



Research Article

A Comparative Evaluation of the Wound Healing Efficacy of Madhvadi Lepa and Silver Nitrate Gel in *Semecarpus anacardium* Linn. Oil-Induced Dermal Toxicity in Albino Mice

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Abstract

Background: *Semecarpus anacardium* Linn.(SA) is widely used in *Ayurveda* but is known to cause dermal toxicity due to irritant phenolic compounds. Modern management relies on symptomatic treatment like Silver Nitrate, whereas the classical formulation *Madhvadi Lepa* is traditionally indicated for toxic skin inflammation. Its efficacy, however, has not been experimentally validated. **Objective:** To evaluate the efficacy of *Madhvadi Lepa* in SA oil induced dermal toxicity in Swiss albino mice, in comparison with Silver nitrate gel. **Methods:** Twenty four female Swiss albino mice (20–30 g) were allocated into four groups (n=6). Normal Control, Disease Control (SA oil only), Standard (SA oil + 0.2% silver nitrate gel), and Test (SA oil + *Madhvadi Lepa*). Dermal parameters including erythema, oedema, scaling, erosion, epithelialization time, wound area, and percentage wound closure were assessed for 14 days as per OECD 404 guidelines. Body weight, food intake, and relative organ weights were recorded. Histopathological examination of skin tissues was performed. Data were analyzed using ANOVA and Tukey's test (GraphPad Prism 8.42). **Results:** The Disease Control group showed persistent erythema, delayed healing (13.50 ± 0.84 days), and only 49.10% wound closure. *Madhvadi Lepa* significantly reduced erythema, erosion, and scaling from Day 5 onward and achieved 83.83% wound closure and epithelialization by 9.83 ± 2.23 days, comparable to silver nitrate ($p > 0.05$). No systemic toxicity was observed. **Conclusion:** *Madhvadi Lepa* effectively attenuates SA induced dermal toxicity and promotes wound healing, exhibiting therapeutic efficacy comparable to Silver nitrate. It represents a safe and economical topical alternative in *Agadtantra* clinical practice.

Keywords: Dermal toxicity, Honey, *Madhvadi Lepa*, *Semecarpus anacardium*, Silver nitrate, Wound healing.

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Introduction

The *Semecarpus anacardium* Linn (SA) is considered a potent drug of great therapeutic importance in *Ayurveda* and is indicated in a wide range of disorders like Amavata (rheumatism), Apasmara (epilepsy), Gridhrasi (sciatica) and Shvasa (Asthma). It is categorized under the Upavisha (semi-poisonous) group in *Ayurved*. It should be administered internally only after proper Shodhana (processing raw materials) procedures. The synonym

Sophahetu, attributed to SA because of its blister causing nature, emphasizes the need for careful handling during collection and processing. (01) The current clinical investigation conducted in Mumbai, Maharashtra, on SA indicates that 25% of patients experience adverse medication reactions characterized by itching and maculopapular rashes. (02) In the general population the occurrence of contact dermatitis has been reported to range from 1.7% to 6.3%. The median prevalence of contact allergy to at least one allergen was 21.1% (12.5-40.6%), derived from data encompassing all age groups and countries. (03)

Despite its toxicity, SA is highly valued for its therapeutic properties. Acharya Charaka described it as Rasayana (rejuvenating), Dipaniya (digestive stimulant), Bhedaniya (purgative), Kuṣṭhaghna (anti-dermatosis), and Mutrasangrahaniya (antidiuretic). The pericarp oil of SA contains phenolic compounds such as anacardic acid, semicarpol, bhillawanol, and

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cardol, which exhibit potent irritant and vesicant actions. Contact with the oil produces erythema, blistering and ulceration. Because of these effects, SA is included under the E1 schedule of India's 1940 drug and cosmetic legislation. (04) In Ayurvedic pharmaceutical practice, accidental contact with SA oil during medicine preparation often results in local skin irritation among Ayurvedic practitioners. Modern medicine doesn't provide a specific antidote for such dermal toxicity and management is largely symptomatic involving topical antiseptics containing Silver Nitrate.) (05) Ayurveda, offers several Vishaghna (antitoxic) formulations that are economical, readily available and effective in the management of Visha-janya-Shotha (toxic inflammation) skin conditions. One such formulation, Madhvadi Lepa is mentioned in Ashtangasangraha (Uttara Tantra 47/32). This combination is traditionally indicated for Visha janya Shotha (toxic inflammation). (06) Each ingredient contributes synergistically to its therapeutic potential. However, despite the supportive evidence, the protective efficacy of Madhvadi Lepa against SA oil induced dermal toxicity has not yet been scientifically validated. Hence, this study was conducted to assess the effectiveness of Madhvadi Lepa in SA oil induced skin toxicity in mice, using Silver nitrate gel as a standard comparator.

