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Efficacy of Silicone Gel Plus Thrombin Adhesives on Cutaneous 2nd Degree Burn Healing

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Abstract
A lot of researchers try many types of therapies for burns healing in Veterinary medicine in the last decades. Silicone gel may decrease the scar tissue, while thrombin may increase the proliferation of cells. This project aims to compare the efficacy of silicone gel combined and thrombin on 2nd degree cutaneous burn healing. Forty female white albino rats, who were housed in plastic boxes divided randomly into four groups 10 rats for each. Control group G1 had no treatment, second group G2 treated with silicone gel, third group treated with autologous thrombin adhesive, fourth group treated with combination of silicone gel plus autologous thrombin adhesive. Under routine surgery with general anesthesia all rats had thermal injuries at their backs. The thermal injuries were then created using a solid aluminum bar with a diameter of 2 cm in diameter had previously been heated to 100°C in boiling water. The bar is kept in touch with the rat's skin in the dorsal proximal region of the back. Hyperemia, edema, scar tissue, and scab were recorded for morphological exam. The therapies were applied topically once daily for 10 days. At the time of burn and on the appropriate days for the biopsies at 7th ,14th,21th, and 28th day, sterile "swabs" were taken and sent to laboratory for bacterial contamination. Biopsies were collected for histopathologic evaluation of healing. The morphologic appearance was recorded at 1 day, 7th day, 14th day,21th day, and 28th day, it was the best in G3. A completed wound healing was recorded highly significant wound contraction in G3 groups. The bacterial colonies count was recorded the lowest in G3.
Histopathologic evaluation showed the superiority of thrombin adhesive than silicone gel for 2nd degree burns healing and this result accompanied with the other parameters. Histological changes post burn injury in thrombin treatment showed complete healing of all skin layers including epidermis, papillary dermis, reticular dermis, and hypodermis, hair follicles, sweat glands, and sebaceous glands were obvious while in silicone gel showed regenerated epidermis layer, gaps between epidermis and dermis, edema or bulla like lesion between epidermis and dermis, subcutaneous edema, and infiltration of inflammatory cell in the dermis mostly lymphocytes.**E**. The study concluded that silicone gel decreases scar formation and thrombin accelerate burn healing.
Keyword: silicone, gel, thrombin, adhesive, cutaneous, burn, healing

Introduction

Burns are severe injuries that may result in loss of tissue fluids and are associated with tissue destruction, infection, pain or even death (1). Burns represent a significant socioeconomic burden, due to loss of income and increasing health care costs. Causative agents include fire, scald and contact with hot/cold objects. Burn injury healing is a complex process involving a dynamic series of events, including clotting, inflammation, granulation tissue formation, epithelialization, collagen synthesis and tissue remodeling. This is still the subject of a large number of studies conducted by several researchers, especially regarding factors that delay the process of tissue healing and treatments for this injury (2). Burn healing is a complex process including dynamic series of events as the clotting, inflammation, granulation tissue formation, epithelialization, collagen synthesis and tissue remodeling (2,3). Healing of burns injury is still difficult to achieve although the evolution of antiseptic, medications and advanced operation procedures. For years silicone has been used as a gold standard for burn related hypertrophic scar management (4; 5). In recent studies, one of the effective non-surgical burn scar treatment methods is using of silicone surfaces. Various mechanisms have been proposed to account for the effect of silicon, including mechanisms of moisture, pressure, temperature, oxygen transfer and silicon absorption. But its mechanism of action is still unclear. In Australia, (6) had used silicone gel to treat burn scars. It is a unique molecule that functions both as a procoagulant and anticoagulant. In its procoagulant role it activates platelets through its receptor on the platelets. It regulates its own generation by activating coagulation factors V, VIII and even XI resulting in a burst of thrombin formation. It activates factor XI, thus preventing fibrin clots from undergoing fibrinolysis. Thrombin not only cleaves fibrinogen to fibrin, but also through the activation of factor XIII effects the cross-linking of fibrin monomers to produce a firm fibrin clot. The aim of the study was to compare the efficacy of silicone gel and thrombin on cutaneous 2nd dgree burn healing.

Materials and Methods

Ethical approval

The ethical committee of the College of Veterinary Medicine at the University of Al-Qadisiyah, Iraq, gave its approval to the current investigation.

Samples

Fourty female white albino rats, who were housed in plastic boxes, divided randomly into four groups 10 rats for each. Control group G1 had no treatment, second group G2 treated with silicone gel, G3 treated with thrombin, and G4 treated with combination of silicone gel plus autologous thrombin. Silicone gel tubes were purchased from a local

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pharmacy. Using 2 ml syringes, heart blood was taken from G3 and G4 in order to make thrombin. Dynamically incubating the filled syringes for 20 min caused thrombin and fibrin to be produced as a result of coagulation. Empty syringes of the same size were placed in the rotor while being hosed to the incubated syringes. Centrifuging the attached syringe pairs at 930 g for 20 min allowed the serum containing thrombin to be separated from the cellular and fibrin clot components. Then, automatically, the thrombin component was injected into the empty syringe (7). Silicone gel was combined with thrombin at a 2 to 1 ratio. The mixture was autoclave sterilized for 15 min. at 121–124°C (8).Under routine surgery with general anesthesia all rats had thermal injuries at the left side of their backs. The thermal injuries were then created using a solid aluminum bar with a diameter of 2 cm in diameter had previously been heated to 100°C in boiling water. The bar is kept in touch with the rat's skin in the dorsal proximal region of the back. Hyperemia, edema, scar tissue, and scab were recorded for morphological exam. The therapies were applied topically once daily for 10 days. At the time of burn and on the appropriate days for the biopsies at 7th ,14th,21th, and 28th day, sterile "swabs" were taken and sent to laboratory for bacterial contamination. Biopsies were collected for histologic evaluation of healing. The morphologic appearance was recorded at 1 day, 7th day, 14th day,21th day, and 28th day. Statistical Analysis: The significance between groups was determined using statistical analysis of the morphometric data using the ANOVA test and Least Significant Difference (LSD) to the level of P≤ 0.05.

