Evaluating the Efficacy of a Herbal Preparation of *Glycyrrhiza glabra* for Topical Treatment of Paronychia

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(Received: July 14, 2025; Accepted: January 28, 2026; Published (web): June 25, 2026)

ABSTRACT: Paronychia is a common tropical infection affecting the periungual skin, often involving bacterial and fungal pathogens, such as *Staphylococcus aureus*, *Pseudomonas* spp. and *Candida albicans*. This study evaluated *in vitro* antimicrobial efficacy of kenicide, a herbal formulation of *Glycyrrhiza glabra*, as a potential topical alternative to conventional antibiotics. Among various extraction methods, the glacial acetic acid extract exhibited superior antimicrobial activity against reference and clinical isolates. All extracts demonstrated hemolytic activity, with the ethanol extract showing the highest toxicity. Cytotoxicity assays in *Artemia* cysts revealed an LD_{50} of 4.88% (w/v), indicating notable toxicity. Given these cytotoxic and hemolytic effects, Kenicide appears suitable only for topical use. Further *in vivo* safety evaluations in animal models are warranted before clinical application.

Key words: Antimicrobial activity, cytotoxicity, *Glycyrrhiza glabra*, paronychia.

INTRODUCTION

Paronychia, a common but often neglected tropical disease, poses significant challenges to individuals in developing and developed countries. Characterized by inflammation of the periungual skin, specifically the nail folds, the disease leads to significant discomfort and functional impairment. It often begins with minor trauma, such as nail biting, manicuring, or occupational injuries that compromise the skin barrier, thereby facilitating pathogen entry. The condition is commonly associated with polymicrobial infections, involving both fungi and bacteria. *Candida albicans* is often implicated in chronic cases, while acute infections are typically driven by *Staphylococcus aureus* and *Pseudomonas* spp., resulting in inflammation, swelling, pain and purulent discharge, collectively known as onychomycosis.[1,2] If left untreated, paronychia may progress to abscess formation, nail deformity, or even permanent nail loss.

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Dhaka Univ. J. Pharm. Sci. **25**(1): 29-37, 2026 (June)
DOI: https://doi.org/10.3329/dujps.v25i1.91121

While acute cases are generally manageable with conservative interventions such as warm compresses and short-term antibiotics, chronic paronychia remains a therapeutic challenge. Recurrent and resistant to conventional treatments, chronic forms are particularly debilitating, often requiring prolonged medical attention, including surgical interventions with inconsistent outcomes. Epidemiological studies have consistently cultured *Candida albicans* from a significant proportion of chronic paronychia cases, reinforcing the need for more effective antifungal strategies.[3]

In resource-limited settings like Bangladesh, the impact of paronychia is especially pronounced. It disproportionately affects manual laborers, domestic workers, and others exposed to repetitive hand or foot trauma, often without access to protective gear or adequate medical care. For these vulnerable populations, standard treatment options, including broad-spectrum antibiotics or surgical debridement are either inaccessible, unaffordable, or insufficient, frequently resulting in recurrence and chronic morbidity.

In light of these limitations, the development of accessible, safe, and effective alternatives is of urgent importance. This study investigates the therapeutic potential of Kenicide, a potential topical alternative to conventional antibiotics in the treatment of paronychia. This product was produced as a solvent-based extract of powdered plant materials of *Glycyrrhiza glabra*, commonly known as licorice, that incorporates a blend of therapeutically active phytochemicals naturally present in licorice. These include glycyrrhizin, glabridin, liquiritigenin and related flavonoids, which have been widely documented for their antimicrobial, anti-inflammatory and immunomodulatory activities.[4] The powdered preparation served as the base material, from which we generated aqueous, ethanol and glacial acetic acid extracts for downstream antimicrobial and safety testing. Since the study focused on evaluating the biological activity of the plant-derived preparation rather than formulating or modifying its components, the powdered Kenicide was used, similar to other studies, assessing crude plant extracts for therapeutic feasibility.

This study aims to evaluate the *in vitro* antimicrobial activity of Kenicide against reference strains and clinical isolates of paronychia-causing organisms. Additionally, it assesses the haemolytic and cytotoxic profiles of its various extracts to determine safety and therapeutic feasibility. By employing a range of microbiological and molecular tools, we seek to bridge the gap between empirical herbal practices and evidence-based therapeutics, ultimately contributing to the development of a sustainable, non-invasive, and low-cost solution for managing a neglected yet impactful disease.

