail with a honeycomb appearance and resolve with crusting. It usually takes 3 weeks to resolve, and relapse is common. A viral swab to confirm the diagnosis of herpes simplex helps direct treatment.

Onychomycosis: According to a study of 15,000 Canadian patients, 16.7% reported abnormal-looking nails, and 8% of the sample had mycologic evidence of toenail or fingernail onychomycosis.[17] Dermatophytes have the ability to invade keratinized tissue, and they accounted for 90.5% of positive cultures; nondermatophyte molds and *Candida* spp accounted for 7.8% and 1.7%, respectively. Individuals such as older adults, those with diabetes, and those with previous trauma to the nail apparatus are prone to developing onychomycosis. Onychomycosis occurs in 4 patterns: distal and lateral subungual onychomycosis (Figure 14); proximal subungual onychomycosis (Figure 15); superficial white onychomycosis (Figure 16); and total nail plate dystrophic onychomycosis (Figure 17).

Distal and lateral subungual onychomycosis is the most common infection affecting the distal portion of the nail. The infection is predominantly caused by *Trichophyton rubrum*. The hyponychium is involved initially with fungal organisms, resulting in local hyperkeratosis that eventually pushes the distal plate up (onycholysis). This is followed by linear streaks that spread back toward the matrix. Often, large toenails first experience asymmetric involvement that gradually spreads to other toenails and less commonly, to the fingernails. Most patients have maceration of the 4th and 5th toeweb (Figure 18).[5] The plantar skin may have a powdery white, dry accentuation of skin surface markings that

Figure 14.
DISTAL AND LATERAL SUBUNGUAL ONYCHOMYCOSIS

Figure 15.
PROXIMAL SUBUNGUAL ONYCHOMYCOSIS

eventually extends around the sides of the foot. This distinctive demarcation is often referred to as the moccasin foot. Check for involvement of the other foot and the hands. Fungal infection usually starts asymmetrically, and with time it can involve both the hands and feet.[5,18]

Proximal subungual onychomycosis is a rare fungal nail infection that is usually caused by *Trichophyton rubrum* invading the proximal portion of nail fold (cuticle and half moon area) and adjacent distal matrix. The distal matrix is responsible for the undersurface of the nail plate, and samples for culture must go through most of the nail plate to ensure an accurate sample for testing. This type is most common in immune-compromised persons and can be the presenting sign of HIV infection.[19]

Figure 16.
SUPERFICIAL WHITE ONYCHOMYCOSIS

Superficial white onychomycosis is mostly found on toenails and due to *Trichophyton mentagrophytes*. Colonies of fungus, yeast, or mold sit on the surface, producing chalky white patches and a crumbly surface without invading the deeper nail plate. This is the only type of fungal nail infection

Figure 17.
TOTAL NAIL PLATE DYSTROPHIC ONYCHOMYCOSIS

Figure 18.
MACERATION

that can be treated topically.[5] A scalpel blade scrapping of the surface colonies helps identify the causative organisms.

Total nail plate dystrophic onychomycosis is an infection of the entire nail. A weakened nail plate may be vulnerable to secondary invasion by yeasts and molds. Treatment addresses both the primary invading organism and potential secondary yeast or mold infection.[20]

Diagnosis of onychomycosis: Several nail disorders may mimic fungal nail infections, and clinicians must differentiate them to choose the appropriate treatment. In a study conducted by Fletcher et al,[21] 32% of nail samples had positive results on both direct examination and culture for fungi; 42% had negative results; and 20% were positive on direct microscopy but negative on culture. However, mycologic examination is the preferred method for diagnosing onychomycosis, despite false-negative culture rates of at least 30%. To ensure that a diagnosis of fungal infection is not missed, clinicians should make 3 culture attempts. Laboratory diagnosis includes a microscopic examination to visualize fungal elements and culture to identify the responsible organism.[20]

Proper specimen collection is essential to accurate diagnosis. The clipped nail, along with soft subungual debris that contains the majority of organisms, should be sent for culture (Figure 19).[5] For *Candida* infection, the material close to the lateral nail edges should be obtained. As much material as possible should be sent to the laboratory to increase the chance of detecting fungal material. A 20% solution of potassium hydroxide will be added to the material on a glass slide. After 15 to 20 minutes, the sample is ready for direct microscopy. The potassium hydroxide dissolves keratin leaving the resistant fungal hyphae.

