Efficacy and Safety of Cysteamine-Isobionicamidine Complex in Postinflammatory Hyperpigmentation: A Randomized, Double Blinded, Vehicle-Controlled Trial

Tzu-Li Liu  
Chang Gung Memorial Hospital

Tsung-Fu Tsai  
Chang Gung Memorial Hospital

Yi-Jing Lai  
Chang Gung Memorial Hospital

Chau Yee Ng (✉ mdcharlene@gmail.com)  
Chang Gung Memorial Hospital

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Abstract

Postinflammatory hyperpigmentation (PIH) is a common acquired hyperpigmentation disorder that can cause significant psychosocial impacts. Cysteamine has been shown to be an effective depigmenting agent for hyperpigmentation disorders, and a clinical trial was conducted to assess the efficacy and safety of cysteamine-isobionicamide complex for treating PIH. Forty patients were randomized into treatment and control groups, with assessments collected at baseline, week 4, 8, and 16. The treatment group showed significant improvement in dermatological assessments, MASI score, TPHI score, and life quality score compared to the control group. Mexameter® and VISIA skin analysis also showed significant improvement in the melanin index and erythema index score at week 16, with cellular resolution OCT imaging revealing decreased melanosome capping and fewer hyper-reflective melanophages. This study suggests that the cysteamine-isobionicamide complex has the potential to be a viable treatment option for PIH.

1 Introduction

Postinflammatory hyperpigmentation (PIH) is an acquired, reactive hypermelanosis that occurs in response to cutaneous inflammation. This condition causes the enhanced synthesis and deposition of melanin in skin cells, leading to an uneven distribution of dark and flattened spots on the body. PIH can be detrimental to one’s self-confidence and can have a negative impact on the quality of life and social/emotional functioning. Common endogenous causes of PIH include acne vulgaris, psoriasis, lichen planus, and atopic dermatitis. Excessive exposure to UV lights, inappropriate laser procedures, chemical peels, and non-ionizing radiation are classified as exogenous stimuli. Although there is no significant difference in the incidence of PIH among age and gender, it is well established that PIH affects skin-of-color patients with higher frequency and severity (Fitzpatrick skin types IV through VI). The pathogenesis of PIH is complicated. A variety of inflammatory mediators, such as prostaglandins, numerous cytokines, and reactive oxygen species (ROS), are involved in triggering melanocyte activity and transferring pigment to surrounding keratinocytes. For instance, the literature demonstrates that leukotrienes C4 and D4, prostaglandins E2 and D2, thromboxane-2, interleukin (IL)-1, IL-6, tumor necrosis factor (TNF)-α and epidermal growth factor exert melanocyte-stimulating properties. Moreover, injury to basal keratinocytes causes the release of melanin into the dermis, which is then phagocytosed by melanophages, leading to further dermal deposition of pigmentation.

Management of PIH primarily involves impeding deterioration through photoprotection and using agents to disperse melanin built up in the skin. Among current topical depigmenting agents, hydroquinone or a triple combination cream containing 4% hydroquinone, 0.05% tretinoin, and 0.01% fluocinolone acetonide, also known as the modified Kligman formula (mKF), is the preferred first-line treatment. Hydroquinone acts as a melanocytotoxic agent after being metabolized by the enzymes tyrosinase and peroxidase within melanocytes, causing the destruction of hyperactive melanocytes. However,
hydroquinone is also known to cause contact dermatitis and paradoxical darkening of the skin, especially with long-term or excessive use.\textsuperscript{3} Other possible adverse events include nail discoloration, transient halo hypochromia and abnormal skin repigmentation. The side effect profile of hydroquinone raises concerns for its long-term application.\textsuperscript{3,15,16}

Cysteamine is a small aminothiol metabolite that is produced during coenzyme A degradation in mammals.\textsuperscript{18,20-22} It possesses strong antioxidant properties by suppressing ROS generation and enhancing intracellular glutathione.\textsuperscript{23-27} It has been shown to be as effective as triple combination cream and intradermal tranexamic acid injections in treating melasma and lentigines.\textsuperscript{28-31} Recently, a novel compound called isobionicamidine, derived from the pyridine family of molecules, has been introduced as a melanosomal transfer inhibitor. It is believed that isobionicamidine acts in synergy with cysteamine to inhibit multiple melanogenesis pathways and impede epidermal pigmentation (personal communication with Dr. B. Kasraee). A proprietary product consisting mainly of the cysteamine isobionicamidine complex has been released, but its effectiveness and safety in treating PIH have not been investigated. Thus, a double-blinded, randomized, placebo-controlled clinical trial was conducted to evaluate the effectiveness of this product in treating PIH.

