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CLINICAL AND PROGNOSTIC VALUE OF HYPONATREMIA IN PATIENTS WITH CHRONIC HEART FAILURE

Abstract

The objective was to assess the short-term prognostic value of different types of hyponatremia in patients hospitalized due to acute decompensated chronic heart failure. **Material and Methods:** A prospective study included 396 patients hospitalized due to acute decompensated chronic heart failure. Hyponatremia was diagnosed in cases of serum sodium level of less than 135 mmol/l. The pre-hospital hyponatremia was defined as a decreased serum sodium level on admission, whereas the hospital hyponatremia was referred to cases occurred during hospitalization. In patients with pre-hospital hyponatremia the reduction of sodium levels by ≥ 3 mmol/l during hospitalization was defined as a progressive hyponatraemia. The influence of different types of hyponatremia on the hospital prognosis was determined, while the combined primary endpoint was all-cause mortality and/or transfer to the intensive care unit. **Results:** Patients with hyponatremia were older and had more severe clinical signs of chronic heart failure, lower left ventricle ejection fraction and more pronounced diastolic dysfunction than normonatremic patients. After adjustment for age, comorbidity and severity of chronic heart failure, the Cox regression showed that hyponatremia was an independent predictor of all-cause mortality and transfer to the intensive care unit (odds ratio 3.1; $p < 0.05$). Pre-hospital hyponatremia had a higher prognostic value for outcome compared with hospital hyponatremia (odds ratio 3.9 versus 2.9, respectively; $p < 0.05$). Progressive hyponatremia was associated with a marked increase of mortality and transfer to the intensive care unit (odds ratio 6.8; $p < 0.05$). **Conclusion:** Pre-hospital and hospital hyponatremia are independent predictors for short-term outcomes in patients hospitalized due to acute decompensated chronic heart failure. Progression of the pre-hospital hyponatremia is associated with significant increase of all-cause mortality and risk of transfer to the intensive care unit.

Key words: *hyponatremia, chronic heart failure, decompensation, severity of the disease, prognosis, mortality*

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CI — confidence interval, OR — odds ratio, FC — functional class, CHF — chronic heart failure

The attention of various clinical specialists is currently focused on the problem of electrolyte metabolism disorders in hospitalized patients. The most common electrolyte abnormality among patients hospitalized for any reason is hyponatremia [1]. It accompanies the course of a number of diseases and is associated with prolonged hospitalization, high costs of treatment and worsening of prognosis [2-4].

Among patients hospitalized with decompensated chronic heart failure (CHF) hyponatremia on admission to hospital is detected in 5-35% of cases [5-8], while its prevalence depends on the patient population and the timing of determining sodium

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level in the blood. In most studies devoted to this issue, the prevalence and prognostic significance of reduced blood sodium levels recorded on admission to hospital [9-13] were studied. At the same time, the incidence and clinical significance of hyponatremia developing during hospitalization have not been studied. However, the normal sodium level in a single measurement in the first days of hospitalization does not exclude the possibility of its further reduction against the background of the natural course of the disease and active diuretic therapy. In addition, the rate of prehospital hyponatremia progression during hospitalization also remains unexplored.

In this regard, the aim of our study was to assess the clinical and prognostic significance of different types of hyponatremia in patients hospitalized due to decompensation of CHF.

Materials and Methods

The authors performed a prospective study involving 396 patients hospitalized in the V.K. Gusak Institute of Urgent and Reconstructive Surgery due to decompensation of CHF for the period 2013-2016. Inclusion criteria in the study were as follows: age over 18 years, hospitalization due to decompensation of CHF and signed informed consent.

The exclusion criteria were as follows: acute coronary syndrome, valvular and septal heart defects, myocarditis, hypertrophic and dilated cardiomyopathy, other conditions associated with hyponatremia (burn disease; acute gastrointestinal disorders; liver cirrhosis; nephrotic syndrome, glomerular filtration rate of <15 ml/min, adrenal insufficiency, acute hypovolemia), decompensation of concomitant disease, pregnancy, cancer, alcohol and drug abuse, participation in another clinical study during the previous 30 days.

