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THE CLINICAL SIGNIFICANCE OF THE DAILY MONITORING OF HOLTER ECG IN CHRONIC GLOMERULONEPHRITIS AT THE PREDIALYSIS STAGE OF THE DISEASE

Abstract

This article presents the results of our own research: comprehensive clinical and laboratory examinations, including data of 24-hour Holter monitoring (HM) in 169 patients with chronic glomerulonephritis at the predialysis stage of the disease. According to HM, 60.3% of the patients examined had episodes of supraventricular ectopic beats, and 28.9% — ventricular ectopic beats. In addition, 11.2% of patients had atrioventricular block (incomplete/partial), 8.8% had atrial fibrillation, and 14.7% had painless ischemia of 1 to 3 episodes per day. Depending on the mean heart rate (HR) according to HM, patients with chronic glomerulonephritis were divided into two subgroups. Subgroup A included 38 patients with heart rate less than or equal to 70 beats/min, subgroup B — 131 patients with heart rate of more than 70 beats/min. With equal values of uric acid, total CL, HDL-C, TG, plasma creatinine and blood fibrinogen in subgroup B there was a significant increase in LDL-C concentration (3.58 (2.74; 5.54) mmol/l vs. 2.82 (2.30; 3.86) mmol/l; $p<0.05$) and a decrease in the estimated GFR (70.4 (48.8; 96.3) ml/min vs. 85.7 (31.5; 103,1) ml/min; $p<0.05$), in comparison with subgroup A. In subgroup B a tendency to increase the degree of daily urine excretion of protein was observed. The data obtained confirm the fact that HM with the analysis of heart rate is of significant clinical importance for the diagnosis of cardiovascular disorders and the prevention of cardiovascular complications in patients with chronic glomerulonephritis at the predialysis stage of the disease.

Key words: *chronic glomerulonephritis, chronic kidney disease, glomerular filtration rate, Holter monitoring, heart rate*

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Hb — hemoglobin, Ht — hematocrit, BP — blood pressure, ASP — atherosclerotic plaque, LVH — left ventricular hypertrophy, LVPW — left ventricle posterior wall, MI — myocardial infarction, LVMI — left ventricle mass index, BMI — body mass index, EDD — end-diastolic diameter, ESD — end-systolic diameter, LV — left ventricle, LA — left atrium, IVS — interventricular septum, CS — cerebral stroke, LVM — left ventricular mass, CCA — common carotid artery, RWT — left ventricular relative wall thickness, GFR — glomerular filtration rate, HM — 24-hour Holter monitoring,

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HF — heart failure, CRP — C-reactive protein, CCC — cardiovascular complications, TG — triglycerides, CIMT — carotid intima-media thickness, EF — ejection fraction, CGN — chronic glomerulonephritis, HR — heart rate, CL — cholesterol, HDL-C — high-density lipoprotein cholesterol, LDL-C — low-density lipoprotein cholesterol, EchoCG — echocardiography

Introduction

Abundant evidence shows that the most frequent forms of cardiovascular damage in CKD are clinically significant cardiac arrhythmias [1, 2], arterio- and atherosclerotic changes in the main arteries [3, 4], left ventricular hypertrophy (LVH) [5, 6], myocardial infarction (MI) [7, 8], acute and chronic heart failure (HF) [9, 10], as well as cerebral strokes (CS) [11, 12]. As a result, studies to find new diagnostic opportunities for early detection of cardiovascular disorders are becoming very relevant. In this area, evaluation of the possibilities for 24-hour Holter monitoring (HM) use in patients with chronic glomerulonephritis (CGN) at the early stages of the disease is of great interest among clinicians and researchers.

Study objective was to investigate clinical value of HM in patients with chronic glomerulonephritis at the predialysis stage of the disease.

Materials and methods

The study included 169 patients aged 17 to 71 years with an established diagnosis of CGN at the predialysis stage of the disease. The mean age of the examined patients at the time of examination was 40.5±13.6 years. The study excluded persons on long-term hemodialysis with the presence of thyrotoxicosis, fever, as well as cancer patients and pregnant women. Along with the recording of complaints and anamnestic data, physical examination of patients with measurement of heart rate (HR) was conducted, blood pressure (BP) was measured and body mass index (BMI) in kg/m^2 was determined. Laboratory examination included evaluation of red blood parameters (determination of hemoglobin (Hb) and hematocrit (Ht), erythrocyte and platelet count) and blood chemistry (concentration of electrolytes, uric acid, fibrinogen, total and C-reactive protein (CRP), creatinine). The parameters of the lipid spectrum in plasma (cholesterol (CL), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C) and triglycerides (TG)) were also studied on the Respons 920 DiaSys Diagnostic System. Glomerular filtration rate (GFR) was calculated using the CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration) formula [13] on the basis of plasma creatinine. Additionally, the magnitude of the daily urine excretion of protein was evaluated in all patients.

