

Haemodialysis-induced electrolyte variation (serum calcium, magnesium and bicarbonate) and intradialytic heart rhythm disorders

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Abstract. Arrhythmias are common in patients with end-stage renal disease. Along with other factors, haemodialysis-related electrolyte changes may have an important pro-arrhythmogenic role. Objectives: to determine the serum ionogram, in order to see the haemodialysis-induced electrolyte variation and to correlate that with intradialytic heart rhythm disorders. Material and method: 42 patients with end-stage renal disease on maintenance haemodialysis were studied, with a mean age of 55 years. Holter ECG examination was performed during dialysis, so all the intradialytic heart rhythm disorders were recorded. Calcemia, magnesemia and serum bicarbonate level were analyzed before and after dialysis. Then the ranges of values were noticed, in order to determine the types of electrolyte variation produced by haemodialysis, as following: A. unchanged level, B. increase and C. decrease. Results: intradialytic heart rhythm disorders had a high prevalence. The main electrolyte changes produced by haemodialysis were the calcemia and serum bicarbonate increase ($p<0.001$), as well as the fall in prevalence of postdialytic hypocalcemia and metabolic acidosis ($p<0.001$). Intradialytic supraventricular premature beats were associated with types A and B of calcemia variation, compared to type C ($p=0.015$). Magnesemia was reduced insignificantly after dialysis. Intradialytic ventricular premature beats were mainly noted in patients who had postdialytic normal magnesemia ($p=0.039$). Haemodialysis significantly improved metabolic acidosis ($p<0.001$), although type C (serum bicarbonate decrease) was associated with atrial fibrillation occurrence, unlike types A and B ($p=0.002$). Discussions and conclusions: Intradialytic heart rhythm disorders have a high prevalence. This might be due to some dialysis-induced electrolyte changes, such as calcemia increase, correction of metabolic acidosis or even its occasional worsening, while magnesemia variation seems to be less important. Though, the pro-arrhythmogenic effect of the electrolytes imbalance and variation during dialysis, may depend on either some other intradialytic risk factors or the organic cardiac suffering.

Key Words: heart rhythm disorders, haemodialysis, electrolyte variation, intradialytic.

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Introduction

Heart rhythm disorders (HRD) are common in patients with chronic renal failure (CRF) on maintenance dialysis (Burton et al 2008). Arrhythmias are more likely to occur if there are electrolytes imbalance, anemia, hypoxemia or blood pressure variations. Uremic cardiomyopathy is characterized by interstitial fibrosis, which may lead to myocardial electrical instability (Leier et al 1992). There are many factors that work together for intradialytic HRD occurrence. Several triggers act on organic heart disease, resulting in the production of arrhythmias during dialysis. Intradialytic variability of these factors, as well as their interdependence, contribute to the validation of the pro-arrhythmogenic effect of dialysis (Kramer et al 1992; Selby et al 2007). Haemodialysis predisposes to sudden changes of serum electrolytes level. Serum calcium, magnesium and blood pH imbalance are usually met in dialyzed patients and are often associated with arrhythmias (Voroneanu et al 2009; Santoro et al 2006).

Our objectives were : recording HRD during haemodialysis, determination of calcemia, magnesemia and serum bicarbonate

level, before and after dialysis and definition of variation types of the analysed electrolytes and their correlation with intradialytic HRD.

Material and method

The study group was made up of 42 patients with chronic kidney disease stage 5 KDOQI undergoing chronic haemodialysis. The sex distribution of the patients was: 23 women (54.8%) and 19 men (45.2%). The patients' age ranged from 18 to 84 (54.94 ± 13.92 years). Haemodialysis was performed using a standard bicarbonate-based dialysis solution. All patients underwent intradialytic ECG Holter registration, in order to record HRD during this time. To determine serum ionogram (calcium, magnesium and bicarbonate), blood samples were taken before and so after dialysis. Electrolyte variation during hemodialysis was noted, taking into account the ranges of values (normal range, less than or more than ordinary), as follows: A. predialytic electrolyte status remains unchanged after dialysis; B. serum electrolyte level increases after dialysis; C. serum electrolyte

level decreases after dialysis. Data were statistically processed in the SPSS program. The results were considered statistically significant if $p < 0.05$.

