

**COMPARATIVE ANALYSIS OF SOME LABORATORY AND FUNCTIONAL PARAMETERS
IN PATIENTS WITH CHRONIC HEART FAILURE WITH AND WITHOUT DIABETES MELLITUS**

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Khasanov Farrukhjon Sherali ugli

Samarkand State Medical University, Republic of Uzbekistan, Samarkand

Abstract. *This scientific article assesses the functional state of the heart using echocardiography in patients with chronic heart failure with diabetes mellitus and without diabetes mellitus. We also compared pro-inflammatory cytokines IL-6, TGF- β 1, potassium, urine albuminuria and glomerular filtration rate with the use of determining cystatin-C in the blood serum of patients. In patients with chronic heart failure and diabetes mellitus, it was found that the quality of life, the level of stability to physical activity and clinical condition are significantly reduced compared with patients without diabetes mellitus.*

Key words: *Chronic heart failure, interleukin-6, cystatin-C, glomerular filtration rate, β -transforming growth factor.*

**СРАВНИТЕЛЬНЫЙ АНАЛИЗ НЕКОТОРЫХ ЛАБОРАТОРНО-ФУНКЦИОНАЛЬНЫХ
ПОКАЗАТЕЛЕЙ У БОЛЬНЫХ ХРОНИЧЕСКОЙ СЕРДЕЧНОЙ НЕДОСТАТОЧНОСТЬЮ С
САХАРНЫМ ДИАБЕТОМ И БЕЗ НЕГО**

Хасанов Фаррухжон Шерали угли

*Самаркандский государственный медицинский университет, Республика
Узбекистан, г. Самарканд*

Резюме. *В данной научной статье проведена оценка функционального состояния сердца с помощью эхокардиографии у больных с хронической сердечной недостаточностью с сахарным диабетом и без сахарного диабета. Также сравнивали провоспалительные цитокины ИЛ-6, TGF- β 1, калий, альбуминурию мочи и скорость клубочковой фильтрации используя определение цистатина- С в сыворотке крови у больных. У больных с хронической сердечной недостаточностью и сахарным диабетом установлено, что качество жизни, уровень устойчивости к физическим нагрузкам и клиническое состояние достоверно снижены по сравнению с больными без сахарного диабета.*

Ключевые слова: *Хроническая сердечная недостаточность, интерлейкин-6, цистатин- С, скорость клубочковой фильтрации, β -трансформирующий фактор роста.*

RELEVANCE

In recent years, it has been noted that high comorbidity reduces the quality of life, leads to impaired social adaptation and increased mortality. The occurrence of comorbidity reaches 69% in 18-44 years, 93% in 45-64 years, and 98% in those over 65 [17]. In many cases, chronic heart failure (CHF) and chronic kidney disease (CKD) comorbidity has a negative impact on the quality and length of life of patients. These cases are more and more often accompanied by diabetes mellitus (DM) or the development of serious complications on its basis. Compared to the general population, the probability of developing SBK is several times higher in patients with cardiovascular diseases, including SUE. Even small declines in renal function are associated with increased cardiovascular risk, independent of other factors. According to the NHANES III registry, the presence of any two cardiovascular risk factors results in a glomerular filtration rate (GFR) of <60 ml per minute [6].

When SYuE occurs in a comorbid condition with type II QD, it has a significant negative impact on the course and prognosis of the disease. Patients with SYuE type II QD with preserved or reduced left ventricular ejection fraction are classified into higher NYHA functional classes (FS) compared to patients with the same left ventricular ejection fraction without QD [18, 14]. Fibrosis processes in the myocardium and kidneys rapidly develop due to the excess of pro-inflammatory cytokines at the tissue level, including IL-6, TGF- β 1 in blood serum in patients with QD [11].

Fibrous changes in the kidneys in patients with SYuE lead to a decrease in CFT and the development of SBK, which is one of the most observed unpleasant complications of the disease [5, 13]. Evaluation of CFT using creatinine level in blood serum does not always allow timely and complete detection of changes in kidneys [9]. Currently, the endogenous marker cystatin-S, which is more reliable than creatinine, is used to determine KFT. With its help, not only glomerular filtration indicators, but also the condition of the kidney's proximal tubules are evaluated. The high level of this marker in the blood is an early indication of the presence of pathological processes in the kidney [3].

