

# Features Of Functional Condition Of Kidney In Patients With Heart Failure Reckoning On The Representation Of The Components Of Metabolic Syndrome

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## ABSTRACT

*The aim of the study was to review the features of the functional state of the kidneys in patients with cardiopathy, betting on the presentation of the components of the metabolic syndrome. For the research we examined 197 male patients with chronic heart condition (CHF) of ischemic genesis of II-III functional class (FC) in line with NYHA. Metabolic syndrome in patients with chronic heart failure HFmrEF exacerbates impaired renal function, which has developed together of the most pathogenetic links in chronic heart condition. it's been established that because the metabolic syndrome progresses (attachment of T2DM to other components of the metabolic syndrome), the phenomena of functional kidney failure increase. Evidence of the importance of the metabolic syndrome within the nature of the clinical manifestations of chronic coronary failure HFmrEF is that the established dependence of the identified disorders on the severity of the metabolic syndrome.*

**Key words:** metabolic syndrome, chronic kidney disease, glomerular filtration rate, heart insufficiency.

## 1. INTRODUCTION

As per various imminent investigations, even atiny low diminishing in kidney work is claimed with an expanded danger of cardiovascular illness (CVI) and mortality (CVM), paying little heed to other danger factors. it has been indicated that the pervasiveness of CVI within the amount of inhabitants in patients with diminished practical capacity of the kidneys is 64% beyond in those with unblemished capacity. A free opposite relationship was found between the glomerular filtration rate (GFR) <60 ml/min/1.73 m<sup>2</sup> and also the expanded

danger of death, complexities and hospitalization [5, 7, 23]. The occurrence of latest cardiovascular intricacies is 4.8% in patients with stage 2 ongoing kidney illness (CKI) and nearly copies little by little 3-4 [17, 25]. The idea of cardiorenal condition (CRC), which infers the speculation of the instruments of CKI improvement in CVI, clarified by the solidarity of danger factors, generally decides the exacerbating of the primary forecast in patients with metabolic syndrome (MS). A striking illustration of this condition is shown by the case of atherosclerosis and abdominal obesity (AO). Hypertriglyceridemia, dyslipidemia and DM, which are parts of MS [3, 20], are additionally the customary revised and normal danger factors for CVI and CKI. The part of the kidneys within the pathogenesis and improvement of CHI is that the topic of a vivacious conversation, the intensity of which is given by the presence of a serious stretch of inert renal brokenness [2, 5, 21]. This condition can keep going for quite very very long time, steadily irritating and forming into an undeniable pathology, showed by clinical markers of constant renal disappointment (CRF) and renal decompensation. Subsequently, it's particularly significant for clinicians to differentiate the underlying time of renal brokenness, when forceful strategies of recommending medications can hinder the cycle of annihilation of the renal glomerulus and alter the long run destiny of the patient.

**Objective:** to check the features of the functional state of the kidneys in patients with cardiopathy, betting on the representation of the components of the metabolic syndrome.

## 2. MATERIALS AND RESEARCH METHODS

To achieve the set objectives, 197 male patients with chronic heart disease (CHF) of ischemic genesis of II-III functional class (FC) in keeping with NYHA [18, 21], aged 40-60 years with a history of infarction from 6 months, were examined. up to five years. betting on the presence of MS components, 3 groups of patients were identified: Group I (n = 70), patients without MS; Group II (n = 67) patients with various combinations of dyslipidemia (HDL-C <1.03 mmol / L; LDL-C > 3.0 mmol / L) with abdominal obesity (AO), AH and hypertriglyceridemia (HTG); Group III (n = 60) patients with various combinations of dyslipoproteinemia (DLP) and sort 2 diabetes (DM2) with AO, AH and hypertriglyceridemia (HTG).

According to the classification of CHF (ESC 2016) [1, 18, 24], looking on the extent of ejection fraction (EF), all examined patients with CHF were also subdivided into subgroups: with normal (preserved EF) -  $EF \geq 50\%$  (HFpEF) and intermediate EF (gray zone) -  $EF = 40-49\%$  (HFmrEF). Thus, in group I patients with CHF without MS, 42 had preserved EF and 28 patients had intermediate EF, in group II, 39 patients had preserved EF, 28 patients with CHF had intermediate EF and in group III, 28 - x retained EF and at 32 intermediate EF (Fig. 1.).

