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Dermabrasion of the Skin: Prevention and/or Treatment of Hyperpigmentation

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ABSTRACT / Dermabrasion of the face for multiple conditions requiring reconstructive surgery is still a valuable tool for the plastic and reconstructive surgeon. This article deals specifically with one of the most important complications, namely, hyperpigmentation.

The specific effect of estrogens on hyperpigmentation and the manner of dealing with it by the use of hydroquinone ointment are discussed. Illustrative case histories and photographs are shown. The reversal of the hyperpigmentation caused by estrogens and treated by hydroquinone ointment are explained, and the conclusion is reached that this management leads to permanent satisfactory results.

KEY WORDS: Dermabrasion, hyperpigmentation, estrogens.

Introduction

Dermabrasion became popular soon after Iversson first introduced it in 1947 as a valuable tool for reconstructive surgery. Conditions treated included revision of traumatic scars and scars due to chicken pox, smallpox, acne vulgaris, and traumatic tattoos; removal of epithelium for overgrafting; preparation of dermal flaps for lymphedema of the lower extremities; and as an ancillary procedure in rhytidectomy of the face.

Then, with the increasing occurrence of complications, as well as unsatisfactory results with acne vulgaris scars, the technique was gradually abandoned by many plastic sur-

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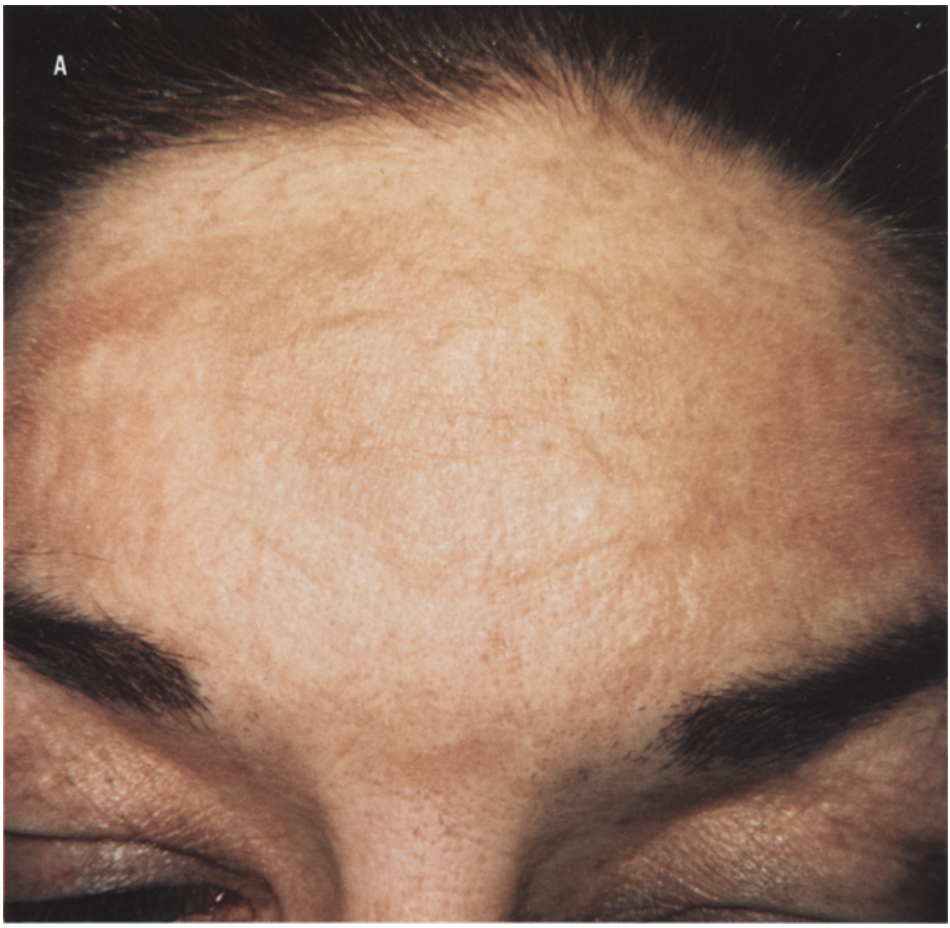

Fig. 1(A): Extensive hyperpigmentation 6 weeks post-dermabrasion.

day, these melanocytes are producing melanin. *In vitro* studies have shown that ultraviolet light injury can cause conversion of dopa without the intervention of tyrosinase, and thus increase the pigment in the injured skin if it has been exposed to ultraviolet light rays at this stage.

