

World Bulletin of Public Health (WBPH) Available Online at: https://www.scholarexpress.net Volume-21, April 2023 ISSN: 2749-3644

### TO INVESTIGATE THE LIFE QUALITY OF THE PATIENTS WITH DIABETES MELLITUS TYPE 2 COMPLICATED WITH CHRONIC KIDNEY DISEASE.

Sadikova N.G., Najmutdinova D.K., Urunbayeva D.A., G`ulomova M.B.

Tashkent medical academy, Uzbekistan					
Article history:		Abstract:			
Received: Accepted: Published:	February 6 <sup>th</sup> 2023 March 6 <sup>th</sup> 2023 April 10 <sup>th</sup> 2023	The clinical course and quality of life of diabetic nephropathy in patients with chronic kidney disease-induced diabetes Type 2 was studied under the SF-36 survey. For the study, 60 patients with chronic kidney disease were taken for examination. Of these, 40 QD type 2 patients make up core group 1. Control group 2 consisted of 20 SBK-infected non-QD patients. A "SF-36" questionnaire was used to assess quality of life indicators in patients on the examination. According to the SF-36 survey, all indicators are separated into physical and mental components. In patients with chronic kidney disease-complicated diabetes Type 2, there was a decrease in quality of life by the physical component by 23.5% and by the mental component by 16.9% compared to patients with chronic kidney disease as a complication of diabetes mellitus were observed to have more severe SBK compared to non-diabetic patients, and quality of life indicators were found to be dependent on the severity of SBK.			
Kaywords: disbates mellitus disbatic nentronathy chronic kidney disease alycated hemoglobin alycemic profile					

**Keywords:** diabetes mellitus, diabetic nephropathy, chronic kidney disease, glycated, hemoglobin, glycemic profile, daily diuresis, questionnaire SF-36.

**RELEVANCE OF THE TOPIC:** The 21st century is characterized by "diabetic" life circumstances in the entire history of mankind. Over the past 25 years, the prevalence of Type 2 diabetes in the United States has almost doubled. In India, Indonesia, China, Korea, and Thailand, the rate increased by 3-5 times. In 2007, 246 million people worldwide suffered from diabetes, which could reach 400 million by 2025. While 310 million people were infected with glucose tolerance disorder, known as "prediabetes", by 2012 the number had increased to 480 million. The prevalence of diabetes has been observed to increase significantly, especially in developing countries. For example, in Mexico, 18% of the adult population is expected to suffer from Type 2 diabetes by 2025. According to the WHO, there will be 130 million diabetics in China and India by 2025, whose treatment will require nearly 40% of the health bujet. This hinders the economic growth of these countries. This trend is especially clearly observed in age groups over the age of 40 and is characterized by Type 2 diabetes, which is 85-90% of the total number of diabetics.

Diabetic nephropathy ranks third after cardiovascular and oncological diseases that cause early disability and high mortality. Only 20% of patients with Type 2 diabetes reach or slightly exceed their average life expectancy. For a long time it was believed that type 2 diabetes is a completely harmless disease, and therefore it was not given enough attention. But the results of a quality of life assessment of patients aged 35-55 with mild type 2 diabetes and impaired glucose tolerance showed that every fifth patient died after 11 years.

Currently, diabetes is the main cause of the development of SBK in developed and developing countries. In 20-40% of patients who began treatment with SBK for the first time in the world, this disease was considered the main diagnosis. The number of new patients with Type 2 diabetes in Australia First increased 5-10 times between 1993 and 2012. In Japan, the number of new patients who started kidney replacement therapy for diabetic nephropathy increased 7 times between 1983 and 2015, accounting for 40% of the total number of new patients. Thus, over the next 10 years, 30% of the \$ 1.1 trillion worldwide - the estimated cost of dialysis treatmentwill be spent on the treatment of DN. A study of diabetes in the United Kingdom found that the rate of newly diagnosed Type 2 diabetes patients moving from normal albuminuria to microalbuminuria, macroalbuminuria and kidney failure was 2-3% per year. With an average medical follow-up of 15 years, 40% of the 4,000 patients included in the study developed microalbuminuria. In a DEMAND study involving 32,280 patients with Type 2 diabetes from 33 countries, microalbuminuria was observed in 39% of patients, and its prevalence rate increased depending on age, duration of diabetes, and the presence of



hypertension. The factors listed above determine the importance of the problem of Type 2 diabetes both socially and medically, and the above facts that indicate its cause in high rates of the development of chronic kidney disease once again confirm the actuality of the subject.

