Chapter II

Warts

Warts are caused by one or more of the various strains of Human Papilloma Viruses (HPVs). They are most frequent in children and young adults. The lesions so produced can be divided into the following clinical types:

- common wart (verruca vulgaris)
- flat wart (verruca plana)
- plantar wart (verruca plantaris)
- epidermodysplasia verruciformis
- laryngeal papilloma
- oral papilloma
- anogenital wart (condyloma acuminatum)
- flat lesions of the uterine cervix (including bowenoid papulosis) (Tagami, Ogushi and Ofuji, 1983)

In any infection, the host immune responses are stimulated. This is also the case with warts. However, warts exhibit one special character for they share features of both infection and tumor (Dahl, 1988). Thus it is quite plausible that regression of warts, if better understood immunologically, might give insight into immunology of neoplasms in man.

Both humoral and cell-mediated immune responses are triggered by HPV infection. However, the latter plays a major part while the former is involved to a lesser degree (Dahl, 1988). A supporting evidence for this statement is the observation that patients with cell-mediated immune deficiency (Hodgkin's disease, chronic lymphocytic leukemia and other malignant lymphomas) appear to be more prone to wart infection as compared with conditions inducing purely humoral immune deficiency such as myeloma (Reid, Fraser and Kernohan, 1976; Dahl, 1988 and Morrison, 1975). Not only are they numerous but they are also unusually resistant to ordinary treatment (Morrison, 1975). Moreover, it is known that the warts of immunosuppressed individuals may undergo malignant transformation as observed in EV and transplant patients (Bender, 1986).

On the other hand, warts tend to regress spontaneously in most patients with normally functioning immune system. In warts showing spontaneous regression T-lymphocytes and Langerhans cells are seen both in the epidermis and upper dermis (Dahl, 1988 and Iwatsuki et al, 1986). Tagami et al., in 1983, also noted satellite cell necrosis in lesions of spontaneously regressing flat warts. These findings are reminiscent of allergic contact dermatitis and GVHR, two processes in which CMI plays crucial roles.

Treatment of few lesions usually involves measures aimed at physical elimination of wart-infected tissues. Commonly

used treatments are electrocauterization, surgery, cryotherapy or the use of various preparations of salicylic acid, lactic acid, podophyllin, cantharidin, trichloroacetic acid, formalin and glutaraldehyde (Taylor, 1988; Mroczkowski & McEwin, 1985; Birkett, 1982). Intralesional Bleomycin and carbon dioxide Laser are also used but they are not without side effects.

Another line of treatment involves immunomodulation. As stated before, patients with a long history of wart infection may have, to some extent, CMI impairment (Morrison, 1975). Persistent viral infection might, in turn, also contribute to depression of T-cell function (Mohanty and Roy, 1984). Topical immunotherapeutic agents such as DNCB, diphenciprone and squaric acid dibutyl ester have been used with some success (Dunagin and Millikan, 1982; Lewis, 1973; Orecchia, 1988).