

ACID-BASE PROFIL IN CHRONIC DIALYSIS PATIENTS:

Comparative study between hemodialysis with bicarbonate and acetate free biofiltration at 84%.

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INTRODUCTION

► The correction of metabolic acidosis by extrarenal purification techniques (ERT) is a therapeutic goal in the treatment of chronic renal failure (CRF).

► Until this day, hemodialysis bicarbonate (HDB) remains a reference of ERT.

► In this technique, the correction of metabolic acidosis may be associated with a post-dialytic hyperbasemia in connection with presence of levels of bicarbonate between 26 and 32 mmol/L in the dialysate.

AIM OF WORK

Compare the acid-base profile in per and inter-dialysis between the technique of hemodialysis bicarbonate (HDB) and the technique of acetate free biofiltration (BSA) at 84 %.

MATERIALS AND METHODS

► A prospective study in cross-over, over a period of 24 months.

► 30 patients (mean age: 50 years; Sex ratio: 1.14) with end-stage of chronic kidney disease, periodically processed by HDB and stable are included in this work.

► Each patient had 6 successive sessions of HDB and 6 successive sessions of BSA at 84 %. The order of techniques is done by a lot.

► A total of 357 sessions are performed, distributed in 180 sessions of HDB and 177 sessions of BSA at 84 %.

► Blood samples and dialysate made during our study are: A blood gas and serum electrolytes with an alkaline reserves before (H0), middle (H2) and after (H4) the dialysis session.

► The study of per-dialysis acid-base balance is based on the calculation of changes in different blood gas parameters between T0 and T4 boat in sessions of BSA 84 % and HDB.

RESULTS AND DISCUSSION

► The analysis of the results shows a largest risk of metabolic alkalosis in post-dialysis with the HDB (51.8% of sessions) compared to the BSA at 84 % (6.4% of sessions) with a statistically significant difference.

CONCLUSION

BSA at 84 % is a safe technique of renal replacement therapy. It ensures a good correction of metabolic acidosis in intracellular with lower risk of post-dialysis hyperbasemia.

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Table 1: Changes in per-dialysis of blood gas parameters during the two techniques: HDB and BSA at 84 %.

	N	HDB	BSA 84%	Test Student (P)	
PH	T0	165	7,397 ± 0,04	7,369 ± 0,04	0.000 (S)
	T2	54	7,417 ± 0,02	7,377 ± 0,03	0.000 (S)
	T4	164	7,477 ± 0,04	7,414 ± 0,03	0.000 (S)
Δ PH	164	0,082 ± 0,05	0,045 ± 0,04	0.000 (S)	
HCO ₃ ⁻ (mmol/L)	T0	164	21,745 ± 3,57	19,529 ± 2,97	0.000 (S)
	T2	54	23,687 ± 2,19	20,661 ± 2,12	0.000 (S)
	T4	163	27,787 ± 3,61	23,305 ± 2,39	0.000 (S)
Δ HCO ₃	163	6,082 ± 3,04	3,309 ± 3,17	0.000 (S)	
PaCO ₂ (mmHg)	T0	163	34,432 ± 4,40	32,605 ± 4,37	0.000 (S)
	T2	58	35,896 ± 2,83	34,136 ± 3,86	0.004 (S)
	T4	162	36,983 ± 4,34	34,501 ± 4,31	0.000 (S)
Δ PaCO ₂	162	2,566 ± 3,95	1,895 ± 3,94	0,093 (NS)	
PaO ₂ (mmHg)	T0	161	103,541 ± 11,54	104,100 ± 13,66	0.576 (NS)
	T2	61	93,895 ± 16,11	91,886 ± 15,05	0.320 (NS)
	T4	162	96,405 ± 14,27	97,756 ± 14,39	0.244 (NS)
Δ PaO ₂	161	- 6,966 ± 14,72	- 6,312 ± 11,48	0.616 (NS)	

► During the HDB sessions, with a standard bath (31 ± 1meq/l), the rate of post-dialysis HCO₃⁻ can reach 27 to 30 mEq/L. This post dialysis alkalosis may have deleterious consequences: respiratory failure, vascular calcification (2-3).

► The absence of post-dialysis alkalosis, in BSA sessions, is due to the fact that there is a phenomenon of self-restraint to prevent the elevation of plasma bicarbonate: the level of bicarbonate in blood tends to stabilize at an equilibrium value for that the loss through the dialyzer is equal to substitution of bicarbonate.

► However, the post-dialysis hyperbasemia risk is not zero in the BSA. In fact, this complication was observed in 6.4% of sessions.

► The average rate of substitution of molar sodium bicarbonate used for these sessions was 4.86 ± 0.32 ml /kg/h and it is significantly higher than that used in risk-free hyperbasemia sessions, where the average rate is 4 ,27 ± 0.68 ml/kg/h.

► In pre-dialysis, the difference in blood gas parameters for the HDB is explained by hyperchloremia observed with the BSA. Indeed, the rise in the plasma concentration of Cl⁻ causes a greater transfer of bicarbonate in intracellular where the slightest elevation of plasma bicarbonate.

► This transfer of bicarbonate dependent of intracellular chloride is one of the mechanisms of regulation of intracellular pH. Despite the low values of pH and plasma alkaline reserves during the BSA at 84 %, the correction of intracellular acidosis would be better.

► Moreover, the removal of chlorine in excess exchanged with bicarbonate ions in gastrointestinal tract increases the pool of bicarbonate and allows better correction of metabolic acidosis in long-term.

