Chelaskin - Stop bruising and flush

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Summary

Company

Mesotech is an innovative company, specialized in providing skin care and beauty solutions from the conception to the manufacturing of products and devices for the aesthetic medical field.

Customer satisfaction and quality are the main priorities for our staff. We develop our range relying on an ongoing dialogue with our customers.

Our Mission

Development of new products from original ideas to a deep research. Original formulations where unique ingredients are selected to restore and correct blemishes and cutaneous.

CHELASKIN: STOP BRUISING AND FLUSH

One of the biggest challenges with aesthetic medical procedures is "how to avoid the down time" and "how to minimize the tell-tale signs "associated with these procedures. The possibility of bruising postprocedure, which is an obvious sign of having had "something done," can be a quandary for many who wish to avoid calling attention to themselves. "What happened to you? Are you safe at home? What does the other guy look like? "These are just a few of the common unsolicited questions clients compare days to weeks after an aesthetic procedure results in bruising.

Our product, Chelaskin, was developed to respond to the growing need of the many patients who, using aesthetic medicine, want to avoid all those annoying cutaneous manifestations, a reason for embarrassment and social unease.

Below is a brief analysis of the main aesthetic problems that occur after cosmetic medicine treatments.

Main skin disorders after aesthetic medicine

Bruises

A bruise, also known as a “contusion” or “ecchymosis,” is a small hemorrhagic spot that results from extravasation of blood; it is found in the skin or mucous membrane and presents as a nonelevated, rounded or irregular, blue or purplish patch (Dorland, 2003).

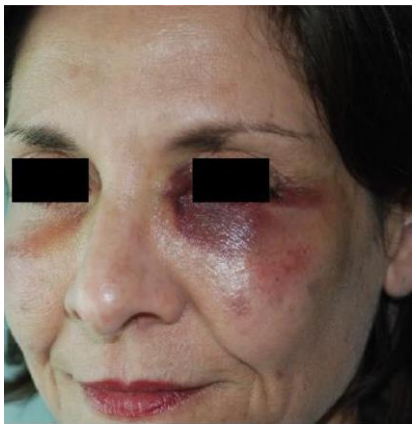


Figure 1. Bruising after cosmetic surgery treatment

Bruises change in appearance over time, and it may be possible to tell how old a bruise is on the basis of its appearance. When it fi rst appears, a bruise will be reddish looking, refl ecting the color of the blood in the skin. By 1–2 days, the reddish iron in the blood undergoes a change and the bruise will appear blue or purple. By Day 6, the color changes to green and by Day 8–9, the bruise will appear yellowish-brown. In general, the bruised area will be repaired by the body in 2–3 weeks after which the skin will return to normal.

Some clients bruise more than others especially if they are taking drugs. Clients tend to bruise more easily because of skin thinning that occurs with age.

All dermal fillers have the potential to cause bruising (Figure 1). Bruising is observed more frequently after injection into the dermal and immediate subdermal planes using fanning and threading techniques.[1] Less bruising is seen when materials are injected using the depot technique at the preperiosteal level.

Short-term post-traumatic edema

Some transient swelling in the immediate postprocedural period is normal and occurs with all dermal fillers. This type of edema occurs very shortly after injection and is related to injection volume and technique.[2]

Flush

Flush (Redness) can be defined as a sensation of warmth accompanied by a visible erythema of the skin.[3] It is associated with an increase in cutaneous blood flow following superficial vasodilation.[3-4] In general, redness occurs on the face,(Fig.2) neck and upper trunk, as it is in these regions that the density of superficial skin vessels and vascular response are more important. The reasons that cause the redness are heterogeneous: it can derive from the direct action of various substances on the smooth muscle fibers of the vessels or it can be related to the nervous system, peripheral or central, through the stimulation of the nerves

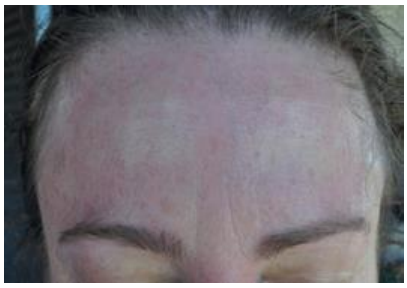


Figure 2. Flush post-procedure

Cutaneous Deposits

The cutaneous deposition disorders are a group of unrelated conditions characterized by the accumulation of either endogenous or exogenous substances within the skin.
These cutaneous deposits are substances that are not normal constituents of the skin and are laid down usually in the dermis, but also in the subcutis, in a variety of different circumstances.
Cutaneous deposits can be due to exogenous or endogenous substances that penetrate the skin because of both voluntary and involuntary reasons, including the particle materials used in tattoos and cosmetic fillers, or the accidental inclusion of external substances secondary to cutaneous trauma. [3]

Iron and heavy metal cutaneous deposits

The hemosiderin is an iron deposit compound, which is generated when the amount of metal is in excess of the normal binding capacity of Ferritin, the protein responsible for iron storage. [5]

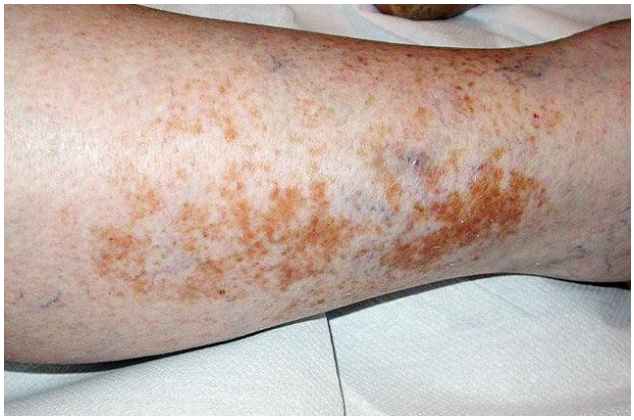


Fig. 3 Hemosiderinic dyschromia

Cutaneous deposits can showed for Chronic venous insufficiency (CVI).[6] Haemosiderinic dyschromia (HD) of CVI is a pathological entity that features a brown colored spot resulting from the deposit of free iron within leg tissues.
Iron is a highly irritative element capable of stimulating free-radical release and of causing leg ulcers, thus producing an ulcerated hemosiderinic dyschromia (UHD). It occurs when blood cells leak out of blood vessels. The hemoglobin from red blood cells is broken down into hemosiderin that is then permanently stored within the tissues. This can take place after a significant injury in the leg and is often worsened by an underlying venous problem. Since extravasated blood cells with hemoglobin are phagocyted by tissue macrophages called siderophages, the accumulation of hemosiderin within the injury area is a characteristic feature of the disease, resulting in the brownish color of the skin[7.8].

