

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 amelanotic or not. (*Muhleman .1985*)

Various theories are suggested for the etiology. The autoimmune hypothesises , neurogenic hypotheses and the self-destructive theory. (*El-Mofty .1979*) (*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*)