Materials and Methodology

Ethical Clearance

"All experimental procedures in this study complied with the OECD 404 guideline and followed the protocol approved by the Institutional Animal Ethics Committee (IAEC) under Project Proposal No. SC/IAEC/2024/029. The study adhered to the regulations of the Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA), as outlined in The Gazette of India dated December 15, 1998. Prior approval from the IAEC was obtained (Approval No. IAEC-17-015)."Ref Anx.01)

Collection and preparation of test reference standard drug

All the drugs were procured from local authorised pharmacy Sanchomee Herboveda Pvt. Ltd, Pune. All the drug samples were sent for standardization to Authentic Research Laboratory. All drugs were standardized according to the specifications outlined in the Ayurveda Pharmacopoeia of India (API)(07)

Preparation of SA Oil - The technique for extracting SA oil is described in Sushruta Chikitsasthana. (08) Two equal-sized earthen jars were obtained. One of the two earthen pots had performed matkapad (smeared with cloth and clay) also known as pishta (Swedish pot), seven times. The SA fruits were stored in a pishta swedan jar after being gently crushed. Iron mesh was then used to cover the pishta Swedish pot's mouth. This pot was set upside down over a second, comparable clay pot, so that the mouths of the two pots were linked. It was then sealed with Multani mitti(Clay) and allowed to dry in the shade. The Yantra was stored in puta after drying. Cakes made from cow dung were stacked and burned over a pishta swedan pot. The earthen pot's bottom accumulated SA fruit oil due to the heat.

For preparation of Madhvadi Lepa

Fine powders of Vara (Terminalia chebula Retz., Terminalia bellirica Roxb. and Emblica officinalis Gaertn.), Vyosha (Piper nigrum L., Piper longum L. and Zingiber officinale Roscoe), Padmaka (Prunus cerasoides D. Don), Devadaru (Cedrus deodara Roxb.), Vidanga (Embelia ribes Burm. f.) and Ushira (Vetiveria zizanioides L.Nash) each weighting of 25 g were individually prepared and sifted through Sieve No. 100 (150 microns particle

size) to ensure fineness.(09) As per Sharangadhara Samhita, Madhyama Khanda, 11/3: "Lepa dravyaani suksma churnani..." (10). These powders were blended thoroughly with 200g of Honey to form a semisolid paste (lepa) suitable for topical application maintaining the appropriate proportion. Standardization of Final product was done at home institute laboratory. (10) Ref. Anx. (02)

Method of Animal Experiment

Experimental animals

The study utilized Swiss Albino mice (6–8 weeks Old. female), with initial body weights ranging from 20 to 30 grams, sourced from Sciore Research Private Limited. The mice were housed under controlled environmental conditions, maintaining a room temperature of $22 \pm 3^\circ\text{C}$, relative humidity of $50 \pm 5\%$, and a 12-hour light/12-hour dark illumination cycle. Three mice were accommodated per polypropylene cage, each equipped with a stainless steel grill top, facilities for food and water bottles, and corn cob bedding. The mice were provided with a standard pelleted feed and water.

Preparation of Animals

Prior to the initiation of the experiment, the body weight of each animal was recorded. The dorsal skin of all 24 Swiss albino mice was shaved using an electric trimmer to expose an area of approximately 2×2 cm. Dermal toxicity was induced by the topical application of 0.1 mL of SA oil uniformly over the shaved dorsal skin of each mouse. The animals were observed continuously for the first 24 hours for the development of erythema, blister formation, and other cutaneous reactions. Skin lesions and associated clinical signs were monitored daily throughout the study period. Following confirmation of dermal toxicity, the respective treatments were applied topically to the affected area once daily for 14 consecutive days. Skin irritation scores, wound healing, blister resolution, and epithelialization were assessed at predetermined intervals in accordance with the study protocol. At the end of the experimental period, the animals were humanely euthanized, and skin tissue samples were collected for histopathological examination. Relative organ weights were also recorded, and the observations were comparatively analysed and tabulated.

