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Disruption of Hpv-16 E1 and E2 regulatory genes as a prognostic factor in cervical cancer

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PV-16 integration into the host chromosome is thought to be an event in cervical malignant transformation, as it may result in uncontrolled expression of viral E6/E7 oncoproteins after disruption or deletion of the viral E2 or E1 regulatory sequences. The purpose of the study was to determine the sites of E1 and E2 HPV16 sequence disruption in cervical cancer (Ca) and in low-grade squamous intraepithelial lesion (LSIL) as a control. The study was conducted in 26 women with HPV-16 infection (INNO-LiPA Genotyping, Belgium), with a diagnosis of Ca (n=11) and LSIL (n=15). Complete E1 and E2 sequences were amplified with 2 and 4 pairs of overlapping primer sets, respectively. The polymorphisms of E1 and E2 were identified by sequencing. The results were compared with the prototype sequence (Gen-Bank no. K02718). In the LSIL group, the deletion of the E1 or E2 sequence of HPV16 occurred at a similar rate (13%), while in 46% of the patients the disruption was detected in both genes. In 4 cases no damage was shown. In cervical cancer, interruption of the E2 sequence occurred in all samples but in 3 together within the E1 sequence. The most frequently occurring damage was the E2 hinge region. Preliminary studies suggest that the site of disruption within the E2 sequence may be a prognostic factor for cervical cancer development. This study was supported by Jagiellonian University Medical College–Statutory Research Katarzyna Sitarz acknowledges the support of InterDokMed project no. POWR.03.02.00-00-I013/16.

Biography

Katarzyna Sitarz received BSc of Biotechnology in 2015 and MSc of Biochemistry in 2017 (both at the Jagiellonian University). Now she is MD student (from 2016) and PhD student (from 2017) at Jagiellonian University Medical College.

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