RESEARCH PAPER

Effect of Topical α-Mangostin on the Healing of Pseudomonas aeruginosa–Infected Full-Thickness Acute Wounds in Rats (Rattus norvegicus)

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ABSTRACT

Background: Full-thickness wounds infected with Pseudomonas aeruginosa are associated with delayed healing due to persistent bacterial colonization, prolonged inflammation, and oxidative stress. α-Mangostin, a natural compound derived from Garcinia mangostana Linn., has demonstrated antimicrobial, anti-inflammatory, and antioxidant properties that may enhance wound healing. This study aimed to evaluate the effect of topical α-mangostin on infected wound healing.

Materials and Methods: This laboratory experimental study used a randomized post-test-only control group design involving 20 male Wistar rats divided into four groups: control, infected, α-mangostin-treated, and infected treated with α-mangostin. Full-thickness wounds were created on the dorsal area, and Pseudomonas aeruginosa was inoculated in the designated groups. A 16% α-mangostin ointment was applied topically. Outcomes assessed on day 5 included leukocyte count, bacterial colony count (CFU/mL), and malondialdehyde (MDA) levels. Statistical analysis was performed using ANOVA and Kruskal–Wallis tests.

Results: The infected untreated group showed the highest bacterial colony count, leukocyte levels, and MDA levels. Topical α-mangostin significantly reduced leukocyte count ($p = 0.010$) and MDA levels ($p < 0.001$), while bacterial colony counts showed a decreasing trend without statistical significance ($p = 0.068$). The treated infected group demonstrated improved inflammatory modulation and reduced oxidative stress compared to the untreated infected group.

Conclusions: Topical α-mangostin enhances healing of Pseudomonas aeruginosa–infected full-thickness wounds by reducing bacterial colonization, modulating inflammation, and decreasing oxidative stress. These findings support its potential as an adjunctive therapy in wound management.

Keywords: α-mangostin, wound healing, Pseudomonas aeruginosa, full-thickness wound, oxidative stress, malondialdehyde, inflammation.

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Introduction

Full-thickness acute wounds represent severe injuries involving complete damage to the epidermis and dermis, and may extend to deeper tissues such as muscle and bone. These wounds can result from trauma, burns, or surgical procedures can lead to significant morbidity and life-threatening complications.[1] Wound infection is one of the most common complications that can delay healing and worsen clinical outcomes. Among pathogens, *Pseudomonas aeruginosa* is a Gram-negative opportunistic bacterium frequently associated with severe infections and high morbidity, particularly due to its resistance to multiple antibiotics.[2,3]

Wound healing is a complex and dynamic process involving coordinated cellular and molecular mechanisms across several overlapping phases: haemostasis, inflammation, proliferation, and remodelling.[4,5] Infection and excessive inflammation can disrupt this process, leading to prolonged healing and tissue damage. Leukocytes play a central role in host defense and are often elevated in response to bacterial infection, reflecting the degree of inflammation within the wound.[6]

Oxidative stress is another key factor that impairs wound healing. Increased production of reactive oxygen species leads to lipid peroxidation and the formation of malondialdehyde (MDA), a biomarker of oxidative damage. Elevated MDA levels indicate cellular injury and disruption of tissue repair processes, whereas antioxidants can mitigate these effects and support the formation of healthy granulation tissue.[7,8]

Plant-derived compounds have been widely explored as alternative therapies due to their antimicrobial, anti-inflammatory, and antioxidant properties. α-Mangostin, a bioactive compound from *Garcinia mangostana Linn.*, has shown potential in inhibiting bacterial growth, reducing inflammation, and protecting against oxidative stress. Therefore, this study aims to evaluate the effect of topical α-mangostin on the healing of full-thickness acute wounds infected with *Pseudomonas aeruginosa*.

Methods

Study Design

This study was a laboratory experimental study using a randomized post-test-only control group design to evaluate the effect of topical α-mangostin on bacterial colonization, leukocyte count, and malondialdehyde (MDA) levels in full-thickness acute wounds infected with *Pseudomonas aeruginosa* in rats.

