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RESEARCH ARTICLE

Histological Parameters to Assess The Impact of Topical Application of Rosemary Cream on Chemically Induced Oral Ulcer (Experimental Study)

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ABSTRACT

Natural herbal compounds are increasingly being used in dentistry to treat one of the most common oral disorders. An oral ulcer is a painful sore that can arise anywhere inside the oral cavity due to various circumstances. In this study twenty-seven Albino Wistar male rats were used and exposed to chemical buccal ulcers, then were categorized into a control (left side) and an experimental side (right side) that was specifically treated with varying concentrations of rosemary cream. The animals were sacrificed, the specimens were collected, and they were assessed to investigate the effect of rosemary cream on buccal ulcers by investigation of histological characteristics.

A wounded area presents early signs of healing on a histological level. Statistically, it records a highly significant difference in inflammatory cell intensity and angiogenesis at varying studied periods of wounding, and it shows increased mean values of the experimental groups treated with rosemary cream independent of concentrations compared to control sides.

The current study revealed that rosemary cream promotes wound healing by modulating the inflammatory response and triggering angiogenesis.

Keywords: Angiogenesis, chemical ulcer, rosemary cream, wound healing.

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1. Introduction

Trauma-induced ulcers damage the mucosa of the oral cavity produced by physical or mechanical trauma or chemical burns, such as acetic acid, piercing by sharp objects, deformed or carious teeth, or sharp edges [1]. Chemical trauma is responsible for oral ulcers which can lead to a poor prognosis and necessitate intensive treatment [2]. Oral chemical burns are typically induced by exposure to acidic or alkaline insults. Both acids and alkalis adversely affect the oral tissues through several mechanisms. Acids induce coagulative necrosis of tissues, resulting in Escher development that restricts the infiltration of substances into the deeper mucosa layers [3]. Alkalis interacts with tissue proteins, resulting in liquefactive necrosis and saponification, infiltrates deeper into tissues, and is associated with markedly poor prognosis [4]. Chemical burns in the oral cavity are a significant cause. Chemical shock to the oral cavity mucosa causes serious tissue damage that jeopardizes tissue integrity, leading to intraoral burns following thermal burns. As necrosis progresses, the tissue sloughs off, exposing the highly vascularized connective tissue caused by a heightened inflammatory response in the affected area. This can occur because of the cytotoxic effects of alkaline and acidic substances, that disrupt tissue integrity. Effective care requires prompt identification of the chemical, the extent of exposure, period between harm and treatment, and location of injury [5], [6].

A chemical burn of the oral mucosa results from a harmful agent coming into direct contact with the mucosa, either by the patient or dentist. Superficial chemical burns of the mucosa can heal rapidly, often within one- two weeks, owing to the rapid turnover of the oral mucosa [7].

The process of wound healing is intricate and multidimensional, involving both the closure and contraction of the wound, as well as the restoration of functional barriers.

Local application of corticosteroids, such as dexamethasone and the use of antibiotics are current therapy methods for oral chemical burn healing; however, they need to be replaced by better alternatives because of their high cost, adverse side effects, and increased antibiotic resistance. Additionally surgical debridement is necessary to eliminate necrotic tissue and hasten the formation of granulation tissue [8].

In addition to helping reduce or eliminate the possible negative effects of chemical drugs, herbal medicines have long been used to prevent and control diseases. Aloe vera, ginger, clove, cinnamon, garlic, neem, miswak, turmeric, tulsi, green tea, chamomile, fenugreek, anise plant, peppermint, bloodroot, caraway, eucalyptus, phyllanthus emblica, black seed, myrrh, and rosemary are herbs that may serve as alternatives to the current treatments for oral conditions such as gingivitis, periodontitis, oral burn, ulcers, inflammation, and caries prevention [9].

