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# Residual Kidney Function in Hemodialyzed Patients and Related Factors

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**ABSTRACT** 

**Objective:** Residual kidney function (RKF) is a strong marker of the remaining capacity of the kidneys in patients with end-stage renal disease (ESRD). The fact that RKF declines in the first year of hemodialysis (HD) has drawn more attention recently. The aim of this study was to determine and analyze the current RKF level and related factors in patients undergoing HD treatment.

**Materials and Methods:** The study was performed at Kars State Hospital with 73 HD patients. Residual renal urea clearance (KRU) was measured to determine RKF. Patient urine volume was determined during the period between the end of the first HD session of the week and the beginning of the next HD session. The patients were classified into 3 groups according to KRU level: <1, 1-2, and >2 mL/minute. The duration of HD treatment, biodemographic characteristics, biochemical and hormonal analyses, HD adequacy, ESRD etiology, and co-morbidities were compared between groups. A p value of less than 0.05 was considered statistically significant (confidence interval: 95%).

**Results:** There was a statistically significant difference between the KRU value and the duration of HD treatment (p<0.001), ESRD etiology (p=0.037), serum potassium level (p=0.028), phosphorus level (p=0.036), urine volume (p<0.001), ultrafiltration (UF) volume (p=0.002), and body mass index (p=0.002). Patients with a urine volume of <100 mL/day had a longer duration of HD treatment of  $6.9\pm4.2$  years (p=0.021), as well as a greater quantity of UF administered  $723\pm230$  mL/hour (p<0.001).

**Conclusion:** Rather than provide an explanation of a cause-effect relationship for RKF loss, the findings of this study may contribute to the monthly interpretation and regulation of HD therapy. Additional studies that include RKF measurement at certain time intervals and long-term observation are needed.

Keywords: Hemodialysis, renal insufficiency, residual kidney function, urine

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## **INTRODUCTION**

Residual kidney function (RKF) is an indicator of the remaining functional capacity of the kidneys, which provide the excretion of uremic toxins and water, in dialysis patients. Generally, hemodialysis (HD) treatment is initiated with 3 sessions a week in HD units without considering RKF, and RKF is known to decrease in the first year of dialysis. Hypotension episodes in HD sessions cause a reduction in RKF, requiring volume replacement and severely threatening the preservation of RKF, which results in loss of this function (1–4).

RKF provides protection from secondary events, such as left ventricular hypertrophy and uncontrolled hypertension caused by volume load, improves inflammatory and metabolic parameters, and results in the need for less erythropoietin (EPO) (1, 5, 6). Small solute clearance from peritoneal and renal clearance predicts mortality in peritoneal dialysis patients in the later stages (3). RKF can be affected by age, gender, etiological cause, body mass index (BMI), catheter infections, dialysis membranes, drugs, and cardiovascular events (7). In previous studies, it has been demonstrated that the mortality risk was 36% less in patients with a daily urine volume of up to 250 mL, and the mortality rate was lower in patients with a urine volume greater than 100 mL in a 2-year follow-up period (8, 9). The small solute clearance and the excretion of middle molecular-weight toxins ensure the control of fluid and phosphorus levels, as well as good quality of life and less dietary restriction (10, 11). Therefore, RKF protection is an important parameter in the treatment of HD patients. In the first months of HD, those with RKF still present should be followed up at 2-month intervals on average, and in the ongoing process, the urine volume and the residual renal urea clearance (KRU) should be measured twice a year until the urine volume decreases to 100 mL per day and the KRU below 2 mL/minute (10). Since the hemodynamics of these patients are not stable and urine sample collection must be performed in the time period between 2 HD sessions, it is difficult and tedious for the patients, resulting in only a small portion of successful RKF measurement collections (8, 12). Therefore, studies have explored and recommended analysis of the C-terminal agrin fragment, β2-microglobulin, cystatin C, and recently, serum bicarbonate and p-cresyl sulfate (1, 7).

The aim of this study was to determine the relationship between the RKF of HD patients and the duration of HD treatment, as well as demographic parameters, such as age and sex, ESRD causes, presence of diabetes mellitus, parameters of dialysis adequacy, and biochemical and hormonal parameters.

