(≥70 years)/33 (≤50 years) cases of Kansai Network and 88 (≥70 years)/69 (≤50 years) cases of TCGA. Median overall survival was 12.8 (≥70 years)/ 21.0 (≤50 years) months in Kansai Network cohort and 8.8 (≥70 years)/ 21.09 (≤50 years) months in TCGA cohort. MGMT promoter was methylated in 50 (54.3%) (≥70 years)/14 (42.4%) (≤50 years) tumors in Kansai Network and 34 (48.6%) (≥70 years)/16 (36.4%) (≤50 years) tumors in TCGA. TERT promoter was mutated in 51 (55.4%) (≥70 years)/13 (39.4%) (≤50 years) tumors in Kansai Network and unknown in TCGA. Significant difference of CNA profiles between ≥70 years and ≤50 years was as follows: PTEN del, 43 (46.7%)/8 (24.2%); CDK4 amp, 17 (18.5%)/1 (3.0%) in Kansai Network and CDKN2A del, 69 (78.4%)/ 42 (60.9%) in TCGA. CONCLUSION: Elderly patients have several potential factors for poor prognosis and different molecular profiles might explain the survival differences among generations.

MPC-05

TUMOR RELATED EPILEPSY AND IDH MUTATIONS IN GLIOMAS

Hikaru Wakabayashi[1], Motoki Inaji[1], Shoko Fujii[1], Jiro Aoyama[1], Satoka Hashimoto[1], Kaoru Tamura[1], Taketoshi Maehara[1]; [1]The Department of Functional Neurosurgery, Tokyo Medical and Dental University, Tokyo, Japan

OBJECTIVE: Tumor related epilepsy (TRE) is an important complication in the treatment of brain tumors. In recent studies, it is assumed that isocitrate dehydrogenase (IDH) mutations are concerned with TRE in gliomas. Here, we examined the association between IDH mutations and TRE in our cases. METHODS: 115 patients who had a supratentorial glioma and were treated in our hospital from February 2009 to November 2018 were retrospectively assessed for IDH mutations and TRE. RESULTS: 38 patients were the IDH mutant group (16 females, mean age 43.7±12.9 years, mean follow-up time 44.0 months). 77 patients were the IDH wild group (35 females, mean age 61.6±16.6 year, mean follow-up time 18.1 months). Compared to the IDH wild group, the IDH mutant group was significantly younger and mean follow-up time was longer. There was no difference in the postoperative radiation and chemotherapy in both groups. The incidence of seizures as presenting symptom was 20 patients (52.6%) in the IDH mutant group and 16 patients (20.8%) in the IDH wild group, and was significantly higher in the IDH mutant group (p<0.01). 27 patients (71.1%) in the IDH mutant group had TRE at least once during follow-up time and 39 patients (50.0%) in the IDH wild group (p=0.06). In addition, the median OS for the group with seizure onset (36 patients) was 69.2 months and the group with the other onset forms (79 patients) was 22.4 months. The seizure onset group had a significantly better prognosis (p<0.05). CONCLUSION: Gliomas with IDH mutations have a higher incidence of TRE. Although IDH mutations are considered to be a risk factor for TRE, which is consistent with previous studies, but it is suggested that differences in survival may have an effect on the incidence of TRE.

MPC-06

LRG1 HAS MULTIPLE POTENTIAL FOR CLINICOPATHOLOGICAL BIOMARKER OF GLIOBLASTOMA

Takuya Furuta[1], Yasuo Sugita, Satoru Komaki, Kouichi Ohshima[1], Motohiro Morioka, Yasuo Uchida, Masanori Tachikawa, Sumio Otsuki, Tetsuya Terasaki, Mitsutoshi Nakada; [1]Department of Pathology, Kurume University, Fukuoka, Japan

BACKGROUND AND AIM: Leucine-rich α-2 glycoprotein 1 (LRG1) is one of the candidate proteins as a diagnostic marker for glioblastoma. Although association with angiogenesis has been reported, it has been suggested that the role as a biomarker differs depending on the tumor types. The role of LRG1 as a biomarker in glioblastoma was examined clinicopathologically. METHODS: Tumor tissues of 156 cases diagnosed as diffuse glioma (27 astrocytomas, 15 oligodendrogliomas, 114 glioblastomas) according to WHO 2016 classification at Kurume University from January 2001 to April 2019 were used. The immunohistochemical intensity of LRG1 was scoring as 4 stages and classified into 2 groups; score 0–1 was defined as low expression and score 2–3 was defined as high expression. Mutations of IDH1/2 and TERT promoter were analyzed by Sanger method. In glioblastoma, the relationship between LRG1 expression and clinical parameters such as age, preoperative Karnofsky Performance Scale, tumor location, extent of resection, MGMT promoter, and prognosis were examined. RESULTS: LRG1 high expression rate was 41.2% (47/114) in glioblastoma, 3.7% (1/27) in astrocytoma, 20% (3/15) in oligodendroglioma, and glioblastoma showed significant higher expression level of LRG1 compared with lower-grade glioma (p = 0.0003). High expression of LRG1 was an independent favorable prognostic factor (HR 0.41, 95% CI 0.18–0.86, p=0.019) in IDH-wildtype glioblastoma, and correlated with gross total resection (p = 0.002) and the tumor location of the non-subventricular zone (SVZ) (p = 0.00007). CONCLUSION: LRG1 demonstrated multiple potential as diagnostic, prognostic, and regional biomarker for glioblastoma.

