CO_2 laser resurfacing can remove freckles and prevent or postpone malignancies in patients with xeroderma pigmentosum

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Background: There is no cure for xeroderma pigmentosum (XP) patients who suffer from persistent freckling and recurrent life-threatening malignancies. We aimed to remove facial lentiginous pigmentations using CO_2 laser resurfacing.

Methods: 5 patients with clinically proven XP living in their third decade were scheduled to be treated with CO_2 laser resurfacing. After tumescent anesthesia, the whole facial skin was treated with 3 UltraPulse® conventional CO_2 ablation passes. The parameters used were 6 mJ, 5 mJ, and 4 mJ for the first to third passes. The mandibular areas were treated with two passes of 4 and 3.2 mJ, while the eye contours were treated with two passes of 3.6 mJ and 3.2 mJ.

Results: The face was edematous and almost free of freckling immediately after resurfacing. The edema persisted for a week. The facial skin oozed within the first three days, followed by crust formation. After a week and after complete shedding of the crusts, smooth and erythematous skin appeared. The erythema persisted for more than two months. The patients were free of malignancy and freckling for up to 16 months follow-up.

Conclusion: CO_2 laser can remove lentiginous pigmentation and prevent or postpone malignancies for a considerable length of time.

Keywords: xeroderma pigmentosum, CO_2 laser, treatment

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INTRODUCTION

Xeroderma pigmentosum (XP) is a rare hereditary and autosomal recessive disorder. Patients with XP are highly sensitive to UV light as they are not able to repair UV-induced DNA damage [1,2]. They die much earlier than the general population [3] because of multiple, repeated, and frequent malignancies of the skin and other organs. It is reported that XP patients can develop skin cancers 10,000 times more than the general population, even before the age of 20 [1,4]. The true incidence of XP is unknown in our area, though there are some statistical data in other countries like the United states, Europe, and Japan [5,6]. Patients with XP are also reported to be immunodeficient [7,8]. This immunodeficiency may be another factor to promote the development of malignancies. There is no cure for the patient suffering from XP. Strict UV protection is our only advice for patients with XP, though they usually do not follow this instruction. This study attempted to remove severe facial lentiginous hyperpigmentation of XP patients using CO_2 laser resurfacing.

PATIENTS AND METHODS

Five clinically proven cases of XP, with the age range of 22 to 29 years, were referred to us within

a period of two years. Three patients suffered from multiple skin malignancies and experienced frequent tumor surgeries. The other two patients were siblings who suffered from profuse sun-exposed lentiginous hyperpigmentation but with no history of malignancy. All patients were intelligent, and as none of them had any neurological problems, the first three can clinically be classified as XPC [6]. The first three patients experienced many tumor surgeries in the past (Figures 1, 3, and 5). The graft surgery results were evident on the nose; this and the hypertrophic scar on the right cheek of patient 1 indicate previous cancer surgeries (Figures 1 and 2). The scar of a huge Z-plasty is also seen on the left cheek of patient number 3 (Figures 5 and 6), who had SCC. There were also multiple scars of BCC surgeries over the same patient's upper labial skin and right maxillary area.

Tumescent anesthesia is the preferred method of anesthesia for CO_2 laser resurfacing because it is long-lasting, and less epinephrine/lidocaine is required for a procedure that may last for more than two hours. After preparation, most areas of the face were anesthetized by tumescent anesthesia. The nerve block was done for the forehead and the nasalis nerves. Some complementary local lidocaine injection was done for the eyelids and tips and sides of the nose to complete the anesthesia before resurfacing. The whole facial skin was treated with three passes of UltraPulse® conventional CO_2 ablation. The CO_2 used was MiXO2 Lasering company Italy. The parameters used were 6 mJ, 5 mJ, and 4 mJ for the first to third passes. The mandibular areas were treated with two passes of 4 and 3.2 mJ, while the eye contours were treated with two passes of 3.6 mJ and 3.2 mJ. After any pass, the coagulated tissues were wiped out using sterile wet gauzes. The procedure may take two hours or more to be completed.

