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Lithium-induced downbeat nystagmus

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ABSTRACT

We report the case of a 76-year old lady under lithium carbonate for a bipolar disorder who presented with a suspected optic neuritis. A typical lithium-induced downbeat nystagmus was observed. Discontinuation of lithium therapy resulted in frank improvement in visual acuity and disappearance of the nystagmus.

1. Case report

A 76-year-old lady with a history of heavy tobacco use, osteoporosis, bipolar disorder stabilized under lithium carbonate (250 mg twice a day), and hypertension treated with ramipril (despite caution against the association of angiotensin converting enzyme inhibitors with lithium) was referred to the Internal Medicine outpatient clinic by her ophthalmologist to investigate a suspected bilateral optic neuritis. Her visual acuity (VA) had dropped from 20/25 (right eye) and 20/32 (left eye) to 20/200 for both eyes in one year. No papilledema was noticed. Brain and optic nerve MRI were normal, and visual evoked potentials were abnormal with a mixed axonal and myelinic pattern. Examination revealed a downbeat nystagmus (**Video**). Biological investigations showed a lithium plasma level of 1.13 mmol/L [therapeutic range: 0.5–1 mmol/L], a slightly elevated calcium level of 2,58 mmol/L, and an estimated creatinine clearance of 75 ml/min/1.73 m^2 (according to CDK-EPI formula). Vitamin B12 and thiamin were within normal levels. Visual symptoms and signs were attributed to lithium therapy despite lack of overt toxicity, and lithium carbonate was discontinued. One month later, VA was respectively 20/25 (right eye) and 20/32 (left eye). Visual field testing, although considered unreliable, showed a bilateral concentric defect. VA remained stable within the next months, and when seen 9 months later, the downbeat nystagmus had markedly diminished.

Supplementary video related to this article can be found at http://dx.doi.org/10.1016/j.ajoc.2017.06.012.

2. Discussion

Lithium-induced downbeat nystagmus (related to brainstem toxicity) has long been described as a side-effect unrelated to lithium plasma levels.[1] A review of 12 cases published in 1989[2] suggested that nystagmus could persist despite discontinuation of the drug. Other ocular motor defects, such as horizontal gaze palsy, may also be lithium-induced.[3] Although optic nerve dysfunction secondary to long-term use of lithium salts have been reported, with or without papilledema,[4] and despite improvement of vision after discontinuation of lithium, we could not definitely document the optic neuritis suspected in that particular case.

3. Conclusion

Ophthalmologists should be aware of this rare complication of lithium therapy.

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Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

Conflicts of interest

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