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Case Vignette

Tics, OCD and Epilepsy: To Lump or Split?

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Abstract

In this report, we describe the case of a 21-year-old man with epilepsy, presenting with obsessive compulsive disorder (OCD) and tics while on levetiracetam monotherapy. Diagnosis of tics in a person with epilepsy (PWE) is challenging and after a detailed evaluation, possibility of drug induced tics was considered. Both symptoms abated on stopping Levetiracetam therapy. Levetiracetam induced various behavioral side effects have been reported in both adult and pediatric patients, but OCD and tics are extremely rare and not known to many. The unique mechanism of action of Levetiracetam has been proposed as the possible explanation for these behavioral manifestations. At the same time there is literature evidence for use of Levetiracetam in Tics. We wish to emphasize on this double edged sword nature and give further insights into it.

Keywords: Tics; OCD; Epilepsy

Introduction

We wish to report a case of levetiracetam (LEV) induced tics in a person with epilepsy(PWE) who presented to our epilepsy clinic. A 21-year-old gentleman, a college graduate presented with history of a single seizure which occurred in the early morning hours, with versive head turn to one side, followed by tonic clonic movements of both upper limbs, lasting around 2 minutes. There was loss of consciousness, post ictal confusion, tongue bite and frothing. This occurred when he was eleven years of age. He has a normal birth and development history, average scholastic performance with no significant family history. He has no history of febrile seizures, nor encephalitis or head trauma. His neurological examination was normal. MRI brain revealed a right temporal dysplastic cortical based lesion involving hippocampus and para-hippocampal gyrus suggestive of a low grade glioneuronal tumor. (Figure 1) The patient was started on levetiracetam 1000 mg/day. It was decided to keep the patient under follow-up with serial imaging since it was a

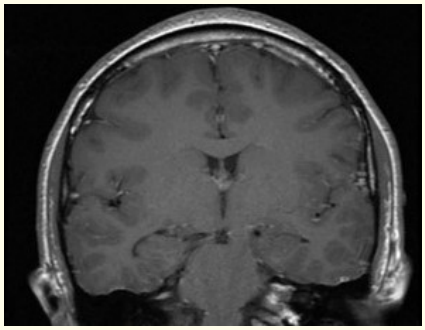

Figure 1: High resolution 3T MRI T1W (A), T2W (B), T2 FLAIR (C) images showing right hippocampus and parahippocampal gyrus altered signal intensity suggestive of a low grade glioneuronal lesion.

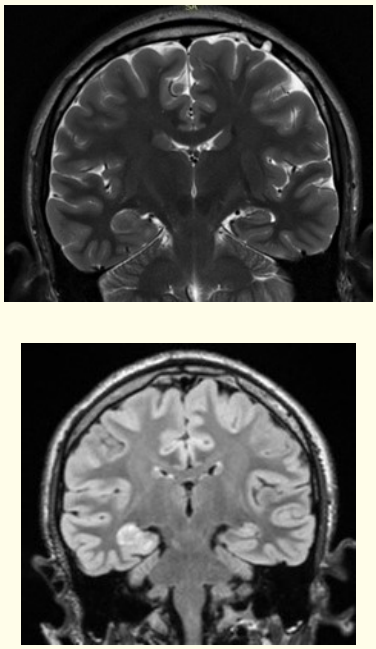

Figure 1: High resolution 3T MRI T1W (A), T2W (B), T2 FLAIR (C) images showing right hippocampus and parahippocampal gyrus altered signal intensity suggestive of a low grade glioneuronal lesion.

single seizure, responded to LEV monotherapy and no increase in size of tumor was noted in serial MRIs over years.

He presented to us six months back with new onset symptoms of one year duration in the form of obsessive-compulsive behaviour for the first time in his life, such as handwashing multiple times, latching doors repeatedly, frequent turning and looking back while walking, climbing on and off his bike etc. He also had frequent throat clearing, all of which was aggravated under stressful situations. He was also noted to have repeated stereotyped movements like tapping the side of his head, wriggling of toes and pursing of lips. There was no history of psychiatric disorders in his family. He had no addictions and sleep pattern was normal. Patient was initially evaluated by a psychiatrist and was initiated on Fluoxetine 20 mg/day and Risperidone 2 mg/day. No substantial change was noted though.

