REVERSIBLE POSTOPERATIVE BLINDNESS DUE TO BILATERAL OCCIPITAL STATUS EPILEPTICUS FOLLOWING NEUROSURGICAL PROCEDURES

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DISCLOSURES

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INTRODUCTION

Post-operative vision loss (POVL) is a rare but devastating complication after a Neurosurgical procedure. The phenomenon is most commonly related to vascular insults to the optic nerve or occipital cortex following prolonged spinal procedures in the prone position. Arterial ischemia may damage the optic nerve resulting in either anterior ischemic optic neuropathy (AION) or posterior ischemic optic neuropathy (PION). Occlusion of the central retinal artery may also cause retinal ischemia and post-operative blindness. Less-common but previously reported causes of POVL include cortical blindness due to Posterior Cerebral Artery occlusion or Posterior Reversible Encephalopathy Syndrome (PRES). Occipital lobe seizures resulting in complete blindness are an entity rarely reported in the literature.[1-4] Referred to as status epilepticus amauroticus (SEA), persistent occipital lobe seizures most often present with visual hallucinations. However, up to one-third of patients with SEA can develop ictal blindness.[5] We report 2 cases of Neurosurgical procedures (a staged thoracolumbar deformity correction & a right lateral temporal bone resection) complicated by complete cortical blindness secondary to SEA.
CASE #1 NEUROSURGICAL TREATMENT

A 67 year-old male with no previous spine surgery presented to clinic complaining of severe low back pain and bilateral L3 radiculopathies. MRI of the lumbar spine revealed central canal and lateral recess stenosis in the mid lumbar spine. Upright long cassette radiographs revealed severe sagittal imbalance, flattening of normal thoracic kyphosis and Lumbar Lordosis - Pelvic Incidence mismatch (Figure 1). A lumbar decompression with T3-ilium deformity correction was recommended to alleviate the patient’s symptoms. This was planned in a staged fashion with 1 day of rest between the two stages.

Preoperative Lumbar T2 weighted MRI scans of the Lumbar spine
CASE #1 CLINICAL COURSE

The patient awoke from the second stage of the procedure at his neurologic baseline with intact vision. Six hours later, he complained of sudden onset visual loss. On examination, he was found to have bilateral blindness with absent perception of light or hand motion. A noncontrast enhanced Computed Tomography of the head was negative for acute pathology. This was followed with Magnetic Resonance Imaging, Magnetic Resonance Angiography and Venography of the brain which were all unremarkable. A comprehensive altered mental status serum lab workup was normal. Carotid duplex and echocardiogram were both normal. Routine EEG revealed slowing of the posterior background, but did not reveal epileptiform discharges nor seizure activity. Ophthalmology examination revealed a left pupil fixed at 5 mm and a right pupil fixed at 1 mm. Bilateral pupils briefly dilated when light was withdrawn, then slowly returned to their previous size without a light stimulus. Switching from dark to light exposure did not elicit pupillary dilation nor constriction. A dilated fundoscopic exam revealed intact bilateral anterior optic nerves and no abnormal retinal findings. Medication side effect was entertained to explain these findings but no clear ophthalmologic etiology could explain these pupillary findings.

As the ophthalmologic exam was inconclusive and the patient continued to be intermittently confused, continuous video-EEG was started two days after the second stage of surgery. This revealed bilateral occipital lobe seizures (Figure 2) with an associated semiology of confusion and blindness only. Nystagmus and eye deviation were absent. The seizures temporarily stopped after the patient received 3 doses of intravenous lorazepam 2 mg, intravenous levetiracetam 1000 mg, and lacosamide 400 mg. However, the patient remained blind. Electrographic bioccipital status epilepticus recurred 1 hour later. The patient was therefore intubated, and a continuous intravenous infusion of midazolam was initiated with a loading dose of 0.2 mg/kg and continued at 0.1 mg/kg/hr with resolution of occipital status epilepticus. The midazolam infusion was slowly titrated off over 3 days with the patient maintained on levetiracetam and lacosamide. There were no recurrent seizures on EEG. The patient was subsequently extubated and found to have regained his baseline vision.
Preoperative lateral (a) and AP (b) standing long cassette radiographs reveal sagittal imbalance with flat back deformity (PI: 50, PT: 29, LL: 8, SVA: 17, TKy: 18). Postoperative lateral (c) and AP (d) studies shows improvement in sagittal balance with an increase in lumbar lordosis and thoracic kyphosis (PI: 50, PT: 29, LL: 25, SVA: 5, TKy: 36).
Initial EEG revealed partial seizures consisting of frequent occipital spikes, isolated or organized in clusters, arising from the right and left occipital regions (a). The partial bioccipital seizures eventually became generalized and persistent (b,).
CASE #2 NEUROSURGICAL TREATMENT

