molecular prognostic factors. In the elderly group, however, there was no significant difference in OS according to MGMT status (methylated = 18.7 vs. unmethylated = 17.1, p = 0.3885) or triple CNA status (triple = 13.6 vs. non-triple = 19.6, p = 0.1734). On the other hand, statistical difference was observed according to NFKBIA status (del = 12.1 vs. non-del = 18.7, p = 0.0157*) even in the elderly cases. CONCLUSION: Prognostic effects of molecular factors might be attenuated in the elderly patients. Further investigation in a larger population is necessary.

MPC-04

UTILITY OF COMPREHENSIVE CANCER GENOME ANALYSIS FOR BRAIN TUMORS

Kazuhiro Miwa[1], Hiroaki Takei[1], Takeshi Ito[1], Kazutoshi Yokoyama[1], Hirohito Yano[2], Jun Shinoda[2], Satoshi Ishihara[3], Noriyuki Nakayama[4], Toru Iwama[4], Yasutka Kato[5]; [1]Department of Neurosurgery, Kizawa Memorial Hospital, Minokamo, Japan

OBJECTIVE: Our hospital has been designated as a cancer genome medical cooperation hospital, and it is our responsibility to play a central role in cancer medicine. We were one of the first local hospitals to clinically apply cancer genome analysis, and in January 2019, we started PleSsision-Rapid testing as a clinical study without patient burden. This study examines data from patients with brain tumors, subjects it to cancer genome analysis, and reports on its utility and efficacy.

METHOD: Genome analysis was performed by PleSsision-Rapid examination for patients with brain tumors who underwent surgery between January 2019 and July 2020. Tissue DNA extracted from pathological specimens was used to perform next-generation sequencing (NGS) analysis. In the PleSsision-Rapid test, 160 genes are comprehensively analyzed, examined by genomics, and evaluated for the presence or absence of actionable and druggable mutations, and the mutation rate is determined.

RESULTS: There were 15 cases total. Histopathological diagnoses included glioblastoma (n=5), diffuse astrocytoma (n=1), metastatic brain tumor (n=4), meningioma (n=2), central nervous system primary malignant lymphoma (n=1), germinoma (n=1), and Langerhans cell histiocytosis (n=1). Of these 15 brain tumor cases, actionable mutations were detected in 80.0% of cases and druggable mutations were detected in 66.6%. The average mutation rate was 8.59±5.32 (range, 1.3 to 22.8) per patient.

Conclusion: Although future improvements will be needed for cancer genome analysis in brain tumors, this strategy may be useful for the selection of molecularly targeted drugs with high antitumor efficacy. We will continue to accumulate and study such cases in the future.

MPC-06

CUTTING-EDGE OF CANCER GENOMIC MEDICINE FOR BRAIN TUMORS

Masahiro Mizoguchi[1], Nobuhiro Hata[1], Daisuke Kuga[1], Ryusuke Hatae[1], Yuhei Sangatsuda[1], Yutaka Fujioka[1], Kosuke Takigawa[1], Yusuke Funakoshi[1], Yuhki Koga[2]; [1]Department of Neurosurgery, Graduate School of Medical Sciences, Kyushu University, Japan

Kyushu University Hospital was designated a Cancer Genome Core Hospital in April 2018, and the multi-gene panel test has been introduced since August 2019. The expert panel has been held for 21 cases of the central nervous system (11 adult glioma, 5 pediatric brain tumors, 5 extramedullary tumors). Actionable gene abnormalities were newly detected in two cases. First case is epithelioid glioblastoma with BRAF V600E mutation, and second is embryonal tumor with VCL-ALK fusion. For the first case, BRAF/MEK inhibitor can be used by the prospective trial of patient-proposed healthcare services with multiple targeted agent based on the result of gene profiling by multigene panel test (NCCH1901). For the second case, we are planning to introduce ALK inhibitor by indicator-initiated clinical trial while continuing ICE therapy. The current approved agents for tumor-agnostic treatment are immune checkpoint inhibitors for mismatch repair deficient (dMMR) cases and TRK inhibitors for NTRK fusion gene-positive cases. We selected microsatellite instability (MSI) test and immunostaining of MMR gene for the indication of immune checkpoint inhibitor for recurrent glioma and Lynch syndrome that require dMMR evaluation, but FoundationOne CDx (F1CDx) allows simultaneous evaluation of MSI and MMR gene abnormalities. Regarding the indication of TRK inhibitors, F1CDx assay is selected as a companion diagnosis for ALK, NTRK1/2/3 fusion gene analysis for pediatric cases. At present, the actionable gene abnormalities are detected by multi-gene panel tests in about 10% of brain tumors. Development of tumor-agnostic treatment will expand the molecular target therapy for brain tumor in the future. Based on the experience of different schemes for molecular targeted therapy, it became clear that it is necessary to establish a cancer genome medical system for prompt introduction of precision medicine for highly malignant brain tumors.

