

Melasma in Men

Helena Kristlova*

Department of Dermatovenerology, Hospital Ceske Budejovice, Czech Republic

Abstract

Melasma (synonymum chloasma) is an acquired hypermelanosis found typically in sun-exposed areas. Melasma affects predominantly women (90%), however it can be a serious cosmetic problem for men, too. This article describes melasma in men, its pathogenesis and treatment options.

Introduction

Melasma is in origin from Greek word melas-black. A synonymum chloasma is used as well, but chloazein, derived also from Greek, means green, so we prefer the term melasma, because hypermelanosis is more brown than green. Melasma is acquired hyperpigmentation of sun-exposed areas, which predominantly affects women, but occasionally can be seen in men. Various endogenous and exogenous factor causing melasma were studied, however, the exact cause of melasma has not been fully clarified.

Clinical Presentation

Melasma is typical for sun-exposed areas. Here, symmetric light or dark brown punctate or confluent macules are found, usually sharply delimited, especially on the forehead, cheeks, upper lip and chin. We can distinguish 3 types of melasma: centro-facial type (involves forehead, cheeks, nose, upper lip and chin), mandibular type (on mandibular ramus) and symmetrical malar type (localised to cheeks and nose) [1]. In men, malar pattern is more common, whereas in women, the centrofacial type is mostly seen [1,2]. Rare variant of melasma can occur on the arms, too [3]. Wood lamp can clinically divide depth of pigmentation: in epidermal type of melasma can be seen highlighting of proliferated pigment (50% of cases in Sarkar et al. study), compared to dermal type (5% in Sarkar st.), where not. In mixed type of melasma (45% of cases in Sarkar study) only partial highlighting of pigmentation is present [2,4]. Clinically, dermal type of melasma appears slightly bluish due to Tyndall effect [3]. The severity of disease can be objectified by Melanin index (MI) values measured by special instrument, Melanin Area and Severity Index (MASI) counting areas and the density of involvement, and patient self-evaluation [5].

Epidemiology

Melasma can affect patient of any race, but is more common in darker phototypes (for example Hispanic, Indian, Guatemalan), most common in individuals with Fitzpatrick skin types IV-VI [1,2,6]. Vazquez et al. described women to men ratio of melasma 9:1, with identical histological picture [7]. However, Sarkar et al screened 200 patients with melasma where men represented 20.5% [2]. Another paper described results of 3 studies evaluating prevalence of melasma in adult male Latino migrant workers to be 36.0%, 7.4% and 14% [6]. Average age of affected men is 33.5 years with a duration of 3.5 years by Sarkar [2], however can be found also in older men and for longer period by other authors [6,7]. It can be a source of embarrassment in men, because of its unsightly appearance and the social stigma of being categorized as a disease in pregnant women [1]. In pregnant women, the estimated prevalence of melasma is up to 70% [1]. Melasma occurs rarely before puberty, most commonly begins in reproductive years. Melasma has a significant emotional and psychological influence on affected patients [8]. Impact on the quality of life of patients with melasma can be objectified by the Melasma Quality of Life Scale (MELASQOL) [3,9] and/or by Dermatology Life Quality Index (DLQI)

[6]. It was proved, that patients having higher DLQI score indicate poorer Quality of Life (QOL) [6].

Pathophysiology

Pathophysiology of melasma is not fully clarified. Recent research tends to indicate to melasma not as an acute inflammatory disease, but as the result of the accumulation of chronic inflammation [5,10,11]. Melasma usually occurs in women after pregnancy or using hormonal contraception. Higher level of estrogen receptor expression were found in affected skin in women on oral contraceptive pills [3,12]. Effect of sun for hypermelanosis is indisputable. Guinot et col. described in a study in 197 Tunisian patients sun exposure as triggering factor in 51% and aggravating factor in 84% of cases [8]. UV radiation increases the level of dermal stem factor and alpha-melanocyte stimulating hormone in the skin, thus increased melanocytosis (higher number of melanocytes) and melanogenesis (excess production of melanin) occurs [3,9]. Among other exogenous factors belong certain cosmetics, especially etheric oils, photosensitising medications (hydantoin, chlorpromazin, isotretinoin) and oral contraceptive pills. Melasma was described as a side effect of diethylstilbestrol in prostate cancer therapy [14]. Also, finasteride-induced melasma in a treatment of men androgenetic alopecia was published by S. Famenini et al. colleagues [14]. In endogenous factors female hormonal activity, pregnancy, mild ovarian or thyroid dysfunction as well as tumors producing estrogens can be count. Estrogens originate in ovaria, placentae and in little amount also in testes and adrenal glands. In skin, estrogens affect keratinocyte differentiation, as well as sebaceous glands functions, pigmentation and kolagen metabolism. In a case of hyperestrogenism naevi aranei, teleangiectasias, palmar erythema, melasma and darkening of genitalia, areolas and naevi can be found [15].