**RESEARCH OBJECTIVE:** Clinical course and quality of life study of diabetic nephropathy in patients with Type 2 diabetes mellitus with chronic kidney disease based on SF-36 survey.

**RESEARCH MATERIALS AND METHODS OF EXAMINATION:** 60 patients with chronic kidney disease were taken for examination. Of these, the 1st core group consists of 40 QD type 2 TTA clinic 3 Endocrinology unit and patients of the rijaetm diabetic nephropathy unit. They have an average age of 56.7±1.0, an average QD duration of 10.5±0.8, and an average SBK duration of 6.2±0.7 years. Control group 2 consisted of 20 SBK-infected non-QD patients. They have an average age of 55.8±0.9, with an average SBK duration 6.9±0.8 of years. In determining the severity of chronic kidney disease in patients examined, two indicators were used: glomerular filtration rate (GFR) and symptoms of kidney damage (proteinuria, albuminuria). Depending on their combination, 5 stages of SBK are distinguished:

Table-1.						
step description GFR, ml/min / 1, 73m2	step description GFR, ml/min / 1, 73m2	step description GFR, ml/min / 1, 73m2				
1 Nephropathy symptoms, normal GFR >90	1 Nephropathy symptoms, normal GFR >90	1 Nephropathy symptoms, normal GFR >90				
2 Nephropathy symptoms,	2 Nephropathy symptoms,	2 Nephropathy symptoms,				
Slight decrease in GFR 60-89	Slight decrease in GFR 60-89	Slight decrease in GFR 60-89				
Average decrease of 3 GFR 30-59	Average decrease of 3 GFR 30-59	Average decrease of 3 GFR 30-59				
4 average decrease in GFR 15-29	4 average decrease in GFR 15-29	4 average decrease in GFR 15-29				

A "SF-36" questionnaire was used to assess quality of life indicators in patients on the examination. The survey is made up of eight scales.

1. Physical function scale (PF) - execution of various physical loads determines the possibility-from minimum (self-service) to maximum (long walks, running, doing sports without restriction).

2. The physical factor scale (RP) is a specific age specific and defined as reflects the ability to perform work that is socially relevant (professional commitment, household).

3. Physical pain scale (P) - significance determined by survey and it can cause simple restriction of patient activity.

4. General health scale (GH) - subjective perception of the previous, current state it allows you to assess how to do and determine its prospects.

5. Survival ability scale (VT) - experience internal energy, reflects the non-existence of fatigue, the desire for energetic actions.

6.1 social aspect scale (SF) - towards full-fledged communication, development determines the feature (family, colleagues, loved ones, etc.).

7. Emotional factor scale (RE) - the emotional status of the patient, his reflects the impact of feelings on everyday activities, the attitude towards those around you resumes. whether there are problems in the lsh process and within the usual activities evaluates.

8. Mental health scale (MH) - level of neurotization, depressive determines predisposition to circumstances, feeling happiness, spiritual peace, calmness.

All indicators according to the SF-36 survey are physical and mental decomposes into components. Physical function (PF), physical factor (RP), physical pain (P), general health (GH) - enters the physical component. Viability ability scale (VT), social aspect scale (SF), emotional factor (RE), and mental health scale (MH) - enters the mental component.



In the survey, answers are scored from 0 to 100 points. How many points the large number indicates that the quality of life in the patient is at such a high level and vice versa.

Glycated to assess the state of diabetes compensation hemoglobin (HbAlc) levels were determined. Glycemia amount glucose-oxidase method with the help determined on an empty stomach and 2 hours after meals.

**RESEARCH RESULTS:** Patients in the main group in 2 groups according to the SBK stage separated: 16

patients in Stage 1 and 2 of Group 1 SBK, average age  $54.8\pm0.6$ , average QD duration  $8.7\pm0.9$ , average SBK duration  $5.2\pm0.7$  years. 24 patients in Phase 3 and 4 of Group 2 SBK, with an average age of  $58.5\pm1.0$ , with an average QD duration is  $12.7\pm0.8$ , average SBK duration is  $7.2\pm0.9$  years (Table 2).

All 20 patients who make up control group 2 have SBK 1 and 2- patients at the stage. They have an average age of  $55.8\pm0.9$ , with an average SBK duration of.  $5.9\pm0.8$  years.