MPC-08

MOLECULAR RISK STRATIFICATION USING GENOME-WIDE DNA METHYLATION DATA OF STANDARD-RISK MEDULLOBLASTOMAS TREATED WITH 18-GY CRANIOSPINAL IRRADIATION

Tomoya Irikura[1], Kohei Fukuoka[1], Makiko Mori[1], Koichi Oshima[1], Yuki Arakawa[1], Yutaka Tanami[2], Koichi Ichimura[3], Atsuko Nakazawa[4], Jun Kurihara[5], Katsuyoshi Koh[1]; [1]Department of Pediatric Hematology/Oncology, Saitama Children's Medical Center, Saitama, Japan

A novel risk stratification of medulloblastoma has been proposed based on retrospective data from patients undergoing standard treatment. However, it remains unclear whether the classification is applicable to patients receiving reduced-dose craniospinal irradiation (CSI). We performed molecular diagnosis and copy number analysis using methylation array on patients with standard-risk medulloblastoma treated with 18 Gy CSI at our institution. Nine tumor samples were available for analysis from seven patients who had a median age of 7.4 years at disease onset and a median observation period of 73 months. Three patients had recurrence, and another patient developed radiation-induced glioblastoma. From the three recurrent cases, one was molecularly diagnosed as SHH subtype with MYCN amplification; another case was a Group 4 tumor without favorable prognostic chromosomal aberrations, and the remaining patient experienced a very late relapse despite low-risk stratification. Of the recurrence-free cases, one was classified as WNT subtype, and another was a Group 4 tumor with chromosome 7 gain, and loss of chromosomes 8 and 11, both of which were associated with good prognosis. Methylation analysis also unveiled the fact that the recurrent tumor diagnosed as relapsing medulloblastoma by conventional diagnostic tools was in fact a radiation-induced glioblastoma. Our data suggested that the new risk stratification may be useful for cases treated with CSI reduced to 18 Gy. However, due to the presence of the late-relapsed case stratified to low risk, further investigations with a larger cohort should be required to confirm the data.

MPC-11

COMPREHENSIVE GENE EXPRESSION ANALYSIS OF IDH-MUTATED ASTROCYTOMAS WITH 19Q-LOSS

Ryohei Otani[1], Akitake Mukasa[4], Takeo Uzuka[2], Fumi Higuchi[2], Hadzki Matsuda[2], Shota Tanaka[3], Phyo Kim[2], Keisuke Ueki[2]; [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. To further explore the biological characteristics of this possible subgroup and obtain insight into the mechanism of their relatively benign clinical behavior, we compared gene expression pattern between five 19q-loss and five 19q-intact IDH-mutated astrocytomas by microarray analysis. By comparing expression levels of genes of 19q-loss astrocytomas to those of 19q-intact astrocytomas,136 up-regulated genes and 203 down-regulated genes were extracted. Down-regulated genes in the 19q-loss astrocytomas were heavily clustered to 19q and 4p, and up-regulated genes to 4q. It was noted that fibroblast growth factor 1 associated with stem cell maintenance was down-regulated in 19q-loss astrocytomas and genes associated with glioma progression were differentially expressed, these results were validated with the independent TCGA data set. On t-SNE analysis of the 19q-loss astrocytomas with other IDH-mutant glioma subgroups from the TCGA datasets, 19q-loss astrocytomas did not shift to oligodendrogliomas with 1p/19q codeletion but were a subgroup in astrocytomas. These results indicated that 19q-loss in astrocytomas is more likely to be an acquired event rather than early event in oncogenesis like 1p/19q codeletion in oligodendrogliomas, and the biological and morphological features of 19q-loss astrocytomas were possibly related to differentially expressed genes associated with stem cell maintenance and glioma progression.

NEUROIMAGING (NI)

NI-01

USEFULNESS OF PREOPERATIVE EVALUATION OF GLIOMA ELASTICITY BY THE MAGNETIC RESONANCE ELASTOGRAPHY

Shinichiro Koizumi[1], Kazuhiko Kurozumi[1]; [1]The Department of Neurosurgery, Hamamatsu University School of Medicine, Hamamatsu, Japan

INTRODUCTION: The elasticity of intracranial tumors is difficult to assess non-invasively because the lesion is surrounded by the skull. Therefore, intracranial tumors have not been verified before surgery in terms of elastic