Figure 1: Autologous thrombin adhesive

Figure 2: Burn induction

Results

Clinical Evaluation

Table-1 showed the clinical signs of the burn along the period of the study 0-day, 7th day, 14th day, 21st day, 28th day. The Clinical signs were hyperemia, edema, crust, and scar tissue. G1 showed sever hyperemia and edema at 0 day while moderate hyperemia and mild edema and crust at 7th day. At 14th day there were mild hyperemia, mild crust, and scar tissue. At 21st day moderate crust and mild scar tissue. At 28th day there were sever crust and mild scar tissue. G2 showed moderate hyperemia and crust with mild edema at 7th day. At 14th day showed mild hyperemia and scar tissue with moderate crust. At 21st day showed mild crust and scar tissue while mild crust in 28th day. G3 showed mild hyperemia and crust at 7th day. At 14th day showed mild crust and scar tissue. At 21st and 28th days showed absent of clinical signs of the burn. G4 showed mild hyperemia and edema with moderate crust at 7th day. At 14th day showed mild hyperemia and scar tissue with moderate crust. At 21st day showed mild crust and moderate scar tissue. At 28th day showed mild crust and scar tissue.

Table 1: Clinical parameters recorded in the experimental rats at different periods.

Groups	periods	Clinical signs			
		Hyperemia	Edema	Crust	Scar tissue
G1 (n=10)	0 day	+++	+++	-	-
	7th day	++	+	+	-
	14th day	+	-	+	+
	21st day	-	-	++	+
	28th day	-	-	+++	+
G2 (n=10)	7th day	++	+	++	-
	14th day	+	-	++	+
	21st day	-	-	+	+

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	28th day	-	-	+	-
G3 (n=10)	7th day	+	-	+	-
	14th day	-	-	+	+
	21st day	-	-	-	-
	28th day	-	-	-	-
G4 (n=10)	7th day	+	+	++	-
	14th day	+	-	++	+
	21st day	-	-	+	++
	28th day	-	-	+	+

* The intensity of clinical signs was scored as -: absent, present: +: mild, ++: moderate, +++: severe.

Wound Contraction: Wound contraction parameter was an essential for burn healing evaluation. Table-2 showed the mean ± SE of the diameter's values of the burn contraction along the period of the study 7th day, 14th day, 21th day, 28th day. G1 recorded significant 1.81±0.02, 1.67±0.03, 1.48±0.04, 1.17±0.02 respectively. G2 recorded significant 1.67±0.03, 1.45±0.03, 0.95±0.03, 0.64±0.04 respectively. G3 recorded highly significant 1.5±0.03, 1.37±0.04, 0.65±0.03, 0.37±0.04 respectively. G4 recorded significant 1.79±0.03, 1.64±0.02, 0.93±0.03, 0.71±0.02 respectively. A completed wound healing was noticed in animals in G3 treated with autologous Thrombin adhesive highly significant wound contraction than the other group (G1, G2, G4) treated animal in all periods of wound healing phases in 7th ,14th ,21st ,28th, respectively.

Table 2: Mean ± SE of the diameters of Burn contraction.

Groups	Diameters of burn (mm.)			
	7th day	14th day	21st day	28th day
G1 (n=10)	1.81±0.02Aa	1.67±0.03Ab	1.48±0.04Ac	1.17±0.02Ad
G2 (n=10)	1.67±0.03Aa	1.45±0.03Bb	0.95±0.03Bc	0.64±0.04Bd
G3 (n=10)	1.5±0.03Ba	1.37±0.04Bb	0.65±0.03Cc	0.37±0.04Cd
G4 (n=10)	1.79±0.03Aa	1.64±0.02Ab	0.93±0.03Bc	0.71±0.02Bd

* Capital letters refers to the vertical statistical comparison, whereas small letters refer to the horizontal statistical comparison.
* Different letters denote to the significant difference at P<0.05, whereas similar letters refer to the no significant difference.

Bacterial contamination

Bacterial contamination might affect the burn healing. Table-3 showed the Mean± SE bacterial colonies counts (CFU/ ml.) post the treatments of the experimental rats along the period of the study 3rd day, 7th day, 14th day, 21th day, 28th day. The bacterial colonies count of G1 recorded 15.2±1.42, 107.1±2.05 , 117.1±1.16 , 133.5±2.68 , 141.2±3.07 , respectively were significantly along the period of the study. G2 recorded 15.10±1.17 , 95.3±1.88 , 66.6±4.33 , 32.4±2.03 , 11.6±0.4 , respectively were significantly at 3,7,14 and 21 day while non-significant at 28 day. G3 recorded 13.3±1.06 , 87±1.98 , 58.7±4.32 , 35.3±3.56 , 8.1±0.61 , respectively were significantly at 3,7,14 and 21 day while non-significant at 28 day. G4 recorded 12.3±1.41 , 96.8±1.31 , 86.2±1.42 , 25.7±3.77 , 17±0.55 , respectively were significantly at 3,7,14 and 21 day while non-significant at 28 day.

Table 3: Mean± SE bacterial colonies counts (CFU/ ml.) post the treatments of the experimental rats.