Iron is thought to be a co-factor or mediator of skin toxicity in a variety of pathological situations, including sunburn,[30] porphyria cutanea tarda,[27] inflammation,[28] and skin cancer,[29] as well as in hereditary hemochromatosis (HH).[31]

Zinc Deposits

Zinc deposition has been reported on the lip after the use of a zinc oxide containing sunblock.[8] It presented as a well-demarcated dark black macule. Histologic examination showed the submucosal deposition of fine golden yellow granules in a superficial location upon the extracellular matrix and around blood vessels.[9]

The new Chelaskin formula

The new formula of Chelaskin is the result of Mesotech's Research and Development Laboratories. Company mission is to develop highly effective products, in order to meet customers 's needs who are looking for very high performance products, without losing sight of the toxicological profile of the raw materials used.

The new Chelaskin formula is even richer in active ingredients, present at high concentrations and with strong soothing, chelating and antiaging properties, able to counteract the phenomena of both biological and chemical-physical nature that underlie the manifestation of these imperfections skin.

Chelaskin helps to remove post-laser redness, skin-roller, mesotherapy, filler, sclerotherapy, dermabrasion, rhinoplasty etc. Thanks to the chelating action of iron and zinc, it reduces dark circles and hyperpigmentation caused by bruising, hemosiderin dyschromia and hematomas. The free radical scavenging activity given by the presence in the formula of the arbutin, that guarantees protection from oxidative phenomena that can trigger following the treatments of aesthetic medicine. It also performs an effective purifying action, removing the dermal deposits of heavy metals that promote aging.

Main active ingredients of chelaskin

Lactoferrin

Lactoferrin is an iron binding glycoprotein that consists of a single polypeptide chain. It is the second most abundant protein in human milk [10,11] and is found in most exocrine secretions including tears, nasal secretions, saliva, intestinal mucus and genital secretions [12,13]. Lactoferrin is a member of the transferrin family of non-heme iron binding proteins [14]. Members of the transferrin family are distinguished from other iron binding proteins by their unique anion requirement for binding of iron. Lactoferrin differs from serum transferrin in its higher iron binding affinity and in its unique ability to retain iron over a broad pH range [15-16]. These differences, together with the differential tissue distribution of lactoferrin relative to other transferrins, contribute to the unique functional properties of lactoferrin.

Modulation of the immune response

Lactoferrin can regulate the cutaneous immune response when applied to the skin as an O / A emulsion. Considering that a) lactoferrin is produced at high levels in human individuals with skin allergic reactions [17], b) the protein is produced locally in the epidermis of normal skin and c) lactoferrin exhibits competitive binding to putative receptors on keratinocyte cells, thereby indicating a potential to directly modulate the function of these epidermal cells [18,19].

Lactoferrin Inhibits allergen induced cutaneous immunity

Analysis of the effects of lactoferrin on oxazolone induced cutaneous immune response demonstrated that administration of the protein by either route resulted in a dose dependent inhibition of Langherans Cell migration and accumulation of Dendritic Cell within the draining lymph nodes. Surprisingly, the inhibitory effect of lactoferrin was independent of its iron saturation status suggesting that its immune regulatory activity may be independent of its iron binding function. The inhibitory effect was also observed when the inflammatory response was initiated in the absence of allergen by IL-1β but was not observed when TNF-α was used as the initiating stimulus. Taken together, these results indicate that lactoferrin functions downstream of IL-1β by interacting directly with keratinocyte cells to downregulate the de novo production of TNF-α. Further, the findings demonstrate that lactoferrin can directly inhibit local inflammatory responses in vivo by a mechanism independent of its ability to bind LPS.[20]

Wound re-epithelialization

Decreased cell proliferation, delayed cell migration and increased cell apoptosis during wound healing are common adverse events that impede tissue repair. Delayed wound healing increases the possibility of open wound infection, which can further impair healing.

The study conducted by L. Tang et al. He stated that Lactoferrin may be useful in wound healing, acting as a growth factor for keratinocytes, and complementary stimulating their activities when the function of EGF is compromised due to a lack of reactivity of keratinocytes or deficiency of EGF receptors, as in chronic wounds. (Fig.4)

Importantly, Lactoferrin greatly enhanced cell viability and decreased cell apoptosis. This protective effect of LF may be another potential beneficial role of LF in wound healing.[21]

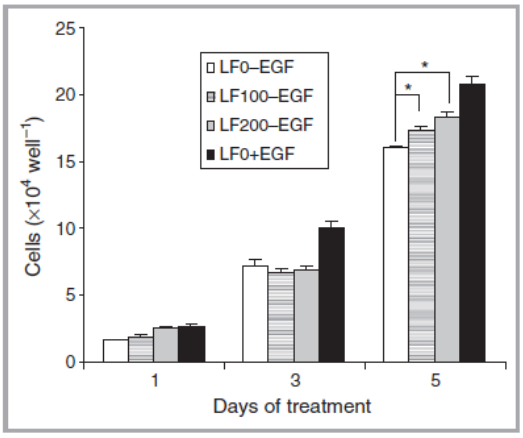


Fig 4. Lactoferrin stimulates the proliferation of human keratinocytes in the absence of EGF

Production of extracellular matrix components

Hyaluronic acid is an important component of the extracellular matrix in the dermis and epidermis and participates in numerous processes involved in wound healing, including cell migration and proliferation and modulation of the inflammatory response [22-24]. Hyaluronic acid is also essential for forming granulation tissue during wound healing and interacts with collagen, fibronectin and fibrinogen present in this specialized tissue, promoting fibrin polymerization and clot formation.

Fibroblasts are the cells responsible for the production of extracellular matrix components. The concentration of these components regulates the activity of fibroblasts, as well as the stimulation given by growth factors and cytokines.

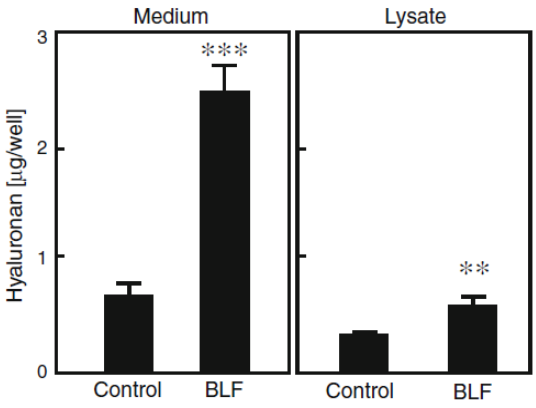


Fig. 5 Effect of BLF on hyaluronan synthesis in NHDFs. Cells were cultured in serum-free DMEM for 4 days in the absence or presence of 2 lM BLF. The amount of hyaluronan in conditioned media or in cell lysates was measured by an ELISA-like hyaluronan competitive binding assay. Error bars show standard deviations of triplicate measurements.

Statistical significance between treated and control cells are indicated as ** P\0.01; *** P\0.001

According to a Saito et al. study [26] showed that BLF-enhanced hyaluronan synthesis in NHDFs (Fig. 5). This study also indicated that Lactoferrin enhanced HAS2 gene transcription (Fig. 6) and increased HAS2 protein expression.

