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CLINICAL NOTE

Pathogenetic Mechanisms of Hypomagnesemia in Alcoholic Patients

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Summary

The aim of our study was to describe the possible pathophysiologic mechanisms of hypomagnesemia in alcoholic patients. A total of 127 chronic alcoholic patients admitted to our university hospital for causes related to alcohol abuse were studied. Hypomagnesemia was the most common electrolyte disturbance observed in 38 patients (29.9 %). In 18 of them inappropriate magnesiuria was evident, possibly due to hypophosphatemia, to metabolic acidosis or to a direct magnesiuric effect of acute alcohol consumption. The causes of hypomagnesemia in the remaining 20 patients were alcohol withdrawal syndrome and diarrhea. Respiratory alkalosis was evident in 10 hypomagnesemic patients and could have played a role in the development of hypomagnesemia. A decreased magnesium intake could also have contributed to the hypomagnesemia, especially in malnourished alcoholic patients. Hypomagnesemic patients more frequently had other acid - base and electrolyte abnormalities, such as hypophosphatemia, hypokalemia, hypocalcemia, and respiratory alkalosis, as compared with the normomagnesemic patients. Moreover, in hypomagnesemic patients serum magnesium levels were correlated with the indices of potassium and phosphorus excretion, suggesting that serum magnesium levels play a central role in the homeostasis of the other electrolytes. In conclusion, hypomagnesemia is the most common electrolyte abnormality observed in alcoholic patients, as a result of various pathophysiologic mechanisms.

Keywords :Alcoholism, hypomagnesemia, magnesiuria, hypokalemia, hypophosphatemia.

Introduction

Electrolyte abnormalities are usually observed in chronic alcoholics (1 - 5). Among them, hypomagnesemia is the most common and clinically significant disturbance in alcoholics admitted to a general medical ward (4,6,7). In a recently published study we have described the acid - base and electrolyte abnormalities in 79 alcoholic patients admitted to our university hospital (6). In the current study, we try to improve our analysis of the possible pathophysiologic mechanisms of hypomagnesemia in a larger group of alcoholic patients.

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Material and Methods

We studied 127 alcoholic patients [120 male, 7 female; mean age (± SD) 44 ± 14, range 29 - 72 years; weight 67 ± 8 kg; body mass index (BMI) 24 ± 2.4 kg/m^2] admitted to our hospital for causes related to alcohol abuse. For inclusion in the study a history of alcohol consumption of at least 3 drinks per day for more than 10 years and of heavy alcohol intake in the preceding year

was needed. The main reasons for the patients' admission are shown in Table 1. Patients with diabetes mellitus, renal failure (serum creatinine > 141.5 μmol/L), ascites, chronic obstructive lung disease, recent bleeding from the gastrointestinal tract, septic shock or other known causes of lactic acidosis, convulsions occurring one hour prior to blood sampling, as well as patients consuming drugs affecting acid - base status and electrolyte levels, such as diuretics, antacids, and potassium, phosphorus, and magnesium supplements were excluded from the study.

On their admission, a physical examination was performed and venous blood was obtained for the determination of serum osmolality (Posm), glucose, urea, creatinine, total proteins, albumin, lipid parameters (total cholesterol, triglycerides), sodium, chloride, potassium, magnesium, calcium, and phosphorus before any therapeutic intervention. Arterial blood was also obtained for blood gas measurements. In cases of hypoalbuminemia the corrected serum calcium was calculated by adding 0.2 mmol/L to the total serum calcium concentration for every 10 g/L decrement in serum albumin from the normal value (assumed to be 40 g/L) (8). At the same time a fresh urine specimen was tested for osmolality (Uosm), creatinine, sodium, chloride, potassium, magnesium, calcium, and phosphorus.

Urine and serum samples were analyzed for sodium, chloride, and potassium with a flame photometer, for calcium and magnesium with an atomic absorption spectrophotometer, and for phosphate by a colorimetric method. Arterial pH and PCO_2 were determined using a pH blood gas analyzer, and serum bicarbonate was calculated from blood hydrogen and blood carbon dioxide tension according to the Henderson - Hasselbach equation with an acidity exponent of 6.10 and a solubility coefficient of 0.0301. Serum and urine osmolality was assayed using a vapor pressure osmometer. Serum total protein concentrations were measured by the Lowry method, serum albumin concentrations by the biuret reaction, while serum and urine creatinine was estimated by a modification of the method of Jaffé. The coefficients of variation for all measured electrolyte parameters were lower than 3 %.

