Recovery of Visual Scotomas by Vortioxetine in a Patient with Symptomatic Occipital Lobe Epilepsy

Sir,

A 52-year-old female patient was admitted to our emergency department after having a first time generalized convulsive seizure (February 2017). On history interrogation, it was learnt that the patient had a resective surgery of edematous, left occipital lobe meningioma (April 2015) which was detected following investigations due to complaints of new-onset headache. The patient was discharged without neurological sequela. Phenytoin therapy was initiated for seizure prophylaxis which was stopped 6 months after surgery. On neurological examination at emergency department, she was evaluated as confused and no lateralized neurological deficit was present, compatible with postictal state. Cranial magnetic resonance imaging showed encephalomalacia in the left occipital lobe [Figure 1]. Due to the clinical manifestation and neuroimaging findings, the diagnosis of symptomatic epilepsy was established and levetiracetam 500 mg BD was started. Routine electroencephalogram (EEG) could be performed 2 days later (it was weekend and EEG was not available) which showed sharp waves over the left occipital region supporting the diagnosis of symptomatic occipital lobe epilepsy [Figure 2]. Two weeks later, the patient admitted to neurology polyclinic for a follow-up visit. She was on levetiracetam 1000 mg therapy, and no seizure recurrence or treatment-related adverse event had occurred. Control EEG was evaluated to be in normal ranges. However, on further interrogation, it was learnt that the patient had been suffering from visual scotomas in the right visual field which were occurring gradually and proceeded nearly for 10 min and repeated multiple times throughout the day (10–20/day). Detailed interrogation of the medical history revealed that these symptoms (scotomas) had started 3 months before the convulsive seizure, and no amelioration

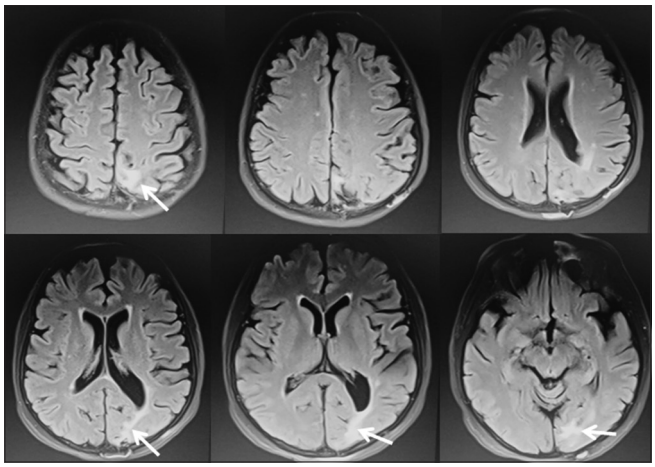

Figure 1: Cranial magnetic resonance imaging (flair sequences) showing encephalomalacia in the left occipital lobe

had occurred following levetiracetam therapy, considering underlying possible mechanisms other than ictogenesis. Levetiracetam dosage was not increased. Nonetheless, the patient was consulted to psychiatry evaluation due to anxiety and depression symptoms which were obviously recognized during the patient interview. She had complaints of a long-term sadness, lack of interest, inability to relax, disturbed sleep, and difficulty in concentration over the last 2 years which had started remarkably soon after the operation. Her complaints had worsened during the last 6 months, and angry outburst and anxiety attacks had occurred such that she was unable to work regularly due to these symptoms. On psychiatry evaluation, organic depression was considered and vortioxetine 10 mg/day was started. Of note, given the possible precipitating effect of levetiracetam, a switch therapy with other antiepileptic drugs such as carbamazepine or valproic acid was considered as an alternative treatment of depression. Nonetheless, it was learnt that depression symptoms were present for the last 2 years and no aggravation in depression had occurred following initiation of levetiracetam for which we referred a psychiatry consultation instead of changing levetiracetam therapy. On the 1st month evaluation, her depressive symptoms recovered moderately. However, more interestingly, visual scotomas were improved significantly following vortioxetine therapy in a gradual course. For an objective assessment, further evaluations were performed. The patient scored 20 on the Beck depression inventory (BDI) (moderate depression), 22 on the Beck anxiety inventory (moderate anxiety), and 14 on the hospital anxiety and depression scale (13 on depression item and 7 on anxiety item). Vortioxetine was increased to 20 mg/day dosage, and polyclinic control was planned. On the 2nd month follow-up visit, BDI score improved to 11 and HAD 'Hospital Anxiety and Depression Scale' depression subscore improved from 13 to 7 in the interval period. Besides, her complaints of visual scotomas were recovered dramatically up to the frequency of once a week and a milder severity following vortioxetine therapy.