For the diagnosis of MS, the standards for the diagnosis of MS of the International Diabetes Federation (IDF, 2009) were used [5, 21]. the most components of MS were considered: abdominal obesity (AO) (> 94 cm for men); triglyceride level (TG > 1.7 mmol / l); lipoprotein cholesterol (HDL-C <1.03 for men); vital sign (SBP > 130 mm Hg; DBP > 85 mm Hg), fasting glucose (> 5.6 mmol / L), or the presence of type 2 diabetes.

The survey didn't include patients with severe CHF (CHF with low EF <40%), CHF of non-ischemic origin, with acute cerebrovascular accidents, previous stroke, severe diabetes and insulin-dependent diabetes, chronic obstructive pulmonary diseases, high-grade arrhythmias, disease, severe nephropathy.

The study of blood biochemical parameters - total cholesterol, high-density cholesterol, triglycerides), fasting glucose was allotted on a biochemical analyzer

SYNCHRON CX SYSTEMS "BECKMAN". The calculation of low-density cholesterol and cholesterol coefficient of atherogenicity was disbursed in step with the formulas:

$$\text{LDLP} = \text{OH} - (\text{HDLP}) - \text{TG}: 2 \text{ and } \text{HCA} = (\text{OH} - \text{HDLP}): 69 (\text{HDLP}).$$

For the study, we used blood from the cubital vein taken from patients of both groups within the morning on an empty stomach.

The study of the functional state of the kidneys included the determination of the amount of serum creatinine, the excretion of albumin within the urine (determination of microalbuminuria (MAU  $\geq 300$  mg / L) in a very single morning urine using indicator strips (Biosensor AN, Russia), the glomerular filtration rate (GFR) calculated by the formula EPI GFR, which takes into consideration race, gender, age, serum creatinine To calculate GFR using the CKI-EPI formula, you'll be able to use special applications for mobile devices (QxMDCalculator) [17].

$$\text{GFR} = 141 \times (0.993)^{\text{Age}} \times \text{Cr} / 0.9) - 0.412 (\text{Whites, male})$$

$$\text{GFR} = 149 \times (0.993)^{\text{Age}} \times \text{Cr} / 0.9) - 0.412 (\text{Asian, male})$$

The stage of chronic nephrosis (CKI) was also determined by the amount of GFR and therefore the combined risk of progression of CKI and cardiovascular complications, counting on the degree of decrease in GFR and albuminuria [5, 17, 25].

The data obtained during the study were subjected to statistical processing on a Pentium-IV pc using the Microsoft Office Excel-2012 software package, including the utilization of built-in statistical processing functions. Methods of variational parametric and nonparametric statistics were used with the calculation of the mean value of the studied indicator (M), variance (SD), relative values (frequency,%), the statistical significance of the measurements obtained when comparing the mean values made up our minds by the Student's test (t) with the calculation of the error probability (R). Comparison of three or more independent groups was applied by one-way analysis of variations ANOVA. the extent of reliability  $P < 0.05$  was taken as statistically significant changes.

### 3. RESULTS AND DISCUSSION

The study of the parameters of the functional state of the kidneys showed that in group I patients with CHF HFpEF in 12 patients (28.5%) out of 42 patients MAU (MAU  $\geq 0,200$ mg / l) was detected. The clearance of serum creatinine during this group was  $88.02 \pm 7.58 \mu\text{mol} / \text{L}$ , and therefore the GFR was  $87.14 \pm 6.13 \text{ ml} / \text{min}$  (Table 1.). In group II, 18 out of 39 patients had MAU (46.2%). There was also a small decrease in GFR, which amounted to  $80.46 \pm 5.76 \text{ ml} / \text{min}$ . At the identical time, there was a rise within the level of serum creatinine by 11.4%, reaching A level of  $98.05 \pm 5.76 \mu\text{mol} / \text{l}$  ( $P < 0.05$ ).

Table # 1.