HM was conducted on the system for daily ECG recording SHILER-102 with 2 modified chest leads close to V1 and V6 chest leads. Nature, incidence, duration of supraventricular and ventricular cardiac arrhythmias were evaluated using HM. Before conducting HM, administration of antiarrhythmic drugs to patients under antiarrhythmic treatment was temporarily stopped 72 hours prior to examination. Non-invasive study of carotid arteries was performed in B-mode by a linear sensor with a frequency of 5–8 MHz on the Philips IE33 X matrix Live 3D ultrasound scanner. The common carotid artery (CCA) was examined, carotid intima-media thickness (CIMT) of the proximal and distal parts of the CCA was measured. Measurement of CIMT was performed three times, the calculation was done using the mean value of CIMT, which was the arithmetic mean CIMT between the right and left CCA. Thickening was indicated by an increase in CIMT by more than 0.9 mm, and atherosclerotic plaque (ASP) — by a CIMT increase of more than 1.5 mm or local compaction by 0.5 mm or 50%, compared with the CIMT value in the adjacent areas of CA [14]. To assess the geometry of the left ventricle (LV), all patients underwent a non-invasive echocardiographic (EchoCG) study on the ultrasound apparatus Sequoia 512 manufactured by Siemens-Acuson Corporation according to the generally accepted method. Thus, wall thickness, LV cavity size, longitudinal size of the left atrium (LA) were assessed from parasternal access with the long axis of the LV. The thickness of the interventricular septum (IVS, cm) and LV posterior wall (LVPW, cm) in diastole was measured, end-diastolic (EDD, cm)

and end-systolic diameters (ESD, cm) of LV were determined. Ejection fraction (EF, %) of LV was also assessed using the L. E. Teichholz formula (1976) in the absence of a- and hypokinetic zones. The mass of LV myocardium (LVM) was calculated according to the formula of R. B. Devereux et al. [15]:

$$LVM\ (g) = 0.8 - \{1.04 - (EDD + IVS + LVPW)^3 - EDD^3\} + 0.6$$

LV mass index (LVMI) was calculated on the basis of indexation of LVM by the body surface of the subject (S, m^2). To assess LV hypertrophy (LVH), LVMI was calculated, the upper value of which was 95 g/m^2 for women and 115 g/m^2 for men. LV relative wall thickness index (RWT) was calculated using the formula:

$$2N/D = (IVSd + LVPWd) / EDD$$

Depending on the values of LVMI and LV RWT the following types of changes in left ventricle geometry were identified [16]: normal LV geometry (RWT<0.42; normal LVMI), concentric remodeling (RWT≥0.42; normal LVMI), concentric hypertrophy (RWT≥0.42; LVMI above the norm), eccentric hypertrophy (RWT<0.42; LVMI above the norm).

Study design

One hundred and sixty-nine patients who underwent HM with different types of CGN were selected by random sampling (Table 1). Type of study — descriptive, with the formation of 2 subgroups based on mean HR according to HM.

Statistical analysis

The results of the study were analyzed using statistical software Statistica 10.0 developed by StatSoft. Verification of distribution normality for quantitative characteristics was carried out using Kolmogorov-Smirnov test. The values of continuous quantities are presented as M±m, where M is the sample arithmetic mean and m is the standard error of the mean. The values of qualitative characteristics are presented in the form of frequencies and percentages. The interquartile range (25th quartile; 75th quartile) in cases of nonparametric distribution of the characteristic was also used in the description of the sample [17]. To assess the significance of differences in mean values, we used the Student t-test for characteristics with normal distribution, to compare two independent groups — Mann-Whitney test. Differences were considered as statistically significant at $p<0.05$.

Study results

Table 1 shows that nephrotic and hypertensive types of the disease prevailed among the examined persons with CGN. The number of patients with the initial stage of renal dysfunction was large (Table 1).