#### MATERIALS and METHODS

This cross-sectional study was performed using the biodemographic characteristics, as well as blood and urine analysis of 104 patients who were undergoing HD treatment 3 times a week for 4 hours using a dialyzer with 1.5-2.0 m<sup>2</sup> surface area and 300-400 mL/minute blood and 500-600 mL/minute dialysate flow rates in the Kars State Hospital HD unit. Biocompatible dialysis membrane and ultrapure dialysate were used in all patients. Hormonal analysis was performed with a Beckman Coulter UniCel DXI 600 autoanalyzer (Beckman Coulter, Inc., Brea, CA, USA), biochemical analysis with a Roche Cobas C501 spectrophotometer (Roche Diagnostics, Risch-Rotkreuz, Switzerland), and blood count with a Horiba ABX Pentra 120 autoanalyzer (Horiba, Ltd., Kyoto, Japan). Blood samples were taken following a session when the blood flow rate was reduced to 100 mL/minute for 15 seconds, as recommended by the National Kidney Foundation Kidney Disease Outcomes Quality Initiative, and at the beginning of a session, before heparin and saline administration (13). The KRU was calculated to determine the residual renal function of the patients. Urine was collected for this purpose in the 44-hour period between the end of the first HD session of the week and the beginning of the next HD session (12).

## **Patient Instructions for Urine Collection**

The patients were asked to collect their total urine output from the period between the end of the first HD session of the week and the beginning of the next session in a 0.5- or 1.0-liter container and to bring it to the HD session. If the patient had a relatively high volume of urine output, they were asked to use the same containers, to urinate before going out for their daily social activities, and to be able to return home quickly in order to perform the collection. The patients were also told to keep their urine containers in a cool place that would not be exposed to direct light.

### **Exclusion Criteria**

A total of 31 patients were excluded from the study: those who did not want to collect urine (n=5), those using diuretics (n=3), those who had only a few drops of urine per day (n=6), those with acute gastroenteritis or fluid loss (n=3), those who had a urinary infection or chronic prostatitis-related infection (n=2), those with a diabetic foot infection (n=1), those who had pulmonary infection (n=3), those who didn't collect urine according to the instructions (n=5), and those who had an assessment of dry weight in the previous month (n=3). The KRU of 73 patients who collected urine was calculated in order to determine the RKF.

Residual renal urea clearance was calculated as follows: [KRU (mL/min)] = urinary urea (mg/dL)  $\times$  urinary volume (mL) / collection time (min)  $\times$  [0.9  $\times$  serum urea (mg/dL)] (10).

Kt/V calculations and the urea reduction rate (URR) were used to determine HD adequacy, (13).

Single-pool (sp Kt/V) was calculated as: -Ln (R-0.008  $\times$  t) + (4-

 $3.5 \times R$ )  $\times$  UF / W (14). R is the ratio of postdialysis to predialysis blood urea nitrogen (BUN), t is the time on HD in hours, UF is the quantity administered in liters, and W is the post-dialysis body weight in kilograms.

URR was calculated as follows: URR =  $100 \times (1 - postdialysis BUN)$  / predialysis BUN) (13).

BMI için hastanın HD sonrası hafif giysiler ile ayakkabılarını giymeden boy ve kilosu belirlenerek şu formül ile hesaplandı:

BMI = The weight (kg) divided by the square of the height (m) (15).

The KRU levels were divided into 3 groups: <1, 1–2, and >2 mL/minute. These groups were compared in terms of the duration of HD treatment; biodemographic characteristics, such as age and sex of the patient; biochemical and hormonal analyses; dialysis adequacy parameters; cause of ESRD; and co-morbidities. The results were examined to determine any relationship to the RKF.

#### **Statistical Analysis**

IBM SPSS Statistics for Windows, Version 21.0 (IBM Corp., Armonk, NY, USA) was used to perform a one-way analysis of variance when the sample quantity was sufficient and there was homogeneous distribution of the variances. The Kruskal-Wallis test was applied when these conditions were not met. A chi-square test and post-hoc analysis were used in the binary comparison of categorical variables. Relation with between UF administered to the patients, HD duration and KRU was evaluated by pearson correlation analysis.

This research was conducted with the approval of the Clinical Research Ethics Committee of Kafkas University Faculty of Medicine.