Groups	Post treatment period				
	3rd day	7th day	14th day	21st day	28th day
G1 (n=10)	15.2±1.42Aa	107.1±2.05Ab	117.1±1.16Ac	133.5±2.68Ad	141.2±3.07Ae
G2 (n=10)	15.10±1.17Aa	95.3±1.88Bb	66.6±4.33Bc	32.4±2.03BCd	11.6±0.4BCa
G3 (n=10)	13.3±1.06Aa	87±1.98Bb	58.7±4.32Bc	35.3±3.56Bd	8.1±0.61Ba
G4 (n=10)	12.3±1.41Aa	96.8±1.31Bb	86.2±1.42Cc	25.7±3.77Cd	17±0.55Ca
G5 (n=10)	9.4±1.03Aa	7.6±13.5Cb	6.7±1.10Da	7.7±0.55Da	6.6±0.70Ba

*Different letters mean the variances were significant at P≤ 0.05.
*LSD = 9.59

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Table 4: Bacterial contamination rate (%) of post burn wounds according to colonies count.

Groups	Colonies count (%)				
	3rd day	7th day	14th day	21th day	28th day
G1	1.5%	30%	15%	38%	50%
G2	1.5%	23%	19%	16%	13%
G3	1.2%	18%	22%	19%	18%
G4	1.2%	15%	13%	12%	9%

Morphological appearance showed different phases of burn healing at 1,7,14,21 and 28 days.

Morphologic appearance of cutaneous burn of the animal groups showed at 0, 7, 14, 21, and 28th days. At 0 day there were pale sharp edge of the burn, at 7th day the burn still covered with scab with clear reduction of the burn size of all groups than G1 (pic.no.1-5) while G3 (pic.no.11-15) showed decrease in size. At 14th day G3 (pic.no.11-15) showed significant decrease in size than other groups, complete cover of scar tissue in most groups. At 21th day, more size reduction occurred in G2 (pic.no.6-10), G3, G4 (pic.no.16-20), when compared with G1 especially with G3.Complete epithelialization was seen with less scar tissue.At 28th day complete healing were noticed in G3 so that it was difficult to distinguish the healed burn from the normal skin.

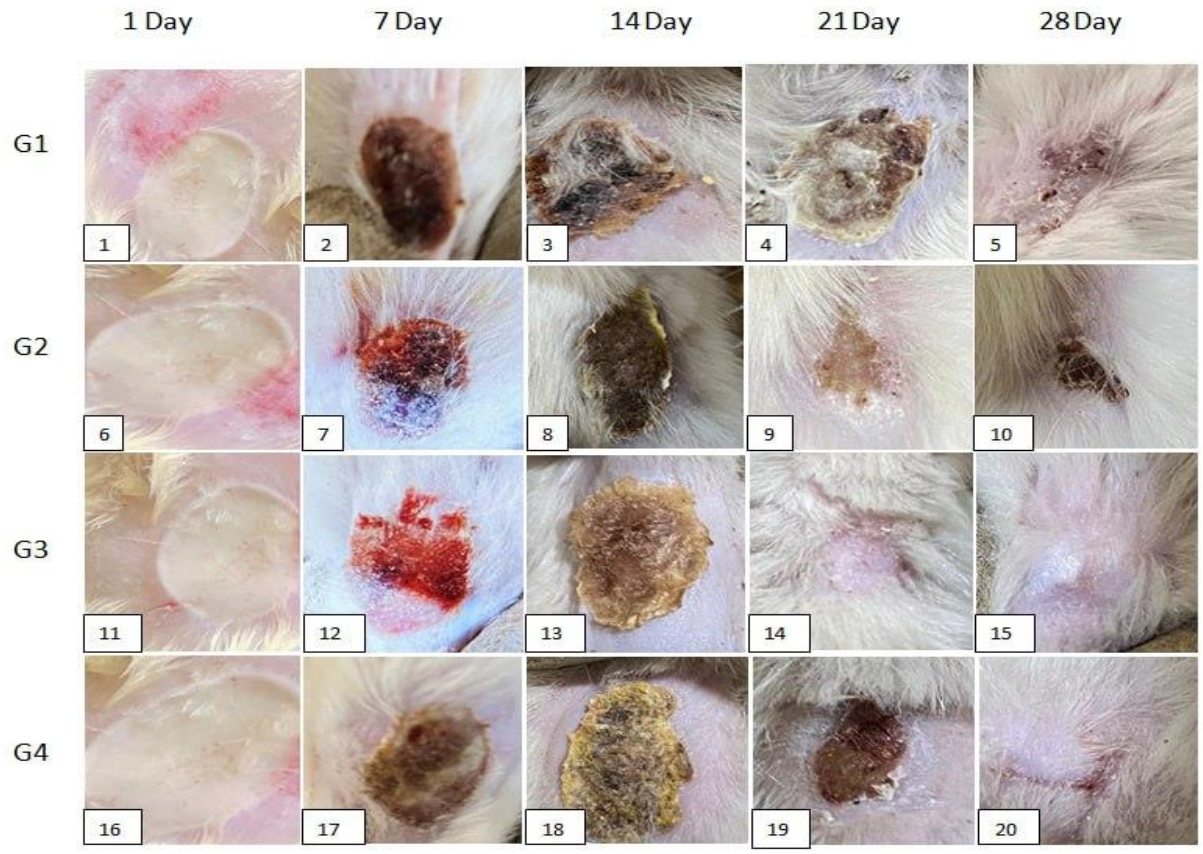

Figure 3: Morphological appearance of burn in all groups at different periods.