Recently, over the past 5 years, FDA has approved three additional drugs for the treatment of major depressive

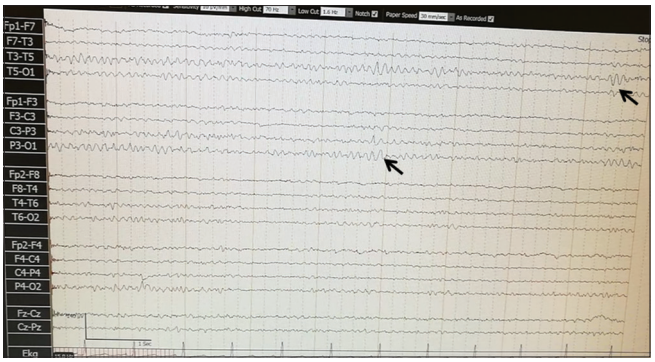

Figure 2: Routine electroencephalogram showing sharp waves over the left occipital lobe

disorder.[1] Vortioxetine is one of these new treatments for depression basically acting as a 5-HT3, 5-HT7, and 5-HT1D receptor antagonist, 5-HT1B receptor partial agonist, 5-HT1A receptor agonist, and serotonin transporter inhibitor.[2] Remarkably, preclinical studies in animals suggested that it may be efficient in improving precognitive functions which is suggested to be mediated through different receptors and brain chemicals other than its efficiency in depression.[3,4] Distinctly, in this report, we present an interesting course of an epilepsy patient whose visual symptoms (scotomas) have given dramatic response to vortioxetine therapy in addition to the depressive disorder. To the best of our knowledge, this report is unique illustrating successful usage of vortioxetine in an epilepsy patient which may give substantial perspectives for future studies.

A very important issue among clinicians is that antidepressant drugs can lower the seizure threshold and this handicap is particularly higher in tricyclic antidepressants subgroups and moderately in serotonin reuptake inhibitors.[5] However, the existence of this side effect in new generation antidepressants remains to be clarified. In our patient with epilepsy, the response to vortioxetine to depressive symptoms was excellent, and more interestingly, visual scotomas had recovered significantly following vortioxetine treatment. One may assert that if the amelioration of visual scotomas was related with vortioxetine or levetiracetam? At 2-week evaluation from initiation of levetiracetam, which is a rapidly titrated and efficient antiepileptic drug, not any improvement in visual scotomas was achieved. In addition, the clinical course of the recovery of these symptoms was in a gradual and progressive manner following initiation of vortioxetine, which is also compatible with temporal evaluation pattern of the efficiency of vortioxetine associated with its pharmacokinetic features. Nevertheless, the issue of by which mechanisms might vortioxetine act resulting in this clinical output can be another matter of debate. The second routine EEG (2 weeks following levetiracetam therapy) showed recovery of sharp waves which was evaluated to be in normal ranges, leading to the consideration of etiologies other than ictogenesis. On the other hand, it is known that an accurate clinical-electrographic correlation in occipital

lobe epilepsy is difficult to be present which has been attributed to the inherent limitations of scalp EEG recordings from the occipital and the calcarine region.[6] Besides, the phenomenology associated with ictal discharges from the occipital lobe is rather subjective according to the observation in temporal lobe epilepsy, making the diagnosis a harder deal. Therefore, we think that ictogenesis as a responsible mechanism underlying visual scotomas cannot be excluded in our patient. In accordance with this consideration, the most common symptoms in association with lesional occipital lobe epilepsy are visual as in our case.[7]

Taken together, based on this unique case, giving conclusions focusing on the mechanisms of the recovery process of visual scotomas (influenced by vortioxetine) may not be rationale. Video EEG monitoring data might give a more accurate demonstration of the pathophysiology of visual auras which was unavailable in our case. However, independently of the responsible mechanisms, this case may rather give a crucial insight regarding the usage of vortioxetine in particularly epilepsy patients for depressive disorder. Future studies reporting the clinical output of usage of vortioxetine in larger groups of depression patients with epilepsy comorbidity may give crucial perspectives for clinical approaches.

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Conflicts of interest

There are no conflicts of interest.

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