#### **RESULTS**

A total of 104 patients were undergoing HD treatment and 73 were included in the analysis after applying the exclusion criteria. There were 27 female patients with a mean age of 64.3 years (range: 24–88 years) and 46 male patients with a mean age of 61.5 years (range: 20–88 years).

As shown in Table 1, there was a statistically significant difference between the KRU and the duration of HD (p<0.001), BMI (p=0.002), urine volume (p<0.001), UF (p=0.002), potassium level (p=0.028), and phosphorus level (p=0.036).

When the KRU was analyzed according to the cause of ESRD, as seen in Table 2, the mean KRU level of patients with diabetic nephropathy was higher than that of patients with other causes of kidney disease (p=0.037).

As illustrated in Table 3, there was no statistically significant difference between the patients with and without co-morbidities according to KRU value (p=0.461).

Table 4 demonstrates that there was a statistically significant difference between the duration of HD treatment and UF volume when compared according to the 24-hour urine level of the patients (p=0.021, p<0.001, respectively).

Analysis of the relationship between vascular access and KRU is shown in Table 5; no statistically significant difference was found (p=0.231).

Table 1. Comparison of residual renal urea clearance with independent variables<sup>a</sup>

Independent variable			Depende	ent variable: KRU (	variable: KRU (mL/min)		
	Mean	MinMax.	KRU >2 Number: 14	KRU: 1-2 Number: 10	KRU <1 Number: 49		
Female, age (years), number: 27	64.3	24–88	60.0	67.5	64.1	0.055	
Male, age (years), number: 46	61.5	20-88	67.6	65.3	58.1	0.897	
Duration on HD (years)	5.8	0.5-18.0	5.1	2.4*	6.7*	< 0.001	
Systolic blood pressure (mm/Hg)	132	90-170	127	140	132	0.086	
Diastolic blood pressure (mm/Hg)	77	50-90	77	74	78	0.300	
Body mass index (kg/m²)	26.2	16.1-39.8	27.9	30.4*	24.9*	0.002	
Urine volume/(mL/in a day)	177.0	0.0-1505.5	641.2*	196.3*	40.5*	< 0.001	
Ultrafiltration (mL/ hour)	625	200-1250	435*	545	696*	0.002	
Hemoglobine (g/dL)	10.4	6.3-13.2	10.0	10.9	10.4	0.402	
C-reactive protein (mg/dL)	1.2	0.0-5.9	1.3	1.6	1.1	0.742	
Albumin (g/dL)	3.7	2.8-4.4	3.6	3.7	3.8	0.582	
Parathyroid hormone (pg/mL)	543.7	12.0-2576.0	375.2	491.4	602.5	0.411	
Ferritin (ng/mL)	496.4	14.4-2001.0	547.3	743.6	431.4	0.359	
Kt/V	1.69	1.30-2.50	1.56	1.55	1.76	0.055	
URR (%)	75.6	62.6-87.5	73.8	73.1	76.6	0.080	
Potassium (mEq/L)	4.8	3.2-6.8	4.4*	4.8	4.9*	0.028	
Calcium (mg/dL)	8.3	4.4-10.0	8.2	8.6	8.2	0.228	
Phosphor (mg/dL)	4.8	2.1-7.8	4.2	5.5*	4.8*	0.036	

ANOVA was used. KRU: Residual renal urea clearance; Min.: Minimum; Max.: Maximum; HD: Hemodialysis; \*Means that diferences between KRU levels with using post hoc analysis (Tamhane's T2); URR: Urea reduction ratio

A scatter plot of the correlation analysis of KRU and HD duration is displayed in Figure 1 (r=-0.239; p=0.042). Figure 2 shows the scatter plot of the correlation analysis between UF administered to the patients and KRU (r=-0.446; p<0.001).

#### **CONCLUSION**

The protection of RKF is valuable in predicting the prognosis of HD patients due to the positive effect it has on both survival and morbidity. Factors such as age, sex, obesity, the cause of ESRD, type of HD, dialyzer membrane properties, catheter-related or other infections, and cardiovascular events can affect RKF (7). Therefore, RKF should be considered in the treatment of ESRD and HD patients (13). Our research revealed a relationship between KRU and HD duration, BMI, urine volume, UF, potassium and phosphorus levels, and the etiology of ESRD.

