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Management of keloid and hypertrophic scars with a topical cream containing vegetal superoxide dismutase (SOD)

Objective. To evaluate the therapeutic activity and tolerance of topical superoxide dismutase (SOD) cream on keloid and hypertrophic scars, taking especially in consideration the evolution of size and volume, and parameters such as color, pruritus, erythema, hardness and pain.

Methods. 27 patients were enrolled at baseline and treated with topical application of SOD cream (SODERMIX® cream) twice a day during 3 months.

Results. At the end of the treatment, there was a significant reduction in the size (37.07 %; $p < 0.05$) and volume (48.44 %; $p < 0.05$) of scars.

Color and pruritus were the best markers of the efficacy of the treatment and were significantly improved from the 1st month onward, such as erythema at a lesser extent.

Tolerance was perfect all along the treatment.

Conclusions. Topical SOD cream provides beneficial effects in the treatment of keloid and hypertrophic scars and could be a valid and safe alternative for treating this pathology.

Key words

Topical superoxide dismutase, SOD, hypertrophic scars, keloid scars.

Keloid and hypertrophic scars (HS) are of major concern for dermatologists and plastic surgeons: patients are often consulting about these pathologies because besides the cosmetic concern which is obvious, they may feel uncomfortable due to itching or pain associated with their occurrence, and even suffer loss of function when overlying a joint.

Hypertrophic scars and keloids are abnormal wound responses in predisposed individuals and feature a connective tissue response to trauma, inflammation, surgery or burns [1].

Keloid and HS are two discrete clinical entities [2].

Clinically, keloids behave like benign dermal fibro-proliferative tumors, extending and infiltrating beyond the confines of the original wound margin without evidence of spontaneous regression [3] whilst HS are typically raised, red or pink [4]. Both of them are usually itchy and may sometimes be painful.

Normal wound healing occurs in three phases [5]:

- 1-The inflammatory phase begins at the time of wounding, when activation of the coagulation cascade causes a release of cytokines stimulating chemotaxis of unspecific immune cells. After 48 to 72 hours it passes to
- 2-the proliferative phase (3—6 weeks): the fibroblasts are attracted into the wound to synthesize granulation tissue composed of pro-collagen, elastin, proteoglycans and hyaluronic acid, allowing vascular growth. Once the wound is closed, immature scar can move to
- 3-the maturation phase, lasting several months.

All stages of the repair process are controlled by a wide variety of growth factors (TGF-β, PDGF, VEGF), pro-inflammatory cytokines and matrix metalloproteinases (MMPs) [6].

Abnormal changes in this complex wound healing process contribute to HS and keloid formation [5].

Among the cytokines and growth factors increased in the occurrence of keloids and HS, let us

mention Transforming Growth Factor beta-1 (TGF-β_1) [7—9], Platelet Derived Growth Factor (PDGF) [10, 11], Vascular Endothelial Growth Factor (VEGF) [11, 12], Insulin-like Growth Factor (IGF) [13, 14], Interleukin-1 (IL-1) [15, 16] and Interleukin-6 (IL-6) [15—17].

MMPs levels are also increased as well in keloids as in HS tissues [18].

On the other hand, a bulk of scientific data suggest that ROS are playing a major role in the formation and maintenance of keloids and HS, by increasing dramatically the levels of a great number of cytokines and growth factors involved in scarring process, especially PDGF [19, 20], VEGF [21—22], IGF [23], but also MMP-2 and MMP-9 [24].

As it seems logical, antioxidant enzymes and especially superoxide dismutase (SOD) were shown to reduce drastically the levels of all cytokines and growth factors susceptible of influencing keloid and HS formation [21, 23, 25—32].

Whilst the most important factor in keloid and HS formation is prevention [1], treatments are often deceptive and unsatisfactory, based principally on the use of pressure, occlusive dressings, silicone sheets and intralesional corticotherapy. Radiation and laser therapy are less commonly used.

Therefore, it was tempting to test topical SOD in the management of keloid and HS.

This is an open, monocentrical, intra-individual study performed in adult patients displaying keloid and HS with more than 3 months of evolution.

The treatment with topical SOD (280 UI/g), which had already demonstrated its efficacy in the treatment of post-irradiation fibrosis [33] consisted in the local application on one or more previously selected scars, twice a day (morning and evening).