The serum sodium level was determined in all patients on admission to hospital and during the hospital treatment period. The study was performed using ion-selective electrodes on an automatic biochemical analyzer Cobas C 311 (Roche Diagnostics, Germany). The electrolyte concentration was expressed in mmol/L. Hyponatremia was diagnosed with a decrease in serum sodium level of <135 mmol/l. The severity of hyponatremia was assessed in accordance with generally accepted recommendations: with sodium levels of 130-134 mmol/l hyponatremia was regarded as mild, with 125-129 mmol/l as moderate, and with <125 mmol/l as severe [14-16]. Hyponatremia was considered as prehospital when detected on admission, as hospital when it developed during inpatient treatment. In order to assess the period of development of hospital hyponatremia, the examination of electrolyte levels was carried out every 3 days. The change of the sodium level during hospitalization was also analyzed in patients with prehospital hyponatremia, while in the case of reduction of sodium levels by ≥ 3 mmol/l from baseline, progressive hyponatremia was diagnosed.

The effect of different types of hyponatremia on the course and hospital prognosis of CHF was determined, with the primary end point of the study being the combined indicator of death and/or transfer to the intensive care unit.

The clinical status of patients was assessed using the clinical assessment scale of CHF in the modification by V.Yu. Mareyev. To objectify the clinical and functional state of the patient and determine their tolerance to physical activity, a 6-minute walk test was performed.

The patients included in the study received medical therapy in accordance with modern standards of treatment [17-19] and followed a diet with restricted water and salt consumption [20, 21]. During the period of active diuretic therapy patients were recommended moderate restriction of sodium consumption with food (<3 g/day) after compensation of the condition — according to the functional class (FC) of CHF. In case of CHF of I FC, patients were advised not to eat salty food (sodium consumption restriction to 3 g/day), in case of II FC, patients were recommended not to add salt to food (sodium consumption restriction to 1.5–2 g/day), in case of III–IV FC, patients were asked to use products with low salt content and prepare meals without salt (sodium consumption restriction to 1 g/day). During active diuretic treatment, patients were prescribed to limit fluid intake to 1.5 L/day, and less than 2 L/day after complete compensation of CHF.

The results were processed on a personal computer using biomedical data statistical analysis packages MedStat and Microsoft Office Excel 2007.

The χ^2 and Shapiro-Wilk W tests were used to check the distribution for normality. For normal distribution, the data were given as mean ± standard deviation (m ± σ) for continuous variables and as a particle (percentage) for categorical variables. In a distribution other than normal one, the data were given as median and interquartile ranges (Me (Q1; Q3)). To compare two samples of continuous variables, which are subject to the normal distribution rule, we used paired and unpaired Student's t-test, and Wilcoxon test in the case of distribution other than normal distribution rule. To compare more than two samples subject to the normal distribution rule, a one-way analysis of variance was used, and in the presence of a statistically significant difference between the groups, a pairwise comparison using the Scheffe test was used, and the Dunnett's test with the control group. If the distribution rule differed from the normal distribution rule, one-way Kruskal-Wallis analysis of variance by ranks was performed, and a comparison was made using Dunn's test if there was a statistically significant difference between the groups. We used a standard method of analysis of contingency tables using the χ^2 test to study the distribution of discrete features in different groups and to compare relative values. Multivariate logistic regression analysis with odds ratio (OR) and 95% confidence interval (CI) was used to study the relationship between the features. The critical level of significance for statistical hypothesis testing was assumed to be 0.05.

Results

Patients with hyponatremia were significantly ($p < 0.05$) older than those with normal sodium levels, among them patients were more likely to have anemia, concomitant chronic obstructive pulmonary disease, type 2 diabetes and renal dysfunction. There were no significant differences in body mass index, prevalence of concomitant hypertension, atrial fibrillation, and myocardial infarction between the groups (Table 1).

Patients with hyponatremia had more pronounced clinical signs of CHF (Table 2). According to echocardiography, they had a smaller ejection fraction and more pronounced diastolic dysfunction of the left ventricle compared to normonatremic patients.

Analysis of drug therapy (Table 3) showed that patients with hyponatremia were more likely to receive thiazide and thiazide-like diuretics compared to patients with normonatremia. In the group with hospital hyponatremia, in addition, the rate of administration of mineralocorticoid receptor antagonists was higher than in other groups of patients.

A detailed analysis of diuretic therapy during the active phase revealed differences in drug doses: patients with hyponatremia were prescribed higher doses of hydrochlorothiazide, indapamide and spironolactone compared with patients with normal sodium levels. The highest doses of spironolactone were taken by patients with hyponatremia which developed during hospitalization (Table 4).

