HIGH INCIDENCE AND NEGATIVE PROGNOSIS OF CARDIORENOHEPATIC SYNDROME IN DECOMPENSATED HEART FAILURE

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Background and Objective

Similar pathophysiologic factors such as venous congestion and hypoperfusion are thought to underlie both heart failure (HF) - induced renal and liver dysfunction[1]. The aim of this study was to assess the prevalence of cardiohepatic syndrome (CHS) and cardiorenal syndrome (CRS) and predictors of simultaneous CHS and CRS in decompensated HF (DHF).

Methods

- Liver function tests (LFT) (alanine transaminase (ALT), aspartate transaminase (AST), direct and total bilirubin (DB and TB), alkaline phosphatase (AP), gamma-glutamyl transpeptidase (GGT)) were measured in 322 patients with DHF on admission (Tab.1).
- CHS was considered when at least one LFT level exceeded upper normal limit.
- Early CRS was diagnosed if serum creatinine decreased ≥26.5 μmol/l in first 48 hours of hospitalization. Simultaneous CHS and CRS were considered as cardiorenohepatic syndrome (CRHS). Mann-Whitney test and multivariate logistic regression analysis were performed. P<0.05 was considered statistically significant.

Table 1. Baseline characteristics

Men, n (%)	190 (59)
Age, years (M±SD)	69.5±10.6
Arterial hypertension, n (%)	280 (87)
Anamnesis of myocardial infarction, n (%)	182 (56.5)
Atrial fibrillation, n (%)	210 (65.2)
Diabetes mellitus, n (%)	134 (41.6)
Chronic kidney disease, n (%)	126 (39.1)
Chronic anaemia, n (%)	94 (29.2)
Chronic obstructive lung disease, n (%)	104 (32.3)
Ejection fraction (EF), % (M±SD)	38±13
EF <35%, n (%)	126 (39.1)
Functional class (NYHA), n (%)	
II	8 (2.5)
III	134 (41.6)
IV	180 (55.9)
Liver cirrhosis, n (%)	32 (9.9)

