both cognitive function and mood symptoms are important to consider in optimizing functioning, but depression appears to vastly outweigh cognitive function in this regard. These preliminary findings highlight the importance of careful attention to these symptoms in survivorship and point to future research directions elaborating on these relationships.

NCOG-11. NEUROPSYCHOLOGICAL OUTCOMES FOLLOWING AWAKE CRANIOTOMY FOR LOW GRADE GLIOMA

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BACKGROUND: Awake craniotomy is an established treatment for tumours in eloquent brain regions. It aims to achieve maximal resection without significant neurological and functional morbidity. There is an emerging evidence base about the neuropsychological outcomes following awake craniotomy for low grade gliomas (LGGs) with inconsistent findings potentially reflective of methodological confounders such as the inclusion of mixed tumour types and the use of varied neurocognitive measures and timescales for repeat testing. We describe detailed neuropsychological outcomes for a cohort of patients following awake craniotomy for LGGs. METHOD: Data were collected from patients undergoing first-time awake resection of presumed LGGs at Leeds Teaching Hospitals, UK between 2010 and 2018. Patients were included if they consented to and completed the full battery of pre- and postoperative neuropsychological testing. Neuropsychometric testing involved assessment of general intellectual functioning, language, memory, executive functioning, perception, processing speed and mood. Data were analysed using paired-samples t-test or Wilcoxon signed-rank test with Bonferroni correction for multiple statistical comparisons. RESULTS: Twenty-two patients undergoing awake craniotomy for presumed supratentorial LGG (13 left hemisphere, 9 right hemisphere; mean age 35 years) met the above inclusion criteria. Tumour location was heterogenous (11 insular, 9 frontal, 1 temporal, 1 parietal). Histopathology confirmed WHO Grade II diffuse astrocytoma (n=11), WHO Grade II oligodendroglioma (n=10) and WHO Grade III anaplastic oligodendroglioma (n=1). No statistical differences in neurocognitive test scores were found pre- and post-neurosurgery (mean follow-up was 61 weeks). Other outcomes including extent of resection and reliable change statistics for neurocognitive tests and measures of mood were also analysed. CONCLU-SION: No significant change in neurocognitive functioning was found in patients following recovery from awake craniotomy for LGG. Our findings suggest that awake craniotomy is a safe treatment for LGG and that neuropsychological input is an important part of the treatment pathway for patients with LGG.

NEURO-IMAGING

NIMG-01. T2WI-FLAIR MISMATCH SIGN IN LOWER GRADE GLIOMA AND DYSEMBRYOPLASTIC NEUROEPITHELIAL TUMOR Shumpei Onishi¹, Fumiyuki Yamasaki², Motoki Takano², Ushio Yonezawa², Akira Taguchi², Kazuhiko Sugiyama², and Kaoru Kurisu², ¹National Hospital Organization Kure Medical Center and Chugoku Cancer Center, Kure, Hiroshima, Japan, ²Hiroshima University Hospital, Hiroshima, Japan

BACKGROUND: T2-FLAIR mismatch sign was reported as a specific imaging marker for diffuse astrocytoma with IDH-mutant and 1p/19q noncodeletion. However, most of the previous studies for T2-FLAIR mismatch were confirmed only among lower grade glioma (LGG). The purpose of this study is to explore the T2-FLAIR mismatch sing in supratentorial nonenhancing tumor including LGG and dysembryoplastic neuroepithelial tumor (DNET) and to unveil the exception rules of the sign. METHODS: Fortyfour patients of non-enhancing LGG and DNET were included in this study. LGG (diffuse astrocytoma with IDH mutant (IDHmut-Noncodel), oligodendroglioma with IDH-mutant and 1p19q codeletion (IDHmut-Codel), diffuse astrocytoma with IDH wildtype (IDHwt)) and DNET were diagnosed based on WHO 2016 classification. The tumors were evaluated MRI by 2 independent reviewers to assess presence or absence of T2-FLAIR mismatch sign. CT was also performed to evaluate the localized thinning of the skull bone. Inter-reviewer agreement with Cohen's kappa (κ) was calculated. RESULT: Ten out of 18 cases (55.6%) of IDHmut-Noncodel presented T2-FLAIR mismatch sign. None of the other LGG (IDHmut-Codel and IDHwt) presented T2-FLAIR mismatch. Eight out of 11 cases (72.7%) of DNET also present the T2-FLAIR mismatch. The overlying part of the skull bone thinning was observed in 5 cases of DNET, but none of LGG presented the localized skull bone thinning. The inter-rater agreement for