#### Table 2.

# Glomerular filtration rate (GFR) and kidney damage chronic in terms of symptoms (proteinuria, albuminuria)

		-			
QD type 2 patients with kidne	y disease com	plications SB	Stage	distribution b	y

SBK stage	number	age	QD duration	SBK duration
1-2	16	54,8±0,6	8,7±0,9	5,2±0,7
3-4	24	58,5±1,0	12,7±0,8	7,2±0,9

In order to determine the level of compensation for carbohydrate metabolism, blood sugar levels and hemoglobin levels glycated were determined. According to this, the average blood sugar content is 6.85±0.12 mmol/l on an empty stomach, 10.54±0.12 mmol/l after 2 hours of meals; the amount of glycated hemoglobin is 7.07±0.12%. The examination included patients with blood sugar levels and glycated hemoglobin indicators close to the state of compensation. The reason is that the decompensation state of QD itself also leads to a deterioration in the quality of life.

١

In all of the controllers, the SF-36 questionnaire was fully completed. According to this, the variation in SBK severity in QD type 2 patients with chronic kidney disease is as follows: Group 1, i.e. QD type 2 patients with stage 1-2 SBK, have decreased PF-15.3%, RP-44.7%, VT-17.7%, SF-9.3%, RE-27.6% compared to those in the control group; Group 2, i.e.3-4 stage SBK 48.9%, P-4.6%, VT-24.2%, SF-16.3%, re-31.4%, MH-4.3% decreased compared to those in the control group; Group 1 patients showed almost no change in P, GH, MH, and Group 2 patients showed almost no change in GH.

Table 3.

## SF-36 survey shows changes in SBK weight levels in QD type 2 patients with chronic kidney disease complications

SF - 36 survey scale	Control group	QD type 2 patients with complications of SBK		
-		SBK 1-2 stage	SBK 3-4 stage	
PF	58,8	49,8	46,4	
RP	61,5	34,0	31,4	
Р	21,75	21,2	20,75	
GH	30,0	30,0	29,8	
VT	61,5	51,0	46,6	
SF	21,5	19,5	18,0	
RE	59,5	43,1	40,8	
МН	62,2	61,6	59,5	

In Group 1 patients, the physical component is 21.5%, the mental component is 14.4% compared to those in the low control group; in Group 2 patients, the physical component is 25.4%, and the mental component is 19.4% compared to those in the low control group



#### World Bulletin of Public Health (WBPH) Available Online at: https://www.scholarexpress.net Volume-21, April 2023 ISSN: 2749-3644

# Table 4. Indicators of physical and mental components according to the SF-36 survey in patients under examination

Components per SF-36	Control group	QD type 2 patients with complications of SBK		
survey		SBK 1-2 stage	SBK 3-4 stage	
Physical component	43,01	33,75	32,09	
Mental component	51,18	43,80	41,23	

#### **CONCLUSION:**

1. Glomerular filtration rate (GFR) and symptoms of kidney damage chronic kidney disease by indications (proteinuria, albuminuria) in QD type 2 patients with complications with SBK stage 1-2 stage 40%

in patients, the 3-4 stage was found in 60% of patients.

2. In patients with Type 2 diabetes mellitus, which is complicated by chronic kidney disease patients with chronic kidney disease who do not have diabetes the relative quality of life rate is 23.5% on the physical component, mental there was a decline of 16.9% by Component.

3. Chronic kidney disease was a complication of diabetes mellitus SBK is more severe in patients than in patients who do not have diabetes performance and quality of life indicators depend on the weight level of SBK.

#### **REFERENCES:**

- 1. Robinson T.W., Freedman B.I. Assessing glycemic control in diabetic patients with severe nephropathy. J. Ren. Nutr. 2013; 23 (3): 199–202.
- ADVANCE Collaborative Group: Patel A., MacMahon S., Chalmers J., Neal B., Billot L., Woodward M. et al. Intensive blood glucose control and vascular outcomes in patients with type 2 diabetes. N. Engl. J. Med. 2008; 358 (24): 2560–72.
- Reidy K., Kang H.M., Hostetter T., Susztak K. Molecular mechanisms of diabetic kidney disease. J. Clin. Invest. 2014; 124 (6): 2333– 40.
- 4. Wada J., Makino H. Inflammation and the pathogenesis of diabetic nephropathy. Clin. Sci. (Lond.). 2013; 124 (3): 139–52.
- Komarova O.V., Kucherenko A.G., Smirnov I.E., Tsygin A.N. The role of disturbances in the processes of apoptosis in the progression of chronic kidney disease in children. Nefrologiya i dializ. 2013: 15 (2): 135–9. (in Russian)