Results

- CHS occurred in 274 (85.1%) patients (Fig. 1). CRS was diagnosed in 60 (18,6%) patients (Fig. 2). Isolated CHS, isolated CRS and CRHS occurred in 78.4, 1.5 and 20.1% patients with hepatic or kidney injury (Fig. 3).
- Patients with versus without CRHS had lower systolic blood pressure (SBP), EF, pulse BP, higher LV mass index, LV end diastolic diameter, higher incidence of severe mitral regurgitation, signs of congestion – jugular venous distension, hepatomegaly, echo-hydropericardium, vasopressor therapy (Tab. 2).
- The independent predictors of CRHS were baseline GFR<45 ml/min/1.73 m^2, SBP<110 mmHg on admission, vasopressor therapy, echo-hydropericardium and EF<35% (Tab. 3).
- Patient with vs without CHRS had worse prognosis: longer length of stay (15.7±6.5 vs 13.5±4.8 days, p<0.05) and trend of increased all-cause death rate in 6 months (31.8 vs 25%, p=0.07).
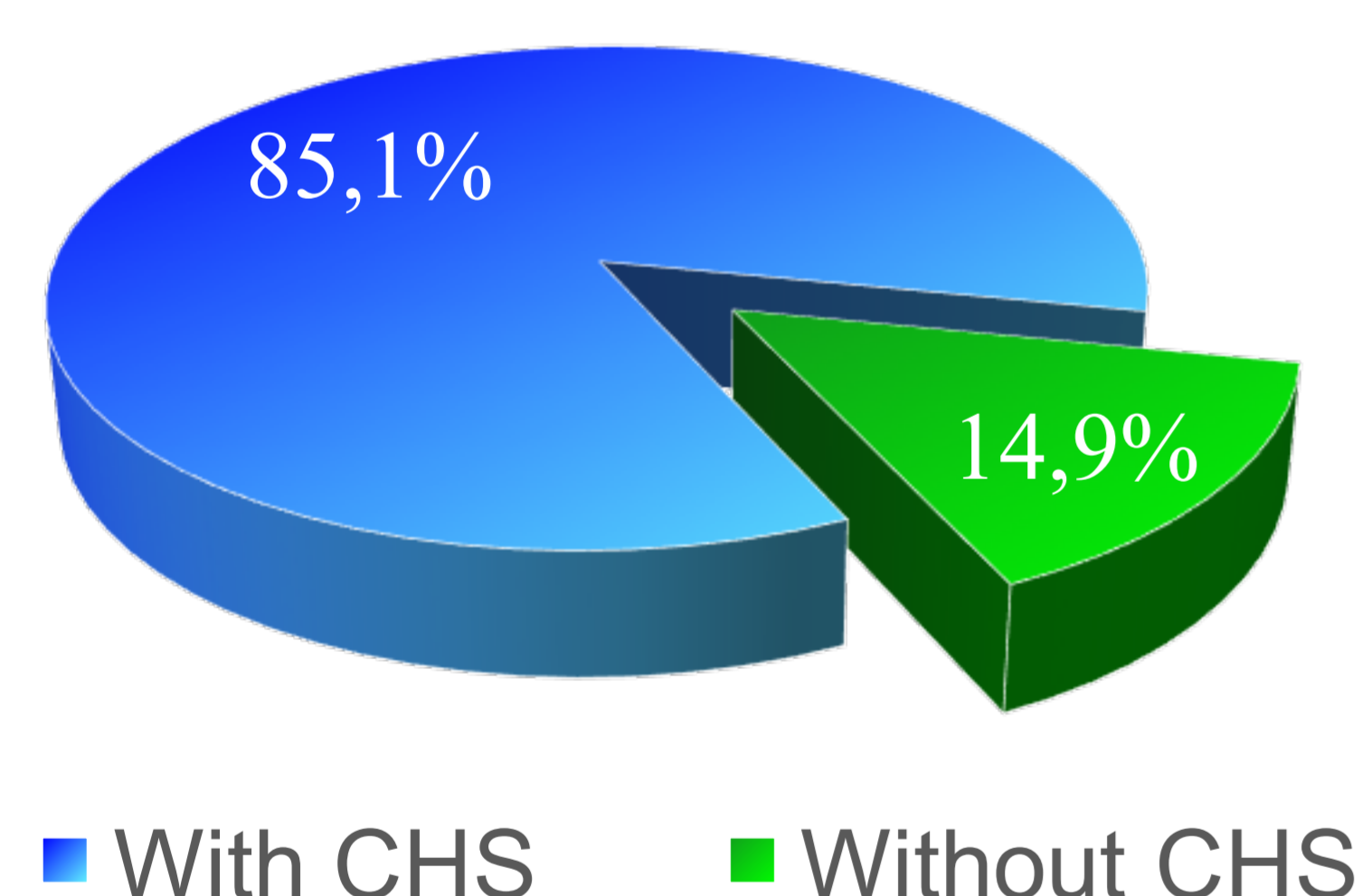
Figure 1. Incidence of CHS

