

NEW HORIZONS IN CARDIORENAL SYNDROME: A COMPLIMENTARY CLASSIFICATION FOR CRS type 1. THE SEARCH FOR A LINK AMONG CLINICAL, PATHOPHYSIOLOGIC EVENTS AND THERAPEUTICS.



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Introduction and Aims

Admissions for acute decompensated heart failure (ADHF) adversely affect patient (P) prognosis. In this population, renal dysfunction (RD) is an already known predictor of poor outcome.

According to our daily practice, we propose a complimentary classification for cardiorenal syndrome (CRS) type 1, based on clinical, pathophysiological mechanisms of renal injury and heart failure phenotype and proposed therapy.

Methods

CRS was defined as an increase or decrease of serum creatinine ≥ 0.3 mg/dl from baseline, or diuretic resistance. CRS was classified in 3 groups due to:

- A) Renal ischaemia, caused by hypoperfusion;
 - B) secondary to right-sided heart failure and an increased intra-abdominal pressure (IAP);
 - C) renal damage secondary to aggressive diuresis or modification of intra-renal hemodynamics (early initiation of renin-angiotensin-aldosterone inhibitors).
- Those P with length of stay (LOS) below 2 days, who received a Heart transplant and those under chronic Dialysis were excluded from this analysis.

Results

A total of 804 consecutive P were admitted between July 2011 and December 2015. RD was detected in 30.3% p at admission, while 70% were hypertensive; 27% diabetics and 35% had atrial fibrillation. A total of 30% of P were octogenarians. According to our CRS definition, 45% developed CRS: 12% corresponded to type A; 35.7% to type B and 52.2% to type C. Hospital mortality was 10.3%.

Group A had lower left ventricular ejection fraction (30 vs 40%; $p < 0.001$), corresponded to idiopathic cardiomyopathy (25.6 vs 8.6%; $p = 0.001$) and had signs of hypoperfusion at admission (55 vs 11%; $p < 0.001$). Diuretic resistance (49 vs 6.7%; $p < 0.001$) and worsening heart failure (58 vs 16.6%; $p < 0.001$) were more frequent in this group. Accordingly, these P often received inotropic drugs (84 vs 20%; $p < 0.001$) and rescue ultrafiltration (20 vs 2.5%; $p < 0.001$). Additionally, type A tended to require chronic dialysis during follow up ($p = NS$). These P had the highest length of stay (LOS) (13 vs 7 days; $p = 0.01$), in-hospital (42 vs 8.4%; OR 7.9; CI95% 4.2–15; $p < 0.001$) and 6-m mortality rates (49 vs 18.5%; OR 4.2; CI95% 2.3-8; $p < 0.001$). No differences were noted in this group concerning with 1 or 6-m readmission rates ($p = NS$).

Group B had prior RD (60 vs 24%; $p < 0.001$), right-sided heart failure (36 vs 23%; $p = 0.004$), low T3 at admission (30 vs 14%; $p < 0.001$) and resistant hyponatremia during hospitalization (24 vs 15%; $p = 0.01$). These P had higher mid-term mortality (29 vs 18%; OR 1.8; CI95% 1.16-2.7; $p = 0.009$) and readmission rates (34 vs 20%; OR 2; CI95% 1.4-3; $p = 0.001$), with differences at comparing in-hospital mortality and 30-d readmission rates ($p = NS$).

Finally, group C identified older (74 vs 68 years old; $p < 0.001$), hypertensive P (systolic arterial pressure at admission 144 vs 120 mmHg; $p < 0.001$) and with pulmonary congestion signs (72 vs 57%; $p < 0.001$). This group had the lowest in-hospital (5.6 vs 11.8%; OR 0.44; 95%CI 0.23-0.85; $p = 0.01$) and 6-m mortality rates (14.4 vs 22%; OR 0.59; CI95% 0.38-0.92; $p = 0.01$).

