



Reconstructive Plastic Surgery Treatment Methods for Hypertrophic and Keloid Scars

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Annotation. Hypertrophic scars and keloids develop as a result of impaired skin regeneration, primarily after burns and traumatic injuries. They are often accompanied by subjective symptoms such as itching, pain, and skin irritation. Despite advances in burn therapy and post-traumatic rehabilitation, the formation of scar tissue—particularly in cases that lead to functional impairments and deformities—remains an urgent problem in clinical practice, even though the condition does not pose an immediate threat to the patient's life.

Keywords: hypertrophic scars, keloid scars, burns, laser technologies.

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Relevance of the topic. Hypertrophic scars and keloids represent an abnormal wound-healing condition that culminates in scar formation. These pathological scars reflect persistent inflammation and are histologically characterized by fibroblast proliferation, neovascularization, and collagen deposition [1,2]. Clinically, hypertrophic scars differ in the extent to which tissue extends relative to the wound margins. Keloids typically develop over months in areas where scars are subjected to tension, including the chest and joints, and may continue to extend beyond the original wound edges [3,4]. Keloid scars most commonly occur in individuals with darker skin phototypes (Fitzpatrick 3–4) and may develop even after minor skin injury [5]. These lesions tend to persist for a long time, often for several years, and are mainly localized on the earlobes, shoulder girdle, and chest. Keloids are frequently accompanied by subjective symptoms such as pain and itching and tend to exhibit progressive growth beyond the site of the initial trauma [6,7]. Various types of skin injury, including burns, surgical interventions, injections, inflammatory dermatoses, and other damaging factors, may contribute to keloid formation [8]. According to the literature, deep thermal burns are one of the most common causes of keloid scar development. Despite a wide range of treatment options, recurrence rates remain high — up to 50–80% for keloids and approximately 10% for hypertrophic scars [9].

Purpose of the study. To evaluate the effectiveness of reconstructive plastic surgery techniques in the treatment of hypertrophic and keloid scars.

I. Materials And Methods

Initially, non-surgical treatment options were prioritized. However, when such methods fail to provide adequate results, surgical intervention becomes the preferred therapeutic approach. Minimally invasive techniques have demonstrated good outcomes for soft and thin scars, whereas stiffer and thicker scars pose greater challenges. Topical agents have limited penetration into deep tissue layers, and injection-based therapies are often obstructed by dense fibrotic tissue. This requires repeated procedures with limited effectiveness.

This study is based on our previous positive experience with the use of CO₂ laser therapy in treating hypertrophic scars. We examined the combined application of lasers in surgical management. The 10,600-nm CO₂ laser, via a fiber-optic delivery system, produces localized thermal effects that rapidly vaporize fibrotic scar tissue, causing cell lysis, necrosis, and coagulation. This results in tissue ablation and controlled inflammation.

A total of 84 patients with hypertrophic scars were included in the study, with a mean age of 14.4 years. The cohort consisted of 60.8% males and 39.2% females. The earliest time point for initiating treatment was 1 year after scar formation. The mean age at which treatment was initiated was 14.5 years. Scars were less common in children aged 1–7 years due to closer parental supervision. In clinical practice, we frequently observed cases managed conservatively at home (commonly with goose fat). These patients were typically admitted later due to scar-related contractures of the face, chin, or periorbital region. The mean scar area was 207.19 cm² (range: 18.00–1063.50 cm²).

Most patients sought treatment due to scars resulting from burns (55.8%), trauma (13.3%), or surgical procedures (20.8%). Interestingly, in 28.3% of cases, patients presented after a prolonged period of waiting without receiving proper medical care following the initial injury.

Based on the nature of the injury, all patients were classified by trauma type, age, sex, and reference data from contemporary literature. In our study group, burn scars constituted 55.8%, traumatic scars 13.3%, postoperative scars 20.8%, and inflammatory conditions 10%. Purulent-inflammatory diseases (e.g., furuncles, furunculosis) were categorized as inflammatory skin disorders. Among burn injuries, scalding with hot water or oil was most common.