The results of epidemiological and population studies testify that early, even subclinical disorders of kidney function lead to a sharp deterioration of the condition of patients with SYuE. According to the data of several authors, kidney dysfunction in SYuE is determined in 32-60% of cases using criteria such as creatinine, creatinine clearance, KFT, cystatin S, microalbuminuria [8].

It is known from numerous studies conducted in different countries of the world and published literature that the main cause of terminal renal failure is type 2 QD [15]. In diabetic nephropathy, the artery, arterioles, kidney balls and tubules are damaged. As a result, diffuse or nodular glomerulosclerosis develops in the kidneys, causing SBK. QD increases the risk of developing SYuE by 2-5 times. In the presence of these two pathological processes, the mortality rate increases by 60-80%. From a clinical point of view, 3 stages of diabetic nephropathy are distinguished. The first of them is

microalbuminuria, the second is proteinuria with preserved renal function, and the third is chronic renal failure.

Advances in molecular medicine and experimental nephrology have made it possible to better study the mechanisms of development of microalbuminuria and proteinuria. According to current research, structural and functional changes develop in the kidneys long before the excretion of albumins in the urine. In this case, podocytes, which are the main component of the diaphragm of the glomerulus, take the leading place. It is shown that the changes noted in them appear long before the occurrence of microalbuminuria. A podocyte is a complex structure that performs a number of functions under physiological conditions and has adaptive properties and is at the same time highly sensitive to various damaging factors. A number of changes (metabolic, toxic, hemodynamic) are observed in podocytes under the influence of various pathogenic agents [12].

The pathogenesis of diabetic nephropathy is complex and involves a number of factors. Metabolic (hyperglycemia, hyperlipidemia) and hemodynamic (glomerular hypertension, arterial hypertension) changes are the most studied and proven among them [1].

Hyperglycemia is one of the important metabolic factors that damage the kidney, and in this condition, stable glycosylation occurs. Autooxidation processes and interaction with cell receptors occur in the human body. As a result of the complex chemical processes that follow, protein structures change, which in turn causes permanent damage to cells. The final products of glycosylation cause changes in the metabolism of the main proteins in the body. They, in turn, increase the proliferation of cells, which aggravates the processes of diabetic nephropathy [7, 16, 10].

Normally, the processes of proliferation and apoptosis are in mutual balance. Due to the activation of a number of factors caused by the development of hyperglycemia and endothelial dysfunction in QD, the balance shifts towards proliferation.

It confirms that nephropathy developed due to QD is a complex process. On the basis of them, the development of SYuE or the last complication together with diabetes not only increase the number of patients in the hospital, but also increase the mortality rate. This, in turn, indicates the need to continue research on the problem.

The purpose of the study is to compare various laboratory and functional indicators in patients with chronic heart failure with diabetes and those without it.

Research source and methods. In the scientific work, 80 patients with chronic heart failure in co-morbidity with diabetes mellitus and without it, and with chronic kidney disease S2 and S3a developed on their basis, were observed. They, in turn, were divided into two groups of 40 patients. A-group was made up of late patients with SYuE+diabetes and B-group without SYuE+diabetes. 45 of them (56.25%) were men and 35 (43.75%) were women. In order to solve the tasks, the scientific research work was carried out as follows.

The diagnosis of SYuE and its functional classes in the patients included in the study were determined according to the criteria of the New York Heart Association (New York

Heart Association, 1964) based on their complaints, anamnesis, objective examination and laboratory-instrumental examinations.

