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The Influence of Magnesium on Visual Field and Peripheral Vasospasm in Glaucoma

Key Words
Calcium channel blockers
Magnesium
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Abstract
Previous studies indicated calcium channel blockers to be of some help for normal-tension glaucoma patients. The present study evaluates the effect of magnesium, a 'physiological calcium blocker', in 10 glaucoma patients (6 with primary open-angle glaucoma, 4 with normal-tension glaucoma). All patients had a digital cold-induced vasospasm. Magnesium (121.5 mg) was administered twice a day for a month. After 4 weeks of treatment, the visual fields tended to improve. All three video-nailfold-capillaroscopic parameters [blood cell velocity (in mm/s) before and after cooling, cold-induced blood flow cessation (in seconds) as well as the number of capillaries per microscopic field which showed such a blood flow cessation] as well as digital tempertature improved significantly. Systemic blood pressure and pulse rate remained stable. In conclusion, magnesium improves the peripheral circulation and seems to have a beneficial effect on the visual field in glaucoma patients with vasospasm.

Introduction

Calcium channel blockers (CCBs) are often used to treat vascular disorders, such as coronary disease, angina pectoris, cardiac arrhythmias, systemic hypertension and Raynaud's phenomenon, as well as for prophylaxis against migraine.

These drugs have been reported to have a beneficial influence on the visual field in some conditions, such as normal-tension glaucoma [1, 2]. This effect might be due to an increase in ocular blood flow [1–7]. However, the effects of CCBs are discussed controversially [8, 9], and, in addition, they often induce side effects, such as swelling of the periorbita or of the legs [10].

Magnesium, 'nature's physiological calcium blocker' [11], is already prescribed in different diseases, such as vascular disorders [12, 13], arrhythmias due to acute myocardial infarction [14], chronic alcoholism [15] as well as toxemia of pregnancy [16] and others.

The present study analyzes the effect of magnesium on the visual field and on the peripheral blood flow in patients with the combination of glaucoma and vasospasm.

Physiological as well as pathological vasoconstrictions are mediated by intracellular influx of calcium, either from the extracellular environment or from intracellular stores [17]. CCBs partially inhibit that influx, and magnesium has a very similar effect. Therefore, it is called 'nature's physiological calcium blocker' [11]. The effect of magnesium on the vascular smooth muscle cells was reported [18] many years ago. High levels of extracellular magnesium lower the baseline vascular tension and decrease the reaction to contractile agonists, while low levels increase contraction and reactivity to contractile agonists, such as catecholamines [19]. Since magnesium is found primarily in the intracellu-

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lar compartment, the circulating magnesium concentration may not accurately reflect intracellular stores [20], and pathological states, such as magnesium deficiency, are difficult to diagnose. In addition, even severe deficiency may be asymptomatic [21]. Furthermore, diuretics decrease magnesium renal tubular reabsorption and magnesuria [22].

In this paper, we describe our first experience with magnesium treatment in 10 glaucoma patients who were progressive despite normal or normalized intraocular pressure. All these patients suffered from a so-called vasospastic syndrome [23].

Subjects and Methods

In this pilot study, we included 10 glaucoma patients, 7 women and 3 men. The mean (±SD) age was 57±13 years. Six patients had primary open-angle glaucoma and 4 normal-tension glaucoma. All patients had marked and progressive visual-field defects despite normal or normalized intraocular pressure and suffered from a vasospastic syndrome confirmed by video nailfold capillaroscopy [24, 25].

For the purpose of this study, magnesium (Magnesiocard® granules, Verla-Pharm) was administered at a dose of 121.5 mg twice a day to all 10 patients for a month. Visual-field, video nailfold capillaroscopy and determinations of systemic blood pressure, pulse rate and finger temperature were performed before and after 1 month of treatment. All patients had had extensive previous experience in Octopus automated perimetry. For each patient one eye was selected randomly. Program G1 [26] was performed before and after treatment. Mean defect (MD) and square root of loss variance (SQRT-LV) were taken as perimetric variables for statistical analysis. Video nailfold capillaroscopy included the measurement of blood cell velocity (in mm/s) before and after cooling, the cold-induced blood flow cessation (in seconds) as well as the number of capillaries per microscopic field which showed such a blood flow cessation. Measurement was done in a room with constant temperature at 23 °C. Paired t tests as well as the nonparametric Wilcoxon tests were performed for statistical analysis.