The mean number of procedures performed per patient was 1.38 (range: 1–3). The mean energy per session was 4486.76 J (range: 388–17 536 J), and the mean power per session was 4 W (range: 3–6 W). The mean follow-up period was 7.2 months (range: 6–12 months).

II. Results

Postoperatively, scar thickness was evaluated at each visit. Doppler ultrasonography demonstrated a reduction of 0.308 ± 0.138 cm after 6 months ($p < 0.05$). Overall, scar thickness significantly decreased compared to baseline (from 0.633 ± 0.306 cm to 0.942 ± 0.377 cm). Scar thickness decreased by 27.7% in hypertrophic scars and by 28.2% in keloids.

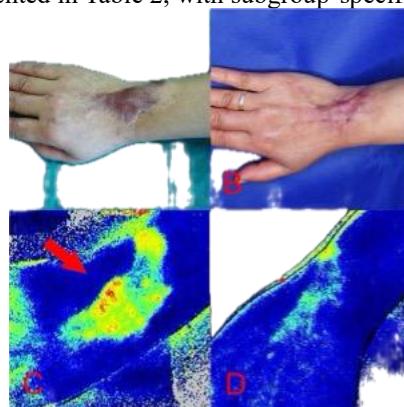
Scar elasticity was also evaluated at each visit (rigidity parameters R0 and Q0 are inversely proportional to numerical elasticity indicators), demonstrating changes of 0.023 ± 0.008 ($p = 0.007$) and 2.616 ± 1.169 units ($p = 0.029$). Elasticity improved as follows: r0 from 1.82 ± 0.056 units to 1.9 ± 0.06 units, and g0 from 365.7 ± 13.113 units to 368.3 ± 12.5 units.

In both subgroups, improvement in the Vancouver Scar Scale was 1.2% for hypertrophic scars ($p < 0.05$) and 0.4% for keloids ($p = 0.26$), the latter not being statistically significant.

Regarding scar pigmentation, no significant improvement was observed in the combined subgroup. The melanin index decreased from 251.413 ± 157.716 units to 234.349 ± 90.708 units after 6 months, a difference of 17.063 ± 131.33 units ($p = 0.308$). However, in the keloid subgroup, pigmentation improved by 21.3% ($p < 0.01$).

Scar vascularization was measured using a PeriCam device and Doppler ultrasonography, demonstrating a reduction of 33.645 ± 15.667 units in vessel perfusion, and a decrease of 17.349 ± 9 units in the erythema index. This indicates that laser treatment significantly reduces scar blood supply (Cam PSI: 118.4 ± 44.593 to 85.3 ± 34.4 , $p < 0.05$; erythema index: 438.8 ± 97.8 to 421.4 ± 97.5 , $p < 0.05$). Additionally, in both subgroups independently, hypertrophic scars showed a 29.6% reduction in perfusion and a 4% reduction in erythema index, whereas keloids exhibited a 22.7% reduction in perfusion and a 3.1% reduction in erythema index (Picture 1).

The overall results are presented in Table 2, with subgroup-specific outcomes shown in Table 3.



Picture 1. A 30-year-old woman with a post-traumatic hypertrophic scar for 2 years before treatment (A); 6 months after surgical and laser treatment (B); a significant reduction in scar vascularity: preoperative value – 105.3 U (C), postoperative – reduced by 28.5% (D); preoperative scar thickness – 0.73 cm (E), postoperative – reduced by 43.84% (F).

Subjective assessment. Pigmentation, height, vascularity, and pliability of the scar during healing were evaluated using the VSS (Vancouver Scar Scale). The results showed that laser-assisted surgery significantly reduces the vascular component of scars, decreases scar thickness, and improves scar pliability. Specifically, the overall VSS score improved by 42% in the hypertrophic scar subgroup and by 37.9% in the keloid subgroup (Table 1).

Table 1.

Changes in scar parameters before and after laser treatment according to the Vancouver Scar Scale.