Rosemary (*Rosmarinus officinalis* L.) is a perennial shrub that is native to southern Europe and Asia, particularly the Mediterranean region, and is grown all over the worldwide. Numerous researches have demonstrated the antibacterial, anti-inflammatory, antioxidant, anti-apoptotic, and anti-tumorigenic properties of rosemary [10]. It works well against Escherichia coli, Vibrio cholerae, Staphylococcus aureus, and Staphylococcus albus [11].

Targeted treatment strategies based on natural bases, which should be created and approved to encourage wound healing and avoid problems, are efficient in treating chemical burns in the oral cavity. Therefore, this study was designed to assess the impact of the topical application of rosemary cream on chemically induced oral ulcers by examining histopathological parameters.

2. Method and Materials

2.1. Animal Preparation

Twenty-seven male Albino Wister rats (body-weight: 300–350 g; 8–16 weeks) were used. All rats were administered intraperitoneal ketamine and xylazine injections (50 mg/kg; 1 ml/kg of body weight, 2%; 0.2 ml/kg) and a single application of 70% acetic acid for 60 seconds in order to cause chemical ulcers. using a micro-dental brush soaked in 70% acetic acid for 3−5 sec. to achieve a uniform ulceration [12]. The left side of the oral buccal mucosa of all animals was considered as the control side and left to heal spontaneously [13], while the right oral buccal mucosa was considered as the experimental side and treated by topical application of rosemary cream at three different concentrations (5%, 10%, and 15%) daily starting a day after ulceration (day 1, Fig. 1). In accordance with the institutional and national rules for the care and use of animals, all rats were treated in accordance with protocols and recommendations.

2.2. Histological Examination

- Histological Parameters: The study was designed to evaluate histological parameters in three healing periods (3, 5, and 7 days) after induction of a chemical ulcer on the buccal mucosa. Buccal mucosa specimens were removed, sectioned into 5 µm slices, fixed with 10% formalin, and stained with hematoxylin and eosin.
- Inflammatory Cell intensity was observed under a light microscope by selecting five random fields under a power of x40. Following a census of the inflammatory cells in each field, the mean number of cells was determined as follows: As stated by Nowicka (2016) [14],

– Score 1: There were either no inflammatory cells or very few cells.

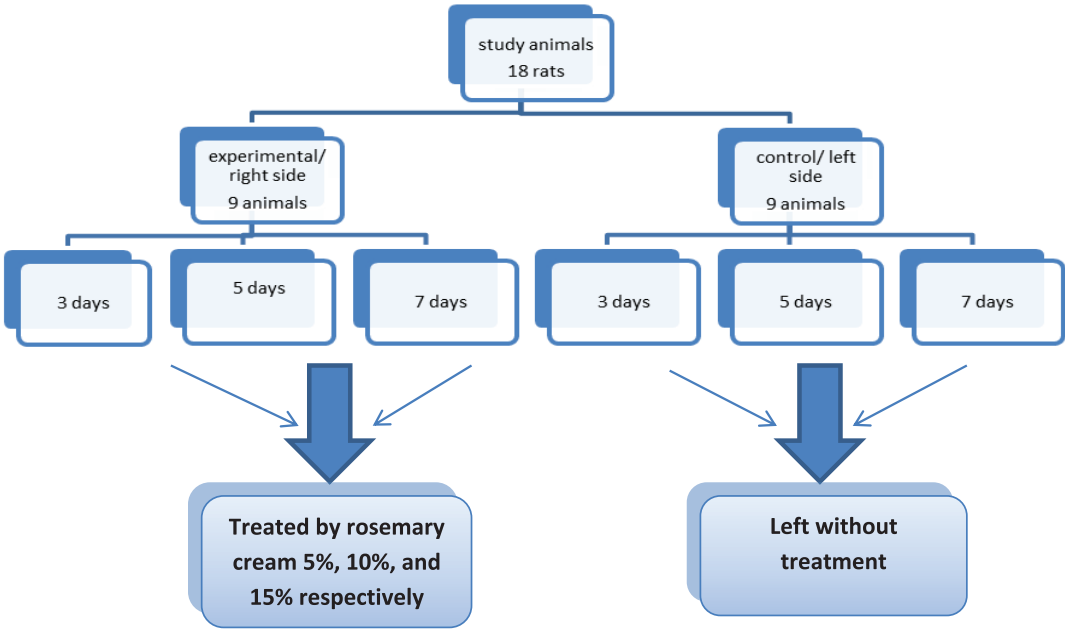

Fig. 1. The design of the study according to the healing periods and concentration of rosemary cream.

