Acute and Chronic Paronychia of the Hand

Abstract

Acute and chronic infections and inflammation adjacent to the fingernail, or paronychia, are common. Paronychia typically develops following a breakdown in the barrier between the nail plate and the adjacent nail fold and is often caused by bacterial or fungal pathogens; however, noninfectious etiologies, such as chemical irritants, excessive moisture, systemic conditions, and medications, can cause nail changes. Abscesses associated with acute infections may spontaneously decompress or may require drainage and local wound care along with a short course of appropriate antibiotics. Chronic infections have a multifactorial etiology and can lead to nail changes, including thickening, ridging, and discoloration. Large, prospective studies are needed to identify the best treatment regimen for acute and chronic paronychia.

Inflammation of the tissue immediately surrounding the nail, known as paronychia, is commonly caused by acute or chronic infection. Paronychia can be acute (<6 weeks duration) or chronic (≥6 weeks duration) and typically develops following a breakdown in the barrier between the nail plate and the adjacent nail fold that is often caused by bacterial or fungal pathogens. However, noninfectious etiologies such as chemical irritants, excessive moisture, systemic conditions, and medications also can cause paronychia. Management options include activity modification along with medical and/or surgical intervention based on the etiology, duration, extent of paronychial involvement, and the associated risk factors present.1,2

Anatomy

The tip of the finger is composed of osseous tissue, soft tissue, and specialized tissues that produce and support the nail distal to the insertions of the flexor and extensor tendons.3 Fibrous septa located within the pulp of the finger stabilize the vascular fibrofatty tissue and bridge the dermis to the periosteum of the distal phalanx. The nail bed, which has a convoluted attachment to the periosteum of the distal phalanx, resists traumatic avulsion. In humans, the fingernail protects the fingertip and enhances its dexterity and sensation by exerting counterpressure for the volar pulp during touch and facilitating skilled hand function, such as the ability to pick up and manipulate small objects.5

The nailbed comprises germinal and sterile matrices, with the germinal matrix located on the palmar aspect of the nail fold and terminating at the distal extent of the lunula. This matrix is more vascular than the remainder of the nail bed and produces nearly all of the nail via gradient parakeratosis.4 Near the periosteum, germinal matrix cells originate as basilar cells. They duplicate and are driven dorsally in columns toward the nail. The cells
flatten and stream distally when they meet the resistance of the nail, leading to longitudinal nail growth. The nail bed and the nail plate are involved in the continuum of nail production at all stages.

The sterile matrix lies distal to the lunula. Its contribution to nail production varies. Cells that originate from the sterile matrix enlarge, flatten, and elongate; large cells eventually break down and are incorporated into the nail. In most people, the nail is thicker distally than proximally, providing evidence of the contribution of the sterile matrix to nail production.

The nail plate is anchored to the underlying linear ridges in the squamous epithelium of the sterile matrix. The nail adheres less to the germinal matrix than to the sterile matrix.

The paronychium is defined as the soft tissue lateral to the nail bed, whereas the term perionychium refers to the paronychium and nail bed (Figure 1). Primate studies suggest that after nail removal, the sterile matrix contributes little to nail regeneration and the nail is primarily reformed by the paronychium. The junction where the sterile matrix of the distal nail bed meets the skin of the fingertip is called the hyponychium. A keratinous plug with abundant neutrophils and lymphocytes composes the hyponychium, which serves as a barrier in preventing microbial invasion of the subungual area.

Acute Paronychia

Etiology and Risk Factors

Most acute paronychias are the result of minor trauma to the nail bed that is often related to onychophagia (ie, nail biting), finger sucking, picking at a hangnail, an ingrown nail, manicures, dishwashing, or puncture-type trauma with or without a retained foreign body. Such trauma disrupts the fingertip’s natural barrier to outside pathogens, resulting in inoculation of the perionychium. In three studies with a total of 61 patients with paronychia, approximately 25% of paronychias were caused by anaerobic bacteria, 25% by aerobic bacteria, and 50% by mixed aerobic and anaerobic bacteria.

