EXPERIMENTAL STUDY

The effect of nicardipine on the zone of stasis in burns: An experimental rat model

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ABSTRACT

BACKGROUND: The zone of stasis in burns is particularly vulnerable to progressive ischemia, making it a critical target for therapeutic interventions. Preventing damage in this zone is essential, as its viability can be preserved with adequate perfusion. Recognizing this, we aimed to investigate the systemic effects of nicardipine, a calcium channel blocker with vasodilatory properties, on the stasis zone in an experimentally induced burn model in rats. We hypothesized that nicardipine could mitigate ischemic progression in the stasis zone and thereby preserve tissue viability.

METHODS: A total of 20 Wistar-Albino rats were included in this study and divided into two groups: a control group (n=10) and a treatment group (n=10). The experimental burn model described by Regas and Ehrlich was employed. Under anesthesia, a 1 x 2 cm metal comb, preheated in boiling water, was applied to the dorsal skin of the rats for 30 seconds to create burn wounds. No treatment was administered to the control group. The treatment group, however, received a daily systemic dose of nicardipine (5 mg/kg) via gastric lavage for three days. Wound healing was monitored daily using a digital camera for three consecutive days. One rat in the treatment group was excluded due to mortality. After three days, the burned areas were excised from the dorsal skin of all rats and subjected to histopathological examination. Additionally, photo analysis of the burn areas was conducted using data obtained from the digital images.

RESULTS: Nicardipine treatment significantly improved burn healing parameters in the stasis zone. Compared to the control group, the treatment group demonstrated lower scores for edema (0.78 vs. 2.80, $p<0.05$), congestion (0.22 vs. 2.80, $p<0.05$), inflammation (0.67 vs. 2.90, $p<0.05$), vascularization (0.11 vs. 2.70, $p<0.05$), and fibrosis (0.22 vs. 2.90, $p<0.05$). Quantitative measurements also revealed a significant reduction in necrosis zone thickness (1079.75 µm vs. 2818.82 µm, $p<0.05$) and necrosis area (249.33 µm² vs. 400.13 µm², $p<0.05$). These findings indicate that nicardipine effectively mitigates ischemic progression and promotes tissue recovery in burn injuries.

CONCLUSION: Our experimental study demonstrated that nicardipine has the potential to prevent and treat damage in the burn stasis zone, suggesting its therapeutic role in burn injuries.

Keywords: Burn; nicardipine; zone of stasis.

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INTRODUCTION

Burns are defined as tissue damage resulting from exposure to heat, electricity, radiation, radioactive rays, or chemical substances. Beyond the physical injury, burns pose significant psychological challenges and are a major cause of mortality and morbidity.[1]

The primary pathological consequence of burns is coagulation necrosis, the depth of which is determined by the intensity and duration of thermal exposure. Burn injuries can be classified into three distinct zones, as described by Jackson in 1947: the zone of coagulation, located at the center of the injury, represents the area of direct contact and irreversible cell damage; the zone of stasis, surrounding the coagulation zone, is characterized by ischemic injury that develops within 24-48 hours post-burn; and the zone of hyperemia, located at the outermost area, demonstrates increased blood flow as part of the inflammatory response and remains viable.[2]

The preservation of the stasis zone is a primary focus in burn treatment, as this area has the potential to maintain its viability with adequate perfusion. Preventing progressive ischemia and subsequent necrosis in the stasis zone is crucial for reducing the depth and extent of the injury, enhancing treatment success, and minimizing complications. Various pharmacological agents have been investigated experimentally to preserve the stasis zone and mitigate ischemic damage.[3-9]

In this study, we aimed to evaluate the effects of nicardipine, a calcium channel blocker and potent vasodilator, on the stasis zone in an experimentally induced burn model in rats. We hypothesized that nicardipine could alleviate progressive ischemia in the stasis zone and preserve tissue viability.[10]

MATERIALS AND METHODS

Ethical approval for the study was obtained from the Ethics Committee of the Ministry of Health, Turkish Public Hospitals Institution, Ankara Province First Regional Public Hospitals Union General Secretariat, and the Health Sciences University Ankara Training and Research Hospital. The ethics committee approval (Approval Number: 782) was granted on July 22, 2024. The study was conducted in accordance with the Declaration of Helsinki.