MATERIALS AND METHODS

Sample collection. Ethical clearance from the Faculty of Biological Sciences, University of Dhaka (reference number 154/ Biol. Scs., dated 28 Nov 2021) and informed consent from participating individuals were obtained at the onset of the study. Dead skin and pus samples (n=8) were collected by trained clinicians from the infected nails of clinically suspected paronychia patients. These patients exhibited symptoms such as pain, swelling, tenderness around the nail, redness of the skin, and pus buildup under the skin around the nail. Patients were recruited from Companigonj Upazila, Noakhali, Bangladesh.

Preparation of plant extract for antimicrobial assay and toxicity assay of Kenicide. Kenicide, as referred to throughout this study, denotes the powdered preparation of *Glycyrrhiza glabra* (licorice), which was used as the starting material for solvent extraction rather than a formulated or commercial drug product. A working solution of Kenicide was prepared using three extraction solvents: i) glacial acetic acid, ii) 95% ethanol and iii) water. Each was used at a ratio of 1:10 *G. glabra* to extraction solvent, where 10 g of *G. glabra* was mixed with 100 ml of each extractant. The solutions were heated at 60°C for 60 minutes and left in a shaking incubator for 24 hours at 37°C. The mixtures were centrifuged at 10,000 rpm for 5 minutes and the supernatants of each extraction were concentrated under 45-50°C using a rotary vacuum evaporator and then dried in an oven at 45°C. The concentrated extract obtained was a dried substance after extraction with distilled water and ethanol, while a thick sticky substance was obtained with glacial acetic acid.[5]

Three different 50% (w/v) Kenicide solutions were prepared with the corresponding extractant and were further diluted to 25, 12.5, 10 and 5% (w/v) using sterile distilled water for antimicrobial, hemolysis and cytotoxicity assays.

Antimicrobial activity test. The antimicrobial activity of Kenicide was tested on eight reference isolates, provided by the Fermentation and Enzyme Biotechnology Laboratory, Department of Microbiology, University of Dhaka. These included three Gram-positive bacteria: *Staphylococcus aureus, Bacillus cereus* and *Listeria monocytogenes* and four Gram-negative bacteria: *Pseudomonas* spp., *Vibrio cholerae, Salmonella enterica* and *Escherichia coli*, and a fungal isolate, *Candida albicans*.[6,7] Among the bacteria and fungi; *Staphylococcus aureus,*

Pseudomonas spp. and *Candida albicans* are established causative agents of paronychia. For this assay, concentrated Kenicide extracts were initially prepared as 50% (w/v) stock solutions using the corresponding extraction solvents. Serial dilutions were made to obtain working concentrations of 25, 12.5 and 10% (w/v).

For the antibacterial assay, the disc diffusion method was employed.[6] Briefly, pure cultures of bacteria were grown on LB broth and incubated at 37^{o}C overnight. Fifty microliters of this culture, diluted to a concentration of 0.5 McFarland turbidity, were spread over Mueller-Hinton agar plates. Discs of 4 mm diameter were prepared from Whatman filter paper No. 1 and sterilized. Thirty microliters of Kenicide solution (50, 25 and 12.5% w/v) were impregnated on 4 mm diameter sterile discs and air-dried. These discs were placed on Mueller-Hinton agar plates previously swabbed with the target bacterial isolate. This preparation was placed at 4^{o}C for 2 hrs for lateral diffusion before incubated overnight at 37^{o}C in an incubator (ESCO Lifesciences, Singapore). Antibacterial activity was estimated as the diameter (mm) of the clear inhibitory zone formed around discs.[8]

For testing the antifungal activity of Kenicide, *Candida albicans* was cultured in potato dextrose broth (PDB) and incubated at 25°C for 72 hrs. Once the cell density reached a concentration equivalent to the 0.5 McFarland standard, the fungal culture was used to assess the antifungal efficacy of Kenicide employing the previously described disc diffusion method. Sterile paper discs (4 mm diameter) were impregnated with 30 µl of each Kenicide solution and air-dried before placement on potato dextrose agar (PDA) plates previously inoculated with the standardized fungal suspension. Plates were incubated at 25°C for 24 hrs and the zones of inhibition were measured in millimeters (mm) across the entire clear zone, including the disc diameter. All assays were performed in triplicate. and the mean ± standard deviation (SD) of the inhibition zones were recorded. Ketoconazole (Himedia Labs, India) and Gentamicin (Oxoid™, Thermo Fisher Scientific, USA) were used as positive controls at a concentration of 50 µg/ml in the final growth media, targeting fungal and bacterial strains, respectively.