The rate of development for combined primary end-point of death and/or transfer to the intensive care unit and its individual components was analyzed (Table 5) to assess the impact of different types of hyponatremia on the course and hospital prognosis of the disease. Adverse outcomes more often developed in patients with hyponatremia, and the period of their stay in hospital was longer compared with persons with normal blood sodium level.

The reasons for the transfer to the intensive care unit were analyzed, while the main reason was selected as the analysed one in the presence of several indications for transfer (Table 6). It was found that patients with hyponatremia compared with patients with normal sodium levels during hospitalization more often experienced hypotension or hypoperfusion of organs requiring treatment in the intensive care unit. Among individuals with pre-hospital hyponatremia, the emergence of resistance to diuretics was more often observed.

According to the results of pathoanatomical studies, cardiovascular diseases dominated in the structure of hospital mortality in both groups of patients, and cardiovascular mortality was higher among patients with hyponatremia. Persons with reduced sodium levels died more often than patients with normal electrolyte levels due to progression of heart failure, and statistical significance of differences was achieved due to a subgroup of patients with prehospital progressive hyponatremia. It is noteworthy that mortality against the backdrop of worsening symptoms of CHF decompensation

Table 1. *Initial clinical characteristics of patients*

Parameter	Prehospital hyponatremia (n=64)	Hospital hyponatremia (n=68)	Normo-natremia (n=264)
Age, years, Me (Q1; Q3)	69 (65; 73.5)*	70 (66; 74.5)*	64 (61; 66.5)
Male, number of patients (%)	38 (59.4%)	49 (72.1%)	157 (59.5%)
BMI, kg/m^2, m±σ	31.4±2.9	30.8±3.1	29.6±2.8
SBP, mmHg, m±σ	119.3±3.6*	118.2±2.9*	132.3±3.7
DBP, mmHg, m±σ	74.2±3.1	70.6±1.9*	76.2±3.9
HR at rest, bpm, Me (Q1; Q3)	88 (80; 94)	85 (79; 93)	86 (78; 96)
Hypertension, number of patients (%)	56 (87.5%)	62 (91.2%)	236 (89.4%)
Myocardial infarction, number of patients (%)	48 (75.0%)	49 (72.1%)	164 (62.1%)
Atrial fibrillation, number of patients (%)	19 (29.7%)	21 (30.9%)	49 (18.6%)
Stroke, number of patients (%)	8 (12.5%)	9 (13.2%)	31 (11.7%)
Chronic obstructive pulmonary disease, number of patients (%)	21 (32.8%)*	28 (41.2%)*	41 (15.5%)
Diabetes mellitus, number of patients (%)	24 (37.5%)*	24 (35.3%)*	49 (18.6%)
Anemia, number of patients (%)	17 (26.6%)*	19 (27.9%)*	40 (15.2%)
Glomerular filtration rate, ml/min, m±σ	42.3±7.4*	44.8±6.4*	58.3±6.5
Serum sodium level on admission, mmol/l, m±σ	132.5 (132; 133.5)*	136.5 (136; 137.5)*	139.5 (138; 142.5)

Note: BMI — body mass index, DBP — diastolic blood pressure, HR — heart rate, SBP — systolic blood pressure; * — differences are significant (p<0.05) compared to patients with normonatremia

Table 2. *The severity of CHF (m ± σ, Me (Q1; Q3))*

Parameter	Prehospital hyponatremia (n=64)	Hospital hyponatremia (n=68)	Normo-natremia (n=264)
NYHA class	IV (III; IV)*	IV (III; IV)*	III (III; IV)*
Signs of fluid retention in two circles of blood circulation, number of patients (%)	56 (87.5%)*	61 (89.7%)*	183 (69.3%)
Anasarca, number of patients (%)	13 (20.3%)	17 (25.0%)	29 (11.0%)
6-minute walk test distance, m, m±σ (n=286)	154.5±9.1*	168.4±11.3*	205.6±13.2
Clinical Assessment Scale of CHF (V. Yu. Mareyev), points, Me (Q1; Q3)	9 (8; 10)*	10 (8; 11)*	7 (6; 8)
Left ventricular ejection fraction, %, m±σ	41.6±6.9*	43.8±7.1*	49.6±7.8
E/e, m±σ	17.8±6.8*	18.4±7.9*	15.6±5.3

Note: * — differences are significant (p<0.05) compared to patients with normonatremia

