


Factors associated with cardiorenal syndrome in patients with decompensated heart failure

Fatores associados à síndrome cardiorenal em pacientes com insuficiência cardíaca descompensada

Factores asociados al síndrome cardiorenal en pacientes con insuficiencia cardíaca descompensada

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Abstract

Objective: to identify cardiorenal syndrome (CRS) prevalence in patients with decompensated chronic heart failure (HF) and its association with sociodemographic and clinical data, admission findings, mortality and length of hospital stay.

Methods: a cross-sectional study with a quantitative approach. The sample consisted of 379 medical records of adult patients with a medical diagnosis of decompensated chronic HF admitted to a public hospital in the state of São Paulo, throughout 2015. Data collection occurred in 2016. Kidney failure was considered in patients with a previous diagnosis of chronic kidney disease (CKD) by glomerular filtration rate (GFR) <89 mL/min/1.73 m². Tests with a p value less than or equal to 0.05 were statistically significant.

Results: CRS prevalence was 54.1%, with 24.8% being type 1 and 29.3% being type 2. The main factors associated with CRS were: higher mean age; women; HF of ischemic etiology; lower ejection fraction; people with diabetes mellitus; coronary artery disease; artificial cardiac stimulator use; hypothyroidism and Chagas disease; hemodynamic profile of HF decompensation in types C and L. Also noteworthy are inappetence, drowsiness, rales on respiratory auscultation, alteration in tissue perfusion, decreased urine output, with increased serum levels of potassium, urea and creatinine in the initial clinical assessment. Patients with kidney failure had higher mortality, with no significant difference in length of hospital stay.

Conclusion: There was a high prevalence of CRS in patients with decompensated chronic HF, associated with higher mortality and several clinical indicators.

Resumo

Objetivo: Identificar a prevalência da síndrome cardiorenal (SCR) em pacientes com insuficiência cardíaca (IC) crônica descompensada e sua associação com os dados sociodemográficos, clínicos, achados admissionais, mortalidade e tempo de hospitalização.

Métodos: Estudo transversal, com abordagem quantitativa. A amostra foi constituída por 379 prontuários de pacientes adultos com o diagnóstico médico de IC crônica descompensada, admitidos em hospital público no estado de São Paulo, ao longo de 2015. A coleta de dados ocorreu em 2016. A disfunção renal foi considerada em pacientes com diagnóstico prévio de doença renal crônica (DRC) pela taxa de filtração glomerular (TFG) < 89 mL/min/1.73 m². Testes com valor de p menor ou igual a 0,05 foram estatisticamente significativos.

Resultados: A prevalência da SCR foi de 54,1%, sendo 24,8% do tipo 1 e 29,3% do tipo 2. Os principais fatores associados à SCR foram: maior média de idade, mulheres, IC de etiologia isquêmica, menor fração de ejeção, portadores de diabetes mellitus, doença arterial coronariana, uso de estimuladores cardíacos artificiais, hipotireoidismo e doença de Chagas, bem como o perfil hemodinâmico de descompensação da IC

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nos tipos C e L. Destacam-se, ainda, inapetência, sonolência, estertores na ausculta respiratória, alteração na perfusão tissular, redução do débito urinário, com aumento dos níveis séricos de potássio, ureia e creatinina na avaliação clínica inicial. Os pacientes com disfunção renal apresentaram maior mortalidade, sem diferença significativa quanto ao tempo de hospitalização.

Conclusão: Houve alta prevalência da SCR em pacientes com IC crônica descompensada, associada à maior mortalidade e diversos indicadores clínicos.

Resumen

Objetivo: Identificar la prevalencia del síndrome cardiorenal (SCR) en pacientes con insuficiencia cardíaca (IC) crónica descompensada y su relación con los datos sociodemográficos, clínicos y descubiertos en la admisión, la mortalidad y el tiempo de hospitalización.

