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Case Report

Preoperative visual field deficits in temporal lobe epilepsy☆

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ABSTRACT

Surgical resection and laser thermoablation have been used to treat drug resistant epilepsy with good results. However, they are not without risk. One of the most commonly reported complications of temporal lobe surgery is contralateral superior homonymous quadrantanopsia. We describe a patient with asymptomatic preoperative quadrantanopsia fortuitously discovered as part of our recently modified protocol to evaluate patients prior to temporal lobe epilepsy surgery. This visual field deficit was subtle and not detected on routine clinical neurological examination. While we understand that this is a single case, we advocate further study for more detailed preoperative visual field examinations to characterize the true incidence of postoperative visual field lesions.

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1. Introduction

Surgical resection has been a well-documented treatment modality in patients with focal epilepsy localized to the temporal lobe. While resection has lead to good postoperative seizure outcomes, surgey is not without risk. Contralateral superior quadrant visual field deficits are one of the most commonly reported adverse outcomes after temporal lobe surgery, occurring in 28% to 52% of patients [1,2]. Multiple techniques and adjuvant intraoperative technologies have been reviewed in an effort to minimize visual field deficits after temporal lobe surgery. In this case report, we describe a patient with subtle preoperative left visual field deficit that was unaltered after right temporal lobe surgery. We postulate that the true rate of postoperative visual field deficits may be lower than the reported rates. We suggest further study to determine if there are subtle visual deficits that may be associated with the patient's underlying epilepsy.

2. Case report

Our patient was a 57-year-old female with adult-onset seizures. She had a 4-year history of seizures with no significant history of trauma or early risk factors for epilepsy. The clinical semiology consistent with focal dyscognitive seizures of temporal lobe origin. Based on some of the clinical history and semiologic features details (lip smacking, left upper extremity dystonic posturing, and head deviation to the left), a clinical localization of right mesial temporal lobe epilepsy was suspected. She had been trialed on 3 first-line antiseizure medications without seizure control. Her antiseizure drugs included 100 mg of lamotrigine twice daily, 100 mg of topiramate twice daily, and 400 mg of eslicarbazepine daily. The patient was unable to tolerate further dose escalation.

She had significant decline in her quality of life because she was legally unable to drive and work because of her seizures. On physical examination performed by the epileptologist, there were no focal deficits and no visual field deficits. The patient denied any visual changes.

As part of our epilepsy protocol, patients undergo neuroophthalmologic evaluation in addition to neuropsychological testing. Upon neuroophthalmologic evaluation, there was concern for a subtle left superior homonymous quadrantanopsia. This was confirmed with computational Goldman perimetry (Fig. 1a). The inpatient epilepsy monitoring unit admission revealed 8 focal seizures with either bitemporal onset or right temporal onset on ictal EEG recording, with rapid spread to the contralateral temporal lobe. Magnetic resonance imaging did not reveal any signs of mesial temporal lobe sclerosis, gray matter heterotopia, or focal cortical dysplasia. Positron emission tomography (PET) scan of the brain revealed interictal right mesial temporal lobe hypometabolism (Fig. 2). Based on these findings supporting MR -/PET + right temporal lobe epilepsy (TLE), the patient elected to undergo laser thermoablation of the right mesial temporal lobe.

As of her last follow-up appointment, the patient had remained seizure free, and neuroophthalmologic evaluation revealed no change in her baseline visual field deficit (Fig. 1b).

3. Discussion

Visual field deficits are one of the most commonly reported complications following temporal lobe surgery. This is likely due to the close