The proportion of male patients was significantly higher compared to females (73% vs. 27%; $p<0.05$). The mean value of BMI in the examined individuals was 27.07±6.36 kg/m^2. The values of systolic and diastolic BP were in the target range, amounting to 142±26 mm Hg and 91±16 mm Hg respectively (Table 2). Mean levels of CL, TG and fibrinogen were higher, and total protein and blood albumin were lower than the established normal value. Median and interquartile range of

Table 1. *Clinical characteristics of examined patients*

Clinical types of chronic glomerulonephritis (n=169)	
Hypertensive type, abs. (%)	48 (28.4)
Latent type, abs. (%)	20 (11.8)
Nephrotic type, abs. (%)	68 (40.3)
Mixed type, abs. (%)	33 (19.5)
The severity of renal dysfunction (KDIGO, 2002) (n=169)	
Stage 1 of chronic kidney disease, abs. (%)	65 (38.4)
Stage 2 of chronic kidney disease, abs. (%)	37 (22.0)
Stage 3 A of chronic kidney disease, abs. (%)	30 (17.7)
Stage 3 B of chronic kidney disease, abs. (%)	15 (8.9)
Stage 4 of chronic kidney disease, abs. (%)	15 (8.9)
Stage 5 of chronic kidney disease, abs. (%)	7 (4.1)

Note: KDIGO — Kidney Disease: Improving Global Outcomes; n — number of patients

plasma creatinine and estimated GFR characterized subclinical renal dysfunction. Since the study was dominated by patients with nephrotic type of CGN, the median daily urinary protein excretion was 1.757 g (Table 2).

According to the results of the instrumental study, shown in Table 3, the mean values of EchoCG indices such as: the longitudinal dimension of LA, LV linear diameters (ESD, EDD, IVST, LVPWT), RWT and indicators of LV systolic function were almost within acceptable limits. When indexing LV mass to the body surface, the magnitude of LVMI was significantly higher thresholds. At the same time, eccentric and concentric variants of LV

Table 2. *Clinical and laboratory parameters of examined patients (n=169)*

Parameters	M±m
Age, years	40.5±13.6
Sex, male/female	124/45
Body mass index, kg/m^2	27.07±6.36
Systolic blood pressure, mm Hg	142±26
Diastolic blood pressure, mm Hg	91±16
Heart rate, beats/min	77±10
Hemoglobin, g/l	135.6±22.9
Hematocrit, %	45.2±7.64
RBC, x10^{12}/l	4.46±0.52
Platelets, x10^9/l	247.7±26.6
Potassium, mmol/l	4.59±0.68
Calcium, mmol/l	1.28±0.48
Sodium, mmol/l	139.4±5.96
Uric acid, mmol/l	0.396±0.097
Total cholesterol, mmol/l	6.31±2.88
High-density lipoprotein cholesterol, mmol/l	1.36±0.44
Low-density lipoprotein cholesterol, mmol/l	3.30 (2.62; 4.84)
Triglycerides, mmol/l	1.95 (1.21; 2.68)
Fibrinogen, g/l	5.108 (3.666; 6.771)
Increased CRP, abs. (%)	59 (35)
Prothrombin index, %	88.3±10.7
Total protein, g/l	57.7±14.8
Albumin, g/l	31.7±11.0
Plasma creatinine, μmol/l	116 (89.0; 184)
Daily urine protein excretion, g	1.757 (0.546; 4.305)
Estimated glomerular filtration rate, ml/min	80.7 (45.0; 109.4)

Note: n — number of patients; CRP — C-reactive protein

Table 3. *Echocardiographic indices of the examined groups of patients with CGN (n=169)*

Parameters	M±m
Left atrium, cm	3.25±0.43
Left ventricle end-systolic diameter, cm	3.32±0.46
Left ventricle end-diastolic diameter, cm	5.13±0.48
Interventricular septum thickness, cm	1.0±0.19
Left ventricle posterior wall thickness, cm	0.98±0.18
Left ventricle mass, g	306.2±94.2
Indexed left ventricle mass, g/m^2	166.3±49.3
Left ventricular relative wall thickness, U	0.385±0.070
Left ventricular ejection fraction, %	64.1±5.56
Normal geometry of left ventricle, abs. (%)	24 (14.2)
Concentric remodeling of left ventricle, abs. (%)	–
Concentric hypertrophy of left ventricle, abs. (%)	44 (26.0)
Eccentric hypertrophy of left ventricle, abs. (%)	101 (59.8)
Calcification and compaction with AV regurgitation, abs.(%)	58 (34.3)
Calcification and compaction with MV regurgitation, abs.(%)	35 (20.7)
Intima-media complex, right common carotid artery, mm	0.5 (0.4; 0.6)
Intima-media complex, left common carotid artery, mm	0.5 (0.50; 0.70)
Mean intima-media complex of common carotid artery, mm	0.5 (0.47; 0.60)
ASP in right common carotid artery, abs.(%)	24 (14.2)
ASP in left common carotid artery, abs.(%)	20 (11.8)
Maximal heart rate, beats/min	123±19
Mean heart rate, beats/min	75±10
Minimal heart rate, beats/min	53±7
Supraventricular ectopic beats (group), abs. (%)	102 (60.3)
High-grade ventricular ectopic beats, abs. (%)	49 (28.9)
Atrioventricular block (I and II degree), abs. (%)	19 (11.2)
Episodes of atrial fibrillation, abs. (%)	15 (8.8)
Transient myocardial ischemia, abs. (%)	25 (14.7)

