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Serum Magnesium, Potassium, Catecholamines and Cortisol after Non-fatal Myocardial Infarction: Possible Interrelations and Importance of Size and Site of Infarction

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Zusammenfassung

Die Autoren haben auf einen Myokardinfarkt folgende Hormon- und Elektrolyt-Veränderungen untersucht. Dazu bestimmten sie die Serumspiegel von Magnesium, Kalium, Katecholaminen und Kortisol im Laufe der ersten vier Tage nach Auftreten unverminderter Brustschmerzen bei 20 Patienten. Diese Messungen wurden 1 Jahr später wiederholt. Die mittlere Magnesi- und Kaliämie war unmittelbar nach dem Infarkt niedriger als ein Jahr später (M ± SD): Mg: 0,76 ±0,01 gegen 0,85 ± 0,02 mmol/l, p < 0,001; K: 4,1 ±0,03 gegen 4,3 ± 0,08 mmol/l, p = 0,02. Die mittlere Magnesiämie hat sich außerdem jedesmal dann als am schwächsten erwiesen, wenn der Infarkt ein größeres Ausmaß hatte (0,72 ± 0,01 gegen 0,79 ± 0,01 mmol/l, p < 0,.001) und mehr die untere Herzwand betraf als die vordere (0,69 ± 0,01 gegen 0,77 ± 0,01 mmol/l, p < 0,001).

Diese Unterschiede lassen sich nicht durch Abweichungen des mittleren Blutdrucks oder eine fraktionsweise unterschiedliche Magnesiumausscheidung durch die Nieren erklären. Ein Jahr nach dem Infarkt lag nur noch der mit dem Ausmaß des Infarkts zusammenhängende Unterschied der Magnesiämie vor (0,08 ±0,02 gegen 0,90 ±0,02 mmol/l, p < 0,001).

Zwischen Katecholaminen und Elektrolytkonzentrationen wurde kein Zusammenhang entdeckt. Die Kortisolämie war umgekehrt mit der Magnesiämie verbunden (p = 0,01).

Summary

Hormonal and electrolyte changes following acute myocardial infarction were studied by the determination of serum magnesium, potassium, catecholamines and cortisol during the first four days after the onset of unrelenting chest pain in 20 patients, and electrolytes were measured again one year later. Mean serum magnesium and potassium were lower shortly after infarction than one year later (M and SEM): Mg: 0.76 (0.01) vs 0.85 (0.02) mmol/l (p < 0.0001); K: 4.1 (0.03) vs 4.3 (0.08) mmol/l (p = 0.02). Mean serum magnesium was lower with larger than with smaller infarctions: 0.72 (0.01) vs 0.79 (0.01) mmol/l (p < 0.0001) and with inferior than with anterior wall infarctions: 0.69 (0.01) vs 0.77 (0.01) mmol/l (p < 0.0001).

These differences could not be explained by differences in mean arterial pressure or fractional renal magnesium excretion. One year later, only the difference in serum magnesium related to the size of infarction remained: 0.08 (0.02) vs 0.90 (0.02) mmol/1 (p < 0.001).

No associations were found between catecholamines and electrolytes. Cortisol was negatively related to serum magnesium (p = 0.01)

Résumé

Les auteurs ont étudié les modifications hormonales et électrolytiques consécutives à un infarctus du myocarde en déterminant les taux sériques de magnésium, de potassium, de catécholamines et de cortisol au cours des quatre premiers jours suivant l'apparition de douleurs thoraciques sans rémission chez 20 patients, puis en répétant les mêmes mesures un an plus tard. La magnésémie et la kaliémie moyennes ont été plus basses immédiatement après l'infarctus qu'un an plus tard (Mg: 0,76 ± 0,01 contre 0,85 ± 0,02 mmol/l, p < 0,0001; K : 4,1 ± 0,03 contre 4,3 ± 0,08 mmol/l, p = 0,02 ; M ± ESM). La magnésémie moyenne s'est également avérée plus faible lorsque l'infarctus était de taille plus importante (0,72 ± 0,01 contre 0,79 ± 0,01 mmol/l, p < 0,0001) et que sa localisation intéressait la paroi inférieure plut^ot qu'antérieure (0,69 ± 0,01 contre 0,77 ± 0,01 mmol/l. p < 0,0001).

Ces différences ne peuvent pas s'expliquer par des écarts entre les pressions artérielles moyennes ou entre les excrétions de magnésium par les différentes fractions rénales. Un an après l'infarctus, seule subsistait la différence de magnésémie liée à la taille de l'infarctus (0,08 ± 0,02 contre 0,90 ± 0,02 mmol/l, p < 0,001).