Hemolytic activity assay

Preparation of blood solution. Three milliliters of sheep blood were washed with 14 ml Phosphate-buffered saline (PBS) at pH 5.7 by centrifuging at 500 g at 4°C for 10 min, followed by careful removal of the supernatant. This washing was repeated 4 times. After the final wash, 300 µl of blood was collected from the bottom of the vial and added to 9.7 ml of PBS (pH 5.7). This RBC suspension was used for the hemolytic assay.[9]

Hemolysis test. Fifty microliters of blood suspension were added to 100 µl of two-fold serially diluted 10% (w/v) Kenicide solution in PBS, resulting in a concentration range from 10% w/v to 0.625% w/v Kenicide. The suspension was incubated at 37°C for 30 min, then centrifuged at 2,500 xg for 6 min. The supernatant was transferred to a microtiter plate, and absorbance was measured at 541 nm to identify hemolysis.[9] Three different Kenicide extracts, prepared using different solvents, were dried using a desiccator and mixed with PBS to 10% w/v Kenicide solutions to mitigate the hemolytic effect of the extraction solvents.

Control preparation. A positive control for complete hemolysis was prepared by adding 50 µL of blood to 100 µl of double-distilled water (ddH_2O), followed by freeze-thaw cycling (-30^{o}C in a freezer vs 42°C in a water bath) 5 times. [9] An internal control was prepared by mixing 100 µl of 20% (v/v) glacial acetic acid in PBS (pH 5.7), serially diluted down to 0.625% (v/v) and adding 50 µl of blood solution. The whole experiment was triplicated. The optical density (OD) values of positive hemolysis and the internal control at 541 nm were compared with the OD values from the hemolysis test for each concentration of Kenicide to calculate the percentage of hemolysis at various concentrations.[9]

Cytotoxicity assay of kenicide using *Artemia salina* (brine shrimp). Brine shrimp cysts were hatched in artificial seawater prepared from sea salt

(40 g/l) supplemented with dried yeast (6 g/l) and oxygenated by an aquarium pump. After incubation at 22 - 29°C for 48 hrs, nauplii were collected by a Pasteur pipette. 2 g of Kenicide extract was mixed with 5 ml of seawater, making a primary working solution of Kenicide with a concentration of 40% w/v. Serial dilution was performed in the sterile glass test tubes using sterile seawater in five steps of two-fold dilution each. A suspension of nauplii containing 10-15 organisms in 1 ml seawater was added to each test tube, making the final Kenicide concentration range from 20% w/v to 0.5% w/v. The covered tubes were incubated at 25°C for 24 hrs. Test tubes were examined with the naked eye, and dead/non-motile nauplii were counted. For the positive control, 1 ml of methanol was added to 1 ml of suspension containing 15 nauplii. Because methanol causes immediate lethality, this suspension was incubated for 15 min, which was sufficient to achieve 100% mortality. In contrast, Kenicide test samples were incubated for 24 hrs to evaluate concentration-dependent mortality.

LC_{50} estimation (Probit model). Mortality data from the brine shrimp (*Artemia salina*) nauplii assay were analyzed using a probit dose–response model with $\log_{10}$-transformed Kenicide concentration (% w/v) as the predictor. The 24 hrs LC_{50} was calculated as the concentration corresponding to 50% predicted mortality from the fitted model. Concentrations producing 0% mortality yield undefined probit values; therefore, such points were recorded descriptively but were not included in the probit regression unless a standard continuity correction based on the number tested (e.g., (dead + 0.5)/(n + 1)) was applied.[9,10]