Calculation of Test Drug Quantity

As described in Sharangdhar Samhita, Vishaghna Lepa should be applied with a thickness of one-third angula (approximately 6–7 mm in humans). Considering the comparatively smaller body size and skin surface area of Swiss albino mice, the thickness of the Lepa was proportionately adjusted. The formulation was applied as a uniform thin layer (approximately 1–2 mm thick) over the entire inflamed area to ensure complete coverage of the lesion without excessive accumulation or spillage beyond the wound margins. The Lepa was freshly prepared and applied once daily throughout the 14-day treatment period.⁸

Grouping of the Animal Experiments

The animal experiment consisted of four groups, each comprising six female Swiss Albino mice. The Normal Control group(NCG) was maintained without any treatment on normal diet. The Disease Control group(DCG) had SA Oil locally applied to their shaved backs. The Standard Group(SG) received SA Oil followed by 0.2% Silver Nitrate gel locally over their shaved backs. The Test Group(TG) was treated with SA Oil followed by Madhvadi Lepa locally over their shaved backs to assess the lepa's efficacy against the induced condition.

Assessment Criteria

The observations were scored as per the reference of OECD guideline 404. Erythema, oedema, scaling, and erosion are graded from 0 to 4 based on severity: 0 indicates no reaction; 1 represents a barely perceptible or very slight reaction; 2 shows a well-defined or moderate reaction; 3 reflects moderate to severe changes such as marked redness, raised oedema, or deep scaling; and 4 denotes severe conditions including eschar formation, oedema extending beyond exposure, or wounds. Weight of the animals were recorded on every seventh day, while absolute organ weights were measured after euthanasia by CO₂ asphyxiation at the end of the experiment. Clinical scores for erythema, edema, scaling, and erosion were documented daily up to Day 14 to assess SA oil-induced skin toxicity and the therapeutic response to Silver Nitrate and Madhvadi Lepa. Wound area, percentage closure and the day of epithelialization were also monitored up to Day 14 to evaluate delayed healing and treatment efficacy. For histopathology, skin tissues were preserved in 10% formalin, followed by routine processing that included dehydration, clearing, and paraffin embedding. Sections of 4–5 µm thickness were prepared, stained with hematoxyline and eosin, and subsequently examined under a microscope.

Figure 1: Image of Drugs Used in Study



Figure 2: Image of Animal Study Experiment



Statistical analysis

GraphPad Prism version 8.42 was employed for data processing and statistical evaluation. The findings were presented as Mean $\pm$ SD. One or two way ANOVA was applied, accompanied by Tukey's multiple comparison test. A P-value of * $P < 0.05$, ** $P < 0.01$, and *** $P < 0.001$ was regarded as statistically significant when compared with the Disease Control group.

Results : (Refer Anx 04 for all the reports)

Body Weight: As per analysis of Figure no 3. All groups (NCG, DCG, SG, TG) showed a steady rise in body weight from Day 0 to

Day 14. The mean gain was consistent (2.2–2.5 g per week), with no reduction or stagnation, indicating absence of systemic toxicity. As per statistical analysis Tukey's test revealed no significant changes between groups at baseline Day 0, Day 7, or Day 14 (all $p > 0.05$).

This confirms that all four groups were well tolerated, maintaining normal physiological growth.

Change in Body Weight: As per Figure no. 04 - All 4 groups showed a steady rise in body weight from Day 0 to Day 14. The mean gain was consistent (2.2–2.5 g per week), with no reduction or stagnation, indicating absence of systemic toxicity. Tukey's test revealed no significant between groups at baseline (Day 0, Day 7 or Day 14 (all $p > 0.05$).