Métodos: Estudio transversal, con enfoque cuantitativo. La muestra estuvo compuesta por 379 historias clínicas de pacientes adultos con diagnóstico médico de IC crónica descompensada, ingresados en hospital público en el estado de São Paulo, durante 2015. La recolección de datos se realizó en 2016. La disfunción renal fue considerada en pacientes con diagnóstico previo de enfermedad renal crónica (ERC) por el índice de filtración glomerular (IFG) $< 89 \text{ mL/min/1.73 m}^2$. Pruebas con un valor de p menor o igual a 0,05 fueron estadísticamente significativos.

Resultados: La prevalencia del SCR fue del 54,1 %, del cual el 24,8 % fue de tipo 1 y el 29,3 % de tipo 2. Los principales factores asociados al SCR fueron: mayor promedio de edad, mujeres, IC de etiología isquémica, menor fracción de eyección, portadores de diabetes mellitus, enfermedad arterial coronaria, uso de estimuladores cardíacos artificiales, hipotiroidismo y enfermedad de Chagas, así como también el perfil hemodinámico de descompensación de la IC en el tipo C y L. Además, se destacan la inapetencia, somnolencia, estertores en la auscultación pulmonar, alteración en la perfusión tisular, reducción del flujo urinario, con aumento del nivel en sangre de potasio, urea y creatinina en la evaluación clínica inicial. Los pacientes con disfunción renal presentaron mayor mortalidad, sin diferencia significativa con relación al tiempo de hospitalización.

Conclusión: Se observó una alta prevalencia del SCR en pacientes con IC crónica descompensada, relacionada con una mayor mortalidad y diversos indicadores clínicos.

Introduction

Heart failure (HF) is a complex and progressive clinical syndrome, reaching epidemic levels of incidence. It is considered one of the priorities among chronic diseases and one of the greatest public health problems, which needs attention from health sectors worldwide.^(1,2)

HF is considered to cause an important physical limitation, in addition to serious psychoemotional and psychosocial consequences. HF has repercussions in reducing the quality of life of individuals with the disease as well as implications for early retirements, high government costs, in addition to major social, economic and human impacts.^(3,4)

Acute kidney injury (AKI) is a clinical condition present in 30–40% of individuals with HF, especially in those with more severe clinical manifestations or related to the coexistence of other chronic comorbidities; and, when present, they are associated with a worse prognosis, and their patients have higher mortality rates, rehospitalizations and costs.⁽⁵⁻⁷⁾

According to the Kidney Disease Improving Global Outcomes (KDIGO) guidelines,⁽⁸⁾ AKI is defined by an increase in serum creatinine $\geq 0.3 \text{ mg/dL}$ within 48 hours; increase in serum creatinine ≥ 1.5 times the known baseline value or that is presumed to have occurred within the last seven days; urine output of $<0.5 \text{ mL/kg/h}$ for six hours.

Chronic kidney disease (CKD) is defined as an abnormality of kidney structure or function, present for more than three months, with health implications.⁽⁹⁾ It is classified as stage 1, when glomerular filtration rate (GFR) is greater than $90 \text{ mL/min/1.73 m}^2$; in stage 2, with GFR of 60 to $89 \text{ mL/min/1.73 m}^2$; in stage 3a, with GFR 45 at $59 \text{ mL/min/1.73 m}^2$; in stage 3b, with GFR of 30 to $44 \text{ mL/min/1.73 m}^2$; in stage 4, with GFR from 15 to 29; and in stage 5, with GFR less than $15 \text{ mL/min/1.73 m}^2$.⁽⁹⁾

The neurohumoral and inflammatory activation present in HF contributes to the progressive loss of renal function, i.e., renal hypoperfusion due to low cardiac output or hypotension is the main factor for the development of kidney failure in patients with chronic HF. Decreased cardiac output, along with blood flow redirection, mainly to the brain and heart, worsens renal perfusion, triggering a series of changes that favor the development of systemic and intrarenal compensatory responses in order to retain fluids and restore the cardiac output. However, with the progression of heart failure, these responses become deleterious, since they will no longer be sufficient to restore cardiac output, thus triggering a progressive worsening of renal function.^(6,7)

