BACKGROUND: Deficits in neurocognitive functioning (NCF) frequently occur in glioma patients. Both treatment and the tumor itself contribute to these deficits. In order to minimize the harmful effects of surgery, it is becoming more common that patients are operated under awake conditions. To investigate if we can indeed preserve cognitive functioning after state-of-the art awake surgery and to identify factors determining the course of NCF after awake surgery, we performed a retrospective cohort study. METHODS: From our consecutive cohort of patients with a diffuse glioma (WHO grade II-IV), we included patients with a clinical indication for awake craniotomy and sufficient data from neuropsychological testing, both pre-operatively and 3–6 months post-operatively. Evaluation covered five neurocognitive domains. We compared pre- and postoperative NCF data for mean scores per domain, and for the percentage of patients that deteriorated after surgery (individual level). We evaluated the value of patient-, tumor- and treatment characteristics for predicting change in NCF, using linear and logistic regression analyses. RESULTS: A total of 163 patients met our inclusion criteria. Mean NCF-scores of psychomotor speed and visuospatial functioning significantly deteriorated after surgery. The percentage of serious neurocognitive impairments (-2SD or worse) increased postoperatively for psychomotor speed, but not for other domains. Extension of the glioma in the left thalamus predicted a decline in the domains of overall-NCF, executive functioning and psychomotor speed. Absence of an IDH-mutation also predicted cognitive decline, for overall-NCF and executive functioning. CONCLUSIONS: In the majority of cognitive domains, cognitive functioning is preserved after surgery. Psychomotor speed seems to be most vulnerable to the effects of surgery. The predictive value of thalamic involvements and IDH-status for cognitive changes after awake surgery support the idea that a combination of locoregional ("mechanical") and biomolecular effects of the tumor determine cognitive performance.

P01.147 RECURRENT GLIOBLASTOMA OR THERAPY-RELATED CHANGES: THE DIAGNOSTIC ACCURACY OF O-(2-[18F]-FLUOROETHYL)-L-TYROSINE PET IMAGING

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BACKGROUND: Discrimination between recurrent glioblastoma and therapy-related changes with conventional magnetic resonance imaging (MRI) is challenging. There is increasing evidence for the clinical value of positron emission tomography (PET) with O-(2-18F-fluoroethyl)-L-tyrosine (FET) in addition to MRI in cases of ambiguous findings in the setting of posttreatment care. The diagnostic accuracy, however, varies among the published studies and is influenced by the composition of the population with different glioma subtypes/grades, lack of histological confirmation, and differences in data processing. Herein, we evaluated the diagnostic accuracy of FET PET scans in glioblastoma patients. MATERIAL AND METHODS: One hundred thirty-seven consecutive patients who had undergone 150 FET PET were reviewed retrospectively. Inclusion criteria were 1) histologically-proven glioblastoma; 2) previous surgery followed by oncological treatment consisting of standard radiochemotherapy or - if at second recurrence - chemotherapy; 3) unexplained constant or increasing contrast-enhancing (CE) lesions on T1, or non-CE lesions on T2 FLAIR MRI later than 6 months after radiotherapy, where the differentiation between disease recurrence or therapy-related changes was uncertain; 4) FET PET for supplementary evaluation; 5) a histological evaluation following surgery less than 3 months after FET PET, or MRI follow-up. FET PET scans were performed as 20-minute static PET/CT acquisitions and evaluated co-registered to T1 post-contrast MRI with measurement of maximum and mean tumor-to-brain ratios ($TBR_{max,}$ TBR_{mean}). Receiver operating characteristics (ROC) analysis was used to determine the optimal threshold of FET parameters. The prognostic influence of FET parameters on overall survival (OS) was investigated with the Cox proportional hazards model. RESULTS: The median time interval from radiotherapy until the radiological progression was 13 months. One hundred twenty-six PET scans demonstrated FET uptake of varying intensity. Surgical interventions were performed following 88 PET scans, while 62 PET scans were evaluated by clinical or MRI follow-up, resulting in 131 glioblastoma recurrences and 19 therapy-related changes. ROC analysis yielded the thresholds of 2.0 for TBR_{max} and 1.8 for TBR_{mean} for differentiation between recurrent disease and therapy-related changes with the best performance of TBR_{max} with a sensitivity of 96% and a specificity of 100% ($p < 0.0001$). Using this threshold, 145 of 150 PET scans of glioblastoma recurrence or therapy-related changes were accurately classified. Multivariate survival analysis showed that $TBR_{max} > 2.0$ predicted independently a shorter OS in patients, who did not undergo subsequent therapy following PET ($p = 0.002$). CONCLUSION: FET PET is a powerful noninvasive tool to distinguish recurrent glioblastoma from therapy-related changes.