In men the cause of melasma is not fully understood. Sialy and colleagues described statistically significant higher levels of luteinizing hormone and lower levels of testosterone in 15 men with melasma. The authors applied the term *subtle testicular resistance* as a potential reason of melasma in men [14]. In laboratory results, significantly higher level of luteinizing hormone and lower level of testosterone were found in melasma cases [14]. Sarkar et al described a common use of mustard oil in Indian men as a potential photosensitising modality for melasma uprise [2]. Among others causative factors are considered nutritional

*Corresponding author: Helena Kristlova, Department of Dermatovenerology, Hospital Ceske Budejovice, Bozeny Nemcove 54, 37001 Ceske Budejovice, Czech Republic, Tel: +420 608 824 723; E-mail: helena.kristlova@yahoo.com

Received: October 08, 2014; Accepted: October 16, 2014; Published: October 18, 2014

Citation: Kristlova H (2014) Melasma in Men. Pigmentary Disorders S1:004. doi: 10.4172/JPD.S1-004

Copyright: © 2014 Kristlova H. The terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

disorder, parasitic infestation and hepatic disorder, although there is no solid supporting evidence [2].

Genetic predisposition can be considerable in the etiology of melasma [2]. In a small trial of 5 men with melasma treated with dual combination, genetic predisposition seemed to correlate with the disease development [16]. Vazquez et al. reported 70% of men in their study had a family history of melasma in their first- or second-degree relatives, although none of them admit melasma in their fathers [7].

Histology

Melasma can be diagnosed just clinically, however, in case of need, histological description can be helpful. Histological finding is similar in men and women [1]. However, the histopathological characteristics of male melasma are not yet clearly defined [17]. Histological features include solar elastosis, flattening of rete ridges and mild inflammatory cell infiltration [5]. Melanin amount is increased in epidermis, dermis or both of these. In epidermis, melanin is found in keratinocytes in basal and suprabasal layer. Number of melanocytes is not raised, but they are larger, with greater melanosomes and more dendritic, thus more active. Dermal melanin arises in superficial and middle dermis within macrophages, often in aggregation in the near of small dilated vessels. Epidermal melanization and presence of melanophages in the papillary dermis can be seen in Haematoxylin-Eosin and Fontana-Masson staining [2]. No evidence of basal layer degeneration was found [2]. Significantly increased expression of stem cell factor (SCF) around dermal fibroblast in dermis and its c-kit receptor in the basal layer of epidermis was found in lesional skin compared to nonlesional [12,17]. Moreover, the expression of SCF was increased in men compared to women [12]. These results suggest that chronic UV radiation associated with signalling of paracrine cytokines would play an important role in the mechanism of melasma in male patients [1].

In differential diagnosis should be mentioned post-inflammatory hyperpigmentation, pigmented contact dermatitis, lichen planus pigmentosus, frictional melanosis, facial acanthosis nigricans, freckles, solar lentigo, nevus of Ota, nevus of Hori and Becker's melanosis [1]. Among others diagnoses melanodermitis toxica – Riehl (syn. Riehl's melanosis – in patients working with antracen derivatives, or associated with cosmetic use, may be synonymous with pigmented allergic contact dermatitis), Berloque dermatitis (photoirritant reaction to bergapentin) and poikiloderma of Civatte (reddish-brown pigmentation, atrophy and teleangiectasia affecting lateral aspect of cheeks and neck, more often in women) can be count [18].