The duration of HD treatment in patients with a KRU <1 mL/minute was 5.8 years ( $0.5\text{--}18\pm6.7$ ), which was higher than those with KRU >1 mL/minute (Table 1; p<0.001). In the correlation analysis, the decrease in KRU over time was moderate and was continuing in a negative direction (Fig. 1; r=-.446, R²=0.019). Urine volume accompanied the decrease in KRU (Table 4; p=0.021). Similarly, in previous studies, the decrease in KRU has been shown to continue in both HD and peritoneal dialysis patients beginning months before dialysis and continuing after dialysis treatment (1, 10). A decrease in RKF has been shown to be strongly

Table 2. Analysis of end-stage renal disease and residual urea clearance<sup>a</sup>

Etiology of ESRD	Number	Dependent variable: KRU (mL/min)				
		Mean±SD	Significant difference**	р		
HT	29	0.691±1.44	-			
DM	21	2.111±2.01	Unknown			
CD	4	0.697±0.69	-	0.025		
Others*	10	1.404±2.04	-	0.025		
Unknown	9	0.416±0.56	DM			
Total	73	1.164±1.72				

One Way ANOVA test was used. ESRD: End-stage renal disease; KRU: Residual renal urea clearance; SD: Standard deviation; HT: Hypertension; DM: Diabetes mellitus; CD: Cystic disease; \*Others: Chronic glomerulonephritis (4), analgesic nephropathy (1), nephrolithiasis (4), vesico-ureteral rephlux (1). \*\*Tamhane's T2 was used for significant differences because of equal variances not assumed

associated with all causes of mortality in the first year of HD treatment (16). In studies conducted at different times, RKF decreased by 0.18–0.33/mL/minute every month. This trend is slower in peritoneal dialysis and hemodiafiltration therapies (4). Exposure to hypovolemia over time as a result of changing volume status is one of the major causes of loss of RKF (4).

Table 3. Comparison of co-morbidity, erythropoietin use, and vitamin D treatment with residual urea clearance

Indeependent variables		Dependent variable: KRU (mL/min)							
	KR	KRU >2 KRU: 1–2		U: 1–2	KRU <1		Total		
	n	%	n	%	n	%	n	%	
Comorbidity									
Yes	10	20.8	8	16.7	30	62.5	48	66	0.461
No	4	16.0	2	8.0	19	76.0	25	34	
Erithropoetin treatments <sup>a</sup>									
Yes	6	15.4	6	15.4	27	69.2	39	53	0.651
No	8	23.5	4	11.8	22	64.7	34	47	
Vitamin D treatment <sup>b</sup>									
Yes	8	22.2	4	11.1	24	66.7	36	49	0.707
No	6	16.2	6	16.2	25	67.6	37	51	
Total							73	100	

Chi-square test was used. KRU: Residual renal urea clearance; Comorbidity: Heart failure (5), coroner artery disease (14), diabetes mellitus (21), chronic obstructive pulmonary disease (4), hypothyroidism (3), epileptic disease (1), patients have two or more than two diseases (15), "Epoetin-alpha, beta or darbepoietin; "Active vitamin –D or calsimimetic or their combinations

Table 4. Analysis of hemodialysis treatment duration and ultrafiltration volume according to urine volume\*

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	A <100 mL Number: 39	B 100-199 mL Number: 15	C 200–499 mL Number: 15	D ≥500 mL Number: 4			
Duration on HD (years) <sup>a</sup>	6.9±4.2	4.6±4.6	3.7±1.4	8.5±5.1	0.021		
Ultrafiltration (UF) (hour) <sup>b</sup>	723±230	498±186	598±229	250±0.0	< 0.001		

ANOVA tests was used. HD: Hemodialysis; UF: Ultrafiltration;  ${}^{\circ}$ Means that differences between group A and C;  ${}^{\circ}$ Means that differences between group A and D, B and D, C and D with post hoc analysis (Tamhane's T2)

**Table 5.** Residual renal urea clearance analysis according to vascular access\*

Dependent variable: KRU (mL/dk)							
Vasculer access	n	%	$X^2$	Mean rank	p		
AVF	56	77	2.9	36.5	0.231		
CVC (cuffed)	15	20		41.4			
Greft (synthetic)**	2	3		15.5			

