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Diplopia and Vertical Gaze Palsy with Punctate Brain Stem Infarct

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Diplopia and Vertical Gaze Palsy with Punctate Brain Stem Infarct

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Methods: Data were collected using EPIC® starting 12 months prior to VNS replacement to Aspire® device to 12 months after implantation date in 3-month blocks. Seizure frequency before and after the VNS implantation as well as medication changes were identified from review of clinic notes. Cost was estimated based on the number of visits for ER, clinic, hospital, and ICU using hospital average cost data. As cost distributions are skewed to the right, we also calculated log total costs for the trend analysis using segmented model. For data analysis segmented Poisson regression was used to establish a trend in usage per quarter.

Results: The trend in Clinic usage was statistically flat ($p > 0.05$) in the pre-placement period while the trend in the post placement was downward with an estimate that for every quarter the natural log of the average number of clinic visits decreases by 0.29. By one-year post placement, the average number of clinic visits had achieved the same level as the pre-placement period. The trends in ER and hospital usage were flat in both the pre and post placement periods. The median number of CPS in the year after placement of the Aspire 106 VNS was statistically smaller by 1 ($p = 0.0161$) CPS than the median number of CPS in the year prior to placement.

Conclusion: Analysis of the above data yielded two main findings: there were no demonstrable cost savings within a 12-month period following VNS implantation, but the median burden of Complex Partial Seizures (CPS) within our cohort revealed a 50% decline, which was statistically significant. However, overall costs post-implantation showed a consistent downward trajectory, with approximation to pre-implantation costs within the 12-month timeframe. ■

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Diplopia and Vertical Gaze Palsy with Punctate Brain Stem Infarct

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Mentor: Sachin Kedar

Program: Neurology

Objective: To describe the clinical features, MRI findings, and neuroanatomy of a patient with a punctate brainstem stroke.

Method: Characterize the eye movement deficits in this patient and correlate the MRI diffusion restriction findings.

Results: This patient presented with vertical gaze palsy and impaired adduction of the left eye. Impaired smooth pursuits up and down clinically localized to damage in the interstitial nucleus of Cajal. Impaired saccades up and down localized to the rostral interstitial nuclei of the medial longitudinal fasciculus. MRI showed diffusion restriction in the left

thalamus-midbrain junction just posterior to the red nucleus.

Conclusion: This case illustrates how a punctate single unilateral lesion can present with vertical supranuclear gaze palsy and horizontal eye movement abnormalities. ■

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Paroxysmal Autonomic Instability and Dystonia Following a Motor Vehicle Accident

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Program: Neurology

Background: We were consulted a young adult patient who presented following a motor vehicle accident. The patient presented with diffuse axonal injury, hemorrhage of left basal ganglia increased intra-cranial pressure and other injuries. The patient was intubated, an intraventricular shunt had been removed and a 7-day course of Keppra prophylaxis had been finished. The patient was stable until the morning we were consulted.

Methods: On exam the patient had episodes of extensor posturing, diffuse dystonia and high frequency, low amplitude tremors, with autonomic instability limited to these spells.

The patient was found to have 3+ reflexes throughout. Brain stem reflexes were intact. Stimuli caused extensor posturing, rigidity and autonomic instability. Continuous VEEG showed epileptiform activity that did not correlate with spells on video. Lab was non-specific expect for a reelevated CK. We loaded Keppra and prn diazepam. This failed to control the symptoms.

Results: We arrived at a diagnosis of paroxysmal autonomic instability and dystonia (PAID) syndrome. Treatment with clonazepam, Gabapentin, baclofen and propranolol stabilized the autonomic system and resolved the dystonia. The CK began to drop and the patient began to respond to commands.

Conclusions: PAID most commonly affects young persons following severe TBI. Potentially caused by disinhibition of sympathoexcitatory regions leading to cortically provoked catecholamine surges causing autonomic instability. Dystonia is secondary to disruption of the pontine and vestibular nuclei. PAID should on the differential diagnosis of TBI patients who are not responding to typical treatments. We had success treating with Clonazepam, gabapentin, baclofen and propranolol. ■

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Diagnostic Challenges in The Radiographic Distinction Between Demyelinating Lesions and Primary Brain Neoplasms

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Program: Neurology

Introduction: The differentiation between demyelinating lesions and primary brain neoplasms is difficult based on radiography alone, resulting in misdiagnoses and delay in treatment. Here we present two cases with MRI abnormalities suggestive of either

demyelination or neoplasm. Case 1 was treated with steroids for a demyelinating process, but was found on biopsy to have an anaplastic astrocytoma that was unresectable by the time of diagnosis. Case 2 was thought to be glioma but was later determined to be tumefactive demyelination. In both cases, there were delays due to the limitations of MR imaging.

Methods: The cases of the two patients were reviewed via EMR. A literature review investigated the most sensitive and specific non-invasive diagnostic modalities for diagnosing demyelinating lesions or primary brain neoplasms.

Results: Review of recent literature shows that conventional MR sequences have