Renal function base-line in patients with CHF HFpEF and MS (M  $\pm$  SD)

Indicators	I group (n=42)	II group (n=39)	III group (n=28)
Creatinine $\mu\text{mol} / \text{L}$	$88,02 \pm 7,58$	$98,05 \pm 7,46^*$	$99,68 \pm 6,94^{**}$
GFR, $\text{ml} / \text{min}$	$87,14 \pm 6,13$	$80,46 \pm 6,76^*$	$75,77 \pm 6,04^{**}$
MAU, $\text{mg} / \text{l}$	12 (28,5%)	18 (46,2%)	19 (67,9%)

Note \* differences regarding data I gr are significant  
(\*\* -  $P < 0.05$ ; \*\* -  $P < 0.01$ ; \*\*\* -  $P < 0.001$ )

Analysis of renal function parameters in group III revealed a big decrease in GFR by 15.0% ( $P < 0.01$ ) with a rise in blood creatinine by 13.3% ( $P < 0.01$ ) in relevancy the information of group I (tab . 1.). during this group of patients, 19 had microalbuminuria,

which was 67.9%. There was a big difference between the GFR values of the II and III groups of patients with CHF HFpEF (preserved EF) by 8.7% ( $P < 0.05$ ). Next, we studied the cardiorenal relationship in CHF and MS by identifying correlations between the extent of GFR and cardiovascular indicators. Thus, in group I patients with CHF preserved EF (HFpEF), there was a moderate indirect correlation between GFR and MS parameters: blood TG level ( $r = -0.37$ ;  $P < 0.05$ ) and SBP level ( $r = -0.40$ ;  $P < 0.05$ ) (tab. 2.)

Table 2  
Indicators of cardiorenal relationship in patients with CHF HFpEF and MS

Indicators	1 group (n=42)	2 group (n=39)	3 group (n=28)
GFR - SBP	$r = -0,37^*$	$r = 0,05$	$r = 0,22$
GFR - TG	$r = -0,4^*$	$r = 0,12$	$r = 0,28$
GFR - HDL	$r = 0,21$	$r = 0,14$	$r = 0,39^*$
GFR - glucose	$r = -0,01$	$r = 0,09$	$r = -0,42^{**}$
GFR -EF	$r = 0,12$	$r = 0,42^{**}$	$r = 0,13$
GFR -RE/RA	$r = 0,08$	$r = 0,40^*$	$r = 0,43^{**}$
GFR -ETDV	$r = 0,09$	$r = 0,42^{**}$	$r = 0,40^*$

Note: at  $r = 0.3-0.4$ :  $P < 0.05$ ; at  $r > 0.41$ :  $P < 0.01$

In group II patients with CHF HFpEF and MS, a moderate correlation was observed between the amount of GFR and therefore the indicator of LV systolic function (EF) - ( $r = 0.42$ ;  $P < 0.01$ ), indicator of LV diastolic function (RE / RA ratio) - ( $r = 0.40$ ;  $P < 0.05$ ) and endothelial function index (ETDV) - ( $r = 0.42$ ;  $P < 0.01$ ).

In group III of the study with CHF preserved EF and MS (+ DM2), there was a major correlation between GFR and therefore the indicator of LV diastolic function (PE/RA ratio) - ( $r = 0.43$ ;  $P < 0.01$ ) and also the indicator of endothelial function (ETDV) - ( $r = 0.40$ ;  $P < 0.05$ ). Also during this group, there was a big correlation between the extent of GFR and MS parameters: HDL ( $r = 0.39$ ;  $P < 0.05$ ) and fasting glucose ( $r = -0.42$ ;  $P < 0.01$ ).

Thus, the violation of the functional state of the kidneys, the severity and nature of this dysfunction depends on the presence and nature of the representation of the components of MS. It had been found that CHF HFpEF without MS proceeds with less pronounced manifestations of renal dysfunction, in contrast to patients with MS. Each component of MS can cause kidney damage. The mixture of AO, arterial hypertension, and hypertriglyceridemia is an independent predictor of a decrease in GFR. AH together with disorders of carbohydrate and lipid metabolism is an independent factor of kidney damage (increased urinary albumin excretion, increased serum creatinine concentration and decreased GFR).

When studying the parameters of the functional state of the kidneys in patients with CHF HFmrEF group 1, MAU was identified in 10 patients (35.7%) out of 28. The serum creatinine clearance during this group was  $89.0 \pm 8.49 \mu\text{mol} / \text{L}$ , and therefore the GFR level was  $88.58 \pm 8.36 \text{ ml} / \text{min}$  (Table 3.). With the event of MS in group II, 16 out of 28 patients had MAU (53.6%). There was also a decrease in GFR by 11.3% ( $P < 0.05$ ) with a rise in serum creatinine by 12.7% ( $P < 0.01$ ), in contrast to the info of group I of the study. Further analysis of renal function parameters in group III CHF HFmrEF revealed a major decrease in GFR (by 23.0%;  $P < 0.01$ ) with a rise in blood creatinine (by 21.8%;  $P < 0.01$ ) in relevancy data from I groups (tab. 3.).