A third mechanism of hyperpigmentation is the effects of alpha and beta melanocyte stimulating hormones (MSH) of anterior pituitary origin (5). A current theory is that corticotropin releasing factor (CRF) is produced in the hypothalamus (8), is transmitted to the anterior pituitary via the hypophyseal portal system, and then stimulates the pituitary to produce ACTH and MSH. This CRF factor is a polypeptide (1) first discovered in 1958 and purified in 1959.

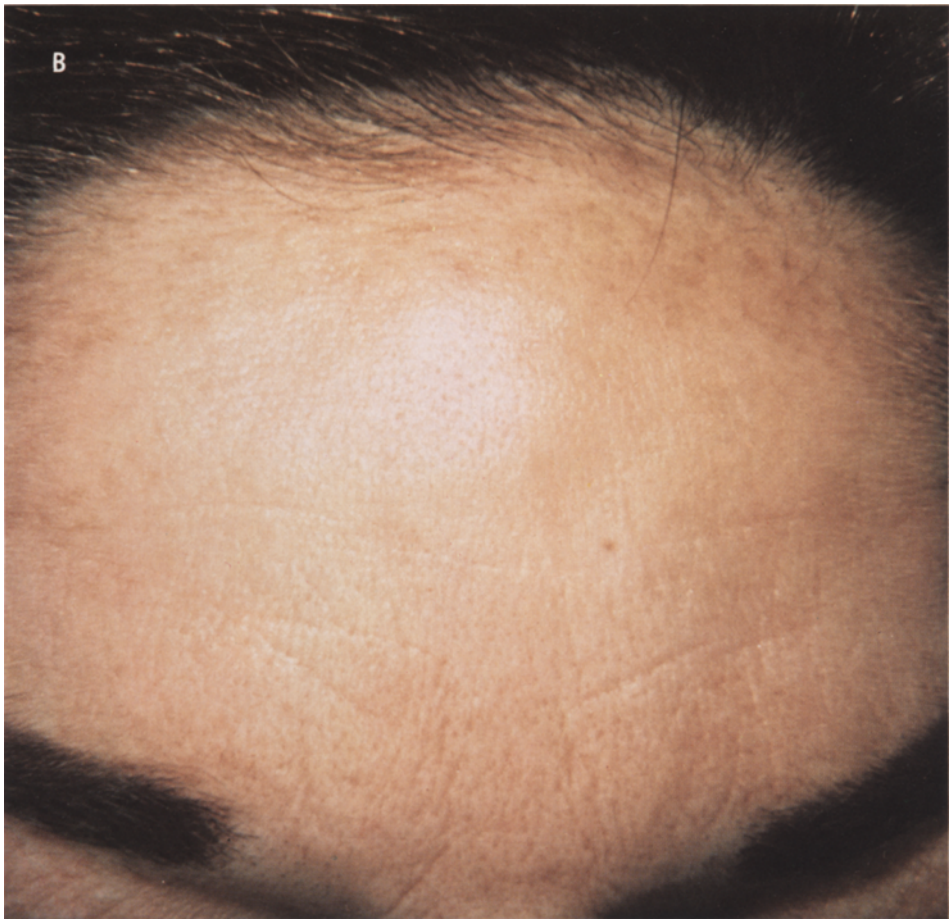

Fig. 1(B): Return to normal 2 months after beginning hydroquinone therapy.

It is of interest to note that ACTH, as well as alpha MSH and beta MSH (more physiologically active), contains the amino acid tyrosine in its chemical structure.
ACTH has two types of secretory control—the feedback inhibition of circulatory cortisol, which in turn acts on the pituitary corticotropin cells. MSH shares in this feedback inhibition by cortisol. An additional site of feedback control may also be in may also be in the hypothalamus.

A fourth mechanism, of special interest in this presentation, is the clinical role played by estrogens. Clinically, estrogens are given therapeutically for supplemental therapy such as in a posthysterectomy syndrome, as a birth control method (oral contraceptives),