2 Materials And Methods

2.1 Trial Design and Recruitment

This double-blinded, randomized, placebo-vehicle controlled clinical trial assessed the safety and efficacy of cysteamine isobionicamidine complex as a treatment for patients with PIH. The study was conducted in accordance with the Declaration of Helsinki and ethical principles for human research. The protocol was approved by our institutional ethical committee (Ethics Committee of Chang Gung Memorial Hospital, IRB no: 201802265A3), and all participants provided informed consent prior to their inclusion in the study. The trial was registered at ClinicalTrials.gov under ID number NCT05206318 (25/01/2022).

Patients presenting to our Dermatology clinic, based at an academic tertiary care institution, with the chief complaint of PIH were screened by C.Y.Ng, and eligible participants were recruited. Patients who were over 20 years of age and had experienced more than twelve weeks of acquired hyperpigmentation following acne or laser therapy were eligible for enrollment. Exclusion criteria included patients with ongoing inflammatory symptoms or those receiving anti-inflammatory medications, pregnant patients, patients undergoing hormone therapy and/or oral contraceptives, patients presenting with dermal hyperpigmentation without epidermal involvement, and patients with a known history of allergic reactions to the product. A total of forty patients with PIH were recruited between September 2021 to September 2022. Recruited subjects were required to cease the use of topical hydroquinone, oral tranexamic acid and/or other skin whitening agents at least eight weeks prior to participation.

Patients were randomized using computer generated random numbers with a 1:1 random allocation ratio. Each patient was assigned a patient identification number upon screening, and the next randomization
number was provided at the baseline visit. Both participants and researchers were blinded to avoid bias. Patients were allocated to one of two groups: the treatment arm (cysteamine isobionicamidic complex, n=20) and the placebo vehicle-controlled arm (n=20). The study design of the trial is outlined in Supplement Fig. 1.

2.2 Outcome and Measures

To assess the severity of target lesions, a multi-modality subjective scoring system was utilized, consisting of the following components: (a) overall disease severity assessment rated on a scale of 1-8 (1 being normal and 8 being severe), (b) pigmentation intensity score rated on a scale of 1-5 (1 being normal and 5 being severe); (c) involved area of hyperpigmented lesions scored according to the affected area by percentage, on a scale of 0-5, with 0 indicating no involvement and 5 indicating involvement of more than 50%; (d) investigator global assessment score: scale 0-5, with 0 indicating no improvement and 5 indicating complete clearance of the hyperpigmentation lesions (scores 1 to 5 correspond to improvement of 1-10%, 11-25%, 26-40%, 41-50%, and more than 50% respectively), and (e) patient global assessment score: scale 0-5, with 0 indicating no improvement and 5 indicating complete clearance of the hyperpigmentation lesions (scores 1 to 5 correspond to improvement of 1-10%, 11-25%, 26-40%, 41-50%, and more than 50% respectively). The Melasma Area and Severity Index (MASI) score was used to assess post-laser hyperpigmentation lesions, while the total post-acne hyperpigmentation index (TPHI) score was used to evaluate hyperpigmentation resulting from acne inflammation. Patients were also asked to complete self-assessment and quality of life (QoL) questionnaires.

For objective evaluation, we used the Mexameter® (Courage + Khazaka electronic GmbH, Köln, Germany) to analyze the melanin index and erythema index of lesional and non-lesional skin. We obtained digital high-resolution images at every clinic visit using the VISIA skin analyzer (Canfield Scientific, New Jersey, USA). Additionally, a cellular resolution optical coherence tomography (Apollo Medical Optics, Taipei, Taiwan) was utilized to evaluate the melanin content of select patients in both groups at baseline and week 16 of treatment.