Table 3. Renal function baseline in patients with CHF HFmrEF and MS ( $M \pm SD$ )

Indicators	I group (n=28)	II group (n=28)	III group (n=32)
Creatinine $\mu\text{mol} / \text{L}$	$89,0 \pm 8,46$	$99,42 \pm 8,17^{**}$	$108,48 \pm 8,10^{***}$

<b>GFR, ml / min</b>	<b>88,58±8,35</b>	<b>79,10±8,01*</b>	<b>71,37±9,03**•</b>
<b>MAU, mg / l</b>	<b>10 (35,75%)</b>	<b>16 (53,6%)</b>	<b>24 (85,7%)</b>

Note \* differences regarding data I gr are significant

(\*\* - P <0.05; \*\* - P <0.01; \*\*\* - P <0.001)

▪ differences between II and III groups (▪- P <0.05)

Also, in group III of patients with CHF and MS (+ T2DM), microalbuminuria was detected in 24 (85.7%) out of 32 patients. When comparing the indices of renal function between groups II and III of the study, there was a major difference within the level of creatinine clearance and glomerular filtration rate by 11.3% and 10.1% (P <0.05), respectively.

Violation of the functional state of the kidneys, the severity and nature of this dysfunction depends on the presence and nature of the presence of MS components. we've got found that CHF without MS occurs with less pronounced manifestations of renal dysfunction, in contrast to patients with MS. Each component of MS can cause kidney damage. the mixture of AO, arterial hypertension, and hypertriglyceridemia is an independent predictor of a decrease in GFR. AH together with disorders of carbohydrate and lipid metabolism is an independent factor of kidney damage (increased urinary albumin excretion, increased serum creatinine concentration and decreased GFR).

Table 4.

Indicators of cardiorenal relationship in patients with CHF HFmrEF and MS

<b>Indicators</b>	<b>1 group (n=28)</b>	<b>2 group (n=28)</b>	<b>3 group (n=32)</b>
GFR - SBP	r= 0,04	r= -0,53**	r= 0,22
GFR -TG	r= -0,46**	r= 0,11	r= -0,63**
GFR -glucose	r= -0,46**	r= 0,19	r= -0,74***
GFR -BMI	r= -0,49**	r= -0,43*	r=-0,22
GFR - EF	r= 0,58**	r= 0,72***	r= 0,55**
GFR -IVRT	r= -0,51**	r= -0,55**	r= 0,13
GFR -RE/RA	r= 0,09	r= 0,22	r= 0,49**
GFR - ETDV	r= 0,69**	r= 0,18	r= 0,17

Note: at r = 0.3-0.4: P <0.05; when r = 0.41-0.6: P <0.01; at r > 0.6: P <0.001.

The study of cardiorenal relationships in patients with intermediate ejection fraction (HFmrEF) also revealed significant correlations between the studied parameters. In group I patients with CHF HFmrEF, there was a mean correlation between GFR and MS parameters: blood TG level (r = -0.46; P <0.01), BMI (r = -0.49; P <0.01) and fasting glucose (r = -0.46; P <0.01). Also during this group, correlations were revealed between the amount of GFR and LVEF (r = 0.58; P <0.01), IVRT (r = -0.51; P <0.01), ETDV (r = 0.69; P <0.001), which confirms the importance of metabolic syndrome within the development and progression of cattle in patients with CHF (Table 4.).

In group II patients with CHF HFmrEF and MS, a major correlation was observed between the extent of GFR and also the indicator of LV systolic function (EF) - (r = 0.72; P <0.001), indicator of LV diastolic function (IVRT) - (r = -0, 55; P <0.01), similarly as between the parameters of MS and also the level of GFR: BMI (r = -0.39; P <0.05) and SBP (r = -0.42; P <0.01) ... In group III of the study with CHF of intermediate EF and MS (+ DM2), there was a big correlation between GFR and therefore the indicator of LV systolic function (EF) - (r = 0.55; P <0.01) and also the indicator of LV diastolic function (PE / RA ratio) - (r = 0.49; P <0.01). Also during this group, there was a big correlation between the

amount of GFR and therefore the parameters of MS: TG ( $r = -0.63$ ;  $P < 0.001$ ) and fasting glucose ( $r = -0.74$ ;  $P < 0.001$ ).