Characteristics	Before Surgery	After Surgery	Difference Before–After	P
Thickness of the scar (cm)	0,95± 0,4	0,52± 0,4	0,29±0,14	<0,001
Scar elasticity				
R0 (units)	1,7± 0,045	1,85±0,06	- 0,024± 0,008	0,006
Q0 (units)	356,7±13,2	370,3± 12,55	– 2,7± 1,17	0,025
Scar Pigmentation (units)				
Melanin index	261,45±158,62	230,29±89,68	16,23±132,4	0,31
Blood supply in the Scar				
Blood perfusion (units)	116,79±43,66	83,322±32,331	32,675±14,554	<0,001
Erythema index (units)	436,654±93,664	421,441±95,432	16,234±8,12	<0,001
VSS Scores				
Pigmentation	2,543± 0,78	2,423± 0,7	0,154±0,41	0,073
Vascularity	3,800± 0,000	2,200± 0,567	2.000± 0.643	<0,001
Pliability	2,342±0,433	1,432± 0,521	1,345± 0,543	<0,001
Height	3,643± 0,532	2.000± 0.665	1,532±0,722	<0,001
Хусусиятлари	Операциягача	Операциядан кейин	Операциядан олдинги операциядан кейинги	P
Қалинлиги чандык (см)	0,95± 0,4	0,52± 0,4	0,29±0,14	<0,001
Чандыкнинг мувофиқлиги				
R0 (бирлик)	1,7± 0,045	1,85±0,06	- 0,024± 0,008	0,006
Q0 (бирлик)	356,7±13,2	370,3± 12,55	– 2,7± 1,17	0,025
Чандыктар пигментацияси (бирликларда)				

меланин индекси	261,45±158,62	230,29±89,68	16,23±132,4	0,31
Чандықдаги қон таъминоти				
Кон перфузияси (бирликда)	116,79±43,66	83,322±32,331	32,675±14,554	< 0,001
Эритема индекси (бирликда)	436,654±93,664	421,441±95,432	16,234±8,12	< 0,001
VSS баллари				
Пигментация	2,543± 0,78	2,423± 0,7	0,154±0,41	0,073
Баландлиги	3,800± 0,000	2,200± 0,567	2.000± 0.643	< 0,001
Кемалар	2,342±0,433	1,432± 0,521	1,345± 0,543	< 0,001
Юмшоқлиги	3,643± 0,532	2.000± 0.665	1,532±0,722	< 0,001

Postoperative pain incidence was 85.71%, with a duration of 2.23 ± 0.8 days and a pain score of 1.2 ± 1.33 units. Local edema occurred in 100% of patients, lasting 7.00 ± 1.2 days. Tissue necrosis was observed in 29.6% of cases, and the healing time averaged 25.23 ± 12.3 days. During the follow-up period of 6 to 12 months, none of the patients demonstrated recurrence of hypertrophic scars after laser therapy. Likewise, no cases of persistent hyperpigmentation, hypopigmentation, or skin infection lasting longer than 3 months were recorded. The summary of the findings is presented in Table 4. Patient satisfaction was high, with more than 85% of participants reporting that they were very satisfied or satisfied with the treatment outcomes.

Table 2
Subgroups of hypertrophic scars and keloids: percentage changes before and after laser treatment

	Thickness	Thickness		Elasticity		Elasticity		
	Hypertrophic	Keloid		Hypertrophic		Keloid		
	Initial	Rapid	Initial	Rapid	Initial	Rapid	Initial	Rapid
Mean ± SD	0,9±0,3	0,56± 0,10	1,2±0,5 5	0,82±0,55	354,7±13 ,5	383± 15,4	364,43±9,2	364,88± 6,0
Min; Max	0,43;0,23	0,40;1,20	0,42; 1,00	0,18;1,75	333;425	350;430	340;378	355; 378
Difference	-0,2	- 0,28		4.2		1.3		
% improvement	- 26 .8	- 27 . 2		1.3		0.4		

