

INTRODUCTION

Scar may be defined as the new tissue that is formed during healing of the wound. Human soft tissue healing is basically non regenerative. That which is destroyed is not restored but replaced by a less well differentiated tissue (scar) (*Thomas,2004*).

Keloid and hypertrophic scars are two kinds of abnormal scar formation in the skin. The mechanism that causes them is not fully understood, but it is thought to involve local histologic factors in connection with a hereditary component in most cases (*Espana et al.,2001*). Traumatic factors that may trigger keloid formation in genetically predisposed individuals include burns, incisional wounds, infections, acne, and other inflammatory disorders (*Fitzpatrick,1999*). Keloid and hypertrophic scars result from excessive collagen deposition. Hypertrophic scars and keloids may lead to significant morbidity as well as pruritus, pain, restriction of motion, or cosmetic disfigurement (*Alster and Tanz,2003*).

There are several non pharmacological methods of treating hypertrophic scars and keloids, but they have drawbacks like high cost (silicone gel sheeting), poor efficacy (Surgery and laser therapy), recurrence (surgery) and adverse effects like malignancy (radiation therapy), pharmacological modes of treatment also have drawbacks like: tacrolimus, bleomycin, interferon alpha, 5 fluorouracil and triamcinolone. The prophylaxis is more effective (*copcu et al ., 2004*).

The efficacy of intralesional corticosteroid injections in the treatment of keloids and hypertrophic scars has been well established, and they have been a mainstay in the treatment of keloids, alone or in

combination with other modalities (*Manuskiatti and Fitzpatrick.,2002*) Corticosteroids reduce excessive scarring by decreasing collagen synthesis, glycosaminoglycans synthesis, the expression of inflammatory mediators, and fibroblast proliferation during wound healing. A welldocumented corticosteroid in the intralesional application is triamcinolone acetonide (TAC) (*Berman and Flores,1998*).

Being depot preparation of triamcinolone acetonide. Triamcinolone injection alone is effective in reducing the volume of lesions in a majority of patients (*Muneuchi et al.,2006*).

In 1990, *Lee and Ping* first demonstrated that calcium antagonists such as verapamil depolymerize actin filaments and consequently modify fibroblasts in normal scars as well as keloids.