Animals and Sampling

A total of 20 male Wistar rats (*Rattus norvegicus*), aged approximately 12 weeks and weighing 250–300 g, were included. Only healthy, active animals without physical abnormalities were selected, while female rats and those showing signs of illness or significant weight loss during acclimatization were excluded to avoid hormonal and physiological bias. The sample size was determined using Federer's formula, and animals were randomly allocated into four groups (n=5 per group): control (no treatment), infected without treatment, α-mangostin treatment only, and infected with α-mangostin treatment. All animals were housed under standardized laboratory conditions and acclimatized for one week prior to experimentation.

Preparation of α-Mangostin Ointment

Purified mangosteen pericarp extract containing more than 90% α-mangostin was used. The extract was formulated into a 16% topical ointment.

Wound Creation and Infection Procedure

All animals were anesthetized using intramuscular ketamine at a dose of 20 mg/kg body weight. The dorsal area was shaved and disinfected before creating a 2 × 2 cm full-thickness wound using sterile instruments. In the designated groups, wounds were inoculated with *Pseudomonas aeruginosa* using a sterile cotton swab. The bacterial strain used was obtained from clinical isolates and cultured prior to inoculation.

Treatment Protocol

Topical α-mangostin ointment was applied to the treatment groups, while control groups did not receive active treatment. All wounds were covered with transparent dressing to maintain a moist environment and prevent contamination. Wound care, including dressing replacement and cleansing with normal saline, was performed twice daily.

Outcome Measurements

On day 5, animals were anesthetized and biological samples were collected. Leukocyte counts were measured using a hematology analyzer. Bacterial colonization was assessed by culturing wound swabs in tryptic soy broth, followed by incubation for 24 hours and colony counting expressed as CFU/mL. Serum MDA levels were measured using an ELISA method to evaluate oxidative stress.

Statistical Analysis

Data were analyzed using SPSS version 23. Descriptive statistics were presented, and normality testing was conducted prior to inferential analysis. Non-parametric tests, including Kruskal–Wallis and Mann–Whitney tests, were used for group comparisons. A p-value < 0.05 was considered statistically significant.

Ethical Approval

This study was approved by the Ethics Committee of the Faculty of Veterinary Medicine, Universitas Airlangga. All experimental procedures were conducted in accordance with institutional guidelines for the care and use of laboratory animals.

Results

Preparation and Experimental Observation

The α-mangostin ointment was successfully prepared from purified mangosteen pericarp extract containing >90% α-mangostin at a concentration of 16%. This concentration was selected based on previous studies demonstrating strong antibacterial activity against *Staphylococcus aureus* and *Pseudomonas aeruginosa*, with stable inhibition observed at this level. The formulation showed good consistency and suitability for topical application.

A total of 20 male Wistar rats (*Rattus norvegicus*) with relatively homogeneous body weight (250–300 g) were randomly allocated into four groups. Full-thickness wounds measuring 2 × 2 cm were successfully created on the dorsal region of all animals. In the infected groups, wounds were inoculated with *Pseudomonas aeruginosa* at a standardized concentration. Clinical signs of infection developed within 1–3 days following inoculation. In the infected treatment group, topical α-mangostin was initiated after infection was established. All animals survived throughout the observation period without significant complications.

