Pathogenesis of Melasma: Where Do We Stand?

Melasma is a common acquired disorder of hyperpigmentation characterized by irregular light brown to dark brown patches of hyperpigmentation commonly affecting the face. The trunk and arms are also occasionally involved. Multiple studies have documented the negative impact of melasma on quality of life as it displays as a phenotype of photodamage. Moreover, new research has led to an increased understanding of the complex pathogenesis of this disorder. Key etiologic factors include a genetic predisposition, solar damage, barrier abnormalities, and unique sensitivities to hormonal changes including pregnancy, oral contraceptives, and hormone replacement therapy. Multiple studies document the role of melanocytes, keratinocytes, and dermal cells including fibroblasts and mast cells in melasma. Transcriptomic studies document upregulation of pigment genes, Wnt genes, and prostaglandins. There is also an increase in melanogenesis, lesional alpha-MSH, solar elastosis, dermal blood vessels, expression of VEGF, and mast cells. Recent studies display that there is altered barrier function in cells due to basement membrane damage, and an aberrant response to estrogen and progesterone. Moreover studies now suggest that inflammatory mediators including nitric oxide play a role in this chronic disease. New technological and pharmacological advances have facilitated expansion of approaches for the prevention, diagnosis, and long term management of this difficult and challenging chronic pigmentary disorder. No cures have been developed as of yet. Evidence based studies suggest the best therapeutic outcomes are achieved with combination therapy or triple combination bleaching with hydroquinone, a steroid, and retinoids. Multiple new non-hydroquinone formulations are now available, and can be used in combination with hydroquinone products in a rotational algorithm for therapeutic intervention. New treatments are needed to address the vascular component of melasma (see references).
References


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