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WARTS, HPV AND HOST IMMUNITY : A COMPLEX RELATION

Dermatology

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A complex relationship between warts, Human papilloma Virus (HPV) and the human host immunity is an area which needs further exploration than what we know now.[1],[2] Skin possesses many connected linked defense mechanisms that include an intact stratum corneum, complement mediated phagocytosis and the adaptive immunity. HPV gains entry through minor skin defects to infect the basal epithelial layer. HPV infections are unlike the short lived acute viral infection influenza which induces a strong immune response. By the property of antigenic variation, genome integration and by residing in sites which are not reachable to immunity cells, HPV can escape detection.

HPV infection can be non-symptomatic or can result in epidermal hyperkeratosis seen in warts or may even cause malignant lesions.[3] The basis of Papillomavirus (PV) taxonomy are genomic they are unsuitable for culturing. They also don't demonstrate robust antibody responses.[4]

Molecular hybridization techniques and polymerase chain reaction (PCR) can identify the virus. More than 150 types of papillomavirus have been identified and this is perhaps the reason for the different forms of warts. The other reason could be its tropism for stratified squamous epithelial cells and different types having specificity to the different anatomical sites. HPV 1 multiply in thick keratinized skin of palms & soles. HPV 16 prefers genital mucosa. HPV 11 in both genital and larynx epithelium. HPV types 10, 1, 27, 3, 4 and 57 are responsible for benign cutaneous warts. HPV 16 & 18 can induce dysplasia hence are high risk. Association of HPV type 16 with insitu genital squamous cell carcinoma and HPV 16 & 18 with cervical cancer is well known and also with a condition called bowenoid papulosis (carcinoma in situ but can spontaneously regress). Epidermodysplasia verruciformis (EV) which is a heritable condition is supportive of this association characterized by multiple plane warts that can turn into squamous cell carcinomas. PVs are different from one another in the type of lesions that develops and also the tumor formation potential. [5],[6]

HPVs target stratified epithelia of skin, ano genital tract & oral cavity hence said to be epitheliotropic. So on top of being tissue specific, they are also species specific. There can be both cutaneous & mucosal types in a genus. One example of it is Alpha genus in which mucosal has high risk and low risk PVs. HPV 57 & 2 which produces common wart are low risk alpha type.[6]

Different transmission strategy and methods exist for propagation in epithelium as well as how it interacts with the host immunity.[7]

HPV infection can evade recognition by the host innate immunity. The life cycle of the virus is inside the epithelium. No viraemia in the blood nor induction of cell death takes place. Multiplication of virus and discharge do not cause inflammation. Down regulation happens to innate immune signalling pathway in the infected cell, pro-inflammatory cytokines and dendritic cell or activation of antigen presenting cell. Inadequate dendritic cells and macrophages are recruited. Shedding of virus proteins occurs from the epithelial surface unnoticed by circulating immunity sentinel cells. Immune tolerance develops in place of a robust T cell action which would have clear away the HPV.[8],[9]

Persistence of HPV infection thereby depends on both viral immunology and the host immunity. Cell mediated immune system has a role in rejection of warts as the group of people with defective cell mediated immunity have higher prevalence of warts besides increased HPV related malignancy. Warts which are resolving have lymphocytic infiltrate which is cytotoxic T cells with predominant T helper-1 immune response. [10],[11]

Therefore, a further research in this field should be in the direction of finding more concrete answers which can unmask the relation between how HPV infection and host immunity interact and manifest in different forms whether benign or malignant. This will also be able to explain the mechanism in more detail of how immunotherapy like Measles,mumps,rubella (MMR) vaccine or Purified protein derivative (PPD) works for a unrelated infection by Human Papilloma Virus.

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