INTRODUCTION

Vitiligo is an idiopathic acquired cutaneous achromia. At least 1% of population is believed to be affected with it. Its exact etiology is still controversial. (Korann, et al., 1988)

The vitiligo is a disorder in which there is a focal defect of pigmentation due to destruction of melanocytes which is thought to be mediated by immunological mechanisms. (Marks, 1993)

It is presented as acquired patchy macular depigmented lesions which gradually progress over many years. (Lorincz, 1975)

Macules appear white in colour within which the hair may become melanotic or not. (Muhleman, 1985)

Various theories are suggested for the etiology. The autoimmune hypothises, neurogenic hypotheses and the self-destructive theory. (El-Mofty, 1968) (Moellman, et al., 1985)

Many therapeutic options are available for treating vitiligo, such as topical application of corticosteroid and photochemotherapy (El-Mofty, 1948; Kandil 1972; Parrish et al. 1976), transplantation of blister tops (Koga, 1988), or minigrafts from normal pigmented areas (Flabella, 1988) and the application of autologous epidermal sheets established in vitro (Olsson, and Juhlin, 1997)
Topical cytotoxic drug was also used for treatment of vitiligo, 5-fluorouracil cream was applied once daily on dermabraded vitiligenous areas for seven to ten days. *(Tsuji and Hamada, 1983)*

Some recent studies have suggested that hair grafts may have some positive effect on the recovery of pigmentation. *(Gunn Yoen, et al 1998)*