\*Kruskall-vallis test was used. KRU: Residual renal urea clearance; AVF: Arteriovenous fistulae; CVC: Central venous catheter; \*\*Grafting was performed in two patients due to AVF loss

In this study, mild excess weight and obesity were related to KRU. When the relationship between BMI and KRU was analyzed, BMI was  $24.9~{\rm kg/m^2}$  in patients with a KRU <1 mL/minute, while it was  $30.4~{\rm kg/m^2}$  in patients with a KRU of  $1-2~{\rm mL/minute}$ , and the difference was statistically significant (Table 1; p=0.002). There are contrasting results from previous studies on the relationship between BMI and RKF. Drechsler et al. (17) suggested in a multi-center cohort study that obesity was a strong risk factor for

decrease in RKF. However, it has also been suggested that obesity can be protective in terms of survival, which is contradictory (18).

Intradialytic hypotension in HD is a threat to RKF. RKF and urine volume decrease as a result of reduction in volume load in instances of hypertension or left ventricular hypertrophy. If appropriate, the use of furosemide in patients with sufficient urine output may protect urine volume and decrease the interdialytic volume load (4, 19, 20). In this study, the mean 24-hour urine volume was 40.5 mL in patients with a KRU of <1 mL/minute and 641.2 mL in patients with a KRU of >2 mL/minute (Table 1; p<0.001). Another study has previously demonstrated that when the RKF was protected, the urine volume load decreased in the interdialytic period, hypertension was reduced, and the left ventricular volume load decreased (7).

Hypovolemia is closely related to RKF and hypotension (4, 20). Evaluation of UF volume and KRU has revealed that the mean UF volume in patients with a KRU <1 mL/minute was 696 mL/hour, while in patients with >2 mL/minute, it was 435 mL/hour (Table 1; p<0.002). Similarly, a moderate correlation has been observed in the reverse direction in correlation analysis between the KRU and the need for UF (Fig. 2; r=-.446,  $R^2$ =0.199; p<0.001). When urine volume was divided into 4 groups and UF volume was ana-

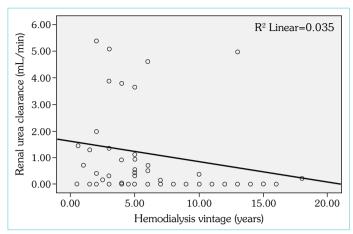


Figure 1. Scatter plot of Pearson's correlation analysis of residual urea clearance reduction and duration of hemodialysis (r=-.239,  $R^2$ =0.035, p=0.042, level of significance in correlation=0.05, number of patients=73)

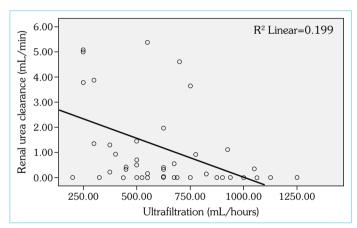


Figure 2. Scatter plot of the correlation analysis between ultrafiltration volume and residual urea clearance (r=-.446,  $R^2=0.199$ , p<0.001, level of significance in correlation=0.01, number of patients=73)

lyzed, the hourly UF requirement was only 250 mL for those with a urine output of 500 mL in 24 hours (Table 4; p<0.001). This level of urine volume seems to be important to the protection of the RFK by reducing the risk of intradialytic hypotension (4, 17, 20). Shemin et al. (9) found a lower mortality rate in patients with a urine volume >100 mL at 2 years of follow-up.

In our study, the phosphorus and potassium levels of patients with a KRU of >2 mL/minute were lower: respectively, they were 4.2 mg/dL and 4.4 mEq/L (Table 1; p=0.036; p=0.028). Preservation of RKF is important for the removal of middle-molecular-weight toxins, control of fluid and phosphorus levels, as well as for better quality of life, a more easily tolerated diet, and protection from the vascular calcification associated with renal osteodystrophy  $(1,\,2,\,10,\,11,\,21,\,22)$ .

