Original Article
İnternal Medicine
DOI: 10.56016/dahudermj.1357040

DAHUDER
Medical Journal
e-ISSN: 2791-9250

The association between serum perilipin-2 and kidney disease progression of patients with autosomal dominant polycystic kidney disease

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ABSTRACT

Objectives: We aimed to evaluate the relationship between serum perilipin-2 / adipophilin (PLIN-2 / ADRP) levels and clinical course in patients with Autosomal Dominant Polycystic Kidney Disease (ADPKD).

Methods: 80 ADPKD patients with Chronic Kidney Disease (CKD) G1-G4 status, among the patients who were regularly followed up in the nephrology outpatient clinic between 2012 and 2019, were included in the study. CKD-G5 patients were excluded from the study. Baseline PLIN-2/ADRP levels were measured. Patients were divided into 2 groups according to the median serum PLIN-2/ADRP level. During the follow-up period, data such as blood pressure, height-adjusted total kidney volume (HtTKV), proteinuria, complete blood count, and biochemical tests were recorded.

Results: In the patients with serum PLIN-2 / ADRP level above the median value (11.675 ng / mL), BMI was significantly higher than the other group ($p<0.001$). The female sex ratio was found to be significantly increased in patients with serum PLIN-2 /ADRP levels above the median value. Serum PLIN-2/ADRP levels were found to increase as eGFR decreased in ADPKD patients, but it was not statistically significant. In patients with high baseline mean PLIN-2/ADRP levels, the mean eGFR decline was found to be 20 ml/min/1.73 m^2. However, the mean eGFR decrease in the other group with a low baseline PLIN-2/ADRP value was found to be 16 ml/min/1.73 m^2 after 7 years of follow-up.

Conclusion: PLIN-2 / ADRP levels increased in female ADPKD patients and it is positively associated with BMI increase. Increased serum PLIN-2 / ADRP levels may be a harbinger of faster kidney function decline.

Keywords: Perilipin-2, Autosomal Dominant Polycystic Kidney Disease, female sex, body mass index

Received: *eptember 8, 2023****; Accepted:*** *January 3, 2024****; Published Online:*** *January 29, 2024*

How to cite this article: *Cetin M, Eroglu E, Karakukcu C, Zararsiz G, Cirak Gursoy A, Kocyigit I. The association between serum perilipin-2 and autosomal dominant polycystic kidney disease.DAHUDER MJ 2024,4(1):7-16. DOI: 10.56016/dahudermj.1357040*

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Autosomal Dominant Polycystic Kidney Disease (ADPKD) is the most common hereditary disease of the kidney that progresses to kidney failure (KF). Genetic causes of ADPKD in the mutations in the PKD1 gene on chromosome 16 and the PKD2 gene on chromosome 4 in the PKD2 genes are formed.[1] The most important complications in ADPKD patients are KF and cardiovascular diseases .[2] ADPKD can be considered as a multisystemic disease in terms of concomitant cystic (such as hepatic, pancreatic) and non-cystic (such as intracranial aneurysm and heart valve disorders) involvement in other organs rather than a kidney disease alone.[3]

Treatment options to slow the progression of the disease in ADPKD are limited. Especially in recent years, positive results have been obtained in preventing the decrease in the estimated glomerular filtration rate (eGFR) with the use of V2 receptor antagonists. However, significant inflammatory and fibrotic changes occur in the renal tubular epithelium before eGFR decline. There are ongoing numerous studies on the role of cell proliferation, apoptosis, fluid secretion, and extracellular matrix in the course of this progressive disease.[4] Recently, studies on ADPKD have been increasing on biomarkers, cytokines, receptors, and molecules that can predict disease progression.[5]

Perilipines (PLINs) are part of the protein group that reacts with the intracellular neutral lipid droplets. PLINs are located on the surface of oil droplets. They highly phosphorylate adipocyte proteins. PLIN is phosphorylated after cyclic adenosine monophosphate (cAMP) activates protein kinase A (PKA). PLIN moves away from the surface of the lipid droplet. It allows hormone-sensitive lipase (HSL) to hydrolyze the triglyceride (triacylglycerol [TAG]) nucleus. The most well-defined and most important droplet proteins are PLINs. There are 5 types currently known: PLIN-1-5.[6]

