P–38; **DETECTING HPV L1 PROTEIN CAPSID IN CERVICAL LESIONS**

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Background: Cohort studies have shown that for developing precancerous lesions of uterine cervix HPV infection is crucial. But the main event in the carcinogenesis is not simple detection of infection, but its persistency and viral integration in association with HPV physical status (episomal or integrated).

Objectives: Aim of the study is to investigate the prevalence of HPV L1 capsid protein in low grade and high grade squamous lesion (LSIL/HSIL) of the uterine cervix using cytoactive method and compare it with findings of p16INK4a protein in HSIL smears.

Materials and Methods: Since the June 2007 – December 2007 we analysed cervical smears from 67 patients. All of them are high risk HPV DNA positive. After decolourising slides we performed immunoassaying with cytoactiv screening set for HPV L1 (cytoimmun diagnostic GbmH, Pirmanses, Germany). In patients with HSIL after decolourising their prior cervical smears we performed p16 INK4a staining using DAKO monoclonal antibody.

Results: Cytological diagnosis of LSIL was made in 53 patients (53/67), and 14 patients (14/67) had HSIL. In all 14 patients HSIL was confirmed by biopsy or cold knife conisation. After immunoassaying positive HPV L1 was found in 60, 4% (32/53) of LSIL and in 21, 4% (3/14) of HSIL specimens. Strong p16INK4a positive stain was seen in eleven HSIL smears (78, 6%).

Conclusions: Lower L1 detection rate in HSIL can be explained by possible loss of viral L genes and consequently lower production of capsid proteins. On the other hand, positive p16INK4a stain represents viral integration in host genome and genetic instability of the host cells which can lead to its malignant transformation. Hence, in L1 negative and p16INK4a positive smears, lesions will most likely progress, so patients can be advised for shorter control period or sooner histological confirmation.