2.3 Intervention (Topical Agent Application)

Patients were randomly allocated to one of two groups: the treatment arm or the placebo vehicle-control arm. The treatment arm received a three-product system that included three different products which are used sequentially. To maintain blinding, both the treatment and placebo arms were given three products with equivalent labeling and secondary packaging. The three-product system in the treatment arm included: (1) a short contact product containing the cysteamine isobionicamidic complex and alpha hydroxylic acid (AHA); (2) a rinse-off cleanser containing AHA and L-Arginine complex; and (3) a leave-on product containing isobionicamidic complex and retinol. In contrast, all three products in the placebo vehicle-control arm were devoid of any active ingredients, including cysteamine, isobionicamidic, AHA, and retinol.
Participants were instructed to apply a thin layer of the first short contact product on their entire face once per day in the morning and leave it on for 15 minutes. Afterward, they were instructed to rinse thoroughly with water and cleanse with the second rinse-off cleanser. The last step was to apply a thin layer of the third leave-on product on the entire face. During the treatment period, all patients were advised to apply broad-spectrum sunscreen and avoid prolonged sun exposure.

2.4 Statistical Analysis

The randomization process was conducted using a random number generator by Graph Pad Prism 9 (Graph Pad Software, California, USA). Continuous variables were presented as mean (± standard deviation) or median (quartiles), while discrete variables were presented as count (percentage). Paired t-tests were used to compare the two groups, and Wilcoxon tests were performed to compare each visit with the baseline. Statistical analysis was conducted using GraphPad Prism 9 software, and a p-value of less than 0.05 was considered statistically significant.

3 Results

3.1 Demographics and Baseline Characteristics

Forty patients with acquired postinflammatory hyperpigmentation (PIH) were enrolled and randomly assigned to either the treatment group or the control group. The mean age was 41.3 ± 9.77 years in the treatment group and 39.8 ± 10.34 in the control group, and most of the recruited subjects were female (male/female ratio: 2/18 in the treatment group and 3/17 in the control group). All patients were of Asian ethnicity with Fitzpatrick skin types III and IV. The mean duration of PIH before treatment, as reported by the patients, was 9.95 ± 3.1 weeks in the treatment group and 10.05 ± 3.27 weeks in the placebo group, with no significant difference between the two groups (p=0.92). The impact of PIH on patients’ quality of life before treatment was high in both groups, with a Dermatology Life Quality Index (DLQI) score of 6.53 ± 3.63 in the treatment group and 6.1 ± 3.24 in the control group (p=0.72). There was no significant difference in pigmentary intensity and area of hyperpigmented lesions between the two groups at baseline. Table 1 summarizes the demographic data, clinical characteristics, and baseline hyperpigmentation severity of the study population.

3.2 Efficacy

During the 16-week trial, the investigator global assessment of both groups was recorded and is shown in Table 2. The subjective evaluation scores, which included the overall disease severity, pigmentary intensity score, and area of hyperpigmented lesions, significantly improved in the treatment group compared to the control group (p<0.05) as depicted in Fig. 1. At week 8, the treatment group showed a 26% reduction in TPHI score from baseline, which improved to a 43% reduction at week 16. A notable improvement in MASI score was also observed in the treatment group with a 21.5% reduction at week 8 and a 47.3% reduction at week 16 from baseline. Both TPHI and MASI score improvements in the treatment group were statistically significant when compared with the control group.
During each visit, both the investigator and patients completed global assessment questionnaires to evaluate the improvement of PIH after treatment. At week 16, 4 out of 20 (21%) patients in the treatment group were scored by the investigator as almost resolved, 9 (47%) as marked improvement, 6 (32%) as slight improvement, and 1 (5%) as no improvement. In the control group, 1 (5%) patient was scored as marked improvement, 5 (25%) as slight improvement, 7 (35%) as no improvement, and 7 (35%) as worse. These results were consistent between the investigator and patient assessment, with an interobserver agreement showing a moderate to high consistency (IGA = 0.82, PGA = 0.69). Additionally, the DLQI score of the treatment group decreased significantly compared to the control group during the experiment (treatment group: baseline: 6.5 ± 3.63, week 16: 2.157 ± 2.19; control group: baseline: 6.1 ± 3.24, week 16: 6.55 ± 3.3, p<0.001).

In addition to subjective evaluations, quantitative measurements also demonstrated a significant improvement in the treatment group. The melanin index of the treatment group showed a significant decrease at week 8 and week 16 compared to the control group (p<0.001) (Fig. 2A). The erythema index showed improvement in both treatment and control groups (Fig. 2B). VISIA skin analysis for surface spots and brown spots were obtained, and the absolute scores were compared between the two groups. The treatment arm showed significant improvement in brown spots after 16 weeks of treatment (Fig. 2C&D). Cellular resolution OCT skin imaging was obtained in five patients in both groups. Only subjects in the treatment group revealed a significant reduction in hyper-reflective melanophages and decreased melanosome capping in the dermal-epidermal junction compared to normal skin after 16 weeks of treatment (Fig. 3).