PLIN-2 / adipophilin (also known as adipocyte differentiation-related protein [ADRP]) is commonly expressed in adipogenic cells in tissues such as the lung, steatotic liver, adrenal cortex, and testis. PLIN-2 / ADRP is associated with droplets and is destroyed in the absence of neutral lipids. Its expression is connected to the number of neutral lipids in the cell and overexpression of PLIN-2 / ADRP occurs in increased droplet formation.[7,8] Diseases associated with lipid droplet (LD) accumulation are on the rise. For this reason, researchers concentrated on studies on PLINs. PLIN-2 / ADRP has been studied as a biomarker in some types of diseases including atherosclerosis and kidney cancer.[9-12]

In recent years, there has been growing interest in the potential role of PLIN-2 / ADRP in kidney disease. This biomarker has been shown to be linked to kidney cancer and atherosclerosis. Studies have shown that PLIN-2/ADRP expression is increased in various renal conditions, such as acute kidney injury (AKI), chronic kidney disease (CKD), and diabetic nephropathy. This increase in PLIN-2 / ADRP expression is thought to be due to several factors, including oxidative stress, inflammation, and metabolic dysregulation. Therefore PLIN-2 / ADRP expression can increase the production of reactive oxygen species (ROS) that can damage kidney cells. The expression of PLIN-2 / ADRP can also block the activity of PPARα, a transcription factor that helps to protect kidney cells from damage. In addition, PLIN-2 / ADRP expression can increase protein secretion from kidney cells, which can lead to proteinuria. Taken together, these findings suggest that PLIN-2 / ADRP is a potential target for the development of new treatments for kidney disease.[13] In addition to being accompanied by vascular dysfunction and renal cystic proliferation, metabolic abnormalities are also evident in patients with ADPKD.[14] Currently, an inexpensive, widely applicable biomarker that can predict the progression to KF in patients with ADPKD has not been fully found. Therefore, we aimed to investigate the relationship between PLIN-2 / ADRP, and ADPKD, in terms of metabolic changes, kidney volume, and kidney disease progression.

METHODS

Patients and study design

We classified ADPKD patients who were consistently monitored at the nephrology outpatient clinic from 2012 to 2019 according to their CKD status, using the estimated glomerular filtration rate (eGFR). 80 ADPKD patients with CKD G1-G4 status were included. CKD G5 patients were excluded from the study. 15 PLIN-2 / ADRP levels were measured from blood stored in a -80 ° C cabinet obtained from the patients at baseline (sera of the patients in 2012). Patients were divided into 2 groups according to the median serum PLIN-2/ADRP level. All patients' data including blood pressure, height, weight measurements, height-adjusted total kidney volume (HtTKV), 24-h urine proteinuria, complete blood count, eGFR, CRP, lipid profile, albumin, uric acid, calcium, phosphorus, proteinuria were recorded during a routine control of

the patients. After all data were obtained, ADPKD patient groups were examined as CKD G1-G4 according to the status of CKD. Rapid progression of ADPKD was defined as an eGFR decrease of ≥ 5 ml/min/1.73m2 yr at 1 year or ≥2.5 ml/min/1.73m2 per yr over 5 yr or Mayo image class 1C, 1D, or 1E .16 An informed consent form was obtained from all patients included in the study. The Ethics Committee of XXXX University Clinical Research granted approval for this study by decision dated November 07, 2018, and number 2018/561.

Statistical analysis

Data normality and variance homogeneity were evaluated using histograms and q-q plot graphs and Shapiro-Wilk's test. Levene's test was used to test variance homogeneity. For continuous variables in between groups, the two-way t-test and Mann-Whitney U test were used. For categorical variables, Pearson's chi-squared test and Fisher's exact test were used. To compare the means between variables with more than two groups, we used one-way ANOVA and Kruskal-Wallis test for continuous variables, and Pearson's chi-squared test, and Fisher's exact test for categorical variables. Pairwise comparisons between groups were performed using Tukey's test, Tamhane's T2 test, Siegel-Castellan test, and Bonferroni-corrected z test. To compare the means between dependent groups, a t-test was used for dependent samples. Receiver operating characteristic (ROC) analysis was used to determine the cut-off point for the PLIN-2 / ADRP variable to assess its relationship with disease progression. The Youden index was used to find the cut-off point. The analyses were performed using R 3.2.0 (www.r-project.org) and TURCOSA Analytical statistical software programs. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Forty-three patients (54%) were female and 37 (46%) were male. Patients were examined in 4 groups according to CKD G1-G4 status. The mean age of the patients was 39.45 ± 10.57, 54.43 ± 7.29, 62.43 ± 12.26, 58.00 ± 4.16, respectively from CKD G1 to G4. Between groups, the presence of hypertension correlated with the severity of kidney failure. The median systolic blood pressure (mmHg) of the patients was 123.43±16.36, 133.81±18.50, 134.29±16.50, 147.14±14.96, respectively from CKD G1 to G4 (p=0.003). Body mass index was significantly increased in CKD G4 patients compared to CKD G1 patients (p=0.006) (Table 1).

