

# Clinical relevance of isolated serum creatinine increase in hospitalizations for acute decompensated heart failure.

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## INTRODUCTION & AIMS:

Patients (P) hospitalized for acute decompensated heart failure (ADHF) may develop impaired renal function during hospitalization. This entity is called cardiorenal syndrome (CRS) and its development is related to a worse prognosis. However, it is not clear whether outcome is affected in all P with CRS alike, and better prognosis in follow-up could be

Identified in any subgroup. **We aimed to assess impact on prognosis of P who increase their serum creatinine (sCr)  $\geq$  0.3 mg/dl during admissions for ADHF.**

Since adverse prognosis has been reported in P who develop resistance to diuretics (RDIUR), these were excluded from this analysis.

## METHODS:

We compared outcome of P hospitalized for ADHF without RDIUR, dividing them into 2 groups: a) P who showed no elevation in sCr; b) those who increased sCr  $\geq$  0.3 mg/dl after admission. RDIUR was defined as the inability to achieve a diuresis  $\geq$  1.5 ml/kg/hour based on a pre-established protocol: initial bolus of 40 mg intravenous (IV) furosemide (FURO) followed by a 5 mg/h for 2 hours IV continuous infusion. In case of not achieving that target FURO dose was doubled for 2 more hours.

Lack to response to this strategy was called "diuretic resistance". We analyzed demographic, biochemical and clinical variables, as well as treatment at admission and outcome during hospitalization, at 6 months and 12 months.

P patients who received heart transplantation or those who were on chronic dialysis were excluded from this analysis.

## RESULTS:

A total of 949 P were admitted consecutively between July 2011 and December 2016. While 461 P (48.6%) did not develop renal dysfunction during hospitalization, 244 (25.7%) P increased their sCr without RDIUR. At admission 73% arterial hypertension, 28% diabetes and 36% had atrial fibrillation. Thirty-one percent were octogenarians. Thirty-one percent had previous chronic kidney disease, and was more frequent in P who increased their sCr during hospitalization (14.8 vs 37.6%;  $p < 0.001$ ; OR 3.5; CI95% 2.4-5). P who increased their sCr were older ( $74 \pm 14$  vs  $70 \pm 16$  years;  $p < 0.001$ ), were admitted with higher blood pressure values (143/81 vs 133/78 mmHg,  $p < 0.01$ ) and had higher Urea and sCr (58/1.3 vs 48/1.05 mg/dl respectively,  $p < 0.001$ ), regardless of gender ( $p = NS$ ).

