

**CHARACTERISTICS OF DAILY BLOOD PRESSURE IN PATIENTS WITH  
CHRONIC GLOMERULONEPHRITIS****Yuldasheva Gulnora****Eshqobilova Madina***Teacher of public health technical college named after Republic No. 1 Abu Ali Ibn Sina*

**Annotation:** *A study of 70 patients with chronic glomerulonephritis with hypertension aged 23 to 50 years was conducted. All patients underwent Doppler echocardiography. Patients with chronic glomerulonephritis have been shown to have more pronounced disturbances of central hemodynamics than patients with hypertension. Left ventricular diastolic dysfunction was reliably noted in this group of patients. It is more common in patients with hyperfiltration and chronic glomerulonephritis, occurs in 100% of cases, and is more pronounced than in patients with significant hypertension. Early deterioration of renal concentration function was established by maintaining nitrogen excretory function long enough.*

**Keywords:** *glomerulonephritis, hypertension, diastolic dysfunction.*

**INTRODUCTION**

Analysis of SMAD results and renal functional status in 70 patients with hypertension and chronic glomerulonephritis aged 23 to 50 years. The study showed that the number of non-dipper patients was significantly higher in the main group (AG + SGN) than in the control group. A sufficient decrease in nocturnal blood pressure indicates a violation of the circadian rhythm of blood pressure and has a significant impact on the development of renal pathology.

In chronic glomerulonephritis (SGN), the prevalence of hypertension is approximately 62%. Therefore, blood pressure is a cardiovascular risk factor. Unfortunately, cardiovascular complications are increasingly common. Therefore, it is important to comprehensively study the characteristics of the daily profile of blood pressure (BP), morphofunctional parameters of the heart, renal status, and metabolism in hypertensive patients. This allows us to assess changes in central hemodynamics for the purpose of early prevention of renal dysfunction and cardiovascular diseases.

**Research objective.** The aim of the study was to evaluate the indicators of daily monitoring of blood pressure (SMAD) and morphological and functional parameters of the heart according to echocardiography.

**LITERATURE REVIEW AND METHODOLOGY.** The study involved 70 patients with AG I-II degree according to the 2008 VNOK classification, aged 23 to 50 years: 36 patients with AG and SG formed 1 (main) group (22 men and 14 women, average age  $36.5 \pm 1.4$  years, in the male subgroup -  $36.5 \pm 1.2$  years, in the female subgroup -  $35.8 \pm 1.2$  years; AG duration  $11.7 \pm 1.2$  years, in the male subgroup -  $10.6 \pm 1.3$  years, in the female subgroup -  $12.7 \pm 1.2$  years. Unilateral kidney damage was observed in 7% of patients, and bilateral kidney damage in 93%. 34 patients with hypertension formed the second (control) group (23 men and 11 women, average age  $36.2 \pm 1.2$  years, AT 11.1 duration.  $\pm 0.9$  years). The

groups are compared by gender, age, duration and prevalence of hypertension. Antihypertensive drugs were canceled in patients 5-7 days before the study of blood pressure. Patients were monitored daily for blood pressure. (SMAD) using a portable device (BAT41-2 LLC ICSTechno) Standard parameters of SMAD were analyzed: maximum, minimum, average systolic (SQB), diastolic (DQB), heart rate (YQ), pulse QB (norm less than 53 mm Hg) electronic values. Time index (VI) SQB and DQB (norm less than 25%, more than 25% - unstable arterial hypertension, more than 50% - stable arterial hypertension), SQB, DQB variability during waking, sleeping and within 24 hours (the norm of VSAD day is less than 15.5 mm Hg, VI SQB at night is less than 14.8 mm Hg, VI SQB of the day is less than 15.2 mm Hg; VI SQB less than 13.3 mm Hg during the day. The severity of the biphasic rhythm was assessed using the traditional criteria for determining the severity of the biphasic rhythm according to the diurnal index (KI): KI value 10-20% (normal), nondipper- KI 0-10% (night blood pressure decrease in), excessive dipper - KI > 20% (excessive decrease in blood pressure at night), at night - KI < 0 (nocturnal hypertension).