Serum uric acid levels were similar in CKD G2 and G3 patients, but there was a statistically significant increase in CKD G4 patients when compared with other groups (p<0.001). When serum PLIN-2 / ADRP levels were compared, no significant difference was found between the groups (p=0.439). However, the mean serum PLIN-2 / ADRP levels were increased in line with the CKD stage progression. When the groups were compared in terms of height-adjusted Total Kidney Volume (HtTKV), there was a significant increase in kidney volume in patients with CKD G4 compared to the other stages (p =0.006). HDL levels were found

Table 1. Demographic and clinical comparison of ADPKD patients with basal values according to stage

Variables	Groups CKD G1 (>90 ml/dk; n=38)	CKD G2 (60-89 ml/dk; n=21)	CKD G3 (30-59 ml/dk; n=14)	CKD G4 (15-29 ml/dk; n=7)	*p*
Age (year)	39.45± 10.57[a]	54.43±7.29[b]	62.43±12.26[b]	58.00±4.16 [b]	**<0.001**
Gender %(Male)	12(31.6)[a]	14(66.7)[b]	8(57.1)[ab]	3(42)[ab]	0.057
Hypertension% (Yes)	19(50)[a]	21(100)[b]	12(85.7)[b]	7(100)[b]	**<0.001**
Status (ex)	0(0)	0(0)	2(14.3)[a]	1(14.3) [a]	0.033
SBP (mmHg)	123.43±16.36[a]	133.81±18.50[ab]	134.29±16.50[ab]	147.14±14.96[b]	**0.003**
DBP (mmHg)	78.95±12.36[a]	84.76±10.66[ab]	80.36±7.95[ab]	92.86± 9.51[b]	0.014
Body Mass Index (kg/m²)	24.26±4.50[a]	26.33±4.19[ab]	27.21±2.88[ab]	29.71±4.27[b]	0.006

Data: n (%), mean ± SD and median (25-75persentyl).
Different superscripts in the same row indicate a statistically significant difference between the groups. SBP: Systolic blood pressure. DBP: diastolic blood pressure
CKD G1–G4: Chronic Kidney Disease GFR category[15]

Table 2. Biochemical comparison of ADPKD patients with basal values according to stage