T-test paired (p)	< 0 . 001	< 0 . 001		< 0 . 05		0,26		
	Blood perfusion	Blood perfusion		Blood perfusion		Blood perfusion		
	ГР	Keloid		ГР		Keloid		
	Initial	Rapid	Initial	Rapid	Initial	Rapid	d Pre	
Mean ± SD	90,2±25,6	71,3±24	132,3±5 1,1	102,3±42, 1	422,7±10 6,1	402,3±10 5,1	444,4±85	433,5±9 2,1
Min; Max	30;143	21;104	31;225	21;178	241;630	221;618	166;570	142;649
Difference	- 28,6	- 28,8		- 16,0		- 12,8		
% improvement	- 29 .3	- 22 .5		- 4 .2		- 3 .2		
T-test paired (p)	< 0 . 001	< 0 . 001		< 0 . 001		< 0 . 05		
	Pigmentation	Pigmentation		Pigmentation		Pigmentation		
	ГР	Keloid		ГР		Keloid		
	Initial	Rapid	Initial	Rapid	Initial	Rapid	Initial	
Mean ± SD	202,3±105, 1	244,6±10 2,2	278,6±1 60	225,2±83, 4	12,5±0,8	7,3± 1,5	12,0±0,6	8,2±2,3

Min; Max	81;481	81;469	81;623	81;322	11;12	5;9	11;13	6;12
Differen ce	42.1		- 61.4		- 5.2		- 4.9	
% improvem ent	21.1		- 22 .1		- 41 . 0		- 36 .7	
T-test paired (p)	0,03		< 0 . 01		< 0 . 001		< 0 . 001	

Table 3
Post-treatment adverse events. SD — standard deviation, VAS — visual analogue scale

Unprocessed events	% / Mean ± SD	Duration
Pain	86,21%	2,2± 0,73
Pain assessment by VAS scale	4,2±1,3	-
Swelling	100%	7,00± 1,2
Partial scar necrosis	28,30%	14,83±3,05
Hyperpigmentation > 3 months	0	0
Hypopigmentation	0	0
Skin infection	0	0
Exacerbation of keloid or hypertrophic scar	0	0



Picture 2. A 46-year-old man with a keloid scar on the chest and arm caused by a burn sustained 2 years earlier (A); after 6 months of follow-up and 3 laser sessions, the patient achieved significant cosmetic and functional improvements (B).

The evolution of laser and surgical treatments has demonstrated a wide range of clinical and cosmetic applications. Based on these innovative devices, a new method of combined surgical treatment for hypertrophic scars has been developed. The surgical treatment technique for hypertrophic scars consists of laser ablation of the scar zone, preserving the boundary between healthy skin and scar tissue in this area, followed by preliminary excision of the superficial layer of scar tissue with an epithelial cover up to 1 mm thick. The procedure is characterized by eliminating the scar with a reduction of the wound surface by 2–3 mm relative to the surrounding healthy skin. A CO₂ laser (Fotona CO₂ Fraxel laser) with a wavelength of 10.05 microns, continuous focused mode, and 20 W power is used. Afterward, the wound surface is treated with yellow laser radiation (Denave

Optiscan – 585 nm, 5 W power, 30 J/cm²) in scanning mode. The pre-excised scar segment is reapplied to the wound surface (with preliminary dermal layer processing and thinning) and fixed along its periphery using biological adhesive. Upon full or partial engraftment of the transplanted tissue segment, photodynamic therapy is performed with a 20% ointment form of 5-ALA photosensitizer, as well as red-light irradiation (PDT laser Deka) within the wavelength range of 600–660 nm, with an exposure time of 5 minutes per field at 1 W power per 1 cm². After epithelialization of the wound surface, laser irradiation is performed (Deka Fraxel CO₂) at 1 W power, 1.06 μm wavelength, frequency of 50 Hz, for 1 minute per 1 cm² of the field, directing the radiation field along the wound perimeter toward the central zone of the scar. Treatment is carried out every 14 days, with a gradual reduction of radiation power to 500 mW and an exposure duration of 30 seconds. Subsequently, for 1 month, elastic adhesive bandages are applied, providing tissue tension up to 100 kPa.