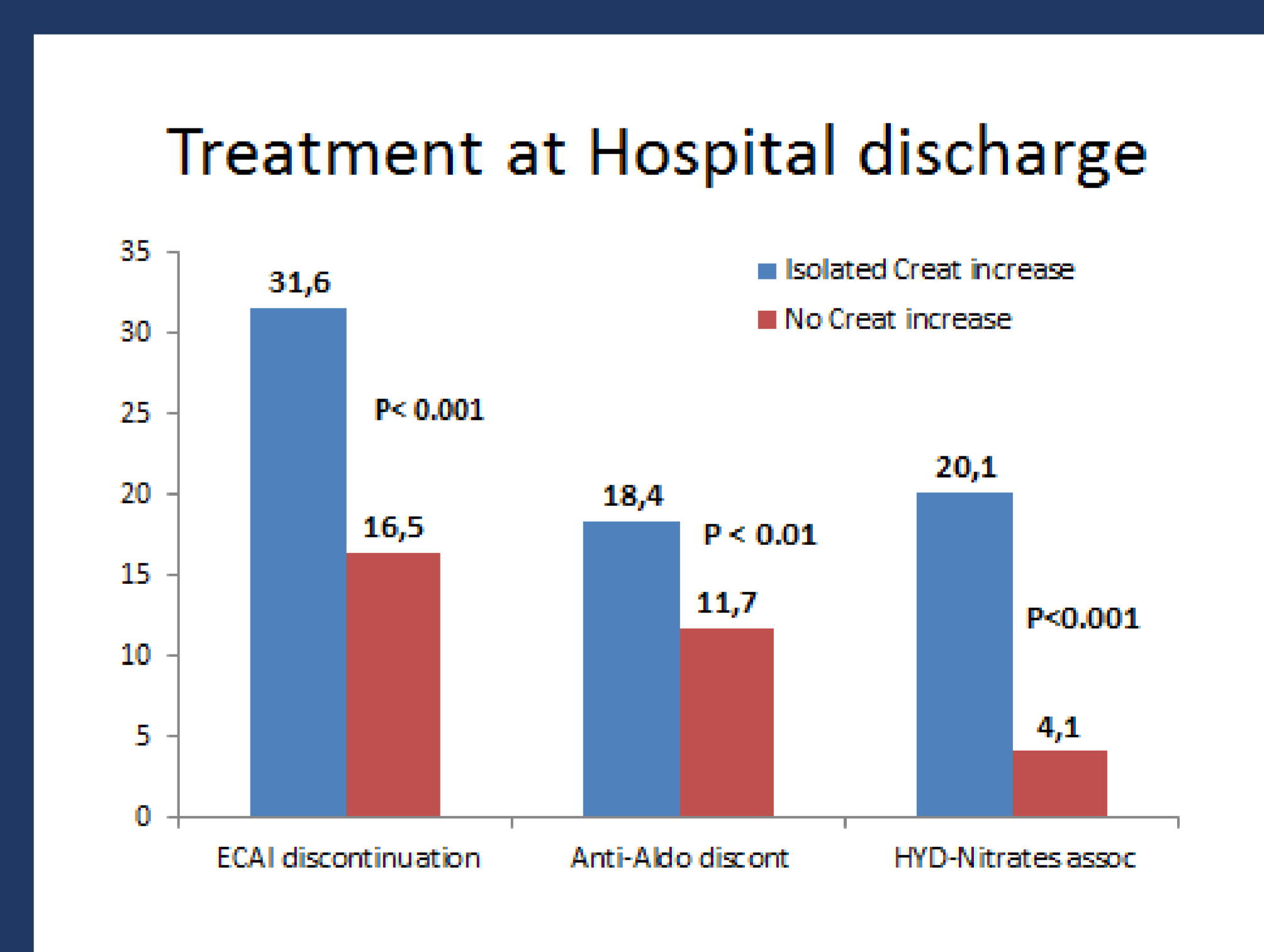
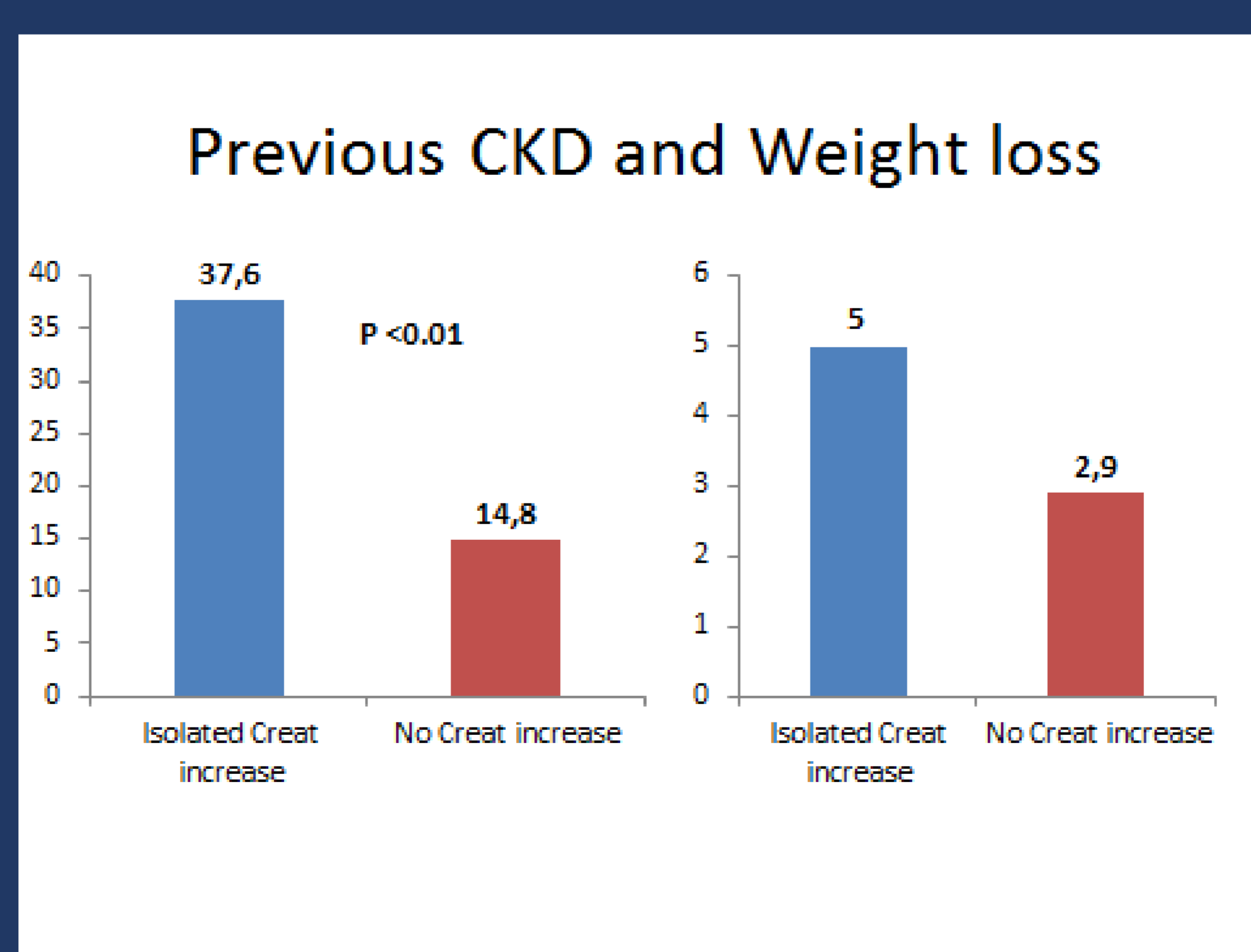
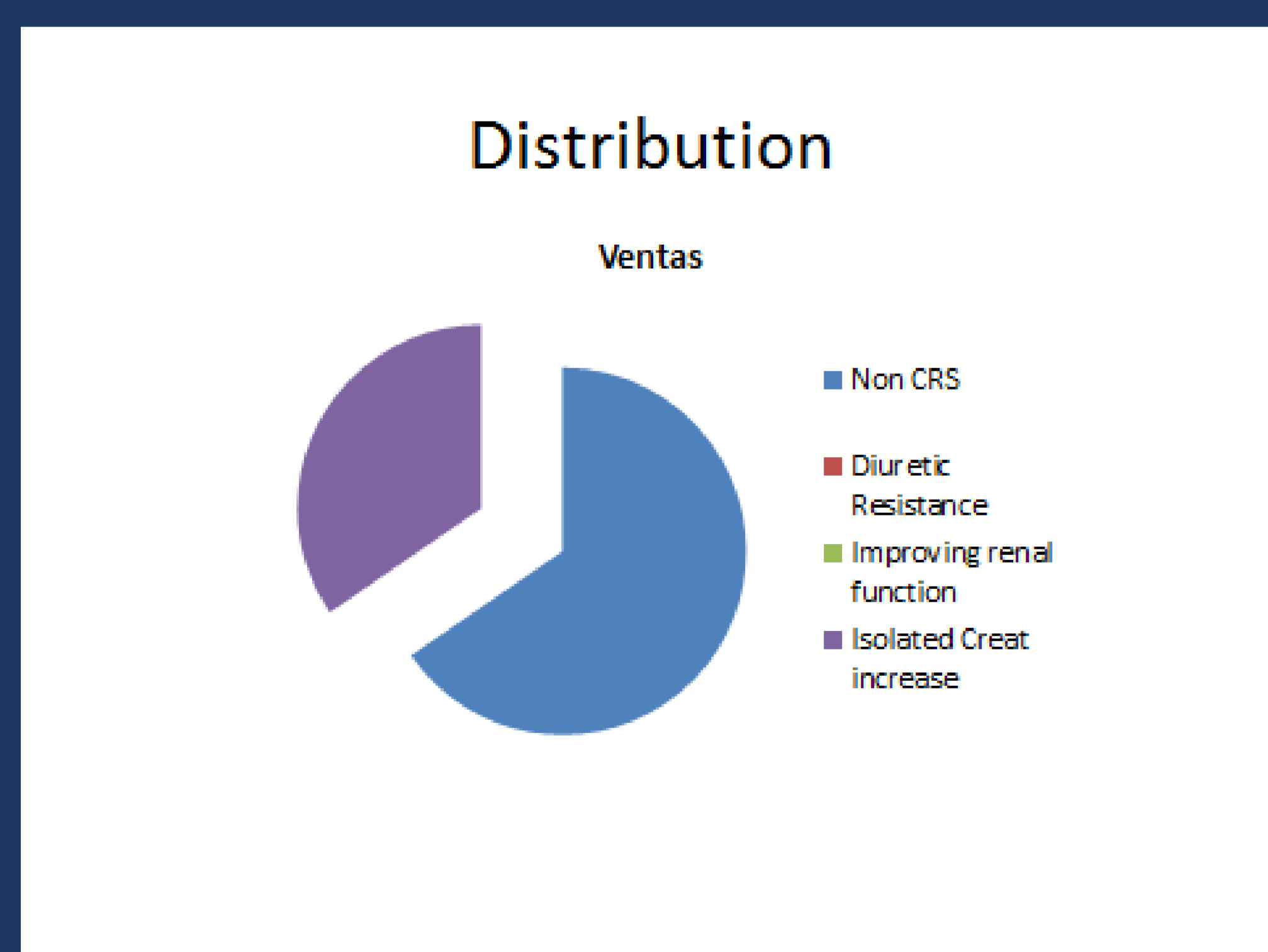
There were no differences neither considering ventricular function (left ventricular ejection fraction 42 vs 41%;  $p = NS$ ) nor considering structural heart disease.