When this phenomenon occurs in the population of patients with HF, it is called cardiorenal syndrome (CRS), as there is a strong pathophysiological interaction between the heart and the kidney.^(10,11) CRS can be classified into

five types: type 1. Acute cardiorenal syndrome; type 2. Chronic cardiorenal syndrome; type 3. Acute renocardiac syndrome; type 4. Chronic renocardiac syndrome; type 5. Secondary cardiorenal syndrome.^(10,11)

Studies⁽¹²⁻¹⁴⁾ showed that GFR <60 mL/min/1.73 and elevated serum creatinine levels at admission and during hospitalization can be predictors of longer hospital stays, re-hospitalizations and mortality. Therefore, serum creatinine, urea and estimated GFR are markers of renal function, with strong and independent prognostic values.

Considering the severity of prognosis of patients with HF who progress to CRS, it is essential that nurses understand this phenomenon and know how to recognize clinical manifestations and predisposing factors in this group of patients, in order to propose specific, assertive and qualified nursing interventions,⁽¹⁵⁾ in order to prevent and monitor this occurrence in clinical practice.

Given these assumptions, this study aimed to identify the prevalence of CRS in patients with decompensated chronic HF and its association with sociodemographic and clinical data, admission findings, mortality and length of hospital stay.

Methods

This is a cross-sectional study, with documentary analysis and based on the STROBE guidelines.⁽¹⁶⁻¹⁷⁾ This study was submitted and approved, under Opinion number 1681988 by the Research Ethics Committees (REC) of the institutions involved.

Medical records of adult patients with a medical diagnosis of decompensated chronic HF, admitted to the emergency unit of a large public hospital in the city of São Paulo, from January to December 2015, were included. We did not include medical records of patients with decompensated chronic HF who had previously been on renal replacement therapy and medical records that did not include all variables relevant to the research.

Data were collected from August to December 2016 through document analysis by the main researcher, using an instrument developed by the authors containing all relevant variables to this study.

Kidney failure at hospital admission was considered as a dependent variable. The criterion considered for screening the presence of kidney failure was a previous diagnosis of CKD and GFR <89 mL/min/1.73m². For patients who had no previous kidney failure at admission, the criterion used was an increase in creatinine by 0.3 mg/dl in the last 48 hours or an increase by 1.5 times in the last seven days, according to a guideline for AKI diagnosis.⁽⁸⁾

The sociodemographic variables analyzed were sex, age, and color, in addition to the clinical characteristics related to the etiology of HF and left ventricular ejection fraction (LVEF). The clinical admission factors analyzed were HF's hemodynamic profile (profile A represents patients with adequate perfusion and without congestion; profile B represents adequate perfusion, but congestion; profile C represents inadequate perfusion and congestion; and profile L represents inadequate perfusion and without congestion), complaints from patients in emergency units, data on propaedeutics, vital sign parameters and laboratory profile. All data were recorded in patients' medical records.

The minimum sample size was calculated based on the results of a meta-analysis study.⁽⁵⁾ Assuming that the prevalence of kidney failure in people with HF is around 40%, calculations were estimated by the Z test statistic (normal distribution), for comparison between two proportions, considering a 10% margin of error, 80% power of test and test's significance level of 5% ($p < 0.05$).

Data were presented using descriptive statistics by calculating absolute and relative frequency for qualitative variables, and mean and standard deviation (SD) for quantitative variables. Statistical tests for comparing patients with the presence and absence of kidney failure were performed using the SPSS program, version 22.0. Pearson's chi-square test and Fisher's exact test or likelihood ratio test between qualitative measures were used to analyze the association. Student's t-test was used for independent samples, and Mann-Whitney U test (when the variables did not have a homogeneous distribution) was used for quantitative measures, being considered statistically significant when p value was less than or equal to 0.05.

Results

Three hundred and seventy-nine medical records of patients with decompensated chronic HF were analyzed. One hundred eleven patients (29.3%) had CKD and 268 patients had no kidney failure prior to hospitalization. Of the total number of patients analyzed without CKD, 94 patients (24.08%) presented worsening of renal function, according to the established criteria (increase of 0.3 mg/dl of serum creatinine).