Figure 19.
NAIL CLIPPINGS

Instruments and sterilization: Nail clippers and any instruments used for fungal nail clippings or skin scrapings of scale should be sterilized with steam and an indicator system for an adequate sterilization cycle. Scalpel blades can be disposable. For personal protection, nonlatex gloves should be worn, and universal precautions used. For nail sanding and drilling, protective eyewear and nose and mouth masks are advised.

Treatment: In recent decades, the Food and Drug Administration (FDA) has approved several treatments for skin infection and onychomycosis. Toenail treatment should not be recommended before mycologic confirmation of infection. The ideal topical antifungal agent should be fungicidal, require a short course, minimize relapses, be conducive to patient compliance, and have minimal adverse effects.

Topical agents (Table 1) cost less than oral agents and have minimal adverse effects. However, they must penetrate a compact layer of nail keratin to reach the organisms causing onychomycosis. Topical treatment is inferior to systemic therapy for nails, but may be indicated for white superficial onychomycosis and distal subungual onychomycosis limited to a few nails. To concentrate the drug on the nail surface, some agents such as amorolfine and ciclopirox are delivered in nail lacquer that is not easily removed by washing or wiping. The nail lacquers are broad-spectrum and provide fungicidal activity against dermatophytes, some nondermatophytes, and yeast. Ciclopirox 8% nail lacquer is used to treat mild to moderate onychomycosis, especially when caused by *Trichophyton rubrum*.[22] It is applied once a day for 8 to 12 months. An effective concentration of ciclopirox can be attained throughout the nail unit during active treatment; 14 days after treatment, the concentration cannot be detected. Amorolfine 5% nail lacquer is applied once or twice weekly to the affected nails for up to 6 months. Studies indicate that amorolfine not only penetrates into subungual debris but the drug concentration continues to be effective 2 weeks after treatment.[22]

Systemic treatment (Table 2) is indicated for proximal subungual onychomycosis and distal subungual onychomycosis involving several digits. Oral antifungals have cure rates of 80% to 90% for fingernail infections and 70% to 80% for toenail infections.[23]

Terbinafine belongs to the allylamines group and is fungicidal, whereas the azoles (ketoconazole, itraconazole, fluconazole) are fungistatic.[24] Terbinafine is superior to itraconazole for dermatophyte onychomycosis.[24] A meta-analysis of 11 trials indicated that allylamines were more efficacious than azoles but were much more expensive.[23] The success rate can be increased with combination therapy using local treatments. Recurrence, which is common, may be

Table 1.
TOPICAL ANTIFUNGAL AGENTS

Class	Examples	OTC	Spectrum	Application	Properties	Notes
Allylamines	• Terbinafine 1% (Lamisil) (30 g)	Yes (by prescription in Canada)	*Candida*, tinea vesicolor, dermatophytes	Once a day	Fungicidal	Clinically effective 90%
	• Naftifine hydrochloride (Naftin 1%) (15 g)	Yes				More efficacious than azoles in treating tinea pedis, tinea cruris, and tinea corporis
Imidazoles	• Clotrimazole (Canestan/Myclo)	Yes	*Candida*, tinea vesicolor, dermatophytes, limited gram-positive bacteria	BID 2 weeks for tinea cruris/ corporis, 4 weeks for tinea pedis	Fungistatic, anti-inflammatory	Clinically effective 70%–80%
	• Miconazole (Micatin/Monostat)					
	• Econazole (Ecostatin)					
	• Ketoconazole (Nizoral)					
	• Tioconazole (Troysd)					
Pyridone	• Ciclopirox (Penlac)	Yes	*Candida*, tinea vesicolor, dermatophytes, limited anti-*Pseudomonas* activity	Once a day	Fungicidal	Clinically effective 60%, available in lacquer for treatment of minor nail involvement (Penlac)
Iodinated tricholorphenols	• Haloprogin (Halotex)	Yes	*Candida*, tinea vesicolor, dermatophytes	BID	Unknown	Clinically effective 29%–35%
Miscellaneous	• Tolnaftate (Tinactin)	Yes	Dermatophytes	BID	Unknown	Inferior to azoles and allylamines in treating tinea pedis
Miscellaneous	• Selenium sulfate (Selsun/Versel)	Yes	Tinea vesicolor	BID	Unknown	Often drying
Short-chain fatty acid	• Undecylenic acid (Zeasorb/Desenex)	Yes	Prevention of *Candida* and tinea vesicolor	BID	Unknown	For prevention only

prevented with regular applications of topical antifungals on previously treated nails.[5]

