PP64

Neuropsychological deterioration predicts tumor progression in a young boy with bithalamic glioma

Laura Peruzzi[1,2], Laura Iuvone[1], Antonio Ruggiero[2], Cesare Colosimo[3], Maria Chiara Stefanini[1], Massimo Caldarelli[4], Riccardo Riccardi[2]

[1] *Don Gnocchi Foundation, Milan, Italy*

[2] *Division of Pediatric Oncology, Catholic University of Rome, Italy*

[3] *Department of Bioimaging and Radiological Sciences, Catholic University, Rome, Italy*

[4] *Pediatric Neurosurgery Unit, Catholic University of Rome, Italy*

Introduction: Primary thalamic gliomas include a distinct type known as bilateral thalamic glioma, which occurs as a large tumor located in symmetrical areas of both thalami.
Behavioural impairments ranging from personality changes, confusion, memory loss, apathy, emotional instability and dementia are described as typical manifestations of these tumors.
Treatment of BTs is not well defined and prognosis is poor, with only 7.6% of all patients surviving for more 12 months.
The focus is on the possible role of neurocognitive decay in predicting tumor enlargement and neuroimaging signs of malignancy.

Methods: The clinical, neuropsychological and neuroimaging evolution of a 12-year–old boy (CG) who presented bilateral thalamic astrocytoma (WHO grade 2) was examined. CG underwent an extensive neuropsychological assessment immediately after biopsy, prior to any medical therapies and was followed up for 3 years until death. Neuropsychological functioning was thoroughly investigated, by means of a detailed battery which included intelligence and cognitive functions.

Results: NA at diagnosis did not reveal cognitive impairment or neuropsychological focal signs.
Evolution was characterized by cognitive deterioration that preceded neuroimaging signs of tumor progression. Starting from normal cognitive organization, the child exhibited visuospatial memory deficits and afterwards diffuse cognitive impairment.
Cognitive testing has been found to have predictive value in numerous neurological disorders, including brain tumors, multiple sclerosis and dementia-related illnesses. Changes in neurological disease status can be identified through variations in cognitive performance, arguably correlating with the degree of disease severity. Interestingly, the onset of cognitive decline in our patient starts before tumor progression as evidenced by cranial MRI at a later stage.

Conclusions: The monitoring of CF in our patient revealed cognitive decline, with progressive decrement of location-related functions. Cognitive deterioration is a typical manifestation of progressive thalamic pathologies and can detect early disease progression.