

Intralesional tranexamic acid as an effective treatment for repigmentation after depigmentation therapy with monobenzyl ether of hydroquinone: a case report

Nasrin Saki, MD ^{1,2}

Alireza Heiran, MD ³

Elham Sheikhi Ghayur, MD ^{1,2}

1. *Molecular Dermatology Research Center, Shiraz University of Medical Sciences, Shiraz, Iran*
2. *Department of Dermatology, Shiraz University of Medical Sciences, Shiraz, Iran*
3. *Student research committee, Medical School, Shiraz University of Medical Sciences, Shiraz, Iran*

Corresponding Author:

Elham Sheikhi Ghayur, MD

Department of Dermatology, Faghihi hospital, Zand Street, Shiraz, Fars, Iran,

Email: elham.shgh@yahoo.com

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Vitiligo is a pigmentation disorder involving 1% of the population. One of the first line depigmenting agents is monobenzyl ether of hydroquinone (MBEH). Repigmentation following sun exposure; however, can occur after successful treatment with MBEH. This study describes a 54-year-old gentleman who presented with a 7-year history of hyperpigmented lesions on his face following depigmentation therapy with MBEH. The patient was successfully treated with intralesional injections of tranexamic acid.

Keywords: hypopigmentation, vitiligo, tranexamic acid

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INTRODUCTION

Vitiligo as the most common pigmentary disorder with a prevalence of 1% in the population is a multifactorial disease. Predominant etiology is an autoimmune process mediated by T cell infiltration. Cytotoxic T cell-induced melanocyte apoptosis leads to depigmented macules and patches ^{1,2}.

Extensive and treatment-resistant vitiligo has a psychosocial impact on the patient's life; therefore, some patients prefer depigmentation therapy to achieve a uniform appearance of their skin. More than 60% to 80% of body surface area involvement, progressive disease, cosmetically sensitive area involvement such as face and hands, and resistance of vitiligo to standard therapies provoke the patient to use depigmenting agents ¹⁻³.

Monobenzyl ether of hydroquinone (MBEH) is

one of the first line depigmenting agents. Irritant and allergic contact dermatitis in the pigmented area rather than vitiliginous skin, exogenous ochronosis, pruritus, xerosis, erythema, rash, and distant depigmentation are among the side effects of this agent ^{1,3,4}.

One of the problems after treatment with MBEH is the high rate of repigmentation of the successfully depigmented areas induced by sun exposure. Hyperpigmentation of MBEH depigmented areas may be preceded by contact dermatitis.

In some patients, the hyperpigmentation after MBEH depigmentation resolves after a while, while in some other, it becomes darker and needs lightening agents or procedures ⁵.

The use of Q-switched lasers for tanning and recalcitrant pigmentation after depigmentation therapy has been reported in some studies ⁶⁻⁸. Some

studies have reported a statistically significant reduction in melasma score following localized intradermal injections of Tranexamic acid (TA)⁹⁻¹⁵. However, until now, no studies have been conducted on the effect of Tranexamic acid on repigmentation after depigmentation therapy with MBEH.

CASE PRESENTATION

An otherwise healthy middle aged man presented with generalized vitiligo since 7 years ago. After several unsuccessful attempts to restore the pigmentation with topical agents and phototherapy, he began depigmentation therapy for residual pigmented patchy areas on the face with MBEH. After 10 months of using this cream, moderate skin irritation appeared on his face that turned into hyperpigmented patches on his cheeks, eyebrows, and chin. His lesions did not respond to hydroquinone, Kligman's formula and Q-switched laser therapy. (Figure 1)

When he referred to our clinic, intradermal injections of tranexamic acid were carried out for him. After cleansing the face with water and soap, topical xylocaine- prilocaine cream was applied over the hyperpigmented areas for 45 minutes. Tranexamic acid (500mg/ml, Caspian Tamin Co,Iran) was drawn in an 0.5 cc BD syringe without dilution and was injected intradermally

with a 1-cm distance between the injection points. The injections were done every month for three sessions.

Before receiving the first session of treatments, a colorimeter, Dermocatch® (Colorix, Neuchatel, Switzerland), was used to quantitatively measure the baseline pigment value, which was repeated in each follow-up visit.

Pigment value decreased considerably from baseline to the last session of treatment. (Figure 2)

DISCUSSION

Tranexamic acid is a synthetic analog of lysine inhibiting melanin synthesis by interfering with keratinocyte- melanocyte interaction. Tranexamic acid blocks plasminogen binding to keratinocytes, leading to a decreased level of arachidonic acid and prostaglandin as the known stimulators of tyrosinase activity. Some studies have reported a statistically significant reduction in melasma score following localized intradermal injection of TA⁹⁻¹⁵.

To the best of our knowledge, this is the first published report of the effectiveness of TA in the treatment of monobenzene-related hyperpigmentation. Given its efficacy in our patient, ease of use, and low side-effect profile, tranexamic acid may emerge as an agent in the treatment of monobenzene-related hyperpigmentation.