Results

After 4 weeks of treatment, the visual-field defects tended to decrease (mean of the index MD±SEM = 9.9±1.8 dB before and 8.9±1.9 dB after treatment). The mean SQRT-LV ±SEM decreased from 6±0.8 to 5.4±0.6 dB. These visual-field defects did, however, not reach statistical significance ($p = 0.09$; fig. 1). If we consider individual patients, 8 improved and 2 deteriorated. One of them deteriorated by several decibels due to an increased cataract.

The parameters of peripheral blood flow improved significantly after 4 weeks of treatment: blood cell velocity was faster after local cooling ($p<0.01$), cold-induced blood flow cessation was shorter ($p<0.001$) and the number of capillaries per microscopic field which showed blood flow cessation during cold provocation was lower ($p<0.05$; table 1).

The mean digital temperature±SEM increased from 2 ±1 to 31±1 °C ($p<0.05$).

Systemic blood pressure and pulse rate remained stable (table 2).

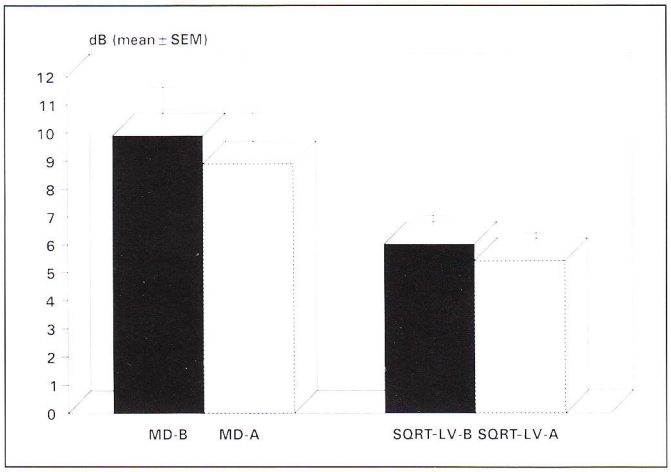

Fig. 1. Mean visual-field indices, MD and SQRT-LV, before (B) and after (A) treatment. A strong tendency for improvement can be observed.

Table 1. Video-nailfold-capillaroscopic variables (means ± SEM) before and after treatment with magnesium

	Before treatment	After treatment
Blood cell velocity after cooling, mm/s	0.05±0.01	0.023±0.07**
Time of cold-induced blood flow cessation, s	75.4±7.5	25.7±11***
Capillaries with blood flow cessation, n/microscopic field	4±1	2±1*

*p<0.05, **p<0.01, ***p<0001, Wilcoxon test.

Table 2. Digital temperature, blood pressure and pulse rate (means ± SEM) before and after treatment with magnesium

	Before treatment	After treatment
Digital temperature, °C	28±1	31±1*
Blood pressure (systolic), mm Hg	130±6	129±5
Blood pressure (diastolic), mm Hg	83±2	82±3
Pulse rate, beats/min	72±3	71±3

*p<0.05.

Discussion

CCBs seem to have a beneficial effect in some glaucoma patients, especially in those cases where the optic nerve head is not yet dramatically damaged [1–3, 5, 7–10]. This present study reports a similar beneficial influence of magnesium. Ten patients who had both glaucoma and vasospasm were treated with magnesium. After 1 month of treatment, the peripheral circulation and the finger temperature improved significantly, whereas the visual field showed a clear tendency towards improvement. However, the visual-field improvement did not reach statistical significance. These first results look quite promising.

Since magnesium is a physiological CCB [11] and induces peripheral and coronary vasodilation [27], it is tempting to speculate that it can also dilate the ophthalmic blood vessels.

As mentioned before, CCBs often induce side effects, whereas the good safety profile of magnesium – concomitantly with its possible ocular vasorelaxing properties – may open up new possibilities in the treatment of glaucoma patients suffering from vascular disorders. Therefore it seems worthwhile to make a placebo-controlled study on a large population.

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