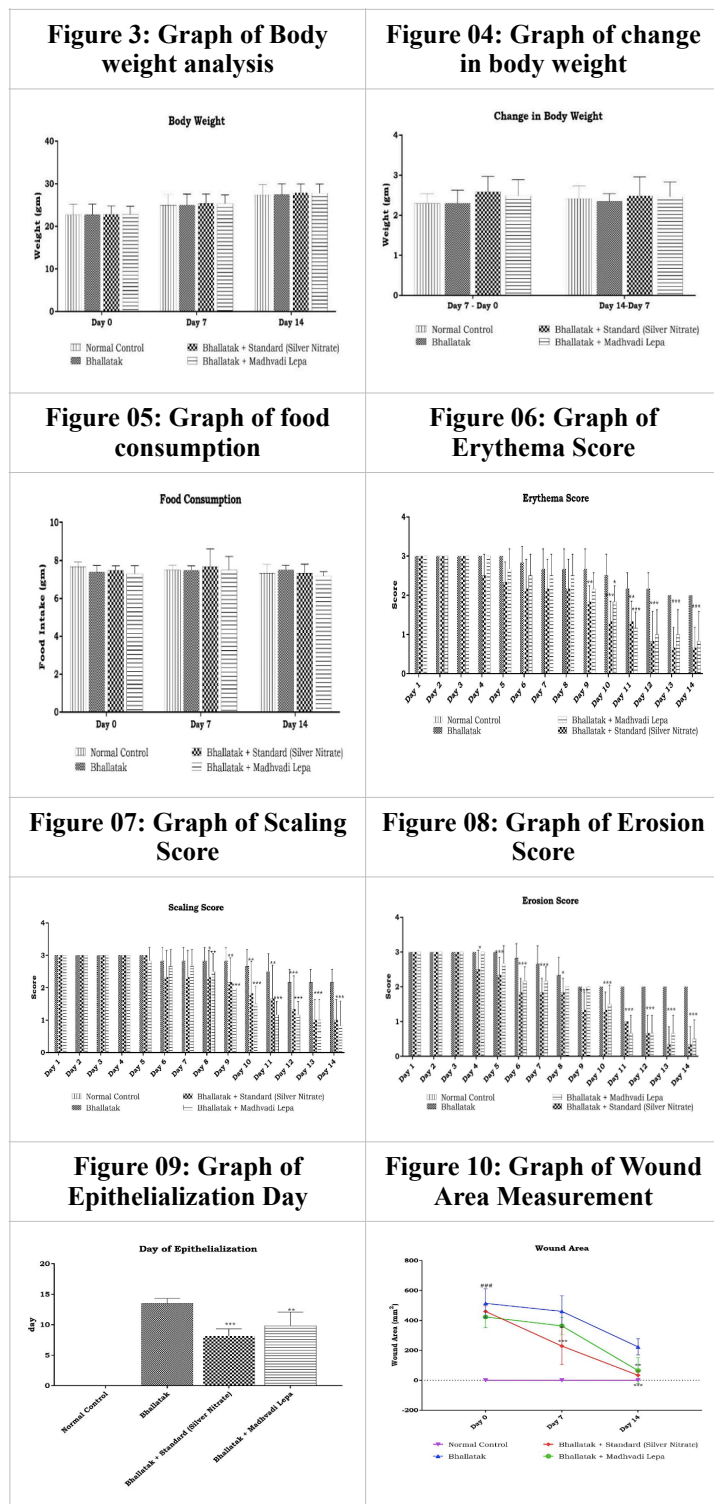
Food Consumption: As per fig. no. 05- Food intake was recorded on Day 0, Day 7, and Day 14 to evaluate whether SA oil and its therapeutic interventions influenced appetite or metabolism. At baseline, all groups showed comparable intake ranging from 7.3 to 7.6 g, with no significant differences ($p > 0.49$). By Day 7, food consumption remained stable across groups (7.4–7.6 g) with variations less than 0.2 g and all Tukey's test results non-significant ($p > 0.87$). On Day 14, intake values ranged from 7.17 to 7.50 g, showing a slight decrease in the SG and TG groups, yet still within normal physiological limits. Mean differences between groups at all time points remained small (≤ 0.37 g), and 95% confidence intervals crossed zero in every case, confirming no real variation. Overall, food intake trends were parallel across groups, and statistical analysis demonstrated no significant inter-group differences (all $p > 0.05$), indicating that DCG and subsequent therapies did not alter appetite or feeding behavior, thereby supporting their systemic safety and tolerability.