Metabolic syndrome could be a unique combination of the foremost important and provoking CVI risk factors. There are various sorts of MS counting on the quantity and combination of symptoms [5, 10, 21]. Its classical form, the most components of which are dyslipidemia and impaired glucose tolerance (IGT / T2DM), is among profound, metabolically interrelated atherothrombotic disorders [2, 23, 26]. The relative risk of developing coronary failure in type 2 diabetes exceeds the relative risk of developing CHF in patients with arterial hypertension, smoking, physical inactivity, and valvular defects [2, 5, 11, 19, 22]. Approximately 40% of patients with T2DM die within 1 year after the primary hospitalization for CHF [4, 9, 11, 23]. Mortality among patients with MS is 2 times more than in patients without metabolic disorders [2, 21, 26]. this can be thanks to the irritating effect of MS components on the event and prognosis of CHF, which is realized through various interrelated mechanisms: insulin resistance, atherosclerosis, inflammation, specific diabetic cardiomyopathy, etc. [6, 8, 16, 23, 26].

Violation of the functional state of the kidneys, the severity and nature of this dysfunction depends on the presence and nature of the presence of MS components. we've found that CHF without MS occurs with less pronounced manifestations of renal dysfunction, in contrast to patients with MS. Each component of MS can cause kidney damage. the mix of AO, arterial hypertension, and hypertriglyceridemia is an independent predictor of a decrease in GFR. AH together with disorders of carbohydrate and lipid metabolism is an independent factor of kidney damage (increased urinary albumin excretion, increased serum creatinine concentration and decreased GFR).

Thus, MS in patients with CHF aggravates renal dysfunction, which has developed collectively of the most pathogenetic links of CHF. it had been found that as MS progresses (joining T2DM to other MS components), the phenomena of functional failure increase.

Heart and kidney disease are interrelated via the sympathetic systema nervosum [5, 11, 12, 19, 25]. In heart disease, renal dysfunction may be a strong predictor of mortality [12, 13, 17, 24]. this means the importance of assessing the combined risk of progression of CKI and also the development of CVI betting on the degree of decrease in GFR and also the severity of albuminuria. The evidence of the importance of MS within the nature of the clinical manifestations of CHF is that the established dependence of the revealed disorders on the severity of MS. Violation of the functional state of the kidneys, the severity and nature of this dysfunction depends on the presence and nature of the presence of MS components. we've got found that CHF without MS occurs with less pronounced manifestations of renal dysfunction, in contrast to patients with MS.

Studies by GUBBIO and DESIRE have shown that a rise in BMI with the manifestation of AO increases the chance of increased urinary albumin excretion [7, 9, 17, 24]. Among the standard metabolic risk factors, the mix of hypertriglyceridemia and T2DM is that the most unfavorable within the development of CKI [2, 3, 16, 18]. The components of MS, especially disorders of lipid and carbohydrate metabolism in patients with CHF and MS, are independent predictors of nephropathy. Insulin resistance, being an integral component of MS, is related to renal dysfunction. Recently, it's become obvious that the decisive role in kidney damage in patients with MS belongs to mediators secreted by adipocytes, which have a harmful effect on the glomerular endothelium and kidney tissue. Under conditions of changes within the vascular bed of the kidneys (AH, DLP, DM) disorders of neurohumoral regulation (activation of RAAS in CHF), hyperfiltration develops within the glomeruli, which is currently considered because the main factor damaging the glomerular membrane and resulting in the death of the nephron [2, 5, 6, 9, 21, 25].

Thus, MS in patients with CHF HFmrEF aggravates renal dysfunction, which has developed united of the most pathogenic links of CHF. It had been found that as MS progresses (joining T2DM to other MS components), the phenomena of functional nephrosis increase. The evidence of the importance of MS within the nature of the clinical manifestations of CHF HFmrEF is that the established dependence of the revealed disorders on the severity of MS. Violation of the functional state of the kidneys, the severity and nature of this dysfunction depends on the presence and nature of the presence of MS components. We found that CHF HFmrEF without MS proceeds with less pronounced manifestations of renal dysfunction, in contrast to patients with MS.

### **CONFLICT OF INTERESTS AND CONTRIBUTION OF AUTHORS**

The authors declare the absence of obvious and potential conflicts of interest related to the publication of this article and report on the contribution of each author.

### **SOURCE OF FINANCING**

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