85,1% With CHS, 14,9% Without CHS
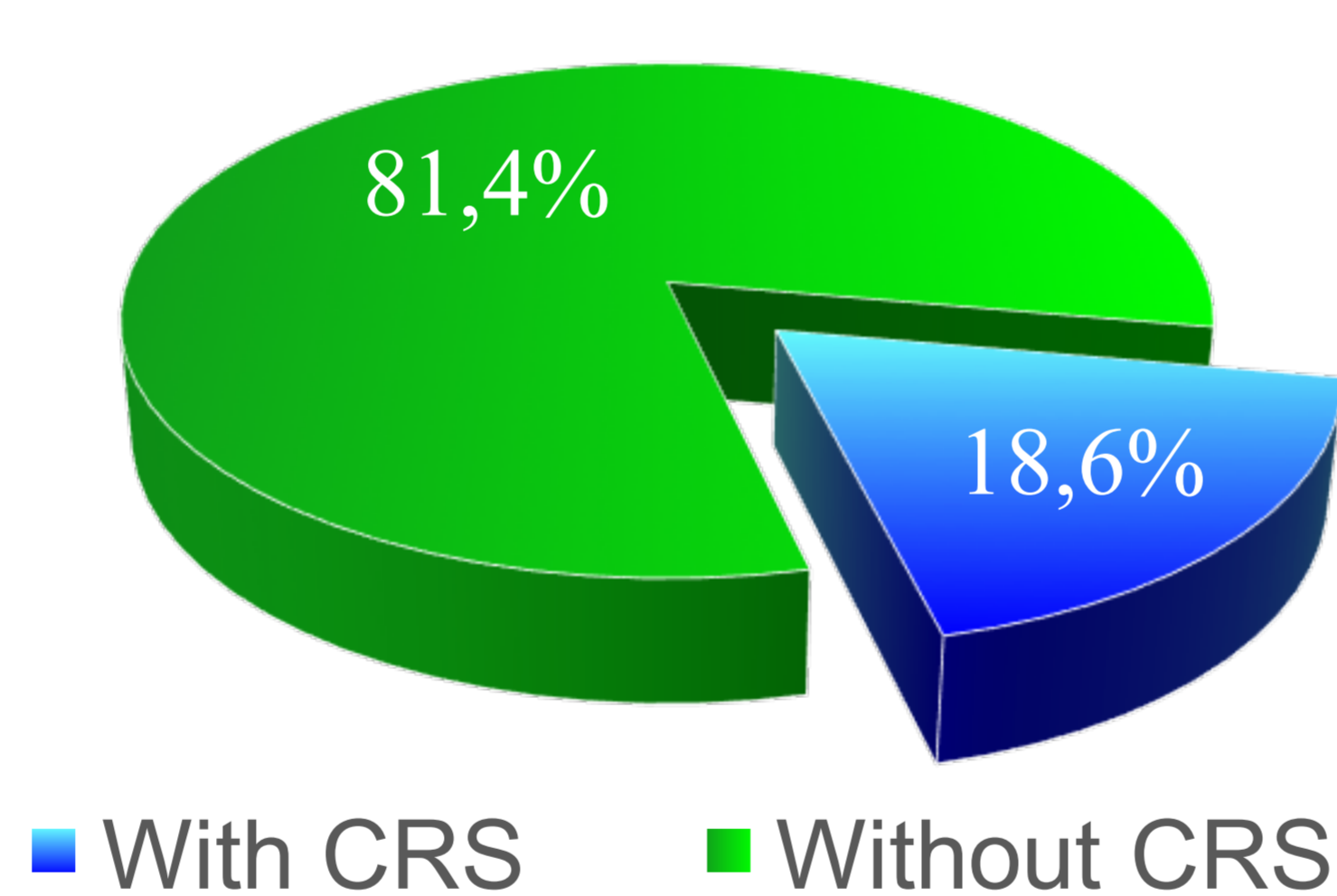
Figure 2. Incidence of CRS