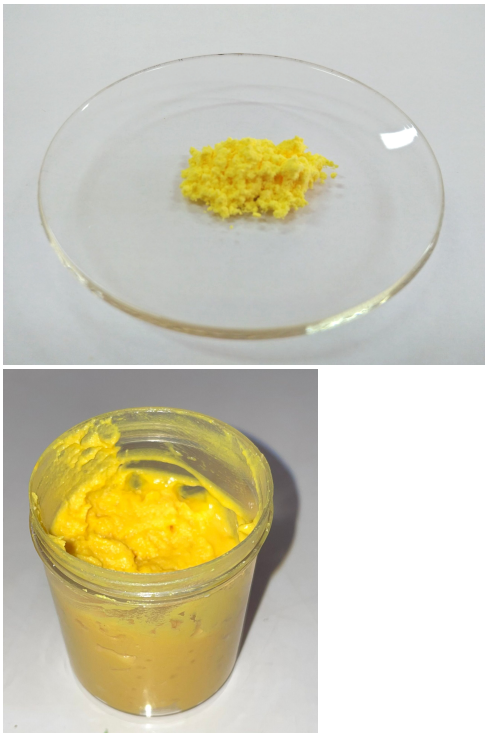

Figure 1. Base material and final product of 16% α-mangostin ointment

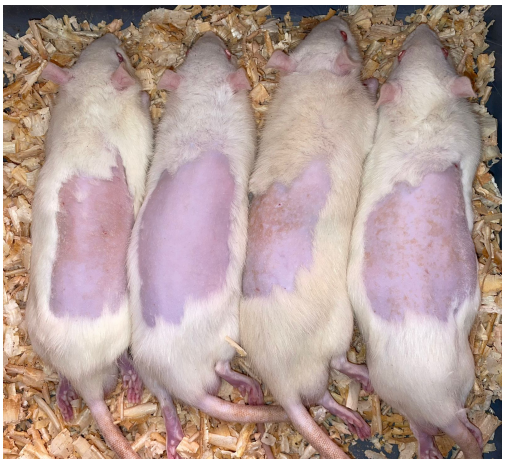

Figure 2. Preparation of the rat dorsal area

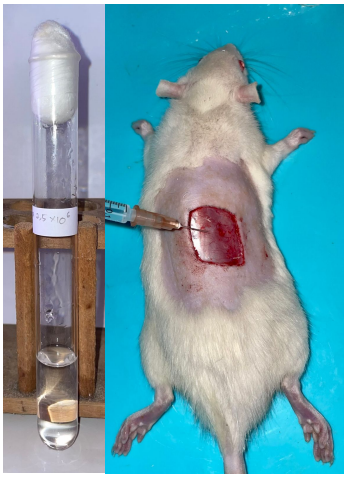

Figure 3. *Pseudomonas aeruginosa* isolate (2.5 × 10^6 CFU/10 mL) (left) and inoculation of 0.5 mL of the isolate onto the full-thickness wound surface (right).

Leukocyte Count Analysis

Leukocyte levels were measured using complete blood count analysis. The results for each group are presented in Table 1.

Table 1. Leukocyte Count (×10^3/mm^3)

Group	Rep 1	Rep 2	Rep 3	Rep 4	Rep 5	Rep 6
Group 1	12	5.1	11.8	7.9	-	-
Group 2	19	16.7	20.1	13.5	11.5	-
Group 3	13.9	14.7	16.4	21.9	17.9	12.1
Group 4	14	13.8	11.5	9.3	12.7	-

Leukocyte levels were generally higher in Group 2 and Group 3 compared to the other groups. Group 2 demonstrated elevated leukocyte counts consistent with an inflammatory response due to *Pseudomonas aeruginosa* infection. Group 3 also showed increased leukocyte levels, possibly reflecting an inflammatory response associated with topical α-mangostin. In contrast, Group 1 (non-infected control) exhibited lower leukocyte counts. Group 4 demonstrated relatively lower and more variable leukocyte levels compared to Group 2, suggesting a potential modulatory effect of α-mangostin on inflammation in infected wounds.

Bacterial Colony Count Analysis

Bacterial colonization was assessed using tissue culture methods, which provide a more accurate representation of microbial invasion within viable tissue compared to surface swab techniques. The results are presented in Table 2.

Table 2. Bacterial Colony Count of *Pseudomonas aeruginosa* (CFU/mL)

Group	Rep 1	Rep 2	Rep 3	Rep 4	Rep 5	Rep 6
Group 1	2480	0	1220	95	-	-
Group 2	730	6,150,000	185	22,500	23,500	-
Group 3	11,200	0	2,120	460	35	1,430
Group 4	0	0	0	1,700	0	-

Group 1 demonstrated low bacterial colony counts, indicating the absence of significant infection. In contrast, Group 2 showed markedly elevated colony counts, confirming successful infection with *Pseudomonas aeruginosa*. Group 3 exhibited higher colony counts compared to the control group, which may be associated with cross-contamination during the experimental process. Notably, Group 4 demonstrated very low to undetectable bacterial counts, indicating that topical α-mangostin effectively suppressed bacterial growth in infected wounds.