In all cases, the diagnosis of diabetes was made after consulting an endocrinologist and confirming it with the help of appropriate laboratory tests, and patients diagnosed with type 2 diabetes with a duration of 3 or more years were included in the follow-up. During the follow-up, the patients were regularly monitored by an endocrinologist and hypoglycemic treatments were carried out according to their recommendations. All patients under observation underwent laboratory-instrumental examinations before the start of treatment and after 3 months. Also, the calculation of KFT based on the level of cystatin-S in the blood was determined according to the formula of Hoek and co-authors (2003). All immunofluorescent and biochemical tests were carried out in the clinical-laboratory diagnostic department of the multidisciplinary clinic of the Tashkent Medical Academy on COBAC 6000 (Germany-Japan) equipment.

MS Excel (2016) package computer program was used for statistical processing of the data obtained in the study. Arithmetic mean and standard deviation ($M \pm m$) of indicators presented in all tables were calculated. The reliability of differences between groups was determined using Student's t-test for odd and even differences.

Research results. From this point of view, we conducted a comparative analysis of indicators such as laboratory, cardiac remodeling and CFT in the group of patients with and without QD with SYUE in our observation. The following table 1 shows the data of echocardiography parameters performed in the main A and B group of patients involved in the study. As shown in the table, the left ventricular end-systolic size was 45.3 ± 1.8 mm and 50.35 ± 1.6 mm in II and III FS of SYUE in group A patients, and 42.6 ± 1.2 mm and 42.6 ± 1.2 mm in group B. was 46.9 ± 1.5 mm, and when they were compared, no reliable differences were found ($R > 0.05$). The diastolic size of the left ventricle was 64.4 ± 1.6 mm and 59.9 ± 1.2 mm in patients with II FS of SYUE in groups A and B, and 69.35 ± 1.5 mm and 65.3 ± 1.2 mm in patients with III FS of SYUE in both groups, respectively. was equal to and a reliable difference was recorded ($R < 0.05$).

The left ventricular end-diastolic volume was 178.3 ± 7.7 ml and 203.9 ± 7.9 ml in II and III FS of SYUE in the first group of patients, and 174.6 ± 4.1 ml and 192.3 ± 6.8 ml in the second group. no reliable differences were observed ($R > 0.05$). The left ventricular end-systolic volume was 97.3 ± 5.2 ml and 83.15 ± 3.4 ml in group A and B patients with FS II of SYUE, and a reliable difference was noted ($R < 0.05$). In patients with III FS, there was no reliable difference between the indicators of both groups (112.3 ± 9.1 ml and 104.15 ± 8.1 ml, respectively, $R > 0.05$).

Left ventricular ejection fraction (LVEF) was $42.1 \pm 1.2\%$ in FS II FS patients with QD and $46.2 \pm 1.0\%$ in FS II FS patients without QD, and a reliable difference was found ($R < 0.05$). In patients with III FS of SYUE, it was $36.5 \pm 0.9\%$ and $41.7 \pm 1.3\%$ in both groups, respectively, and a highly reliable difference was noted ($R < 0.01$). Left ventricular myocardium weight was 233.5 ± 4.4 g and 247.5 ± 6.2 g in II and III FS of SYUE in group A

patients, and 220.3 ± 3.9 g and 240.9 ± 5.5 g in group B and when they were compared, a reliable difference was found in patients with II FS ($R < 0.05$). Based on the goals and objectives, we studied pro-inflammatory cytokines IL-6, TGF- β 1, potassium, urinary albuminuria and cystatin-S in the blood serum of patients involved in the study, and the speed of glomerular filtration using it. In addition, we compared the indicators of physical load resistance, quality of life and clinical conditions of the main group A and B patients with QD and those without it. Table 2 and Figure 1 below show the results obtained.