Histologic evaluation:

The histologic assessment reveals the following changes of each group for four weeks. G1-1w.: Histological changes following burn injury showed the presence of remnants of the epidermis layer full of infiltrating inflammatory cells, the presence of a gap between the epidermis layer and the dermis, the infiltration of a large number of inflammatory cells in the boundary between the two layers below the gap mostly lymphocytes, extravasated RBCs (EVRBCs), edema, accumulation of few fibroblast cells , and the absence of the characteristic hair follicles and sebaceous and sweat glands as showed in fig.-4. Fig.-5: w1-G2. Histological changes following burn injury

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showed severe destruction and necrosis of the epidermis layer, presence of gap and edema between epidermis and dermis, Loss of the normal microarchitecture of the epidermis and hair follicle, severe infiltration off inflammatory cells (red arrow; INFLC) mostly neutrophils (N) surrounding a bulla like lesion (B) that forming edema (ED) in dermis. H&E, 40X, 100X, and 400X. Fig.-6: W1-G3. Histological changes following burn injury showed severe destruction and necrosis of the both epidermis layer, presence of gap and edema between dermis and epidermis, severe edema, but initial re-epithelization and hair follicles were seen, as well as infiltration of inflammatory cells beneath epidermis were evident. H&E, 40X, 100X, and 400X. Fig.-7: W1-G4. Histological changes following burn injury showed severe destruction and necrosis of the both epidermis (EP) and dermis (DE) layer, presence of gap between dermis and epidermis, severe edema, no hair follicles, sweat, and sebaceous glands were seen, as well as severe infiltration of inflammatory cells in the subcutaneous layer. H&E, 40X, 100X, and 400X. Fig.-8: W2-G1. Histological changes following burn injury showed destructed epidermis and thick scab formation, subepidermal and dermal edema and severe inflammatory cells infiltration (INFLC). The inflammatory infiltrated cells are mostly epidermis neutrophils (green arrow) and macrophages as well as extravasated RBCs were seen. H&E, 40X, 100X, and 400X.Fig.-9: W2-G2. Histological changes following burn injury showed formation of thick scab covered by inflammatory cells, destruction and necrosis of the epidermis layer, Loss of the normal microarchitecture of the dermis, and infiltration of inflammatory cells (INLFC). H&E, 40X, 100X, and 400X. Fig.-10: W2-G3. Histological changes following burn injury showed well stage of re-epithelization, but gaps between the re-epithelized epidermis and dermis layers was evident, a new formatted blood vessels were seen surrounded by inflammatory cells. H&E, 40X, 100X, and 400X. Fig.-11: W2-G4. Histological changes following burn injury showed partial re-epithelization of stratum basale layer, destructed epidermis, subepidermal edema (ED), and infiltration of the neutrophils (N) were evident. H&E, 40X, 100X, and 400X. Fig.-12: W3-G1. Histological changes following burn injury showed partial re-epithelization (blue arrow) of stratum basale (2) and stratum spinosum (1), subepidermal edema (ED) were evident. H&E, 40X, 100X, and 400X. Fig.-13: W3-G2. Histological changes following burn injury showed partial re-epithelization, thick scab formation, subepidermal, severe infiltration of inflammatory cells (INFLC) in the epidermis and dermis, neutrophils (blue arrow) and lymphocytes (pink arrow) were evident. H&E, 40X, 100X, and 400X. Fig.-14:W3-G3. Histological changes following burn injury showed complete re-epithelization of epidermis, where all layers of epidermis were seen, but a subepidermal and dermal edema (ED), infiltration of inflammatory cells (N) and fibroblasts (blue arrow) were evident. H&E, 40X, 100X, and 400X. Fig.-15:W3-G4. Histological changes following burn injury showed uncovered area of skin, partial re-epithelization of two layers of epidermis including stratum basale and stratum spinosum layers, large subepidermal (ED), infiltration of inflammatory cells in the epidermis and dermis, mostly neutrophils (N), were evident. H&E, 40X, 100X, and 400X. Fig.-16:W4-G1. Histological changes following burn injury showed complete re-epithelization of epidermis (EP), but a subepidermal edema was evident, newly formed hair follicles (HF), sweat glands (SWG), and sebaceous glands (SBG) were seen, but subcutaneous edema (SCE) also present. H&E, 40X, 100X, and 400X. Fig.-17:W4-G2. Histological changes following burn injury showed regenerated epidermis layer , gaps between epidermis and dermis, edema or bulla like lesion (ED) between epidermis and dermis, subcutaneous edema (SED), and infiltration of inflammatory cell in the dermis (INFLC) mostly lymphocytes . H&E, 40X, 100X, and 400X. Fig.-18:W4-G3. Histological changes following burn injury showed complete regenerated skin, all layers of the epidermis are normal, a new formed hair follicles were seen in the dermis. H&E, 40X, 100X, and 400X. Fig.-19:W4-G4. Histological changes following burn injury showed complete healing of all layers of skin including epidermis (EP), papillary dermis (PD), reticular dermis (RD), and hypodermis. Normal epidermis layers: stratum basal (1), stratum spinosum (2), stratum granulosum (3), stratum lucideum (4), and stratum corneum (5), hair follicles sweat glands, sebaceous glands, and arrector pili muscles were obvious in the dermis. H&E, 40X, 100X, and 400X.