RESULTS

After the procedure, the whole face was quite edematous, and the lips and the eyelids were swollen due to tumescent anesthesia. The faces were almost free of freckling immediately after complete resurfacing. Patients were quite alert with no pain and discomfort. Silver sulfadiazine was applied

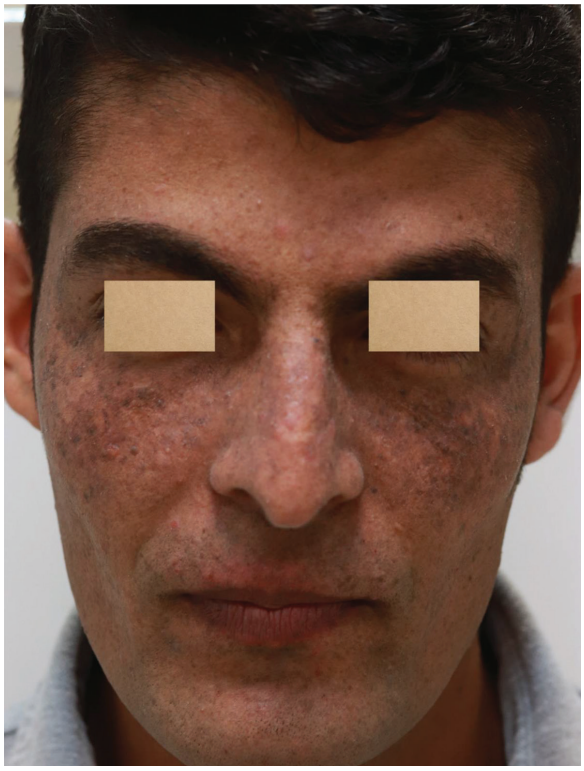

Figure 1. Patient 1, before treatment

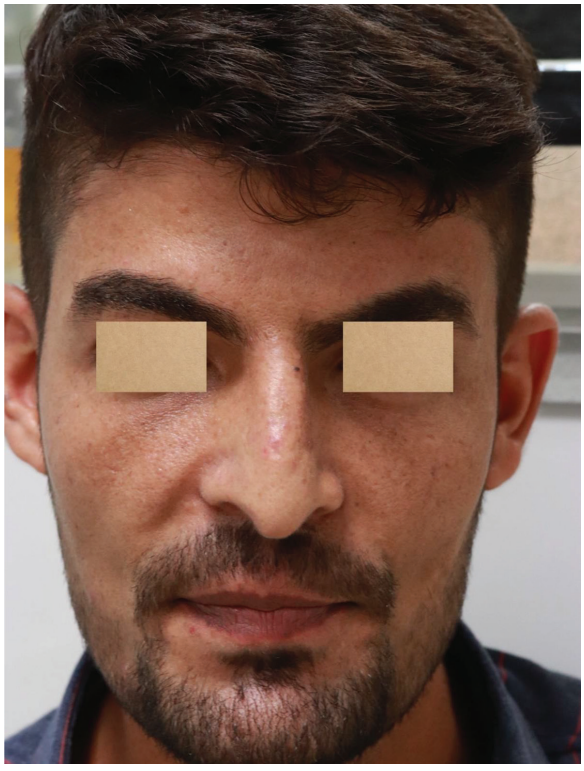

Figure 2. Patient 1, sixteen months after treatment

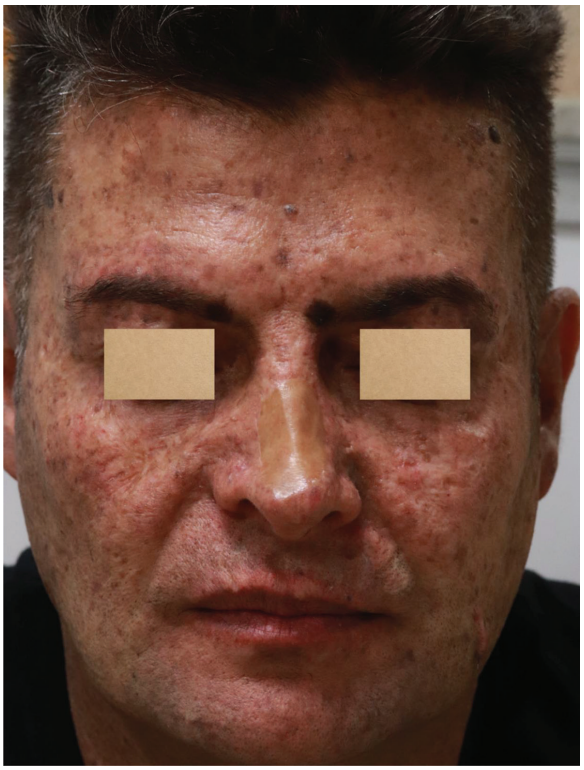

Figure 3. Patient 2, before treatment

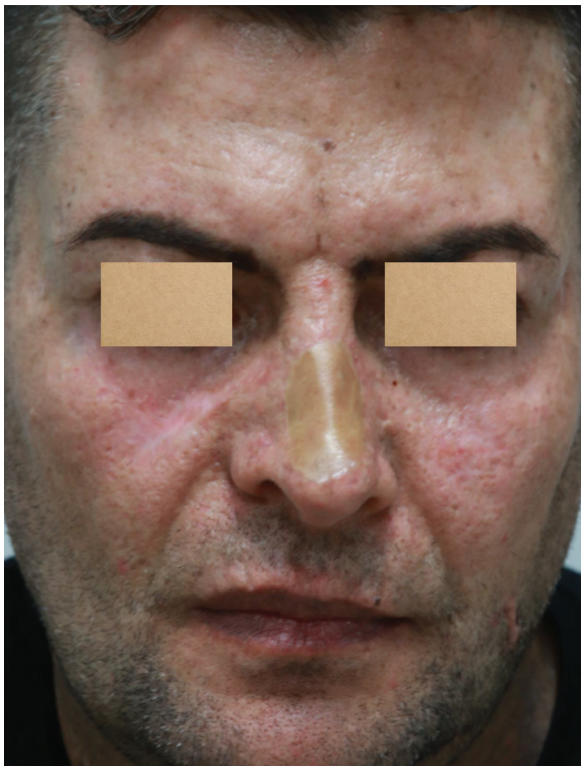

Figure 4. Patient 2, eight months after treatment

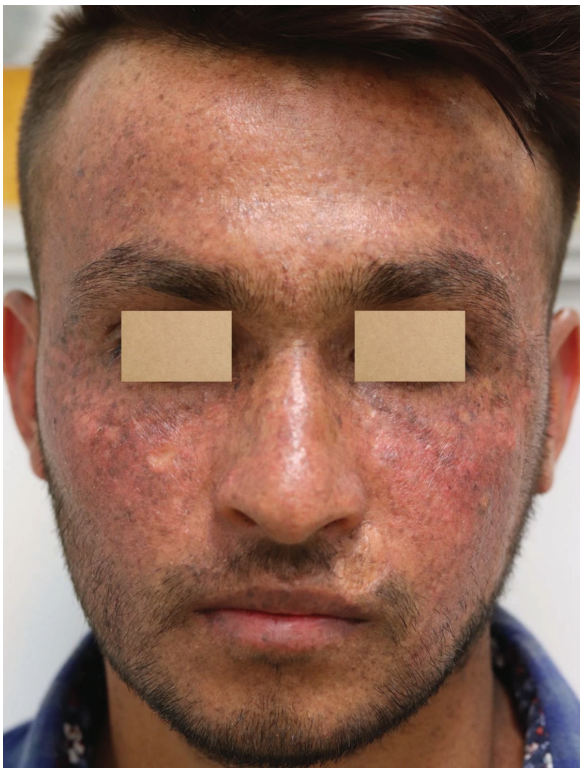

Figure 5. Patient 3, before treatment

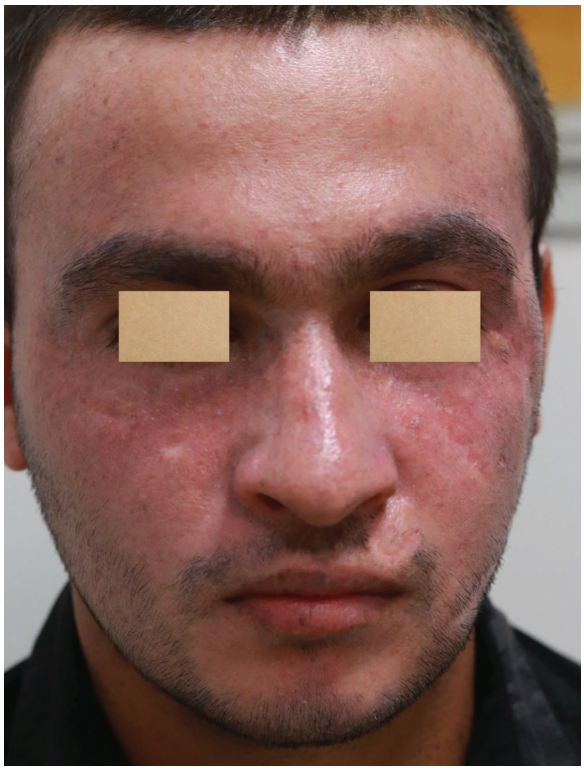

Figure 6. Patient 3, three months after treatment

throughout the face and then covered by sterile gauze and fixed with non-allergenic adhesive tapes. The mouth and eyes were left open. Facial edema may persist for a week due to the inflammatory reaction of laser therapy and slow absorption of tumescent fluid. The facial skin oozed within the first three days, and the patients were advised and instructed to change the dressing twice a day or more with frequent bathing and gentle washing of the face. From the third day on, the face became crusted. The crust started to shed out from day 3 to 6. After the complete shedding of the crusts, smooth, even, and erythematous skin appeared. The erythema was remarkable for the first two months, with gradual fading thereafter. Patients were followed for 12 to 16 months. During the follow-up period, the patients had normal-looking skin with only a few sporadic freckles and no new cancer developed during the course of follow-up (Figures 2, 4, and 6).