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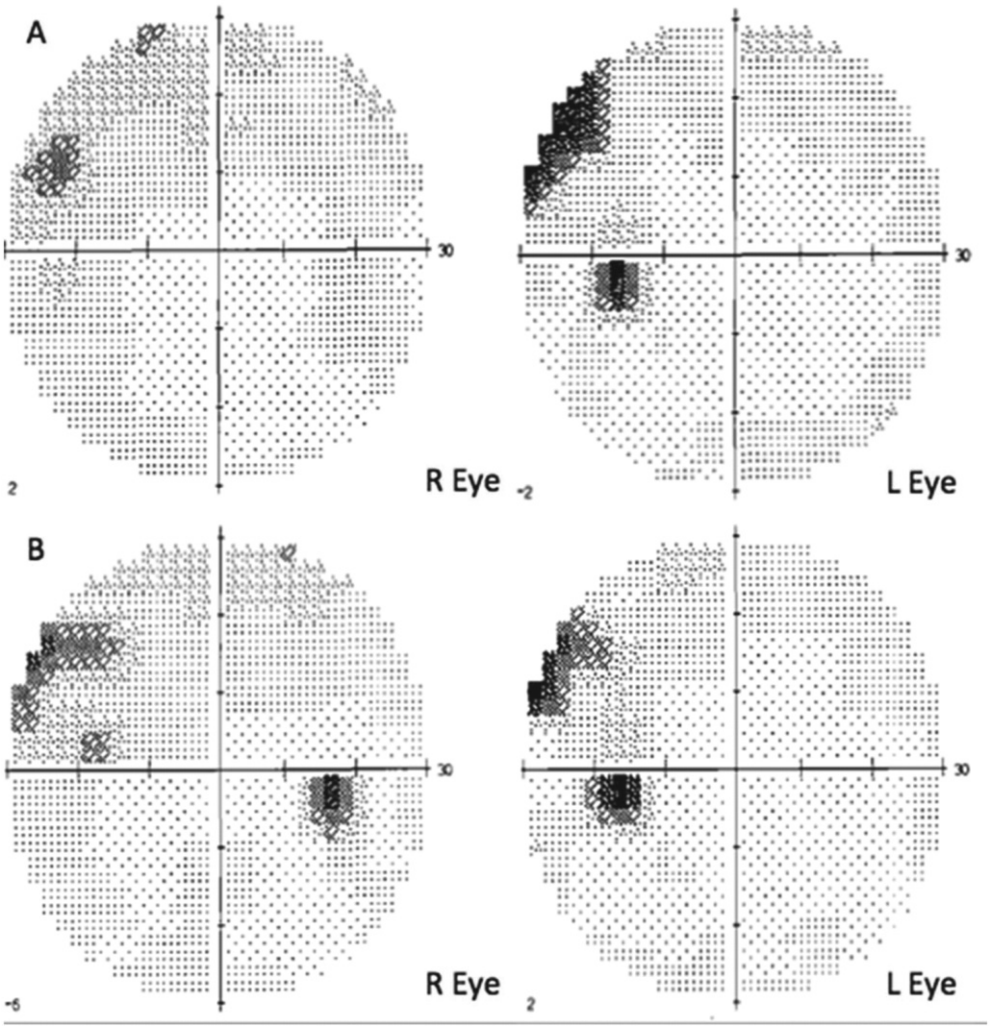

Fig. 1. Visual fields, A (presurgical)/B (postsurgical); images reveal a stable left incomplete homonymous quadrantanopsia.

