Dear Sir,

Hyperkalemia is a frequent encountered problem in patients with chronic renal failure (CRF). The principal cause of hyperkalemia is reduced renal potassium excretion, but the extrarenal potassium homeostasis may also be impaired [1]. Besides dialysis, it can be treated effectively with intravenous administration of insulin and glucose. Beta-adrenergic agonists that facilitate intracellular potassium uptake may be a cause of hypokalemia [2]. Several studies have reported a decrease in plasma potassium levels in uremic patients treated with salbutamol, a β2-adrenergic agonist [3-5]. In this study, we examined effectiveness of aminophylline, a methylxanthine derivative, in the treatment of hyperkalemia in patients with CRF.

Forty-five patients with CRF whose plasma potassium level was higher than 6 mEq/l were included in the study. None of these patients was taking digitalis, β-blockers, insulin or β-adrenergic agonist, and was known to have asthma, diabetes mellitus or heart disease. Twenty-five patients, 15 male and 10 female, were treated with 480 mg aminophylline in 100 ml of 0.9% NaCl solution administered intravenously over a period of 30 min (group A). Twenty patients, 12 male and 8 female, were treated with 10 units of regular insulin in 100 ml of 30% dextrose solution infused over a period of 30 min (group B). Immediately before, and 60, 180 and 360 min after the infusions, blood pressure, heart rate, plasma potassium, sodium, glucose and creatinine levels were measured. Results are expressed as mean ± SD.

### Table 1. Mean plasma potassium levels before and after aminophylline (group A) or insulin (group B) infusions in hyperkalemic patients

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean Plasma Potassium Level (mEq/l)</th>
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<tbody>
<tr>
<td>A</td>
<td><strong>p &lt; 0.001</strong> vs. before treatment; <strong>p &lt; 0.0001</strong> vs. before treatment.</td>
</tr>
<tr>
<td>B</td>
<td></td>
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Statistical analyses employed paired or unpaired Student’s t test for means, and Fisher exact test for ratios.

The changes in plasma potassium levels observed after both treatments are shown in table 1. A significant decrease in the mean plasma potassium level was observed after both treatments, and maximal decrements were achieved at 180 min in both groups. There was no significant difference at any time between groups A and B. The maximal decrease in plasma potassium levels after aminophylline infusion was 0.92 ± 0.54 mEq/l, and it was 0.98 ± 0.43 mEq/l after insulin infusion (p > 0.05). Five patients in group A (20.0%) and 3 patients in group B (15.0%) did not respond to the therapy (p > 0.05) (maximal decrease of plasma potassium < 0.5 mEq/l). Both treatments were well tolerated and no serious side effects were observed. There were no significant changes in plasma creatinine, glucose, sodium and blood pressure in any group. In the patients receiving aminophylline, heart rate was moderately increased (p > 0.05).

Our results suggest that aminophylline is at least as effective as insulin in the treatment of hyperkalemia in patients with CRF. Aminophylline acts by either inhibiting phosphodiesterase enzyme or blocking aden-osoine receptors, resulting in an activation of the intracellular cAMP which in turn affects the Na⁺-K⁺ pump and stimulated intracellular potassium uptake [6].

References

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