DISCUSSION

Freckling, although quite disfiguring, it is not a disease. It may be a reaction of the deeper structures to protect them from further damage. This explanation is not acceptable for the patients, who seek to treat their embarrassing facial pigmentations. The lentiginous pigmentation of these patients cannot be treated or even fainted by topical therapies. Sunscreens and physical protections may slow down the pigmentation, but they cannot block it completely, and the freckling continuously increases with age. The pathology of freckling is located in the epidermis, but xeroderma pigmentosum (XP) has both epidermal and dermal pathology [9]. In XP, it is not only the epidermal cells that have defective DNA repair; the fibroblasts in dermal tissue also lack the ability to repair UV-induced DNA damage [6,10]. This means that in XP, both epidermal and dermal components should be included in any decision or treatment. We surprisingly found that the patients developed no malignancies during the follow-up, which lasted from 12 to 16 months. The first three patients had frequent skin cancers and surgeries more than six times a year before laser therapy.

How can we explain the controversy between the removal of superficial pigmentation as a protective measure and the reduction in the occurrence of malignancies? By CO_2 laser resurfacing, all UV-damaged structures, including the entire epidermis as well as the papillary dermis, were removed and replaced by new, healthy, and non-UV irradiated cells. After removal of the epidermis, the epithelial cells from the deeper portions of hair follicles and sweat glands migrate and cover the dermis. The migrated epithelial cells proliferate and make a new epidermis with healthier and non-UV irradiated cells. Dermal fibroblasts are also reported to have defective DNA repair [6,10]. The replacement of both dermal and epidermal tissues by new and non-UV irradiated cells may be the reason for preventing further cancer cells from developing or postponing this issue. Long-term follow-up and more cases of XP are required for a better conclusion.

CONCLUSION

CO_2 laser can remove lentiginous pigmentation and prevent or postpone malignancies for a considerable length of time.

REFERENCES

1. DiGiovanna JJ, Kraemer KH. Shining a light on xeroderma pigmentosum. J Invest Dermatol. 2012;132(302):785-796.
2. Bensenouci S, Louhibi L, De Vemeuil H, et al. Diagnosis of xeroderma pigmentosum groups A and C by detection of two prevalent mutations in west algerian population: a rapid genotyping tool for the frequent XPC mutation c.1643_1644delTG. Biomed Res Int.; 2016.
3. Kraemer KH, Lee MM, Scotto J. Xeroderma pigmentosum. Cutaneous, ocular, and neurologic abnormalities in 830 published cases. Arch Dermatol. 1987;123(2):241-50.
4. Bowden NA, Beveridge NJ, Ashton KA, et al. Understanding xeroderma pigmentosum complementation groups using gene expression profiling after UV-Light exposure. Int J Mol Sci. 2015;16(7):15985-96.
5. Lehmann AR, McGibbon D, Stefanini M. Xeroderma pigmentosum. Orphanet J Rare Dis. 2011;6:70.
6. Jennifer O. Black. Xeroderma pigmentosum. Head Neck Pathol. 2016;10(2):139–144.
7. Gennery AR, Cant AJ, Jeggo PA. Immunodeficiency associated with DNA repair defects. Clin Exp Immunol. 2000;121(1):1–7.
8. Mariani E, Facchini A, Honorati MC, et al. Immune defects in families and patients with xeroderma pigmentosum and trichothiodystrophy. Clin Exp Immonol. 1992;88(3):376-382.
9. Zheng JF, Mo HY, Wang ZZ. Clinicopathological characteristics of xeroderma pigmentosum associated with keratoacanthoma: a case report and literature review. Int J Exp Med. 2014;7(10):3410–3414.
10. Chacón-Solano E, Leon C, Diaz F, et al. Fibroblast activation and abnormal extracellular matrix remodelling as common hallmarks in three cancer-prone genodermatoses. Br J Dermatol. 2019;181(3):512–522.