The molecular weight of hyaluronan is an important factor for its biological functions, and is dependent on which HAS is used for hyaluronan synthesis. HAS2 produces high molecular weight (HMW) hyaluronan, whereas HAS1 and HAS3 produce smaller hyaluronan[25]. The effect of HAS2 on cell motility was more prominent than that of HAS1 or HAS3 [25]. Thus, BLF potentially promotes fibroblast motility by stimulating HAS2 gene expression, while the HAS2 expression level is varied in the types of fibroblasts and their physiological status. Finally, this study showed that Lactoferrin promotes COL1A1 mRNA transcription and collagen synthesis in a dosedependent manner (Fig. 4). In addition to hyaluronan, type-I collagen is a major component of the extracellular matrix in dermis and promotes wound healing.[26]

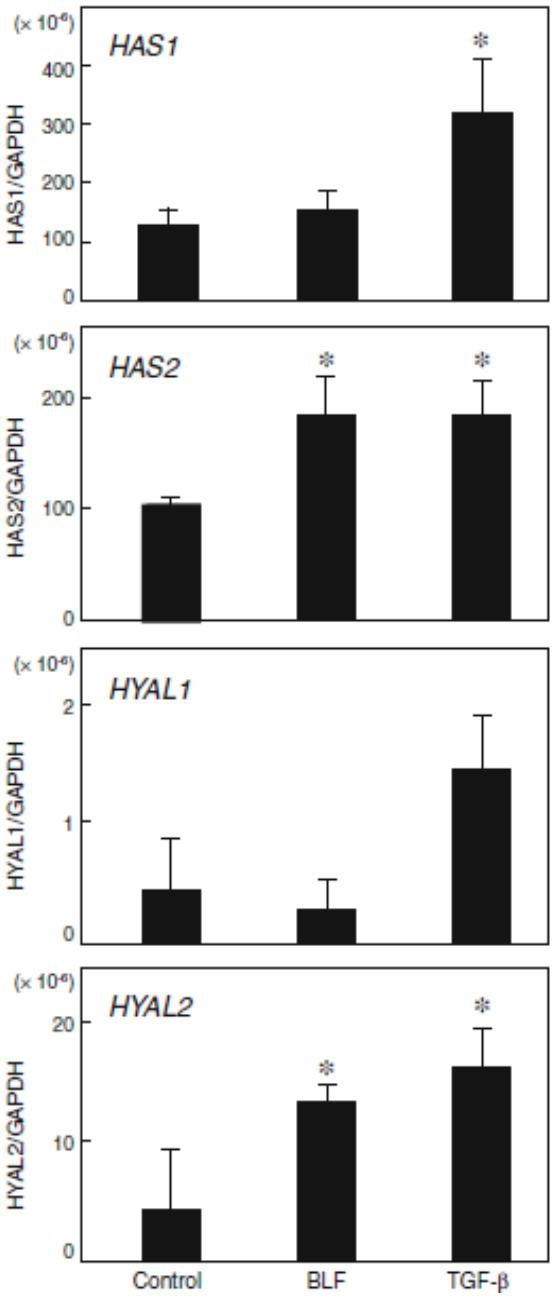


Fig. 6 Effect of BLF on HAS and HYAL mRNA expression in NHDFs. Cells were cultured for 16 h in the absence or presence of 1 lM BLF or TGF-b (10 ng/mL). The relative mRNA expression levels of HAS1, HAS2, HYAL1, and HYAL2versus GAPDH were measured by real-time PCR. Error barsshow standard deviations of triplicate measurements.

Statistical significance between treated and control cells are indicated as * P\0.05

Treatment of hemosiderin dyscromias and iron deposits

Ochre dermatitis is a secondary pigmentary disorder of venous stasis in which the increase in intravascular pressure and endothelial alterations cause extravasations of erythrocytes, hemosiderin-laden macrophages, and melanin deposits. [32]

9 patients presenting with haemorrhages and hemomideric dyschromia, both associated with refractory ulcers, were the subject of the study conducted by Brizzio et al. 2012 [33]. The main finding was that topic application of Lactoferrin allowed a fast and progressive reduction in the dimensions of the area of the ulcer in 9 of 9 patients and complete closure in 7 of 9 cases. The 90 days of evolution evidenced an important improvement in the injuries, with a reduction in the intensity of the brown color of the spot (9 of 9) and time to complete scarring ranging from 15 to 180 days (7 of 9).

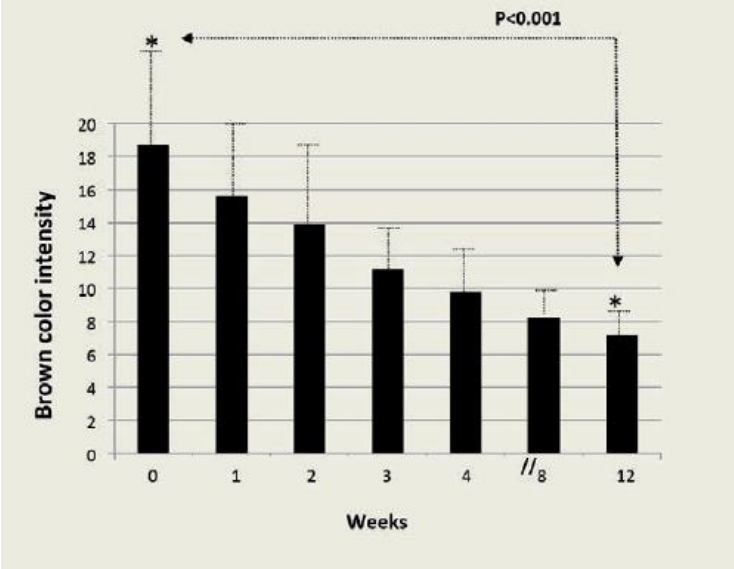


Figure 7 Variation of the value in the blue scale during treatment.

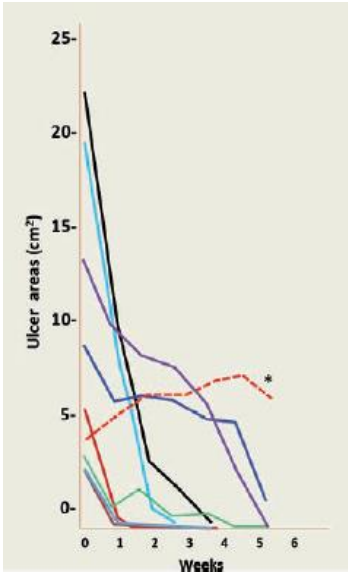


Figure 8 Decreased ulcer area during 6 weeks

One of the most remarkable findings was the significant decrease, in all cases, of the brown color of the HD and the size of the ulcerous areas (Figures 5 and 6), with a concomitant goniometric improvement and complete closure of lesions in 7 cases after six months of treatment. The rate of healing was independent of baseline or recurrent ulcers (Figure 7). In all patients, clinical improvement of thewounds (10 ulcers) was associated with a significant decrease in pain and improvement in quality of life, except in one case (*case no. 3161) due to a domestic accident on the lesion, which showed no clinical improvement and led to the patient discontinuing treatment (Figure 8).