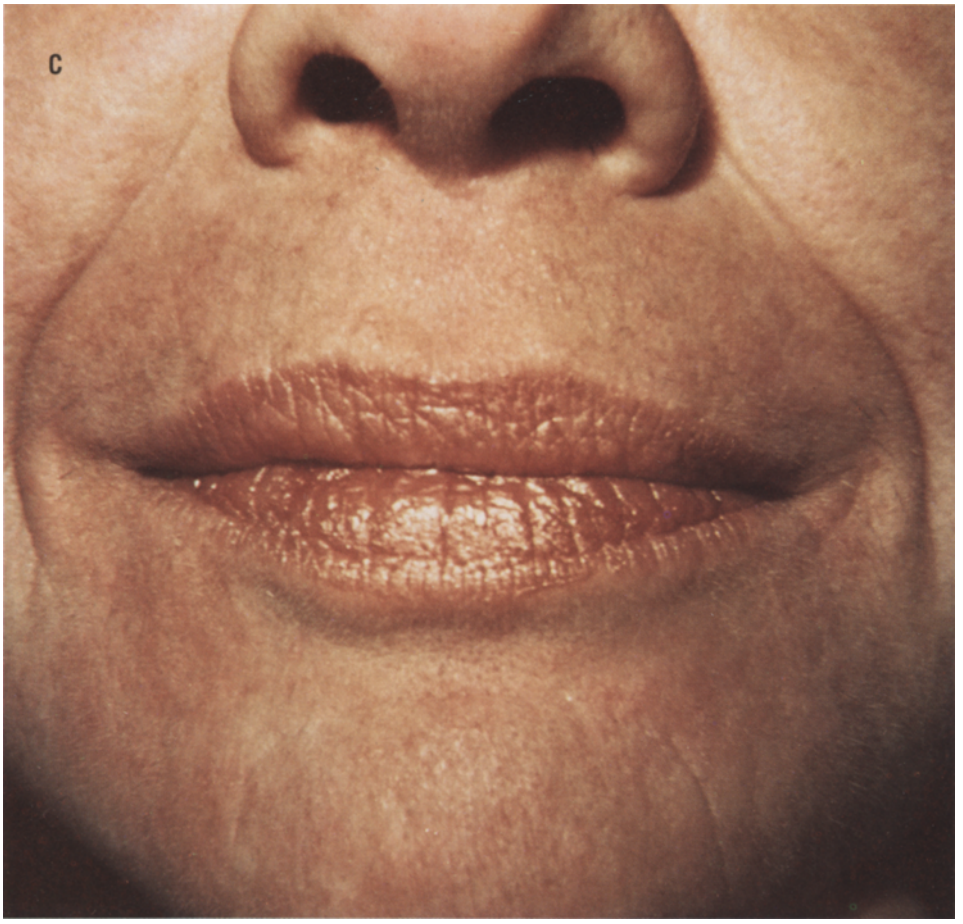

Fig. 1(C): Extensive hyperpigmentation 6 weeks post-dermabrasion.

or in a postmenopausal syndrome. In any case, they can suppress the gonadotropin (follicle stimulating hormone (FSH) and luteinizing hormone (LH) through a feedback mechanism of the pituitary gland. The gonadotropins FSH and LH are glycoproteins with the amino acid tyrosine in their molecules (8). They are regulated by the hypothalamic hormone luteinizing release hormone (LRH) and by feedback inhibition by sex hormones. Estrogens are the most potent inhibitors.

Estrogens are known to produce a pleiotypic response at the cell level by increasing RNA synthesis (8) (Fig. 3). Therefore, this increase of RNA synthesis may in turn increase the amount of melanin formation, which then leads to hyperpigmentation (Figs. 2 and 3).

Specifically relating this theory to the clinical cases presented in this article, I have

