EFFECT OF BICARBONATE DIALYSIS ON GAS ANALYSIS IN PATIENTS WITH AKI AND ACUTE RESPIRATORY FAILURE

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Introduction and Aims

Acid base abnormalities often emerge in uremic patients undergoing hemodialysis(HD). Several studies have shown that the only way bicarbonate HD compensates uremic acidosis is by eliminating the exceeding CO_2 from dialysate solution through the lungs. CO_2 concentration is higher in dialysate solution than in blood and therefore, high dialysance of CO_2 facilitates its rapid diffusion to the blood compartment. There is no much evidence about the effect of bicarbonate hemodialysis on gas exchange in subjects with acute kidney injury (AKI) requiring renal replacement therapy and associating acute or chronic respiratory failure. The aim of this study was to determine if bicarbonate HD influences blood gases, pH and exacerbates respiratory acidosis in AKI patients with acute versus chronic respiratory failure.

Material and method

The study included 28 AKI individuals requiring HD associating respiratory failure without the need of mechanical ventilation, who were admitted in our Department between December 2012 and November 2013 -17 subjects with AKI and acute respiratory failure (A1) and 11 with AKI and chronic respiratory failure (A2). Acid-base balance, blood gases (arterial blood), were initially analyzed at the beginning of dialysis, during HD, at 1hr (m1), 2 hrs (m2) and 4 hrs (m3). All groups were homogeneous for age, time on dialysis (4hrs), and male/female ratio.

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Results

At the beginning of dialysis, acid-base balance and blood gasses were comparable between patients of both groups. There were no statistically significant differences between the 2 groups. At m1 a statistically significant difference was observed for PaO_2 (A1: 73.7 3.40, A2: 79.29 3.91; p=0.001). At m2 PaO_2 decreased in 23,52% subjects in A1 group. There were no important differences of PaO_2 in A2 group between m1 and m2. At the end of HD (m3), 2 patients from A1 were intubated. PaO_2 was lower in A1 (71.3 2.39) versus A2 (78,3 3.01; p=0.001). In both groups, $PaCO_2$ increased from the beginning of HD to m1 (at M1 $PaCO_2$ was higher in group A1 versus A2; p=0.003). At m2, $PaCO_2$ was higher in A1 compared to A2 ($p<0,0001$) and at M3, no differences were noted.Over the 4 hr treatment, HCO_3^- and ph increased in both groups but ph was lower in A1 than in A2 (p=0.001). No significant difference was observed between A1 and A2 for HCO_3^-.

Conclusions

This study shows that association between AKI requiring bicarbonate HD and acute respiratory failure may aggravate respiratory failure by increasing the $PaCO_2$ and decreasing PaO_2 compared to people with chronic respiratory failure and AKI. 2 patients from A1 group required mechanical ventilation after HD, reinforcing the ideea that bicarbonate HD on AKI patients with acute respiratory failure may worsen the respiratory failure. Therefore, it is recommended a special consideration of dialyzed AKI patients with acute respiratory failure versus chronic respiratory failure, because HD per se can aggravate respiratory failure and the need of mechanical ventilation and assisted advanced life support.

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