Variables	Groups				*P*
	CKD G1 (>90 ml/dk; n=38)	CKD G2 (60-89 ml/dk; n=21)	CKD G3 (30-59 ml/dk; n=14)	CKD G4 (15-29 ml/dk; n=7)	
Hemoglobin (g/dl)	13.59±1.74[a]	14.34±1.6[a]	13.71±1.97[a]	13.04±2.31[a]	0.312
PLIN-2/ADRP (ng / mL)	11.83 (10.18-12.73)[a]	11.12 (9.60-12.11)[a]	12.12 (9.92-12.95)[a]	12.84 (7.30-13.56)[a]	0.439
CRP (mg/L)	3.40 (3.25-3.45) [a]	3.40 (3.28-6.76) [a]	3.61 (3.19-7.05) [a]	3.40 (3.17-7.20) [a]	0.461
eGFR* (ml/min/1.73m^2)	114.50 (106.50-121.0)[a]	72.0 (65.90-80.0)[cd]	36.40 (31.60-54.72)[bd]	26.10 (21.50-27.0)[b]	**<0.001**
Total cholesterol (mg/dl)	189.61±351[a]	195.86±34.65[a]	190.71±47.8[a]	183.71±15.41[a]	0.871
Triglycerides (mg/dl)	100.5 (77.0-129.75)[a]	171.0 (104.5-258.5)[b]	179.5 (108.5-260.75)[b]	105.0 (84.0-126.0)[ab]	**0.001**
LDL (mg/dl)	113.76± 32.52[a]	121.62±29.05[a]	123.29±38.27[a]	114.00±20.78[a]	0.704
HDL (mg/dl)	49.26±11.11[a]	41.48±8.49[bc]	37.71±12.88[b]	44.00±7.23[abc]	**0.003**
Albumin (g/dl)	4.20 (3.90-4.50) [a]	4.20 (3.95-4.35) [a]	3.80 (3.45-4.22)[a]	4.0 (3.20-4.20)[a]	0.066
Uric acid (mg/dl)	4.32± 1.10[a]	6.21±1.48[b]	7.40±1.69[bc]	8.11±1.20[c]	**<0.001**
Calcium (mg/dl)	9.32±0.43[a]	9.34±0.41[a]	9.10±0.65[a]	9.27±0.41[a]	0.450
Phosphorus (mg/dl)	3.27±0.57[a]	3.09±0.46[a]	3.22±0.45[a]	3.40±0.56[a]	0.491
Proteinuria (g/24h)	0.10 (0.08-0.14) [a]	0.10 (0.09-0.17) [a]	0.31 (0.16-0.65) [b]	0.20 (0.10-0.90) [ab]	**<0.001**
HtTKV (ml/m)	502.50 (271.0-848.0)[a]	870.50 (352.50-1246.50)[b]	1124.0 (666.25-1303.5)[b]	1782 (995.5-3230.0)[b]	**0.006**

* Calculated by CKD-EPI method.

Different superscripts in the same row indicate a statistically significant difference between the groups. eGFR: Glomerular filtration rate
CRP: C reactive protein,
DBP: Diastolic blood pressure, SBP: Systolic blood pressure, LDL: Low-density lipoprotein,
HDL: High-density lipoprotein, HtTKV: height-adjusted Total Kidney Volume
PLIN-2/ADRP: Perilipin-2/Adipose Differentiation-Related Protein
CKD G1–G4: Chronic Kidney Disease GFR category[15]

to be higher in G1 CKD patients than in G4 CKD patients. This difference was statistically significant (p=0.003).

Proteinuria level was found to be higher in G4 CKD patients than in G1 CKD patients, consistent with CKD stages (p<0.001) (Table 2).

The median serum PLIN-2 /ADRP level measured in patients with ADPKD was 11.675 ng/mL. The female patients were found to be significantly increased in patients with serum PLIN-2 /ADRP levels above the median value (p<0.001). Similarly, body mass index was significantly higher in patients with serum PLIN-2 / ADRP levels above the median value (p <0.001) The hemoglobin median value was found to be significantly decreased in patients with serum PLIN-2/ ADRP levels above the median value (p<0.001). By following the GFR values of the patients between 2012 and 2019, the percentage change in the average annual glomerular filtration rate (ΔGFR%) was calculated. ΔGFR was observed to be increasing in patients with serum PLIN-2/ADRP levels above the median value. However, it is not statistically significant (p=0.343) (Table 3).

A cut-off value for serum PLIN-2 / ADRP was found 11.97 in ROC analysis (Table 4). When the effect of serum PLIN-2 / ADRP level on rapid progression was examined in the ROC curve analysis, the area under the ROC curve was found to be 0.53 (0.40-0.67). There was no statistically significant difference (p=0.237) (Figure 1).

Table 3. Demographic, clinical, and biochemical comparison of ADPKD patients with baseline values according to median serum PLIN-2 / ADRP level

