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Case Report

The Treatment of Café Au Lait Spot Using Dr. Hoon Hur's Golden Parameter Therapy

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Abstract

Café au lait spot (CALS) is a light or dark brown spot that has various sizes (diameter of 0.5cm-30cm). Solitary or multiple lesions may occur on any parts of body except palms and soles at birth or in infancy. Histopathologically, since nevus cell does not exist in CALS and CALS is not changed into malignant lesion, therefore CALS is a benign pigmented disease [1,2]. That said, treatment is not necessary for CALS except cosmetic concerns [1,2-5]. However, treatment for CALS without side effects such as postinflammatory hyperpigmentation (PIH), scars and recurrences cannot be found in any literature yet. Therefore, the author introduces Dr. Hoon Hur's Golden Parameter Therapy (GPT), using a 1064nm Q-switched Nd:YAG laser without the side effects or recurrences.

Keywords: *Café au lait spot, Dr. Hoon Hur's Golden Parameter Therapy, No side effects, No recurrences*

Introduction

In general, CALS can be classified into two types: non-syndromic solitary CALS and multiple café au lait spots (CALSs) associated with genetic syndrome [1,2]. Clinically, non-syndromic solitary CALS is relatively common and multiple CALSs with genetic syndrome is rare (Table 1) [1,2].

Report of cases

Thirty two Korean patients with CALS (age range : 2-36 years old, mean age : 14.75 years) participated in this study. All patients were clinically diagnosed with CALS. Otherwise, the patients had no significant medical or familial history. After obtaining written informed consent, all of the 32 patients were subjected to 50 treatment sessions of a 1064nm Q-switched Nd:YAG laser (Spectra Laser, Lutronic, South Korea) at a one-week interval with a spot size of 7mm, a fluence of 2.4J/cm^2 and a pulse rate of 10Hz with slowly one pass by sliding-stacking technique to the CALS. Ice packs were immediately applied to the entire face after laser treatment sessions, and patients were instructed to use a broad-spectrum sunscreen daily. Patient photos were obtained on the day of treatment and 4 weeks after the final session. The evaluation was performed by standardized digital photography using a Canon Camera G11(Japan). Patients were asked to report any side effects, pain or discomfort during the treatment. All patients were satisfied with the results, and no any significant side effects, including purpura, postinflammatory hyperpigmentation and scarring except slight pain during the laser treatment.

Table 1: Conditions associated with the development of cafe au lait spots (CALSs).

Conditions associated with the development of cafe au lait spots (CALSs)
Ataxia telangiectasia
Bloom syndrome
Fanconi anaemia
Gaucher's disease
Marfan syndrome
McCune–Albright syndrome
Multiple endocrine neoplasia type 1
Neurofibromatosis type 1(NF1)
Neurofibromatosis type 1-like syndrome
Noonan syndrome
Peutz-Jeghers syndrome
Silver–Russell syndrome
Tuberous sclerosis
Von Hippel–Lindau disease

Table 2: Indication of Dr. Hoon Hur's Golden Parameter therapy.

Indication of Dr. Hoon Hur's Golden Parameter therapy
Cafe au lait spot
Agminated lentiginosis
Becker's nevus
Nevus spilus
Linear and whored nevoid hypermelanosis
Incontinentia pigmenti
Acquired bilateral nevus of Ota-like macules(ABNOM)
Melasma
PIH
Erythema dyschronicum perstans
Pityriasis rotunda
pigmented contact dermatitis(Rielh's melanosis)
Dowling-Degos disease
Onychomycosis
Verruca plana
Removal of vellus hair

Discussion

In non-syndromic solitary CALS, NF1 somatic mutations do not exist in melanocytes and keratinocytes in the epidermis and fibroblasts in the dermis. The epidermal melanocytes have no

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macromelanosomes. In addition, the NF1 somatic mutations cannot be found in the surrounding normal skin [6,7]. On the other hand, with regard to multiple CALSs with neurofibromatosis type1, NF1 somatic mutations can be seen in the melanocytes, keratinocytes in the epidermis and fibroblasts in the dermis. Unlike solitary CALS, the epidermal melanocytes have marcromelanosomes and NF1 somatic mutations can be found in the surrounding normal skins [6,7].