Table 3. *Hospital medical therapy (number of patients, %)*

Group of drugs	Prehospital hyponatremia (n=64)	Hospital hyponatremia (n=68)	Normo-natremia (n=264)
ACE inhibitors	54 (84.4%)	53 (77.9%)	221 (83.7%)
ARBs	10 (15.6%)	15 (22.1%)	38 (14.4%)
β-blockers	56 (87.5%)	59 (86.8%)	238 (90.2%)
Loop diuretics	64 (100.0%)	68 (100.0%)	264 (100.0%)
MCR antagonists	54 (84.4%)	66 (97.1%)#*	202 (76.5%)
Thiazide and thiazide-like diuretics	19 (29.7%)*	28 (41.2%)*	42 (15.9%)

Note: ACE — angiotensin converting enzyme, ARBs — angiotensin-2 receptor ablockers, MCR — mineralocorticoid receptors; * — differences are significant (p<0.05) compared to patients with normonatremia; # — differences are significant (p<0.05) compared to patients with prehospital hyponatremia

Table 4. *Types and daily average doses of diuretics in the period of active diuretic therapy*

Group of drugs	Prehospital hyponatremia (n=64)	Hospital hyponatremia (n=68)	Normo-natremia (n=264)
Loop diuretics:			
Furosemide, number of patients (%)	52 (81.3%)	46 (67.7%)	194 (73.5%)
Torasemide, number of patients (%)	12 (18.8%)	22 (32.4%)	70 (26.5%)
Average daily dose (in terms of furosemide), mg, (Me (Q1; Q3))	80 (60; 120)	80 (80; 120)	80 (60; 120)
Thiazide diuretics (hydrochlorothiazide):			
Number of patients (%)	11 (17.2%)	27 (39.7%)*	29 (11.0%)
average daily dose, mg, (Me (Q1; Q3))	25 (12.5; 25)*	25 (12.5; 25)*	12.5 (12.5; 25)
Thiazide-like diuretics (indapamide):			
Number of patients (%)	8 (12.5%)	11 (16.2%)*	13 (4.9%)
average daily dose, mg (Me (Q1; Q3))	1.25 (1.25; 1.5)*	1.25 (1.25; 1.5)*	0.625 (0.625; 1.25)
Mineralocorticoid receptor antagonists:			
Spironolactone, number of patients (%)	51 (79.7%)	62 (93.9%)*#	188 (71.2%)
Spironolactone, average daily dose, mg (Me (Q1; Q3))	100 (75; 100)*	150 (100; 150)*#	75 (50; 100)
Eplerenone, number of patients (%)	3 (4.7%)	4 (5.9%)	14 (5.3%)
Eplerenone, average daily dose, mg (Me (Q1; Q3))	25 (25; 25)	25 (25; 25)	25 (25; 25)

Note: * — differences are significant (p<0.05) compared to patients with normonatremia; # — differences are significant (p<0.05) compared to patients with prehospital hyponatremia

Table 5. *Primary endpoint and duration of hospitalization*

Group of patients	Parameter			
	The combined primary endpoint, number of patients (%)	Hospital mortality, number of patients (%)	Transfer to intensive care unit, number of patients (%)	Duration of hospitalization, days, m±σ
Hyponatremia, all types (n=132)	38 (28.8%)*	16 (12.1%)*	22 (16.7%)*	19.6±3.8*
Prehospital hyponatremia, all types (n=64)	20 (31.3%)*	9 (14.1%)*	11 (17.2%)*	18.2±3.7*
Prehospital non-progressive hyponatremia (n=48)	12 (25%)*	3 (6.3%)	6 (12.5%)	17.9 ± 2.9
Prehospital progressive hyponatremia (n=16)	8 (50%)*	6 (37.5%)*	5 (31.3%)*	19.8±4.0*
Hospital hyponatremia (n=68)	18 (26.5%)*	7 (10.3%)	11 (16.2%)*	19.2±3.5*
Normonatremia (n=264)	27 (10.2%)	14 (5.3%)	13 (4.9)	16.2±3.2

Note: * — differences are significant (p<0.05) compared to patients with normonatremia

Table 6. *Reasons for the transfer of patients to the intensive care unit, number of patients (%)*