A 68-year-old female with right-sided otalgia and was found to have a mass in her right external auditory canal. She underwent a CT of the temporal bones which revealed a destructive soft tissue lesion of the external auditory canal. There was erosion into the mastoid bone with posterior extension to the sigmoid sinus. (Figures 1 and 2) Biopsy of this lesion showed invasive squamous cell carcinoma. Her past medical history included breast cancer treated with lumpectomy, axillary lymph node dissection, and adjuvant chemoradiation greater than 10 years prior. The patient was treated with a right lateral temporal bone resection, superficial parotidectomy, modified radical neck dissection, and microvascular free flap reconstruction. The bone over the sigmoid sinus was resected but the tumor did not invade into the sigmoid sinus or dura.

Axial and coronal sections of the patient’s preoperative CT scan of the temporal bone showing an erosive soft tissue mass of the right external auditory canal and mastoid (white arrows).
CASE #2 CLINICAL COURSE

The patient was brought to the post anesthesia recovery unit (PACU) with stable vital signs with heart rate in the 60s and systolic blood pressure in the 120s. Shortly after arriving in the PACU, she experienced an episode of tachycardia and hypertension with a heart rate in the 120s and systolic blood pressure in the 180s. She also became unresponsive at this time. After approximately 1 minute she started responding to questions and commands. She was noted to have peripheral right sided facial weakness. There were no other focal neurologic deficits. Approximately 50 minutes following this episode, the patient reported acute vision loss bilaterally with no change in her overall physical examination.

A CT scan of the head and CT angiogram of the head and neck were normal. On postoperative day 1 continuous video electroencephalogram (EEG) revealed a partial seizure of the left occipital lobe with secondary involvement of the right occipital lobe. Levetiracetam was started but there remained concern for posterior reversible encephalopathy syndrome (PRES) given the transient hypertensive episode in PACU. The patient reported some improvement in vision and was able to perceive light and motion. On postoperative day 2 the implantable Doppler was removed so the patient could undergo MRI of the brain and MR venography. There was no evidence of occlusion of the dural venous sinuses. There were ill defined T2 and flair hyperintensities within the bilateral thalami and periatrrial white matter without associated diffusion abnormalities. On postoperative day 3 the patient developed intermittent visual hallucinations. By postoperative day 4 the patient had significant improvement in visual acuity with 20/30 in the right eye and 20/25 in the left eye. Neurology originally felt the T2 hyperintensities seen on MRI may have represented a subtle case of PRES. Multiple subsequent MRIs, however, showed stable nonspecific white matter T2 signal abnormalities which were attributed to chronic small vessel ischemia. Neurology hence felt PRES was unlikely given the stability of the lesions on imaging. She was successfully weaned off antiepileptic medications. She is currently over two years out from surgery and there have been no additional episodes of acute visual loss or seizure like activity. There is no evidence of local or metastatic cancer recurrence.
DISCUSSION & SUMMARY POINTS

POVL is a devastating complication after any surgery. It is most frequently reported after spine and cardiac surgery with estimates of 0.2% and 4.5%, respectively. [6, 7] Etiologies for POVL after spine surgery include AION, PION, central retinal artery occlusion (CRAO), PRES and cortical blindness due to PCA territory ischemia. Occipital lobe seizures and SEA presenting as complete blindness may represent an etiology for POVL, and should be considered in patients with or without a seizure history. Clinical suspicion is raised if seizure semiology is accompanied by altered mental status, eye deviation, or body shaking and rigidity. Continuous EEG is required for diagnosis as routine EEG may not capture non-convulsive seizures and SEA. Treatment with AED’s should be initiated for occipital lobe seizures and may require intubation for continuous intravenous infusions (midazolam, pentobarbital, ketamine, volatile anesthetics) if SEA develops.

This report describes SEA as a novel cause of POVL that requires a heightened degree of clinical suspicion. If left untreated, SEA may result in permanent visual loss. However, as demonstrated in this case report, early recognition and aggressive anti-seizure treatment can result in an excellent outcome.