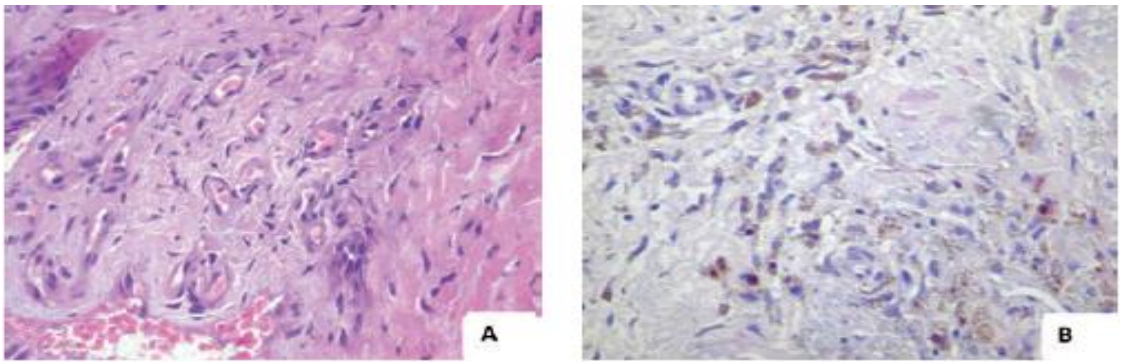


Figure 9. (A) Histological features before treatment: presence of fibrin sleeves, small vessels, extravasations of red blood cells, fibrosis, chronic inflammatory pattern. (B) Histological features after treatment (4 weeks): presence of new vascular structures, extravasations of red blood cells, fibrosis and granulation tissue-granulation, chronic repairing inflammatory pattern

All biopsies showed changes in cytological patterns (Figure 9). In several cases, a decrease was seen in the high level of staining for HS in periulcerous and ulcer fundus biopsies present during the initial control and this associated with a significant improvement in the edema and ulcerous areas after treatment. [33]

Arbutin

Arbutin (Fig. 10) Is a glycosylated hydroquinone that is abundant in the leaves of several plant species such as wheat, pear, and bearberry plants in the genus Arctostaphylos. Arbutin demonstrates excellent safety and causes no adverse health effects such as toxicity and irritation[34]. In erythrocytes, arbutin shows long-lasting radical scavenging properties and also protects a membrane lipid from oxidative stress in human skin fibroblasts [35-37]. Recently, arbutin was effective in post-inflammatory hyperpigmentation (PIH), which is a reactive hypermelanosis and sequela of various inflammatory skin conditions [38], suggesting that arbutin can regulate not only radical-mediated stress, but also the inflammatory response.

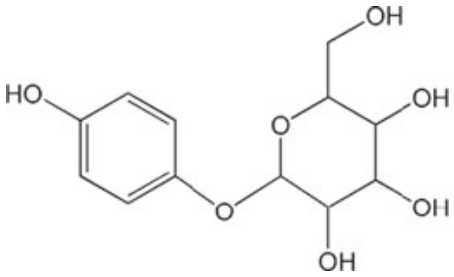


Figure 10 Molecular structure of Arbutin

Free Radical Scavenging Activity

The association of reactive oxygen species (ROS) and free radicals with many disease states is now well recognized and antioxidants have attracted considerable attention [39,40]. Antioxidants have an important role in the skin to prevent skin ageing, skin disorders and skin diseases [41]. One of the most abundant and most famous antioxidants in nature is Vitamin C. Its activity is

given by the presence in the structure of two hydroxyl groups able to neutralize the oxidizing compounds.

The arbutin has a hydroxyl -OH group similar to 2 stable derivatives of ascorbic acid 2-O-α-D-Glucopiranosyl-L-ascorbic acid (AA-2G) and 2-O-β-D-glucopyranosyl-L-ascorbic acid (AA-2βG), and for this reason, numerous scientific studies have been conducted to ascertain the antioxidant activity. In particular, the study conducted by Takebayashi et al. showed that arbutin possesses a long-lasting radical scavenging property by DPPH radical-scavenging assay and ABTS1 scavenging assay and that the antioxidant activity of arbutin was comparable to or even superior to that of hydroquinone in the ORAC assay and two cell-based antioxidant assays using erythrocytes and human skin fibroblasts. [42] (Figure 11)

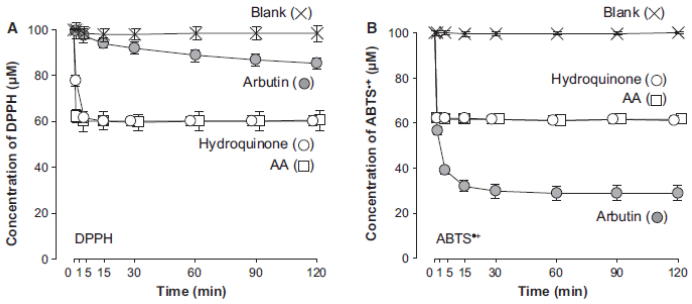


Figura 11. Time courses of DPPH radical- (A) and ABTS•_-scavenging (B) reaction of arbutin, hydroquinone and AA. Arbutin, hydroquinone or AA (20 □M) and DPPH radical or ABTS•_ (100 □M) were incubated at 25°C in 60% ethanol/40% citrate buffer (10 mM, pH 5) or citrate buffer (50 mM, pH 5), respectively. Changes in the remaining radicals were measured at the indicated times. Each value is themean _ SD of three separate experiments. Absence of SD bar means that the SD bar is within the symbol.

Anti-inflammatory effect

When an inflammatory process begins, macrophages proliferate, differentiate, or become activated through the effect of interleukins or growth factors [43]. Under certain circumstances, when chronic inflammation occurs, macrophages have a harmful effect, promoting inflammation, extracellular matrix (ECM) destruction, apoptosis, cell proliferation, and angiogenesis [44,45]

Lipopolysaccharide is an important pro-inflammatory factor that can cause endotoxemia and even shock and multiple organ dysfunction syndromes [46]. Stimulation with LPS can induce the expression of iNOS, COX-2, proinflammatory cytokines, and chemokines in microglia/ macrophages.[47] Subsequently, activated microglia/macrophages may secrete many pro-inflammatory cytokines, including IL-1b and TNF-a, which in turn can act on these cells in an autocrine manner [48].