The most common aerobic pathogens responsible for acute paronychia include Staphylococcus aureus, gamma-hemolytic streptococci, Eikenella corrodens, group A B-hemolytic streptococci, and Klebsiella pneumoniae. Common anaerobic bacteria responsible for paronychia include Bacteroides species, gram-positive anaerobic cocci, and Fusobacteria species. Enterococcus faecalis, Proteus species, and Pseudomonas aeruginosa are other isolated organisms that can cause paronychia. In addition, nonbacterial pathogens such as yeast (Candida albicans) and viruses (eg, herpes simplex) have been identified as causative organisms. A specific trauma or inciting event may not be identified in all cases of acute paronychia.
Clinical Presentation

Patients with acute paronychias typically present with localized pain, redness, inflammation, and edema of the paronychium that is typically limited to a single digit. The timing of presentation varies, but is often 2 to 5 days after the initial trauma. Fluctuance of the paronychium may not be observed with early presentation. In patients with delayed presentation, fluctuance may extend around the nail, involving the eponychium as well as the paronychium on both the radial and ulnar sides of the digit (ie, runaround infection). Purulence may develop underneath the nail plate, causing the nail plate to pull away from the sterile matrix; this may be more accurately described as a peri-onychial infection (Figure 2).

Diagnosis

Diagnosis of acute paronychia is based on the patient’s history and physical examination. A detailed history is crucial for evaluation of risk factors that may be associated with an atypical causative organism. For example, contact with oral secretions may provide exposure to specific anaerobic bacteria such as *Eikenella corrodens* or the herpes virus. Exposure to animals may result in an increased risk of infection with gram-negative organisms such as *Pasteurella multocida*.

Turkmen et al described the use of a digital pressure test to identify the presence and extent of paronychial abscesses. The test is performed by applying light pressure to the distal volar aspect of the affected digit and observing for blanching in the area of the paronychia (Figure 3). Blanching may indicate the presence of an abscess. Typically, radiographs and laboratory tests are not needed for diagnosis of acute paronychia.

Differential Diagnosis

Although gram-positive bacterial infections account for most cases of acute paronychia, a wide differential should be considered. Herpetic whitlow is a manifestation of herpes simplex infection and presents as one or more blisters grouped on the distal aspect of the digit (Figure 4). The blisters are typically filled with serous-type fluid, but the fluid may be more opaque and can be easily mistaken for purulence. Herpetic whitlow is often seen in healthcare professionals (eg, dental professionals) who are at risk of topical exposure to the virus, but the condition may also be seen in persons with a primary herpes simplex infection. A definitive diagnosis is made based on Tzanck smear or viral culture results. Incision and drainage are contraindicated.

In addition, to herpetic whitlow, other conditions such as psoriasis, Reiter syndrome, and pemphigus vulgaris can mimic acute or chronic paronychia. Medications such as retinoids, antiretrovirals, and chemotherapeutics can cause paronychial inflammation, as well.

Nonsurgical Management

Management of paronychia depends largely on the amount of inflammation and whether an abscess is present. In patients who present with a minimal amount of inflammation and no abscess formation, frequent soaks with warm water, aluminum acetate (Burrow solution), vinegar, a dilute povidone-iodine solution or chlorhexidine may be sufficient. However, no studies have...

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**Figure 2**
A, Photograph of a fingertip demonstrating an acute paronychia and its sequelae. The patient presented with acute onset of pain and swelling. The abscess spontaneously decompressed under the nail fold and nail plate. B, Photograph of the fingertip obtained 3 weeks later. The infection resolved and a new nail is growing to replace the one present at the time of infection.

**Figure 3**
Photograph of a fingertip demonstrating an abscess, which is evident from the blanched area caused by simple digital pinch pressure. (Courtesy of Robert Strauch, MD, New York, NY.)

**Figure 4**
Photograph of a fingertip demonstrating herpetic whitlow. (Reproduced with permission from Usatine RP, Titinigan R: Nongenital herpes simplex virus. *Am Fam Physician* 2010;82(9):1075-1082.)
evaluated the effectiveness of soaks alone.

A topical antibiotic may be added to the treatment regimen in patients with minimal erythema and no abscess formation. Topical and/or oral antibiotics should be used in patients with substantial erythema and abscess formation. Topical antibiotics may be used alone or in combination with a corticosteroid. Wollina\textsuperscript{17} compared the efficacy of fusidic acid and betamethasone versus gentamycin ointment for acute paryonychia in a nonblinded study. Erythema, swelling, exudation, and pain were graded on a scale of 0 (absent) to 3 (heavy). The author reported a 50\% reduction in pain in the fusidic acid and betamethasone group after 3.5 ± 2.0 days compared with a 50\% reduction in pain after 5.1 ± 3.1 days in the gentamycin group. Both regimens were effective and had no associated complications.