Variables	PLIN-2/ADRP (ng / mL) ≤11.675 (n=40)	>11.675 (n=40)	*p*
Age (year)	48.48±14.37	49.58± 12.76	0.718
Gender %(Male)	31(77)	6(15)	**<0.001**
Hypertension% (Yes)	31(77.5)	28(70)	0.446
Status (ex)	2(5)	1(2.5)	0.556
SBP (mmHg)	130.13±19.59	130.13±16.73	0.999
DBP (mmHg)	80.38±11.11	83.50±12.04	0.232
Body Mass Index (kg/m²)	24.08±3.57	27.53±4.56	**<0.001**
Hemoglobin (g/dl)	14.54±1.63	12.985±1.65	**<0.001**
CRP (mg/L)	3.40(3.21-3.45)	3.40(3.28-4.60)	0.598
eGFR* (ml/dk)	87.55± 31.66	80.90± 37.08	0.513
Total cholesterol (mg/dl)	185.30±36.85	196.65±34.44	0.162
Triglycerides (mg/dl)	113(84-174)	124(95-193)	0.624
LDL (mg/dl)	113.38±32.81	121.15±30.39	0.307
HDL (mg/dl)	41.83±11.40	47.65±10.85	0.022
Albumin (g/dl)	4.17(3.82-4.30)	4.15(3.8-4.40)	0.739
Uric acid (mg/dl)	5.86± 1.94	5.52±1.91	0.427
Calcium (mg/dl)	9.33±0.47	9.25±0.47	0.452
Phosphorus (mg/dl)	3.18±0.526	3.26±0.52	0.583
Proteinuria (g/gün)	0.13(0.10-0.28)	0.10(0.09-0.19)	0.301
HtTKV (ml/m)	848(436-1236)	739(239-1122)	0.255
ΔGFR %	22.05(11.13-42.63)	24.90(15.03-42.07)	0.343

* Calculated by CKD-EPI method.
Data: n (%), mean ± SD and median (25-75persentyl).
SBP: Systolic blood pressure DBP: Diastolic blood pressure
eGFR: Glomerular filtration rate, CRP: C reactive protein, LDL: Low-density lipoprotein,
HDL: High-density lipoprotein HtTKV: height-adjusted Total Kidney Volume
PLIN-2/ADRP: Perilipin-2/Adipose Differentiation-Related Protein
ΔGFR: Percent change in average annual glomerular filtration rate

The relationship between annual eGFR values and serum PLIN-2 / ADRP level between 2012-2019 of the patients was analyzed. Mean eGFR values were higher in patients with serum PLIN-2 / ADRP levels below the mean value in both 2012 and 2019. In this group, the eGFR was 87.55 ± 31.66 ml/min/1.73 m2 in 2012. The mean eGFR value of the patients with serum PLIN-2 / ADRP levels above the mean value was 80.90 ± 37.08 ml/min/1.73 m2 in 2012. In ADPKD patients with high baseline mean PLIN-2/ADRP levels, the mean eGFR loss at the end of 2019 was found to be approximately 20 ml/min/1.73 m2. However, the mean eGFR loss in the other group was found to be approximately 16 ml/min/1.73 m2 at the end of 2019 (Figure 2 and Figure 3).

Table 4. ROC analysis results for rapid progression of serum PLIN-2 /ADRP levels in ADPKD

Variable	CUT-OFF	Diagnostic Measurements SEN (95% CI)	SPE (95% CI)	PPV (95% CI)	NPV (95%CI)
PLIN-2/ADRP	11.97	0.55 (0.40-0.69)	0.61 (0.42-0.78)	0.69 (0.50-0.80)	0.46 (0.32-0.66)

CI: Confidence interval, SEN: Sensitivity, SPE: Specificity, PPV: Positive Predictive Value,
NPD: Negative Predictive Value Değer PLIN-2/ADRP: Perilipin-2/Adipose Differentiation-Related Protein