– Score 2: Mild (less than 10 inflammatory cells on average).
– Score 3: Moderate, with 10–25 inflammatory cells on average.
– Score 4: Severe (>25 inflammatory cells on average).

- Angiogenesis (new blood vessels formation) was determined by counting four different visual fields per section and the mean values were calculated by [15].

2.3. *Statistical Analysis*

Statistical analysis were performed by using SPSS (v 20). The obtained data were subjected to a t-test to compare the experimental and control groups for each concentration at the studied healing periods, while one-way ANOVA was used to compare the mean value of the three concentrations at the same time. Statistical significance was set at P >0.05, while a p value ≤0.05 was significant or highly significant respectively. Estimation of the correlation coefficient between different parameters in this study.

3. Results

3.1. *Evaluation of Histological Parameters*

- At Day 3 of Wounding (Fig. 2): the control side showed marked necrosis of fibrous tissue & infiltration of leukocytes (a). While the experimental side treated with 5% rosemary cream showed marked ulceration with a wide area of tissue necrosis and profound infiltration of leukocytes (b); the wounded area treated with 10% cream showed focal necrosis of fibrous tissue with focal aggregation of leukocytes and vascular congestion (c), and additionally, the wounds treated with 15% rosemary cream showed necrotic fibrous tissue with observed infiltration of inflammatory cells (d).
- At Day 5 of Wounding (Fig. 3): the control side showed marked ulceration with a wide area of fibrous tissue necrosis, little formation of granulation tissue, and profound infiltration of leukocytes (a); in contrast, the experimental sides showed marked ulceration with a marked necrotic tissue plug, an inflammatory zone, and degeneration of necrotic tissue with severe infiltration of leukocytes (b), (c). Additionally, the side treated with the 10% cream showed good angiogenesis (d).
- At Day 7 of Wounding (Fig. 4), the control side showed ulceration with loss of lining cells, infiltration of leukocytes, and mild angiogenesis (a). The experimental side treated with 5% showed less inflammatory response than the 15% -treated area (b), (c) respectively, where as 10% rosemary cream provoked significant angiogenesis (d).

3.2. *Statistical Results*

A highly significant increase ($p < 0.001$) in inflammatory cell intensity and angiogenesis was observed in the experimental groups compared to the controls on day 3, 5, and 7 of wounding, Across all treatment periods and concentrations of rosemary cream, the experimental groups consistently exhibited higher mean values, with the most pronounced effect recorded on day 3 of wounding in the 5% rosemary cream group (Table I and II), while the highest overall mean value was observed in the 10% rosemary cream group on day 5 of wounding, Fig. 5 further confirms these findings.