Oral antibiotic regimens such as trimethoprim-sulfamethoxazole, cephalixin, amoxicillin and clavulanate, or clindamycin (in patients with sensitivity to penicillins) should provide coverage against gram-positive organisms, including \textit{S. aureus} and streptococci.\textsuperscript{1} Tosti and Ilyas\textsuperscript{18} recommend the use of agents that are effective against methicillin-resistant \textit{S. aureus}, such as oral trimethoprim-sulfamethoxazole, if this bacterium has been documented in >10\% of community-acquired hand infections at a given institution. In the setting of suspected infection with oral flora, broad-spectrum antibiotics such as amoxicillin/clavulanate or clindamycin should be used to provide coverage against anaerobic bacteria.\textsuperscript{1}

**Surgical Management**

In general, surgical management of acute paronychia is reserved for patients with a discrete abscess, failure of nonsurgical care, and/or extensive involvement of the eponychium. Numerous surgical techniques have been described for management of acute paronychia, and each technique has a role based on the structures affected and the extent of involvement. To our knowledge, no studies have compared the efficacy of administering oral antibiotics alone versus drainage.\textsuperscript{19} Some authors recommend a course of oral antibiotics after drainage,\textsuperscript{20} whereas others recommend drainage and local wound care alone.\textsuperscript{2} No studies have compared the efficacy of administering oral antibiotics after drainage with that of drainage and wound care alone.

To drain an abscess, a No. 11 or 15 scalpel blade (with sharp edge pointed away from the nail), a freer elevator, or a small hemostat is inserted into the nail sulcus and beneath the nail fold until the abscess is decompressed (Figure 5). This obviates the need to create a skin incision in the lateral nail fold, which can put the intervening tissue at risk for skin bridge necrosis.\textsuperscript{21} However, this treatment option requires that the abscess be immediately adjacent to the nail sulcus. A piece of mesh gauze may be placed beneath the nail fold to allow for continued drainage.\textsuperscript{21}

Ogunlusi et al\textsuperscript{22} described a more limited approach for draining an abscess. The tip of a 21- or 23-gauge needle is used to lift the nail fold, allowing egress of purulence. Drainage is followed by oral antibiotic therapy. The authors used this technique in 8 patients with 10 paronychias and noted resolution of acute paronychia in all patients after 2 days. The authors concluded that neither anesthesia nor daily dressing changes were required with this technique.

Extensive abscesses or those not immediately adjacent to the nail sulcus may require the creation of skin incisions to promote drainage. A small incision made in the paronychium directly over the abscess can facilitate drainage (Figure 6). The sharp edge of the blade should be pointed away from the nail to avoid injury to the matrix, which can lead to subsequent nail deformity. Additionally, a longitudinal incision can be made in line with the lateral nail fold to decompress the abscess.\textsuperscript{15} In the setting of eponychia or runaround infection, longitudinal incisions can be made on both sides of the nail. If incisions are made in line with the nail folds, the fold can be reflected proximally, irrigated, and returned to its original position (Figure 7). A piece of gauze may be placed under the nail fold to facilitate continued drainage. In cases in which the abscess has spread beneath the nail plate to form a subungual abscess, partial or complete removal of the nail plate may be indicated, particularly if the paronychia is related to an ingrown nail. Complete removal of the nail plate is reserved for cases in which spreading infection has undermined the entire plate, causing complete separation of the nail plate from the underlying sterile matrix.

![Figure 5](https://example.com/figure5.png)

**Figure 5**

Illustrations demonstrating decompression of an abscess using a blade (A) or an elevator (B). The paronychia is elevated from the nail via the nail sulcus with care taken to avoid injury to the nail bed.
Treatment failure and recurrence are uncommon after appropriate management of acute paronychia. Failure to recognize a significant abscess or incomplete drainage or débridement may result in persistent or recurrent infection. Local factors such as the extent of infection or host factors (eg, diabetes mellitus, other immunocompromised states) may play a role in impaired clearance of infection; however, no studies have demonstrated the role of host factors with respect to acute paronychia. Several studies cite treatment failure as a risk factor for the development of chronic paronychia, but sparse evidence has been reported with regard to factors that contribute to failed management of acute paronychia. To our knowledge, no evidence exists to suggest that improper management of paronychial infection leads to felon or osteomyelitis.