A total of 20 male Wistar-Albino rats, weighing an average of 270 grams (range: 250-290 g), were used in the study. The rats were housed in groups of five per cage at room temperature under a 12-hour light/dark cycle and had ad libitum access to water and standard laboratory food containing 20% protein. Prior to surgery, the rats were fasted for at least 12 hours without solid food intake.

To induce anesthesia, 50 mg/kg ketamine (Ketalar®) and 5 mg/kg xylazine (Rompun®) were administered intramuscularly. The depth of anesthesia was confirmed by assessing skeletal muscle tone. The dorsal skin of each rat was shaved before the procedure.

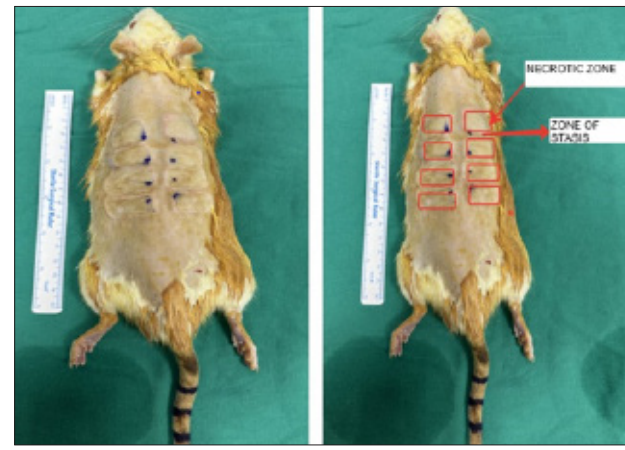

Figure 1. Burn wounds on dorsal skin with standardized positioning and spacing.

To create burn wounds, 1x2 cm brass metal combs were preheated in boiling water for 15 minutes and applied to the dorsal skin without pressure for 30 seconds. This resulted in a total of eight 1x2 cm burn areas per rat (four on the right and four on the left), positioned 0.5 cm lateral to the vertebrae with 0.5 cm spacing between each burn (Fig. 1).

The rats were randomly assigned to two groups: a control group (Group 1, n=10) and an experimental group (Group 2, n=10). The control group (Group 1) received no medication. The experimental group (Group 2) was administered nicardipine (Ninax®) at a dose of 5 mg/kg via gastric lavage, given once daily at the 0, 24, and 48 hours, for a total of three doses.

The healing of the burn areas was monitored daily using a digital camera for three days. A millimeter ruler was included in each photograph for calibration purposes. One rat in the experimental group died on the first day and was excluded from the study.

At the end of the three-day study period, the rats were sacrificed, and the burn areas were excised. The samples were fixed in formalin solution and sent to the pathology unit for histopathological examination.

Photoanalysis

Daily photographs of the burned areas in both groups were taken, with the initial surface area of each burn standardized as 200 mm². The AutoCAD® software program was used to calculate the burned areas in the photographs. A millimeter ruler placed in each photograph allowed for calibration, ensuring accurate calculation of the burned area in square millimeters (Fig. 2). Necrotic areas were measured, and the obtained values were recorded (Table 1). At the end of the third day, the total necrotic areas between the two groups were compared.

Figure 2. Burn area measurement using AutoCAD® with calibrated photographic analysis.

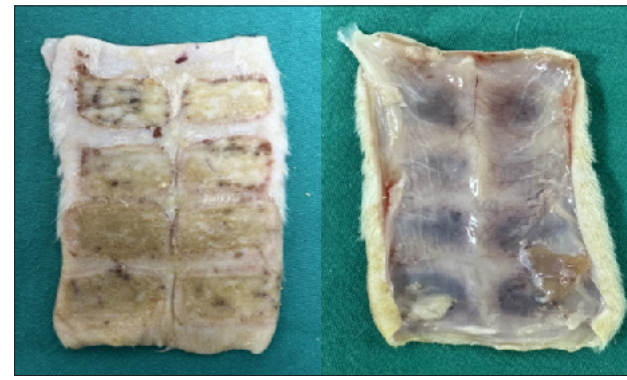

Figure 3. Excised burn wounds preserved in formaldehyde for pathological examination.