Cystatin-C values were 1.26 ± 0.04 $\mu\text{g/ml}$ and 1.15 ± 0.05 $\mu\text{g/ml}$ in patients with SYuE II-FS in group A and B, respectively, and no significant difference was detected ($R > 0.05$). SYuE-III FS was 1.56 ± 0.08 $\mu\text{g/ml}$ in the first group and 1.25 ± 0.05 $\mu\text{g/ml}$ in the second group, and the differences were reliable ($R < 0.01$). Interleukin-6 indicators were 11.59 ± 0.8 pg/ml in group B patients with SYUE II-FS and 15.39 ± 1.5 pg/ml in group A, and were 1.33 times higher, and a reliable difference was noted ($R < 0.05$). SYuE-III FS was 23.74 ± 1.7 pg/ml and 14.7 ± 0.55 pg/ml in group A and group B, respectively, which was 1.46 times higher ($R < 0.001$). When TGF β 1 indicators were compared in group A and group B patients with SYuE II-FS, the difference was reliable (4.87 ± 0.3 ng/ml and 3.58 ± 0.27 ng/ml , respectively, and 1.36 times higher, $R < 0.01$). SYuE-III FS was 7.4 ± 0.3 ng/ml in the first group and 5.89 ± 0.4 ng/ml in the second group, which was 1.27 times higher and a reliable ($R < 0.01$) difference was noted. Although serum potassium was within the normal range in all groups, it was significantly higher in patients with QD (4.42 ± 0.08 and 4.2 ± 0.07 mmol/l between groups II and III FS, respectively, $R < 0.05$, 5.02 ± 0.07 and 4.5 ± 0.08 mmol/l , $R < 0.001$). A highly reliable ($R < 0.001$) difference of 351.45 ± 10.2 mg and 298.96 ± 10.5 m was recorded in patients A and B who underwent QD with SYuE II FS in urine.

In patients with FS III in both groups, this indicator was 298.96 ± 10.5 mg and 345.95 ± 9.7 mg, and a highly reliable ($R < 0.001$) difference was observed. We evaluated the level of endurance of the patients through a 6-minute walking test. In this, patients with SYuE II FS in the main group A walked an average of 312.2 ± 16.50 meters for 6 minutes, and group B patients walked 363.2 ± 12.31 meters, and the difference between them was reliable. In both groups of patients with SYuE III FS, this indicator was highly reliable ($R < 0.01$) different from each other (192.4 ± 12.2 meters and 249.4 ± 12.50 meters in the corresponding approach, 1.3 times less). The following results were noted when the indicators of the quality of life of patients were studied using the Minnesota questionnaire. 55.6 ± 1.9 and 48.9 ± 2.1 points were found in patients with SYuE II FS in group A and B, respectively, and the difference between them was reliable. 70.4 ± 2.1 points were recorded in patients with SYuE III FS in group A, and it was also 1.2 times higher than patients in the second group ($R < 0.001$). The following results were obtained when the patients involved in the study were evaluated using the KPSH. A reliable ($R < 0.05$) difference was noted in both FS patients with SYuE QD compared to those without QD (means 6.0 ± 0.3 and 7.84 ± 0.27 points between groups, respectively, 5.2 ± 0.19 and 7.08 ± 0.3 points). Also, as we mentioned above, KFT was calculated based on cystatin-S

indicators in patients involved in the study. Figure 1 below provides this information. As shown in this figure, glomerular filtration rate was $56.75 \pm 2.2 \text{ ml/min/1.73m}^2$ and $65.8 \pm 2.89 \text{ ml/min/1.73m}^2$ in group A and group B patients with SYuE II FS, respectively, and reliable differences were found when they were compared ($R < 0.05$). In patients with SYuE III FS QD, this indicator was $45.8 \pm 2.6 \text{ ml/min/1.73m}^2$ and in those without SYuE III FS QD, it was $57.7 \pm 2.7 \text{ ml/min/1.73m}^2$, and a highly reliable difference was found ($R < 0.01$).

Summary. The conducted analyzes showed that when SYUE is comorbid, including with QD, there are negative changes in intracardiac hemodynamic indicators in accordance with its FSs compared to those without QD. These were evident in left ventricular end-diastolic size, end-systolic volume, myocardial weight, and ejection fraction. Patients with SYuE II-III FS with QD had cystatin-S, IL-6, TGF- β 1 in blood and albuminuria in urine similar to patients without QD. - was noted to be reliably higher. Also, the low CFT determined by cystatin-S in patients with QD indicates that irreversible pathological changes in the kidneys are observed in this group of patients, and as a result, they develop premature SBK. quality and clinical status showed a correspondingly reliable adverse effect of QD compared to those without.

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