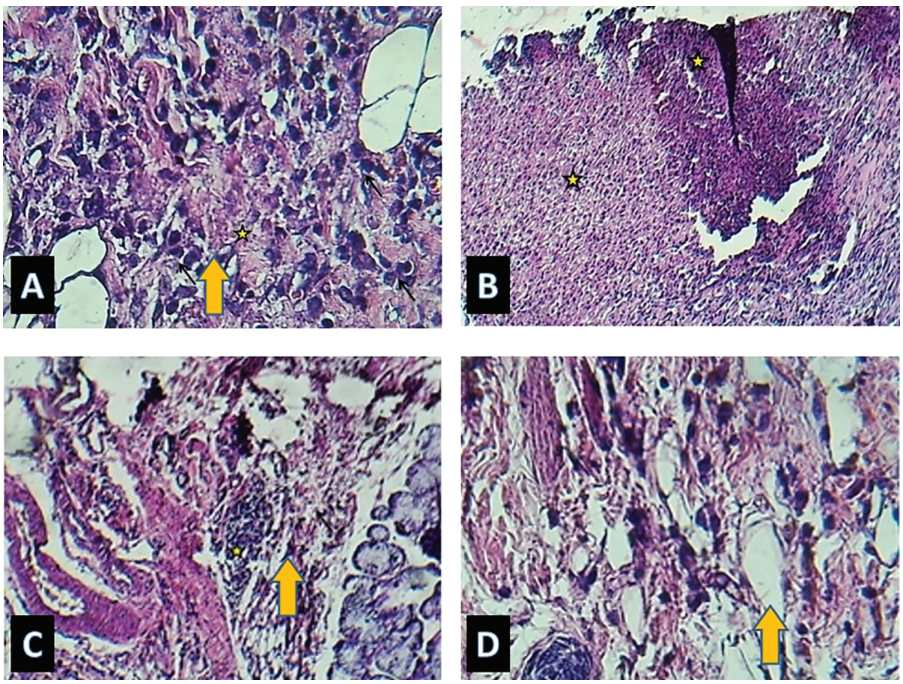

Fig. 2. Histological view of buccal mucosa on day 3. (A) Necrotic tissue (asterisks), inflammatory cells aggregation (yellow arrow), H&E 40x, (B) leukocytes infiltration (Asterisks), H&E 10x, (C) necrotic tissue (yellow arrow), H&E 10x, (D) infiltration of leukocytes (yellow arrow), H&E 40x.

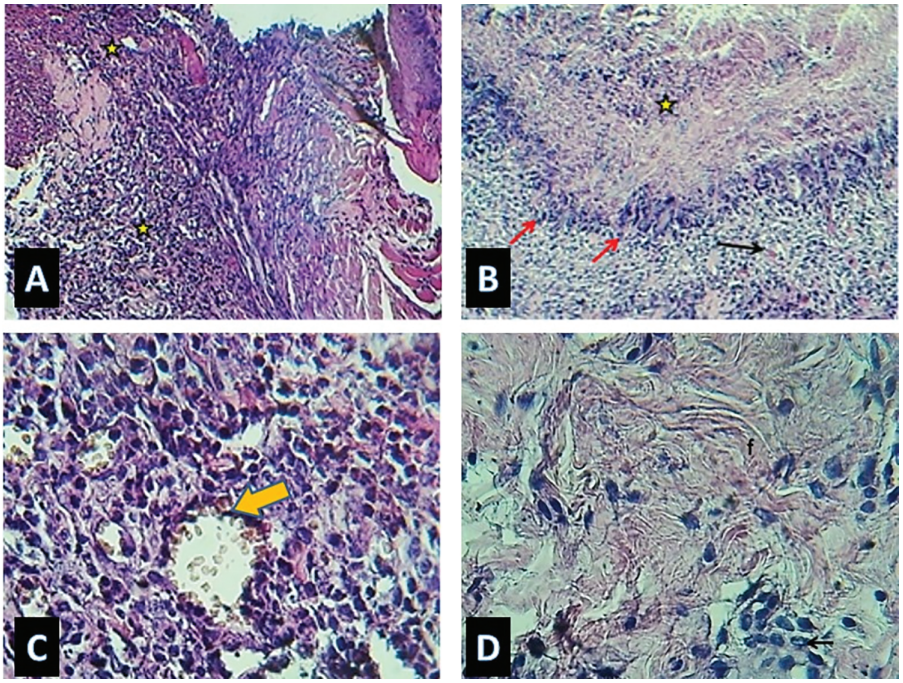

Fig. 3. Histological view of wound site on day 5. (A) Infiltration of leukocytes (asterisks), H&E 10x, (B) inflammatory zone (red arrows), and infiltration of leukocytes (black arrows), H&E 40x, (C) well angiogenesis (yellow arrow), H&E 10x, (D) fibrous tissue -f-, H&E 40x.

