in avoiding PLA. In 17 patients (9.9%), CT revealed unexpected pathologic findings, which were succeeded by other invasive or non-invasive examinations.
Conclusions: The sensitivity of CT in this study is extremely low, corresponding to earlier findings. CT does not meet the demands of a meaningful diagnostic tool for this group of patients. The disadvantages are obvious: Exposition to unnecessary radiation, prolongation of the time of treatment and redundant expenses. We propose that CT should consequently not be performed in the staging of PC.

N6

BRCA2 gene mutation characterization in hereditary prostate cancer patients in Latvia

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Introduction and Objectives: Prostate cancer is one of the most commonly diagnosed tumors affecting men in Latvia. A significant factor in prostate cancerogenesis is genetic. The failure to identify highly penetrant genes in hereditary prostate cancer may result from the fact that multiple genes with a small to moderate effect are involved. Aim of the study is to evaluate the epidemiological features and BRCA2 gene mutation variations in hereditary prostate cancer in Latvia, as well as to consider association with other malignancies.
Material and Methods: In total 855 prostate cancer families were selected for our study that was identified from Nov-2003 to Jan-2009. Data were collected on their clinical characteristics; additionally blood samples were collected for DNA tests. Family cancer histories were analyzed according to clinical diagnostic criteria of hereditary prostate cancer, if at least one of the following criteria applied: (a) at least 3 affected blood relatives at any age; or (b) 2 affected blood relatives at age ≤55 years. Complete sequencing of the BRCA2 gene was performed for 9 probands from selected hereditary prostate cancer families.
Results: 855 family histories of prostate cancer patients were analyzed; from those 11 (1.3%, 95% CI: 0.7–2.3) families matched clinical diagnostic criteria of hereditary prostate cancer. The median age at diagnosis in this group was 65.4 (range 54–74) years. Besides BRCA2 polymorphisms in all analyzed cases, the novel missence mutation V2419D was detected in 73 years old proband with family history of 2 prostate, 1 lung and 1 stomach cancer. Along with prostate cancer at least 1 other localization malignancy was found among blood relatives in 9 (81.8%, 95% CI: 52.3–94.9) families of hereditary prostate cancer group. Distribution of the most frequent malignancies are: lung in 4 (36.4%) cases, uterine in 3 (27.3%) cases, stomach in 2 (18.2%) cases, breast in 2 (18.2%) cases, kidney in 1 (9.1%) case, leukemia in 1 (9.1%) case, unknown in 1 (9.1%) case.
Conclusions: Hereditary prostate cancer according to definitive clinical diagnostic criteria was found in 1.3% (95% CI: 0.7–2.3) of prostate cancer cases in Latvia. Further research of first degree family members and control group needed to confirm clinical significance of detected novel missense mutation V2419D in predisposition to hereditary prostate cancer and other localization malignancies.

N7

Trinucleotide repeat length polymorphisms of the androgen receptor gene – a step forward in understanding the pathogenesis of prostate cancer

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Introduction and Objectives: The pathogenesis of prostate cancer (PC) and the mechanisms responsible for progression of this tumor are not fully understood. The fact of androgen dependence of PC is known since 1940s. Action of androgens in prostate gland is mediated through the cytoplasmic molecule of androgen receptor (AR), which in turn stimulates expression of target genes correlated with regulation of cell cycle. Three regions of trinucleotide repeats were identified in the sequence of exon 1 of AR gene, namely (CAG)n, (GGN)n and (CCN)n. Two former are characterized by repeat length polymorphisms, which influence the activity and protein yield of AR. Data from literature suggest that shorter (CAG)n and (GGN)n regions are associated with higher transactivation activity and higher protein yield of the AR and may therefore constitute a risk factor for PC. The aim of our study was to evaluate the correlation between (CAG)n, (GGN)n and (CCN)n repeat length polymorphism of the AR gene and prostate cancer risk in polish population.
Material and Methods: The study was approved by local ethics committee. Written informed consent was obtained from 120 consecutive patients with pathologically confirmed prostate cancer, hospitalized in department of urology in Szczecin between 2004 and 2006. Population control group comprised genomic DNA samples isolated from umbilical cord blood samples of 120 male newborns born in the neonatology unit between 2004 and 2005. Amplification of polymorphic regions (CAG)n, (GGN)n and (CCN)n was performed using PCR method with fluorochrome stained primers. Length of microsattelites was assessed by means of capillary electrophoresis and sequencing. Statistic analysis was performed with Statistica 7.0. software.
Results: There were repeat length polymorphisms identified in (CAG)n and (GGN)n regions. The number of CCN repeats remained constant in both groups. There was no significant correlation between number of CAG repeats and number of GGN repeats. In comparison with control group, patients with PC had a significantly ($p < 0.05$) higher frequency of shorter CAG ($n \leq 18$ repeats) and GGN ($n \leq 19$ repeats) regions.
Conclusions: Shorter variants of (CAG)n and (GGN)n microsatellites were already shown to be associated with higher biological activity of androgen receptor. Our current study demonstrated that they are more frequently found in patients with prostate cancer than in population control. These findings suggest their role in pathogenesis of prostate cancer, and make them candidates for future genetic models of PC risk assessment.