Treatment and Management

Men have a tendency to avoid complex and time-consuming regimens in melasma treatment. Unfortunately, there is no quick-fix for melasma to date [1]. In addition, it is believed that each type of melasma (epidermal, dermal or mixed) should have its most appropriate treatment [5,19].

Strict avoidance to sunlight is essential, using hat is recommended, together with daily applications of broad spectrum sunscreen, rather physical blockers with titanium dioxide or zinc oxide. Nonetheless, compliance can be a problem regarding sunscreen use in men [1]. No estrogens exposure is advised. Some types of melasma can fade during a time, but others may be permanent. Here a topical therapy can help. Over-correction and feminization should be avoided in the approach to men melasma [1]. Generally, men prefer using a solution rather than cream or ointment [16]. Subsequently, stinging sensation due to high ethanol content can be experienced [1].

Two categories of therapies for melasma are known: biologic modulators and locally destructive agents [3]. Biologic modulators, also known as topical hypopigmenting agents, can be divided into biochemical and physiologic modulators [3]. Biochemical modulators (for example hydroquinone, azelaic acid, kojic acid and ascorbic acid) inhibit tyrosinase and thus significantly reduce melanin synthesis [3]. Physiologic modulators (including tretinoin and glycolic acid) interfere with normal melanin distribution.

Another division of melasma treatment can be into tyrosinase inhibitors, those which inhibit transfer of melanosomes, melanocytotoxic agents and antioxidants [20].

Criterion standard in melasma therapy is topical hydroquinone (HQ) in concentration 2-4%. HQ is hydroxyphenolic chemical which inhibits tyrosinase and thus decreased a production of melanin [9]. With higher concentration of HQ better effect can be seen, but also adverse effects can arise: irritation, phototoxic reaction with secondary postinflammatory hyperpigmentation, irreversible exogenous ochronosis.

Tretinoin (trans-retinoid-acid) is used in monotherapy or also in combination with HQ and/or corticosteroids. Tretinoin inhibits tyrosinase by decreasing melanin synthesis affect melanin transfer to keratinocytes, moreover increases the penetration of bleaching agents and accelerates epidermal turnover [16].

Triple-combination cream (TCC) containing HQ 4%, tretinoin 0.05%, and fluocinolone acetonide 0.01% was successfully applied in some patients series [9]. However, due to a risk of facial atrophy has this TCC cream limited use to no more than twice daily for 6 month [9].

Arbutin is derived from hydroquinone and is felt to have fewer adverse effects than HQ [9].

Ellagic acid is an antioxidant and also a substrate of tyrosinase, thus a possible inhibitory effect on melanogenesis can be suggested [9].

Rucinol – is a derivative from resorcinol and inhibits tyrosinase-related protein 1 (TRP1) and tyrosinase [9].

Oligopeptides as a new class of tyrosinase inhibitors may also be beneficial alternatives to hydroquinone, due to their minimal cytotoxicity and improved inhibitory efficacy [3].

Azelaic acid (in 20% concentration cream-formula) influence melanocytes by reducing DNA-synthesis and mitochondrial cellular energy products inhibition. It is a naturally occurring dicarboxylic acid synthesised by the yeast *Mallessezia furfur* and is associated with the hypopigmented macules seen in tinea versicolor [9]. Azelaic acid targets only hyperactive melanocytes, therefore will not fade skin with normally functioning melanocytes. Benefit of azelaic acid is in adverse effect, where skin irritation can be found, but there is no risk of phototoxic or photoallergic reactions.

A successful treatment with combination of Mequinol 2% and Tretinoin 0.01% in a topical solution was described in 4 from 5 cases of melasma in men. Four patients achieved complete clearance of melasma in 12 weeks, one patient showed moderate improvement [17]. Mequinol is a phenolic derivative of hydroquinone and is used as potent bleaching agent. However, the mechanism of action is largely unknown [17]. Mequinol, as a phenolic derivative also has potential to cause exogenous ochronosis, though it has not been reported [17].

Among newer and experimental agents for melasma treatment were

used aloesin, flavanoids, ellagic acid, gentisic acid, hydroxycoumarins, natural derived botanical extracts, rucinol, soy, silymarin, alphalipoic acid, diolic acid, green tea, octadecenedioic acid, orchid extracts, mequinol, linoleic acid, magnolinan, tranexamic acid and cinnamic acid [20].