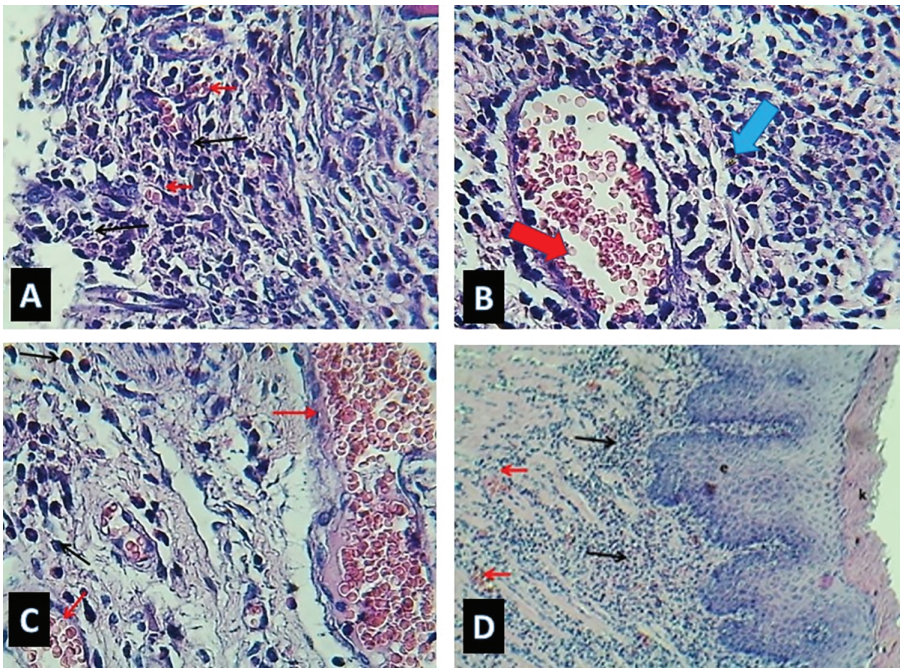

Fig. 4. Histological view of wound site on day 7, H&E 40x. (A) Infiltration of leukocytes (black arrows) mild angiogenesis (red arrows), (B) infiltration of leukocytes (blue arrow), vascular congestion (red arrow), (C) vascular congestion (red arrows) with infiltration of leukocytes (black arrows), (D) hyperkeratosis [k] acanthosis of lining cells -e-, vascular congestion (red arrows) with little infiltration of leukocytes (black arrows).

The current results showed that treatment with rosemary cream enhanced angiogenesis in experimental groups relative to the controls. Among the concentrations tested, 5% of rosemary cream demonstrated the most sustain action, maintaining elevated angiogenesis up to day 7. In comparison, 10% rosemary cream group exhibited a peak angiogenic response on day 5 followed by regression, while the 15% rosemary cream group induced the greatest vascular response at day 3 but decline thereafter, reaching its lowest level on day 7 (Fig. 6).

4. Discussion

Oral ulceration refers to the disruption of the oral mucosa. It is a debilitating condition marked by painful, superficial ulcerations that can effectively impair a patient's daily activities, dietary intake, and general quality of life [16]. Beginning with hemostasis and progression through inflammation, proliferation, and remodeling, wound healing is a four-phase process involving interactions between various cells and tissue components. Immunosuppression, whether local or systemic, extends

TABLE I: Group Comparison in Mean of Inflammatory Cell Intensity in Different Healing Periods and Various Cream Concentrations

Inflammatory cell intensity					
Time duration	Tested groups		Mean ± SE		P value
3 Days	Control side	9.6 ± 0.6	23 ± 2.5	25.3 ± 1.5	0.001
	Experimental side	5%	10%	15%	0.002
		11.3 ± 2.6	22 ± 4.3	20.6 ± 3.2	
	P value	0.05	0.17	0.04	
5 Days	Control side	9 ± 1	22.3 ± 5.3	18 ± 1.4	0.001
	Experimental side	5%	10%	15%	0.001
		13 ± 2.1	25 ± 3.6	21 ± 2.5	
	P value	0.04	0.05	0.05	
7 Days	Control side	10.6 ± 1.7	22.3 ± 2.1	20 ± 3.1	0.001
	Experimental side	5%	10%	15%	0.001
		14.6 ± 1.3	22.6 ± 1.2	20.3 ± 1.4	
	P value	0.05	0.87	0.91	