Results

Most of the patients (97.6%) had intradialytic HRD. The prevalence and type of them are shown in fig. 1.

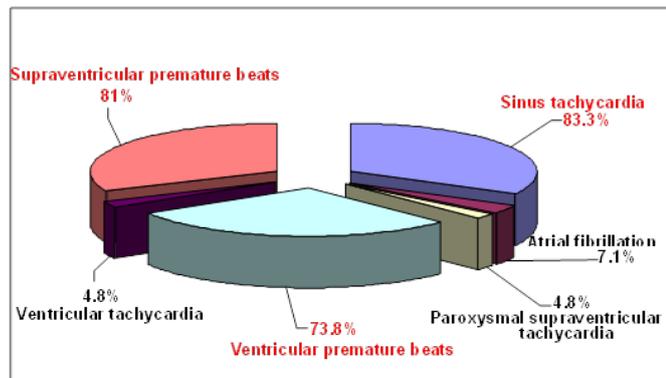


Fig. 1. Prevalence and type of the intradialytic HRD

Haemodialysis-induced electrolytes changes were statistically highly significant, regarding the postdialytic rise of calcemia ($p < 0.001$) and serum bicarbonate level ($p < 0.001$) (fig. 2). Besides, a falling in the prevalence of postdialytic hypocalcemia and metabolic acidosis appeared (Table I). Instead, magnesemia decreased insignificantly after dialysis (fig. 2).

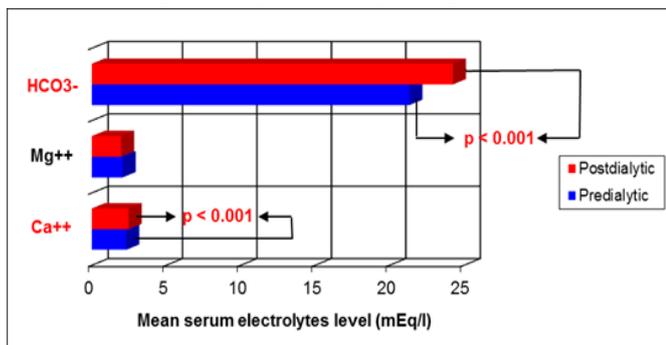


Fig. 2. Electrolyte changes after haemodialysis

Serum calcium

Electrolyte variation type A (unchanged calcemia during dialysis) has been observed in 23 patients (54.8%). The calcemia increase (type B) had a higher frequency rate (16 patients; 38.1%) than the decrease of it (type C), which only has been met in 3 patients (7.1%) ($p = 0.003$). Intradialytic arrhythmias have been especially recorded in type A. Unlike type C, types A and B were associated with the intradialytic occurrence of supraventricular premature beats (sVPB) ($p = 0.015$) (fig. 3).

Serum magnesium

The prevalence of intradialytic ventricular premature beats (VPB) was higher in patients with postdialytic normal serum level of magnesium, compared to those who had hypo- or hypermagnesemia at that time ($p = 0.039$) (fig. 4). Predialytic serum magnesium status remained unchanged after dialysis in 32 patients (76.2%) (electrolyte variation type A). The magnesemia decrease (type C) has been noticed in 7

patients (16.7%), while its increase (type B) only appeared insignificantly in 3 of them (7.1%). In comparison with types B and C, the higher prevalence of intradialytic HRD was associated with type A, without having any statistical significance.

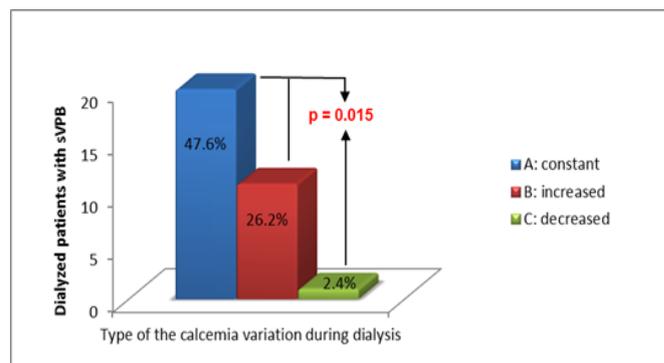


Fig. 3. Intradialytic sVPB in relation to haemodialysis-induced variation of calcemia