Superficial chemical peels and laser therapy may be effective, but also can cause subsequent hyperpigmentation. This kind of additional treatment induced localized tissue destruction and after, the effect of hypopigmentation can be seen [3]. Among superficial chemical peels modality alpha and beta hydroxy peels like glycolic, salicylic, Jessner's, TCA and azelaic acid have been studied for their therapeutic benefits in melasma treatment [20]. Deeper peels are generally inadvisable in melasma treatment, for their possible complications (hypo- and hyperpigmentation, scarring and keloid formation, secondary infection etc.) [20]. Glycolic acid is an alpha hydroxy acid thins the stratum corneum, disperse melanin in the epidermis and improves the distribution of other drugs in the skin [3,9]. Salicylic acid is a beta hydroxy acid with keratolytic and antiinflammatory properties [9]. Trichloroacetic acid (TCA) is a derivative of acetic acid and causes protein denaturation, with depth of necrosis proportional to concentration [9]. Aminofruit acids are carboxylated amino-acids, also potent antioxidants [9]. Jessner's solution is a combination of salicylic acid 14%, lactic acid 14% and resorcinol 14% in an alcohol base. Through its keratolytic activity it acts as a superficial chemical peeling agent [20].

Lasers may be the recent treatment of choice only for resistant melasma, especially in fair-skinned patients [20]. Different laser and light treatment including O-switched neodymium: yttrium-aluminium-garnet laser, Q-switched ruby laser, Q-switched alexandrite laser, copper bromide laser, erbium:YAG laser, 1550 nm erbium-doped fractional laser, carbon dioxide fractional ablative laser and intense pulsed light have been tried as treatment options [1,9].

A study with 50 patients was published to evaluate the efficacy and safety profile of a 1064 nm Q switched neodymium doped yttrium aluminium garnet (QS Nd:YAG) laser in the treatment of melasma. Here, Nd: YAG laser was used at low energy levels weekly for nine sessions. Melanin index (MI) and Melasma Area Severity Index (MASI) scores were evaluated for all patients. Q-switched lasers are based upon the theory of selective photothermolysis, targeting lesional melanocytes and its melanin particles within melasma. This melanolysis probably resulted from an inhibition of melanin synthesis in melanocytes, melanin transfer from melanocytes to keratinocytes and excretion of melanin particles. The conclusion of this study was a confirmation of effectivity and safety of this laser in melasma treatment, however recurrence rates remain high (64% at the 3 month follow-up) and further adjunctive therapy is needed. Moreover, this study disclosed a negative correlation between therapeutic outcome and severity of disease on the baseline. Here, when melasma is more severe, less improvement can be expected. On the other hand, a good response was found in both epidermal and dermal type of melasma, independently on Fitzpatrick skin type, may be due the deep penetration of this laser with wavelength of 1064 nm [5].

1550 nm fractional nonablative laser is recently it is investigated in melasma treatment [9]. A trial in 51 patients with melasma was described by Karsai and colleagues. Here, four sessions of nonablative fractional photothermolysis (NFP) in combination with broad-spectrum sun-screen were performed in 3 weeks intervals, compared to use sun-screen alone. Patients were evaluated in baseline and 12 after treatment.

The result of this trial was not really encouraging. In both groups MASI was reduced significantly after therapy. Thus, these findings do not support the hypothesis of NFP providing a substantial benefit in treating melasma.

In summary, resurfacing procedures, including chemical peels and lasers, are best used in combination with topical therapy [20].

Oral antioxidants (Procyanidin), vitamin A, C taken orally can have also positive effect in melasma treatment [9].

Conclusion

Melasma therapy in men can be a long-term and ungrateful challenge, due to worse compliance of patients and often relapses. Epidermal type of melasma shows clinical improvement with topical hypopigmenting agents, compared to dermal type, which can be resistant and may require additionally laser therapy or superficial dermabrasion [3].