SOD is sourced from tomato, in order to avoid any potential risk related to the use of animal extracts. Tomato SOD is mainly constituted by the CuZn form of the enzyme, and its molecular weight is around 31'500 daltons.

Methods

The study was performed in the Dermatological Clinic SKINMED, at Córdoba, Argentina between August 2005 and March 2006.

Patients of both sexes, older than 18, with keloid or HS with more than 3 months of evolution before their inclusion were recruited, after signature of informed consent.

We excluded pregnant and suckling women, patients with keloid or HS of less than 3 months of evolution before the date of inclusion, patients having received systemic or local treatments for their scars in the course of 4 weeks previous to their inclusion, or treated with any systemic, topical or cosmetic treatment susceptible of interfering with the parameters of the study, and patients with an allergy to any of the components in the formula.

Evaluation

Clinical evaluation was undertaken at monthly intervals for three months.

Scars were assessed by a dermatologist, grading the patients' scars at baseline and then evaluating the changes in relation to a series of criteria, using a three point scale (1 = none, 2 = moderate, 3 = marked).

The parameters were the following:

- color (assessed by investigator);
- pruritus (assessed by patient);
- erythema (assessed by investigator);
- pain (assessed by patient);
- hardness (assessed by investigator).

A global assessment of the clinical course of scar development was evaluated using a Therapeutic Index (TI) calculated as follows after each visit:

TI = index of color + index of pruritus + index of erythema + index of pain + index of hardness.

TI values improvements were classified in four groups to define the degree of improvement:

- complete healing: reduction > 85 % of TI;
- clear improvement: reduction > 50 % and < 85 % of TI;
- moderate improvement: reduction > 20 % and < 50 % of TI;
- poor improvement: reduction < 20 % of TI.

On the other hand, digital photographs were taken at baseline and at each visit, and surface and volume analysis of the scars were performed with SigmaScan Pro5 Software (available from SYSTAT software Inc.). With this software five non-destructive overlay planes allow to collect measurements such as intensity, hue, saturation, distance, perimeter, slope, angle, area, volume, and center of mass simultaneously.

Tolerance was also evaluated at each visit, by collecting the unwanted effects according to a four degree scale (0 = None, 1 = Mild, 2 = Moderate, 3 = Severe).

Treatment Procedures

Patients were taught to apply the topical SOD cream (SODERMIX® cream, Life Science Investments Ltd, London, UK) twice a day (morning and evening) with a slight massage following the application.

Statistics

The model used was the generalized linear model log-linear Poisson, with logarithmic binding function, $g(\mu) = \log(\mu)$ and linear predictor $\eta = \mu_0 + \alpha_i$, with α effect of view, $i = 1, 2$.

From these models were obtained the estimations of odds ratios (OR) in order to interpret the correlation with time. When a parameter is featuring a significant change from one visit to another, it is labeled as «significant marker» of efficacy in the treatment.

These models were adjusted and estimated in Statistica Software (StatSoft Inc. 2005).

Results

Of the 27 patients who entered the study (20 females, 7 males, mean age 42,7 years), 25 (92.6 %) attended the first visit at D30, 16 patients (59.2 %) came back at D60 and only 8 of them (29.6 %) at D90.

This poor compliance, typical of our country, was also explained by the rapidity of occurrence of improvement, and further, the study took place during the summer holidays season, which made the compliance more difficult.

None of the patients stopped the treatment because of intolerance to the product.

Scar duration was ranging between 3 months and 27 years, with a mean value of 8.6 years.

Scars were classified as keloid in 14 patients (52 %) and HS in 13 patients (48 %).

Phototypes of the patients were ranging from I to IV according to Fitzpatrick: 7 patients (26 %) were phototype I, 14 (52 %) were phototype II, 5 (18 %) were phototype III and 1 patient (4 %) was phototype IV.

Color parameter

At baseline, 50 % of patients were displaying marked pigmentation.

At D30, there was no more patient with marked pigmentation, but only moderate or normal ones.

There is a strong relationship ($p < 0.001$) between color and time of treatment, as the proportion 1 : 2 observed between normal and moderate pigmentation at D30 significantly reverts, as in practical, there is only normal pigmentation in all patients after 2 months.

Color is a good marker of the treatment, and permits to evaluate the efficacy of the same. It was the most sensitive marker in this trial (fig. 1).