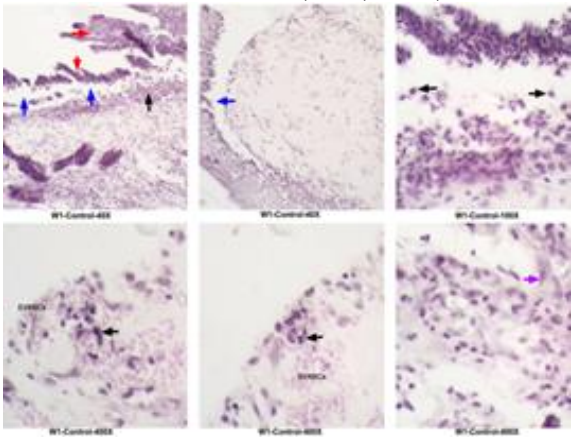

Figure 4: G1-1w. Histological changes following burn injury showed the presence of remnants of the epidermis layer full of infiltrating inflammatory cells (red arrows), the presence of a gap between the epidermis layer and the dermis (blue arrows), the infiltration of a large number of inflammatory cells in the boundary between the two layers below the gap (black arrows) mostly lymphocytes, extravassated RBCs (EVRBCs), edema, accumulation of few fibroblast cells (pink arrow), and the absence of the

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characteristic hair follicles and sebaceous and sweat glands. H&E, 40X, 100X, and 400X.

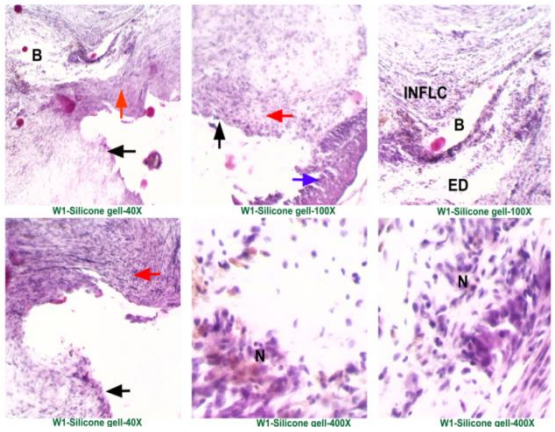

Figure 5: W1-G2. Histological changes following burn injury showed severe destruction and necrosis of the epidermis (black arrows) layer, presence of gap and edema between epidermis and dermis (blue arrow), Loss of the normal microarchitecture of the epidermis and hair follicle, severe infiltration off inflammatory cells (red arrow; INFLC) mostly neutrophils (N) surrounding a bulla like lesion (B) that forming edema (ED) in dermis. H&E, 40X, 100X, and 400X.

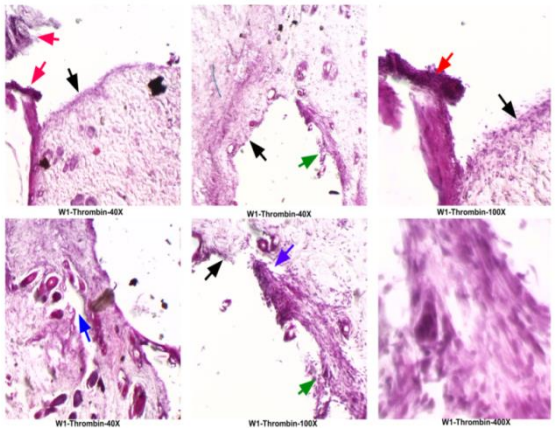

Figure 6: W1-G3. Histological changes following burn injury showed severe destruction and necrosis of the both epidermis (red arrows) layer, presence of gap and edema between dermis and epidermis, severe edema (blue arrows), but initial re-epithelization (green arrows) and hair follicles were seen, as well as infiltration of inflammatory cells beneath epidermis were evident. H&E, 40X, 100X, and 400X.

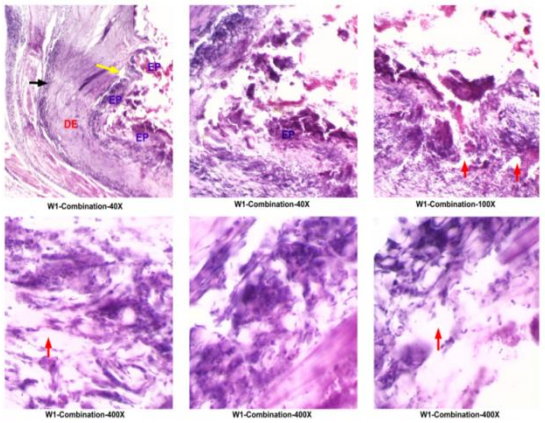

Figure 7: W1-G4. Histological changes following burn injury showed severe destruction and necrosis of the both epidermis (EP) and dermis (DE) layer, presence of gap between dermis and epidermis (yellow arrow), severe edema (red arrows), no hair follicles, sweat, and sebaceous glands were seen, as well as severe infiltration of inflammatory cells in the subcutaneous layer (black arrow). H&E, 40X, 100X, and 400X.

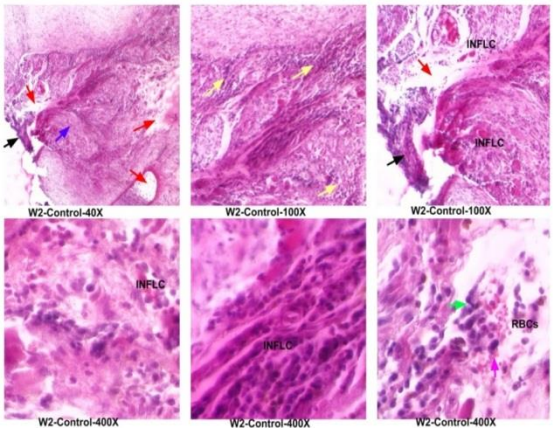

Figure 8: W2-G1. Histological changes following burn injury showed destructed epidermis (black arrows) and thick scab formation (blue arrows), subepidermal and dermal edema (red arrows) and severe inflammatory cells infiltration (INFLC). The inflammatory infiltrated cells are mostly epidermis neutrophils (green arrow) and macrophages (pink arrow) as well as extravassated RBCs were seen. H&E, 40X, 100X, and 400X.

