to the heterogeneity of cell populations that could contribute to the TSPO-PET signal. We therefore dissected TSPO labelling in connection with the underlying histopathological and molecular features in biopsy samples from glioma patients. Comparing regions of high and low TSPO-PET signal in 58 patients, we assessed TSPO protein expression and expression of cell differentiation markers by use of immunohistochemistry on consecutive sections and by multiplex stains. Our results suggest that apart from microglia and macrophages the glial tumor cells relevantly contribute to the overall TSPO signal in glioma patients. To identify hallmarks and GO terms associated with TSPO labelling/expression we next performed RNAseq in 33 patients. Using DESeq2 followed by FUMA and Reactome as well as GSEA with normalized counts, we identified three TSPO-dependent functional clusters, i.e. apoptosis/DNA repair, extracellular matrix organization and immune system. On the DNA level, we analyzed the TSPO promoter by direct bisulfite sequencing suggesting that a loss of TSPO methylation may mechanistically contribute to the TSPO overexpression observed in high-grade gliomas. Exceeding our investigations on glioma biopsies we stained tissue microarrays (130 individuals) for TSPO that cover a broader spectrum of both human brain pathologies (13 different entities) and non-neoplastic tissues (14 different brain regions). We here found a high degree of heterogeneity of TSPO expression between entities and regions. Merging this information with TSPO-PETs from respective patients/brain regions allows to generate a map of TSPO expression in healthy and diseased brain for clinical use. Altogether, our approach of integrating histological, molecular and imaging data will provide unique insights into TSPO-PET enrichment patterns which may help to better understand and to comprehensively describe the clinical relevance of this novel imaging biomarker.

NIMG-107. THE LANDSCAPE OF BRAIN TUMOR MIMICS IN NEURO-ONCOLOGY PRACTICE

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BACKGROUND: Differentiating neoplastic and non-neoplastic brain lesions is essential to make management recommendations and convey prognosis, but the distinction between brain tumors and their mimics in practice may prove challenging. The aim of this study is to provide the incidence of brain tumor mimics in the neuro-oncology setting and describe this patient subset. METHODS: Retrospective study of adult patients referred to the Division of Neuro-oncology for a presumed diagnosis of brain tumor from January 1, 2005 through December 31, 2017, who later satisfied the diagnosis of a non-neoplastic entity based on neuroimaging, clinical course, and/or histopathology evaluation. We classified tumor mimic entities according to clinical, radiologic, and laboratory characteristics that correlated with the diagnosis. RESULTS: The incidence of brain tumor mimics was 3.4% (132/3897). The etiologies of the non-neoplastic entities were vascular (35%), inflammatory non-demyelinating (26%), demyelinating (15%), cysts (10%), infectious (9%), and miscellaneous (5%). In our study, 38% of patients underwent biopsy to determine diagnosis, but in 26%, the biopsy was inconclusive. DISCUSSION: Brain tumor mimics represent a small but important subset of the neuro-oncology referrals. Vascular, inflammatory, and demyelinating etiologies represent two-thirds of cases. Recognizing the clinical, radiologic and laboratory characteristics of such entities may improve resource utilization and prevent unnecessary as well as potentially harmful diagnostic and therapeutic interventions.

NIMG-108. PHYSICAL STRESSES IN BRAIN TUMORS

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Solid stress, distinct from fluid pressure, is a physical force contained in and transmitted by solid components of the brain tumor, including cells and the matrix they produce. Solid stress has been shown to promote tumor progression, and decrease anticancer therapy efficacy. This is especially relevant in brain tumors, as the rigid skull results in these trapped forces, increasing intracranial pressure, and potentially leading to other complications, including neuronal cell death. Here we present a novel method of quantifying these physical stresses in situ in both mice (glioblastoma [U87], brain metastasis [BT474], and ependymoma models) and patients. Briefly, following a craniotomy, mechanical forces that include solid stress are released, which causes the tissue to deform in peaks (areas under compression) and valleys (areas originally under tension). This tissue deformation is imaged via high-resolution ultrasound and analysed via custom MATLAB code to produce an accurate 3D model of the entire mouse brain, including the tumour region. For human samples, a pre-operative MRI is used to generate a detailed 3D model of the human brain. During surgery, the trapped physical stresses results in a bulge of the dura post craniotomy. We use BrainLab to measure the craniotomy induced brain deformation, which is then registered to the pre-operation MRI before being analysed in an identical fashion to the murine models using SolidWorks and Abaqus. We further show that in the brain metastases model, chemotherapy reduces compression stresses by 51%. Further, our technique results in fast processing time (~ 15 minutes), and has the potential to prevent the need for intraoperative MRI based on position simulations. As such, solid stress measurements provide a new class of mechanical biomarkers that can be correlated to clinical outcomes for predictive and prognostic value.

NIMG-109. A PILOT STUDY OF LYMPHOSCINTIGRAPHY WITH TRACER INJECTION IN THE HUMAN BRAIN

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INTRODUCTION: Many groups have reported lymphatic and glymphatic structures in animal and human brains, but tracer injection into the human brain to demonstrate real-time lymphatic drainage and mapping has not been reported. Technetium (^{99m}Tc) tilmanocept is a radiopharmaceutical agent that binds CD206 and maps draining lymph nodes. We hypothesized that peritumoral ^{99m}Tc-tilmanocept injection into the brain parenchyma would map drainage to cervical lymph nodes. Here we show a paucity of tracer movement after injection due to a presumed inefficiency of glymphatic and lymphatic drainage. METHODS: We enrolled 14 patients undergoing resection or stereotactic biopsy of suspected benign or malignant intracranial tumors. Patients received peritumoral injections of ^{99m}Tc-tilmanocept followed by planar or tomographic imaging. One patient was excluded from analysis because of tracer leakage during injection. RESULTS: No patients showed drainage of ^{99m}Tc-tilmanocept to regional lymph nodes. On average, after correcting for radioactive decay, 70.1% (95% CI: 60.0%, 81.6%) of the tracer in the injection site and 78.1% (95% CI: 71.1%, 85.1%) in the whole-head on the day of surgery remained the morning after, with variable radioactivity in the subarachnoid space. The remaining fraction at the injection site was much larger than the expected fraction of 1.12% (95% CI: 1.07%, 1.17%) based on previously reported clearance rates from non-brain injections. We determined that this lack of drainage was not due to sequestration by a large population of CD206$^+$ cells via immunohistochemistry of peritumoral tissue sections. Patients with benign or malignant tumors and a patient who underwent or minimally invasive stereotactic biopsy all showed no drainage. DISCUSSION: In this pilot study, the lymphatic tracer ^{99m}Tc-tilmanocept was injected into the brain parenchyma, and there was an absence of drainage to the cervical lymph nodes. Our work demonstrates an inefficiency of drainage from the brain parenchyma and highlights an opportunity to improve brain immunosurveillance.