In the sample analysis, 205 patients (54.1%) had CRS; 111 (29.3%) had CRS type 2; 94 (24.80%) had type 1 CRS, making two comparative groups, i.e., 205 patients with kidney failure (AKI and CKD) and 174 patients (45.9%) without kidney failure.

Most patients were men, with a mean age of 65.8 years, white, with HF of ischemic etiology followed by idiopathic and valve etiology, with a mean LVEF of 40%.

There was a predominance of women with higher mean age, HF of ischemic etiology and lower LVEF in the group of patients with kidney failure, after comparing the two groups. Valve etiology was highlighted in patients without kidney failure, as shown in Table 1.

The main comorbidities presented by patients were systemic arterial hypertension (73.0%), atrial fibrillation (42.5%), diabetes mellitus (38.8%), dyslipidemia (38.3%), coronary artery disease (34.3%), and smoking (37.5%) as a life habit. In a comparative analysis between groups, it was observed that patients with kidney failure had a higher prevalence of diabetes mellitus (62.6% versus 37.4%, $p < 0.01$), coronary artery disease (63.1% versus 36.9%, $p = 0.01$), artificial cardiac stimulator use (74.4% versus 25.6%, $p = 0.01$), hypothyroidism (65.9% versus 34.1%, $p = 0.01$) and Chagas disease (73.0% versus 27.0%, $p = 0.01$).

Regarding HF's hemodynamic profile at admission, it was identified that most patients included in the study had decompensation profile B (n 253, 6%), followed by profile C (n 114, 3%) and profile L (n 12.3%). In a comparison between groups, a higher prevalence was identified in the group of

Table 1. Sociodemographic and clinical characterization associated with kidney failure in patients with decompensated HF (n=379)

Variables	Total n(%)	Kidney failure		P value
		Present n(%)	Absent n(%)	
Sex n(%)				
Male	194(51.2)	75(61.3)	119(38.7)	<0.01*
Female	185(48.8)	99(53.5)	86(46.5)	
Mean age (SD)	65.8(14.3)	68.4(12.3)	62.8(15.8)	<0.01†
Skin color				0.40 ^o
Yellow	2(0.5)	2(100)	0(0.0)	
White	236(62.3)	125(53)	111(47)	
Black	64(16.9)	34(53.1)	30(46.9)	
Mixed-race	77(20.3)	44(57.1)	33(42.9)	
HF etiology				<0.01*
Ischemic	115(30.3)	71(61.7)	44(38.3)	
Valvar	96(25.3)	44(45.8)	52(54.2)	
Hypertensive	21(5.5)	3(14.3)	18(85.7)	
Puerperal	3(0.8)	1(33.3)	2(66.7)	
Chagas	39(10.3)	28(71.8)	11(28.2)	
Alcoholic	6(1.6)	5(83.3)	1(16.7)	
Not compressed	1(0.3)	1(100)	0(0.0)	
Idiopathic	97(25.6)	52(53.6)	45(46.4)	
Amyloidosis	1(0.3)	1(100)	0(0.0)	
LVEF (%) (SD)	40.4(16.3)	37.7(15.4)	43.6(16.7)	<0.01†

HF - Heart Failure; LVEF - Left Ventricular Ejection Fraction; SD - Standard Deviation; *Pearson's Chi-square test; V - Likelihood Ratio Test; † - Student's t-test for independent samples

patients with kidney failure, with profile C (71.9% versus 28.1%, $p < 0.01$) and profile L (75% versus 25%, $p < 0.01$), and in patients without kidney failure, profile B (45.1% versus 54.9%, $p < 0.01$).

The most prevalent admission complaints in patients were dyspnea (91.6%), progressive dyspnea (49.9%), orthopnea (49.1%), paroxysmal nocturnal dyspnea (42%), edema (39.3%) and decreased urine output (19.5%). In a comparative analysis, it was identified that patients with kidney failure had a higher prevalence of inappetence (71.1% versus 28.9%, $p = 0.01$) and decreased urine output (74.3% versus 25.7%, $p < 0.01$). Meanwhile, in patients without kidney failure, the most prevalent complaints were progressive dyspnea (47.6% versus 52.4%, $p = 0.01$), paroxysmal nocturnal dyspnea (47.2% versus 52.8%, $p = 0.02$), edema (47% versus 53%, $p = 0.01$), orthopnea (51.1% versus 48.9%, $p = 0.04$) and palpitation (25% versus 75%, $p = 0.04$).