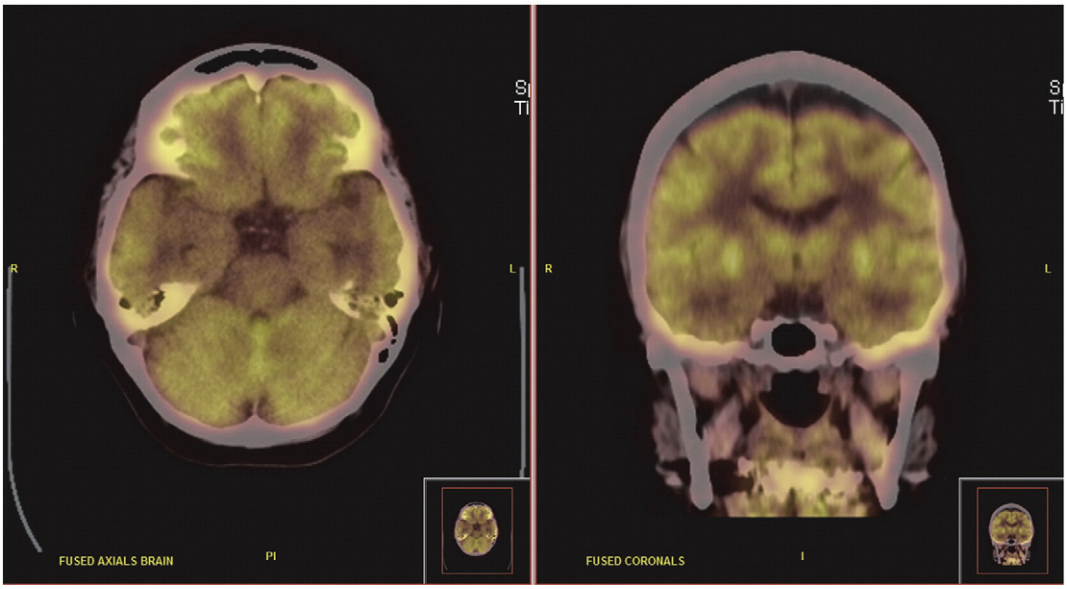

Fig. 2. Axial and coronal positron emission tomography (PET) scans revealing mild right mesial temporal lobe hypometabolism.

proximity of the visual fibers to the epileptogenic zone in TLE. Nasal fibers from the superior temporal visual field pass through the optic nerve and decussate in the optic chiasm. These fibers synapse in layers 1, 4, and 6 of the lateral geniculate body and then travel anteriorly and inferiorly to loop around the temporal horn of the lateral ventricle to their eventual destination synapsing in the primary visual cortex. The ventral aspect of the optic radiations passing anteriorly around the temporal horn is referred to as Meyer's loop.

It was originally thought that one could avoid Meyer's loop by limiting the posterior extent of a temporal lobectomy to 6 cm from the temporal pole [3]. This was subsequently revised to 4 to 5 cm [4]. However, recent studies suggest a high degree of variability in the location of the temporal radiations, with variations both between subjects, as well as between the left and right temporal lobes, with Meyer's loop typically being more anterior in the left temporal lobe [5–7]. Due to this variability, surgeons have increasingly employed intraoperative adjuncts, such as neuronavigation with tractography, in an attempt to minimize postoperative visual field deficits. In addition, cortical-sparing approaches have been employed, such as selective amygdalohippocampectomies through a subtemporal approach or trans-sulcal approaches, and most recently, laser thermoablation.

Despite the use of cortical-sparing approaches and intraoperative adjunctive tools, visual field deficits remain a potentially significant morbidity [1,2,8,9]. Fortunately, many of these deficits are clinically insignificant and do not preclude patients from driving [5].

This case illustrates a patient with a clinically “silent” contralateral superior homonymous quadrantanopsia following laser thermoablation of the mesial temporal lobe. In most retrospective studies and series, this would have been attributed to a lesion of Meyer's loop from surgery. However, due to our case, we have instituted a recent protocol detail preoperative visual field examination. Our patient's visual field deficit was recognized preoperatively and was noted to have remained stable after the procedure. While antiseizure drugs such vigabatrin, topiramate, and carbamazepine may be associated with visual disturbances or visual field deficits, it is unlikely that an isolated superior homonymous quadrantanopsia was the result of medication [10]. While the precise pathophysiology is unknown, we postulate that the patient's visual field deficit could result from aberrant neuronal dysfunction in the mesial temporal lobe, as depicted in the PET scan. While the onset of seizures is generally cortical in location, seizures or underlying pathology can involve through white matter tracts (Meyer's loop), and the chronic effects of recurrent seizures could potentially lead to the subtle visual field defect detected in our patient.

While we acknowledge the limitations of a single case report, the authors believe that subtle preoperative visual field deficits are likely an underreported entity and artificially inflate to the high incidence of postoperative visual field deficits. We advocate detailed preoperative visual field testing for patients undergoing temporal lobe surgery for epilepsy.

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