A study conducted by Lee et Al., investigated about the effect of arbutin on the expression of inflammation-related genes in LPS-stimulated BV2 microglial cells. results showed that arbutin inhibited LPS induced iNOS expression and NO production. Additionally, arbutin attenuated LPS-induced gene expression and the release of pro-inflammatory cytokines such as IL-1b, TNF-a, and IL-6, as well as the chemokine MCP- and CXCL1. Furthermore, this suppression was associated with a decrease in NF-jB transcriptional activity. Taken together, our data suggest an antiinflammatory effect of arbutin on LPS-stimulated BV2 microglial cells. Finally, arbutin decreased the expression of Ninj1, which is an important adhesion molecule. We also found that

arbutin can attenuate pro-inflammatory molecules without cellular toxicity in murine BV2 microglial cells.[49]

Aloe Vera

Several studies have reported the anti-inflammatory, cell proliferative, immune modulating, collagen stimulatory, antioxidative, and angiogenic activity of A. vera.

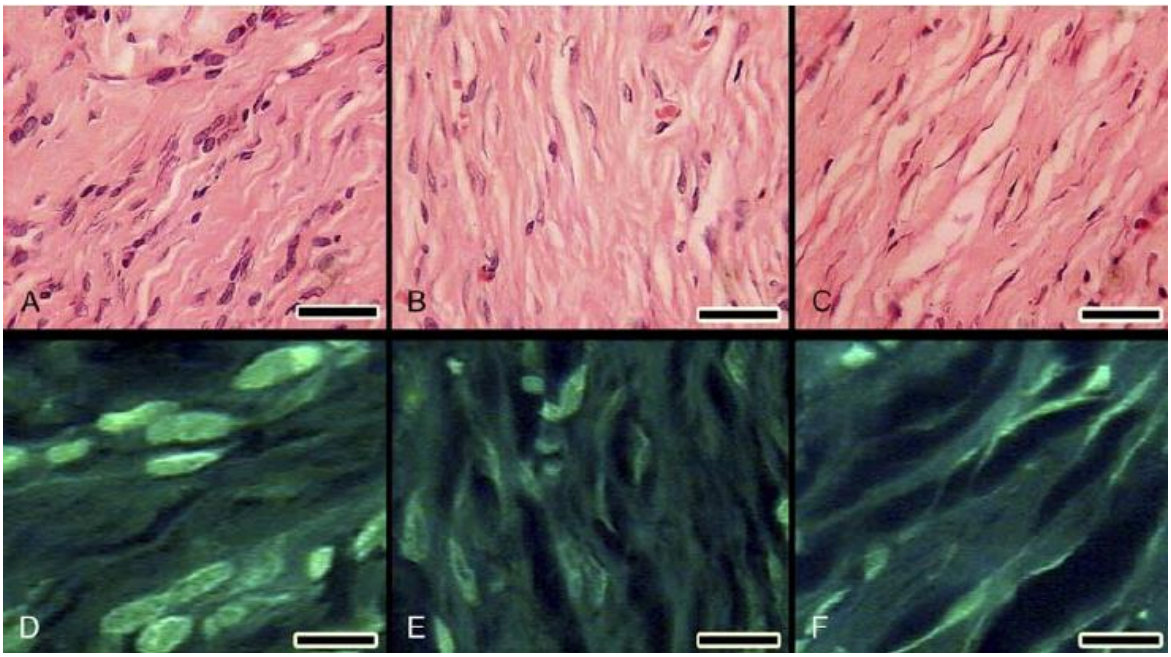


Fig. 12 Sezioni longitudinali del controllo (A, D), 25 mg / ml di A. vera (B, E) e 50 mg / ml di A. vera (C, F) a 30 DPI. Dalla A alla C sono macchiate da ematossilina e eosina. D a F sono figure invertite per una migliore chiarificazione delle strutture. Le barre della scala da (A) a (C) sono pari a 12,5 µm e (D) a (E) sono uguali a 6,25 µm. L'aloe vera ha ridotto in modo dose-dipendente i fibroblasti immaturi e le cellule infiammatorie ma ha anche migliorato la maturazione e l'allineamento dei fibroblasti a 30 DPI in modo che i fibrociti più maturi fossero presenti nelle lesioni trattate rispetto a quelli dei controlli.

A. vera has been proven to increase the rate of wound reduction, epithelialization and maturation.[50] At the earlier stages of wound healing, the reduced total cellularity, edema, and fibrin clot together with the elevated number of macrophages, fibroblasts, and large blood vessels observed in the treated lesions, compared to the controls, suggest that A. vera enhances the rate and quality of the inflammatory phase of wound healing.[51] Increase in the number of blood vessels in the treated lesions could indicate the angiogenic activity of A. vera at earlier stages of wound healing which established a better perfusion and appropriate circulation in the injured area. The extracellular matrix that is produced by fibroblasts is made up of collagen, glycosaminoglycans, and elastin.[52]

Furthermore, it indicated that higher number of fibroblasts and fibrocytes at different stages of wound healing had a strong positive correlation with the tissue level of collagen and glycosaminoglycans and A. vera increased the amount of these items in the healing treated

tissues compared to the unassisted control lesions. Once the tissue level of glycosaminoglycans increased and the fibroblasts proliferated, these cells become mature and their behavior would change.[52,53] At mid to late stages of fibroplasia, their matrix production is shifted from glycosaminoglycans to collagen production.[52,53] By increasing the collagen content of the healing tissue, a scar tissue forms and the tissue becomes mature.[52,54] Therefore, in the remodeling phase of wound healing, size of the scar tissue decreases because the collagen fibers are aligned and the free spaces between them decrease.[52,53]

At 30 DPI, the results suggest that A. vera significantly reduced the total cellularity and immature blood vessels. It also increased fibrocytes and caliber of the blood vessels and enhanced alignment of the collagen fibers in the injured area. Aloe vera also significantly increased the tissue level of collagen and dry matter content compared to the controls. In addition, it interestingly decreased the scar size at this stage. The remodeling phase could be divided into 3 stages of alignment, maturation, and consolidation.[50]

On the basis of the results of the present study, the treated wounds at 30 DPI were in the alignment and maturation stages of wound healing because the epidermis was fully regenerated, the wound contraction completely occurred, and the wounds closed. In addition, the scar tissue formed in the injured area was aligned and mature. The consolidation stage of wound remodeling lasted up for months. In this stage, the healing tissue may gain almost its normal mechanical strength.[55]
All these results suggest that A. vera improved the structural organization of the healing tissue and this enhanced hierarchical organization was responsible for the superior biomechanical performance of the healing treated tissues compared to the controls.

Licorice extract

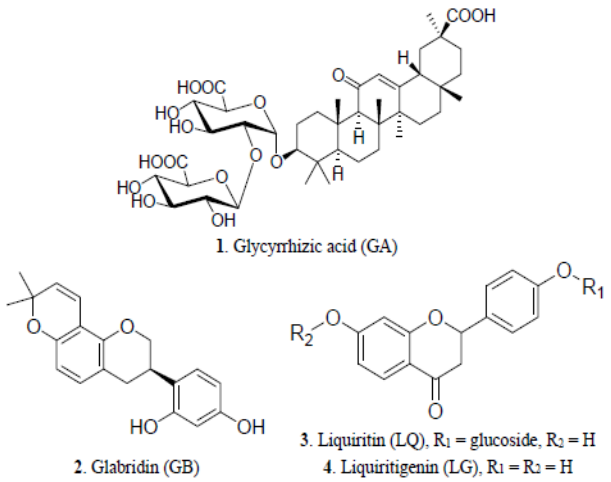


Figura 13. Molecular structures of the biologically active components of licorice extract.