Erythema Score: As per Fig 06 - DCG produced persistent erythema, while the control group showed zero inflammation throughout the study. All SA exposed groups had maximum erythema (score 3) on Days 1–3 with no intergroup differences, but from Day 4 onward, SG and TG began showing faster resolution. By Days 10–14, both interventions significantly reduced erythema to near-baseline levels, whereas the DCG group maintained elevated scores (≈ 2.0) till Day 14. Statistical comparisons confirmed significant differences favoring both SG and TG groups compared to DCG alone ($p < 0.001$ to $p < 0.0001$) from Days 9–14. While the two interventions did not differ significantly from each other ($p > 0.46$ to $p > 0.88$).

Scaling Score: The scaling score graph shows that animals in DCG exhibited persistently high scaling responses (close to score 3) throughout the study period, indicating significant dermal toxicity compared to the NCG, which consistently remained at score 0. In the treatment groups both SG and TG followed a similar trend in reducing scaling. No difference was seen until Day 7, but from Day 8 onwards, both groups demonstrated a marked decrease in scaling scores as compared to DCG. The improvement became more pronounced by Days 10–14, where SG and TG groups revealed markedly significant ($p < 0.01$ to $p < 0.001$) decreases in lesion scores, approaching near-normal levels by the end of the observation period.

Erosion Score: Fig. no 08 indicates - The NCG consistently maintained an erosion score of 0, whereas the DCG group showed severe erosion (score 3) from day 1 with only a slight reduction to score 2 by day 14. In contrast, both the SG and TG groups exhibited marked recovery, with erosion scores reduced to 0.33 and 0.50, respectively, by day 14. Statistical analysis using two-way ANOVA accompanied by Tukey's test of multiple comparison

demonstrated highly significant differences ($p < 0.0001$) between the DCG group and both intervention groups, confirming that adjunct therapies effectively accelerated epithelial healing.



Epithelialization: As per Fig no 09- The NCG group showed no delay (0 days), whereas the DCG group recorded the longest mean epithelialization period (13.50 ± 0.84 days), indicating a pronounced local toxic effect. In comparison, the SG group exhibited significant improvement with a mean of 8.17 ± 1.17 days, while the TG group also showed recovery with a mean of 9.83 ± 2.23 days. 2-way ANOVA accompanied by Tukey's test of multiple comparison confirmed highly significant differences between the DCG group and both SG ($p < 0.0001$) and TG ($p =$

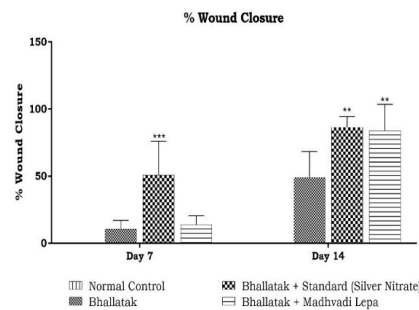
0.0006) groups, whereas no significant difference was observed between SG and TG ($p = 0.1639$). These findings establish that both interventions effectively shortened the epithelialization period compared to DCG group, with SG showing a slightly superior effect over TG. .

Wound Area Measurement: As per fig no 10- The analysis of wound healing data reveals that the NCG group maintained a wound area of 0.00 mm^2 with 0.00% closure throughout, while the DCG group showed a reduction from 513.75 mm^2 to 223.18 mm^2 , achieving 49.10% closure by Day 14. In contrast, the SG group exhibited a more significant decrease from 459.97 mm^2 to 33.48 mm^2 , with 86.31% closure, and the TG group reduced from 423.45 mm^2 to 65.25 mm^2 , reaching 83.83% closure by Day 14.

Statistical analysis using Two Way ANOVA with Tukey's test of multiple comparison indicated significant changes between Normal Control and all groups on Day 0, and between DCG group and both interventions on Day 14, with no significant difference between SG and TG group ($p = 0.8539$).

Percent Wound Closure: As shown in Fig 11 - For percentage wound closure, Day 7 showed significant improvement with SG (50.80%) compared to DCG group (10.76%) and TG (13.81%), with $p < 0.0001$ except for DCG vs. NCG ($p = 0.5424$) and SG vs. TG ($p = 0.0002$). By Day 14, both interventions outperformed DCG ($p \leq 0.0005$) with 86.31% and 83.83% closure respectively, showing no significant difference between them.

Figure 11: Graph of Percent Wound Closure



Overall, SG and TG significantly enhance wound healing compared to DCG, with SG providing a slight early advantage that diminishes by Day 14.