**Chronic Paronychia**

**Etiology and Risk Factors**

Chronic paronychia is inflammation of the perionychium that has been present for >6 weeks. This inflammation can have many causes and often is related to repeated exposure to environmental irritants, with colonization by fungal or bacterial pathogens that occurs after disruption of the barrier formed by the eponychium and nail vest. Exposure to irritants can take many forms and persons with a higher risk of chronic paronychia include those with frequent exposure to moisture and/or chemical irritants. Homemakers, bartenders, barbers, dishwashers, cooks, food handlers, swimmers, and nurses are commonly identified as having an increased risk of chronic paronychia. Conditions such as diabetes mellitus and immunosuppression also predispose patients to development of chronic paronychia. *C. albicans* is a pathogen commonly associated with chronic paronychia; this fungus has been found in cultures in 40% to 95% of cases. The exact role that it plays in the development and maintenance of chronic...
paronychia is unclear. The presence of Candida may represent a secondary colonization of the nail fold that can contribute to the development of chronic paronychia by inducing an additional persistent inflammatory response. In a study of chronic paronychia, Stone and Mullins soaked fingers in water until they were macerated and then inoculated the perionychium with either viable or nonviable Candida. Inflammatory conditions similar to chronic paronychia developed in both groups, demonstrating the inflammatory effect caused by the pathogen. Tosti et al compared the use of topical steroids versus systemic antifungals for management of chronic paronychia and found that the patients treated with topical corticosteroid alone showed more clinical improvement than those treated with antifungal agents alone. Eradication of Candida was associated with cure in only 2 of 18 patients who tested positive for infection at the outset of the study. The authors concluded that, in patients with chronic paronychia, Candida is a secondary colonizer of the nail fold, and chronic paronychia is an inflammatory disorder rather than a primary mycotic infection.

**Clinical Presentation and Diagnosis**

Typically, a thorough history and physical examination is sufficient to diagnose chronic paronychia. The patient’s history typically reveals exposure to one or more risk factors. Chronic paronychia presents with erythema, swelling, and pain, although the degree of erythema and swelling is often less than that associated with acute paronychia. In general, symptoms are present for >6 weeks at the time of diagnosis. Epidemic exacerbation of symptoms can occur, and such episodes may follow exposure to moist environments. The proximal nail fold may become raised and separated from the underlying nail. Chronic paronychia may have associated nail changes including ridging, grooving, discoloration, and/or rounding of the nail plate (Figure 8). Further diagnostic testing may be warranted in atypical cases in which malignancy or systemic etiologies are suspected.

**Differential Diagnosis**

Certain antiretroviral and chemotherapeutic medications have been implicated in the development of acute and chronic paronychia. Anti-retroviral medications (eg, indinavir, lamivudine), have been associated with the development of paronychia and periungual pyogenic granulomas. The toes are often involved but finger involvement has been described, as well. The similarity between the cutaneous side effects associated with protease inhibitors and those associated with retinoid-based therapies have led to the theory that protease inhibitors alter retinoid metabolism, resulting in the aforementioned cutaneous side effects. Toma et al found that indinavir and several other protease inhibitors can significantly increase plasma retinoic acid concentrations. Some of the proposed mechanisms include enhanced conversion of retinol to retinoic acid caused by an indinavir-mediated increase in the activity and/or expression of retinal dehydrogenase, inhibition of cytochrome P450-mediated catabolism of retinoic acid, and/or increased activity of retinoid-responsive gene products. Anti-epidermal growth factor receptor (EGFR) chemotherapeutic agents (eg, cetuximab, gefitinib, lapatinib) have been associated with the development of paronychia, as well. The inhibition of EGFR by anti-EGFR agents has been implicated in the development of chronic paronychia.

Malignancies of the periungual region and paraneoplastic conditions may mimic acute or chronic paronychia. Several conditions cause a paronychia-like presentation, including squamous cell carcinoma, melanoma, Kaposi sarcoma, digital papillary adenocarcinoma, myeloma-associated systemic amyloidosis, bronchogenic carcinoma, renal cell carcinoma, subungual keratoacanthoma, and leukemia cutis. These diagnoses should be considered in patients who present with signs and symptoms of paronychia, particularly in those with
recalcitrant paronychia or those with a history of cancer.