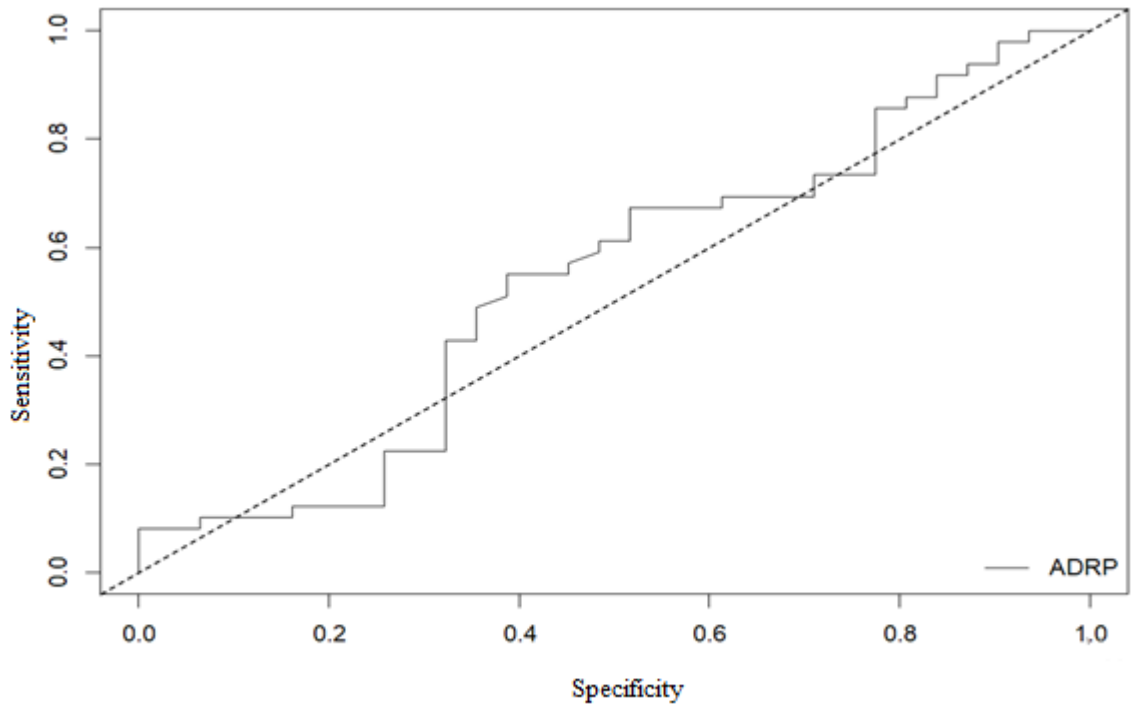

Figure 1. ROC analysis curve of the sensitivity and specificity of the effect of serum PLIN-2 / ADRP level on rapid progression in ADPKD

DISCUSSION

ADPKD stands as the most prevalent hereditary renal disorder, affecting millions of individuals worldwide. Extensive research has uncovered numerous crucial molecular and cellular mechanisms that contribute to the development of ADPKD. However, despite these advancements, many aspects of the disease still remain elusive. Unraveling the complex molecular and cellular mechanisms underlying this debilitating disease, as well as finding biomarkers that can predict disease progression, will pave the way for the

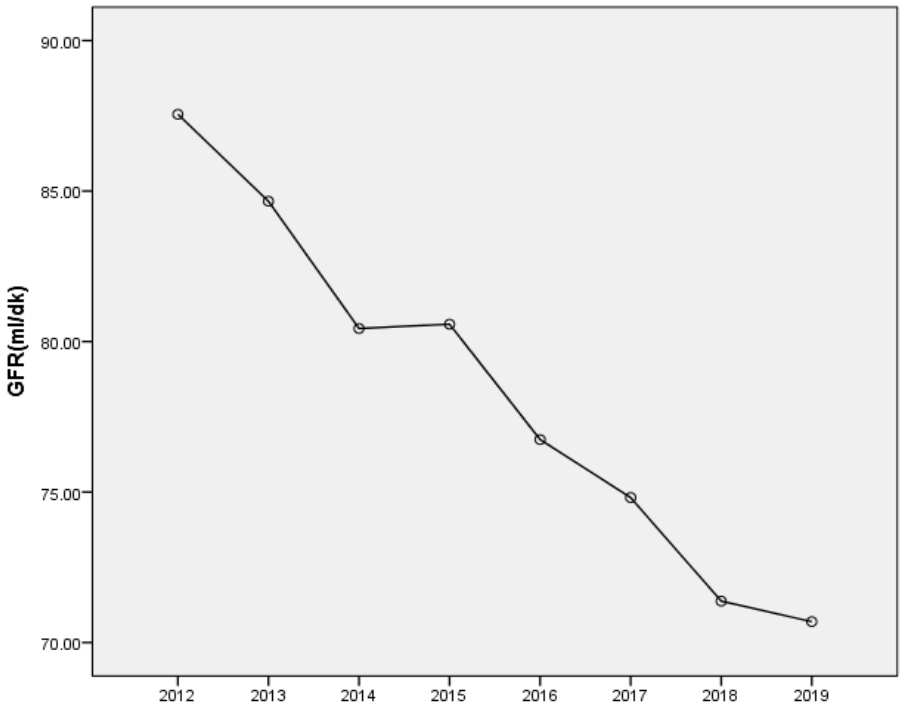

Figure 2. Mean eGFR course between 2012-2019 in patients with serum PLIN-2 / ADRP levels below the mean value