In non-syndromic solitary CALS, the expression of endothelin-1 is increased in the keratinocytes and the expression of stem cell factor (SCF) is increased in the fibroblasts compared to those of normal skins. The increased expression of endothelin-1 and SCF activates the melanocytes and increases melanin synthesis, therefore causing CALS [6-9].

In multiple CALS with neurofibromatosis type1 and normal skins of neurofibromatosis, the expression of endothelin-1 in the keratinocytes is more highly increased than in non-syndromic solitary CALS [6-9]. The expression of SCF in the fibroblasts is highly increased as well. Thus, this further increased expression of endothelin-1 and SCF activates the melanocytes and increases melanin synthesis, which may cause multiple CALS [6-9]. However, basic fibroblast growth factor (bFGF) that may provoke melasma and PIH [6-9]. Given that bFGF in the keratinocytes of CALS is not increased, bFGF may not cause CALS. Following up a giant solitary CALS for 20-30 years, NF1 somatic mutations have not been observed in melanocytes and keratinocytes in the epidermis and fibroblasts in the dermis [1]. Moreover, the giant solitary CALS may not be developed into neurofibromatosis [1,6,7]. On the other hand, when more than 6 multiple CALSs without neurofibromatosis exist, NF1 somatic mutations may either occur or not in the epidermal melanocytes. More than 6 multiple CALSs without neurofibromatosis may eventually develop into neurofibromatosis [1,6,7].

A histopathological finding of CALS shows moderate elongation of epidermal ridge, increased number of melanocytes, increased melanin deposition in the epidermis, and a few melanophages in the upper dermis [2]. According to the histopathological finding [2],a Golden Parameter with a 1064nm Q-switched Nd:YAG laser may destroy melanocytes without keratinocyte damage, and the products of damaged melanocytes will be removed through transepidermal elimination [3,4,10]. Then, basement membrane breaks down and epidermal melanocytes secrete melanin into the upper dermis. Thus, after phagocytizing this melanin, dermal melanophage is eliminated through dermal lymphatics [3,4,10]. By apoptotic melanocytic cell death and homeostasis, normal melanocytes of hair outer root sheath migrate to basal layer and abnormal melanocytes are displaced into normal melanocytes in the basal layer. It is the author's theory that this laser treatment can be performed complete clearance of the CALS without side effects or recurrences.

There are possible four reasons of failure when treating CALS with traditional laser therapy. First, 532nm of Q- Swithched Nd:YAG laser, 694nm of Ruby laser, 755nm of alexandrite, and 515-755nm of IPL absorb much more melanin compared to 1064nm of Q-Swithched Nd:YAG laser. Thus, fluence that destroys epidermal melanocytes injuries the surrounding keratinocytes, and the damaged keratinocytes secrete endothelin-1, α-MSH, ACTH, prostaglandin (PGE2,PGF2α) and nitric oxide. These cytokines activate melanocytes and increase melanin synthesis, therefore causing PIH and worsening CALS [6-9].

Second, the keratinocytes secrete urokinase type plasminogen activator (U-type PA) that converts plasminogen into plasmin. This plasmin stimulates the keratinocytes, which secrete bFGF that activates the melanocytes and increase melanin synthesis, therefore causing PIH and worsening CALS. The author believes that bFGF, a major cytokine, causes PIH and melasma because tranexamic acid (500mg/day for 90 days), which suppresses U-type PA and plasmin, improves melasma and PIH [6-9].

Third, as the traditional laser therapy causes petechiae and crusts, irradiated fluence may damage fibroblasts, mast cells, lymphocytes, macrophages, and vascular endothelium. Then, fibroblasts mainly secrete SCF and mast cells, in which the activation of arachidonic metabolites occurs, secrete histamine. The arachidonic metabolites and histamine cause inflammatory reaction that activates melanocytes and increases melanin synthesis, eventually causing PIH and worsening CALS [6-9].