In the analysis of the relationship between ESRD and RKF, patients with diabetic nephropathy had a KRU of 2.1 mL/minute, which was higher than that of any other cause (p=0.025). However, the results of previous studies are different. lest et al. (23) reported a more rapid decrease in RKF in cases of diabetic nephropathy, and

Haynes et al. (24) found the annual decrease in RKF to be 3.8 and 2.5 mL/minute in patients with cystic kidney disease and diabetic nephropathy, while glomerulonephritis patients had the lowest decrease at 1.9 mL/minute.

In the present study, 56 patients had an arteriovenous access site, 15 used a cuffed catheter, and a graft (synthetic) was used in 2. There was no significant difference between the KRU level and the presence of an HD access path (Table 5; p=0.231). However, infections caused by bacterial colonization in tunneled central venous catheters have a negative effect on RKF (25).

The presence of a co-morbidity, the administration pf EPO, and the activated vitamin D level demonstrated no significant difference according to KRU (Table 3; p>0.05). However, co-morbidities such as cardiovascular disease, congestive heart failure, or obesity have previously been shown to lead to a decrease in RKF in those who previously had HD treatment (1, 8, 18). Vitamin D is widely administered to HD patients. However, it is not yet known whether or not it prevents the loss of RKF (18). In patients with an RKF of  $\geq 1$  mL/minute, a low dose of EPO is suggested (1, 6, 8).

RKF is often not measured in clinical practice. Strengths of this study include the determination of urine volume in the time period between 2 HD sessions and the calculation of the KRU according to this value, as well as analysis of factors that may be related. The relatively small number of patients and the lack of a cohort limit the study results. Therefore, there is still a need for large-scale studies that include follow-up of RKF at frequent intervals beginning at the time of HD onset and the observation of factors that may affect RKF.

Ethics Committee Approval: This research was conducted with the approval of the Clinical Research Ethics Committee of Kafkas University Faculty of Medicine (Date: 03.04.2019, Number: 80576354-050-99/79).

**Informed Consent:** Written informed consent was obtained from patients who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – HİE, FK; Design – HİE, FK; Supervision – HİE, FK; Resource – HİE, FK; Materials – HİE, FK; Data Collection and/or Processing – HİE, FK; Analysis and/or Interpretation – HİE; Literature Search – HİE; Writing – HİE; Critical Reviews – HİE, FK.

Conflict of Interest: The authors have no conflict of interest to declare.

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#### **REFERENCES**

- Hur I, Lee YK, Kalantar-Zadeh K, Obi Y. Individualized Hemodialysis Treatment: A Perspective on Residual Kidney Function and Precision Medicine in Nephrology. Cardiorenal Med 2019; 9(2): 69–82. [CrossRef]
- Lowenstein J, Grantham JJ. Residual renal function: a paradigm shift. Kidney Int 2017; 91(3): 561–5. [CrossRef]
- 3. [No authors listed] Adequacy of dialysis and nutrition in continuous peritoneal dialysis: association with clinical outcomes. Canada-USA (CANUSA) Peritoneal Dialysis Study Group. J Am Soc Nephrol 1996; 7(2): 198–207.
- Krediet RT. Preservation of Residual Kidney Function and Urine Volume in Patients on Dialysis. Clin J Am Soc Nephrol 2017; 12(3):