Table 1. Main reasons for patients'admission

Cause	Patients	
	Number	%
Alcohol withdrawal syndrome	25	19.7
Increased serum liver enzymes and / or hepatomegaly	27	21.2
Acute intoxication	26	20.5
Anemia	11	8.7
Diarrhea	7	5.5
Chronic alcoholic pancreatitis	7	5.5
Gastrointestinal symptoms (nausea, vomiting, gastritis, dyspepsia, epigastralgia)	6	4.7
Chronic myopathy	4	3.1
Peripheral neuropathy	6	4.7
Epistaxis	3	2.4
Ataxia	5	3.9

Standard formulas were used to calculate the fractional excretion of potassium, magnesium, and phosphorus. The transtubular potassium gradient (TTKG) was calculated from the equation :

$$TTKG = \frac{\text{Urine potassium} \div \text{Uosm / Posm}}{\text{Serum potassium}} \quad (9,10).$$

The renal tubular threshold concentration for phosphate ($TmPO_4^{---}$ / GFR) was determined by the nomogram of Walton et al. (11).

Abnormalities of serum electrolytes or acid - base metabolism were defined as follows : hyponatremia, by a serum sodium concentration < 135 mmol/L; hypernatremia, by a serum sodium concentration > 146 mmol/L; hypokalemia, by a serum potassium concentration < 3.5 mmol/L; hypomagnesemia, by a serum magnesium concentration < 0.65 mmol/L; hypocalcemia, by a serum calcium concentration < 2.10 mmol/L; hypophosphatemia, by a serum phosphate concentration < 0.77 mmol/L; hyperphosphatemia, by a serum phosphate concentration > 1.45 mmol/L; acidemia, by a blood pH < 7.34; respiratory alkalosis, by a blood pH > 7.46 and PCO_2 < 36 mmHg; and metabolic alkalosis, by a blood pH > 7.46 and serum bicarbonate concentration > 26 mmol/L. These cut - off values come from our laboratory after we examined a considerable number of samples from normal subjects and are in agreement with those stated by the manufacturers.

Acid - base and electrolyte parameters of the alcoholic patients were compared with those of 203 normal subjects who abstained from alcohol or consumed only very small amounts of alcohol matched for age and sex .

Statistical analysis was performed by the unpaired t - test, or by the x^2 test . Linear regression analysis was used for the correlation between parameters.

Results

Sixty - nine out of the 127 patients (54.3 %) had at least one acid - base or electrolyte abnormality (Table 2). The serum electrolyte and blood acid - base values are shown in Table 3. As compared with the normal subjects, the alcoholic patients had lower serum concentrations of potassium, magnesium, bicarbonate, calcium and phosphate, as well as lower values for arterial PCO_2, whereas there was no significant difference between the groups in

Table 2. Acid-base and electrolyte abnormalities in 127 alcoholic patients

Abnormalities	Patients	
	Number	%
Respiratory alkalosis	20	15.7
Metabolic alkalosis	7	5.5
Acidemia#	14	11
Hyponatremia	16	12.6
Hypernatremia	3	2.3
Hypokalemia	16	12.6
Hypomagnesemia	38	29.9
Hypocalcemia	26 (18)*	20.5 (14.2)*
Hypophosphatemia	37	29.1
Hyperphosphatemia	2	1.6

Due either to pure metabolic acidosis or mixed acid - base disorders

* The values in parentheses represent the number of patients and the percentage with true hypocalcemia

Table 3. Acid-base and electrolyte parameters in alcoholic patients and in normal subjects

Parameters	Alcoholic patients (n = 127)	Normal subjects (n = 203)	p
Arterial pH	7.41 ± 0.08	7.40 ± 0.04	NS
PCO_2 (mmHg)	35 ± 6	39 ± 3	< 0.05
in serum (mmol/L)			
bicarbonate	21.2 ± 3	24.2 ± 2.0	< 0.01
sodium	138 ± 3	140 ± 3.5	NS
chloride	100 ± 3	99 ± 4	NS
potassium	3.8 ± 0.3	4.4 ± 0.3	< 0.01
magnesium	0.7 ± 0.2	0.9 ± 0.3	< 0.01
calcium	2.15 ±0.15	2.4 ± 0.12	< 0.05
phosphorus	1.0 ± 0.26	1.23 ± 0.23	< 0.01