**Nonsurgical Management**

The first step in management of chronic paronychia is avoiding irritants and moisture. Topical and systemic therapies can be used, as well. Tosti et al performed a double-blind randomized controlled trial to compare the effectiveness of systemic antifungal medications (250 mg of terbinafine daily or 200 mg of itraconazole daily) with that of a topical corticosteroid (0.1% 5 mg of methylprednisolone aceponate daily). A placebo was provided for each group. The treatment period lasted 3 weeks. At 3 weeks, the authors noted a significant increase in the clinical cure rate of the topical corticosteroid group compared with that of the antifungal group ($P < 0.01$). The authors reported improved or cured paronychia in 42 of 48 nails (87.5%) in the corticosteroid group compared with 22 of 64 nails (34.4%) in the itraconazole group and 19 of 57 nails (33.3%) in the terbinafine group. Tosti et al concluded that topical corticosteroid therapy should be used as a first-line treatment for chronic paronychia. They also recommended that chronic paronychial infection should be regarded as an inflammatory disorder of the nail fold rather than an onychomycosis.

In a study of 17 patients with chronic paronychia, Daniel et al reported good results with a combined irritant avoidance regimen and topical application of a 0.77% ciclopirox suspension, a broad-spectrum antifungal with anti-inflammatory properties, for 6 to 12 weeks. Chronic paronychia resolved in all 17 patients. The authors recommended management of primary factors (eg, exposure, irritants, inflammation) and secondary fungal colonization to reduce recurrence and avoid treatment failure caused by the inflammatory effects of secondary fungal colonization. The addition of a topical antifungal agent to topical corticosteroid therapy has been described; however, the use of a topical antifungal and corticosteroid has not been shown to be superior to the use of a topical corticosteroid alone.

In a study of 45 patients with chronic paronychia, Rigopoulos et al compared the safety and efficacy of twice-daily application of 0.1% betamethasone 17-valerate ointment with application of 0.1% tacrolimus ointment or an emollient over a 3-week period. Both the betamethasone and tacrolimus groups demonstrated statistically significant improvement ($P < 0.001$) in cure or improvement rate versus the emollient group, with tacrolimus therapy demonstrating the highest efficacy. Thus, a 1- to 2-week course of 0.05% betamethasone cream or 0.1% betamethasone solution or lotion has been recommended for management of chronic paronychia. For refractory cases of chronic paronychia, some authors have recommend a trial of a systemic antifungal before proceeding with an invasive procedure. In an earlier study on paronychia, Rigopoulos et al described the use of a short course of systemic corticosteroids for patients with severe involvement of multiple fingers.

**Surgical Management**

Surgical management of chronic paronychia is typically reserved for refractory cases. Several surgical techniques have been described and involve excision or elevation of the involved tissue of the eponychium. Chronic inflammation of the eponychium leads to progressive fibrosis, edema, induration, and rounding of the cuticle border, which act together to compromise the natural barrier function and impair blood flow to the affected tissues, making spontaneous healing difficult.

In 1976, Keyser and Eaton described the use of eponychial marsupialization for management of chronic paronychia. The original technique involved excision of a crescent-shaped area of the dorsal aspect of the proximal nail fold without concomitant nail removal. The excision area begins 1 mm from the distal border of the eponychium and extends approximately 6 mm proximally and from one lateral nail fold to the other to include all inflamed tissue. Excision is followed by hydrogen peroxide soaks and dressing changes until reepithelialization occurs (typically within 2 weeks). The authors noted excellent results with this technique, with chronic paronychia cured in 28 of 31 digits. The exact mechanism by which marsupialization promotes healing is not well understood.

In 1981, Baran and Bureauc described a technique that involved en bloc excision of the proximal nail fold without nail plate removal. A 5- to 6-mm-wide section of involved eponychial tissue spanning from one lateral nail fold to the other was excised and, in contrast to marsupialization, a distal rim of tissue was not spared. After en bloc excision, postoperative care consisted of dressing changes and application of a topical antibiotic preparation. The proposed benefit is a simpler and more effective technique than marsupialization with concomitant nail plate removal that provides satisfactory functional and cosmetic results. However, objective outcome data were not provided in the study.

