



5as. JORNADAS DE INICIAÇÃO À INVESTIGAÇÃO CLÍNICA

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Poster 5. MINERALOCORTICOID RECEPTOR ANTAGONISM IN ACUTELY DECOMPENSATED CHRONIC HEART FAILURE

Autores: *João Pedro Ferreira, MD¹, Mário Santos, MD¹, Sofia Almeida, PhD², Irene Marques, MD¹, Paulo Bettencourt, MD, PhD³, Henrique Carvalho, MD, PhD¹*

Afilições: ¹Centro Hospitalar do Porto (CHP), Porto, Portugal; ²Climate Change Impacts, Adaptation and Mitigation Research Group (CC-IAM), Faculdade de Ciências, Universidade de Lisboa (FCUL), Lisboa, Portugal; ³Centro Hospitalar de São João (CHSJ), Porto, Portugal.

Contatos: *João Pedro Ferreira*, médico interno de Medicina Interna do CHP, aluno de doutoramento do ICBAS/UP e do CHP: jp7ferreira@hotmail.com

INTRODUCTION: Aldosterone levels are elevated in patients with acutely decompensated chronic heart failure (ADCHF) despite the use of angiotensin-converting enzyme inhibitors (ACEi), angiotensin receptor blockers (ARB), and beta-blockers (BB), and may contribute to cardiorenal dysfunction, increasing the risk of death and ventricular arrhythmias. Therefore, mineralocorticoid receptor antagonists (MRAs) use in ADCHF treatment has two major putative advantages: improve congestion and hypervolemia through its diuretic effect and prevent the neurohormonal activation that characterizes ADCHF, and that is enhanced by loop diuretics. The impact of MRAs in ADCHF patients has not been studied.

OBJECTIVES: We aimed to evaluate the short-term clinical effect and safety of the MRA antagonist spironolactone in worsening chronic HF patients.

MATERIAL AND METHODS: Prospective, experimental, single-center, and single-blinded trial. Patients were eligible for enrollment if they presented with decompensation of chronic HF with symptoms leading to hospitalization. Patients were treated by the assistant physician with: standard AHF therapy or oral spironolactone 50 - 100 mg/d plus standard ADCHF therapy. An assessment of biomarkers and short-term effects on clinical status and symptoms was performed.

RESULTS: A total of 100 patients were enrolled. Fifty patients were included in the treatment group. Mean (SD) spironolactone dose at day 1 was $94,5 \pm 23,3$ mg and at day 3 was $62,7 \pm 24,3$ mg. Patients in the control group were significantly older (mean (SD), $78,8 \pm 9,3$ vs. $73,2 \pm 11,7$ years; $p = 0,01$). The study groups were well balanced. Greater proportion of patients in the treatment group were free of congestion at day 3: no edema (32% vs. 66%; $p = 0,001$), no rales (24% vs. 66%; $p < 0,001$), jugular venous pressure (JVP) ≤ 8 cm (90% vs. 100%; $p = 0,02$) and no orthopnea (76% vs. 96%; $p = 0,004$). In addition, a significantly higher proportion of patients were on oral furosemide at day 3 (44% vs. 82%; $p < 0,001$). Worsening renal function (increase in pCr $\geq 0,3$ mg/dL from day 1 to day 3) was more likely to occur in control group (20% vs. 4%; $p = 0,038$). Serum potassium (K⁺) levels did not differ significantly between groups. Plasma N-terminal pro brain natriuretic peptide (proBNP) had a significant decrease in spironolactone group at day 3 (median [IQR], 2488 [4579] vs. 1555 [1832]; $p = 0,05$).

CONCLUSIONS: High dose spironolactone in ADCHF is safe, provides faster resolution of congestive signs, and reduction of respiratory effort, allowing an earlier switch to oral furosemide. These findings were translated into a more pronounced natriuretic peptide reduction and lower levels of indirect end-organ damage markers. Our results are a first step towards the potential use of this therapy in patients with worsening chronic HF.