Resolution of superior oblique myokymia with memantine

Saurabh Jain, MRCPht,a Shegufta J. Farooq,b and Irene Gottlob, MDb

We describe a novel treatment of superior oblique myokymia. A 40-year-old woman was treated with gabapentin for this disorder with partial success and reported significant side effects including loss of libido and weight gain. After a drug holiday, memantine therapy was initiated resulting in a substantial improvement in her symptoms with far fewer side effects and stability on long-term maintenance therapy.

Superior oblique myokymia is an uncommon, monocular eye movement disorder involving contractions of the superior oblique muscle causing monocular oscillopsia and diplopia.1,2 We describe a novel treatment for this disorder using memantine.

Case Report

A 40-year-old white woman presented with a 2-year history of intermittent episodes of paroxysms of right torsional oscillations with associated right eye pain. Her symptoms worsened with alcohol and were relieved by holding the right lid down. These symptoms lasted between 5 and 10 seconds and recurred many times a day. Visual acuity was 6/6 in both eyes with no obvious strabismus or fourth nerve weakness. Episodes of fast pendular torsional movements of the right eye lasting approximately 5 seconds were detected on slit-lamp examination. The rest of the ocular examination was unremarkable and magnetic resonance imaging (MRI) was normal. Eye movements were recorded three-dimensionally using a video oculography technique at a sampling rate of 50 Hz (Strabas system, Sensomotoric, GmbH, Teltow, Germany; see Yousry et al3 for methods). They showed a pendular torsional nystagmus (amplitude of approximately 2° occurring at 1.4 Hz) without vertical component in the right eye, which coincided with the patient perceiving oscillopsia (Figure 1A).

Superior oblique myokymia was diagnosed and oral gabapentin with incremental doses of 300 mg at weekly intervals, until a total daily dose of 2400 mg, was prescribed. This controlled the symptoms for 12 months (Figure 1B) but the patient reported side effects consisting of tiredness, weight gain, and loss of libido. However, over the next 3 months she noticed a recurrence of sharp eye pains, double vision, and headaches, and we decided to change her medication.

The gabapentin was discontinued for 2 weeks and the patient was started on oral memantine at 5 mg daily increasing by 5 mg every 3 days to a maximum dose of 20 mg in two divided doses.

She reported a substantial improvement in her symptoms with the memantine, with far fewer headaches, no more diplopia, and no systemic side effects. Eye movement recordings did not show any abnormal torsional movements with memantine (Figure 1C). The patient had been on the treatment for 6 months at the time of this report and remained symptom free with no side effects.

Discussion

Superior oblique myokymia is typically an idiopathic disorder with a variable clinical presentation. In some cases researchers have described an association with vascular compression of the trochlear nerve at the root exit zone, usually by a branch of the posterior cerebral or superior cerebellar artery. This vascular compression may not be detected on a routine MRI scan and therefore a specific
protocol may be required. This association has also resulted in the technique of microvascular decompression as a surgical treatment modality for superior oblique myokymia. Williams and colleagues recently emphasized the potential benefits of medical treatment for superior oblique myokymia in contrast to surgical options. Carbamazepine, the initial drug of choice, had serious potential side effects including leucopenia, acute renal failure, thromboembolism, and arrhythmias, and the blood counts and hepatic and renal function needed to be monitored regularly. Therefore drugs with a more favorable safety profile were evaluated and phenytoin, propanolol, baclofen, and oral and topical beta-blockers have all been used with varying degrees of success. In addition to the drugs listed above, gabapentin has recently been reported as an effective drug for controlling superior oblique myokymia.

Gabapentin acts as a glutamate antagonist by inhibiting N-methyl-d-aspartate receptors or by influencing voltage-sensitive sodium and calcium channels. We therefore decided to use memantine, which is an N-methyl-d-aspartate receptor antagonist with similar antiglutaminergic action.

We have found gabapentin and memantine to be effective in the treatment of congenital and acquired nystagmus. Both drugs lead to subjective improvement in oscillopsia and reduction in amplitude of nystagmus in both groups. Interestingly, in patients with multiple sclerosis, memantine has been reported to reduce nystagmus that was unresponsive to gabapentin. Its action in superior oblique myokymia could be similar to the one in nystagmus.

Although no definitive recommendations can be made based on this single report, in this case, memantine was effective in controlling symptoms and was tolerated well by the patient.

Acknowledgments

This study was supported by the Ulverscroft Foundation.

References