On evaluation, he had both obsessions and compulsions which were persistent causing marked distress in him for one year, meeting the DSM-V criteria for OCD [1]. We kept the following possibilities for his stereotyped movements viz., compulsions, habitual actions, epilepsia partialis continua (EPC), stereotypies, chorea or other movement disorders, psychogenic and antiseizure medication(ASM) induced (Levetiracetam in this case). His movements were variable, distractable and exacerbated by stress suggestive of motor tics. Vocal tics also were noted. Though these have been present for a year, the age of onset did not favor Tourette disorder according to DSM- V criteria [1]. Work-up for other underlying movement disorders were negative. Video EEG was done to rule out the possibility of EPCs and record showed focal interictal epileptiform abnormalities over right anterior temporal region. Stereotyped movements were noted during the record and had no EEG correlate.

After ruling out all other causes relevant to his symptoms and signs, we tapered and stopped levetiracetam. Both OCD and tics reduced drastically in 48 hours and disappeared in a week.

Discussion

Psychiatric and behavioral side effects as undesirable effects associated with ASM use and are reported to occur in 7.6% of patients [2]. History of pre-existing psychiatric conditions and drug-resistant epilepsy are associated with increased incidence of these side effects. Levetiracetam is the ASM most frequently reported to cause behavioral side effects. These include irritation, aggression, depression, nervousness, suicidal ideation and psychotic symptoms but reports of levetiracetam induced OCD and tics are extremely rare [2].

Çökmüş et al reported a case of OCD symptoms in a 55-year-old epileptic female patient who did not have any previous psychiatric disorder, and received levetiracetam treatment [3]. Fujikava., *et al*. has reported a 13-year-old female child who after two months of initiation of levetiracetam with favorable seizure control, started to show OCD [4]. Peters., *et al*. in a pragmatic review has identified 43 cases of tics induced by ASMs published in the medical literature, 40 from case reports, two reported in a randomized control trial [5]. Carbamazepine, clonazepam, lacosamide, lamotrigine, levetiracetam, phenytoin and phenobarbital have been identified as causative ASMs. Phenomenology had facial/truncal predominance and most tics resolved or improve with either ASM withdrawal or dose reduction [5].

There is emerging evidence that glutamate transmission abnormalities in cortico-striato-thalamo-cortical circuits may contribute to the pathogenesis of OCD [6,7]. Modulation of the balance of the cortico-striato-thalamo-cortical loops below a 'tic threshold', in favor of disinhibition, in susceptible individuals is hypothesized to cause tics. Levetiracetam exerts antiepileptic effect by its unique mechanism of action by binding to synaptic vesicle protein (SV2A) and controlling SV2A mediated glutamate release [8]. Modulation of the glutamate system by levetiracetam may be the plausible explanation for the behavioral effects.

Interestingly, a number of anticonvulsants have been investigated for their efficacy in OCD as well as tics. Wang et al reviewed the potential role of anticonvulsants in OCD and found that except for two negative studies, anticonvulsants were effective in OCD [9]. A prospective, open-label study by Avaad., *et al.* has evaluated the effects of levetiracetam on motor and vocal tics in children and adolescents with tics and Tourette syndrome. All the 60 patients, 18 years of age enrolled in the study showed improvement based on Yale Global Tic Severity Scale [10].

Conclusion

Through this clinical commentary, we wish to highlight the challenges involved in diagnosis of tics and OCD in an epilepsy patient. Detailed evaluation is warranted to rule out other causes. Drug induced tics should always be kept as a differential diagnosis. Levetiracetam being a broad-spectrum antiepileptic is widely used and there are several reports of behavioral side effects, but OCD and tics are extremely rare and therefore overlooked. Nevertheless, there is literature evidence for the use of Levetiracetam for treatment of OCD and tics. This double edge sword nature of Levetiracetam is unknown to many and hence we wish to emphasize this enigma through this case vignette.

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