Values are means ± SD

serum sodium and chloride concentrations, as well as in arterial pH. Hypomagnesemia was the most common electrolyte disturbance: 38 patients (29.9 %) had decreased serum magnesium (< 0.65 mmol/L) with a range of 0.28 - 0.62 mmol/L. Twenty of them had a very low magnesium excretion ($FeMg^{++}$ < 2.5 %) suggestive of an magnesium conservation in the face of magnesium depletion. Sixteen of these twenty patients had alcohol withdrawal syndrome and 4 a chronic diarrheal syndrome. In the remaining 18 patients inappropriate magnesiuria ($FeMg^{++}$ > 2.5 %) was evident. Nine of them had severe hypophosphatemia (serum phosphorus < 0.64 mmol/L), six had acidemia (arterial pH < 7.34), while 3 patients were admitted to the hospital with symptoms of acute alcohol intoxication. Respiratory alkalosis was evident in 10 hypomagnesemic patients (seven with alcohol withdrawal syndrome). Ten hypomagnesemic patients were malnourished, with a serum albumin level lower than 28 g/L and a total lymphocyte count lower than 1.2 X 10^9 cells/L. Patients with hypomagnesemia more frequently had hypokalemia, hypophosphatemia, hypocalcemia, and respiratory alkalosis compared to the normomagnesemic patients (Table 4). Moreover, a significant decrease in serum potassium, bicarbonate, calcium and phosphate concentrations, as well as in PCO_2, and a significant increase in arterial pH were found in hypomagnesemic patients in comparison to patients with serum magnesium levels within normal limits (Table 5). All but one of the hypomagnesemic patients with hypokalemia had inappropriate kaliuria (FeK^+ > 6.5 %, TTKG > 2) (9,10,12). Moreover, the vast majority of hypomagnesemic patients with hypophosphatemia also had inappropriate phosphaturia ($FePO_4^{---}$ > 20 %, $TmPO_4^{---}$ / GFR < 0.87 mmol/L) (11,13). Serum magnesium levels in hypomagnesemic patients correlated well with the indices of both potassium and phosphorus excretion. Specificallly, an inverse correlation between serum magnesium levels and FeK^+ (%), (r = - 0.57, p < 0.001), TTKG (r = - 0.39, p < 0.02), and $FePO_4^{---}$ (r = - 0.47, p < 0.01) and a positive correla-

Table 4. Acid-base and electrolyte abnormalities in hypomagnesemic vs. normomagnesemic patients

Abnormalities	Hypo-magnesemic patients (n=38) number (%)	Normo-magnesemic patients (n=89) number (%)	p
Respiratory alkalosis	10 (26.3)	10 (11.2)	< 0.05
Metabolic alkalosis	2 (5.2)	5 (5.6)	NS
Acidemia#	6 (15.8)	8 (9)	NS
Hyponatremia	5 (13.1)	11 (12.3)	NS
Hypernatremia	1 (2.6)	2 (2.2)	NS
Hypokalemia	12 (31.6)	4 (4.5)	< 0.001
Hypocalcemia[1]	9 (23.7)	9 (10.1)	< 0.05
Hypophosphatemia	19 (50)	18 (20.2)	< 0.001
Hyperphosphatemia	1 (2.6)	1 (1.1)	NS

[1]true, # pH ≤ 7.34 due either to pure metabolic acidosis or mixed acid - base disorders

Table 5. Acid-base and electrolyte parameters in hypomagnesemic vs. normomagnesemic patients

Parameters	Hypo-magnesemic patients (n=38)	Normo-magnesemic patients (n=89)	p
	Mean values (± SD)		
Arterial pH	7.43 ± 0.08	7.40 ± 0.06	< 0.05
PCO_2 (mmHg)	32 ± 5	36.5 ± 5	< 0.01
in serum (mmol/L)			
bicarbonate	20 ± 3	23 ± 2.5	< 0.01
sodium	137.5 ± 3.2	138.6 ± 2.8	NS
chloride	99 ± 3	101 ± 3	NS
potassium	3.6 ± 0.4	4.0 ± 0.2	< 0.01
calcium	1.9 ± 0.16	2.2 ± 0.15	< 0.05
phosphate	0.90 ± 0.29	1.05 ± 0.21	< 0.01

tion between serum magnesium levels and $TmPO_4^{- -}$ / GFR ($r = 0.55$, $p < 0.001$) were observed.

