MAGNESIUM THERAPY

Strong theoretical basis for role of magnesium deficiency in glaucomatous disease

by Roibeard O’hEineachain in Copenhagen

Eyes with primary open-angle glaucoma (POAG) have lower levels of magnesium than do eyes without the condition. Moreover, their condition appears to respond well to treatment with a medication containing magnesium, said Lusine Arutyunyan MD, Helmholtz Research Institute of Eye Diseases, Moscow, Russia, delivering a paper that reported a study initiated by Prof Elena Iomdina of same institute.

“Trace element analysis revealed a deficiency of magnesium in all the media and tissues of glaucomatous eyes we looked at. In addition, our first results with magnesium-based treatment showed a positive effect on IOP and the biomechanical properties of conical scleral capsule and on the structural and functional conditions of eyes with primary open angle glaucoma,” Dr Arutyunyan told the 10th European Glaucoma Society Congress in Copenhagen.

In one study, the Moscow Helmholtz Institute researchers obtained 0.1ml to 0.2ml samples of aqueous humour as well as scleral tissue samples from 18 POAG patients aged 49 to 86 who were undergoing sinus trabeculectomy with preventive posterior scleral trepanation. They also obtained aqueous samples from 16 cataract patients without glaucoma. They then performed trace element analysis of the fluids and tissue using a mass spectrometer with ionisation in inductively coupled plasma (ELAN DRC II, Perkin Elmer, USA).

They found that the mean magnesium concentration in scleral samples of eyes with initial/moderate POAG was 19.3 mg/l, and in eyes in the advanced stages of the disease it was 17.5 mg/l, she noted. That compares to a mean magnesium concentration of 177 mg/l in normal anterior sclera (p < 0.001), she said.

Magnesium levels were also lower in aqueous samples from eyes with glaucoma, where the mean concentration was 5.9 mg/l in initial to moderate POAG and as low as 3.0 mg/l in eyes with advanced disease. That compared to a mean magnesium concentration of 6.7 mg/l in eyes without glaucoma.

Magnesium therapy

To assess the potential of magnesium therapy in the treatment of glaucoma, Dr Arutyunyan and her associates conducted a trial in which 28 POAG patients ranging in age from 42 to 72 years received Magnerot (Wörwag Pharma), a magnesium-containing drug, at a dosage of two tablespoons, amounting to one gram of magnesium, three times a day during the first week, then one to two tablespoons two to three times a day. A further 16 POAG patients did not receive magnesium therapy. All received standard IOP-lowering medication.

Dr Arutyunyan noted that at final follow-up, the tear film concentration of magnesium in the eyes of magnesium-treated patients had increased from a baseline value of 0.0034 mg/l to 0.28 mg/l, approaching the normal value of 0.66 mg/l. In addition, computer perimeter showed that there was a significant increase of the mean total visual field, from 426.5 degrees to 452.5 degrees among magnesium-treated patients (p < 0.05).

The improvements in visual field occurred mainly in patients with moderate glaucoma.

Overall, among patients receiving magnesium therapy the visual fields improved in 71.9 per cent, remained unchanged in 20.8 per cent, and got worse in 7.3 per cent. By comparison, among patients in the control group, visual fields improved in 33 per cent of patients, remained unchanged in 25 per cent, and got worse in 42 per cent.

The magnesium-treated patients also had a significant reduction in mean IOP from initial values and a significantly lower mean IOP than the control group by the end of the trial (p < 0.05). That is, in the magnesium treatment group IOP measured with the ORA (Reichert) and calculated according to Goldmann tonometry was 18.6 mmHg before treatment and 15.3 mmHg after treatment. By comparison, the mean IOP in the control group was 18.7 mmHg at baseline and 18.5 mmHg at the end of the trial.

Trace elements imbalance

Dr Arutyunyan noted that there are several ways in which a magnesium deficiency might bring about glaucomatous changes in the eye. Magnesium deficiency can cause impairment of the neural, circulatory and connective tissues throughout the body and including the eye, she noted. “We know that magnesium is needed to protect neuron elements of the retina and the optic nerve against neurodegeneration and to regulate the metabolism of pathologically changed connective tissue structure of glaucomatous eyes,” she said.

She noted that magnesium deficiency can lead to trace element imbalance which in turn impairs the enzyme system of neural mitochondria, provoking caspase activation, leading ultimately to apoptosis. In addition, magnesium deficiency excites the glutamate and NMDA receptors that contribute to apoptosis.

Moreover, research has shown that magnesium deficiency can increase the viscosity of blood to the point that it decreases the blood supply to the glaucomatous eye.

Furthermore, magnesium deficiency activates the cross-linking of collagen and elastin which, combined with the impairment of matrix metalloproteinase activity, brings about a granularisation of the connective tissue, increasing scleral rigidity in glaucoma as a consequence, she pointed out.

Commenting on the study, Keith Martin PhD, Cambridge University Centre for Brain Repair, Cambridge, UK, said that there is a strong theoretical basis for a role of magnesium deficiency in glaucomatous disease. However, he pointed out that the positive effect of magnesium therapy on visual fields seen in this pilot study will require confirmation by a placebo-controlled trial. He cautioned that the dosages used in the trial might be toxic to some patients, particularly those with renal complaints.