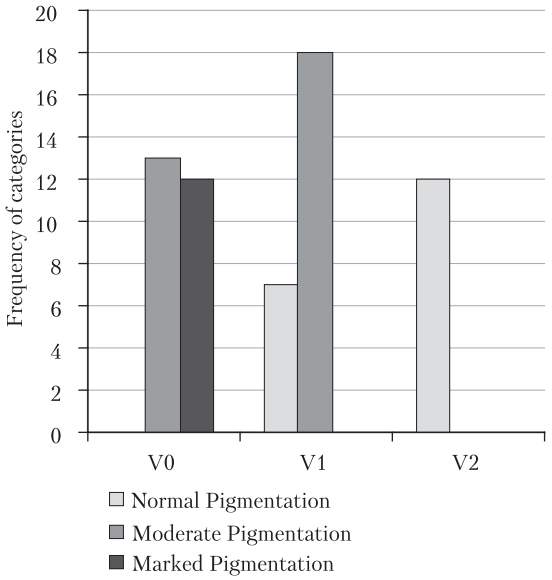

Fig. 1. **Evaluation of color along time**

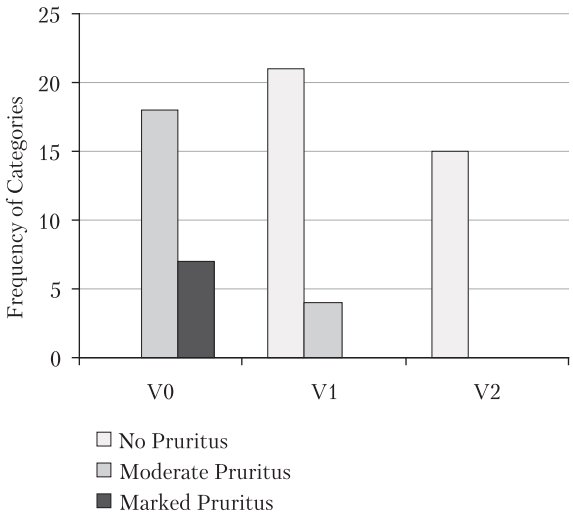

Fig. 2. **Evolution of pruritus along time**

Pruritus (itch) parameter

When starting the treatment, 7 patients of 27 (25.9 %) were suffering severe pruritus, and none of them remained with severe pruritus at D30.

In total, 25 of 27 patients (92.6 %) were suffering any grade of pruritus at D0, whilst at D30 there were only 4 patients (16 %) with moderate pruritus, and at D60 pruritus had disappeared in all patients.

There was a strong relationship ($p < 0.01$) between the presence of pruritus and the duration of treatment (OR = 0.33; IC 95 %: [0.135—0.48]; $p < 0.01$).

Pruritus was also found to be a good marker for evaluating the efficacy of treatment (fig. 2).

Erythema parameter

At the beginning of the treatment only 1 patient (4 %) was displaying marked erythema, but 9 (33 %) were showing moderate erythema.

At D30 there was no more patient with marked erythema, and only 5 (20 %) with moderate erythema.

The association between the reduction of erythema and time was significant. (OR = 0.67; $p < 0.05$).

Table 1. **Index values for each parameter along study**

	Color	Pruritus	Erythema	Hardness	Pain
Baseline D0	2.48 ± 0.51	2.33 ± 0.48	1.44 ± 0.58	1.44 ± 0.58	1.37 ± 0.56
D30	1.72 ± 0.46	1.16 ± 0.37	1.24 ± 0.44	1.20 ± 0.41	1.16 ± 0.37
D60	1.25 ± 0.45	1.06 ± 0.25	1.12 ± 0.34	1.00	1.12 ± 0.34
D90	1.00	1.00	1.00	1.00	1.00

Table 2. **Evolution of Therapeutic Index values along the study**

	Therapeutic Index (TI)
Baseline D0	8.89 ± 1.83
D30	6.40 ± 1.44
D60	5.56 ± 1.03
D90	5.00

Table 3. **Healing rate of patients along time according to therapeutic index classification**

Healing Grade	D30 N° (%)	D60 N° (%)	D90 N° (%)
Complete healing	0	0	0
Clear improvement	2 (8)	12 (75)	5 (63)
Moderate improvement	17 (68)	4 (25)	3 (37)
Poor improvement	6 (24)	0	0
Total	25 (100)	16 (100)	8 (100)