MPC-07

MECHANISMS OF BETTER PROGNOSIS IN IDH-MUTATED ASTROCYTOMA WITH 19Q-LOSS

Ryohei Otani[1], Takeo Uzuka, Fumi Higuchi, Hadzki Matsuda, Phyo Kim, Keisuke Ueki; [1]Department of Neurosurgery, Tokyo Metropolitan Komagome Hospital

We previously reported that there was a subgroup of IDH-mutated astrocytomas harboring only 19q-loss showing oligodendroglioma-like morphology and significantly longer overall survival (OS) compared with 19q-intact astrocytomas (Otani et al. Cancer Sci 2018). The purpose of the present study was to reveal how 19q-loss contributed to better prognosis and the morphology in the subgroup. We compared expression pattern between five 19q-loss and five 19q-intact IDH-mutated astrocytomas by microarray analysis.

136 up-regulated genes and 203 down regulated genes were extracted in 19q-loss astrocytomas compared with 19q-intact astrocytomas. Significantly changed genes distributed throughout all chromosomes, but more down-regulated genes were on 19q and 4p, and more up-regulated genes were on 4q.

Genes associated with apoptosis, cell adhesion, and antigen presentation were up-regulated, and genes associated with Ras signaling pathway were down-regulated. These changes could result in better prognosis. By contrast, there was few expression changed gene associated with oligodendroglioma-like morphology although up-regulation of genes associated with axon guidance and down-regulation of genes associated with cell shape might result in the morphology or neuronal differentiation. Expression pattern of 19q-loss astrocytomas indicated no tendency of oligodendroglial differentiation.

Better prognosis of 19q-loss astrocytomas was derived from expression changes associated with tumor proliferation and tumor immunity.

MPC-08

CLINICOPATHOLOGICAL ANALYSIS OF 12P GAIN IN INTRACRANIAL GERM CELL TUMORS

Kaishi Satomi[1], Hirokazu Takami, Shintaro Fukushima, Yoichi Nakazato, Shota Tanaka, Nobuhito Saito, Masayuki Kanamori, Toshihiro Kumabe, Keiichi Kobayashi, Motoo Nagane, Toshihiko Iuchi, Koji Yoshimoto, Kaoru Tamura, Taketoshi Maehara, Keiichi Sakai, Kazuhiko Sugiyama, Kiyotaka Yokogami, Hideo Takeshima, Masahiro Nonaka, Akio Asai, Ryo Nishikawa, Masao Matsutani, Koichi Ichimura; [1]Department of Diagnostic Pathology, National Cancer Center Hospital, Tokyo, Japan

BACKGROUND: Gain of short arm of chromosome 12 (12p) is commonly observed in testicular germ cell tumors (tGCTs). 12p gain is also frequently seen in intracranial GCTs (iGCTs). However, little is known about the clinical significance of 12p gain in iGCTs. MATERIALS AND METHODS: We have collected over 200 fresh frozen tissue samples of iGCTs through the Intracranial Germ Cell Tumor Genome Analysis Consortium in Japan. Firstly, we analyzed DNA methylation status in 83 iGCTs, 3 seminomas and 6 normal control samples using Infinium Human Methylation 450K BeadChip array (Illumina, CA). Idat files were processed using R (Version 3.5.3) and minfi package (1.30.0) to generate copy number variations. Compared with average genome-wide copy number level, 12p gain was determined. Then, 58 iGCTs with clinicopathological information were analyzed for progression-free survival (PFS) and overall survival (OS). Those tumors that consist of only either germinoma and/or mature teratoma components were classified as Favorable Histology (FH) and all the others that contains malignant histological components were classified as Unfavorable Histology (UFH). RESULT: 12p gain was observed in 100% (3/3) of seminoma, 13.6% (3/22) of germinoma, 16.7% (1/6) of mature teratoma, 25% (1/4) of immature teratoma, 55% (11/20) of mixed germ cell tumor, 100% (4/4) of yolk sac tumor, 100% (1/1) of embryonal carcinoma, and 100% (1/1) of choriocarcinoma. In total, 44.6% (37/83) of iGCT showed 12p gain. Regarding histological classification, the 12p gain rate in UFH (72%, 18/25) was significantly higher than that in FH (12.1%, 4/33, P<0.01). Both PFS and OS were significantly worse in iGCTs with 12p gain (PFS: P=0.027, OS: P=0.0012). DISCUSSION: 12p gain can be a molecular marker to predict prognosis and histological malignancy in iGCTs.

MPC-09

THE OPTIMIZATION OF TREATMENTS FOR SO-CALLED PRIMITIVE NEUROECTODERMAL TUMORS WITH MOLECULAR ANALYSIS

Mario Suzuki[1], Naohide Fujita[1], Ikuko Ogino[1], Junya Fujimura, Akihide Kondo[1]; [1]Department of Neurosurgery, Juntendo University School of Medicine, Tokyo, Japan

INTRODUCTION: In the previous WHO classification of central nervous tumors, the supratentorial tumors comprise small round blue cells with aggressive clinical features had been defined as primitive neuroectodermal tumor (PNET). Recent molecular analysis revealed that they do not belong to a single entity, but they are re-classified as the tumors of other well-defined tumors and newly defined tumor species. These facts were reflected to the new classifications. While, there are few studies those showed the