Isolation and identification of the causative agent from paronychia patients. The causative agents of paronychia were isolated from the collected dead skin and pus samples by inoculating them individually into Alkaline Peptone Water (Oxoid, UK) and incubating in an orbital shaker at 120 rpm for 4 hrs at 37°C (Gallenkamp, Germany). Following incubation, 10 µl of the sample suspension was inoculated onto selective media: mannitol salt agar and cetrimide agar for preferential selection of *Staphylococcus* spp. and *Pseudomonas* spp. respectively. The cultures were then incubated at 37°C for 24 hrs under aerobic conditions. The appearance of bacterial colonies, if any, on the selective media was initially selected based on the colony morphology observed. Presumptive identification of the selected isolates was carried out using Gram staining and subsequent biochemical tests in appropriate media (Oxoid, UK). These biochemical tests included sugar fermentation, H_2S production, indole production, methyl red test, Voges-Proskauer test, citrate utilization test, catalase test, oxidase test, and motility test.[12]

RESULTS AND DISCUSSION

Antimicrobial activity test. The antimicrobial activities of various fraction extracts of Kenicide were evaluated using the agar-disc diffusion method, with gentamicin (Oxoid, UK) serving as the positive control. Zones of inhibition were measured to assess antibacterial efficacy. The results showed that the antibacterial and antifungal activities of Kenicide varied according to both the extraction method and the concentration. Kenicide demonstrated inhibitory effects against seven bacterial isolates and one fungal isolate, with the acetic acid extract generally producing the most pronounced activity.

At a 12.5% (w/v) concentration, the water-based Kenicide extract produced zones of inhibition measuring 5.5 mm for *S. aureus* and 4.3 mm for *V. cholerae*, which were comparatively larger than those produced by other extracts at the same concentration (Figure 1A). In contrast, an acetic acid-based extract of Kenicide demonstrated larger zones of inhibition against *B. cereus*, *Pseudomonas* spp. and *C. albicans*. None of the three extracts demonstrated any activity against *L. monocytogenes*, *E. coli*, and *S. enterica*. As anticipated, the 25% (w/v) concentration of the drug followed a similar pattern of antimicrobial activity, albeit with higher efficacy (Figure 1B). Moreover, both the acetic acid and ethanol extracts showed measurable zones of inhibition against *E. coli*. A higher concentration of 50% (w/v) produced an

increased growth inhibition compared to the 25% concentration (data not shown). To balance antimicrobial efficacy with safety and minimize potential toxicity, 12.5% and 25% concentrations were selected for further experimentation.

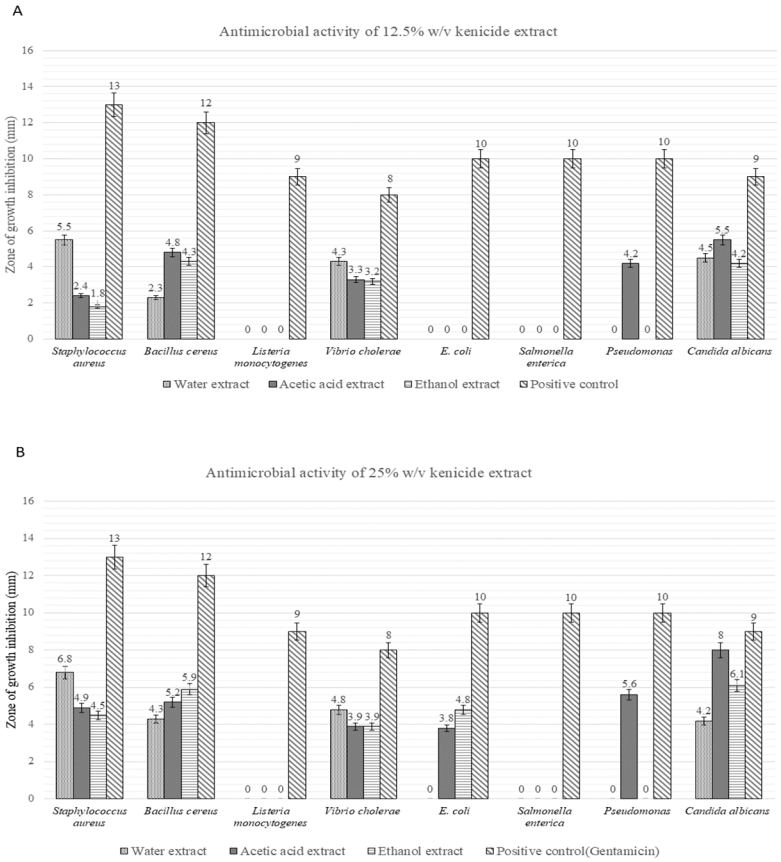

Figure 1. The antimicrobial activities of different Kenicide extracts: aqueous, acetic acid, and ethanol, on seven microorganisms at 12.5% (A) and 25% (B) (w/v) concentrations by disc diffusion method. Data are presented as mean ± standard deviation (SD) from three independent experiments. Error bars indicate standard deviations.