The most prevalent findings in admission tests were: state of normal consciousness, presence of rales on respiratory auscultation, absence of abnormalities on cardiac auscultation, presence of jugular stasis and lower limb edema. Findings with signifi-

cant association in patients with kidney failure were guidance in time and space, drowsiness, rales on respiratory auscultation and changes in tissue perfusion. In an analysis of vital parameters, a lower blood pressure level and lower heart rate are observed in the group of patients with kidney failure, as shown in Table 2.

Table 2. Association between the findings of admission tests and vital parameters with presence of kidney failure (n=379)

Variables	Total n(%)	Kidney failure		P value
		Present n(%)	Absent n(%)	
Orientation in time and space	321(84.7)	166(51.7)	155(48.3)	0.02*
Drowsiness	41(10.8)	30(73.2)	11(26.8)	<0.01*
Confusion	17(4.5)	9(52.9)	8(47.1)	0.92*
Respiratory auscultation with rales	324(85.5)	182(56.2)	142(43.8)	0.04*
Respiratory auscultation with wheezing	36(9.5)	17(47.2)	19(52.8)	0.38*
Respiratory auscultation with rhonchi	17(4.5)	7(41.2)	10(58.8)	0.27*
Normal cardiac auscultation	217(57.3)	113(52.1)	104(47.9)	0.36*
Systolic murmur in mitral focus	156(41.2)	107(42.6)	49(38.3)	0.63 ^F
Diastolic murmur in mitral focus	15(4.0)	11(4.4)	4(3.1)	0.84 ^F
Hepatomegaly	117(30.9)	65(55.6)	52(44.4)	0.70*
Hepatojugular reflux	85(22.4)	47(55.3)	38(44.7)	0.80*
Ascites	75(19.8)	43(57.3)	32(42.7)	0.52*
LL edema	295(77.8)	162(54.9)	133(45.1)	0.54*
Jugular stasis	169(44.6)	97(57.4)	72(42.6)	0.24*
Reduced tissue perfusion	121(31.9)	85(70.2)	36(29.8)	<0.01*
Anasarca	32(8.4)	17(53.1)	15(46.9)	0.90*
Dehydration	5(1.3)	4(80)	1(20)	0.38 ^F
SBP mmHg (Mean (SD))	111(30.4)	103(28.6)	120(29.9)	<0.01 ^U
DBP mmHg (Mean (SD))	69(18.5)	64(16.1)	74(19.7)	<0.01 ^U
HR bpm (Mean (SD))	88(25)	85(22.5)	93(27.0)	<0.01 ^U
SatO2% (Mean (SD))	90(9.9)	91(9.2)	90(10.6)	0.65 ^U
RR ipm (Mean (SD))	21(4.1)	21(4.0)	21(4.0)	0.16 ^U

LL - lower limbs; SBP - systolic blood pressure; DBP - diastolic blood pressure; HR - heart rate; RR - respiratory rate; SatO2 - oxygen saturation; bpm - beats per minute; ipm - incursions per minute; mmHg - millimeters of mercury; * Pearson's Chi-square test; ^F - Fisher's exact test; ^U - Mann-Whitney U test

In an analysis of laboratory tests, it was observed that the group of patients with kidney failure had a lower level of platelets, a higher serum concentration of potassium, urea and creatinine, according to Table 3.

With regard to mortality, 108 patients (28%) died during hospitalization, with a higher prevalence and greater chance of developing this outcome in patients with kidney failure (73.1% versus 26.9%, $p < 0.01$, Odds Ratio of 3.13). Regarding length of hospital stay, there was no statistically significant difference ($p = 0.49$) between patients with kidney failure (mean of 18.2 days; SD of 27.13) and patients without kidney failure (mean 16.3 days; SD of 26.07).