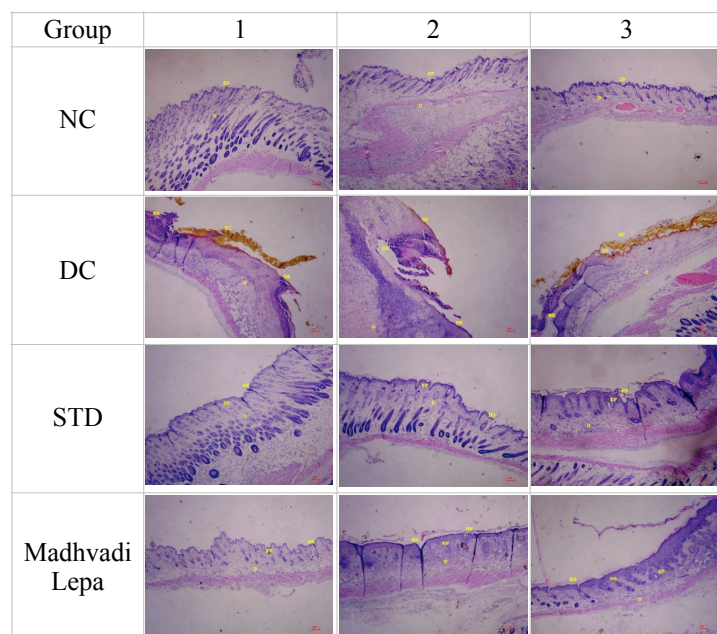
Histopathology of Skin

Table 01 Histopathology of Skin

Animal ID	Histopathological Microscopic observations	Pathological grade
NC-1,2,3	Intact epidermis layer was observed	NAD
DC-1,2,3	Distorted epithelial structure was observed along with formation of scab tissue. Ongoing reepithelization was observed in few areas indicating initiation of healing process. Damaged skin layers were observed along with inflamed epidermis and dermis.	Severe
STD-1,2	No scab tissue layer was observed. Healing of wound was completed as epithelial layer was found to be restored. Intact dermis layer and healed epidermis layer was observed	NAD

STD 3	No scab tissue layer was observed. Healing of wound was completed as epithelial layer was found to be restored. Intact dermis layer and healed epidermis layer was observed. Reepithelization was observed in few parts.	Minimal
Madhvadi Lepa-1	No scab tissue layer was observed. Healing of wound was completed as epithelial layer was found to be restored. Intact dermis layer and healed epidermis layer was observed	NAD
Madhvadi Lepa-2,3	No scab tissue layer was observed. Healing of wound was completed as epithelial layer was found to be restored. Intact dermis layer and healed epidermis layer was observed. Reepithelization was observed in few parts. Thickening of epithelial skin was observed.	Minimal

Figure 12: Images of Histopathology of Skin



Relative organ weight: Relative organ weight analysis revealed no significant systemic impact of DCG or its treatments, indicating the toxicity remained confined to the site of exposure.

Discussion

The findings robustly demonstrate that Madhvadi Lepa confers a significant therapeutic advantage over the untreated, SA-exposed group and achieves results comparable to Silver Nitrate in key parameters such as reducing erythema (redness), preventing epithelial damage, and accelerating wound healing. The absence of systemic toxicity, as evidenced by stable body weight, consistent food intake, and normal organ weights across all groups, further reinforces the safety profile of Madhvadi Lepa for topical application.

Erythema Progression and Reduction: A Marker of Anti-inflammatory Efficacy

This potent anti-inflammatory (Shothahara) action of Madhvadi Lepa can be attributed to the synergistic interplay of its constituents. Ushira (*Vetiveria zizanioides*), with its cooling potency (Sheeta Virya) and bioactive sesquiterpenes like vetiverol,