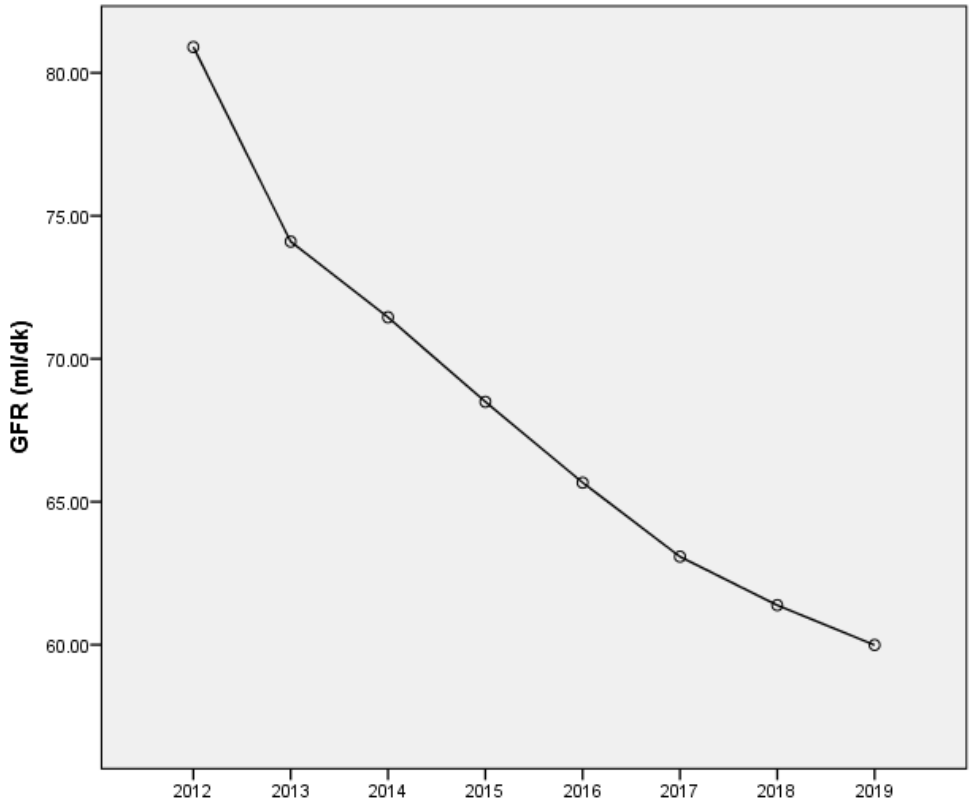

Figure 3. Mean eGFR course between 2012-2019 in patients with serum PLIN-2 / ADRP levels above the mean value

development of new targeted therapies that will alleviate the burden of ADPKD and improve the quality of life of affected individuals. The clinical course of ADPKD is variable, but the disease typically progresses slowly over time. The factors that influence prognosis in ADPKD include the age of onset, the severity of the disease, and the presence of other medical conditions.

Our study presents three significant discoveries. Firstly, high levels of PLIN-2 / ADRP are associated with increased BMI which may affect the clinical course of ADPKD. Secondly, PLIN-2 / ADRP is increased in female patients with ADPKD compared to male counterparts. Lastly, patients with high levels of PLIN-2 / ADRP are prone to faster kidney disease progression compared the patients with low levels of PLIN-2 / ADRP however the difference was not found to be statistically significant.

Perilipines (PLINs) have emerged as part of the group of proteins that react with the intracellular neutral lipid droplets. PLINs localized on the surface of the oil droplets have been found to highly phosphorylate adipocyte proteins, regulating lipolysis via cAMP and PKAF.[6] Based on metabolic disorders in uremic patients, by Axelsson *et al*., examined human adipocyte cells that were kept in uremic serum. In the study, it was found that the rate of spontaneous lipolysis was 30% higher in cells kept in uremic serum. In a study evaluating uremic serum and human adipocyte cells, the relationship between PLIN and uremia was investigated. It has been proposed that the increase in lipolysis in CKD may be associated with a decrease in PLIN synthesis.[17]

