Abstracts

PNR-10. MOLECULAR CHARACTERIZATION OF PEDIATRIC GLIONEURONAL TUMOR WITH NEUROPIL-LIKE ISLANDS: A GENOME-WIDE COPY NUMBER ANALYSIS

Laura Giunti¹, Anna Maria Buccoliero², Marilena Pantaleo¹, Silvia Guarducci¹, Maurizio Luchessa³, Viviana Palazzo³, Mälena Guidi³, Lorenzo Genitori⁴, Sabrina Giglio¹,⁵, and Iacopo Sardi³; ¹Medical Genetics Unit, Meyer Children’s University Hospital, Florence, Italy; ²Anatomic Pathology Unit, Meyer Children’s University Hospital, Florence, Italy; ³Neuro-Oncology Unit, Department of Pediatric Oncology, Meyer Children’s Hospital, Florence, Italy; ⁴Neurosurgery Unit, Department of Neuroscience, Meyer Children’s Hospital, Florence, Italy; ⁵Medical Genetics Unit, Department of Clinical and Experimental Biomedical Sciences ‘Mario Serio’, University of Florence, Florence, Italy

Glioneuronal tumor with neuropil-like islands (GTNI) are a rare group of Central Nervous System (CNS) tumors with a variable clinical spectrum aggressiveness. GTNI, also known as rosetted glioneuronal tumor, are described for the first time about 16 years ago and many pathologists consider this tumor as an astrocytoma with an aberrant neuronal differentiation. Most of the clinical and molecular information of GTNI originate from studies in adult case. In recent years the use of array-CGH has improved the understanding of the biology of various types of cancers, including those of the CNS, identifying Copy Number Alterations, (CNA), useful for a better understanding of the molecular mechanisms that are at the base of the progression of these tumors. By array-CGH we studied four pediatric GTNI. Moreover in 3 cases it was possible to compare the results with the non-tumor DNA with the aim to identify possible specific CNA. Unlike our previous study on pediatric glioblastomas (Giunti L, et al. Am J Cancer Res. 2014; 4: 293) we have not identified specific and recurrent CNA. In two of the four cases of GTNI we found both a duplication of the entire chromosome mosaic 8 and a duplication in 5q14.1 containing DMGDH, BHMT2 and BHMT genes. In conclusion our preliminary results will allow us to better understanding the mechanisms underlying the genesis and progression of this rare glioma variant.