DISCUSSION AND RESULTS. When analyzing the results of SMAD (Table 1), it was found that the systolic (VI SQB) and diastolic VI time coefficient (VI DQB) during the day were significantly higher in patients with AG than in patients with essential AG. ( $39.0 \pm 3.1\%$  versus  $43.2 \pm 3.2\%$  and  $43.8 \pm 3.6\%$  versus  $41.1 \pm 3.8\%$ ). The number of patients with increased variability of SQB and DQB (SMAD) during the day was greater among patients with hypertension and chronic hepatitis than among those with essential hypertension ( $49.1\%$  versus  $21.1\%$  and  $45.3\%$  versus  $22.5\%$ ).  $p < 0.05$ ). An increase in the VI variability index is a reflection of the involvement of vital organs in the process and the acceleration of the development of renal failure. Results of 24-hour blood pressure monitoring in the patients under study

Indicators 1st group AG + SGN 2nd group GK

SBP average, mm s.u.t,  $146.0 \pm 1.5$   $141, \pm 1.6$   
 SBP average, mm s.u.t,  $88.8 \pm 1.2$   $85.9 \pm 1.3$   
 SBP average, beats/min  $74.4 \pm 1.2$   $73.9 \pm 1.3$   
 SBP average, mm s.u.t,  $57.2 \pm 0.8$   $55.3 \pm 0.9$   
 VI SBP daytime, %  $69.0 \pm 3.1^*$   $53.2 \pm 3.2$   
 VI DBP daytime, %  $63.8 \pm 3.6^*$   $51.1 \pm 3.8$   
 VI SBP evening, %  $69.9 \pm 3.7$   $62.1 \pm 3.6$   
 VI DQB late,%  $68.4 \pm 3.7$   $67.4 \pm 3.5$   
 OSKB daily, mm.s.u.t.  $16.0 \pm 0.5^*$   $14.2 \pm 0.6$   
 OSKB milk >15.2 (%)  $49.1^*$   $21.1$   
 O' DQB daily, mm.s.u.t.  $12.6 \pm 0.5$   $10.8 \pm 0.4$   
 O' DQB milk >12.3 (%)  $45.3^*$   $22.5$   
 OSQB day, mm.s.u.t.  $14.9 \pm 0.5$   $14.7 \pm 0.4$   
 OSQB day, >15.5(%)  $34$   $32.5$   
 ODQB day. mm.s.u.t  $11.4 \pm 0.6$   $11.3 \pm 0.5$   
 ODQB day >13.3(%)  $18.9$   $23$

VSQB evening, mm s.u.t,  $12.0 \pm 0.5$   $12.2 \pm 0.5$

VSQB evening,  $\gt 14.8(\%)$  24.5 19.1

VDQB evening, mm s.u.t,  $9.3 \pm 0.5$   $9.4 \pm 0.5$

VDQB evening,  $\gt 11.3(\%)$  18.9 15.3

SQB, mm s.u.t,  $48.4 \pm 2.7$   $49.7 \pm 3.2$

DQB, mm s.u.t,  $34.5 \pm 1.6$   $34.6 \pm 2.1$

SQB, mm s.u.t,  $23.6 \pm 2.7^*$   $19.6 \pm 1.9$

SGN - chronic glomerulonephritis; GB - hypertension; SQB - systolic blood pressure; DQB - diastolic blood pressure; YQS - heart rate; QTU - pulse rate; VI SQB - SQB time indicator; VI DQB - DQB time indicator; OSQB - SQB variability; OSQB- DQB variability.

There were no significant differences in the level of pulse blood pressure (PQB), but in both groups the average values exceeded the norm ( $57.2 \pm 0.9$  mm Hg in hypertension and chronic hepatitis.  $55.3 \pm 0.9$  mm Hg in patients with significant hypertension). PQB  $\gt 53$  mm Hg. Art. is associated with increased large vessel stiffness and is an independent predictor of cardiovascular mortality.