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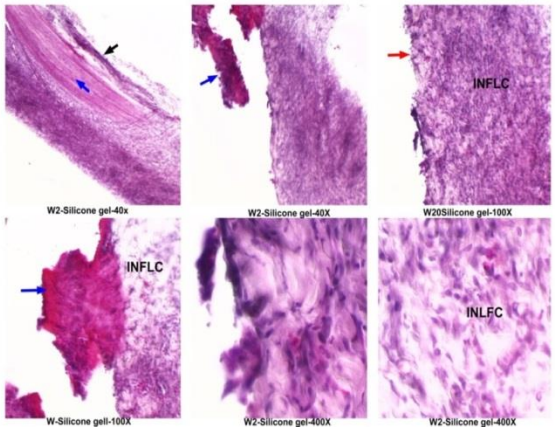

Figure 9: W2-G2. Histological changes following burn injury showed formation of thick scab(blue arrows) covered by inflammatory cells (black arrows), destruction and necrosis of the epidermis (red arrows) layer, Loss of the normal microarchitecture of the dermis, and infiltration of inflammatory cells (INLFC). H&E, 40X, 100X, and 400X.

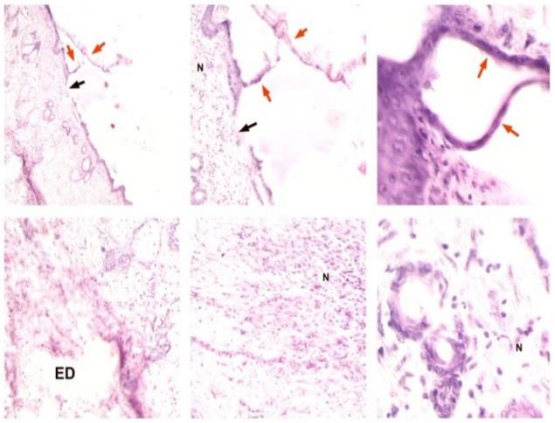

Figure 11: W2-G4. Histological changes following burn injury showed partial re-epithelization (red arrows) of stratum basale layer, destructed epidermis (black arrows), subepidermal edema (ED), and infiltration of the neutrophils (N) were evident. H&E, 40X, 100X, and 400X.

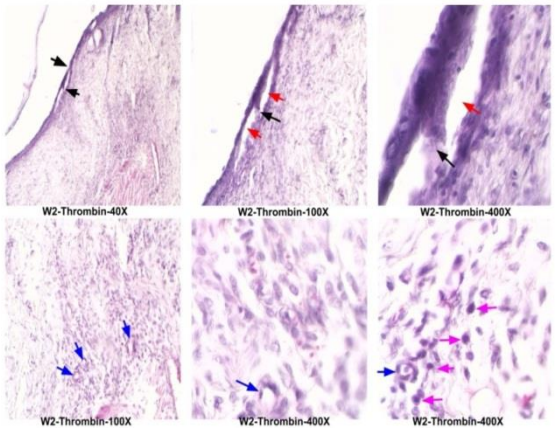

Figure 10: W2-G3. Histological changes following burn injury showed well stage of re-epithelization (black arrows), but gaps (red arrows) between the re-epithelized epidermis and dermis layers was evident, a new formatted blood vessels (blue arrows) were seen surrounded by inflammatory cells (pink arrows). H&E, 40X, 100X, and 400X.

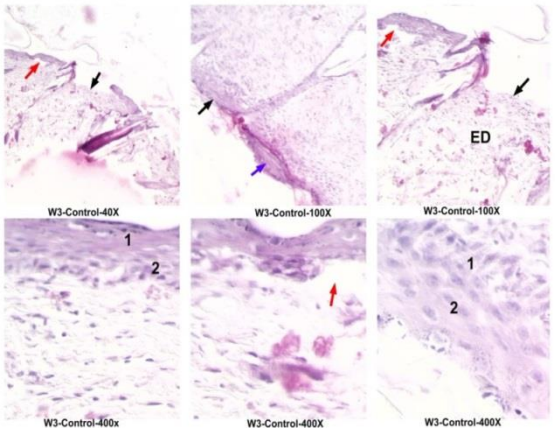

Figure 12: W3-G1. Histological changes following burn injury showed partial re-epithelization (blue arrow) of stratum basale (2) and stratum spinosum (1), subepidermal edema (ED) were evident. H&E, 40X, 100X, and 400X.

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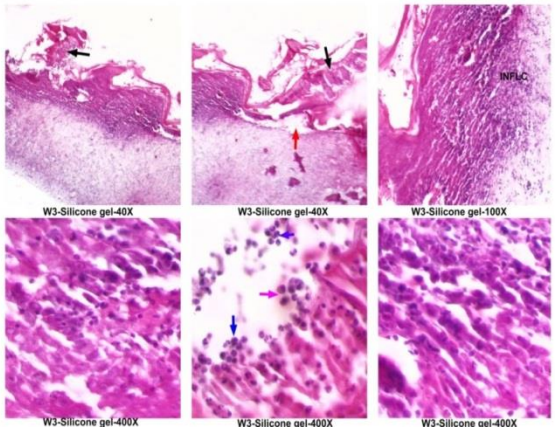

Figure 13: W3-G2. Histological changes following burn injury showed partial re-epithelization, thick scab formation (black arrows), subepidermal (red arrow), severe infiltration of inflammatory cells (INFLC) in the epidermis and dermis, neutrophils (blue arrow) and lymphocytes (pink arrow) were evident. H&E, 40X, 100X, and 400X.