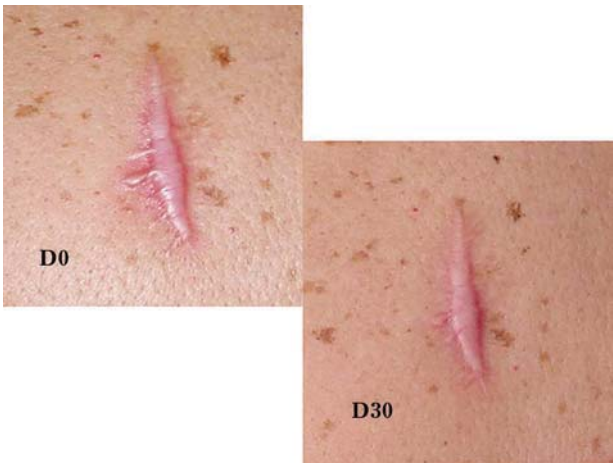

Fig. 3. **Female, age 27, phototype II, keloid with 11 years of evolution following excision of nevus on the breast. Unsatisfactory previous treatment with intralesional CTC. Day0 and D30 (30 days treatment)**

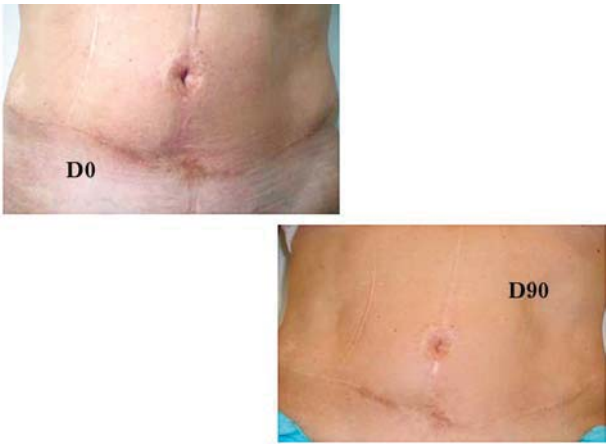

Fig. 4. **Female, age 50, phototype II, keloid with 10 years of evolution following abdominal trauma. No previous treatment. Day0 and Day90 (after 90 days-treatment)**

Erythema was also a good marker of the efficacy of treatment, although at a lesser extent than color or pruritus.

Pain and Hardness parameters

As regards hardness, there was a discrete tendency to improvement, although there was no significant association ($p = 0.362$) between its intensity and time.

This tendency was more pronounced as regards pain.

Therapeutic Index (TI)

Index values were calculated separately for each parameter. All data presented are mean ± standard deviation (table 2).

Therapeutic Index values were calculated separately for each patient. All data presented are mean ± standard deviation (table 3).

Planimetric analysis

The photographs realized along the study were analyzed by SigmaScan Pro5 Software (fig. 3, 4).

The percentages of improvement as regards surface and volume of the scars were analyzed as regards their evolution along time.

Independent of the number of visits for each patient, the mean values for the percentages of improvement (along with their standard deviations) are featured in Table 4, and there is a marked positive correlation ($p < 0.05$) between these variables (0.793).

Following this therapy, the improvement observed is highly significant (Table 5 and Table 6).

It can be observed that evaluating this improvement by measuring the volume is much more precise than by evaluating the surface.

Discussion

Pharmacologic therapeutic agents are commonly used in the treatment of keloid and HS, with irregular results.

The anti-inflammatory and anti-fibrotic properties of SOD were already investigated and demonstrated in vitro and in vivo, and topical SOD was found to be effective on lesions caused by progressive systemic fibrosis, systemic lupus erythematosus, Behçet's disease, herpes simplex and burns [34, 35].

On the other hand, several studies conducted on human skin radiofibrosis (post-radiotherapy) reported that topical SOD appeared to reduce collagen accumulation in the dermis of irradiated skin [36] and reduce post-irradiation fibrosis in breast cancer patients [33].

Meanwhile, in spite of the similarities existing in the physiopathology of skin fibrosis and keloid and HS, no study was published in the literature on topical SOD in scar management.

In our study, the improvement of size and volume was shown to be strongly significant and there is a marked correlation between these results and time.

There was also a significant improvement on the color of the scars, itching (pruritus) and inflammation, while the activity of topical SOD was not significant on the pain and hardness.