Clinical phenotype did not differ between groups ( $p = NS$  after

comparing hypoperfusion vs congestion), as incidence of worsening heart failure (8.2 vs 7.4;  $p = NS$ ) or length of hospital stay (5 vs 7 days,  $p = NS$ ) did not differ between groups.

While in-hospital mortality was 11%, readmission rate during one year follow-up reached to 35.4%. Anyway, death or readmission did not differ between groups at 1; 6 or 12 months ( $p = NS$ ).

FURO doses were not different at admission and discharge ( $p = 0.06$  in both cases), or during the first 5 days: 376 vs 424 mg;  $p = 0.20$ ). Weight loss was higher in P who increased sCr during admission: 5 vs 2.9 kg ( $p < 0.01$ ). P who increased their sCr required discontinuation of renin-angiotensin system inhibitors more frequently (31.6 vs 15%,  $p < 0.001$ ; OR 2.6; CI95% 1.8-3.8) and anti-aldosterone agents (18.4 vs 11.7%,  $p < 0.01$ ; OR 1.7; CI95% 1.1-2.6), while use of alternative vasodilator regime with hydralazine/nitrate association raised in this group (20.1 vs 4.1%,  $p < 0.001$ ; OR 5.8; CI95% 3.4-10).



## CONCLUSIONS:

Isolated sCr increase was prevalent in our P. It was associated with lower renal reserve and a more aggressive fluid balance. Its development had no impact on short and mid-term prognosis, but led to changes in discharge pharmacological therapy, with a lower prescription rate of drugs effective for survival improvement in heart failure patients.

Authors declare that they have no conflict of interest regarding the material discussed in the present poster