TABLE II: Experimental Sub-Groups Comparison of Mean Angiogenesis at Different Healing Periods and Cream Concentrations

Angiogenesis					
Periods of healing	Tested groups		Mean ± SE		P value
3 Days	Control side	13.67 ± 3.2	30.3 ± 4.5	29.3 ± 5.13	**0.002**
	Experimental side	5%	10%	15%	**0.001**
		7.76 ± 1.5	19 ± 3.5	17.3 ± 3.4	
	P value	**0.02**	**0.001**	**0.001**	
5 Days	Control side	14 ± 2	30.3 ± 2.5	26.3 ± 1.5	**0.001**
	Experimental side	5%	10%	15%	**0.001**
		15 ± 4.4	22.67 ± 5.03	16.33 ± 5.13	
	P value	0.78	**0.02**	**0.01**	
7 Days	Control side	10.67 ± 1.1	15 ± 1	12 ± 1.2	**0.002**
	Experimental side	5%	10%	15%	0.001
		15.3 ± 3.01	22 ± 3.6	12.3 ± 1.1	
	P value	**0.05**	**0.02**	0.89	

the inflammatory phase and postpones wound healing. [17] Thus, the current study focused on this phase and investigated the histological parameters to assess the effectiveness of topical application of rosemary cream at various concentrations. Hemostasis and chemotaxis are the first steps in the inflammatory phase. Thrombocytes and white blood cells increase the inflammatory process [18], and discovered the antithrombotic properties of rosemary, which significantly reduced platelet reactivity and improved flow-mediated vasodilation. This finding may explain the pronounced inflammatory effects observed during various healing periods. Recent measurements of the mean values of inflammatory cells in the experimental groups treated with varying cream concentrations exhibit significant results corroborating [19], who indicated that rosmarinic, another component of rosemary, is associated with inflammation triggering through modulation of the transcription factor (NF-κB) pathway, In addition to its antioxidant impact and increased collagen cross-linking, it is a crucial regulator of the immune system and inflammatory response [20].

Animal studies have suggested that rosemary oil may aid in the recovery of burns. Numerous studies have demonstrated that rosemary extract at concentrations of 1.5%–3% or its separated constituents at 2–128 µg/mL exhibit greater antimicrobial activity against S. aureus than lower concentrations or controls [21]. Unfortunately, few clinical studies have been conducted to assess the potential advantages of rosemary in burn healing.

Additional mediators and cytokines, including transforming growth factor alpha (TNF-α) and interleukins (IL-1, IL-6, and IL-8), are released during the inflammatory phase to facilitate chemotaxis. Apart from platelet-derived growth factors, a number of other factors promote fibroblast transformation, collagen breakdown, angiogenesis, and re-epithelialization. Together with transforming growth factor (TGF), platelet-derived growth factor draws fibroblasts and encourages their division and proliferation. Fibroblasts subsequently make collagen [22], which aligns with the present histological findings, indicating that the wounded area on experimental sides treated with rosemary cream, regardless of concentration, exhibited significant angiogenesis, inflammatory response, collagen deposition, and restoration of the mucosal epithelial discontinuities resulting from chemical ulceration, particularly on day 7 of injury (Figs. 3 & 4). This is also consistent with the findings of [23], as the antioxidant properties of rosemary extract are particularly important in directly eliminating reactive oxygen species (ROS), which prevent oxidative damage to biological molecules and programmed cell death (apoptosis). It is also linked to a significant reduction in IL-6 synthesis, caspase-3 and