Group of patients	Reasons				
	Hypotension / hypoperfusion of organs	Pulmonary edema	Diuretic resistance	Arrhythmias	Other reasons
Hyponatremia, all types (n=132)	7 (5.3%)*	5 (3.8%)	6 (4.5%)	2 (1.5%)	2 (1.5%)
Prehospital hyponatremia, all types (n=64)	3 (4.7%)	3 (4.7%)	4 (6.3%)*	-	1 (1.6%)
Prehospital non-progressive hyponatremia (n=48)	2 (4.2%)	1 (2.1%)	2 (4.2%)	-	1 (2.1%)
Prehospital progressive hyponatremia (n=16)	1 (6.3%)	1 (6.3%)	3 (18.8%)*	-	-
Hospital hyponatremia (n=68)	1 (1.5%)	3 (4.4%)	4 (5.9%)	1 (1.5%)	2 (2.9%)
Normonatremia (n=264)	2 (0.8%)	2 (0.8%)	3 (1.1%)	2 (0.8%)	4 (1.5%)

Note: * — differences are significant (p<0.05) compared to patients with normonatremia

Table 7. *Structure of hospital mortality, number of patients (%)*

Group of patients	Reasons of death								
	Cardiovascular death	Heart failure	Thrombosis and thromboembolism	Myocardial infarction	Stroke	Other cardiovascular causes	Not cardiovascular death	Pneumonia	Other reasons
Hyponatremia, all types (n=132)	12 (9.1%)*	8 (6.1%)*	3 (2.3%)	1 (0.8%)	-		4 (3.0%)	3 (2.3%)	1 (0.8%)
Prehospital hyponatremia, all types (n=64)	7 (10.9%)*	5 (7.8%)*	1 (1.6%)	-	1 (1.6%)		2 (3.1%)	1 (1.6%)	1 (1.6%)
Prehospital non-progressive hyponatremia (n=48)	3 (6.3%)	2 (4.2%)			1 (2.1%)				
Prehospital progressive hyponatremia (n=16)	5 (31.3%)*	5 (31.3%)*#	-	-	-		1 (6.3%)	1 (6.3%)	
Hospital hyponatremia (n=68)	5 (7.4%)	2 (2.9%)	1 (1.5%)	1 (1.5%)	-	1 (1.5%)	2 (2.9%)	1 (1.5%)	1 (1.5%)
Normonatremia (n=264)	9 (3.4%)	4 (1.5%)	2 (0.8%)	1 (0.4%)	1 (0.4%)	1 (0.4%)	5 (1.9%)	3 (1.1%)	2 (0.8%)

Note: * — differences are significant (p <0.05) compared to patients with normonatremia, # — differences are significant (p <0.05) compared to patients with hospital hyponatremia

Table 8. *Relation of different types of hyponatremia with the outcomes*

Group of patients	Endpoints					
	Combined primary endpoint		Hospital mortality		Transfer to the intensive care unit	
	OR	95% CI	OR	95% CI	OR	95% /CI
Hyponatremia, all types (n=132)	3.1*	2.0-6.6	2.2*	1.2-4.9	2.6*	1.7-6.4
Prehospital hyponatremia, all types (n=64)	3.9*	2.1-8.2	2.6*	1.1-6.1	3.6*	1.3-8.1
Prehospital non-progressive hyponatremia (n=48)	2.6*	1.2-6.1	1.1	0.3-3.9	1.8	0.9-8.2
Prehospital progressive hyponatremia (n=16)	6.8*	2.8-23.1	8.7*	5.4-31.2	6.3*	2.3-25.0
Hospital hyponatremia (n=68)	2.9*	1.2-5.2	1.9	0.9-4.3	2.6*	1.4-6.5

Note: OR — odds ratio, CI — confidence interval, * — p < 0.05

among this cohort of patients was several times higher than in the subgroups of patients with hospital and prehospital non-progressive hyponatremia and reached 31.3% (Table 7).

Age-, comorbidity- and CHF severity-adjusted regression analysis showed that the presence of hyponatremia was associated with a significant increase in the risk of death and transfer to the intensive care unit (Table 8). In this case, prehospital hyponatremia had a greater prognostic value in relation to unfavorable outcome compared to the hospital one. It is noteworthy that the progression of hyponatremia which existed at admission significantly increased the risk of the end point achievement. At the same time, in-hospital non-progressive hyponatremia retained its influence in the multivariate model only on the risk of developing a combined end point, while its prognostic value for individual components of the latter did not reach statistical significance.