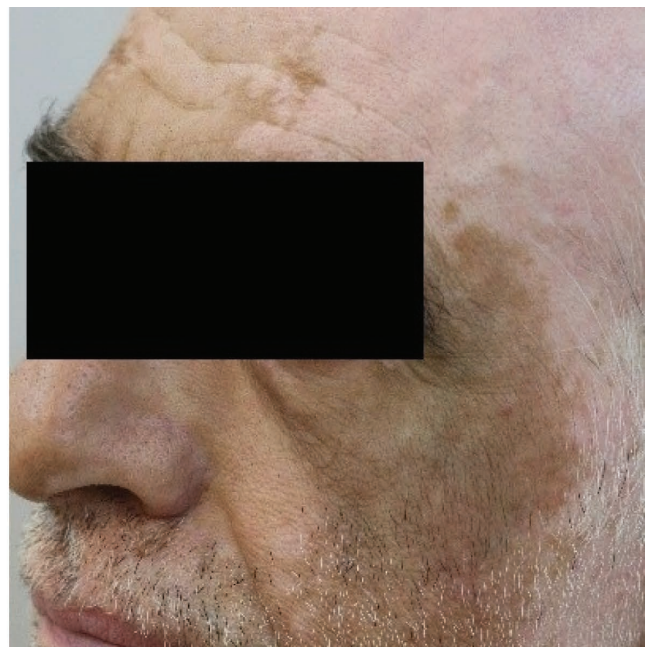
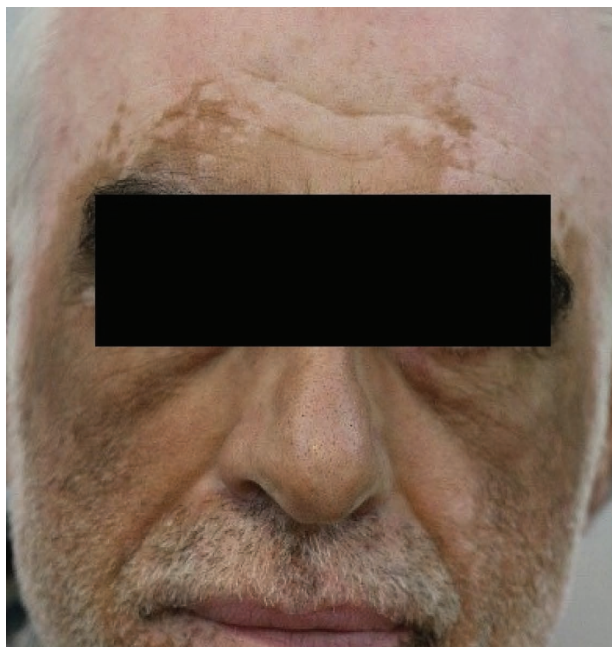


Figure 1. Before treatment with intradermal tranexamic acid injection.

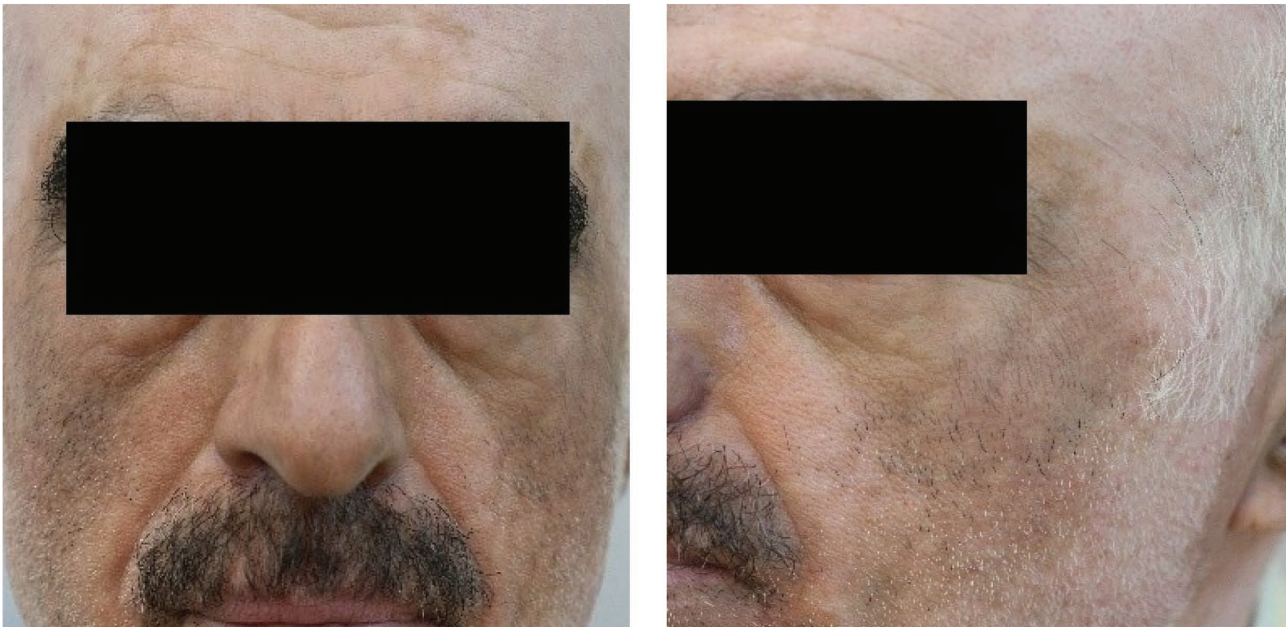


Figure 2. After treatment with intradermal tranexamic acid injection.

CONCLUSION

Tranexamic acid can be effective in the treatment of monobenzone-related hyperpigmentation. It is recommended that a more extensive controlled study be conducted to find its efficacy.

Conflict of Interest: None declared.

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