Table 3. Association between laboratory data and presence of kidney failure (n=379)

Variables (Mean/SD)	Total	Kidney failure		P value
		Present	Absent	
Hb g/dL	12.5(2.2)	12.4(2.3)	12.7(2.1)	0.18 ^t
Ht%	38.4(6.8)	38(7.2)	38.9(6.2)	0.19 ^t
Platelets mil/mm ³	207(91.23)	192(88)	226(91)	<0.001 ^U
Na mmol/L	135(5.34)	135(5.0)	135(5.6)	0.75 ^U
K mmol/L	4.54(0.86)	4.66(0.91)	4.39(0.76)	<0.01 ^U
Cr mg/dL	1.88(1.17)	2.55(1.18)	1.04(0.25)	<0.01 ^U
U mg/dL	94.2(55.5)	125(53.8)	55(25.5)	<0.01 ^U
Leukocytes mil/mm ³	8.61(3.60)	8.49(3.6)	8.76(3.5)	0.54 ^U
PCR mg/dl	6.25(9.2)	5.85(8.35)	6.73(10.1)	0.38 ^U

SD - standard deviation; Hb - hemoglobin; Ht - hematocrit; Na - plasma sodium; K - plasma potassium; Cr - serum creatinine; U - urea; CRP - C-reactive protein; U - Mann-Whitney U test; t - Student's t-test

Discussion

As found in our study, we obtained a prevalence of 54.1% of patients with CRS. Of these 54.1%, 29.3% had chronic CRS, and 24.8% had acute CRS. Compared to a meta-analysis study,⁽⁵⁾ it was shown that in the populations of patients with HF, CKD prevalence was around 49%, whereas AKI was observed around 23% to 35% of patients.

In this study, it was identified that the presence of CRS was associated with a higher mean age and in women; these data are consistent with other studies,^(6,18,19) showing that CRS incidence increases with age, justified by a progressive loss of GFR with aging and also associated with a higher prevalence of comorbidities such as HF.⁽²⁰⁾

In two multicenter clinical studies^(14,21) that assessed CRS in patients with HF, males figured prominently in the sample; however, this difference was statistically significant only in the study conducted by Al-Jarallah⁽¹⁴⁾ and collaborators, in which men figured prominently, with prevalence in the group of patients without CRS. In the study conducted by Lala et al.⁽¹³⁾, women figured prominently, mainly in the group of patients with intermediate LVEF HF who presented AKI during hospitalization, focusing on the importance of this clinical characteristic in the female population and which corroborates the findings of this study.

Some clinical characteristics showed an association in patients with CRS, such as HF of ischemic etiology, lower ejection fraction, patients with diabetes mellitus, coronary artery disease, artificial cardiac stimulator use, hypothyroidism and Chagas disease.

Comorbidities, such as CKD, arterial hypertension, diabetes mellitus, coronary artery disease, chronic lung disease, dyslipidemia and vascular diseases are risk factors for patients with HF developing CRS.^(13,14,20-24)

In a French study,⁽²¹⁾ it was found that patients with HF with moderate or severe kidney failure (GFR <60 mL/min/1.73 m²) had more often pacemaker implants and implantable cardioverter defibrillator (ICD) than the group of patients without kidney failure (4.78% vs 0%), supporting the findings of our study.

The main etiology of HF found in the study for the group of patients with CRS was that of ischemic etiology. According to Brazilian and American HF guidelines,^(22,23) ischemic etiology of HF, secondary to coronary artery disease, is one of the main causes of HF with reduced ejection fraction. Also, a progressive increase in coronary atherosclerosis increases myocardial dysfunction and, consequently, CRS.