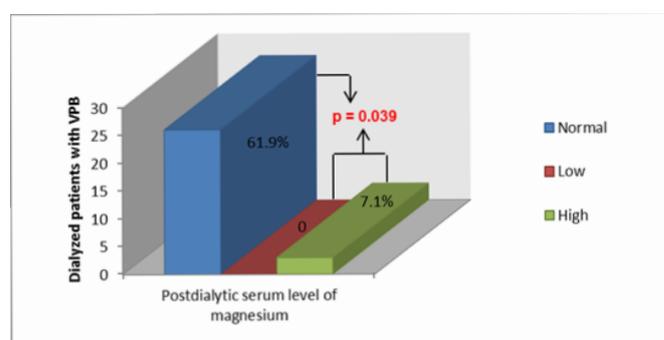


Fig. 4. Intradialytic VPB in relation to postdialytic serum level of magnesium

Serum bicarbonate

Twenty-three patients (54.8%) had the same postdialytic level of serum bicarbonate as before dialysis (electrolyte variation type A). One patient (2.4%) developed postdialytic metabolic acidosis (type C). Unlike this, in the case of other 18 patients (42.8%) haemodialysis improved previous acidosis (type B) ($p < 0.001$). Except atrial fibrillation (AF), the highest prevalence of intradialytic HRD has been observed at types A and B compared to type C, although it was statistically insignificant. AF instead was associated with type C, unlike types A and B ($p = 0.002$) (fig. 5).

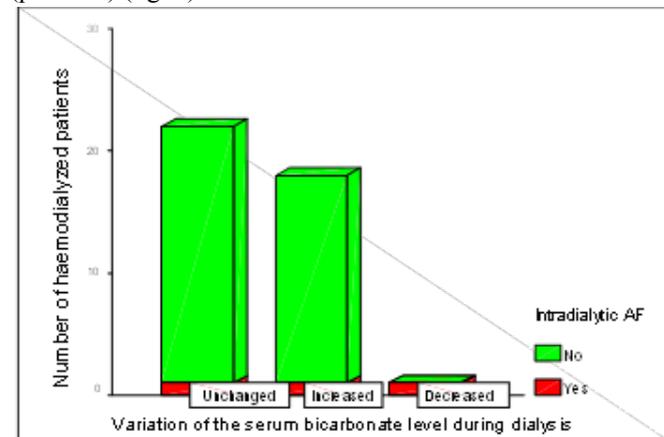


Fig. 5. Intradialytic AF in relation to haemodialysis-induced variation of the serum bicarbonate level

Table 1. Prevalence and type of electrolyte imbalance before and after dialysis

Type of electrolyte imbalance	Predialytic		Postdialytic		Wilcoxon	
	(a)		(b)		(a) vs (b)	
	No. of patients	Percentage	No. of patients	Percentage	z	p
Hypercalcemia	4	9.5%	7	16.7%	-1	0.317
Hypocalcemia	15	35.7%	2	4.8%	-3.606	<0.001
Hypermagnesemia	7	16.7%	3	7.1%	-1.414	0.157
Hypomagnesemia	2	4.8%	2	4.8%	0	1
Metabolic acidosis	32	76.2%	14	33.3%	-3.9	<0.001
Metabolic alkalosis	0	0%	1	2.4%	-1	0.317

Discussions

A number of studies have shown that in dialyzed patients with CRF, HRD occur chiefly during dialysis and in the first hours after its conclusion, including evidences for and against the role of haemodialysis in the genesis of arrhythmias (Erem et al 1997; Switalski et al 2000; Wizemann et al 1985). Our results confirm the high prevalence of intradialytic HRD. Haemodialysis contributes to the correction of certain electrolytes imbalance (acidosis, hypocalcemia, hypermagnesemia) but sometimes, serum ionogram variations can generate HRD (Santoro et al 2006; Yetkin et al 2000).