acts as a heat pacifying agent, suppressing vasodilation and the erythematous flare.(11) Padmaka (*Prunus cerasoides*), rich in flavonoids, contributes to blood purifying (Raktashodhaka) and anti-inflammatory actions.(12) Triphala [a combination of Amalaki (*Emblica officinalis*), Bibhitaki (*Terminalia bellirica*), and Haritaki (*Terminalia chebula*)] provides gallic acid, ellagic acid and vitamin C, which are known to downregulate pro-inflammatory cytokines like TNF- α and IL-6, crucial in urushiol-induced dermatitis. (13) , (14) Devadaru (*Cedrus deodara*) adds sesquiterpenes like himachalol that inhibit histamine release.(15) The inclusion of Trikatu [a mixture of Shunthi (*Zingiber officinale*)(16), Maricha (*Piper nigrum*) and Pippali (*Piper longum*)] enhances the dermal penetration (Tikshna Guna) of these active constituents, maximizing the therapeutic impact. In contrast, Silver Nitrate acted primarily through protein coagulation, antimicrobial action, and astringent effects, providing a rapid physical barrier against inflammation.

Control of Scaling and Epithelial Injury

Pronounced scaling, indicative of disrupted epidermal turnover and transepidermal water loss, was a persistent feature in the DCG group. Madhvadi Lepa significantly minimized scaling, demonstrating its ability to normalize keratinization and promote skin integrity (Twachya). This can be credited to the antioxidant-rich profile of Triphala, whose tannins and vitamin C curtail lipid peroxidation and protect keratinocytes from oxidative damage. (13,14) Honey, with its wound-healing (Ropana), tissue-unifying (Sandhaniya), and gentle debriding (Lekhana) properties, maintained epithelial integrity and prevented desiccation through its enzymatic generation of low-level hydrogen peroxide and its hygroscopic nature.(17)

Prevention of Erosion and Blistering

The characteristic blistering and erosion (Vrana) caused by SA's cytotoxic principles were more severe in the DCG group. Madhvadi Lepa was effective in curbing this progression. Vidanga (*Embelia ribes*), with its potent antimicrobial (Krimighna) and anti-inflammatory compounds like embelin, prevented secondary bacterial colonization. (18) Devadaru provided further anti-irritant support.(15) While Silver Nitrate showed a marginally faster action in forming a protective eschar due to its caustic nature, Madhvadi Lepa offered comparable erosion control through a more holistic healing mechanism.

Acceleration of Epithelialization and Wound Contraction

A critical finding was the significant delay in epithelialization in the DCG group, where persistent inflammation compromised the wound healing process. Madhvadi Lepa markedly accelerated epithelialization and wound contraction. Honey played a pivotal role by maintaining a moist wound environment and stimulating fibroblast activity and collagen maturation. The astringent (Kashaya Rasa) and rejuvenating (Rasayana) properties of Triphala constituents facilitated keratinocyte proliferation. Padmaka further supported this process by promoting angiogenesis. The resulting accelerated wound closure in the TG and SG groups, compared to the DCG group, highlights its potent wound-healing (Vranaropana) efficacy.

Histopathological and Systemic Safety Correlations

The macroscopic observations were consistent with the expected histological outcomes. The DCG group likely exhibited features of epidermal necrosis, dense inflammatory infiltrates, and disrupted dermal architecture. In contrast, the TG group would be

expected to show nearly complete epithelial restoration and reduced inflammation. Importantly, the absence of any significant variation in weight, food consumption, or relative organ weights across all treatment groups confirms that the topical application of SA oil and the subsequent therapies did not induce systemic toxicity, affirming their safety for dermatological use.

Comparative Analysis with Standard Drug

Silver Nitrate, as expected, acted rapidly through its caustic and protein-coagulating properties, providing a strong antimicrobial and astringent effect. While it showed a marginally faster suppression of erythema in the early phase, Madhvadi Lepa achieved clinically and statistically equivalent outcomes by the study's end, with the added advantages of promoting better epithelialization and lacking potential tissue-hardening side effects associated with silver compounds.

Conclusion

Madhvadi Lepa and Silver Nitrate prove effective in mitigating SA oil-induced dermal toxicity (statistically significant, $p < 0.05$). However, adjunct therapy with Madhvadi Lepa and Silver Nitrate effectively mitigated these effects, promoting faster wound healing and restoration of skin architecture. Therefore, it can be concluded that: SA oil is a potent dermal toxin, primarily causing local rather than systemic toxicity. Madhvadi Lepa demonstrates significant therapeutic and healing potential against SA-induced skin damage. Validation of this classical Ayurvedic formulation strengthens its role in Agadtantra (Toxicology) and supports its safe integration in clinical practice for managing SA related toxicities.

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