- Komarova O.V., Tsygin A.N., Kucherenko A.G., Smirnov I.E. Cystatin C as a marker of renal function in children with chronic renal failure. Nefrologiya i dializ. 2010: 4: 272–4. (in Russian)
- Kacso I.M., Kacso G. Endothelial cell-selective adhesion molecule in diabetic nephropathy. Eur. J. Clin. Invest. 2012; 42 (11): 1227–34.
- Eleftheriadis T., Antoniadi G., Pissas G., Liakopoulos V., Stefanidis I. The renal endothelium in diabetic nephropathy. Ren. Fail. 2013; 35 (4): 592–9.
- Demirel F., Tepe D., Kara O., Esen I. Microvascular complications in adolescents with type 1 diabetes mellitus. J. Clin. Res. Pediatr. Endocrinol. 2013; 5 (3): 145–9.
- Joshi M.S., Berger P.J., Kaye D.M., Pearson J.T., Bauer J.A., Ritchie R.H. Functional relevance of genetic variations of endothelial nitric oxide synthase and vascular endothelial growth factor in diabetic coronary microvessel dysfunction. Clin. Exp. Pharmacol. Physiol. 2013; 40 (4): 253–61.
- 11. Cherney D.Z., Scholey J.W., Daneman D., Dunger D.B., Dalton R.N., Moineddin R. et al. Urinary markers of renal inflammation in adolescents with Type 1 diabetes mellitus and normoalbuminuria. Diabet. Med. 2012; 29 (10): 1297–302.
- 12. Har R., Scholey J.W., Daneman D., Mahmud F.H., Dekker R., Lai V. et al. The effect of renal hyperfiltration on urinary inflammatory cytokines/chemokines in patients with uncomplicated type 1 diabetes mellitus. Diabetologia. 2013; 56 (5): 1166–73.
- Prunotto M., Ghiggeri G., Bruschi M., Gabbiani G., Lescuyer P., Hocher B. et al. Renal fibrosis and proteomics: current knowledge and still key open questions for proteomic investigation. J. Proteomics. 2011; 74 (10): 1855–70.
- 14. Ponchiardi C., Mauer M., Najafian B. Temporal profile of diabetic nephropathy pathologic



changes. Curr. Diabet. Rep. 2013; 13 (4): 592–9.

- 15. Brenner B.M., Lawler E.V., Mackenzie H.S. The hyperfiltration theory: a paradigm shift in nephrology. Kidney Int. 1996; 49 (6): 1774–7.
- Palatini P. Glomerular hyperfiltration: a marker of early renal damage in pre-diabetes and prehypertension. Nephrol. Dial. Transplant. 2012; 27 (5): 1708–14
- 17. Andreev I.L., Nazarova L.I. Bitter sugar diabetes. Vestnik RAN. 2014; 84 (2): 170–5. (in Russian)
- 18. Satirapoj B. Nephropathy in diabetes. Adv. Exp. Med. Biol. 2012; 771: 107–22.
- Shestakova M.V., Chugunova L.A., Shamkhalova M.Sh., Dedov I.I. Diabetic nephropathy: advances in diagnosis, prevention, treatment. Sakharnyy diabet. 2005; 3: 22–4. (in Russian)
- Bondar' I. A., Klimontov V.V. Early markers of diabetic nephropathy. Klinicheskaya nefrologiya. 2010; 2: 6–65. (in Russian)
- Otu H.H., Can H., Spentzos D., Nelson R.G., Hanson R.L., Looker H.C. Prediction of diabetic nephropathy using urine proteomic profiling 10 years prior to development of nephropathy. Diabet. Care. 2007; 30 (3): 638– 43.
- 22. Lebedeva N.O., Vikulova O.K. Pre-clinical markers for diagnosis of diabetic nephropathy in patients with diabetes mellitus type 1. Sacharnyy diabet. 2012; 2: 38–45 (in Russian)
- Gu H.F., Brismar K. Genetic association studies in diabetic nephropathy. Curr. Diabet. Rev. 2012; 8 (5): 336–44.
- 24. Araki S. APOE polymorphism and diabetic nephropathy. Clin. Exp. Nephrol. 2014; 18 (2): 230–3.
- Rizvi S., Raza S.T., Mahdi F. Association of genetic variants with diabetic nephropathy. World J. Diabet. 2014; 5(6): 809–16.
- 26. Arora M.K., Singh U.K. Molecular mechanisms in the pathogenesis of diabetic nephropathy: an update. Vasc. Pharmacol. 2013; 58 (4): 259–71.
- 27. Dronavalli S., Duka I., Bakris G.L. The pathogenesis of diabetic nephropathy. Nature Clin. Pract. Endocrinol. Metab. 2008; 4 (8): 444–52.