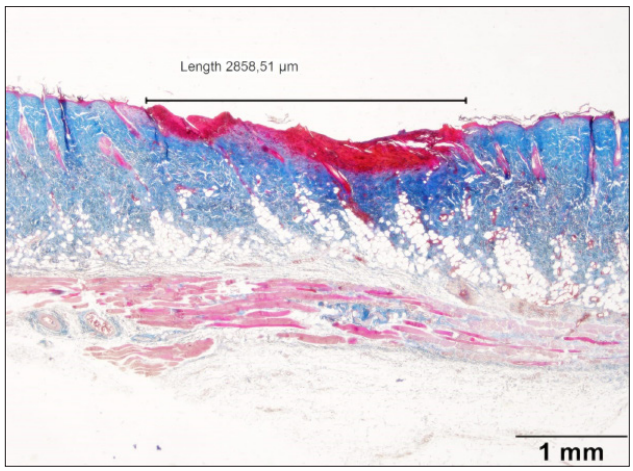

Figure 4. Control group necrosis zone.

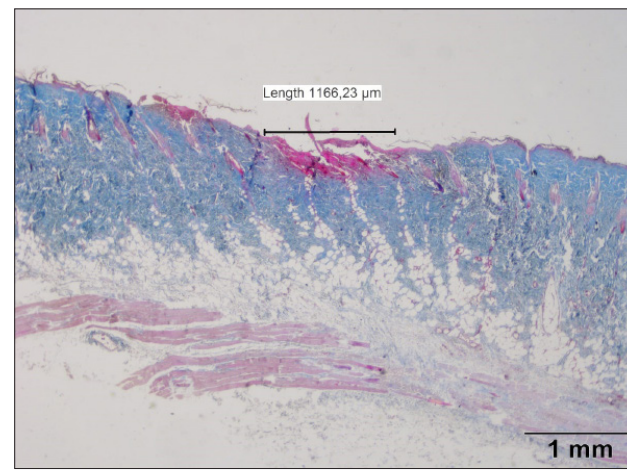

Figure 5. Nicardipine Group Necrosis Zone.

Histological Analysis

After the rats were sacrificed, the burned skin areas on their backs were excised and preserved in formaldehyde solution for pathological examination (Fig. 3). Hematoxylin-eosin staining was performed. Semi-quantitative scoring was conducted on a scale from 0 to 3 for parameters such as edema, congestion, inflammatory infiltration, vascular proliferation, and fibrosis. Numerical values were obtained by analyzing slide images of the necrosis zone (Fig. 4 and 5), and average values were calculated from the selected preparations. The results of these analyses are summarized in Table 1.

Statistical Analysis

The effect of nicardipine on the healing of burn areas in rats was investigated. Nineteen rats were included in the study, with 10 assigned to the control group and nine to the treatment group. The comparison of edema, congestion, inflammation, vascularization, fibrosis, necrosis zone thickness (µm), and necrosis area values between the control and treatment groups is presented in Table 2. Statistical analyses were performed using the Independent Samples T-Test and Chi-Square Test (Tables 2 and 3).

RESULTS

The parameters of edema, congestion, inflammation, vascularization, fibrosis, necrosis zone thickness (µm), and necrosis area were analyzed, and significant differences were observed between the control and treatment groups (Table 3).

The mean edema score was significantly lower in the nicardipine-treated group (0.78±0.12) compared to the control group (2.80±0.15) ($p<0.05$). Similarly, the mean congestion score in the nicardipine group (0.22±0.10) was markedly reduced compared to the control group (2.80±0.20) ($p<0.05$). The treatment group also exhibited a significant reduction in inflammation, with a mean score of 0.67±0.18 compared to 2.90±0.25 in the control group ($p<0.05$).

Vascularization scores followed the same trend, with the nicardipine-treated group showing a mean score of 0.11±0.05, substantially lower than the control group's mean score of 2.70±0.18 ($p<0.05$). Fibrosis was also significantly reduced in the treatment group, with a mean score of 0.22±0.08 compared to 2.90±0.20 in the control group ($p<0.05$).

In addition to these qualitative parameters, quantitative measurements revealed significant differences in necrosis zone

Table 1. Macroscopic photoanalytical data (necrotic burn area) and histopathological data