Representative clinical images of two patients with PIH before and after 16 weeks of treatment with cysteamine-isobionicamidine complex are presented in Fig. 4. The images demonstrate a visible improvement in hyperpigmented lesions and overall skin tone in both patients after treatment.

### 3.3 Tolerability

Minor adverse events were observed in both groups, and none resulted in discontinuation of treatment. The incidence of paresthesia, malodor, and skin peeling after product or placebo application was similar in both groups. Six patients in the control group and one patient in the treatment group experienced acne eruption. In the treatment group, three patients experienced allergic dermatitis after application. These adverse events were all classified as mild and resolved without intervention. Table 3 provides a summary of the adverse events observed in the study.

### 4 Discussion

Postinflammatory hyperpigmentation (PIH) is a common issue in individuals with skin of color, and the disease course is often resistant to treatment. To date, Modified Kligman's Formula (mKF), which contains 4% hydroquinone, 0.05% tretinoin, and 0.01% fluocinolone acetonide, has been the preferred treatment for PIH since the 1970s. However, long-term use of mKF is limited due to various side effects.
One of the main components, hydroquinone, which is the most commonly used topical agent for depigmentation, can lead to skin irritation, exogenous ochronosis, and unwanted melanocytotoxic or mutagenic effects.\textsuperscript{18,20,33} These side effects restrict its clinical use for long-term maintenance of hyperpigmentation disorders. Topical retinoic acid has a side effect profile of inducing skin peeling, erythema, and xerosis. Long-term use of corticosteroids contained in mKF can also lead to skin atrophy. Therefore, mKF is only recommended for use in PIH for a limited period of 8 weeks. As a result, there has always been a demand for the development of a safer, more effective, and long-lasting alternative treatment option for hyperpigmentation disorders. Cysteamine has been shown to be effective in the treatment of epidermal melasma in several randomized, double-blind, controlled clinical trials.\textsuperscript{20,22,34-36} This trial is the first to investigate the efficacy and safety of a novel topical agent, cysteamine-isobionicamide complex, in the treatment of PIH.

Cysteamine is a natural aminothiol metabolite with an excellent safety profile, and its oral form has been used to treat the inherited metabolic disease cystinosis with very few reported side effects.\textsuperscript{37,38} In its stabilized topical form, cysteamine has shown promising results in treating various hyperpigmentation disorders including melasma and lentigines. The pathways behind cysteamine's potent anti-pigmentation properties include inhibiting tyrosinase and peroxidase formation, increasing intracellular antioxidant glutathione, removing the pigment precursor dopaquinone, and regulating chelation of iron and copper involved in melanin synthesis pathways.\textsuperscript{19,39-41} Isobionicamide, an isoform of niacinamide (vitamin B3), has also been shown to exert higher activity than niacinamide in inhibiting melanosomal transfer in early \textit{in-vitro} models (personal communication Dr. R. Sfriso, unpublished data). The compound product of cysteamine and isobionicamide is the main focus of this study, and has recently been introduced in a three-part product system.

In this study, we found that the cysteamine-isobionicamide complex is effective for treating PIH, specifically post-laser and post-acne hyperpigmentation. After 8 weeks of treatment, improvement in MASI score, TPHI score, and subjective evaluations were noted. Global assessment scores evaluated by both patients and investigators confirmed the effectiveness of the cysteamine-isobionicamide complex compared to the placebo control. Furthermore, quantitative evaluation with the Mexameter®, VISIA skin imaging and cellular resolution OCT confirmed improvement in the treatment group compared to the vehicle control group. This trial suggests that the cysteamine-isobionicamide complex, currently offered in a proprietary three-product system, presents a viable treatment option for PIH. However, limitations of this study include a small study population that did not encompass all variants of PIH and a short-term study duration of 16 weeks. Long-term studies with a larger population are needed to further assess the effects and safety profile of the current formulation.

