Introduction

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Warts are contagious, benign tumors of the epidermis. However they may heal spontaneously without leaving scar, treatment should never be more trouble than the disease itself ***(Munkvad et al., 1983)***. Warts are rarely a serious problem except in instance of failure and /or recurrence ***(Amer et al., 1988)***.

Bleomycin, the generic name for sulfur-containing polypeptide antibiotic derived from Streptomyces verticillus, has been used as a single agent or in combination for treatment of various neoplasms ***(Shumer et al., 1983)***.

The intralesional injection of warts with the anti-neoplastic /antibiotic drug bleomycin has unusual properties of preferential binding in squamous cells, DNA strands scission and restricted toxicity ***(Bunney et al., 1984)***.

Intralesional bleomycin therapy has been used in the treatment of viral warts since 1970s ***(Sollitto et al., 1989)***. Although it was an effective method of treatment of resistant warts, it is still not widely used ***(Munn et al., 1996)***. Little is known about the action of bleomycin or its systemic absorption after injection into viral warts ***(James et al., 1993)***.

The mechanism of action of bleomycin in wart therapy has been assumed to be secondary to DNA and antiviral effects, it is theoretically possible that induction of local tumor necrosis factor

(TNF) production by bleomycin may partially account for the observed effect of bleomycin on warts. Tumor necrosis factor is also known to up-regulate the expression of intercellular adhesion molecule-1(ICAM-1), endothelial leukocyte adhesion molecule-1(ELAM-1), and vascular cell adhesion molecule-1 (VCAM-1) to induce a tissue factor-like procoagulant activity on human endothelial cell, and to cause hemorrhagic necrosis of the tumor ***(Templeton et al., 1994)***.