Note: AV — aortic valve; MV — mitral valve

Table 4. *Comparison of laboratory parameters in examined subgroups*

Parameters	Group A (n=38)	Group B (n=131)
Uric acid, mmol/l	0.402±0.080	0.394±0.101
Total cholesterol, mmol/l	5.30 (4.02; 5.96)	5.54 (4.39; 7.82)
HDL-cholesterol, mmol/l	1.0 (0.88; 1.30)	1.0 (0.90; 1.39)
LDL-cholesterol, mmol/l	2.82 (2.30; 3.86)	3.58 (2.74; 5.54)*
Triglycerides, mmol/l	1.59 (1.21; 2.76)	2.02 (1.22; 2.62)
Fibrinogen, g/l	4.995 (3.443; 5.882)	5.328 (3.886; 7.770)
Plasma creatinine	136 (100.5; 227.0)	113 (88.0; 181.0)
Daily urine protein excretion, g	1.570 (0.664; 3.987)	1.815 (0.539; 4.366)
Estimated GFR, ml/min	85.7 (31.5; 103.1)	70.4 (48.8; 96.3)*

Note: n — number of patients; HDL — high-density lipoproteins; LDL — low-density lipoproteins; GFR — glomerular filtration rate; * — $p<0.05$

structural changes were revealed more often, and the number of patients with normal LV geometry decreased (Table 3). There were no cases of LV concentric remodeling in the sample. The presence of atherosclerotic and calcified changes on the aortic (34.3%) and mitral valves (20.7%) was more often recorded during ultrasound imaging of the heart valve structures.

According to HM, episodes of supraventricular ectopic beats were identified in 60.3% of the examined individuals, and ventricular ectopic beats — in 28.9% (Table 3). In addition, 11.2% of the patients had atrioventricular block (incomplete/partial), 8.8% had atrial fibrillation, and painless ischemia, 1 to 3 episodes per day, occurred in 14.7%.

It has now been established that patients with HR of more than 70 beats/min have a higher risk of developing CCC compared to patients with HR of less than 70 beats/min. This concept was the basis of the separation of the examined persons with CGN into 2 subgroups depending on the mean heart rate according to HM.

Subgroup A included patients with HR less than 70 beats per minute, subgroup B — patients with HR more than 70 beats per minute. With equal concentrations of uric acid, total CL, HDL-C, TG, plasma creatinine and blood fibrinogen, in subgroup B there was a significant increase in LDL-C (3.58 (2.74; 5.54) mmol/l versus 2.82 (2.30; 3.86) mmol/l; $p<0.05$) and a decrease in estimated GFR (70.4 (48.8; 96.3) ml/min versus 85.7 (31.5; 103.1) ml/min; $p<0.05$) compared with subgroup A. In subgroup B there was a tendency towards an increase in the degree of daily urinary protein excretion (Table 4).

Discussion

Our study focused on the evaluation of the clinical value of HM in the early diagnosis of lesions of the cardiovascular system at the CGN. Currently, there is sufficient evidence of the critical role of renal dysfunction in determining CVR in the general population [18]. Thus, patients with GFR of 60–30 ml/min/1.73 m^2 regardless of the type of CGN are at high risk, and patients with GFR≤30 ml/min/1.73 m^2 — at very high risk of CVD [19]. According to the existing recommendations [20, 16], in our study, the examined patients with CGN in terms of the severity of renal dysfunction were in the zone of high risk of cardiovascular complications (CCC) (Table 1). As early as 2004 A. S. Go et al. found that CVD prevalence in a population of patients with renal dysfunction is 64% higher than in individuals with normal renal function [21]. However, the authors were able to demonstrate the independent inverse relationship between a decrease in GFR of less than 60 ml/min/1.73 m^2 and an increase in the risk of death, CCC and hospitalization. Similar data were obtained in recent clinical and instrumental studies [22, 23].

