Correlation of E6 and E7 levels in high-risk HPV16 type cervical lesions with CCL20 and Langerhans cells

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ABSTRACT. The human papillomavirus (HPV)16 E6 and E7 correlation with chemokine ligand (CCL)20 expression and Langerhans cells (LCs) in cervical lesions was investigated. We enrolled 43 patients with surgically treated cervical lesions from the Department of Gynecology in our hospital, and 20 controls without cervical lesions. Subjects were divided by pathology: HPV16(-) and HPV16(+) normal cervical groups (N = 10 each), and HPV16(+) cervical intraepithelial neoplasia (CIN), cervical invasive carcinoma (N = 15 each), and in situ carcinoma (N = 13) groups. E6, E7, the LC surface marker CD1a, and CCL20 were analyzed by immunohistochemistry. E6 and E7 in HPV16-type lesions were correlated with CCL20 and LCs. The average high power field cell numbers of CD1a+ LCs in the HPV(-) and HPV(+) normal cervix groups, and the CINI-II, CINIII in situ and cervical carcinoma groups were 22.89 ± 4.84, 13.7 ± 2.26, 9.2 ± 1.68, 5.9 ± 1.59, and 5.5 ± 1.58, respectively. Significant between-group differences existed except between cervical carcinoma and CINIII groups (P < 0.05). CCL20+ rates in each group were 70, 60, 60, 15.38, and 13.33%, respectively.
E6/E7-positive expression rates in each group were 20/20, 66.7/66.7, 76.9/69.2, and 86.67/73.3%, respectively. CCL20 was positively correlated with CD1a (r = 0.649), and negatively correlated with E7 (r = -0.946) and E6 (r = -0.949). CD1a was negatively correlated with E6 (r = -0.632) and E7 (r = -0.632). Downregulation of CCL20 leading to LC decline is a key factor in cervical lesions. High-risk HPV-type lesions might inhibit the chemokine CCL20 through E6 and E7 to escape the immune response.

**Key words:** Cervix; CCL20; Langerhans cells; E6; E7