Licorice (Glycyrrhiza glabra) is a traditional medicinal, sweet and soothing herb growing in several regions of the world. It is known that licorice has anti-inflammatory, anti-bacterial, antioxidative, anti-viral and expectorant properties [56-58] and is effective in the detoxification and protection of the liver[59].

The biologically-active components of licorice are well known as glycyrrhizic acid (GA, glycyrrhizin), liquiritin (LQ), glabridin (GB) and

liquiritigenin (LG) (Figure 13).

Recently, various studies have been performed that analyzed and characterized the primary and secondary metabolites of licorice its active components [60,61]. These studies demonstrated that the whole liquorice extract can be as effective as corticosteroids in the treatment of dermatitis, eczema and psoriasis. The inhibition of the inflammatory cascade may allow the repair of tissue damages and the prevention of carcinogenesis. The topical administration of anti-inflammatory and antioxidant products can substantially ameliorate the impaired conditions, by restoring the physiological balance and skin functionality. In this framework, glycyrrhizin and licorice extract may be promising candidates to prevent and treat local inflammatory pathologies and skin injuries.[62]

Free Radical Scavenging Activity

It is known that compounds with antioxidant properties may exert anti-inflammatory effects. The antioxidant effect of the licorice extract and its three active ingredients was then measured using a cell-free system. These data showed that the licorice extract and three compounds, GA, LQ and LG, have antioxidant effects [63](Figure 14)

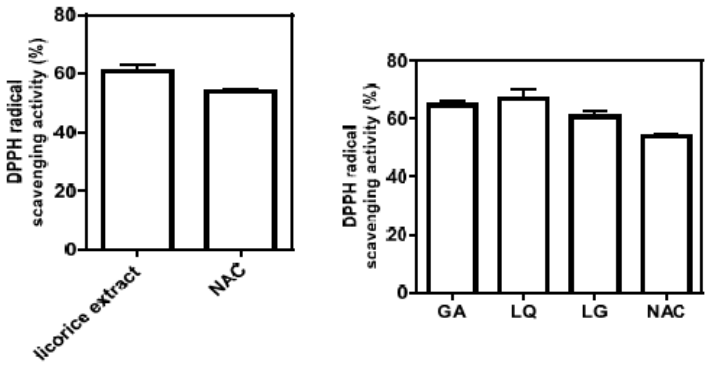


Figure 14. Free-radical scavenging activity of licorice extracts and three ingredients were measured by using the DPPH assay. The direct scavenging activity of licorice extract and its ingredients (GA, LQ and LG) on DPPH radicals was expressed as the % of control at 10 mg/mL of licorice root extract and 100 µM of GA, LQ and LG. NAC (N-Acetyl cysteine) 10 µM was used as positive control. The results are the means ± SD of three separate experiments.

Anti-inflammatory effect

The study performed by Ji-Yeon Yu et al. 2015, showed that the main components of the licorice extract GA, LQ and LG inhibited the LPS-stimulated NO production in a dose-dependent manner (Figure 15A). Furthermore, GA, LQ and LG inhibited the iNOS and COX-2 protein expressions in LPS-stimulated cells (Figure 15B). These result showed that bioactive compounds of licorice extract have anti-inflammatory activities through the inhibition of pro-inflammatory cytokine expression.[63]

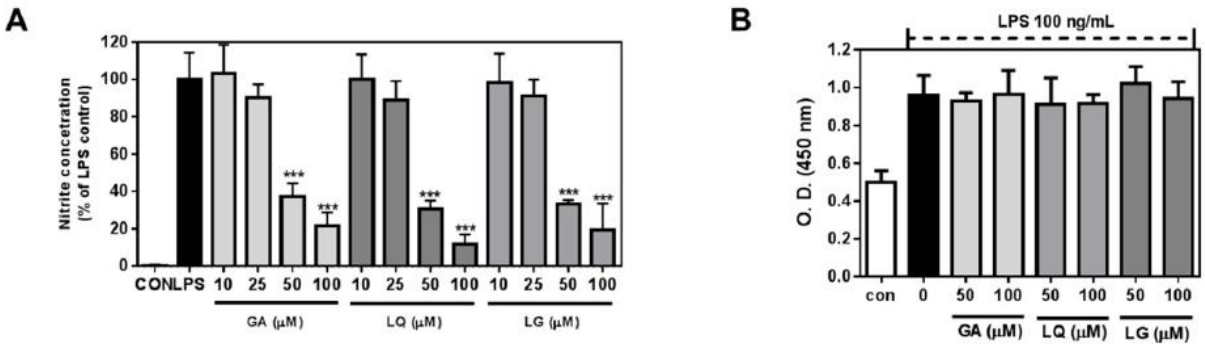


Figure 15. GA, LQ and LG suppressed the NO production and pro-inflammatory gene expression in LPS-stimulated BV2 cells. (A) Cells were exposed to each compound (50 and 100 μM) for 24 h in the presence of 100 ng/mL LPS. Cell viability was measured by theCCK-8 assay kit; (B) Cells were exposed to each compound (10 to 100 μM) for 18 h in the presence or absence of 100 ng/mL LPS. NO production was measured as nitrite concentration in the culture media

Efficay Trials

Fractional laser and Chelaskin combination

Dr. Cristina Rosati

Specialist in general surgery

Expert and consultant of aesthetic medicine – Agorà - Milano

Introduction

One of the most frequent question that patients ask when they come to our medical-aesthetic clinic, is how they can improve face photo-aging without “transforming” their face injecting or implanting extraneous substances.

For this reason we had the idea to use, in our patients affected by photoaging, laser in combination with a very interesting, natural molecule which is extracted from milk: Lactoferrin. We decided to conceive the fractional laser as a means to convey this multi properties molecule, because we’re convinced that if the fractional laser is already an excellent way to fight photo-aging, it can be even more efficient if “helped” by the properties of Lactoferrin.

A dual source fractional laser (erbium and thullium: Fraxel Restore Dual) has been used as a means to vehiculate lactoferrin; The fractional laser remodels dermal matrix layer through various steps: an early inflammation, damaged collagen removal, fibroblasts activation and subsequent neocollagenesis.

So, having available such important laser technology , like Fraxel Restore Dual, we asked why don’t we combine it to lactoferrin to take advantage of their synergistic features?