Group	Necrotic Burn Area (mm²)	Edema (0-3)	Congestion (0-3)	Inflammation (0-3)	Vascular Proliferation (0-3)	Fibrosis (0-3)	Necrotic Zone (μm)
C1	434.08	2	2	2	2	2	2642.13
C2	461.58	3	3	3	3	3	2833.72
C3	393.15	3	3	3	3	3	2715.62
C4	324.48	3	3	3	3	3	2858.51
C5	428.15	2	3	3	2	3	2794.36
C6	379.89	3	3	3	3	3	2903.17
C7	428.16	3	3	3	3	3	2891.67
C8	344.99	3	3	3	2	3	2795.12
C9	409.47	3	3	3	3	3	2817.07
C10	397.34	3	3	3	3	3	2936.82
N1	120.20	1	0	0	0	0	1061.94
N2	184.88	2	2	2	1	2	1207.23
N3	204.12	0	0	0	0	0	1142.36
N4	243.67	1	0	1	0	0	1018.49
N5	309.89	2	0	2	0	0	1198.07
N6	275.16	0	0	0	0	0	953.18
N7	274.83	0	0	0	0	0	1166.23
N8	293.59	1	0	1	0	0	1005.96
N9	337.64	0	0	0	0	0	964.27

Control Group (C1-C10) and Nicardipine Group (N1-N9). One rat was lost during the study.

Table 2. Comparison of edema, congestion, inflammation, vascularization, fibrosis, necrosis zone (μm), and necrosis area values between control and treatment groups (test statistic: independent samples t-test)

Variable	Group	N	Mean	SD	p
Edema	Control	10	2.80	0.42	0.000**
	Nicardipine	9	0.78	0.83	
Congestion	Control	10	2.80	0.42	0.000**
	Nicardipine	9	0.22	0.67	
Inflammation	Control	10	2.90	0.32	0.000**
	Nicardipine	9	0.67	0.87	
Vascularization	Control	10	2.70	0.48	0.000**
	Nicardipine	9	0.11	0.33	
Fibrosis	Control	10	2.90	0.32	0.000**
	Nicardipine	9	0.22	0.67	
Necrosis Zone (μm)	Control	10	2818.82	88.93	0.000**
	Nicardipine	9	1079.75	100.32	
Necrosis Area	Control	10	400.13	41.92	0.000**
	Nicardipine	9	249.33	68.60	

thickness and necrosis area between the groups. The mean necrosis zone thickness was 2,818.82±123.45 μm in the control group and 1,079.75±98.35 μm in the treatment group ($p<0.05$). Similarly, the mean necrosis area was 400.13±15.72 μm² in the control group and 249.33±12.88 μm² in the nicardipine-treated group ($p<0.05$).

Table 3. Comparison of Edema, Congestion, Inflammation, Vascularization, Fibrosis, Necrosis Zone (μm) and Necrosis Area Values of Rats in Control and Treatment Groups.

Variable		Control Group		Treatment Group		
		f	%	f	%	p
Edema	Absent	0	0.0	4	44.4	0.000**
	Mild	0	0.0	3	33.3	
	Moderate	2	20.0	2	22.2	
	Severe	8	80.0	0	0.0	
Congestion	Absent	0	0.0	8	88.9	0.000**
	Mild	2	20.0	1	11.1	
	Severe	8	80.0	0	0.0	
Inflammation	Absent	0	0.0	5	55.6	0.000**
	Mild	0	0.0	2	22.2	
	Moderate	1	10.0	2	22.2	
	Severe	9	90.0	0	0.0	
Vascularization	Absent	0	0.0	8	88.9	0.000**
	Mild	0	0.0	1	11.1	
	Moderate	3	30.0	0	0.0	
	Severe	7	70.0	0	0.0	
Fibrosis	Absent	0	0.0	8	88.9	0.000**
	Moderate	1	10.0	1	11.1	
	Severe	9	90.0	0	0.0	

*p<0.05, **p<0.01, Chi-Square Test.

Proportional analyses further supported these findings (Table 3). Severe edema, congestion, inflammation, vascularization, and fibrosis were consistently more prevalent in the control group, while these parameters were significantly reduced or absent in the nicardipine-treated group. For instance, severe edema was observed in 80% of the control rats, whereas only 20% of the treated rats exhibited mild edema, and 40% had no edema. Severe congestion was present in 80% of the control group, but only 10% of the treated group showed moderate congestion, with the remaining rats showing no signs of congestion.

These results indicate that nicardipine significantly improves key histopathological parameters in burn injuries, including reducing necrosis and inflammation and promoting better tissue recovery in the stasis zone. These findings align with previous studies emphasizing the efficacy of pharmacological agents in improving outcomes in experimental burn models.