Lastly, free radical oxygen and peroxide from the keratinocytes also activates melanocytes and increase melanin synthesis, eventually causing PIH and worsening CALS [6-9]. To solve the side effects such as PIH and worsening CALS of the traditional laser therapy, the author devised Dr. Hoon Hur's Golden Parameter Therapy (GPT). The author would like to name this therapy "Golden Parameter Therapy" because the author believes that it can improve various skin diseases (Table 2).

Dr. Hoon Hur's GPT can be summarized as follows

First, the mechanism of GPT is to minimize the epidermal damage and destroy melanosomes in the epidermal melanocytes, which are changed into ghost cells due to the loss of function. Then, weekly GPT destroys melanocytes completely, accelerates apoptotic melanocyte cell death, thus removing abnormal epidermal melanocytes [3,4,10].

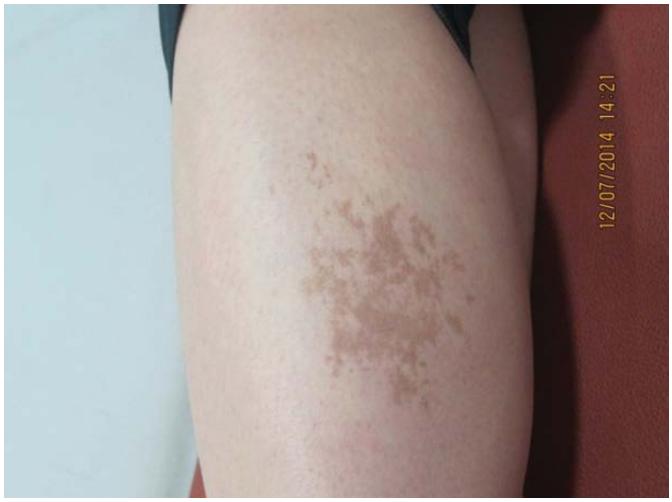

Figure 1: A brown reticulate patch on the left lower leg (before treatment:7/12/2014)

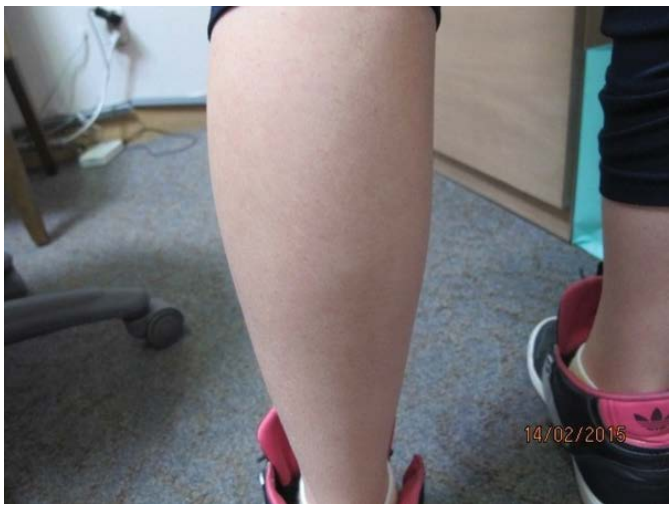

Figure 2: A complete clearance of café au lait spot (after treatment with Golden Parameter:2/14/2015)