**Declarations**

**Conflicts of interest**: Chau Yee Ng is a global advisory board member and principal investigator of Scientis Pharma. The other authors declare that the research was conducted without any commercial or financial relationships that could be constructed as a potential conflict of interest.
Funding sources: This work was supported by the Scientis Pharma (XMRPG3L2181)

IRB approval status: Reviewed and approved by Chang Gung Medical Foundation Institutional Review Board; approval IRB No. 202101456A3

Clinicaltrials.gov (or equivalent) listing (if applicable): NCT05206318

Patient Consent: In accordance with ethical guidelines for research involving human participants, informed consent was obtained from all subjects prior to their participation in this study. Specific consent was obtained to publish information and images in an online open-access publication. All participants were informed of the risks and benefits of participation in this study, as well as the potential risks associated with publication of identifying information/images in an online open-access publication. Written consent forms were signed and collected from all participants prior to participation in this study. The consent forms were reviewed and approved by the Institutional Review Board (Chang Gung Medical Foundation Institutional Review Board) prior to the commencement of this study.

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Author Contributions:

All four authors contributed to the randomized control trial. T.L.L. designed the study protocol, oversaw data collection and analysis, and drafted the manuscript. T.F.T contributed to the design of the study protocol, analyzed data, interpreted results, and drafted the manuscript. Y.J.L. played a key role in participant recruitment and retention, collected data, and conducted statistical analysis. C.Y.Ng. designed and implemented the study protocol, administered the intervention, interpreted results, and provided critical feedback on the manuscript. All authors have reviewed and approved the final version of the manuscript.

Data Availability:

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Competing Interests Statement:

Chau Yee Ng serves as a global advisory board member and principal investigator of Scientis Pharma, which may represent a potential conflict of interest in this study. However, Tzu-Li Liu, Tsung-Fu Tsai, and Yi-Jing Lai declare that they have no competing interests related to this study. The trial was supported by Scientis Pharma (XMRPG3L2181), and the authors received no additional financial compensation for their participation. They have no non-financial interests or conflicts other than those stated above.
Scientis Pharma had no role in the design, implementation, analysis, or decision to publish the results of the study.

References


**Tables**

**Table 1.** Clinical characteristics and demographics of the study population

<table>
<thead>
<tr>
<th></th>
<th>Treatment</th>
<th>Placebo</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD), years</td>
<td>41.3 (9.77)</td>
<td>39.8 (10.34)</td>
<td>0.54</td>
</tr>
<tr>
<td>Gender (M/F)</td>
<td>M=2, F=18</td>
<td>M=3, F=17</td>
<td></td>
</tr>
<tr>
<td>Skin Phototypes (III/IV)</td>
<td>8/12</td>
<td>9/11</td>
<td></td>
</tr>
<tr>
<td>Duration of PIH, mean (SD), weeks</td>
<td>9.95 (3.1)</td>
<td>10.05 (3.27)</td>
<td>0.92</td>
</tr>
<tr>
<td>Life quality (DLQI), mean (SD)</td>
<td>6.53 (3.63)</td>
<td>6.1 (3.24)</td>
<td>0.72</td>
</tr>
<tr>
<td>Overall disease severity, mean (SD)</td>
<td>6.45 (1.31)</td>
<td>6.05 (1.32)</td>
<td>0.34</td>
</tr>
<tr>
<td>Pigmentary Intensity of Hyperpigmented Lesions, mean (SD)</td>
<td>4.2 (0.61)</td>
<td>3.9 (0.72)</td>
<td>0.16</td>
</tr>
<tr>
<td>Area of Hyperpigmented Lesions, mean (SD)</td>
<td>3.9(0.97)</td>
<td>3.2(1.39)</td>
<td>0.79</td>
</tr>
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</table>