DISCUSSION

Recent advancements in burn research have focused on pharmacological interventions to preserve the stasis zone. Various agents have demonstrated efficacy in experimental models. Nitroglycerin, a topical anti-ischemic agent, has been shown to enhance vascular proliferation and improve burn wound healing.[3] Sildenafil and N-acetylcysteine have demonstrated antioxidative and vasodilatory effects, reducing oxidative stress and accelerating wound healing.[4] Similarly, udenafil, a potent phosphodiesterase inhibitor, has been found to reduce necrotic areas and inflammation while improving the histological viability of the stasis zone.[5]

Systemic treatments have also shown promise. Pentoxifylline has been reported to accelerate epithelialization, reduce necrotic areas, and prevent the deepening of burn wounds, while low molecular weight heparin (LMWH) enhances angiogenesis, fibroblast proliferation, and capillary density in the stasis zone.[6,7] Advanced therapies, such as platelet-rich plasma (PRP), have demonstrated significant benefits, including reduced apoptosis, increased viable tissue, and decreased inflammation in the stasis zone.[11] Similarly, Tarantula Cubensis extract (TC) has been shown to improve macroscopic viability and promote epithelial regeneration in the stasis zone, facilitating overall burn wound healing.[12]

Other agents, such as melatonin, taurine, apocynin, and magnesium-adenosine triphosphate (ATP) nanoliposomes, have demonstrated efficacy in experimental burn models. For example, magnesium-ATP nanoliposomes have been found to enhance blood perfusion in ischemic areas, preserving tissue

viability and supporting healing in the stasis zone. These studies highlight the multifactorial nature of stasis zone injury and the importance of targeting ischemia, inflammation, and oxidative stress simultaneously.[8-9,13]

Furthermore, calcium channel blockers have been studied in rat models for their potential to increase flap viability. Pal et al.[14] investigated the effect of nifedipine on ischemic skin flap survival in rats. Similarly, Kılınç et al.[15] studied the effects of verapamil, nifedipine, and daflon on the viability of reverse island flaps in rats. However, the number of studies examining calcium channel blockers in burns remains limited. While it has been shown that a verapamil hydrochloride (HCl)-based biopolymer enhances burn wound healing, neither the systemic nor the topical form of nicardipine has been studied in burns to date.[16]

In our study, we evaluated the effects of nicardipine, a calcium channel blocker with potent vasodilatory properties, on the stasis zone in an experimental burn model in rats. Nicardipine was hypothesized to mitigate progressive ischemia, improve perfusion, and preserve tissue viability. The findings of our study demonstrated significant improvements in key histopathological parameters, including reduced necrosis and inflammation and increased vascularization in the nicardipine-treated group compared to controls. These results align with previous studies highlighting the benefits of vasodilatory agents in preserving the stasis zone.[3-6]

Our study has some limitations; we have not examined the side effects of nicardipine. However, these effects have been previously documented and include hypotension, impaired perfusion, and increased peripheral edema.[17] Due to these systemic side effects, the use of nicardipine may exacerbate hypovolemia and peripheral edema in burn patients. On the other hand, McCann et al. highlighted in their review that burns involving more than 25% of the total body surface area (TBSA) are associated with a systemic inflammatory response.[18] Based on this, non-topical use of nicardipine may be less likely to aggravate burn-related systemic complications in patients with burns covering less than 25% TBSA. For more severe burns, where a systemic inflammatory response is more likely, the topical application of nicardipine, similar to nitroglycerin, may be considered a safer alternative.

In our study model, the non-topical administration of nicardipine limits the generalizability of our recommendations regarding its topical use. Another limitation is the lack of investigation into its optimal dosage. Future studies should focus on optimizing its topical formulation, determining the ideal dosage, and exploring its potential combination with other agents. Additionally, further research is needed to evaluate its safety and efficacy in clinical settings.

Ethics Committee Approval: Ethical approval for the study was obtained from the Ethics Committee of the Ministry of Health, Turkish Public Hospitals Institution, Ankara Province First Regional Public Hospitals Union General Secretariat, and the Health Sciences University Ankara Training and Research Hospital (Date: 22.07.2024, Decision No: 782).

