Compulsive gambling possibly associated with antiepileptic medication

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1. Introduction

Compulsive gambling has been reported with treatment of Parkinson’s disease but not with epilepsy. This report provides the first possible connection between gambling and treatment of epilepsy.

2. Case

A 46-year-old female, diagnosed with idiopathic generalized epilepsy at the age of 18, was first seen in the clinic in 2000, when she had nonconvulsive status epilepticus. She had trialed carbamazepine, valproate (VPA), topiramate, gabapentin (GPT), and lamotrigine (LTG), prior to attending the clinic, to which had been added clobazam without seizure control. Levetiracetam (LEV) was introduced in 2001, at which time she was on VPA and GPT. Lamotrigine was reintroduced in 2006, leaving her on LEV and LTG. With increased LTG dosage, she lost $50,000, prompting discovery of her gambling.

3. Discussion

GSK (Glaxo Smith Kline) and UCB (United Chemical Belgium) were contacted, and GSK reported intrusive repetitive behavior (similar to punding) associated with LTG but that there had been no reports of excessive gambling. UCB reported that LEV is associated with depression and other emotional issues, but there are no known cases of gambling with LEV. Evidence of an association between LEV and a genetic variation in dopaminergic activity and hence the risk for psychiatric complications has been previously reported [1]. As it is presumed to be the dopaminergic effect of dopamine agonists that provoke the compulsive gambling in Parkinson’s disease, this may offer a plausible explanation in this case.

Dopaminergic medications, in the form of Madopar (L-dopa combined with benserazide), have been trialed in 3 patients with intractable epilepsy, resulting in seizure reduction in 2 patients, thereby suggesting possible reinforcement of the hypothesis of a dopaminergic mechanism of seizure control in idiopathic generalized epilepsy [2,3].
There have been other reports of obsessive behavior with LEV that dissipated upon stopping LEV [4], which also indicates it to be a potential provocateur in this case. Lamotrigine has also been associated with obsessive symptoms, which appeared dose-dependent in one case [5]. In that case, it was hypothesized that LTG inhibited the presynaptic release of glutamate with altered striatal dopamine uptake, which again is relevant to the reputed cause of gambling associated with Parkinson’s disease and dopaminergic therapy. Others [6] have also noted the emergence of obsessional behavior in association with LTG and questioned glutamatergic regulation. Animal studies have demonstrated stereotyped behavior provoked by dopaminergic mechanisms induced by LTG. The hypothesis is that LTG may directly stimulate the postsynaptic striatal D2 and D1 dopamine receptors or indirectly release dopamine from the nigrostriatal dopamine neurons [7].

These reports, when seen in the context of this patient, raise serious concerns that this patient’s gambling behavior may well be AEM-provoked and could be due to either the LTG or the LEV or the combination of both. The emergence of compulsive gambling in Parkinson’s disease is a relatively newly recognized phenomenon and has become a major focus of patient monitoring. This patient initially concealed her gambling, and without such behavior being recognized as a possible adverse event of AEMs, it might have remained (and may remain) unexplained.

4. Conclusion

To our knowledge, this case represents the first report of possible compulsive gambling associated with the use of AEMs, either LTG or LEV or their combination. It highlights a potential adverse effect of AEMs that may be more widespread if actively sought as this patient was able to initially conceal it until the problem became unmanageable.

Contributorship statement

Roy G Beran —study concept and design.
Susanne Storrier —acquisition of data.

Susanne Storrier, Roy G Beran —analysis and interpretation.
Roy G Beran —critical revision of the manuscript for important intellectual content.
Roy G Beran —study supervision.

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