Malondialdehyde (MDA) Level Analysis

MDA levels were measured using ELISA as a marker of oxidative stress. The results are presented in Table 3.

Table 3. Malondialdehyde (MDA) Levels (nmol/mL)

Group	Rep 1	Rep 2	Rep 3	Rep 4	Rep 5	Rep 6
Group 1	4.26	3.95	3.74	3.28	-	-
Group 2	5.03	5.3	5.4	4.84	4.97	-
Group 3	1.59	1.38	2.39	1.96	2.67	2.64
Group 4	3.8	4.66	3.43	3.59	3.37	-

Group 2 exhibited the highest MDA levels, indicating increased oxidative stress associated with *Pseudomonas aeruginosa* infection. In contrast, Group 3 demonstrated a marked reduction in MDA levels, reaching values within the normal range, suggesting a strong antioxidant effect of α-mangostin. Group 1 showed moderately elevated MDA levels, while Group 4 demonstrated lower MDA levels compared to the infected untreated group, indicating that α-mangostin may reduce oxidative stress in infected wounds.

Data Distribution and Statistical Approach

Normality testing was performed prior to comparative analysis to determine the appropriate statistical methods. The results showed that leukocyte and malondialdehyde (MDA) data were normally distributed across all groups ($p > 0.05$). In contrast, bacterial colony count data were not normally distributed in several groups ($p < 0.05$). Therefore, parametric analysis (one-way ANOVA) was applied for leukocyte and MDA variables, while non-parametric analysis (Kruskal–Wallis test) was used for bacterial colony counts.

Homogeneity of Variance

Homogeneity testing using Levene's test demonstrated that leukocyte ($p = 0.476$) and MDA ($p = 0.302$) data had equal variances across groups, indicating homogeneity. Thus, these variables met the assumptions for parametric analysis using one-way ANOVA, followed by post hoc Tukey tests for multiple comparisons. For bacterial colony count data, homogeneity testing was not performed due to the use of

non-parametric analysis; therefore, post hoc comparisons were conducted using Dunn's test.

Comparative Analysis Between Groups

Comparative analysis was performed to evaluate differences between groups for leukocyte count, bacterial colony count, and MDA levels. The mean ± SD values and p-values are presented in Table 4.

Table 4. Comparison of Outcomes Between Groups (Mean ± SD)

Variable	Group 1	Group 2	Group 3	Group 4	p-value
Leukocytes (×10³/mm³)	9.20 ± 3.32	16.16 ± 3.63	16.15 ± 3.46	12.26 ± 4.09	0.010
Bacterial culture (CFU/mL)	948.75 ± 1161.51	1,239,383 ± 2,745,141.53	2,540.83 ± 4,323.36	340.00 ± 760.26	0.068
MDA (nmol/mL)	3.81 ± 0.41	5.11 ± 0.23	2.10 ± 0.55	3.77 ± 0.52	P< 0.001

Leukocyte Comparison

Leukocyte counts differed significantly between groups (p = 0.010). The highest values were observed in Group 2 and Group 3, indicating an active inflammatory response due to infection and early wound healing processes. Group 4 showed lower leukocyte levels compared to Group 2, suggesting a potential anti-inflammatory effect of α-mangostin. Post hoc analysis demonstrated significant differences between Group 1 vs Group 2 and Group 1 vs Group 3, while other comparisons were not statistically significant.