As for the hemodynamic profile of clinical presentation of patients with HF, a greater association of profiles L and C with CRS was identified. Our findings support other studies, which also demonstrated that patients with CRS had greater severity of clinical symptoms and worse functional class (III and IV) of the *New York Heart Association*.^(13,21)

Signs and symptoms, such as inappetence, drowsiness, decreased urine output and reduced tissue perfusion, which showed a significant association in patients with CRS, are related to a greater severity and mortality of HF decompensation secondary to reduced cardiac output.^(13,21)

The presence of rales on respiratory auscultation is synonymous with congestion. A study⁽²⁵⁾ showed that congestion is the main reason for hospitalization and rehospitalization due to decompensated HF, resulting in higher rates of CRS and death. Another study⁽²¹⁾ observed that increased level of brain natriuretic peptide hormone (BNP), equivalent to a greater congestion condition in patients with HF, was directly related to renal function worsening.

As for clinical parameters, patients with CRS had lower blood pressure and heart rate, and these findings were also found in other studies.^(14,21) The justification for these data is related to a greater severity of

cardiac function, which causes lower blood pressure values and, consequently, greater need for optimization of beta-blocking drugs, in an attempt to postpone the harmful compensatory mechanisms of HF; however, it is possible to identify findings antagonistic to ours, with higher values of systolic pressure.⁽²⁶⁾

Some studies^(13,14,21) showed that serum creatinine and urea values were higher in the groups of patients with HF, associated with kidney failure and anemia. According to Uduman,⁽²⁴⁾ even small increases in creatinine can be associated with worse results, considering a high prevalence of CRS within acute and chronic heart diseases.

With regard to the serum potassium level, higher values were identified in patients with CRS, denoting the importance of a careful assessment in admission of these patients. Hyperkalaemia is a serious clinical condition that can cause severe and fatal cardiac arrhythmias, in addition to being associated with cardiovascular diseases, hospitalization, progression of kidney disease and increased risk of mortality.⁽²⁷⁾ Therefore, these patients must remain under multidisciplinary surveillance until their clinical stabilization.^(2,21-23)

It was also observed that, in the group of patients with kidney failure, patients were more thrombocytopenic. Not many studies have been found comparing platelet values in the groups of patients in question. However, it is understood that thrombocytopenia may be a clinical sign of severity or an unfavorable outcome in patients with CRS.

Regarding length of hospital stay and analysis of mortality, higher mortality was found in patients with kidney failure; however, in relation to the length of hospitalization, there was no difference between groups. This same result is supported by other studies,^(13,14,19,28-30) which also showed a significant increase in the mortality rate when patients had worsening renal function associated with HF (with preserved or decreased LVEF). No difference was found between groups regarding mortality in another study,⁽²¹⁾ however, hospitalization length was longer in patients with kidney failure. The study conducted by McCallum et al.⁽³¹⁾ showed that GFR decline was not significantly associated with death and readmission.

Nurses and multidisciplinary teams, given the recognition of these factors associated with CRS, should implement nursing interventions focused on patients' clinical compensation, mainly related to hydroelectrolytic, acid-base, renal function, hemodialysis therapy and low cardiac output control, through independent or dependent actions by other professionals.⁽¹⁵⁾

In addition to the nursing interventions focused on patients' clinical compensation, it is necessary to intensify educational actions in order to slow CRS progression, improve self-care, increase the level of medication and non-medication adherence to reduce hospitalization rates due to HF decompensation,⁽³²⁾ especially in patients at higher risk of worsening clinical signs and developing CRS.

It is believed that the main limitations of this study are the fact that it was conducted in a single hospital institution through data assessment in medical records and retrospectively, which makes it impossible to guarantee the reliability of some registered data.

Conclusion

There was a high prevalence of CRS in patients with decompensated chronic HF, which reflected in the outcomes of higher mortality. The factors associated with CRS were high mean age, female gender, etiology of ischemic and Chagas HF, lower ejection fraction, comorbidities, of type C and L HF's hemodynamic profiles, inappetence, drowsiness, crackles in respiratory auscultation, reduced tissue perfusion and decreased urine output, with increased serum levels of potassium, urea and creatinine. The results of our study can serve as a basis for constructing clinical assessment care protocols performed by nurses, aiming at prioritizing the initial care of patients in emergency services.

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Collaborations

Zhao LM, Lopes JL, Lopes CT, Santos VB and Barros ALBL declare that they contributed to the study design, data analysis and interpretation, article writing, critical review of the manuscript and approval of the final version to be published.

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