The role of hypertension in the prognosis of cerebrovascular and cardiac complications in patients with CKD can hardly be overestimated. Timely and adequate correction of hypertension reliably delays the onset of the dialysis-dependent stage of renal dysfunction. As can be seen from Table 1, in our study, the proportion of hypertensive glomerulonephritis, i. e. persons with CGN+AH was 28.4%, and the mean BP values were 142 mm Hg for

systolic pressure and 91 mm Hg for diastolic pressure. According to some researchers, the frequency of hypertension is up to 40% at stage 1–2 of CKD, that is close to the frequency of hypertension in the general population [24, 25]. In our opinion, the relatively low prevalence of hypertension in the examined group is associated with the homogeneity of patients included in the study. In addition, our patients had median estimated GFR equal to 80.7 ml/min/1.73 m^2 (Table 2), i. e. the initial stage of the disease was revealed. According to some authors, at GFR below 60 ml/min/1.73 m^2, the frequency of AH increases sharply, and at GFR below 30 ml/min/1.73 m^2, it reaches 75% [25, 26].

In our study (Table 3) according to HM, episodes of AF were detected in 8.8% of patients. On the other hand, supraventricular and ventricular ectopic activity was observed in 60.3% and 28.9% of patients respectively. It is clear that on the one hand, high group ectopic electrical activity of the myocardium is a predictor of AF, and on the other hand, it is a predictor of the development of LV geometry impairment (Table 4). However, the mean values of the longitudinal dimension of LA in the examined persons did not go beyond the established normal values for adults. These facts are quite consistent with the results of previous studies [27, 28]. In the prospective study ARIC (Atherosclerosis Risk in Communities Study), GFR inhibition in the zone of <45 ml/min is clearly accompanied by an increase in the risk of AF by 35% [29].

Epidemiological studies have shown that the prevalence of heart failure (HF) increases concurrently with deterioration of renal function [30]. The adverse effect of reduced GFR on the structural rearrangement of arterial vessels [31] and LV was demonstrated, regardless of the presence of traditional risk factors in CGN [32]. The strongest predictor of an elevated risk of HF symptoms is concentric LVH [33, 34, 35]. We were also able to demonstrate (Table 2, 3) high incidence of eccentric (59.8%) and concentric (26.0%) types of LVH in patients with CGN at the predialysis stage of the disease (Table 3). At the same time, the mean indices of LV contractile function (EF) were preserved.

The appearance of ASP and increase in CIMT among CKD patients at the predialysis stage of the disease were obtained in the work of O. V. Pyankina et al. [36]. Life-time study of ASP structure revealed its increased vulnerability in case of renal dysfunction [37]. In normal CIMT, ASP is often detected in the carotid arteries, which was confirmed in our study (Table 3). With median of mean CIMT (0.5 mm), the presence of ASP was found in 14.2% in the right and 11.8% in the left vascular region of CCA.

HR is a specific marker of life expectancy, reflecting the state of metabolism in the body [38]. Slowing the heart rate improves the balance between myocardial oxygen supply and demand in patients with IHD and significantly reduces the risk of cardiovascular complications and death. Increased HR is one of the predictors of hypertension and kidney hemodynamic stress development [39]. Our work revealed (Table 4) a significant increase in LDL-C level and decrease in estimated GFR in the subgroup of persons with HR over 70 beats/min. In the Framingham Heart Study, the overall mortality and mortality from CVD in people with hypertension almost doubled with an increase in HR for every 40 beats per min, regardless of additional risk factors [40]. At the same time, an increase in heart rate at rest can be a marker of imbalance of the autonomic nervous system, i. e. suppression of vagal activity or increasing sympathetic activity [41]. High HR increases the risk of ASP damage due to hydrodynamic disorders, which underlies the development of acute cardiovascular and nephrocerebral events [42]. The mechanism of anti-atherosclerotic action of decreased HR is probably due to a positive effect on arterial stiffness. The increase in HR can lead to atherosclerotic induration of the arteries, which is associated with an increase in pulse wave velocity. Certainly, autoregulation of blood flow in the brain and kidneys is disturbed due to non-uniform elasticity, the presence of multiple arterial branches and low resistance of blood vessels.

Conclusion

The performance of HM in patients with CGN at the predialysis stage of the disease showed important clinical value for early diagnosis of cardiovascular disorders and prevention of their complications.

Conflict of interests

The authors declare no conflict of interests.

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