Terbinafine, administered in a dosage of 250 mg daily for 12 weeks, clears 77% of toenail infections.[23] It is effective against dermatophytes and should not be used for nondermatophyte infection. Terbinafine should be used for 6 to 8 weeks for fingernail and 12 to 16 weeks for toenail involvement.[24] It may be associated with headache (13%), gastrointestinal intolerance (5%), skin rash

Table 2.
SYSTEMIC ANTIFUNGAL AGENTS

Class	Examples	Application	Drug interactions	Side effects	Notes
Allyamines	Terbinafine (Lamisil 250 mg)	Scalp: 250 mg once daily x 4–8 weeks	• Increased clearance with rifampicin	• GI upset	• Superior to azoles
		Fingernails: 250 mg x 6 weeks	• Decreased clearance with cimetidine	• Occasional skin rashes	
		Toenails: 250 mg x 12–16 weeks		• Temporary lost of taste	
Azoles	Itraconazole (Sporanox)	Scalp: 3–5 mg per kg per day for 30 days Nails: 200 mg BID x 1 week/month x 2 months or 200 mg q24 hours x 3 months	• Drugs especially metabolized by CYP3A4 (cisapride, dofetilide, ergot alkaloids, lovastatin, pimozide, and simvastatin, midazolam nisoldipine, quinidine, and triazolam) are contraindicated	• Rare hepatotoxicity • Variable absorption • Drug resistance	• Similar cure rates against *Candida*, not aspergillus
	Ketoconazole (Nizoral 200 mg)	Scalp: 200 mg once daily x 2 wk after clear Nails: 200 mg x 18 mo	• HMG CoA- reductase inhibitors (lovastatin and simvastatin) and ergot alkaloids are contraindicated	• Rare hepatotoxicity • Variable absorption • Drug resistance	• Poor penetration into CNS • Inhibits synthesis of cortisol and testosterone

(1%–2%), loss of taste (1 in 800), and a transient decrease in lymphocyte count.[25] Itraconazole pulse therapy is administered at a dosage of 200 mg twice a day for 1 week a month for 2 cycles for fingernail infection and 3 to 4 cycles for toenail infection. It is recommended as second-line therapy.[22] Intermittent or pulse therapy reduces the cost of systemic treatment as well as total drug exposure. Other options are outlined in Table 1.

SUMMARY

Nail disorders are common, but only a small percentage result from fungal infection. Thus, practitioners should not use oral antifungal agents without microscopic or cultural confirmation of a fungal infection. Fungal infection of the nails and skin can represent a disruption of the cutaneous barrier that leads to secondary bacterial infection in chronic wounds. Several common nail disorders provide cutaneous clues to systemic disease and may be associated with local trauma or physiologic changes.

CASE 1 CONCLUSION

Infection occurs when an imbalance exists between organism proliferation and host resistance. John has diabetes, which produces immune deficiency, and he has atherosclerosis, which is provoked by smoking. His moist hands, which are related to his job, predispose him to fungal infection. The fungal smear confirmed the presence of *Trichophyton rubrum*, and a culture of a scraping and nail clipping identified the fungus. John was given recommendations for improving glucose control, trying to stop smoking, and avoiding continuous moisture. After 12 months of therapy with oral terbinafine and topical antifungal treatments, healing was complete.

CASE 2 CONCLUSION

Clinicians are continuously faced with the dilemma of accurately diagnosing and treating chronic nail disorders. David is concerned about his chronic nail changes because they interfere with his social life. He has tried different antifungal creams and treatments, and he has had 2 negative scrapings for fungus. Additional history revealed skin lesions and dactylitis. David is suffering from chronic psoriasis, and his nail involvement is part of his systemic disease, psoriatic arthritis. Such involvement occurs in about 20% of affected persons.

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