Introduction

Psoriasis is an immune-mediated, multifactorial skin disease with hyperproliferation and altered differentiations of keratinocytes, linking the pathways of angiogenesis and inflammation (**Simonetti et al., 2006**).

Vessels expansion seems to play an important role in the evolution of psoriatic plaques (**Henno et al., 2010**). Several growth factors, which include platelet derived growth factor (PDGF), fibroblast growth factor, epidermal growth factor and transforming growth factors (TGF)-α and –β, demonstrate angiogenic activity. However, vascular endothelial growth factors (VEGF) is recognized as a pivotal factor responsible for angiogenesis in different tissues (**Chua and Arbiser , 2009**).

Overexpression of VEGF can promote new blood vessel formation and account for the chronicity in psoriatic lesion (**Ferrara, 2000**). In vitro culture studies revealed that VEGF in the skin is secreted predominantly by keratinocytes and its concentration is enhanced in skin of patients with psoriasis (**Bhushan et al., 1999**). Moreover, constant delivery of VEGF in the skin in the transgenic mouse results in development of psoriasis-like inflammation **(Teige et al., 2009).**

Serum or plasma concentrations of some inflammatory cytokines (IL-4, IL-6, MCP-1, VEGF, TGFβ1, TIMP and PDGF) which are elevated in severe forms of psoriasis stimulate VEGF production by epidermal keratinocytes **(Deeva et al. ,2010).** It can result in enhanced inflammation

accompanying excessive angiogenesis and vasodilation in psoriatic skin lesions **(Kakurai et al., 2009).**

Tazarotene, the only approved topical retinoid available for treatment of mild psoriasis, acts on psoriatic lesions after conversion by esterases in the skin to the active form, tazarotenic acid. The retinoid selectively binds to β and γ-retinoic acid receptors (RAR) on the cell membrane of keratinocytes and is then transported to the nucleus, altering transcription of genes in keratinocytes ***(Mason et al., 2009)***.

For more extensive lesions Several modalities of treatment are available. However, phototherapy is still the mainstay in the treatment of the disease ***(Naldi and Griffiths, 2007)***.

Narrow band UVB is used in plaque psoriasis involving more than 20% of the body surface area. It utilizes the effective UVB range and excludes Erythema-inducing rays, thus having a definite advantage over broad band UVB, and PUVA that requires pretreatment with psoralin that may cause phototoxicity, xerosis or hyperkeratosis. The advantages of UVB are shorter sessions and suitability in children and pregnant women **(Housman et al., 2002)**.