Our study results demonstrated that the use of a laser with a wavelength of 10,600 nm contributes to the reduction of inflammatory reactions and decreases tissue perfusion, which collectively prevents the development of pathological scar changes. Application of this method led to a significant reduction in scar thickness (by almost 30%) and a decrease in the severity of inflammation within keloid scar areas ($p < 0.05$).

In scar tissues, collagen fibers undergo degenerative changes and contraction; the thermal energy diffusing into surrounding tissues causes protein denaturation and structural coagulation. These processes enhance the increasing synthesis of components within the collagen and extracellular matrix, which leads to an increase in scar density and the number of type I and type III collagen fibers. The resulting fibrous structures raise tissue rigidity and reduce its elasticity.

In stabilizing the collagen molecule, a special role is played by its triple-helix structure reinforced by hydrogen bonds via 4-hydroxyproline. Under thermal exposure, this structure loses its organization and transitions into an amorphous state of random tangling. This leads to contraction of approximately one-third of the collagen fibers, as well as changes in their strength and elastic properties.

According to the data obtained in this study, the elasticity index of scar tissues significantly decreased after laser therapy, indicating softening of the tissue and improvement of its elastic properties ($p < 0.05$). In the overall group, a statistically significant improvement of 1.2% was recorded ($p < 0.05$), whereas the 0.4% improvement observed in the subgroup of patients with keloid scars was not statistically significant ($p = 0.26$), which reflects only partial modification of scar tissue. Thermal exposure disrupts intermolecular cross-linking and destabilizes the triple-helix structure of collagen, resulting in its contraction and subsequent degradation.

According to the literature, the most common adverse reactions after laser scar treatment are postoperative hyperpigmentation, depigmentation, and persistent erythema. However, our findings demonstrated that the use of a low-intensity laser with a wavelength of 980 nm does not enhance melanogenesis in the area of keloid scars and therefore does not lead to the development of hyperpigmentation. Furthermore, pigmentation intensity of scar tissue decreased by 21.3%, which may indicate the ability of the laser to eliminate melanin accumulation and help reduce the severity of pigmentation.

Different researchers reported an increase in fibroblast proliferation within scar tissues, intensification of collagen synthesis, and activation of cellular metabolism, which promote neovascularization and the formation of new capillaries that provide transport of nutrients. Ogawa et al. (11, 41) revealed that the level of the angiogenic cytokine VEGF and the density of vascular structures in scars were significantly higher than in normal tissues, demonstrating the crucial role of vascular supply in maintaining metabolic processes within scar tissue.

When evaluating changes in blood perfusion in scar tissues using the erythema index, a significant decrease in scar perfusion was detected ($p < 0.001$). Colorimetric assessment of erythema revealed a statistically significant difference compared to the preoperative state. Specifically, vascular blood flow decreased by 29.3% in the hypertrophic scar (HS) group and by 22.5% in the keloid scar group; the erythema index improved by 4.2% in HS and by 3.2% in the keloid subgroup. These results differ from the data reported in a controlled study by Wittenberg et al. (42), where redness of the scar disappeared within one year after treatment with a pulsed dye laser.

A possible explanation for this discrepancy is the use of an innovative surgical treatment technique in combination with laser therapy, which helps shorten the inflammatory phase of scar tissue and, as a result, accelerates its maturation stage.

III. Conclusion

This scientific study achieved satisfactory results confirming the effectiveness of the applied method in reducing the volume of scar tissue, improving its elasticity, and decreasing the severity of erythema, pigmentation, and vascular prominence within the scar. It should be emphasized that the penetration depth of laser irradiation is limited to 2–3 mm, which indicates the necessity of a combined therapeutic approach—both surgical intervention and laser therapy—for scars protruding more than 2 mm above the skin surface. In this regard, it is advisable to apply this laser technique in combination with other types of laser treatment or pharmacological agents.

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