81,4% Without CRS, 18,6% With CRS

Figure 3. Incidence of CRHS

78,4% Isolated CHS, 1,5% Isolated CRS, 20,1% CRHS
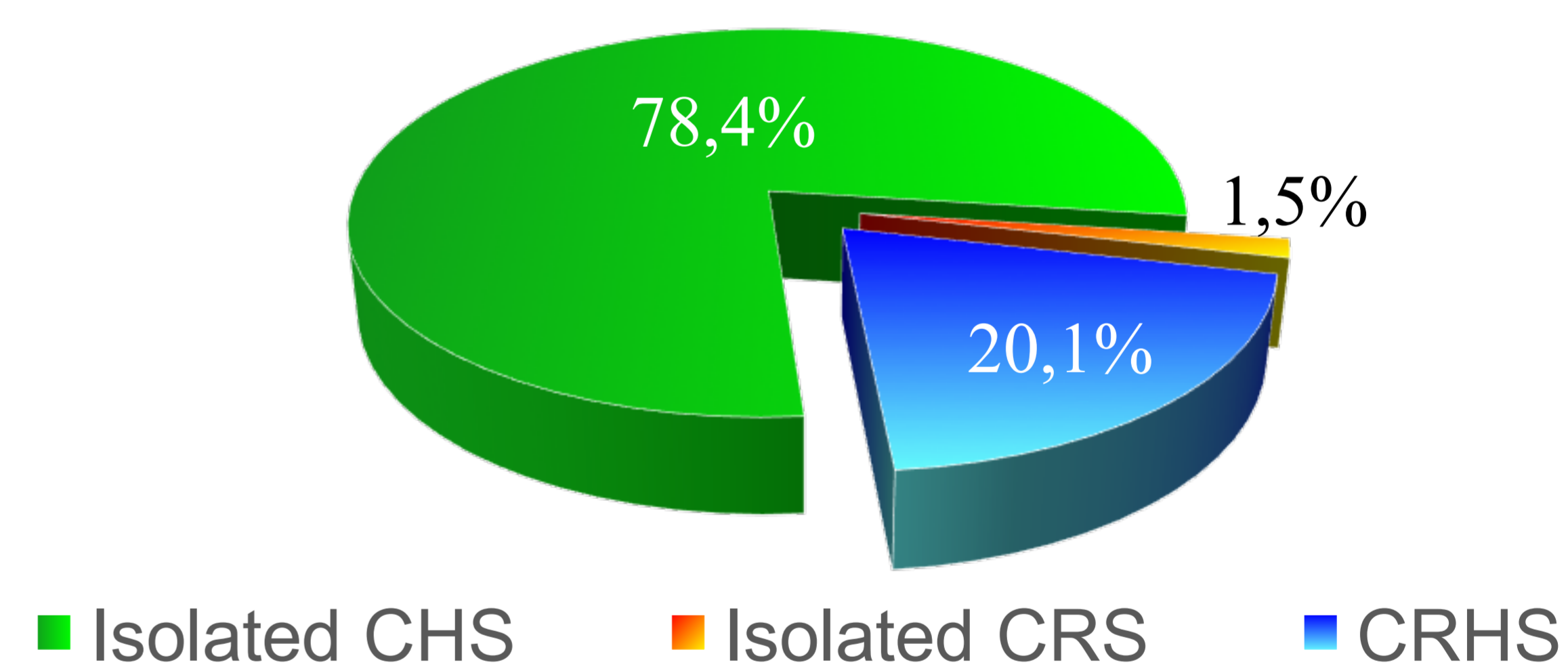
Table 2. Differences of groups depending on presence of CHRS

Parameter	Without CRHS	With CRHS
SBP, mm Hg (M±SD)	138±19	130±18**
EF, % (M±SD)	38±13	32±10**
Pulse pressure, mm Hg (M±SD)	56±15	49±16**
LV mass index, g/m² (M±SD)	178±52	200±50**
LV end diastolic diameter, mm (M±SD)	56±9	62±6***
Severe mitral regurgitation, %	39.6	64.3***
Jugular venous distension, %	39.6	57.1*
Hepatomegaly, %	70.3	85.7*
Echo-hydropericardium, %	22.5	46.4***
Vasopressor therapy, %	6.3	17.9**

*p<0.05, **p<0.01, ***p<0.001

Table 3. Predictors of CRHS

Parameter	OR	CI
GFR <45 ml/min/1.73 m²	3.95	2.15-7.21**
SBP <110 mm Hg on admission	3.51	1.55-7.94*
Vasopressor therapy	3.23	1.35-7.73*
Echo-hydropericardium	2.98	1.62-5.50**
EF <35%	2.96	1.61-5.44*

OR – odds ratio, CI – confidential interval
*p<0.05, **p<0.01

[1] Brisco M. et al. Journal of Cardiac Failure 2013;19(11):739-745.

Conclusions

Isolated CHS, isolated CRS and CRHS occurred in 78.4, 1.5 and 20.1% patients with DHF and hepatic or kidney injury. The independent predictors of CRHS were baseline GFR<45 ml/min/1.73 m^2, SBP<110 mm Hg on admission, echo-hydropericardium and EF<35%. CRHS is associated with longer length of stay and trend of increased all-cause death rate in 6 months.

Declaration of interest: nothing to declare