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Visual Disturbances Secondary to the Use of Lamotrigine in a Patient Suffering from Bipolar Depression: Case Report

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Abstract **Case Report**

Lamotrigine is a second-generation anticonvulsant approved for the treatment of epilepsy. Like other antiepileptics, lamotrigine is also used off-label in bipolar disorders as a mood stabiliser. We describe the case of a patient suffering from bipolar disorder type 2 who developed ocular toxicity following the use of lamotrigine in the management of his mental illness. The vast majority of these visual side effects are non-serious, rare, of mild to moderate intensity. And they appear to be reversible once treatment is stopped.

Keywords: mental illness, Lamotrigine, epilepsy, bipolar disorders.

1. INTRODUCTION

Lamotrigine is a second-generation anticonvulsant approved by the FDA for the treatment of epilepsy. Like other antiepileptic drugs, lamotrigine is also used off-label in bipolar disorder as a mood stabiliser.

We describe the case of a patient suffering from bipolar disorder type 2 who developed ocular toxicity following the use of lamotrigine for the management of his mental illness.

2. CASE CLINIQUE: OBSERVATION

- Mr M.E, 28 years old, single; no stable occupation.
- No particular pathological antecedents,
- The patient presented to the psychiatric consultation at the HIN hospital for a depressive episode characterised by bipolar disorder type 2, which necessitated the use of a thymoregulator (Lamotrigine).
- The dose of lamotrigine was gradually increased (to 300 mg/d after 6 weeks).
- Good improvement in psychiatric symptoms was noted.
- He was then identified as having symptoms consistent with ocular toxicityThe patient presented a slight reduction in vision and significant visual blur, which worsened with the progressive increase in the dosage of lamotrigine.
- A clinical ophthalmological examination showed no abnormalities.
- Decreasing the dose produced a clear clinical improvement in visual symptoms
- At a dose of 50 mg/d of lamotrigine: total disappearance of visual blur
- Lamotrigine was subsequently replaced by Quetiapine (300 mg/day).

3. DISCUSSION

Lamotrigine can cause a number of undesirable effects, and visual disorders have been described in several studies.

Among these effects, blurred vision was reported by 23% of patients receiving lamotrigine monotherapy in a study conducted by Arndt CF and colleagues in 2005 [1].

Diplopia may occur in cases of acute toxicity resulting from the pharmacological interaction of lamotrigine co-administered with carbamazepine [2, 3], and when lamotrigine is administered alone at high doses [3]. In a pooled comparison of two clinical databases of patients taking lamotrigine, diplopia was reported as an adverse event in 5.4% of patients treated with lamotrigine, compared with 0.6% receiving placebo (Messenheimer *et al*., 2000) [4].

Rotatory nystagmus may occur in acute toxicity following lamotrigine overdose (O'Donnell. J *et al*., 2000) [5].

Only high-dose lamotrigine patients showed visual field constriction, but visual field normalisation after lamotrigine dose reduction suggests that retinal damage is reversible [1].

No irreversible visual field damage has been observed in patients treated with LTG, although dose-dependent retinal toxicity may have been present [1].

Lamotrigine is a broad-spectrum antiepileptic drug. It acts by inhibiting sodium channels and inhibiting calcium currents [6]. Lamotrigine influences the release of excitatory neurotransmitters, in particular by inhibiting glutamate [7].

Lamotrigine treatment may induce a significant increase in GABA levels in the brain; consequently, retinal GABA levels may also be higher. This mechanism of action may explain the electrophysiological changes observed after lamotrigine treatment.

4. CONCLUSION

In recent years, the effects of thymoregulatory drugs on visual function have been widely collected and studied.

Of these commonly used drugs, the vast majority are associated with non-serious visual adverse effects of mild to moderate intensity.

Lamotrigine causes only very rare, reversible and dose-dependent symptomatic visual problems.

These adverse reactions, although they appear to be reversible after treatment has been stopped, should be systematically investigated after each use of this therapy.

A reminder to the prescribing physician of the ophthalmological side-effects is therefore of obvious importance.

REFERENCES

1. Arndt, C. F., Husson, J., Derambure, P., Hache, J. C., Arnaud, B., & Defoort-Dhellemmes, S. (2005). Retinal electrophysiological results in patients receiving lamotrigine monotherapy. *Epilepsia*, *46*(7), 1055-1060.
2. Besag, F. M. C., Berry, D. J., Pool, F., Newbery, J. J., & Subel, B. (1998). Carbamazepine toxicity with lamotrigine: pharmacokinetic or pharmacodynamic interaction?. *Epilepsia*, *39*(2), 183-187.
3. Loiseau, P. (1996). Tolerability of newer and older anticonvulsants: a comparative review. *CNS Drugs*, *6*, 148-166.
4. Messenheimer, J. A., Giorgi, L., & Risner, M. E. (2000). The tolerability of lamotrigine in children. *Drug Safety*, *22*, 303-312.
5. O'Donnell, J., & Bateman, N. (2000). Lamotrigine overdose in an adult. *Journal of Toxicology: Clinical Toxicology*, *38*(6), 659-660.
6. Stefani, A., Spadoni, F., Siniscalchi, A., & Bernardi, G. (1996). Lamotrigine inhibits Ca2+ currents in cortical neurons: functional implications. *European journal of pharmacology*, *307*(1), 113-116.
7. Meldrum, B. S. (1996). Update on the mechanism of action of antiepileptic drugs. *Epilepsia*, *37*, S4-11 (suppl 6).