Diabetologia (1995) 38: 629–634

Diabetologia

Letters to the editor

Hypomagnesaemia in childhood IDDM and risk of nephropathy

Dear Sir,

In their recent letter Pickup and colleagues [1] mentioned that they could not demonstrate any gross deviation of magnesaemia from normal levels in proteinuric diabetic patients. Although there are some published studies on the role of hypomagnesaemia in the development of certain complications of non-insulin-dependent diabetes mellitus (NIDDM) [2, 3] and on Na^+-dependent inositol transport in vitro [4] its effect in insulin-dependent diabetes (IDDM) has not been documented. Therefore, we investigated the relationship between magnesaemia and albuminuria in children with IDDM. We studied 80 children and adolescents with IDDM of differing durations (Table 1). Two serum samples obtained at different times were analysed for magnesium using calmagite kits. Twenty-four-hour urine collections were made at least three times at monthly intervals. Albumin was measured using double antibody radioimmunoassay. The mean values for two serum magnesium levels and three urinary albumin excretion rates were calculated. Hypomagnesaemia was found in 24 patients according to the reference criteria of our laboratory (< 0.70 mmol/l). Microalbuminuria was found in 30 patients (38 %) defined as urinary albumin excretion greater than 20 mg/min; 11 of the patients (14 % of total) were ascertained as having incipient nephropathy (microalbuminuria in at least two of three urine samples).

We compared the mean urinary albumin excretion rates of the hypo- and normomagnesaemic groups. Hypomagnesaemic patients had significantly higher albuminuria ($p < 0.05$). Age, diabetes duration and glycated haemoglobin did not differ significantly between the two groups (Table 1). We also found a negative correlation between serum magnesium levels and urinary albumin excretion ($r = -0.23$, $p < 0.03$). We then compared serum magnesium levels of the microalbuminuric and normoalbuminuric patients without finding any significant difference. However, serum magnesium levels did differ significantly when we compared the patients with and without incipient nephropathy ($p < 0.05$).

Although serum magnesium measurement is not a sensitive indicator of total body magnesium it is specific and can be used in almost all cases except in aminoglycoside treatment and severe diuresis [5]. We concluded that hypomagnesaemia has a relationship with "persistent" microalbuminuria and can be regarded as a risk factor for diabetic nephropathy.

Yours sincerely,

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References

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Table 1. Clinical characteristics of patients with normo- and hypomagnesaemia

	Age (years)	Sex (female/male)	Diabetes duration (years)	HbA_1 (%)	Mean urinary albumin excretion rate (μg/min)
Normomagnesaemic patients ($n = 56$)	10.9 ± 3.9	30/26	3.8 ± 3.1	9.7 ± 2.4	10.8 ± 10.8
Hypomagnesaemic patients ($n = 24$)	12.5 ± 4.8	14/10	4.6 ± 3.4	11.1 ± 3.9	20.7 ± 21.0

Results are mean ± SD; HbA_1 glycated haemoglobin

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