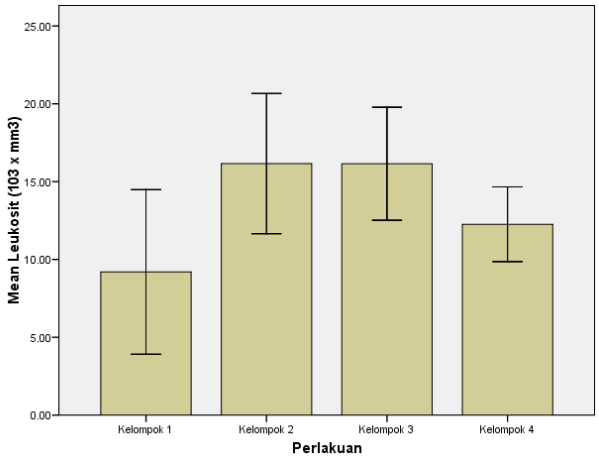

Figure 4. Mean leukocyte levels (×10³/mm³) across experimental groups.

Bacterial Colony Comparison

Bacterial colony counts were highest in Group 2, confirming active infection, while Group 4 showed the lowest values, indicating suppression of bacterial growth following α-mangostin treatment. Although the difference was not statistically significant (p = 0.068), a clear biological trend was observed, with reduced bacterial burden in treated groups, particularly in infected wounds receiving α-mangostin.

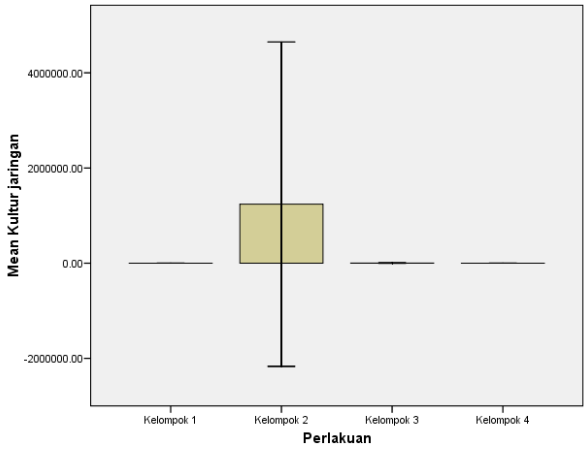

Figure 5. Mean bacterial colony count (CFU/mL) across experimental groups

Malondialdehyde (MDA) Comparison

MDA levels differed significantly between groups (p < 0.001). The highest levels were observed in Group 2, reflecting increased oxidative stress due to infection. In contrast, Group 3 demonstrated the lowest MDA levels, indicating a strong antioxidant effect of α-mangostin. Group 4 showed reduced MDA levels compared to Group 2, suggesting that α-mangostin

mitigates oxidative stress in infected wounds. Post hoc analysis revealed significant differences between most groups, except between Group 1 and Group 4.

Table 5. Post Hoc Analysis of MDA Levels (p-values)

Comparison	p-value
Group 1 vs Group 2	0.003
Group 1 vs Group 3	0.000
Group 1 vs Group 4	0.999
Group 2 vs Group 3	0.000
Group 2 vs Group 4	0.001
Group 3 vs Group 4	0.000

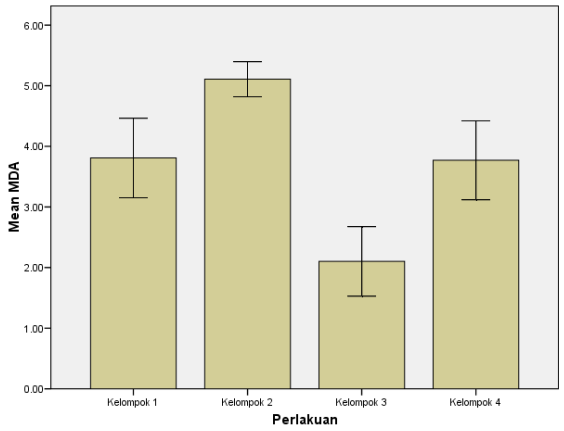

Figure 6. Mean MDA levels (nmol/mL) across experimental groups.