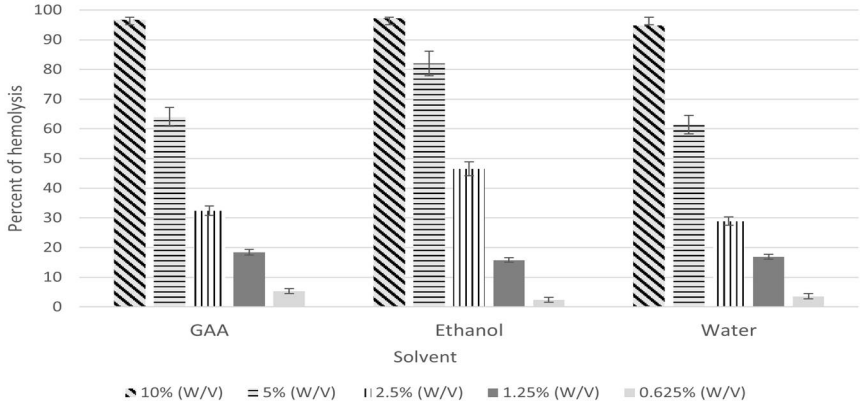

Figure 2. Hemolytic activities of different extracts: glacial acetic acid (GAA), ethanol, and water, of Kenicide at various concentrations. Data are presented as mean ± standard deviation (SD) from three independent experiments. Error bars indicate standard deviations.

Hemolytic activity analysis of Kenicide. Kenicide exhibited hemolytic activity across all types of extracts. A 50% hemolysis (HD_{50}) of red blood cells was observed at a concentration of 5% (w/v) irrespective of the solvent used during the extraction process (Figure 2). However, the hemolytic effect was more pronounced in the ethanol-based extract.

Cytotoxicity assay. The cytotoxicity of Kenicide was evaluated using *Artemia salina* (brine shrimp) cysts. Nauplii were exposed for 24 hours to different concentrations of Kenicide in aqueous solution, alongside a control group that received no Kenicide. After the incubation period, mortality rates of 15% and 70% were recorded at Kenicide concentrations of 1% (w/v) and 5% (w/v), respectively. These results indicate a concentration-dependent cytotoxic effect of Kenicide, with higher concentrations exhibiting greater lethality. Kenicide produced concentration-dependent naupliar mortality after 24 h exposure, with observed mortality of 15%, 70%, 85%, and 95% at 1%, 5%, 10% and 20% (w/v), respectively, while 0.5% (w/v) resulted in 0% mortality. Probit regression using $\log_{10}$ (concentration) for doses producing non-zero mortality estimated a 24-h LC_{50} of 3.06% (w/v) (Figure 3; Table 1).

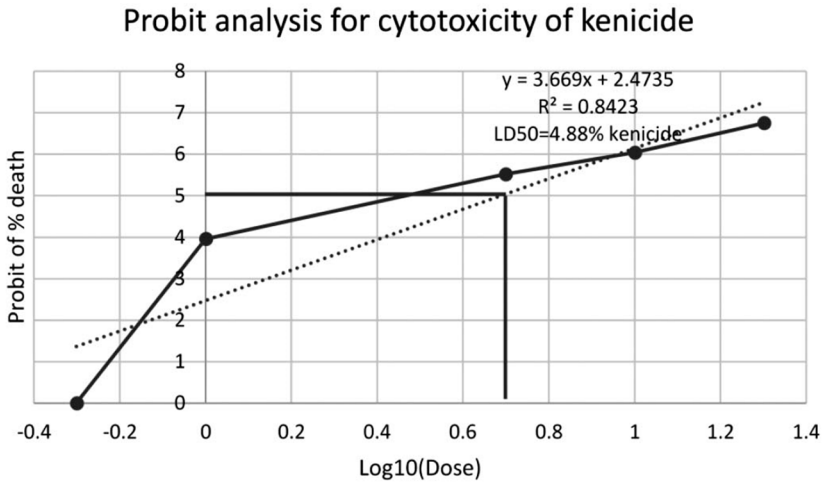

Figure 3. Probit dose–response model for Kenicide cytotoxicity in *Artemia salina* nauplii after 24 h exposure. Mortality proportions were analyzed using a probit model with log10-transformed concentration (% w/v). The 24-h LC_{50} was calculated as the concentration corresponding to 50% predicted mortality from the fitted regression.