In a long-term study of 25 patients (28 fingers) with chronic paronychia treated with eponychial marsupialization with or without nail removal, Bednar and Lane reported better results in the group that underwent simultaneous removal of the nail plate (Figure 9). Of the 23 patients with nail irregularities, the first seven were treated with eponychial marsupialization without nail removal. Two of
these patients had a recurrence. The remaining 16 patients underwent marsupialization with nail removal, with no recurrence reported ($P < 0.05$). This technique differed slightly from the technique described by Keyser and Eaton in that a 3-mm crescent of tissue was excised and the underlying fat and subcutaneous tissue were left in place. In addition to marsupialization, all patients were treated with hydrogen peroxide soaks followed by cleansing with chlorhexidine gluconate and, in patients with positive cultures, oral antibiotics for 14 days or until cultures were negative. Grover et al. examined the effectiveness of en bloc excision of the proximal nail fold with and without nail removal for management of chronic paronychia. En bloc excision of the proximal nail fold was performed followed by a 5- to 7-day course of oral antibiotics as well as daily cleansing with antibiotic solution and application of topical antibacterial, antifungal, and corticosteroid creams. Of 30 patients, 12 in group I (en bloc excision without nail removal) and 13 in group II (en bloc excision with nail removal) completed the treatment protocol. The authors reported that 70% of patients in group II were cured versus only 41% in group I.

Pabari et al. described the use of the Swiss roll technique for management of both acute and chronic paronychias with runaround infection of both nail folds. The eponychium is elevated by making an incision on both sides of the nail fold and then the eponychium is reflected proximally. The wound is irrigated and the fold is rolled back over a roll of nonadherent gauze dressing and then anchored in place with nonabsorbable sutures (Figure 10). Postoperative application...
of topical medications, either antibiotics or steroids, was not described. The dressing and anchoring sutures were removed 2 to 7 days postoperatively. The nail fold was allowed to return to its original position and heal by secondary intention. Proposed benefits of this technique include the ability to retain the nail and to avoid creating a skin defect. The authors achieved “excellent cure rates” with this technique.

With regard to treatment failure or recurrence of symptoms after treatment, a paucity of data are available. This is partly due to variability in reporting outcomes as improved versus cured. In a study of patients treated with marsupialization with or without nail removal, Bednar and Lane\(^5\) reported two recurrences in the group that did not undergo nail removal. Both recurrences were treated with marsupialization with nail removal. One went on to cure and the other had persistent nail deformity and tenderness. No recurrences or treatment failures were reported in a group treated with marsupialization with nail removal. Daniel et al\(^45\) treated simple chronic paronychia with a combined regimen of ciclopirox and irritant avoidance and reported a cure rate of 100%. Although the authors found that the use of a topical antifungal was effective for preventing recurrence of chronic paronychia, no control group was available for comparison.

Studies have emphasized irritant avoidance as a key factor in achieving satisfactory results in management of chronic paronychia.\(^1,2,14,15,21,45\) When patients are unable or unwilling to comply with this recommendation, treatment failure is likely. However, a thorough evaluation should be performed in recalcitrant cases of chronic paronychia to rule out atypical etiologies such as the use of specific medications (eg, antiretrovirals) or malignancies.

Summary

Acute and chronic paronychias of the hand caused by infections are common. Acute paronychia typically indicates an acute bacterial infection. Cases without an associated abscess often can be treated successfully with an oral antibiotic regimen and soaks. The presence of an abscess is an indication for surgical drainage, which can be accomplished through various techniques based on the extent of infection, presence of an ingrown nail or subungual abscess, and surgeon preference. Further research is needed to determine whether oral antibiotic therapy is needed in addition to surgical drainage. Therefore, the decision to prescribe antibiotic therapy after drainage is based on clinical judgment and the extent of infection.

Chronic paronychias represent persistent inflammation of the nail fold that is multifactorial in origin. Exposure to environmental and occupational irritants appears to be the primary cause of this clinical entity, with secondary fungal colonization common and a likely contributor to ongoing inflammation. Management of chronic paronychia begins with avoidance of irritants and topical anti-inflammatory medication. The addition of topical antifungal agents has been recommended; however, this is controversial.

Surgical management is reserved for refractory cases of paronychia. Several surgical procedures have been described, with good to excellent results reported. Nail removal in conjunction with tissue excision seems to portend better results. Larger, prospective studies are needed to identify a treatment regimen that is clearly superior for management of acute and chronic paronychias.

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References

Evidence-based Medicine: Levels of evidence are described in the table of contents. In this article, references 17, 24, 45, 46, and 51 are level II studies. References 23 and 25 are level III studies. References 48 and 49 are level IV studies. References 1, 8, 11, 14, 15, 18-22, 50, and 52 are level V expert opinion.

References printed in bold type are those published within the past 5 years.

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