In literature, it is mentioned the important role of calcium imbalances in the pathogenesis of arrhythmias during dialysis in uremic patients, as it regards both postdialytic hypocalcemia and hypercalcemia (Tong et al 2006; Toussaint et al 2006; Näppi et al 2000). In our study group, a standard dialysis solution, with a mean calcium concentration of 1.5 mmol/l has been used. This dialysis regimen has led to a lower prevalence of postdialytic hypocalcemia. Despite the low proportion of hypo- and hypercalcemia, intradialytic HRD were common. The rise of serum calcium level during dialysis (type B) is consistent with the observations of other authors (Yetkin et al 2000; Tong et al 2006). Instead, in more than half of the patients, hemodialysis has not altered serum calcium level (type A). Statistical analysis showed an association between electrolyte variation types A and B and the high prevalence of intradialytic sVPB. Similarly, some studies have shown that intradialytic HRD occur more frequently if serum calcium level increases during haemodialysis (Tong et al 2006; Nishimura et al 1992). Other studies have demonstrated the opposite, namely the correlation of arrhythmias with intradialytic decrease of calcemia (Toussaint et al 2006; Näppi et al 2000). Some authors have even noted the lack of connection between HRD in the dialyzed and serum calcium level (Erem et al 1997). These conflicting data suggests that the pro-arrhythmogenic effect of the electrolytes imbalance and variation during dialysis may depend on either the magnitude and speed of their production, or some other intradialytic risk factors, or the preexisting cardiac disease (Wizemann et al 1985), or the severity of uremic cardiomyopathy.

In our study, intradialytic VPB occurred mainly in patients with postdialytic normal magnesemia. Comparatively, in literature it is mentioned the involvement of serum magnesium imbalance in the pathogenesis of haemodialysis-related arrhythmias

(Voroneanu et al 2009; Santoro et al 2006). However, some authors showed a cardioprotective role of hypermagnesemia in dialyzed patients with CRF, namely the magnesium contribution to maintaining the electric stability of the myocardium (Yokoyama et al 2005). Taking into account these observations, the normal or low serum magnesium levels could have a high arrhythmogenic risk in this category of patients. The most patients of our roster had unchanged magnesemia during dialysis. Although statistically insignificant, this type of electrolyte variation was associated with the presence of intradialytic HRD. Unlike our results, other studies have shown that haemodialysis decreases serum magnesium level, but they have not been able to determine a correlation between this type of electrolytic change and the risk of ventricular arrhythmias (Yetkin et al 2000). According to data, our results suggest that intradialytic imbalance and variability of magnesemia probably have a lower pro-arrhythmogenic effect, compared to other intradialytic factors, or to the severity of the organic heart disease.

There are evidences that metabolic acidosis but also intradialytic increase of blood pH, are associated with arrhythmias in dialyzed patients (Voroneanu et al 2009; Santoro et al 2006; Yetkin et al 2000). Consistent to the data found in literature (Tong et al 2006; Bruges et al 1994; Fantuzzi et al 1991), in our study haemodialysis improved significantly predialytic acidosis (electrolyte variation type B), while we used a bicarbonate-based dialysate. Even though, more than half of the patients had the same level of serum bicarbonate, before and after dialysis (type A). Types A and B have been associated with the presence of most intradialytic HRD, without having any statistical significance. Instead, AF was associated with the decrease of serum bicarbonate (type C). Compared to our results, other studies have shown an association between HRD and the correction of acidosis during haemodialysis, meaning that predialytic blood pH levels may not have a direct arrhythmogenic effect (Yetkin et al 2000; Rombolà et al 1992). However, the described relationship depends on the speed and consistency of the acidosis correction, as it is known that bicarbonate haemodialysis is less arrhythmogenic than the acetate one (Fantuzzi et al 1991).

Conclusions

1. Intradialytic HRD have a high prevalence.
2. Haemodialysis increases serum calcium level and improves metabolic acidosis.

3. The rise of calcemia, and also the unchanged level of serum calcium during haemodialysis, are associated with a high prevalence of intradialytic sVPB.
4. Intradialytic VPB mainly occur in patients with postdialytic normal level of serum magnesium.
5. The postdialytic worsening of metabolic acidosis is associated with intradialytic AF.

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