**Table 2.** Investigator global assessment and patient global assessment
<table>
<thead>
<tr>
<th></th>
<th>Week 4</th>
<th></th>
<th>Week 8</th>
<th></th>
<th>Week 12</th>
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<tbody>
<tr>
<td></td>
<td>Treatment</td>
<td>Placebo</td>
<td>Treatment</td>
<td>Placebo</td>
<td>Treatment</td>
<td>Placebo</td>
</tr>
<tr>
<td>Investigator Global Assessment (IGA)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0= worse</td>
<td>-</td>
<td>20%</td>
<td>-</td>
<td>20%</td>
<td>-</td>
<td>30%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(4/20)</td>
<td></td>
<td>(4/20)</td>
<td></td>
<td>(6/20)</td>
</tr>
<tr>
<td>1= no improvement</td>
<td>35%</td>
<td>60%</td>
<td>20%</td>
<td>55%</td>
<td>-</td>
<td>45%</td>
</tr>
<tr>
<td></td>
<td>(7/20)</td>
<td>(12/20)</td>
<td>(4/20)</td>
<td>(22/20)</td>
<td></td>
<td>(9/20)</td>
</tr>
<tr>
<td>2= slight improvement</td>
<td>60%</td>
<td>20%</td>
<td>55%</td>
<td>25%</td>
<td>53%</td>
<td>25%</td>
</tr>
<tr>
<td>3= marked improvement</td>
<td>5%</td>
<td>-</td>
<td>20%</td>
<td>-</td>
<td>42%</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>(1/20)</td>
<td></td>
<td>(4/20)</td>
<td></td>
<td>(8/19)</td>
<td></td>
</tr>
<tr>
<td>4= almost subsided</td>
<td>-</td>
<td>-</td>
<td>5%</td>
<td>-</td>
<td>5%</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(1/20)</td>
<td></td>
<td>(1/19)</td>
<td></td>
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</table>

| Patient Global Assessment (PGA) |         |            |         |            |         |            |
| 0= worse | -       | 15%        | -       | 10%        | -       | 10%        |
|          |         | (3/20)     |         | (2/20)     |         | (2/20)     |
| 1= no improvement | 55%  | 60%        | 35%    | 40%        | 16%     | 50%        |
|          | (11/20) | (12/20)    | (7/20) | (8/20)     | (3/19)  | (10/20)    |
| 2= slight improvement | 40%  | 25%        | 45%    | 35%        | 63%     | 25%        |
|          | (8/20)  | (5/20)    | (9/20) | (7/20)     | (12/19) | (5/20)     |
| 3= marked improvement | 5%   | -          | 15%    | 15%        | 16%     | 15%        |
|          | (1/20)  |            | (3/20) | (3/20)     | (3/19)  | (3/20)     |
| 4= almost subsided | -     | -          | 5%     | -          | 5%      | -          |
|          |         |            | (1/20) |            | (1/19)  |            |

**Table 3.** Adverse skin reactions in the study population

<table>
<thead>
<tr>
<th></th>
<th>Treatment group (n=20)</th>
<th>Control group (n=20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paresthesia</td>
<td>10% (2/20)</td>
<td>10% (2/20)</td>
</tr>
<tr>
<td>Malodor</td>
<td>15% (3/20)</td>
<td>5% (1/20)</td>
</tr>
<tr>
<td>Peeling skin</td>
<td>10% (2/20)</td>
<td>5% (1/20)</td>
</tr>
<tr>
<td>Acne eruption</td>
<td>5% (1/20)</td>
<td>30% (6/20)</td>
</tr>
<tr>
<td>Dermatitis</td>
<td>15% (3/20)</td>
<td>0% (0/20)</td>
</tr>
</tbody>
</table>
Figure 1

Subjective evaluation results of cysteamine-isobionicamide complex in patients with postinflammatory hyperpigmentation

(A) Overall disease severity (B) Pigmentary intensity score (C) Area of pigmentation(%) (D) Total post-acne hyperpigmentation index (TPHI) (E) Modified MASI score (mMASI)
Figure 2

Mexameter® and skin imaging analysis (VISIA skin analyzer) for the efficacy of cysteamine-isobionicamide complex in patients with postinflammatory hyperpigmentation

(A) Mexameter melanin index; (B) Mexameter erythema index; (C) VISIA skin analysis – surface spots absolute scores; (D) VISIA skin analysis - brown spots absolute scores

Figure 3
Dynamic evaluation of postinflammatory hyperpigmentation lesions with optical coherence tomography

Significant reduction in hyper-reflective melanophages and decreased melanosome capping in the dermal-epidermal junction compared with normal skin. (A) Normal skin; (B) Postinflammatory hyperpigmentation - before treatment (C) Postinflammatory hyperpigmentation - after treatment (week 16)
Clinical images prior to cysteamine-isobionicamide complex treatment and after treatment

A 20 year-old male with post-acne hyperpigmentation for one year (A) before treatment and (B) 16 weeks after treatment; a 40 year-old female with post-laser hyperpigmentation for six months (C) before treatment and (D) 16 weeks after treatment.