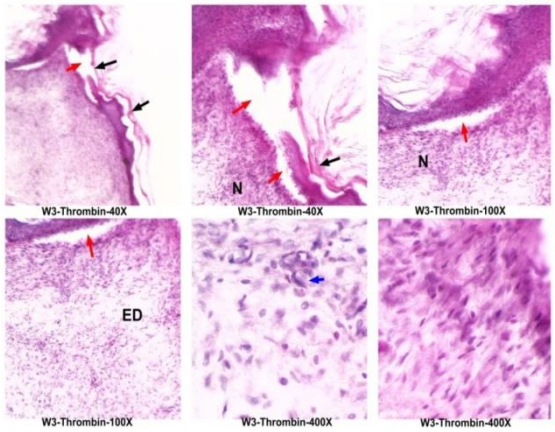

Figure 14: W3-G3. Histological changes following burn injury showed complete re-epithelization (black arrows) of epidermis, where all layers of epidermis were seen, but a subepidermal (red arrows) and dermal edema (ED), infiltration of inflammatory cells (N) and fibroblasts (blue arrow) were evident. H&E, 40X, 100X, and 400X.

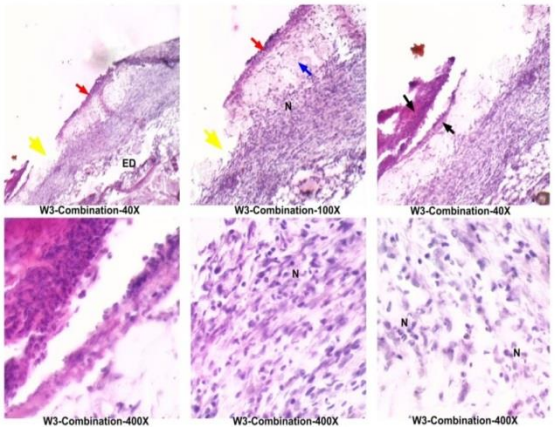

Figure 15: W3-G4. Histological changes following burn injury showed uncovered area of skin (yellow arrow), partial re-epithelization (black arrows) of two layers of epidermis including stratum basale (blue arrow) and stratum spinosum (red arrow) layers, large subepidermal (ED), infiltration of inflammatory cells in the epidermis and dermis, mostly neutrophils (N), were evident. H&E, 40X, 100X, and 400X

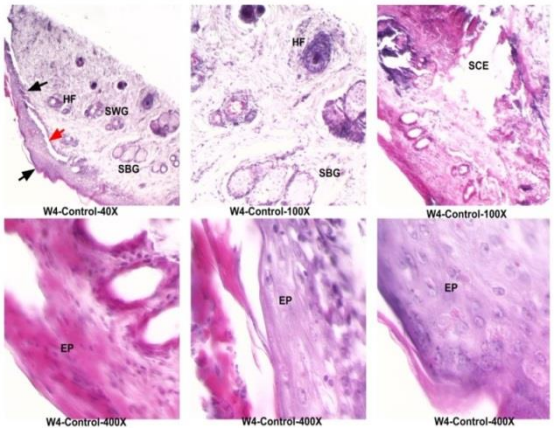

Figure 16: W4-G1. Histological changes following burn injury showed complete re-epithelization (black arrows) of epidermis (EP), but a subepidermal edema (red arrows) was evident, newly formed hair follicles (HF), sweat glands (SWG), and sebaceous glands (SBG) were seen, but subcutaneous edema (SCE) also present. H&E, 40X, 100X, and 400X.

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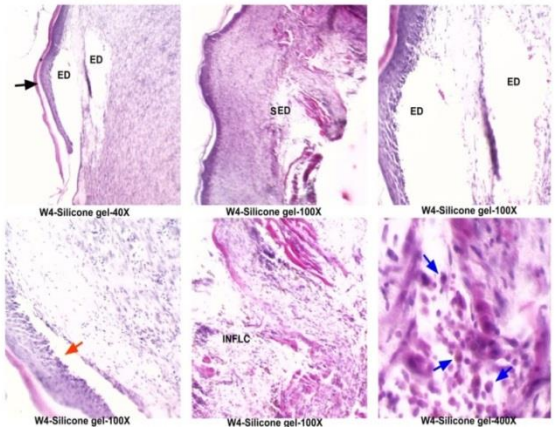

Figure 17: W4-G3. Histological changes following burn injury showed regenerated epidemis layer (black arrows), gaps between epidermis and dermis (red arrows), edema or bulla like lesion (ED) between epidermis and dermis, subcutaneous edema (SED), and infiltration of inflammatory cell in the dermis (INFLC) mostly lymphocytes (blue arrows). H&E, 40X, 100X, and 400X.

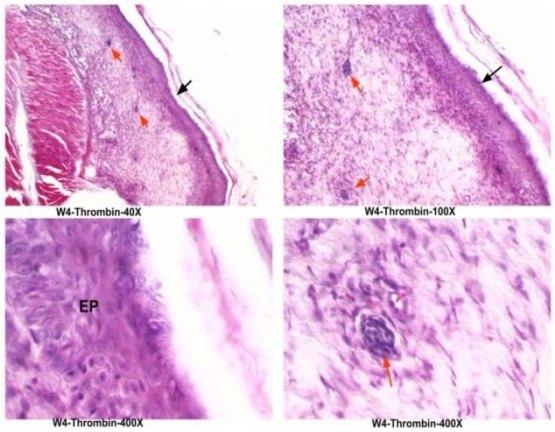

Figure 18: W4-G3. Histological changes following burn injury showed complete regenerated skin, all layers of the epidemis (black arrows, EP)are normal, a new formed hair follicles (red arrows) were seen in the dermis. H&E, 40X, 100X, and 400X.

