

## Original article

# Acute effect of citrate bath on postdialysis alkalaemia

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### ABSTRACT

**Introduction:** The correction of metabolic acidosis caused by renal failure is achieved by adding bicarbonate during dialysis. In order to avoid the precipitation of calcium carbonate and magnesium carbonate that takes place in the dialysis fluid (DF) when adding bicarbonate, it is necessary to add an acid, usually acetate, which is not free of side effects. Thus, citrate appears as an advantageous alternative to acetate, despite the fact that its acute effects are not accurately known.

**Objective:** To assess the acute effect of a dialysis fluid containing citrate instead of acetate on acid-base balance and calcium-phosphorus metabolism parameters.

**Material and methods:** A prospective crossover study was conducted with twenty-four patients (15 male subjects and 9 female subjects). All patients underwent dialysis with AK-200-Ultra-S monitor with SoftPac® dialysis fluid, made with 3 mmol/L of acetate and SelectBag Citrate®, with 1 mmol/L of citrate and free of acetate. The following were measured before and after dialysis: venous blood gas monitoring, calcium (Ca), ionic calcium ( $\text{Ca}_i$ ), phosphorus (P) and parathyroid hormone (PTH).

**Results:** Differences ( $p<0.05$ ) were found when using the citrate bath (C) compared to acetate (A) in the postdialysis values of: pH, C: 7.43 (0.04) vs. A: 7.47 (0.05); bicarbonate, C: 24.7 (2.7) vs. A: 27.3 (2.1) mmol/L; base excess (BEecf), C: 0.4 (3.1) vs. A: 3.7 (2.4) mmol/L; corrected calcium ( $\text{Ca}_c$ ), C: 9.8 (0.8) vs. A: 10.1 (0.7) mg/dL; and  $\text{Ca}_i$ , C: 1.16 (0.05) vs. A: 1.27 (0.06) mmol/L. No differences were found in either of the parameters measured before dialysis.

**Conclusion:** Dialysis with citrate provides better control of postdialysis acid-base balance, decreases/avoids postdialysis alkalaemia, and lowers the increase in  $\text{Ca}_c$  and  $\text{Ca}_i$ . This finding

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is of special interest in patients with predisposing factors for arrhythmia and patients with respiratory failure, carbon dioxide retention, calcifications and advanced liver disease.

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## Efecto agudo del baño con citrato sobre la alcalemia postdiálisis

### R E S U M E N

#### Palabras clave:

Hemodiálisis  
Hemodiafiltración  
Bicarbonato  
Acetato  
Citrato  
Acidosis metabólica  
Alcalosis metabólica

**Introducción:** La corrección de la acidosis metabólica provocada por la insuficiencia renal se consigue aportando bicarbonato durante la diálisis. Para evitar la precipitación de carbonato cálcico y magnésico que se produce en el líquido de diálisis (LD) al añadir bicarbonato, es necesario añadir un ácido, habitualmente acetato, que no está exento de efectos secundarios. Así, el citrato se presenta como una alternativa ventajosa al acetato, aunque sus efectos agudos no se conocen con precisión.

**Objetivo:** Evaluar el efecto agudo sobre los parámetros del equilibrio ácido base y del metabolismo calcio-fósforo con la utilización de un líquido de diálisis con citrato en lugar de acetato.

**Material y métodos** Estudio prospectivo y cruzado realizado en veinticuatro pacientes (15 hombres y 9 mujeres). Todos los pacientes se dializaron con monitor AK- 200-Ultra-S con líquido de diálisis SoftPac®, elaborado con 3 mmol/l de acetato y con SelectBag Citrate®, con 1 mmol/l de citrato, libre de acetato. Se extrajeron pre y post-diálisis: gasometría venosa, calcio (Ca), calcio iónico (Cai), fósforo (P) y hormona paratiroides (PTH).

**Resultados** Encontramos diferencias ( $p < 0,05$ ) cuando utilizamos el baño con citrato (C) frente a acetato (A) en los valores postdiálisis de: pH (C: 7,43 (0,04) vs. A: 7,47 (0,05)), bicarbonato (C: 24,7 (2,7) vs. A: 27,3 (2,1) mmol/L), exceso de base (BEecf) (C: 0,4 (3,1) vs A: 3,7 (2,4) mmol/L), calcio corregido (Cac) (C: 9,8 (0,8) vs A: 10,1 (0,7) mg/dl) y Cai (C: 1,16 (0,05) vs A: 1,27 (0,06) mmol/L). No encontramos diferencias en ninguno de los parámetros medidos prediálisis.

**Conclusión** La diálisis con citrato consigue un mejor control de equilibrio ácido base postdiálisis disminuyendo/evitando la alcalemia postdiálisis y un menor aumento de Cac y Cai. Este hallazgo es de especial interés en pacientes con factores predisponentes a arritmias, pacientes con insuficiencia respiratoria, retención de carbónico, calcificaciones y hepatopatía avanzada.

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## Introduction

The correction of metabolic acidosis is one of the treatment goals for chronic kidney disease. To achieve so in patients who are undergoing haemodialysis, bicarbonate is added during the sessions. The optimal bicarbonate concentration these patients should maintain is not accurately known. The KDOQI guidelines recommend maintaining a predialysis bicarbonate of 22 mEq/L in all patients<sup>1</sup>, while the UK Renal Association suggests different targets for patients undergoing peritoneal dialysis (25-29 mmol/L) and haemodialysis (HD) (20-26 mmol/L)<sup>2</sup>.

We could define the optimal bicarbonate concentration of DF as that which prevents interdialysis metabolic acidosis and avoids intradialysis and postdialysis alkalosis. Achieving this goal is not an easy task, as patients who are undergoing haemodialysis have progressive bicarbonate depletion in the interdialysis period and a sudden bicarbonate overload takes place during dialysis.

From a technical point of view, in order to avoid precipitation of calcium carbonate and magnesium carbonate that takes place in the DF when adding bicarbonate, it is necessary to add an acid. Thus, a DF generation system is used with 2 concentrates: one with bicarbonate and the other with acid. Acetic acid is most generally used, at concentrations ranging from 3 to 10 mmol/L. This small amount causes an acetate transfer to the patient during HD, increasing its blood concentration, since the DF has concentrations which are 30 to 40 times greater than the normal blood values (0.1 mmol/L). This exposure to acetate increases in online haemodiafiltration (HDF) techniques<sup>3</sup>, due to the higher amount of infused fluid. Among the side effects described with acetate, hemodynamic instability caused by vasodilation mediated by nitric oxide release<sup>4</sup> and the activation of proinflammatory cytokines by hypoxia<sup>5</sup> are worth mentioning due to their importance during HD. Even compared to a DF with low concentrations of acetate (3 mmol/L), a lower risk of hemodynamic complications has been described when patients undergo dialysis with an acetate-free DF<sup>6</sup>.

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