Table 1. Probit analysis for the cytotoxicity of kenicide in *Artemia salina* cysts.

Concentration (% w/v)	Nauplii mortality (%)	LOG_{10} (dose)	Probit
20	95%	1.30103	6.75
10	85%	1	6.04
5	70%	0.69897	5.52
1	15%	0	3.96
0.5	0%	-0.30103	0

Isolation of causative agents of paronychia from nail wounds of patients. Paronychia is commonly caused by *Staphylococcus aureus*, *Streptococcus* spp. *Pseudomonas aeruginosa* and *Candida albicans*. To isolate bacterial pathogens, samples of dead skin and pus collected from patients were initially enriched in alkaline peptone water and incubated at 37°C for 4 hours. Subsequently, aliquots were streaked onto mannitol salt agar (MSA) and cetrimide agar to selectively isolate *S. aureus* and *Pseudomonas* spp. respectively. Plates were incubated at 37°C for 24 hours.

From the MSA plate, 27 strains were isolated, while 24 strains were obtained from the cetrimide agar, based on colony morphology, cellular characteristics, and Gram staining. Biochemical identification confirmed 8 strains as *S. aureus* and 5 strains as *Pseudomonas* spp.

Antimicrobial activity of Kenicide on causative agents isolated from participants. The antimicrobial efficacy of Kenicide was assessed against the *S. aureus* and *Pseudomonas* spp. strains isolated from patients using the disc diffusion method. Among the different extraction solvents, the glacial acetic acid-based extract of Kenicide exhibited the most potent antimicrobial activity, producing significantly larger zones of inhibition compared to ethanol and aqueous extracts. This difference was statistically significant according to a paired t-test ($p < 0.005$) (Figure 4).

Notably, the zone of inhibition produced by the 25% (w/v) glacial acetic acid extract of Kenicide against *S. aureus* was comparable to that of the positive control (Gentamicin, 10 µg disc). ANOVA analysis revealed that this difference was statistically non-significant (data not shown) (Figure 4A). On the contrary, the zone of inhibition by kenicide extracts against *Pseudomonas* spp. was smaller than that of the positive control (Gentamicin, 10 µg disc) (Figure 4B).