Discussion

The results of the study confirm the literature data that hyponatremia is a common disorder of the water-electrolyte balance in patients hospitalized for decompensated CHF. According to our data, in general, its rate reached 33.3%, while 48.5% of patients had it at the prehospital stage and 51.5% during treatment in the Department. Hospital hyponatremia developed on average by the end of the first week of hospital stay. Interestingly, at the time of discharge, spontaneous normalization of sodium levels occurred only in 7.8% of patients, and on the contrary, there was a progression of hyponatremia in 25% of patients. In the vast majority of cases, patients with decompensated CHF experienced mild hyponatremia, while the incidence of moderate and severe hyponatremia was low (84.8% and 4.6%, respectively).

It is believed that a decrease in blood sodium level in CHF is a variant of hypervolemic hypernatremia and is caused mainly by a violation of water excretion by the body [22, 23]. In this case, the pathophysiological basis for the development of hyponatremias is excessive neurohumoral activation in conditions of reduction of cardiac output. Despite the absolute excess of water in the extracellular space of the body in decompensated CHF, the effective volume of circulating blood remains low, which promotes non-osmotic stimulation of antidiuretic hormone secretion and the effectors of the renin-angiotensin-aldosterone system through the system of baroreceptors. Such neurohumoral activation is compensatory in nature and is aimed at normalization of perfusion pressure by limiting the excretion of sodium and water. The release of antidiuretic hormone directly increases the reabsorption of water in the collecting tubules of the kidneys, while angiotensin-2 and noradrenaline limit the delivery of water to the kidneys by reducing their perfusion and therefore contribute to the reduction of its excretion [24]. In addition, the decrease in cardiac output and a high level of angiotensin-2 are potent stimuli of thirst, which leads to an increase in water consumption.

The degree of neurohumoral activation in CHF, and therefore the risk of hyponatremia usually correlate with the severity of cardiac dysfunction, as confirmed in our study. Indeed, patients with hyponatremia had more severe clinical (FC by NYHA, the severity of congestion, distance in 6-minute walk test) and echocardiographic (the degree of systolic and diastolic dysfunction) signs of CHF compared with patients with normal blood sodium level.

It can not be excluded that active diuretic therapy has a certain contribution to the development of hospital hyponatremia. Thus, according to the results of our study, patients with hyponatremia significantly more often and in higher doses received thiazide, thiazide-like diuretics and spironolactone. It is noteworthy that the highest doses of spironolactone were taken by patients with hyponatremia which developed during hospitalization. Of course, providing adequate sodium consumption and diuresis is an important factor in the prevention of symptoms of decompensated CHF, and a necessary condition for their elimination in the development of hypervolemia signs. However, it is worth remembering that the use of diuretic therapy may be accompanied by the development of adverse side effects, among which the leading position is occupied by various electrolyte disorders including hyponatremia. According to literature, hyponatremia is most often observed with the use of thiazide and thiazide-like diuretics, and more rarely with loop and potassium-sparing diuretics [22].

Thiazide diuretics have a mixed mechanism of hyponatremic action: they enhance the effect of antidiuretic hormone at the level of collecting tubules and at the same time stimulate natriuresis. The development of hyponatremia when using spironolactone, in addition to the natriuretic effect of the drug, is associated with its ability to block the release of sodium from the cell, which leads to an increase in the intracellular electrolyte content and a decrease in its blood concentration.

Thus, active diuretic therapy in patients with decompensated CHF, especially with the use of thiazide, thiazide-like diuretics and high doses of spironolactone, should be carried out with caution, under the control of blood electrolytes, which is regulated by modern guidelines [21].

It remains unclear whether hyponatremia per se is a factor determining the deterioration of the prognosis for CHF, or whether it only acts as a laboratory marker of disease severity. Further extensive studies are needed to answer this question. However, in any case, reducing the level of blood sodium can serve as a simple and reliable tool for risk stratification in patients with decompensated CHF, as it is associated with deterioration of the hospital prognosis and prolongation of hospitalization.

Conclusion

Hyponatremia is a frequent disorder of the water-electrolyte balance in patients hospitalized due to decompensated CHF. In general, its rate reaches 33.3%, while 48.5% of patients experience it at the prehospital stage and 51.5% during treatment in the Department. By the time of discharge, spontaneous normalization of sodium levels occurs only in 7.8% of patients, and on the contrary, there is a progression of hyponatremia in 25% of patients.

Presence of both pre-hospital and in-hospital hyponatremia is associated with more severe CHF, poor in-hospital prognosis and prolonged period of stay in the hospital. The progression of existing hyponatremia significantly increases the risk of death and transfer to the intensive care unit.

Conflict of interests

The authors declare no conflict of interests.

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