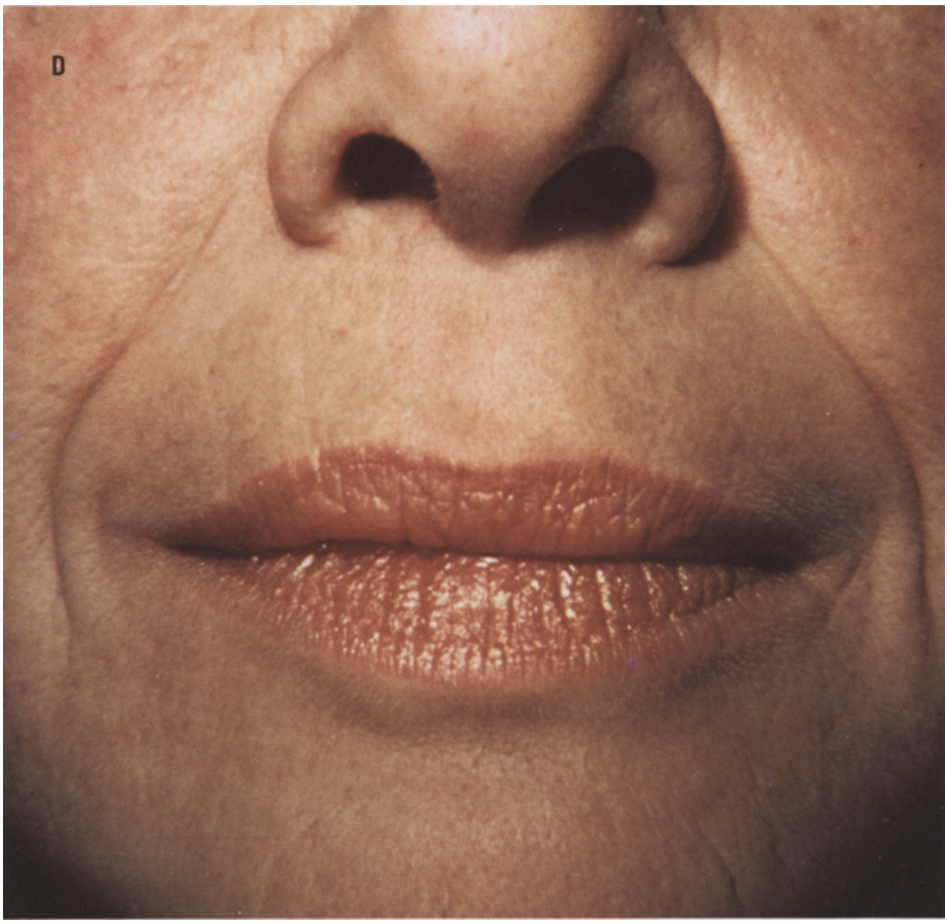

Fig. 1(D): Return to normal 2 months after hydroquinone therapy.

found that the hyperpigmentation seen postoperatively was definitely due to the estrogen factor and nothing else.

Practical application and treatment

The treatment for the hyperpigmentation complication in the three cases presented was specific. In addition to the normal postoperative care, hydroquinone ointment was used twice a day on the hyperpigmented area. After a length of time varying with the individual patient and the degree of hyperpigmentation, the skin color returned to normal. The patient must be cautioned against the possibility of irritation from the hydroquinone ointment; if this occurs, treatment is discontinued for a few days until the irritation disappears.

geons. Some of these complications include hyperpigmentation, edema, erythema, milia, linear scarring, hypertrophic scarring, infection, and marginal demarcation.

The purpose of this article is to reaffirm that dermabrasion is still a valuable procedure in plastic surgery. When the technique is used properly, large scars can be revised and hyperpigmentation can be controlled and prevented. Moreover, infection can be prevented with topical antibiotic ointments, linear scarring can be prevented by improved techniques, and hypertrophic scarring can be greatly avoided by preoperative selection and skin testing of a prospective patient.

In my opinion, hyperpigmentation is one of the most serious complications, and this article is limited to discussion of its prevention and/or treatment.

The problem

Hyperpigmentation of the skin of the face in a postdermabraded patient can occur within the first 3 months of convalescence and is usually due to the effect of ultraviolet rays of the sun. Various substances currently on the market prevent this action of the sun. One of these products, containing dioxybenzone and oxybenzone, has given consistently good results for me in the postdermabraded patient.

In my experience, several patients have developed hyperpigmentation of the dermabraded area of the face in spite of proper attention to postoperative care recommendations as well as the use of a good sunscreen lotion. A review of the case histories of the patients involved revealed a common factor, i.e. taking supplemental estrogens orally, for various medical conditions.

Operative technique

The patient is sedated preoperatively with a standard routine of Nembutal and Vistaril. The area to be dermabraded is carefully prepared with a pHisoHex scrub and draped with sterile drapes. The local anesthetic used is Xylocaine 0.5 with epinephrine 1:200,000 and is injected subdermally. Diamond dust burrs are used and are driven by an electric power unit at 25,000 rpm's. The edges of the area dermabraded are feathered with a sterile pumic stone. The dressing consists of sterile saline sponges, and once hemostasis is achieved a topical ointment of neomycin-steroid is used. Postoperative care includes careful cleansing of the dermabraded area with plain soap and water and use of the neomycin-steroid ointment for 10 to 21 days. Some patients with no history of a previous allergic reaction developed an allergy to neomycin. This was treated successfully by discontinuing the neomycin ointment and continuing only with one of the steroid ointments. Cosmetics can be used after this period. The sunscreen lotion is used thereafter for 3 months.

Case reports

A 42-year-old white female had a revision of a traumatic scar on the left side of the nose, in the radix area, by excision and dermabrasion. Three to six weeks postoperatively she developed hyperpigmentation of the dermabraded area. This was treated successfully by reversing the pigmentation with the use of hydroquinone ointment for 2 months. The mechanism of action of hydroquinone will be explained later.