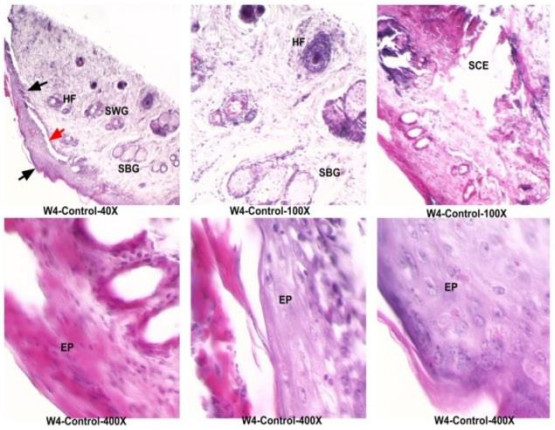

Figure 19: W4-G4. Histological changes following burn injury showed complete re-epithelization (black arrows) of epidermis (EP), but a subepidermal edema (red arrows) was evident, newly formed hair follicles (HF), sweat glands (SWG), and sebaceous glands (SBG) were seen, but subcutaneous edema (SCE) also present. H&E, 40X, 100X, and 400X.

Discussion

The creation of new biopolymeric materials with specific properties to create the ideal wound dressing is required for the development of dressings that enable quick healing with few visible scars on the body's surface. It's important to note.to be easily applied, to maintain local moisture, to ensure an appropriate exchange of gases (O2 and CO2), to absorb exudates that develop on the lesion site (9), to stimulate angiogenesis, to protect against external pathogens, to clear the injured tissue, to eliminate nonviable tissues, to reduce the exposed area (10), to be easily removed and replaced (11), Additionally, materials for wound dressings must be elastic, sterile, non-adherent, and non-allergenic (12), as well as affordable and able to insulate heat (13).One of the most effective non-surgical treatments for burn scars, according to recent studies, is the use of silicone surfaces. This finding was confirmed in the current investigation, as indicated in table-1 in G2 and G4. Numerous mechanisms, including those involving moisture, pressure, temperature, oxygen transfer, and silicon absorption, have been proposed to explain the effects of silicon. In Australia, silicone gel was administered to burn scars by (6). They showed that the silicone-based burn scar therapy had significantly improved. Although there is no physiopathological explanation for this phenomenon, silicone gel alone provides the utmost flexibility and softness for scars (without applying pressure to the scar). Silicone gel is effective at both preventing and treating hypertrophic scars, according to (14). Therefore, it would be conceivable to concur with (6;14) who found that silicone gels minimize scar tissue and hasten the healing of wounds.Coagulation and hemostasis are produced by thrombin through a variety of biological processes that have been extensively studied. In addition to the coagulation cascade, thrombin can interact directly with a variety of cell types when there is bleeding or tissue damage. It causes smooth muscle cell vasoconstriction, which aids in hemostasis. By stimulating platelets, it promotes platelet aggregation at the thrombus location. Activated thrombin also serves as a mitogen and chemo attractant

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for neutrophils and fibroblasts for the aim of tissue and vascular remodeling and repair. The current study's findings concur with those of (15; 16; 17); absorbable hemostatic agents include gelatin sponge, oxidized regenerated cellulose, and microfibrils collagen. It even stimulates the production of vascular endothelial growth factor (VEGF). These substances may be used singly or in conjunction with thrombin. They are all porous structures that serve as a foundation for the activation of platelets and coagulation factors. This suggests that for a clot to form, functional coagulation components must be present (18). It may be possible to observe the results of our experiment on rats using topical thrombin on the burn site, and we obtained a good wound healing without scar tissue, making it difficult to distinguish the recovered tissue from the normal tissue. This is because the intrinsic and extrinsic coagulation pathways are both highly active, which cleaves the prothrombin protein into the active thrombin molecule. Due to maintaining a sufficient temperature, hyperemia as shown in table (4-1) was mild only after the seventh day. Edema was absent in G5 due to promoting blood circulation, and these results are consistent with (19). The crust was mild at day 7 and then disappeared at days 14, 21, and 28 in G5 as a result of maintaining local moisture, which is in accordance with (20). In comparison to silicone gel in G2, thrombin, as shown in table -1 in G3, exhibited a greater efficacy.

Wound contraction: G3 showed a substantial superior contraction. This result, which is consistent with (19), was caused by a speeding up of fibroblast growth, promotion of blood circulation, and stimulation of cell expansion, as shown in table-2.

Bacterial contamination: As shown in table -3 also had the best effect on bacterial contamination in G3 due to act as antibacterial activity by protect against extraneous pathogens, to clear the injured tissue, to eliminate nonviable tissues, and to reduce the exposed area. These findings are consistent with those of (10) and (21). According to this study of (22), silicone gel functions, at least in part, by raising bFGF levels. Based on past research of bFGF's effects, it would be expected that a higher concentration will decrease collagen growth. By using antibody tests of wound fluid retained in an occluded wound, growth factors like platelet-derived growth factor and bFGF accumulated under occlusive dressings. As was already indicated, variations in cytokine expression were discovered to be brought on by both silicone gel and occlusive wound dressing in (23).

Conclusions: The study concluded that silicone gel decrease scar formation and thrombin accelerate burn healing.

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Conflict of interest

No conflict of interest was detected

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