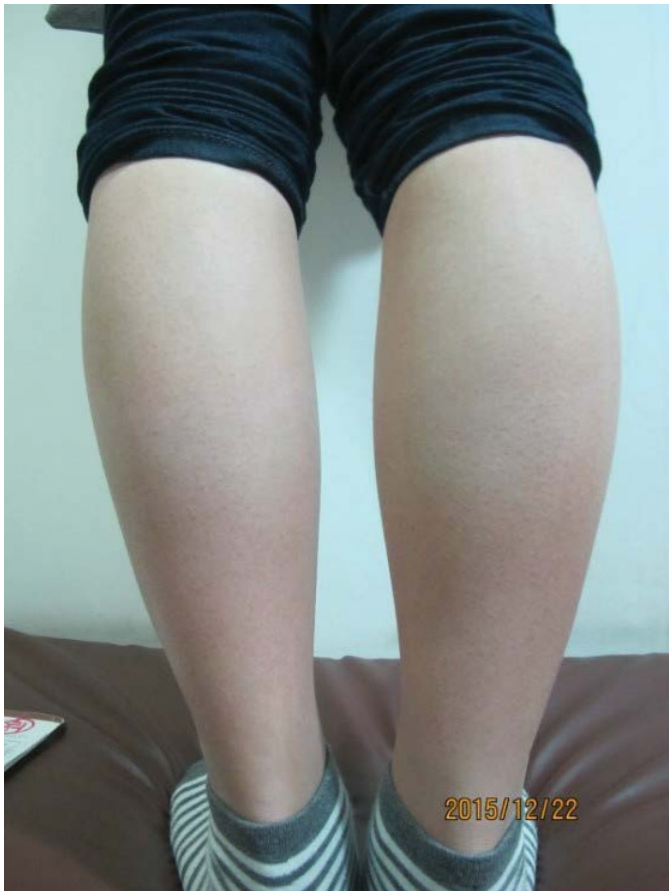

Figure 3: There is no recurrence at 10 months follow-up (12/22/2015)

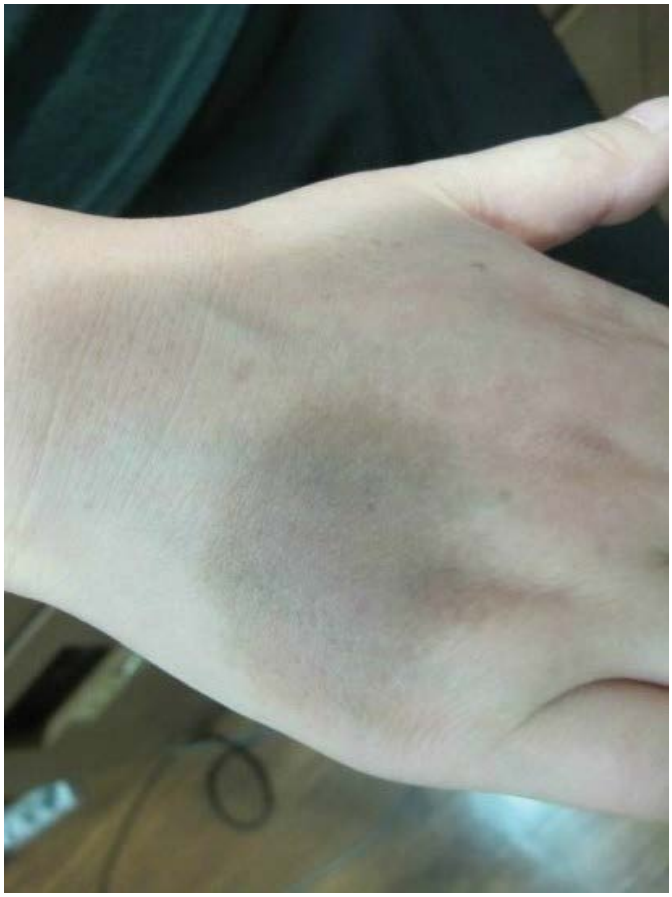

Figure 4: A solitary round brown patch on the right hand (before treatment:2/15/2014)