the T2-FLAIR mismatch and the localized thinning of the skull bone were excellent (κ = 1.00). CONCLUSION: The T2-FLAIR mismatch sign was specific marker for IDHmut-Noncodel among LGG. However, DNET also presented the T2-FLAIR mismatch sign. The localized skull bone thinning could be useful for differentiating between IDHmut-Noncodel and DNET

NIMG-02. NON-INVASIVE DETECTION OF IDH MUTANT 1p19q NON-CODELETED GLIOMAS USING THE T2-FLAIR MISMATCH SIGN

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OBJECTIVE: To assess the validity and pathophysiology of the T2/ FLAIR mismatch sign for non-invasive identification of IDH-mutant 1p/19q non-codeleted glioma. METHODS: MRI scans from 408 consecutive patients with newly diagnosed glioma (113 lower-grade glioma and 295 glioblastoma) were evaluated for the presence of a T2/FLAIR-mismatch sign (defined as complete/near-complete hyperintense signal on T2w, simultaneous hypointense signal on FLAIR except for a hyperintense peripheral rim) by two independent reviewers. Sensitivity, specificity, accuracy, positive and negative predictive value (PPV, NPV) were calculated to assess the performance of the T2/FLAIR-mismatch sign for identifying IDH-mutant 1p/19q non-codeleted tumors. An exploratory analysis of spatial differences in ADC and rCBV values comparing the FLAIR-hypointense core vs. hyperintense rim in cases with presence of a T2/FLAIR-mismatch sign was performed. RESULTS: There was substantial interrater agreement to identify the T2/FLAIR-mismatch sign (Cohen's Kappa = 0.75 [95% CI 0.57-0.93]). The T2/FLAIR-mismatch sign was present in 12 cases with lower-grade glioma (10.6%), all of them were IDH-mutant, 1p/19q non-codeleted tumors (sensitivity=10.9%, specificity=100%, PPV=100%, NPV=3.0%, accuracy=13.3%). The T2/FLAIR-mismatch sign was not identified in any other molecular subgroup, especially not in any of the IDH-mutant glioblastoma cases (n=5). In tumors with T2/FLAIR-mismatch sign the ADC values were significantly lower in the rim as compared to the core (p=0.0005) whereas there was no difference in rCBV values (p=0.4258). CONCLU-SION: This study confirms the high specificity of the T2/FLAIR-mismatch sign for non-invasive identification of IDH-mutant 1p/19q non-codeleted gliomas, although sensitivity is low and applicability is limited to lower-grade gliomas. The identified spatial differences in ADC values between the core and rim of tumors with a T2/FLAIR-mismatch sign potentially reflects differences in tumor cellularity and microenvironment.

NIMG-03. PROSPECTIVE PHASE II RANDOMIZED TRIAL COMPARING PROTON THERAPY VS. PHOTON IMRT FOR GBM: SECONDARY ANALYSIS COMPARISON OF PROGRESSION FREE SURVIVAL BETWEEN RANO VS. CLINICAL AND RADIOLOGICAL ASSESSMENT

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PURPOSE: To compare tumor progression based on clinical radiological assessment and on Response Assessment in Neuro-Oncology (RANO) criteria between GBM patients treated with proton radiotherapy (PT) vs. photon intensity modulated radiotherapy (IMRT). METHODS: Eligible patients were enrolled on the described prospective phase II trial and had MR imaging at baseline and follow-up beyond 12 weeks from treatment completion. 'Clinical' progression was based on a radiology report of progression in combination with changes in treatment due to suspected disease progression. A single blinded observer applied RANO criteria to determine the RANO-based tumor progression. RESULTS: Of 90 enrolled patients, 66 were evaluable, with median follow-up of 19.8 (Range: 3.2–65.1) months; median of 22.6 months for PT (n=25) vs. 18.9 months for IMRT