P01.148 INTRA-TUMOR HETEROGENEITY ANALYSIS OF LOW-GRADE GLIOMAS. A POLA NETWORK STUDY

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BACKGROUND: Low-grade gliomas (LGG) are divided into three histomolecular groups: i) IDHwt, ii) IDHmut and 1p19q intact and iii) 1p19q co-deleted with IDH1 mutation. The current classification has improved the clinical stratification and its reproducibility. However, LGGs are still associated with an important degree of clinical heterogeneity.We sough to analyze the genomic and genetic heterogeneity by re-analyzing next-generation sequencing data of two different cohorts of LGG to dissect the potential role of clonal architecture on the clinical evolution of LGG. MATERIAL AND METHODS: We have re-analyzed whole-exome sequencing (WES) using LGG TCGA dataset and a cohort of 40 LGG analyzed by WES from the POLA Network. We have obtained the clonal architecture, the mutation burden, the number of clones, the clonal and subclonal distribution of genetic and genomic alterations and the distribution of the somatic signatures. In addition, we have also analyzed 5 1p19q co-deleted samples at different timepoints to further dissect the clonal evolution of this entity. RESULTS: As expected, the mutation burden of LGG is low (median somatic mutation per sample 34). Surprisingly, the number the subclones was higher in the 1p19q co-deleted subgroup.LGG were enriched in somatic signatures associated with hydrolytic deamination of methylated CpG and with deficiency of mismatch repair. Interestingly, only within 1p19q co-deleted gliomas, the number of subclones, the mutant-allele tumor heterogeneity (MATH) score and the signature associated with hydrolytic deamination of methylated CpGwere associated with poorer outcome in univariate analysis. Finally, only the enrichment of the mutational signature associated with hydrolytic deamination of methylated CpG had prognostic impact in 1p19q co-deleted LGG in Cox multivariate analysis afteradjusting by age, mutational clones number, MATH score and loss of CDKN2A loss. CONCLUSION: The clonal architecture of LGG may provide additional clinical relevant data within 1p19q co-deleted tumors. The enrichment of a somatic signature associated with hydrolytic deamination of methylated CpG is independently associated with poor overall survival.

P01.149 LOW-GRADE GLIOMA POST-RADIATION THERAPY SURVEILLANCE WITH ADVANCED IMAGING TECHNIQUES

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BACKGROUND: Optimal sequential strategy in LGG patients is complex: beyond the role of each individual treatment surgery (S); radiation therapy (RT); chemotherapy (CT), second effects can potentiate the effect of each treatment. With late toxicities concerns as median survival range 5 to 15 years with better prognosis and treatment response for pts with 1p/19q codeletion and IDH1 mutation. Symptoms mainly decreased seizures and magnetic resonance (MRI) evaluation is part of clinical follow-up.Using advanced MRI techniques such as Perfusion can help distinguish pseudoprogression (PP) from true progression (TP), which is a major concern since PP in LLG can occur up to 20%. MATERIAL AND METHODS: Review in Mosaiq® and PACS imaging database, adults with primary CNS tumors. Between January 2007 and December 2017, primary CNS tumors 947 pts with were treated in our radiation therapy department. According to 2016 WHO CNS classification 22% LGG. MRI imaging follow-up was assessed 1–3 months after RT. The protocol included T2FLAIR 3D, T13D before and after gadolinium and perfusion weighted-imaging T2* (which measures cerebral blood volume -CBV). RESULTS: We present 7 pts with LGG diagnosis who underwent surgery/biopsy, molecular features were 57% codeletion 1p19q and 72% IDH1 mutation. MRI alterations observed were increased T2FLAIR and 57% pts enhancement in T1 after gadolinium both in PP and TP but in one with PP the CBV was reduced whereas in the other patients with TP it was increased. CONCLUSION: MRI is very useful in the follow up of LGG and advanced techniques such spectroscopy and perfusion-weighted help distinguish PP from TP.

P01.150 HYPERMUTATIONS IN GLIOBLASTOMA ARE ASSOCIATED WITH INCREASED RESPONSE TO IMMUNOTHERAPY

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