THE EGYPTIAN JOURNAL OF IMMUNOLOGY Vol. 25 (1), 2018
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Human Leukocyte Antigen-G (HLA-G) Expression in Precancerous and Cancerous Cervical Lesions: Association with Human Papilloma Virus Infection and Host Immune Response

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Human leukocyte antigen-G (HLA-G) is a non-classical HLA-class Ib molecule with multiple immunoregulatory properties. Its main functions in physiological conditions are to abolish maternal immune cell activity against fetus and to establish immune tolerance at the maternal-fetal interface. Cervical tumor cells have been reported to express HLA-G which is one of the immunomodulatory molecules that is involved in every phase of cancer immunoediting. It has inhibitory functions against natural killer (NK) cells, T-lymphocytes, and antigen-presenting cells (APCs). The purpose of this study was to investigate the HLA-G expression in precancerous (squamous intraepithelial cervical lesion) and cancer cervix and determine HLA-G expression relation to HPV infection as well as host immune response. The study included 48 paraffin embedded cervical tissue sections [32 squamous intraepithelial lesion (SIL) {16 low grade lesions (LSIL) and 16 high grade lesions (HSIL)} and 16 cervical cancer tissue sections]. All tissue sections were examined for HLA-G expression by real time PCR and for host immune response by estimating the number of tumor infiltrating lymphocyte (TIL) and NK $CD57^+$ cells. HLA-G expression increased progressively from precancerous and cancerous cervical lesions. There was an inverse relationship between HLA-G expression and estimated number of TILs and NK $CD57^+$ cells. No significant statistical difference between HPV positive and HPV negative cervical lesions as regards HLA-G expression was detected. In conclusion, HLA-G is a potential biomarker for the diagnosis and prognosis of cervical cancer as there was a progressive increase in expression of HLA-G in precancerous and cancerous cervical lesion. It is functionally involved in tumor escape mechanisms as observed by inhibition of host immune response and more studies are needed to design strategies for blockade of HLA-G expression or elimination of HLA-G expressing cancer cells as this may be important to the efficacy of anticancer therapies.