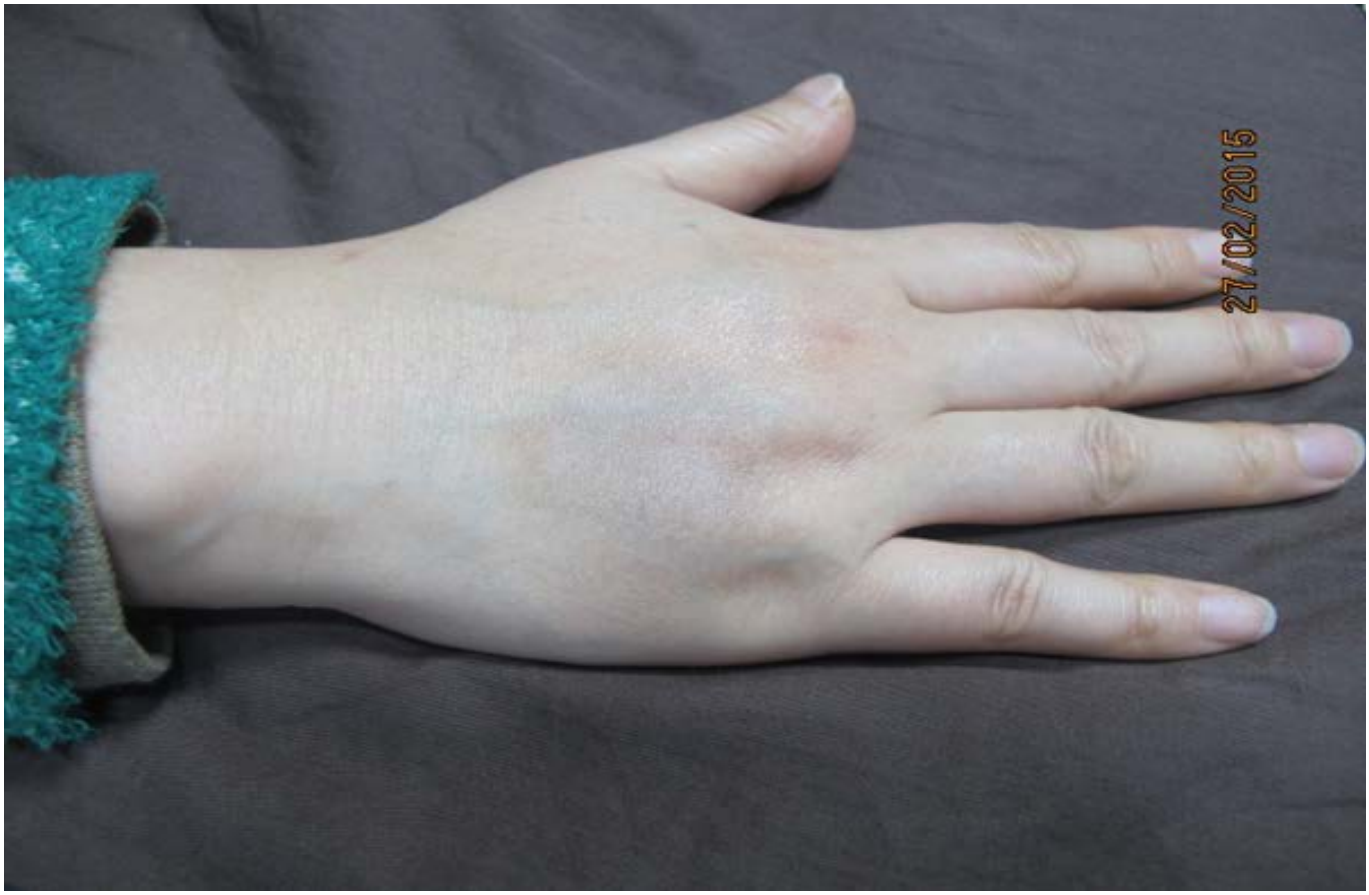

Figure 5: A complete clearance of café au lait spot on the right hand (after treatment with Golden Parameter:2/27/2015)

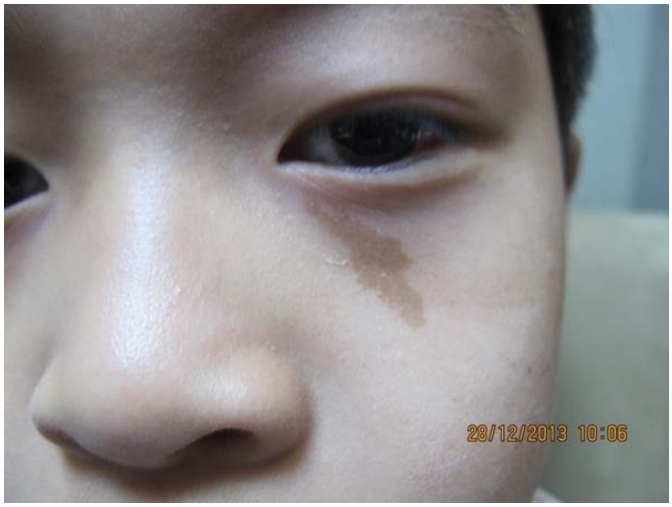

Figure 6: A solitary brown band-like patch on the left infraorbital area (before treatment:12/28/2013)