Unfortunately, there is currently no study in the literature that reveals the correlation between serum PLIN-2/ADRP levels and eGFR. However, in our study, eGFR values were significantly decreased in patients above the median value of PLIN-2/ADRP. While the mean eGFR value was 80.90±37.08 ml/min in this group, the mean eGFR was 87.55 ± 31.66 ml/min in patients with below the PLIN-2 / ADRP median value. In addition, the annual average percentage of eGFR change (ΔGFR) was evaluated. It was found that the change was higher in patients with serum PLIN-2 / ADRP levels above the median value. The BMI value was statistically significantly increased in patients with serum PLIN-2 / ADRP levels above the median value ($p<0.001$). A preclinical study conducted on mice has revealed a significant connection between PLIN-2/ADRP and obesity. This study demonstrates that hepatocyte-specific actions of PLIN-2/ADRP are central to the initiation and pathological progression of non-alcoholic fatty liver disease (NAFLD) through its effects on immune cell recruitment and fibrogenesis in obese and insulin-resistant mice. Conversely,

extra hepatocyte PLIN-2/ADRP actions support NAFLD pathophysiology through effects on obesity, insulin resistance, and inflammation.[18] A recent clinical study found that PLIN-2/ADRP levels do not change with age, but women have higher PLIN-2/ADRP levels than men. In addition, a strong relationship was found between PLIN-2/ADRP levels and BMI.[19] In our study, patients with high PLIN-2/ADRP levels mostly consisted of obese patients. A significant correlation was found between high PLIN-2/ADRP levels and BMI in our study, consistent with the literature ($p<0.001$). Furthermore, a significant correlation was found between female ADPKD patients and PLIN-2/ADRP levels. Supporting the previous knowledge, PLIN-2/ADRP levels were higher in female patients than in males ($p<0.001$).

In a 2017 study by Nowak *et al*., overweight and especially obesity were connected with the rate of progression in patients with early-stage ADPKD. In another study of this group, this relationship was once again demonstrated.[20, 21] In addition, Kocyigit *et al*. in a study with ADPKD patients who met the criteria for metabolic syndrome progressed significantly more rapidly than those who did not, during the 12-month follow-up period.[22] These studies mentioned above clearly demonstrated the relationship between obesity and kidney disease progression in ADPKD patients. In our study, it was shown that decreased eGFR was associated with increased BMI, consistent with the current literature.

In a large retrospective cohort study of ADPKD patients followed for many years, men had worse kidney function at a given age. In another retrospective study, the median age of onset of KF was 4 years younger in men than in women (52 versus 56 years).[23] The actual gender effect is unknown, as none of these studies were population-based. Larger population numbers are needed to fully see the gender relationship. However, the male gender stands out as a risk factor for ADPKD patients. In our study, patients with high PLIN-2/ADRP levels were women. Therefore, it suggests that high PLIN-2/ADRP levels may be associated with female gender and fat mass. In other words, PLIN-2/ADRP levels in ADPKD patients may be related to factors such as female hormones, BMI, and fat mass.

CONCLUSION

In conclusion, PLIN-2 / ADRP is associated with increased BMI and female sex in the ADPKD population. Taking into account these findings along with other research studies, the rise in serum PLIN-2 / ADRP levels provides valuable insights into the prediction of rapid progression. Although the current findings may not have been deemed statistically significant, it is crucial to clarify the link between the level of serum PLIN-2 / ADRP and ADPKD. This can be achieved through the utilization of a more extensive sample size and an extended follow-up duration.

Conflict of Interest

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding Sources

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Ethical Approval

The Ethics Committee of Erciyes University Clinical Research granted approval for this study by decision dated November 7, 2018 and number 2018/561.

Authors' Contribution

Study Conception: MC, EE, IK; Study Design: MC, EE, IK; Supervision; EE, IK; Funding: MC; Materials: CK, ACG; Data Collection and/or Processing: CK, GZ, ACG; Analysis and/or Data Interpretation: GZ; Literature Review: MC, EE; Critical Review: MC, EE, IK; Manuscript preparing: MC, EE, IK.

Main points

•As BMI increases in the ADPKD population, the median value of serum PLIN-2/ADRP level increases.

•The median value of serum PLIN-2/ADRP level was higher in female ADPKD patients than in males.

•As eGFR decreased in ADPKD patients, serum PLIN-2 / ADRP levels were found to increase, but it was not statistically significant.

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