and suggestive symptoms are associated with NM, we found very poor Sn/Sp for elevated protein (61%/61%), low glucose (35%/85%), elevated lactate (35%/91%), and symptoms consistent with NM (74%/73%). Of these tests at least an elevated lactate and symptoms were predictive of a positive CSF cytology. No variable predicted a negative cytology, and 22.8% of patients with a positive CSF cytology had completely normal CSF findings. CONCLUSION: Generally accepted concepts about the frequency of CSF and clinical findings in patients with NM are based on biased estimates in the literature. No CSF chemistry or clinical finding reliably predicts the presence of NM, and almost a quarter of patients with cytologically proven NM have completely normal findings. In patients with positive CSF cytologies, about 73% are detected after one LP, and more than 90% after 3, but again these findings represent overestimates because of pervasive spectrum bias and differences in sample acquisition/handling. Better diagnostic techniques are desperately needed.

**POSTER PRESENTATIONS**

**P01 COGNITION IN BRAIN TUMORS**

**P01.01.A. LESION-FUNCTION ANALYSIS FROM MULTIMODAL IMAGING AND NORMATIVE BRAIN ATLASES FOR PREDICTION OF COGNITIVE DEFICITS IN GLIOMA PATIENTS**

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BACKGROUND: Cognitive deficits are common in glioma patients following multimodality therapy, but the relative impact of different types and locations of treatment-related brain damage and recurrent tumors on cognition is not well understood. MATERIAL AND METHODS: In 121 WHO Grade III/IV glioma patients, structural MRI, O-2-[18F]fluoroethyl)-L-tyrosine PET, and neuropsychological testing were performed at a median interval of 14 months (range, 1-214 months) after therapy initiation. Resection cavities, T1-enhancing lesions, T2/FLAIR hyperintensities, and FET-PET positive tumor sites were semiautomatically segmented and dynamically registered to a normative resting state (RS) fMRI-based functional cortical network atlas and to the JHU atlas of white matter (WM) tracts, and their influence on cognitive test scores relative to a cohort of matched healthy subjects was assessed. RESULTS: T2/FLAIR hyperintensities presumably caused by radiation therapy covered more extensive brain areas than the other lesion types and significantly impaired cognitive performance in many domains when affecting left-hemispheric RS-nodes and WM-tracts as opposed to brain tissue damage caused by resection or recurrent tumors. Verbal episodic memory proved to be especially vulnerable to T2/FLAIR, but it is not clear whether these findings affect the nodes in the temporal lobes. CONCLUSION: In order to improve radiotherapy planning, publicly available brain atlases, in conjunction with elastic registration techniques, should be used, similar to neuronavigation in neurosurgery.

**P01.02.B. CASE REPORT: DISRUPTION OF RESTING-STATE NETWORKS AND COGNITIVE DEFICITS AFTER WHOLE BRAIN IRRADIATION FOR SINGULAR BRAIN METASTASIS**

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BACKGROUND: Long-term survivors of whole brain radiation (WBRT) are at significant risk for developing cognitive deficits, but knowledge about the underlying pathophysiological mechanisms is limited. Therefore, we here report a rare case with a singular brain metastasis treated by resection and WBRT that survived for more than 10 years where we investigated the integrity of brain networks using resting-state functional MRI. MATERIAL AND METHODS: A female patient with a left frontal non-small cell lung cancer (NSCLC) brain metastasis had resection and postoperative WBRT (30.0 in 3.0Gy fractions) and stayed free from brain metastasis recurrence for 11 years. Structural MRI and amino acid [O-2-[18F]fluoroethyl)-L-tyrosine] positron emission tomography (PET) were repeatedly acquired. At the last follow up, neurocognitive functions and resting-state functional connectivity (RSFC) using resting-state fMRI were assessed. Within-network and inter-network connectivity of seven resting-state networks were computed from a connectivity matrix. All measures were compared to a matched group of 10 female healthy subjects. RESULTS: At the 11-year follow-up, T2/FLAIR MR images of the patient showed extended regions of hyper-intensities covering mainly the bilateral dorsal frontal and posterior regions, while sparing most of the temporal lobes. Compared to the healthy subjects, the patient performed significantly worse in all cognitive domains that included executive functions, attention and processing speed, while verbal memory, learning and memory, and visuospatial ability were left mostly unaffected. The connectivity matrix showed a heavily disturbed pattern with a widely distributed, scattered loss of RSFC. The within-network RSFC revealed a significant loss of connectivity within all seven networks where the dorsal attention and frontoparietal control networks were affected most severely. The inter-network RSFC was significantly reduced for the visual, somato-motor, and dorsal and ventral attention networks. CONCLUSION: As demonstrated here in a patient with a metastatic NSCLC and long-term survival, WBRT may lead to extensive white matter damage and cause severe disruption of the RSFC in multiple restate networks. In consequence, executive functioning which is assumed to depend on the interaction of several networks may be severely impaired following WBRT apart from the well-recognized deficits in memory function.