morbidity in adults is the exclusive occurrence of subependymal giant cell astrocytoma (SEGA) which responds in at least 35% of cases to everolimus, mTOR inhibitor. However, drug treatment is associated with 33% rate of adverse events and requires long-term treatment **Methods:** In this report, we present a case of 49-year old female with TSC and a left enlarging SEGA that was approached endoscopically in order to minimize morbidity associated with open surgical approaches. **Results:** The use of NICO Myriad system is described in this case to achieve successful tumor debulking without post-operative neurologic morbidity. **Conclusions:** This report reveals the value of minimally invasive neuroendoscopic techniques in the management of challenging intraventricular tumors while avoiding injury to crucial deep venous structures.

P.051

5-hydroxymethylcytosine profiling identifies differential targeting in IDH1 mutant versus IDH1 wild-type high-grade gliomas

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Background: Gliomas demonstrate epigenetic dysregulation highlighted by the Glioma CpG-Island Methylator Phenotype (G-CIMP) seen in IDH1 mutant tumors. IDH1 mutation perturbs the balance between 5-methylcytosine (5mC) and 5-hydroxymethylcytosine (5hmC) by inhibiting TET-mediated active demethylation. The role 5hmC plays in *IDH1* mutant tumors remains poorly understood. **Methods:** We profiled 5hmC in high grade *IDH1* mutant (n = 12) and wild-type (n = 9) tumors on the Illumina MethylationEPIC Beadchip. We examined regions with high 5hmC abundance (top 1% probes), and differentially hydroxymethylated regions (DHMR). 5hmC profiles were correlated with gene expression. **Results:** Mean 5hmC b-values were 4.6%% and 3.8% for IDH1 mutant and wildtype tumors, respectively. Top 1% and DHMR probes demonstrated increased 5hmC among IDH1 mutants. 5hmC enriched for enhancer and super-enhancers. Among G-CIMP target genes, 22/50 were hydroxymethylated in our IDH1 mutant cohort, suggesting that 5hmC contributes to their overall methylation. Gene expression was associated with gene body 5hmC. 48 genes differentially expressed between IDH1 cohorts showed a positive Spearman correlation between 5hmC and gene expression, in particular for genes upregulated in IDH1 mutants. Conclusions: Locus-specific gain of 5hmC, targeting regulatory regions and associated with over-expressed genes, suggests a significant role for 5hmC in IDH1 mutant HGG.

P.052

Case Report: Brentuximab associated toxic neuropathy

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Background: A 28 year old male with a previous diagnosis of Ewing's Sarcoma in 2008, and a revised diagnosis to Hodgkin's lymphoma in 2016, presented to the Neurology service 6 months after starting the novel monoclonal antibody, Brentuximab. Concurrent therapy included adriamycin, vincristine and daunorubicin. He

was referred for progressive weakness and sensory symptoms starting in the legs and spreading to the arms over 6 months. Methods: Examination demonstrated distal symmetric weakness with power of 3 proximally and distally in the lower extremities. Reflexes were absent at the ankles and severely reduced at the patella. Gait was consistent with a sensory ataxia, and there was pseudoathetosis of the left hand. Results: MRI demonstrated no relevant abnormalities. Electrophysiology was consistent with a motor predominant, distal symmetric sensorimotor axonal neuropathy. Conclusions: A review of the literature demonstrated that the monoclonal antibody brentuximab has a high incidence (48%; n = 89) of a reversible distal symmetric polyneuropathy. The mechanism likely relates to microtubule dysfunction by the conjugated compound monomethyl auristatin E. This case adds to the existing body of literature around a severe but potentially reversible neuropathy, resulting from the new monoclonal antibody brentuximab, which may also serve as a model of disease in neuropathy with a well elucidated mechanism of toxicity.

P.053

Expanded endoscopic endonasal approach for petrous apex lesions: our clinical experience and surgical techniques

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Background: Traditionally petrous apex lesions surgical approach is associated with multiple complications including brain injury secondary to brain retraction. Expanded endoscopic endonasal trans-clival (EEET) can be used in selected patients with minimal complications. Methods: We are presenting our experience over the last three years in patients who underwent EEET resection of petrous apex lesions: 8 patients underwent such procedure. All patients underwent extensive workup including MRI and CTA to identify the relation of the carotid to the lesion. All surgeries were done with neuro-physiological monitoring. Intraoperative neuronavigation and endoscopic Doppler were used to identify the petrous segment of the internal carotid artery. Our follow up ranged from 6 months to 2.5 years. Results: All patients presented with severe neurologic symptoms related to either fifth cranial nerve, sixth cranial nerve or brain stem compression. Pathologies included chondrosarcoma, cholesterol granulomas and lymphangioma. All patients demonstrated improvement in their symptoms. None of our patients had intraoperative vascular injury. There was no post-operative CSF leak or infection. Postoperative imaging demonstrated excellent resection with no clear residual. Three patient required adjuvant stereotactic radiosurgery because of their underlying pathology. Conclusions: The expanded endoscopic endonasal approach for petrous apex lesion should be considered as a minimally invasive approach in selected cases.