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Oral Presentation
OP-11

METHYLATION-SPECIFIC PCR REVEALED ABERRANT PROMOTER GENE METHYLATION OF *p16, MGMT* AND *SPOCK2* IN DIFFUSE LARGE B CELL LYMPHOMA

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DNA methylation silences the gene through addition of methyl group.p16, a tumor suppressor gene that inhibits cyclin-dependent kinase, inactivates the Rb protein and blocks G1 phase in a normal cell cycle.A DNA repair gene, MGMT removesalkyl adduct to a cysteine residue within the protein, thus preventing lethal cross-links. p16 and MGMTmethylationhas been reported to associate with DLBCL. A member of the extracellular chondroitin and heparinsulfate proteoglycans, SPOCK2functions mainly in extracellular matrix for cell adhesion. Uniquely, SPOCK2 (testican 2) abolishes the inhibition of membrane-type 1-matrix metalloproteinase by other testican family which mightenhance the angiogenesis.In this study, we aimed to screen for aberrantly methylated genes which might contribute to the pathogenesis of DLBCL using methylation specific PCR (MSP). p16 methylation was identified in 64 (73%)of 88 samples. On the other hand, SPOCK2 was found to be unmethylatedin 30 (34%) samples. Interestingly, MGMT methylation was detected in all cases. We also found an association between p16 methylation status with patients aged >50 years old (p= 0.023).This finding is parallel with an animal study showing that aging increases p16 methylation. No association was found between the methylation of other genes with age. Unmethylation of SPOCK2 might cause testican 2 expressions, which has been suggested to contribute toward malignant behaviour. MGMT was reported to be methylated amongcancer patients who smoke, drink and are non-vegetarian. Thus, it is hypothesized that lifestyle might affect MGMT methylation in this study population.