Il n'y a eu aucune relation entre les taux de catécholamines et les concentrations d'électrolytes. La cortisolémie a été inversement liée à la magnésémie (p = 0,01).

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Introduction

Metabolic changes may be of great importance after myocardial infarction. It is well known that hypokalaemia, as well as hypomagnesaemia may promote ventricular arrhythmias. Furthermore, decreased serum magnesium levels make coronary arteries contract, thus hindering myocardial perfusion. Hypokalaemia is thought to occur as a consequence of increased beta-2-receptor activity due to increased levels of catecholamines. This beta-2 activity seems also to be of importance in decreasing serum magnesium, in some way related to stimulation of lipolysis. We have been interested in investigating the short-term and possible long-term alterations in serum potassium and magnesium levels after myocardial infarction, and in looking at the putative association between catecholamines or cortisol and the profiles of these electrolytes.

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Study group

We studied 25 consecutive patients with AMI, admitted to a district

hospital. Five patients died shortly after admission due to extensive infarction and were left out of the study. Serum electrolytes, catecholamines and cortisol, and urinary magnesium excretion were measured every twelve hours after the beginning of infarction, i.e. the beginning of an unrelenting chest pain. These measurements were obtained for a total of 8 periods, i.e. 4 days. Arterial pressure was recorded as well, and serum magnesium and potassium were measured again after one year.

These patients were then subdivided according to site and size of infarction: according to the peak serum level of glutamate oxaloacetate transferase (GOT), the size of the myocardial infarction was considered small (GOT < 80 U/l) or large (GOT > 80 U/l), whereas the site of infarction was defined according to the ECG changes, with 8 patients sustaining an anterior wall myocardial infarction, 5 with a posterior wall myocardial infarction, while 7 could not be classified.

The whole study group consisted of 20 patients, 15 men and 5 women, with a mean age of 63.3 years. The mean duration between symptoms and admission was 12.8 hours.

Results

Hypomagnesaemia, as defined by a serum level of less than 0.70 mmol/l, was present in 6 patients, while hypokalemia, defined as a serum level of less than 3.5 mmol/l, occurred in 4 patients. Hypomagnesaemia was thus at least as common as hypokalaemia.

Shortly after infarction, both serum magnesium and potassium were lower than one year later, which is consistent with a temporary decrease of both cations early after infarction. Norepinephrine, epinephrine and cortisol were initially elevated, though the rise in catecholamines were smaller than reported in the literature.

Patients with a large infarction had on the average a lower serum magnesium level than those with a small infarction, both early and late after infarction. No such pattern was found for potassium. This difference could not be explained by differences in urinary magnesium excretion or arterial pressure. Fractional magnesium excretion was actually higher in patients with small infarctions who had a higher serum magnesium. Surprisingly, catecholamines were not different between those with a small infarction and those with a large non-fatal infarction. This lack of difference could not be explained by the use of morphinomimetics. Cortisol levels in patients with large infarctions was higher than in patients with small infarctions.

Patients with an inferior wall infarction had early after infarction a lower magnesium level than those with an anterior wall infarction. One year later, this difference had disappeared. No such pattern was found for potassium. The early difference could not be explained by differences in urinary excretion of magnesium, or differences in arterial pressure. Serum epinephrine was higher in patients with anterior wall infarction, who also had higher serum magnesium levels.

Linear regression analysis showed a consistent negative relation between serum magnesium level and cortisol levels, whereas no negative correlation could be found between the catecholamine levels and serum magnesium or serum potassium levels. We could even not corroborate the expected negative association between epinephrine and potassium levels. This might be explained by the fact that this hypokalaemic effect of epinephrine is usually seen immediately after elevation of its level; in our study, the time period studied was 96 hours.

Conclusions

From our data, we may conclude that early after myocardial infarction, both serum magnesium and serum potassium levels are temporarily decreased: hypomagnesaemia is at least as common as hypokalaemia. The decrease is related to the size and to the site of infarctions, in some way related to an altered internal distribution. The size of infarction has a long lasting influence or serum magnesium: late after infarction, patients with a large infarction still had a lower serum magnesium level than those with a small one. Cortisol levels disclosed a negative correlation to serum magnesium levels: this role of cortisol and the importance of size and site of infarction deserve further study, as well as long-term changes in serum magnesium, namely with respect to potential prognostic influences.

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