- 377-9. [CrossRef]
- Daugirdas JT, Greene T, Rocco MV, Kaysen GA, Depner TA, Levin NW, et al. Effect of frequent hemodialysis on residual kidney function. Kidney Int 2013; 83(5): 949–58. [CrossRef]
- Vilar E, Wellsted D, Chandna SM, Greenwood RN, Farrington K. Residual renal function improves outcome in incremental haemodialysis despite reduced dialysis dose. Nephrol Dial Transplant 2009; 24(8): 2502–10. [CrossRef]
- Liu X, Dai C. Advances in Understanding and Management of Residual Renal Function in Patients with Chronic Kidney Disease. Kidney Dis (Basel) 2017; 2(4): 187–96. [CrossRef]
- Shafi T, Jaar BG, Plantinga LC, Fink NE, Sadler JH, Parekh RS, et al. Association of residual urine output with mortality, quality of life, and inflammation in incident hemodialysis patients: the Choices for Healthy Outcomes in Caring for End-Stage Renal Disease (CHOICE) Study. Am J Kidney Dis 2010; 56(2): 348–58. [CrossRef]
- Shemin D, Bostom AG, Laliberty P, Dworkin LD. Residual renal function and mortality risk in hemodialysis patients. Am J Kidney Dis 2001; 38(1): 85–90. [CrossRef]
- Mathew AT, Fishbane S, Obi Y, Kalantar-Zadeh K. Preservation of residual kidney function in hemodialysis patients: reviving an old concept. Kidney Int 2016; 90(2): 262–71. [CrossRef]
- van der Wal WM, Noordzij M, Dekker FW, Boeschoten EW, Krediet RT, Korevaar JC, et al. Full loss of residual renal function causes higher mortality in dialysis patients; findings from a marginal structural model. Nephrol Dial Transplant 2011; 26(9): 2978–83. [CrossRef]
- Kjaergaard KD, Jensen JD, Peters CD, Jespersen B. Preserving residual renal function in dialysis patients: an update on evidence to assist clinical decision making. NDT Plus 2011; 4(4): 225–30. [CrossRef]
- National Kidney Foundation. KDOQI Clinical Practice Guideline for Hemodialysis Adequacy: 2015 update. Am J Kidney Dis 2015; 66(5): 884–930. [CrossRef]
- Daugirdas JT. Second generation logarithmic estimates of single-pool variable volume Kt/V: an analysis of error. J Am Soc Nephrol 1993; 4(5): 1205–13. [CrossRef]
- 15. Misra A, Dhurandhar NV. Current formula for calculating body mass

- index is applicable to Asian populations. Nutr Diabetes 2019; 9(1): 3.
- Obi Y, Rhee CM, Mathew AT, Shah G, Streja E, Brunelli SM, et al. Residual Kidney Function Decline and Mortality in Incident Hemodialvsis Patients. J Am Soc Nephrol 2016; 27(12): 3758–68, [CrossRef]
- Drechsler C, de Mutsert R, Grootendorst DC, Boeschoten EW, Krediet RT, le Cessie S, et al. Association of body mass index with decline in residual kidney function after initiation of dialysis. Am J Kidney Dis 2009; 53(6): 1014–23. [CrossRef]
- Patel N, Hu SL. Preserving residual renal function in dialysis: what we know. Semin Dial 2015; 28(3): 250–8. [CrossRef]
- Hiramatsu T, Hobo A, Hayasaki T, Kabu K, Furuta S. A Pilot Study Examining the Effects of Tolvaptan on Residual Renal Function in Peritoneal Dialysis for Diabetics. Perit Dial Int 2015; 35(5): 552–8. [CrossRef]
- Sjolund J, Garcia Anton D, Bayes LY, Hoekstra T, Dekker FW, Munoz Mendoza J. Diuretics, Limited Ultrafiltration, and Residual Renal Function in Incident Hemodialysis Patients: A Case Series. Semin Dial 2016; 29(5): 410–5. [CrossRef]
- 21. Penne EL, van der Weerd NC, Grooteman MP, Mazairac AH, van den Dorpel MA, Nubé MJ, et al. Role of residual renal function in phosphate control and anemia management in chronic hemodialysis patients. Clin J Am Soc Nephrol 2011; 6(2): 281–9. [CrossRef]
- Kalantar-Zadeh K, Unruh M, Zager PG, Kovesdy CP, Bargman JM, Chen J, et al. Twice-weekly and incremental hemodialysis treatment for initiation of kidney replacement therapy. Am J Kidney Dis 2014; 64(2): 181–6. [CrossRef]
- lest CG, Vanholder RC, Ringoir SM. Loss of residual renal function in patients on regular haemodialysis. Int J Artif Organs 1989; 12(3): 159–64. [CrossRef]
- 24. Haynes R, Staplin N, Emberson J, Herrington WG, Tomson C, Agodoa L, et al. Evaluating the contribution of the cause of kidney disease to prognosis in CKD: results from the Study of Heart and Renal Protection (SHARP). Am J Kidney Dis 2014; 64(1): 40–8. [CrossRef]
- Kang JS, Jang HR, Lee JE, Park YJ, Rhee H, Seong EY, et al. The bacterial colonization in tunneled cuffed dialysis catheter and its effects on residual renal function in incident hemodialysis patients. Clin Exp Nephrol 2016; 20(2): 294–301. [CrossRef]