804 consecutive pts

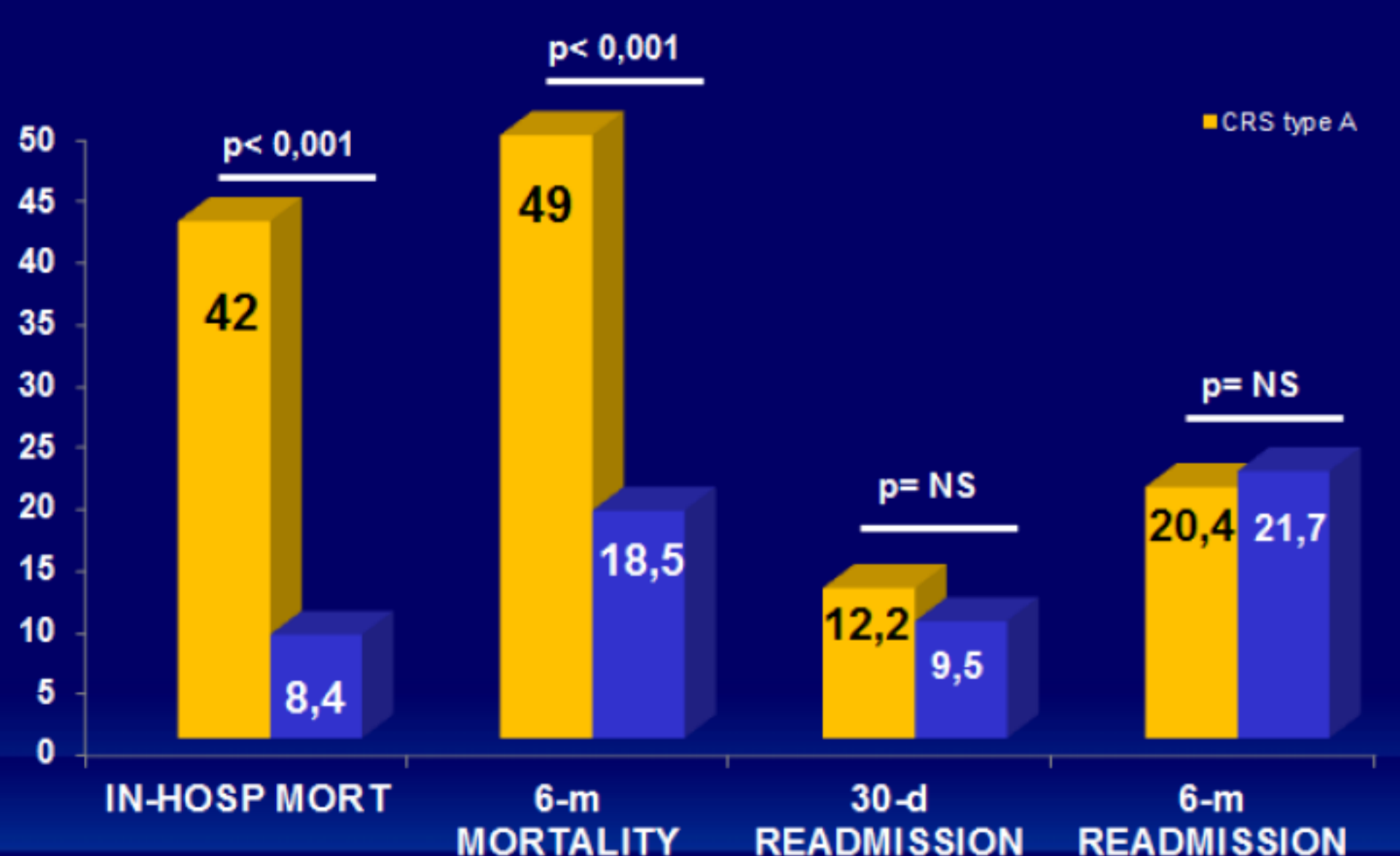
Total CRS



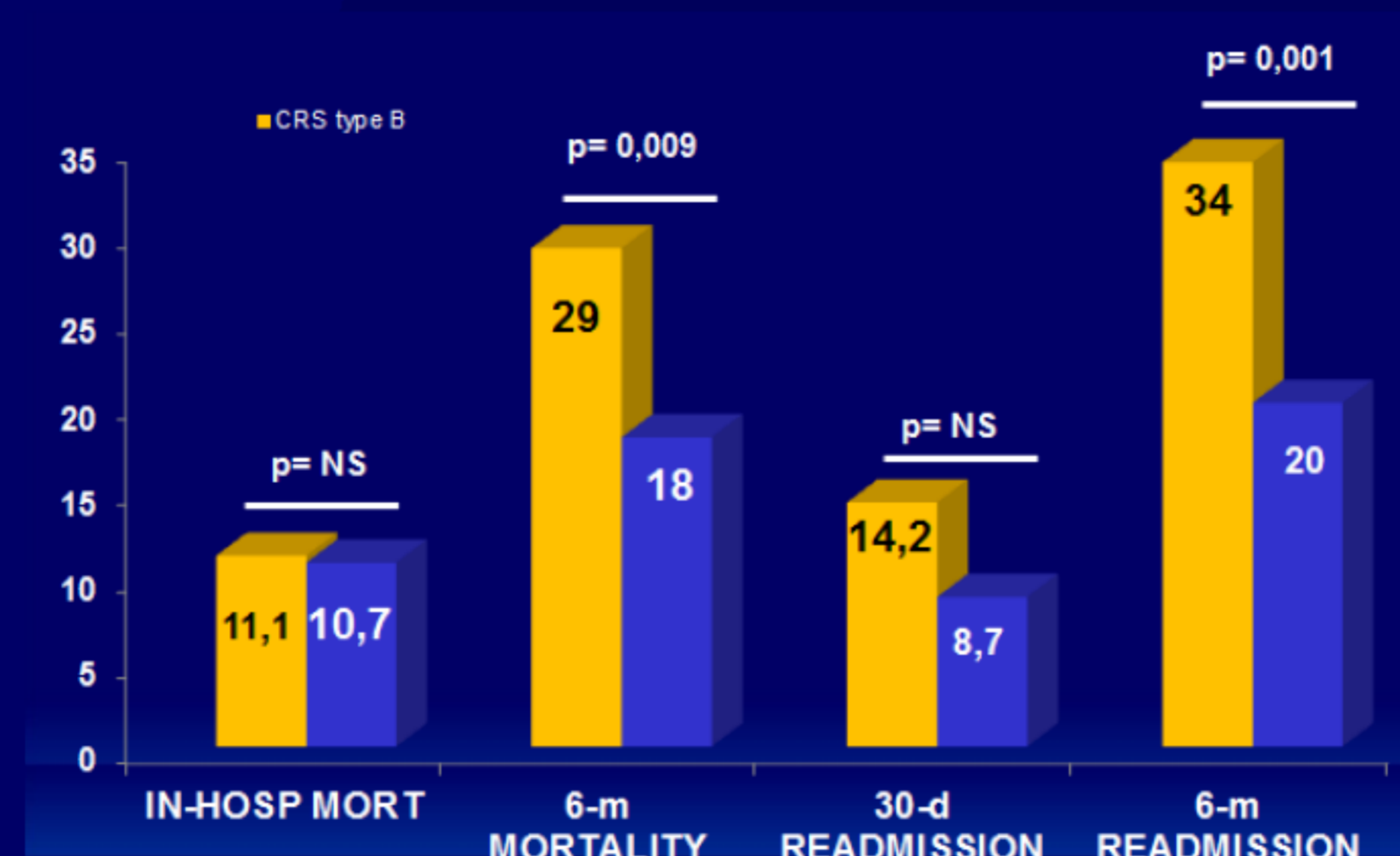
Sub-type A

Sub-type C

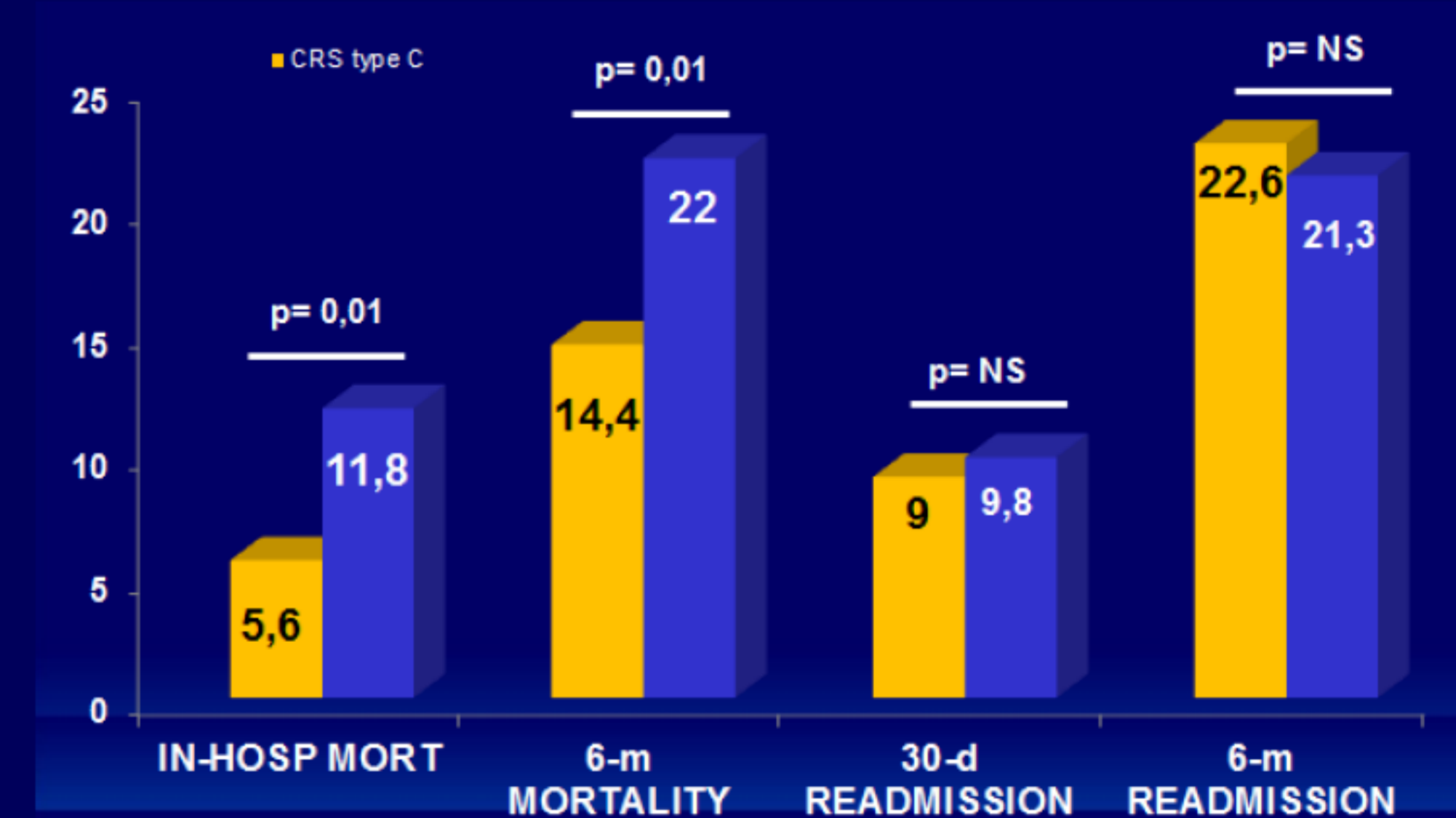
Sub-type B



Type A



Type B



Type C

Conclusions and perspectives

The classification identified 3 different groups with distinctive clinical features and prognosis. Identifying a link between renal and cardiovascular dysfunctions might aid in decision making. Considering CRS as a retrospective diagnosis, recognition of different mechanisms that lead to renal dysfunction in ADHF would allow early implementation of personalized therapies to improve adverse prognosis.

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

