DOSE-COMPARISON STUDY OF PREDNISONE FOR RENAL WATER AND SODIUM EXCRETION IN DECOMPENSATED HEART FAILURE

Poster Contributions
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Background: Recent evidence reveals glucocorticoids potentiate renal responsiveness to natriuretic peptides (NPs) by increasing the density of natriuretic peptide receptor A in the kidney, leading to a dramatic enhancement in diuresis and natriuresis in heart failure. The purpose of the study was to determine the effect of different doses of prednisone on renal water and sodium excretion in decompensated heart failure.

Methods: Thirty-eight patients with symptomatic heart failure were randomized to receive standard heart failure care (n=10), or low-dose of prednisone (15mg/day n=8), or medium-dose of prednisone (30mg/day n=10) or high-dose of prednisone (60mg/day, n=10) for 10 days. We recorded 24-hour urinary output for 10 days, and 24-hour urinary sodium at baseline, day 5 and day 10. We also monitored the change of concentrations of serum creatinine, angiotensin II, aldosterone, high-sensitive C-reactive protein (hs-CRP), tumor necrosis factor (TNF)-α, interleukin-1β and interleukin-6.

Results: Prednisone induced a slow but potent diuresis and natriuresis during the study period, leading to a dramatic weight body reduction (1.5±1.1 kg in standare care group, 3.0±1.8 kg in low-dose prednisone group, 3.9±3.2 in medium-dose of prednisone group, and 4.1±2.8 kg in high-dose of prednisone group). High-dose of prednisone induced a more potent natriuresis than those treated with medium- or low-dose prednisone. However, there was no difference in diuresis between low-dose, medium-dose and high-dose of prednisone treated groups. Despite of potent diuresis and natriuresis induced by prednisone, serum creatinine, angiotensin II and aldosterone levels were not elevated. Prednisone therapy also was not associated with suppression of pro-inflammatory cytokines, such as hs-CRP, TNF-α, interleukin-1β and interleukin-6.

Conclusion: Either low-dose or high-dose of prednisone can induce a potent diuresis and natriuresis. The diuretic effect induced by prednisone is not associated with renal function impairment, renin-angiotensin-aldosterone system activation and inflammation suppression.