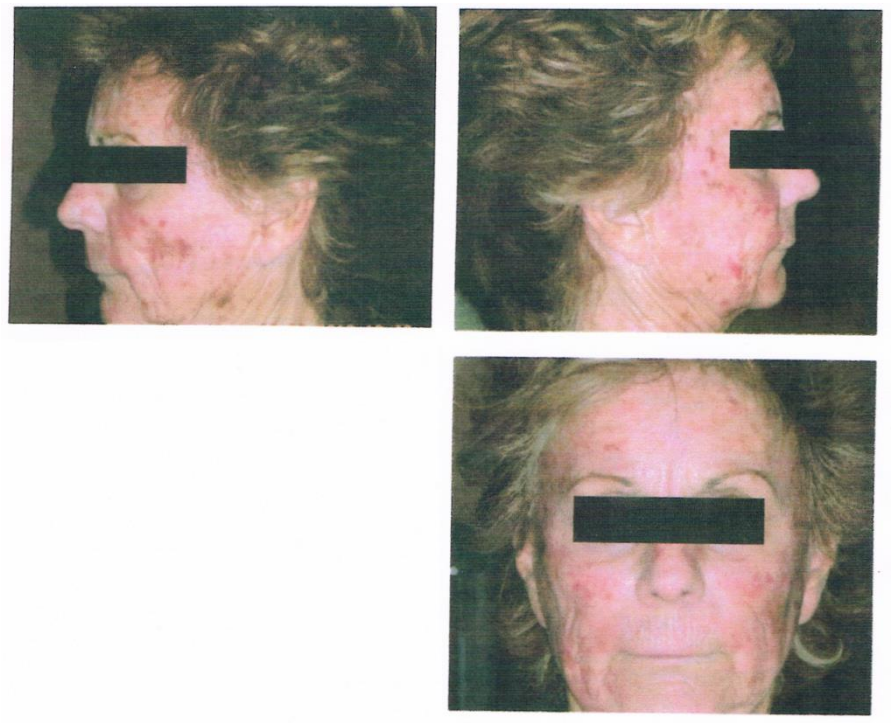


Figura 16. Photoaging case. After one day of treatment with Fraxel Restore Dual the classic post-laser triad, erythema, edema and swelling, are clear. Down time has been deeply reduced with the use of Chelaskin cream.

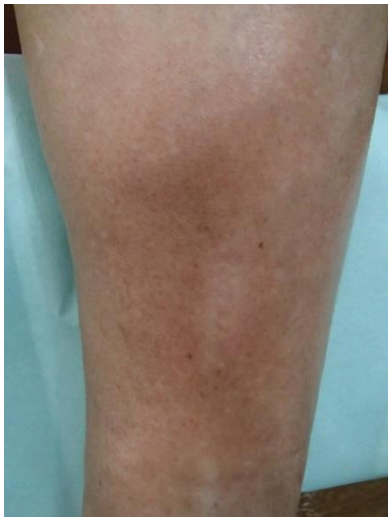
Method

As a protocol of our study we have used lactoferrin (Chelaskin cream) just after laser treatment to take advantage of its feature of modulating the inflammation; Than we continued at home with Chelaskin cream (2 times a day for one week) thanks to it's capacity to regenerate the epithelia and substituted it to the usual "cosmetic night cream" to take advantage from lactoferrin antioxidant features and it's capacity of stimulation on fibroblasts and on the production of hyaluronic acid and collagen. In case of severe photo-aging, being much more aggressive with the use of high energies, the prescription of lactoferrin has been useful for its safety into preventing infections.

Patients seemed very interested in this new "laser/Chelaskin" combined method and, when we described the reasons of our choices, they go on with home therapy with great motivation.

Congestive hyperpigmentation due to varicose vein

Chelaskin was applied twice a day for 2 weeks on the leg of 81 years old lady.



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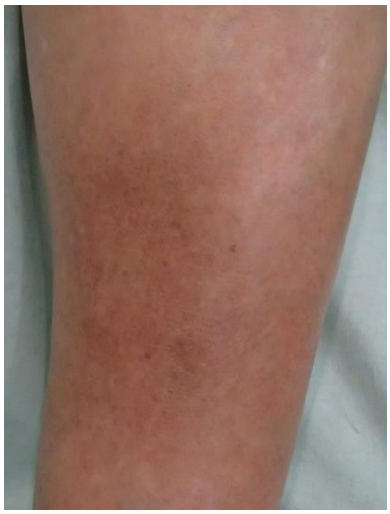

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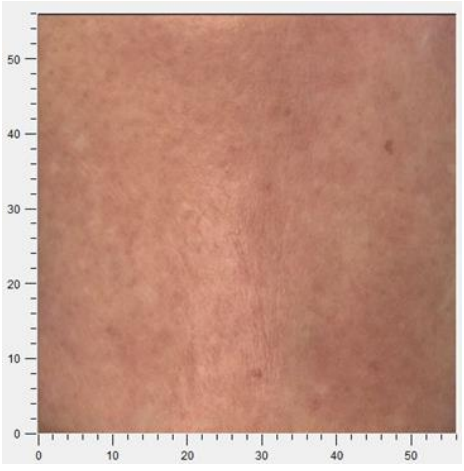
Hyperpigmentation decreased significantly

Images obtained through the use of Antera 3D

Detail of the treated area



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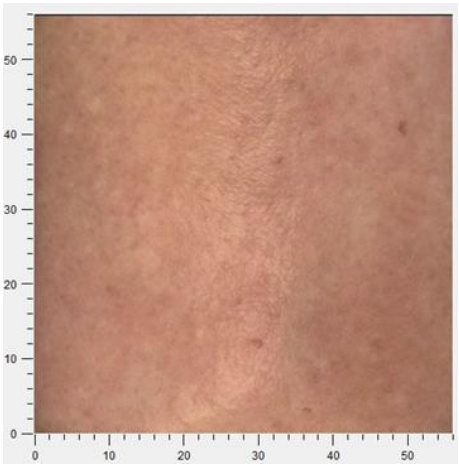

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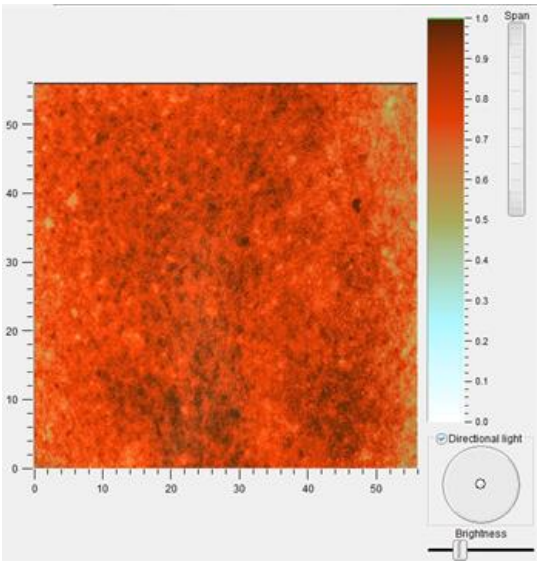
Detail of the treated area (Melanin)