Regarding the latter, we must regret the too-short duration of the study, as hardness is a result at least in part of dermal fibrosis, and takes a longer time than 3 months to improve.

We must also regret the poor compliance of the patients; a better compliance would maybe have been susceptible of achieving better and more significant results.

Anyhow, the results appeared to be positive, all the more given the short duration of study.

Besides, tolerance was excellent and we have not observed any adverse effect in the course of this trial.

Table 4. **Means and standard deviations of improvements of surface and volume of the scars along the study**

Variable	Means and standard deviations		
	Means	Standard Dev.	N
Improv. Surface	37.07	23.24	27
Improv. Volume	48.44	29.71	27

Conclusions

Topical SOD provides beneficial effects in the treatment of keloid and HS, and could be a valid and safe alternative for treating this pathology.

We were particularly surprised by the significant effect of topical SOD on pruritus, which was never documented earlier.

Success would certainly be increased when this treatment is followed on a longer period of time and possibly when combined with other modalities.

Hence it would be worth conducting further clinical trials on longer periods, with higher number of patients and achieving better compliance of the same.

Table 5. **Test of means against reference constant for surface improvement**

Variable	Test of means against reference constant (value equal 0)							
	Mean	St Dev	N	Std. Err.	Ref. Cons.	t-value	df	p
Imp.Surf	34.07407	23.24605	27	4.473705	0,00	7.616523	26	0.00

Table 6. **Test of means against reference constant for volume improvement**

Variable	Test of means against reference constant (value equal 0)							
	Mean	Std.Dev.	N	Std. Err.	Ref. Cons.	t-value	df	p
Improv.Vol	48.44444	29.70539	27	5.716805	0.00	8.474042	26	0.00000