Figure 7: A complete clearance of café au lait spot on the left infraorbital area (after treatment with Golden Parameter:7/12/2014)

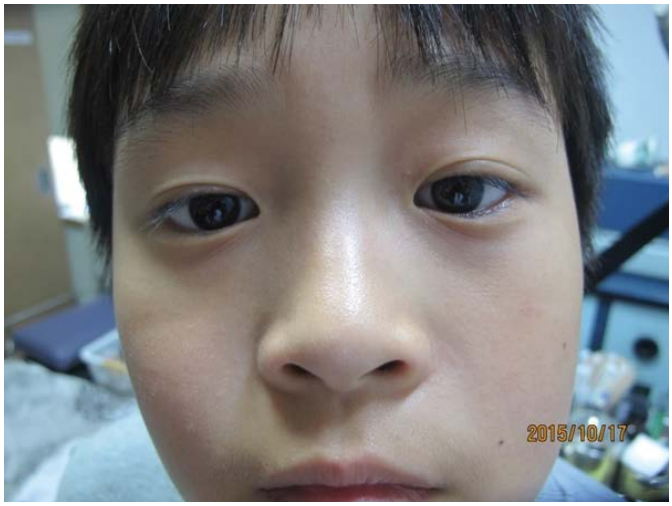

Figure 8: There is no recurrence at 15 months follow-up (10/17/2015)

Eventually, abnormal melanocytes are displaced into normal melanocytes which migrate from hair outer root sheath [3,4,10]. In conclusion, complete clearance of CALS without side effects and recurrences can be achieved.

Second, patients with CALS are treated with 30-50 sessions of a 1064nm Q-switched Nd:YAG laser (Spectra Laser, Lutronic, South Korea) at a one-week interval with a spot size of 7mm, a fluence of 2.4J/cm^2 and a pulse rate of 10Hz with slowly one pass by sliding-stacking technique to the CALS. One pass of fluence by sliding-stacking technique is very important to minimize the epidermal damage. If two passes of fluence by sliding-stacking technique were performed, the epidermal damages might have occurred and the damaged keratinocytes might have secreted cytokines such as endothelin-1, α-MSH, ACTH, bFGF, prostaglandin (PGE2,PGF2 α) and nitric oxide which could cause PIH and worsen CALS [6-9].

Third, in case of CALS on face, 30 GPT sessions are performed once a week. Small lesion with a diameter of less than 2cm requires 30 treatment sessions on a weekly basis. And large lesion with a diameter of larger than 3cm needs 50 treatment sessions once a week. In case of CALS on other parts of body (arms, legs, and torso), more than 50 GPT sessions should be performed once a week regardless of the size of lesion. The advantage of GPT is that it minimizes the epidermal damage without petachiae and crusts so this therapy does not cause PIH. But GPT requires a long-term treatment for one year. In this study, 32 patients with a solitary CALS (Figure 1,4 and 6) were treated with GPT of a 1064nm Q-switched Nd:YAG laser. A total of 32 patients with a solitary CALS was achieved complete clearance of CALS (Figure 2,5 and 7). There is no recurrences at 12 months follow-up (Figure 3 and 8).

Conclusion

The parameter for each GPT was a spot size of 7mm, a fluence of 2.4J/cm^2 with one pass and a pulse rate of 10Hz. This parameter does not provoke side effects such as petechiae, crusts, pain, and PIH during the laser treatment. Therefore, the author suggests GPT achieves complete clearance of CALS with a very safe and effective profile.

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