A 74-year-old white female had a rhytidectomy of the face. Three months later she had a dermabrasion of the lips and chin as an ancillary procedure. Approximately 4 to 6 weeks post-dermabrasion she developed hyperpigmentation of the treated area (Fig. It took 6 months of therapy with hydroquinone ointment to reverse the effects of the Hyperpigmentation and return the skin color to normal.

A 48-year-old white female had a rhytidectomy of the face, followed by a dermabrasion of the lips, chin, and forehead, 2½ months after the first surgical procedure. Approximately 6 weeks postdermabrasion, hyperpigmentation developed in all of the areas treated (Fig. 1 A-D). Two months of therapy with hydroquinone ointment was required to reverse the process completely.

Discussion

An excellent review of skin pigmentation has been published recently (6). Briefly, pigmentation of the skin involves synthesis of melanin in the melanocytes and its transfer to keratinocytes (2) and then to the basal layer of the epidermis.

A biochemical illustration of melanin synthesis can be seen in Figure 2.

Histopathology

Melanin is synthesized in the melanosome, a membrane-bound organelle found in the cytoplasm of the melanocyte (9). The biochemical constituents of melanin include the enzyme tyrosinase and a structural protein (2).

Among Negroes and Orientals, there is a predominance of stages three and four in the maturation of the melanosome with its darker melanoprotein. Caucasians have a predominance of stages one and two with lighter melanoprotein (11).

Hyperpigmentation of the skin through the mechanism of melanogenesis can be produced in several ways. One of the most common ways is through the overexposure to ultraviolet rays of the sun, as occurs when one obtains a suntan. The mechanism here is increased production of stages three and four of the melanosome.

A second way of developing hyperpigmentation is through injury. It has been observed (3,10) that after dermabrasion and laceration of the skin, melanocytes migrate from hair follicles and the healing edge of the wound into the healing skin. By the eighth to tenth

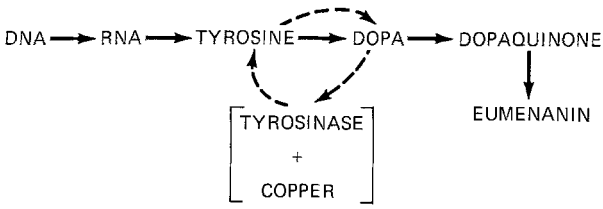

Fig. 2: Simplified illustration of melanin synthesis.

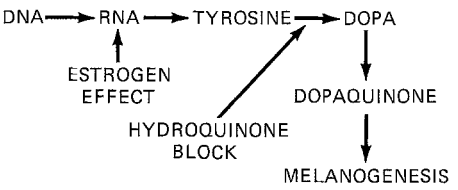

Fig. 3: Pleiotynic response site of estrogen and site of hydroquinone block in reversing pigmentation.

Hydroquinone is known to produce a reversible pigmentation in the skin by interfering with the conversion of tyrosine to dopa and dopa to dopaquinone, partly by inhibition of tyrosinase activity (4).

When a patient gives a preoperative history of taking supplemental estrogens, the medication is discontinued for 1 month and for 3 months postoperatively. Occasional side effects of this abstinence are treated with Bellergal Spacetabs in appropriate dosage.

Since the inception of this pre- and postoperative abstinence of supplemental estrogens in the dermabraded patient, there have been no further recurrences of the hyperpigmentation in numerous cases.

It is also of interest to note that prior to dermabrasion, these patients did not have hyperpigmentation as is sometimes seen in women who take oral estrogens. This hyperpigmentation resembles chloasma or "the mask of pregnancy." When the hyperpigmentation did occur after dermabrasion, the combination of complete abstinence of supplemental estrogens plus the use of the hydroquinone ointment produced a complete and permanent reversal of skin coloring to normal tones. Once the skin color returned to normal and supplemental estrogens were taken again, the hyperpigmentation did not recur. All of these patients were followed postoperatively for at least 1 year.

Summary

Dermabrasion is a useful and valuable surgical procedure for the plastic and reconstructive surgeon and should not be abandoned.

The most common and annoying complication of dermabrasion is hyperpigmentation. The specific effect of supplemental estrogens on hyperpigmentation is discussed in this article.

The specific prevention of hyperpigmentation is discontinuation of supplemental estrogens for 1 month preoperatively and 3 months immediately postoperatively. The specific treatment of hyperpigmentation is complete abstinence from supplemental estrogens for the period of time necessary for the topical application of the hydroquinone ointment to reverse the hyperpigmentation completely. Once the skin color returns to normal, this result is permanent and the supplemental estrogens can be renewed without creating a second episode of hyperpigmentation.

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