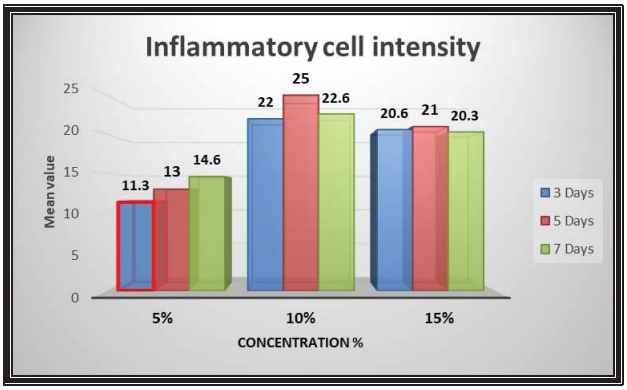

Fig 5. Mean values of inflammatory cell intensity among experimental sub-groups treated with various concentrations of rosemary cream.

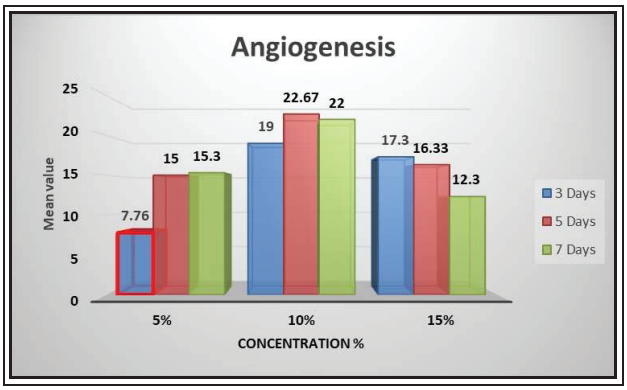

Fig 6. Mean values of angiogenesis (new blood vessels formation) among experimental groups treated with various concentrations of rosemary cream.

-9 activities, and DNA damage. Furthermore, previous studies on wound healing demonstrated that adding 0.25–0.75 weight percent powdered rosemary ethanolic extract to polylactic acid (PLA) enhanced the biocompatibility of the extracted product while simultaneously boosting its antibacterial and antioxidant qualities [24].

The current findings demonstrate that rosemary cream significantly enhanced the mean value of angiogenesis compared to the control side. This contrasts with previous researches regarding the anti-angiogenic properties of two key constituents of rosemary extract, specifically carnosol and carnosic acid, which may aid in the treatment of certain angiogenesis-related malignancies due to their chemo-preventive, anti-tumoral and anti-metastatic activities by inhibiting endothelial cell proliferation and exerting an effect on flow- mediated vasodilation [25].

The treatment with rosemary cream significantly enhanced angiogenesis in the experimental groups compared to the controls. This effect may attributed to Rosmarinus officinalis essential oil (ROEO), a major constitute of rosemary, which has antioxidant properties and is known to increase serum vascular endothelial growth factor (VEGF) a key regulator of angiogenesis in wound healing [26].

4.1. Limitation of The Study

This study has some limitations that should be declared: First, the small size was relatively small due to expensive experimental procedures that may limits the generalizability of the present findings.

Second, the experimental duration was short. Furthermore, the current study relied on histological evaluation; incorporating additional molecular markers or biochemical assays. Further experimental and clinical researches addressing these limitations would strengthen the evidence for the therapeutic efficacy of rosemary cream in wound healing.

5. Conclusion

Chemical injury is one of the many causes of oral ulcers, and proper management begins with early diagnosis and use of recommended natural extracts treatments to reduce pain and ulcer duration. This study revealed that rosemary cream triggers the inflammatory activity and enhances wound healing. Topical rosemary cream at various concentrations (5%, 10%, and 15%) improved the histological characteristics of chemically induced ulcers.

5.1. Recommendation for Further Studies

Based on the current findings, it is suggested that rosemary cream can be further evaluated in other oral mucosal injury models, such as traumatic, thermal ulcer and recurrent aphthus stomotities.

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Conflict of Interest

None.

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