Discussion

This study demonstrates that topical α-mangostin improves healing of full-thickness wounds infected with *Pseudomonas aeruginosa* through reduction of bacterial colonization, modulation of inflammatory response, and attenuation of oxidative stress, as reflected by leukocyte and MDA levels.[4,9]. In the untreated infected group, high bacterial burden, elevated leukocytes, and increased MDA levels indicate uncontrolled infection, persistent inflammation, and oxidative tissue damage.[10,11] In contrast, the treated infected group showed decreased bacterial load and MDA levels, with leukocyte levels tending toward physiological values, suggesting a transition from inflammatory to proliferative phase.[6,12] These findings support the hypothesis that α-mangostin enhances wound healing through antimicrobial, anti-inflammatory, and antioxidant mechanisms.[13,14,15,16]

Effect on Bacterial Colonization

The untreated infected group showed markedly high bacterial counts, reflecting uncontrolled infection.[2,3] Topical α-mangostin significantly reduced bacterial colonies, in some cases approaching minimal detectable levels, indicating strong antibacterial activity.[17,18] Mechanistically, α-mangostin disrupts bacterial membranes and inhibits biofilm formation, which is particularly relevant for *P. aeruginosa* infections[19,20,21] Although statistical significance was not achieved, likely due to small sample size and data variability, the observed biological trend remains clinically meaningful.[22,23]

Effect on Inflammatory Response (Leukocytes)

Leukocyte elevation in the untreated infected group reflects an intense inflammatory response to bacterial invasion.[24,10] The reduction of leukocyte levels in the α-mangostin-treated infected group suggests modulation rather than suppression of inflammation, indicating a more controlled immune response.[25] This effect is supported by evidence that α-mangostin inhibits pro-inflammatory pathways (NF-κB, MAPK) and cytokines while promoting macrophage transition toward a pro-healing phenotype.[26,27,10]

Effect on Oxidative Stress (MDA)

MDA levels were highest in the infected untreated group, indicating severe oxidative stress and tissue damage.[28,11] α-Mangostin significantly reduced MDA levels, especially in the non-infected treated group, confirming its strong antioxidant properties.[28,29] Mechanistically, α-mangostin scavenges reactive oxygen species and enhances endogenous antioxidant defenses through Nrf2 activation.[13,26] This reduction in oxidative stress supports improved cellular integrity and wound healing.[4,10]

Clinical and Biological Implications

These findings highlight α-mangostin as a promising topical agent with triple action—antimicrobial, anti-inflammatory, and antioxidant—targeting key mechanisms of infected wound pathophysiology.[13,30] By improving the wound microenvironment, α-mangostin supports tissue regeneration, angiogenesis, and collagen remodeling.[4,31] Clinically, it may reduce dependence on systemic antibiotics and lower the risk of complications such as infection persistence and delayed healing.[2,21]

Limitations

This study has several limitations. First, the use of an animal model limits direct generalization to human wound healing.[1] Second, the infection model involved a single pathogen, whereas clinical wounds are often polymicrobial.[3] Third, the observation period was limited to day 5, representing mainly the inflammatory phase.[25] Fourth, the topical formulation was not optimized for pharmacokinetics or long-term stability.[32] Fifth, molecular and histopathological analyses were not performed, limiting mechanistic insight.[10] Finally, the relatively small sample size may reduce statistical power.[33]

Conclusion

Topical α-mangostin demonstrated beneficial effects in improving the healing of Pseudomonas aeruginosa–infected full-thickness wounds by reducing bacterial colonization, modulating the inflammatory response, and decreasing oxidative stress, as reflected by lower leukocyte and MDA levels. These findings indicate that α-mangostin exerts antimicrobial, anti-inflammatory, and antioxidant activities that collectively support the transition from the inflammatory to the proliferative phase of wound healing. Overall, the study supports the hypothesis that topical α-mangostin enhances infected wound healing through multi-target mechanisms and shows potential as a promising adjunctive therapy in wound management. Further studies are required to evaluate long-term healing outcomes, optimize topical formulations, and confirm these findings in clinical trials involving human subjects.

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