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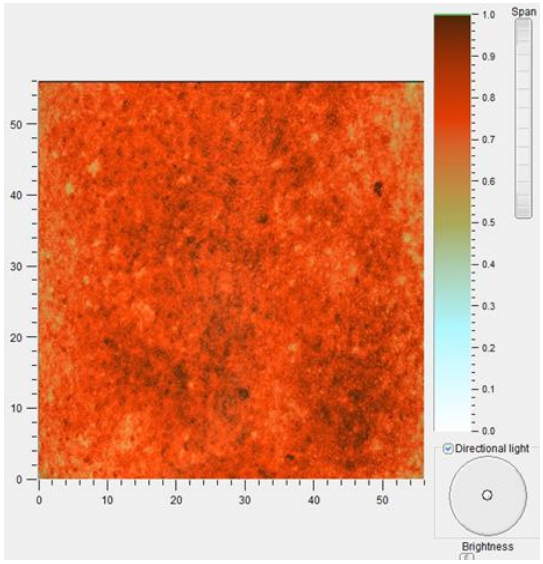

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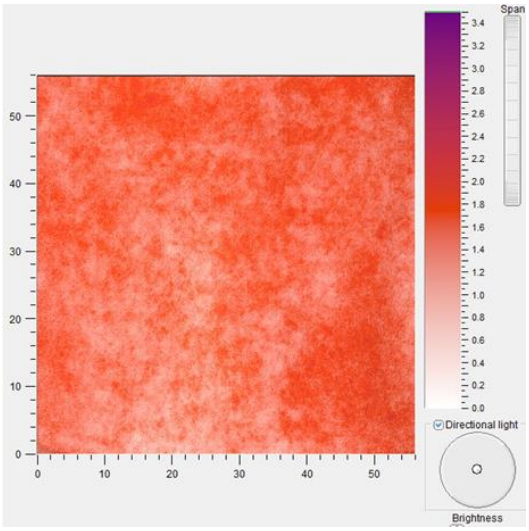
Detail of the treated area (Hemoglobin)



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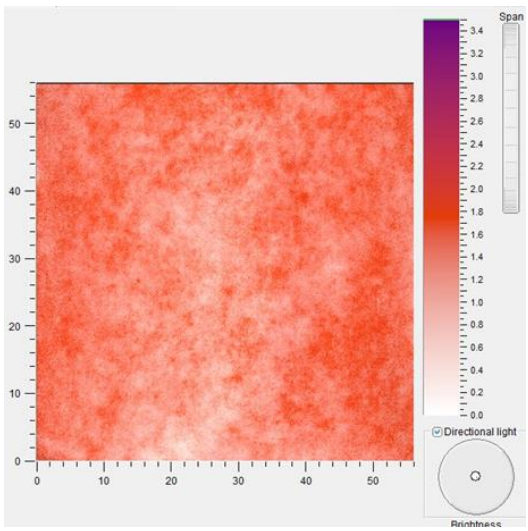

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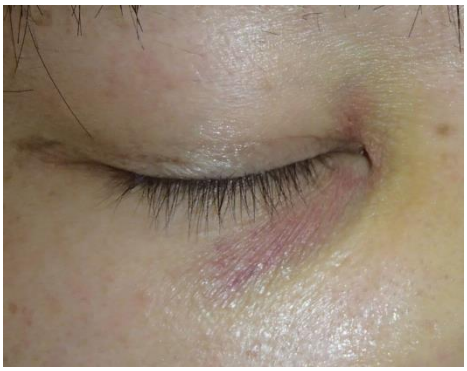
After a blow in inner canthus



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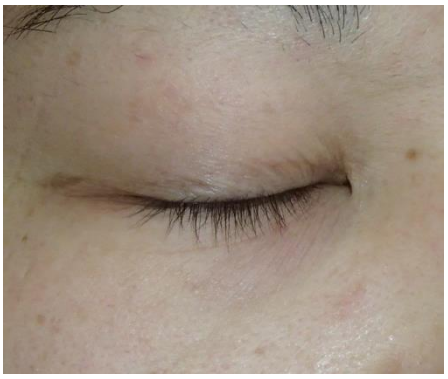

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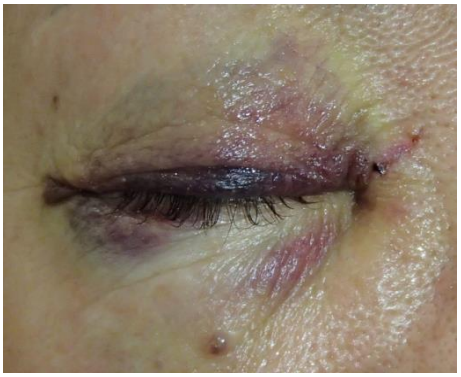
After a contusion



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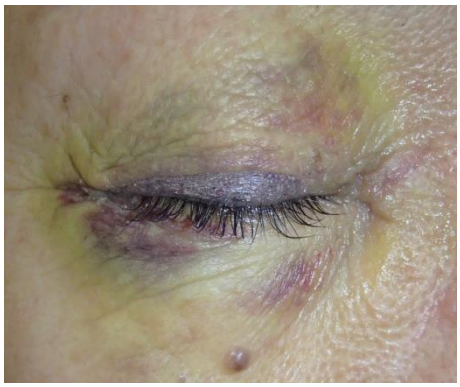

T=2

Medical aesthetic treatments that benefit the use of Chelaskin cream

- Non-ablative fractional laser: The method is based on a pixel system that creates some micro-necrosis areas without damaging the epidermis.
- Ablative fractional laser: In general they are CO_2 based and produce microscopic holes in the skin thickness by vaporization process (explosive distruction caused by very rapid increase of temperature). To use on intact skin only.

Both lasers stimulate new collagen production.

- Radiofrequency: You get it by collagen fibers heating resulting in their shortening and therefore a remodeling of treated area (face, neck, body)
- IPL (Intense Pulse Light): It's an electronic device commonly used for photo-epilation, but also for photo- rejuvenation.
- Diode laser: Used in aesthetic for hair removal
- Alexandrite laser: Post-epilation
- ND:YAG:Laser: Capillaries
- Ultrasound: Stimulate the birth of new collagen. *Indications:* skin laxity.
- Peeling: It's a treatment that stimulates exfoliation by the application of a chemical substance. So you get a smoothing effect and a rejuvenation of the face.

  Peelings are:

  - very superficial: glycolic acid (% less then 30%)
  - superficial: salicylic acid, glycolic acid, trichloroacetic acid 15-20%, Jessner solution;
  - medium depth: trichloroacetic acid 35%,
  - deep: : trichloroacetic acid or phenol.
- Bio-revitalizers: In general it's based on hyaluronic acid or on an amino acids cocktail. Some micro-injections are done in the derma layer to restore elasticity and hydration in the skin.
- Filler: Infiltration of of hyaluronic acid for filling purpose.

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