

Serum Lipids and Magnesium Status in Haemodialysis Patients

I. Tzanakis¹, K. Virvidakis², A. Tsomi³, St. Kagia¹, N.Girousis¹, VL. Spadidakis¹,
A. Papadaki¹, N. Karefylakis¹, N. Kallivretakis¹

¹Nephrological Department, General Hospital of Chania, Chania,

²Third Medical Department and ³Laboratory of Pathological Physiology, University of Athens, Athens

Introduction

Studies in lab-animals provide enough evidence that magnesium (Mg) deficiency is associated with hypercholesterolemia and/or hypertriglyceridemia, and that Mg supplementation ameliorates these abnormalities^{1,2}. Controversial reports are available regarding the effect of Mg on lipid profile in humans. Population studies have shown a positive or a negative or no correlation between serum magnesium and serum cholesterol in healthy subjects³. Data in renal patients are limited. Magnesium was given in patients with mild to moderate renal failure and hypercholesterolaemia and resulted to a significant reduction of serum cholesterol after 12 weeks⁴. In the only study concerning terminal renal patients Markel et al. found elevated serum lipids in association with low free Mg in patients with end-stage renal disease (ESRD) as well as in renal transplant recipients⁵. Mg supplementation has recently been found to result to significant decrease of serum lipids in hypomagnesemic renal transplant recipients⁶. Furthermore, an association between hypomagnesaemia and both types of diabetes has been found, while magnesium repletion has been resulted in a significant improvement of glycaemic control in these patients⁷.

The aim of this study was to examine the relationship between serum lipids and Mg status in haemodialysis (HD) patients.

Patients and methods

Intracellular (IcMg) and serum Mg (sMg) levels were measured in 94 HD patients (56 males and 38 females, mean age 65±15; range 37-83 years, 19 diabetic) and in 29 healthy controls (16 males and 13 females, mean age 63±14; range 40-79 years). Total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, lipoprotein (a) (Lpa) levels were also obtained from the monthly follow-up of these patients for the last 12 months and the mean values were used for statistical analysis. IcMg was estimated by determination of this ion in isolated peripheral lymphocytes using atomic absorption spectrometry. Mean values of sMg levels corresponding to the monthly follow-up of these patients for the last 12 months were used. Serum Mg was measured by autoanalyser (Hitachi 912, Tokyo; Boehringer Mannheim Diagnostics, Germany). Normal values: sMg 1.2-2.1 mEq/l, IcMg 40-65 nmol/g protein. An unpaired two-tailed student's t-test was applied to compare differences of magnesium concentrations between the examined groups. A multiple regression linear

analysis was performed to examine the correlation between both sMg and IcMg levels and the serum lipids based on the Wald's statistic and using 5% of the probability for enters and 10% of the probability of removal a variable from the model. P value of <0.05 was considered statistically as significant.

Results

Table 1. Correlations between sMg and serum lipids in patients

	Non diabetic patients R, P-value	Diabetic patients R, P-value
t. Cholesterol	-0.16, ns	0.05, ns
HDL-Cholesterol	-0.03, ns	0.30, ns
LDL-Cholesterol	-0.15, ns	-0.23, ns
Triglycerides	0.13, ns	0.30, ns
Lipoprotein (a)	0.17, ns	0.16, ns

sMg, serum magnesium

Table 2. Correlations between IcMg and serum lipids in patients

	Non diabetic patients R, P-value	Diabetic patients R, P-value
t. Cholesterol	0.17, ns	0.02, ns
HDL-Cholesterol	-0.07, ns	0.16, ns
LDL-Cholesterol	0.18, ns	0.11, ns
Triglycerides	0.14, ns	0.02, ns
Lipoprotein (a)	0.04, ns	0.31, ns

IcMg, intracellular magnesium

Mean values of sMg were 2.81 ±0.32 in diabetic patients and 2.91±0.33 in non-diabetic patients (p=ns) and 1.72±0.20 mEq/l in controls (vs all patients, p<0.05). IcMg were 57.33±20.25 in diabetic patients and 57.14±11.25 in non-diabetic patients (p=ns) and 58.79±11.25nmol/g protein controls (vs all patients, p=ns). Patients' serum lipids were as follows: Total cholesterol 247.93±75.53, LDL-cholesterol 152.06±44.66, HDL-cholesterol 41.13±8.55, triglycerides 276.13±113.02, Lp(a) 48.46±46.31 mg/dl in

HD diabetic patients, and total cholesterol 245.04 ± 51.19 , LDL-cholesterol 157.45 ± 47.46 , HDL-cholesterol 43.37 ± 7.65 , triglycerides 227.41 ± 112.19 , Lp(a) 56.46 ± 54.59 mg/dl in non-diabetic HD patients. No significant correlations between sMg or IcMg and each one of the serum lipid parameters neither in diabetic nor in non-diabetic HD patients were found (Tables 1 and 2).

Conclusion. Elevated serum but normal intracellular magnesium levels were found in the haemodialysis patients we studied. Serum lipids were not correlated with intracellular and serum magnesium neither in diabetic nor in non-diabetic HD patients. It is very likely that magnesium status modulates serum lipids only in cases of magnesium deficiency.

References

1. Altura BM, Altura BT. Magnesium and cardiovascular biology: an important link between cardiovascular risk factors and atherogenesis. *Cell Mol Biol Res* 1995; 41: 347-59
2. Baydas B, Karagoz S, Meral I. Effects of oral zinc and magnesium supplementation on serum thyroid hormone and lipid levels in experimentally induced diabetic rats. *Biol Magnes Res* 2002; 88: 247-253
3. Purvis JR, Movahed A. Magnesium Disorders and Cardiovascular Diseases. *Clin Cardiol* 1992; 5: 556-568
4. Kirsten R, Heintz B, Nelson K, Sieberth HG, Oremek G, Hasford J, Speck U. Magnesium pyridoxal 5-phosphate glutamate reduces hyperlipidaemia in patients with chronic renal insufficiency. *Eur J Clin Pharmacol* 1998; 34(2): 133-137
5. Markel MS, Altura BT, Sarn Y et al. Deficiency of serum ionized magnesium in patients receiving hemodialysis or peritoneal dialysis. *ASAIO J* 1993; 39: 801-804
6. Gupta BK, Glicklich D, Tellis VA. Magnesium repletion therapy improved lipid metabolism in hypomagnesaemic renal recipients: a pilot study. *Transplantation* 1999; 67(11): 1485-1487
7. Lima MD, Cruz T, Pousada JC et al. The effect of magnesium supplementation in increasing doses on the control of type 2 diabetes. *Diabetes Care* 1998; 21: 682-686