When analyzing the distribution of patients according to the daily SQB index (VI SQB), the number of patients with non-dipper and peak nighttime curves was higher in the study group than in the control group (48.8% vs. 25.4%, respectively,  $p < 0.05$ ). Among patients with hypertension combined with chronic hepatitis, the number of patients with VI DQB  $< 10\%$  was also significantly higher (34.1% vs. 18.2%, respectively). An insufficient decrease in blood pressure at night, provided that the sleeping person does not have subjective sensations, indicates a violation of the circadian rhythm of blood pressure and has a significant impact on the development of renal pathology.

**CONCLUSION.** This study showed that in patients with hypertension, combined with chronic hepatitis, compared with patients with essential hypertension, diurnal blood pressure control is significantly impaired - high levels of VI SQB and DBP day and night, often absent or insufficient nighttime blood pressure decrease, as well as excessive fluctuations in blood pressure during the day.

#### REFERENCES:

1. Terentyev V.P., Batyushin M.M. Nephrogenic arterial hypertension. - Rostov-on-Don: RostGMU Publishing House, 2004. - 62 p.
2. Rogoza A.N. Daily monitoring of arterial pressure (based on materials from the methodological "ESH Recommendations 2003) // Functional Dia- Ymstika. - 2004. - No. 4. - P. 29-44.
3. Schiller N.B., Osipov M.A. Clinical echocardiography. - M.: Practice, 2005. - 344 p.
4. Yarmukhamedova S. H., Normatov M. B., Vafaeva N. A. Osobennosti sutochnogo profilya arterialnogo davleniya and bolnykh khronicheskim glomerulonephritom // Dostizheniya nauki i obrazovaniya. - 2020. - No. 11 (65).
5. Nephrology. Rukovodstvo dlya vrachey / Pod Tsd. I.E. 'Garee- wow. - M.: Medicine, 2000. - 688 p.

6. Verdecchia P., Schillaci G., Reboldi G. et al. Differential prognostic impact of 24-hour mean blood pressure and pulse pressure on stroke and coronary artery disease in essential hypertension // *Circulation*. - 2001. - Vol. 103, No. 21. - P. 2579-2584.
7. Yarmukhamedova S. Kh., Normatov M. B. Izuchenie osobennostey sutochnogo monitorirovaniya arterialnogo davleniya u bolnyx kronicheskim glomerulonephritom // *Molodoy uchenyy*. - 2020. - No. 38. - S. 48-51.
8. Belenkov Yu.N., Mareev V.Yu. Serdechno-sosudistyy sauce distyy continuum // *Serdechnaya dostatochnost*. \* 2002. - T. 3, No. 1, - S. 7-11.
9. European Best Practice Guidelines for Hemodialysis. Part 1 // *Nephrol, Dial. Transplant* 2002. - Vol. 17 (Suppl. 7). - P. 7-15.
10. Mukhin H.A. Izbrannye lektsii po vnutrennim boleznyam. - M.: Izd-vo "Litterra", 2006. - 240 p.
11. Vafoeva N. A., Gaffarov Kh. X. Osobennosti kliniko-laboratornoy diagnostiki kronicheskogo pyelonephrita u genshchin // *Natsionalnaya Assotsiatsiya Uchenykh*. - 2016. - no. 1. - S. 20-21.
12. Mancia G., De Backer G., Dominiczak A. et al. 2007 Guidelines for the management of arterial hypertension: the Task Force for the management of arterial hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC) // *J. Hypertens*. - 2007. - Vol. 25, No. 6. - P. 1105-1187.
13. K/DOQI clinical practice guidelines for managing dyslipidemias in chronic kidney disease // *Am. J. Kidney Dis*. - 2003. - Vol. 41 (Suppl. 4). - P. S1-S92.