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Authorship Contributions: Concept: R.D., M.İ., N.T.B., N.A., N.Y., N.B., U.Z.; Design: R.D., M.İ., N.T.B., N.A., N.Y., N.B., U.Z.; Supervision: R.D., M.İ., N.T.B., N.A., N.Y., N.B., U.Z.; Resource: R.D., M.İ., N.T.B., N.A., N.Y., N.B., U.Z.; Materials: R.D., M.İ., N.T.B., N.A., N.Y., N.B., U.Z.; Data Collection and/or Processing: R.D., M.İ., N.T.B., N.A., N.Y., N.B., U.Z.; Analysis and/or Interpretation: R.D., M.İ., N.T.B., N.A., N.Y., N.B., U.Z.; Literature Review: R.D., M.İ., N.T.B., N.A., N.Y., N.B., U.Z.; Writing: R.D., M.İ., N.T.B., N.A., N.Y., N.B., U.Z.; Critical Review: R.D., M.İ., N.T.B., N.A., N.Y., N.B., U.Z.

Conflict of Interest: None declared.

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DENEYSEL ÇALIŞMA - *ÖZ*

Nikardipin'in yanık staz zonu üzerine etkisi: Ratlarda deneysel bir çalışma

AMAÇ: Yanık staz zonu, uzayan iskemiye karşı çok hassastır ve bu iskemiyi engellemek yanık tedavisinin bir hedefi haline gelmiştir. Yanık staz zonundaki olası hasarı önlemek önemlidir, çünkü bu bölgenin canlılığı yeterli perfüzyon sağlanırsa korunabilir. Çalışmamızda ratlarda deneysel olarak oluşturulan bir yanık modelinde bir vazodilatör ve kalsiyum kanal blokörü olan Nikardipin'in sistemik olarak uygulanmasının yanık staz zonu üzerinde etkilerini araştırmayı amaçladık. Nikardipin'in yanık staz zonundaki iskemik süreci aaltabileceğini ve böylece doku perfüzyonunu ve canlılığını koruyabileceğini düşündük.

GEREÇ VE YÖNTEM: Çalışmaya toplam 20 Wistar-Albino rat dahil edildi. Ratlar 10 tanesi kontrol grubu 10 tanesi tedavi grubu olmak üzere 2 gruba ayrıldı. Regas ve Ehrlich tarafından tanımlanan deneysel yanık modeli kullanıldı. Yanık yaraları oluşturmak için kaynar suda önceden ısıtılmış 1x2 cm'lik metal çubuklar, anestezi altındaki ratların sırt derisine 30 saniye boyunca uygulandı. Kontrol grubuna hiçbir tedavi uygulanmazken, tedavi grubuna 3 gün boyunca gastrik lavaj yoluyla sistemik olarak 5 mg/kg dozunda Nikardipin verildi. Yara iyileşmesi 3 gün boyunca dijital kamera kullanılarak takip edildi. Tedavi grubundaki ratlardan biri exitus nedeniyle çalışmadan çıkarıldı. 3. Günün sonunda ratların sırt derisi yanık alanları dahil edilerek eksize edildi ve histopatolojik incelemeye gönderildi. Ek olarak, dijital görüntülerden elde edilen veriler kullanılarak yanık alanlarının fotoğrafik analizi yapıldı.

BULGULAR: Nikardipin tedavisi staz zonundaki yanık iyileşme parametrelerini önemli ölçüde iyileştirdi. Kontrol grubuyla karşılaştırıldığında, tedavi grubu ödem (0.78'e karşı 2.80, $p<0.05$), konjesyon (0.22'ye karşı 2.80, $p<0.05$), inflamasyon (0.67'ye karşı 2.90, $p<0.05$), vaskülarizasyon (0.11'e karşı 2.70, $p<0.05$) ve fibrozis (0.22'ye karşı 2.90, $p<0.05$) için daha düşük skorlar gösterdi. Kantitatif ölçümler ayrıca nekroz bölgesi kalınlığında (1079.75 µm'e karşı 2818.82 µm, $p<0.05$) ve nekroz alanında (249.33 μm^2'ye karşı 400.13 μm^2, $p<0.05$) önemli bir azalma ortaya koydu. Bu bulgular, nikardipinin yanık yaralanmalarında iskemik ilerlemeyi etkili bir şekilde azalttığını ve doku iyileşmesini desteklediğini göstermektedir.

SONUÇ: Ratlarda yapmış olduğumuz deneysel çalışma, Nikardipin'in yanık staz zonundaki hasarı önleme ve tedavi etme potansiyeline sahip olduğunu ve yanık yaralanmalarında terapötik bir rol oynayabileceğini göstermektedir.

Anahtar sözcükler: Nikardipin; staz zonu; yanık.

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