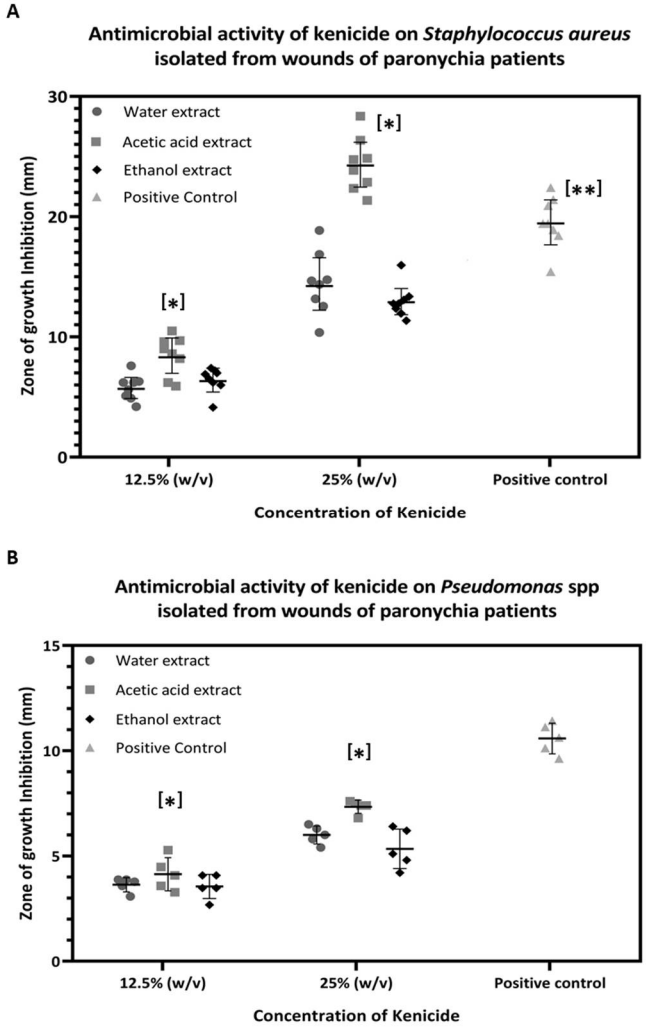

Figure 4. Zones of inhibition observed in disc diffusion assays evaluating the antimicrobial activity of Kenicide at 12.5% and 25% (w/v) concentrations, prepared using three different extraction methods: water, ethanol and glacial acetic acid. Inhibition zones against *Staphylococcus aureus* strains (A) and *Pseudomonas* spp. strains (B), both isolated from the nail wounds of paronychia patients. Each point represents a mean zone of inhibition from three replicates for each bacterial strain and error bars indicate the mean ± standard deviation (SD). Gentamicin (10 µg/disc) was used as the positive control. Statistical significance was assessed using a paired *t*-test [*] ($p<0.005$) and one-way ANOVA [**] ($p < 0.05$).

Paronychia is an inflammatory condition affecting the tissue folds surrounding fingernails or toenails, leading to significant changes in the nail's shape, color, or texture. This condition can be caused by various pathogens, including bacteria, fungi, and yeast. Common triggers include trauma from nail biting, cuticle picking, or aggressive manicures, which allow pathogens, particularly *Staphylococcus aureus* and *Candida* spp., to enter the soft tissue surrounding the nail. Symptoms typically include swelling, redness, pain and the formation of pus-filled lesions.

Acute paronychia, usually caused by bacterial infection, develops rapidly and is often resolved within six weeks.[13] In contrast, chronic paronychia can persist for longer and is often linked to ongoing exposure to irritants or allergens, with fungal infections, especially *Candida*, frequently involved. This chronic form is more difficult to treat and often recurs, particularly in individuals with compromised immunity (e.g., diabetes, cancer, HIV)[14,15] or those exposed to wet environments, such as housekeepers or food service workers.[16]

While *S. aureus* and *Candida* are the most common pathogens, other organisms, including *Pseudomonas aeruginosa* and even Herpes simplex virus[2] can also cause paronychia. Non-infectious factors, such as irritant contact dermatitis, allergic reactions, or certain medications, can also lead to the development of this condition.[13] The multifactorial etiology complicates diagnosis and treatment, often requiring both antimicrobial and anti-inflammatory approaches.

Conventional treatment options, including topical or oral antifungals and antibiotics, are often insufficient, especially in chronic cases.[17] Surgical intervention may be needed, yet outcomes remain inconsistent, and there is no definitive cure. These limitations underscore the need for alternative therapies.

Licorice (*Glycyrrhiza glabra*), long used in traditional medicine in Asia[18,19] is known for its broad pharmacological properties, including antiviral and antimicrobial properties.[20-23] In this study, we explored Kenicide, a herbal formulation derived from licorice, for its potential use against paronychia-causing pathogens.

Kenicide demonstrated notable antimicrobial activity against *S. aureus* and *Pseudomonas* spp., strains isolated from patients. Notably, the acetic acid extract at 25% (w/v) produced inhibition zones comparable to those of gentamicin (10 µg), a commonly prescribed antibiotic (Figure 1B). Its effectiveness was significantly greater against the wild-type clinical isolates than that of laboratory strains, suggesting strong real-world applicability. Although Kenicide inhibited the growth of patient-derived *Pseudomonas* spp, the magnitude of inhibition was lower than that observed for *S. aureus* (Figure 4B). This indicates that Kenicide may exhibit greater antimicrobial activity against Gram-positive bacteria, a trend that has been reported for *G. glabra* [24] and attributed in part to structural differences between Gram-positive and Gram-negative cell envelopes.

However, toxicity assessments revealed cytotoxic and hemolytic effects even at low concentrations, indicating that Kenicide, the *G. glabra* (licorice) extract, should not be administered systemically. Its application should therefore be restricted to topical use on affected areas. Despite this limitation, the preparation showed considerable promise in pre-clinical *in vitro* evaluations. Future work should focus on validating Kenicide's safety and efficacy *in vivo*, beginning with animal models. If found safe, this could pave the way for clinical trials and potential integration into the therapeutic arsenal for paronychia.

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