Discussion

Hypomagnesemia along with hypophosphatemia were the most common electrolyte abnormalities in our alcoholic patients, who also exhibited a variety of acid - base and electrolyte disturbances. Decreased magnesium intake could have played a prominent role in hypomagnesemia, at least in some malnourished patients. A diet in which the primary source of calories is alcohol is severely magnesium deficient. Increased magnesium entry into cells, on the other hand, could have contributed to the development of hypomagnesemia in many patients. The possible causes of increased magnesium entry into cells were : a) respiratory alkalosis, which has been described as creating an acute decrease in serum magnesium levels due to a shift of magnesium to the intracellular compartment (14); b) alcohol withdrawal syndrome with excessive catecholamine release, as it is well known that exogenous catecholamines can significantly influence the transcellular magnesium shift (15). It has been reported, however, that hypomagnesemia in withdrawing alcoholics might be spurious to a certain extent. Plasma catecholamines are elevated in alcohol withdrawals, and the associated lipolysis allows mobilization of free fatty acids (FFA) which can precipitate with magnesium, thus contributing to the hypomagnesemia (16); c) Increased gastrointestinal magnesium losses in patients suffering from a chronic diarrheal syndrome.

Inappropriate magnesiuria was present in 18 out of the 38 patients and was of paramount importance for the development of hypomagnesemia. In some of these patients phosphate depletion could have been responsible for the inappropriate magnesiuria, which has been shown to arise from reduced magnesium reabsorption (17 - 20). Micropuncture experiments in dogs showed that this defect occurred in the loop of Henle as well as in the distal tubule and can be corrected by the administration of parathyroid hormone or phosphate (18). Metabolic acidosis may also be associated with renal magnesium excretion, which appears to result from diminished magnesium reabsorption in the thick ascending limb of Henle (21). In the 3 patients with acute alcohol consumption a direct magnesiuric effect of ethanol may have played a significant role in the increased magnesium excretion (22 - 24). Although the mechanism of acute alcohol - induced magnesiuria is unknown, two possibilities are favored; either a direct effect of ethanol on the tubular reabsorption of magnesium, or an increased production of a metabolic intermediate (ie, lactate) with the potential to bind magnesium when it is excreted by the kidney (25). The latter theory is supported by the strong correlation observed between urinary magnesium and lactate excretion following alcohol ingestion (26). However, studies have shown that the initial magnesiuria caused by ethanol decreased as hypomagnesemia developed (22 - 24, 27).

Recently, De Marchi et al (4) suggested a reversible tubular defect responsible for the enhanced urinary magnesium excretion that was seen in 21 % of chronic alcoholics. The inappropriate magnesiuria, as well as the other tubular dysfunctions observed, disappeared after 4 weeks of alcohol abstinence (4). Furthermore, transient hypoparathyroidism was reported during alcohol intoxication and might have enhanced renal magnesium excretion (28).

Hypomagnesemic patients more frequently had other acid - base and electrolyte disorders such as hypokalemia, hypocalcemia, hypophosphatemia and respiratory alkalosis as compared with the normomagnesemic patients.

Hypokalemia was the result of inappropriate kaliuresis, as it is well known that in hypomagnesemia an inability of the kidneys to conserve potassium is commonly seen (29,30). In fact, an inverse correlation between serum magnesium levels and indices of potassium excretion was found in our hypomagnesemic patients.

Hypomagnesemia can also lead to hypocalcemia. The mechanism whereby magnesium deficiency leads to hypocalcemia is multifactorial. Parathyroid gland function is abnormal, largely because of the impaired release of PTH. In addition, there is strong evidence for a skeletal resistance to the action of PTH (31,32).

Even though hypophosphatemia could be the cause of inappropriate magnesiuria and hypomagnesemia in some cases, it might also be the result of hypomagnesemia, since in experimental magnesium depletion phosphaturia is a common finding and seems to be corrected with magnesium repletion (33,34). In fact, in hypomagnesemic patients a good correlation between serum magnesium levels and indices of phosphorus excretion was noticed.

The coexistence of hypomagnesemia and respiratory alkalosis might be ascribable to the transfer of magnesium from extracellular to intracellular fluid due to respiratory alkalosis (14). However, this coexistence might also be the result of a common underlying cause such as alcohol withdrawal syndrome and acute intoxication.

In conclusion, hypomagnesemia is the most common electrolyte abnormality observed in alcoholic patients, resulting from various pathophysiologic mechanisms.

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