Bibliography

1. English R.S., Shenefelt P.D. Keloid and hypertrophic scras // Dermatol Surg.— 1999.— Vol. 25.— P. 631—638.
2. Burd A., Huang L. Hypertrophic response and keloid diathesis: two very different forms of scar // Plastic. Reconstr. Surg.— 2005.— Vol. 116.— P. 150—157.
3. Ehrlich H.P., Desmouliere A., Diegelmann R.F. et al. Morphological and Immunological differences between keloid and hypertrophic scars // Am. J. Pathol.— 1994.— Vol. 145 (1).— P. 105—113.
4. Slemp A.E., Kirschner RE. Keloids and scars: a review of keloids and scars., their pathogenesis, risk factors and management // Curr. Opin. Pediatr.— 2006.— Vol. 18.— P. 396 —402.
5. Wolfram D., Tzankov A., Pülzi P., Piza-Katzer H. Hypertrophic scars and keloids – A review of their pathophysiology, risk factors and therapeutic management // Dermatol. Surg.— 2009.— Vol. 35 (2).— P. 171—181.
6. Werner S., Grose R. Regulation of wound healing by growth factors and cytokines // Physiol. Rev.— 2003.— Vol. 83.— P. 835—870.
7. Lee T.Y., Chin G.S., Kim W.J. et al. Expression of transforming growth factor beta-1, -2 and -3 proteins in keloids // Ann. Plast. Surg.— 1999.— Vol. 43 (2).— P. 179—185.
8. Hanasono M.M., Kita M., Mikulec A.A. et al. Autocrine growth factor production by fetal, keloid and normal dermal fibroblasts // Arch. Facial. Plast. Surg.— 2003.— Vol. 5 (1).— P. 26—30.
9. Tang S., Pang S., Cao Y. Changes in TGF-beta1 and type I, III procollagen gene expression in keloid and hypertrophic scar // Zhonghua Zheng Xing Shao Shang Wai Ke Za Zhi.— 1999.— Vol. 15 (4).— P. 283—285.
10. Niessen F.B., Andriessen M.P., Schalkwijk J. et al. Keratinocyte derived growth factors play a role in the formation of hypertrophic scars, J Pathol 2001.— Vol. 194 (2).— P. 207—216.
11. Jiang D.Y., Fu X.B., Chen W., Sun T.Z. Relationship of overexpression of angiogenesis factors and their receptors with invasive growth of keloids // Zhonghua Zheng Xing Wai Ke Za Zhi.— 2004.— Vol. 20 (2).— P. 128—131.
12. Gira A.K., Brown L.F., Washington C.V. et al. Demonstrate high-level epidermal expression of vascular endothelial growth factor // J. Am. Acad. Dermatol.— 2004.— Vol. 50 (6).— P. 850—853.
13. Ghahary A., Shen Y.J., Nedelec B. et al. Enhanced expression of mRNA for insulin-like growth factor-1 in post-burn hypertrophic scar tissue and its fibrogenic role by dermal fibroblasts // Mol. Cell. Biochem.— 1995.— Vol. 148 (1).— P. 25—32.
14. Yoshimoto H., Ishihara H., Ohtsum A. et al. Overexpression of insulin-like growth factor-1 receptor and the invasiveness of cultured keloid fibroblasts // Am. J. Pathol.— 1999.— Vol. 154.— P. 883—889.
15. Polo M., Ko F., Busillo F. et al. The 1997 Moyer Award. Cytokine production in patients with hypertrophic burn scars // J. Burn. Care Rehabil.— 1997.— Vol. 18 (6).— P. 477—482.
16. Duong H.S., Le A., Messadi D.V. Role of Proinflammatory cytokines in keloid pathogenesis // Free Comm. Epith. Cell. Biol.— 2004, Hawaï.
17. Xue H., Cauley R.L., Zhang W. Elevated interleukin-6 expression in keloid fibroblasts, // J. Surg. Res.— 2000.— Vol. 89 (1).— P. 74—77.
18. Neely A.N., Clendening C.E., Gardner J. et al. Gelatinase activity in keloids and hypertrophic scars, // Would Repair. Regen.— 1999.— Vol. 7 (3).— P. 166—171.
19. Curcio F., Pegoraro I., Dello Russo P. et al. SOD and GSH inhibit the high glucose-induced oxidative damage and the PDGF increased secretion in cultured human endothelial cells // Thromb. Haemost.— 1995.— Vol. 74 (3).— P. 969—976.
20. Min D.S., Kim E.G., Exton J.H. Involvement of tyrosine phosphorylation and protein kinase C in the activation of phospholipase D by H^2O^2 in swiss 3T3 fibroblasts // J. Biol. Chem.— 1998.— Vol. 273 (45).— P. 29986—19994.
21. Grzenkowicz-Wydra J., Cisowski J., Naconieczna J. et al. Gene transfer of CuZn superoxide dismutase enhances the synthesis of vascular endothelial growth factor // Mol. Cell. Biochem.— 2004.— Vol. 1—2.— P. 169—181.
22. Cho M., Hunt T.K., Hussain M.Z. Hydrogen peroxide stimulates macrophage vascular endothelial growth factor release // Am. J. Physiol. Heart Circ. Physiol.— 2001.— Vol. 280.— P. H2357—2363.
23. Delafontaine P., Ku L. Reactive oxygen species stimulate insulin-like growth factor I synthesis in vascular smooth muscle cells // Cardiovasc. Res.— 1997.— Vol. 33 (1).— P. 216—222.
24. Rajagopalan S., Meng X.P., Ramasamy S. et al. Reactive oxygen species produced by macrophage-derived foam cells regulate the activity of vascular matrix metalloproteinases in vitro // J. Clin. Invest.— 1996.— Vol. 98 (11).— P. 2572—2579.
25. Lefaix J.L. et al. Successful treatment of radio-induced fibrosis using Cu/Zn superoxide dismutase and MnSOD: an experimental study // Int. J. Radiot. Oncol. Phys.— 1996.— Vol. 35 (2).— P. 305—312.
26. Delanian S. et al. Cu/Zn superoxide dismutase modulates phenotypic changes in cultured fibroblasts from human skin with chronic radiotherapy damage // Radiother. Oncol.— 2001.— Vol. 58 (3).— P. 325—331.
27. Tamagawa K., Taooka Y., Maeda A. et al. Inhibitory effects of a lecithinized superoxide dismutase in bleomycin – induced pulmonary fibrosis in mice // Am. J. Respir. Critic. Care Med.— 2000.— Vol. 161 (4).— P. 1279—1284.
28. Kelner M.J., Uglik S.F. Superoxide dismutase abolishes the platelet-derived growth factor-induced release of prostaglandin E2 by blocking induction of nitric oxide synthase: role of superoxide // Arch. Biochem. Biophys.— 1995.— Vol. 322 (1).— P. 31—38.
29. Jurpaev K., Litvinov D., Dubovaya V. et al. Induction of vascular endothelial growth factor by nitric oxide in cultured human articular chondrocytes // Biochimie.— 2001.— Vol. 83 (6).— P. 515—522.
30. Lange R.W., Germolic D.R., Foley J.F., Luster M.I. Antioxidants attenuate anthralininduced skin inflammation in BALB/c mice: role of specific Proinflammatory cytokines // J. Leukoc. Biol.— 1998.— Vol. 64 (2).— P. 170—176.
31. Chang S.C., Kao M.C., Fu M.T., Lin C.T. Modulation of NO and cytokines in microglyal cells by Cu/Zn superoxide dismutase // Free Radic. Biol. Med.— 2001.— Vol. 31 (9).— P. 1084—1089.
32. Yin M., Wheeler M.D., Connor H.D. et al. Cu/Zn superoxide dismutase gene attenuates ischemia-reperfusion injury in the rat kidney // J. Am. Soc. Nephrol.— 2001.— Vol. 12.— P. 2691—2700.
33. Campana S., Zervoudis S., Perdereau B. et al. Topical superoxide dismutase reduces post-irradiation breast cancer fibrosis // J. Cel. Mol. Med.— 2004.— Vol. 8 (1).— P. 109—116.
34. Mizushima Y., Hoshi K., Yanagawa A., Takano K. Topical application of superoxide dismutase cream // Drugs Exp. Clin. Res.— 1991.— Vol. 17 (2).— P. 127—131.
35. Vorauer-Uhl K., Fürnschlief E., Wagner A. et al. Topically applied liposome encapsulated superoxide dismutase reduces postburn wound size and edema formation // Eur. J. Pharm. Sci.— 2001.— Vol. 14 (1).— P. 63—67.
36. Benyahia B., Campana F., Perdereau B. et al. Effects of superoxide dismutase topical treatment on human skin radiofibrosis: a pathological study // Breast.— 1996.— Vol. 5.— P. 75—81.