References

- Vachiramon V, Suchonwanit P, Thadanipon K (2012) Melasma in men. *J Cosmet Dermatol* 11: 151-157.
- Sarkar R, Puri P, Jain RK, Singh A, Desai A (2010) Melasma in men: a clinical, aetiological and histological study. *J Eur Acad Dermatol Venereol* 24: 768-772.
- Jadotte YT, Schwartz RA (2010) Melasma: insights and perspectives. *Acta Dermatovenerol Croat* 18: 124-129.
- R.Konkolova (2001) Korektivne dermatologicke metody. (1stedtn). Maxdorf s.r.o., Prague.
- Zhou X, Gold MH, Lu Z, Li Y (2011) Efficacy and safety of Q-switched 1,064-nm neodymium-doped yttrium aluminum garnet laser treatment of melasma. *Dermatol Surg* 37: 962-970.
- Pichardo R, Vallejos Q, Feldman SR, Schulz MR, Verma A, et al. (2009) The prevalence of melasma and its association with quality of life in adult male Latino migrant workers. *Int J Dermatol* 48: 22-26.
- Vázquez M, Maldonado H, Benmamán C, Sánchez JL (1988) Melasma in men. A clinical and histologic study. *Int J Dermatol* 27: 25-27.
- Guinot C, Cheffai S, Latreille J, Dhaoui MA, Youssef S, et al. (2010) Aggravating factors for melasma: a prospective study in 197 Tunisian patients. *J Eur Acad Dermatol Venereol* 24: 1060-1069.
- Ball Arefiev KL, Hantash BM (2012) Advances in the treatment of melasma: a review of the recent literature. *Dermatol Surg* 38: 971-984.
- Hernández-Barrera R, Torres-Alvarez B, Castaneda-Cazares JP, Oros-Ovalle C, Moncada B (2008) Solar elastosis and presence of mast cells as key features in the pathogenesis of melasma. *Clin Exp Dermatol* 33: 305-308.
- Kang HY, Hwang JS, Lee JY, Ahn JH, Kim JY, et al. (2006) The dermal stem cell factor and c-kit are overexpressed in melasma. *Br J Dermatol* 154: 1094-1099.
- Jang YH, Sim JH, Kang HY, Kim YC, Lee ES (2012) The histopathological characteristics of male melasma: comparison with female melasma and lentigo. *J Am Acad Dermatol* 66: 642-649.
- Sialy R, Hassan I, Kaur I, Dash RJ (2000) Melasma in men: a hormonal profile. *J Dermatol* 27: 64-65.
- Famenini S, Gharavi NM, Beynet DP (2014) Finasteride associated melasma in a Caucasian male. *J Drugs Dermatol* 13: 484-486.
- Cetková P, Pizinger K, Stork J (2010) Kožní změny u interních onemocnění (Skin changes in internal diseases) - Grada Publishing a.s. 2010, U Pruhonu 22: 95.
- Keeling J, Cardona L, Benitez A, Epstein R, Rendon M (2008) Mequinol 2%/tretinoin 0.01% topical solution for the treatment of melasma in men: a case series and review of the literature. *Cutis* 81: 179-183.
- Kang HY, Hwang JS, Lee JY, Ahn JH, Kim JY, et al. (2006) The dermal stem cell factor and c-kit are overexpressed in melasma. *Br J Dermatol* 154: 1094-1099.
- Lonsdale-Eccles AA, Langtry JA (2005) Melasma on the nape of the neck in a man. *Acta Derm Venereol* 85: 181-182.

-
19. Prignano F, Ortonne JP, Buggiani G, Lotti T (2007) Therapeutical approaches in melasma. *Dermatol Clin* 25: 337-342, viii.
20. Sarkar R, Chugh S, Garg VK (2012) Newer and upcoming therapies for melasma. *Indian J Dermatol Venereol Leprol* 78: 417-428.
21. Karsai S, Fischer T, Pohl L, Schmitt L, Buhck H, et al. (2012) Is non-ablative 1550nm fractional photothermolysis an effective modality to treat melasma? Results from a prospective controlled single-blinded trial in 51 patients, *JEADV* 26: 470-476.

This article was originally published in a special issue, [Melasma](#) handled by Editor(s). Dr. Serena Gianfaldoni, University of Pisa, Italy