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Лікування келоїдних і гіпертрофічних рубців місцевим кремом, що містить рослинну супероксиддисмутазу (СОД)

Мета роботи — оцінити терапевтичну активність і толерантність місцевого використання крему супероксиддисмутази при келоїдних і гіпертрофічних рубцях з урахуванням еволюції їх розмірів та об'єму, а також таких параметрів, як колір, свербіж, еритема, твердість та біль.

Методи. У дослідженні взяли участь 27 пацієнтів, які місцево 2 рази на добу протягом 3 місяців наносили крем з вмістом СОД (SODERMIX® cream).

Результати. Наприкінці лікування спостерігалося значне зменшення розмірів (37,07 %; $p < 0,05$) та об'єму (48,44 %; $p < 0,05$) рубців. Нормалізація кольору та значне зменшення свербежу протягом місяця, а також еритеми були найкращими показниками ефективності лікування. Толерантність була хорошою протягом усього терміну лікування.

Висновки. Місцеве використання крему з вмістом СОД продемонструвало ефективність при келоїдних і гіпертрофічних рубцях та може бути альтернативним лікуванням при цій патології.

Ключові слова: місцева супероксиддисмутаза, СОД, гіпертрофічні рубці, келоїдні рубці.

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Лечение келлоидных и гипертрофических рубцов местным кремом, содержащим растительную супероксиддисмутазу (СОД)

Цель работы — оценить терапевтическую активность и толерантность местного использования супероксиддисмутазы при келлоидных и гипертрофических рубцах с учетом эволюции их размеров и объема, а также такие параметры, как цвет, зуд, эритема, твердость и боль.

Методы. В исследовании приняли участие 27 пациентов, которые местно 2 раза в сутки в течение 3 мес наносили крем с содержанием СОД (SODERMIX® cream).

Результаты. В конце исследования наблюдалось значительное уменьшение размеров (37,07 %; $p < 0,05$) и объема (48,44 %; $p < 0,05$) рубцов. Нормализация цвета и значительное уменьшение зуда в течение месяца, а также эритемы были лучшими показателями эффективности лечения. Толерантность была хорошей в течение всего срока лечения.

Выводы. Местное применение крема с содержанием СОД продемонстрировало эффективность при келлоидных и гипертрофических рубцах и может быть альтернативным лечением при этой патологии.

Ключевые слова: местная супероксиддисмутаза, СОД, гипертрофические рубцы, келлоидные рубцы.

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