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ROLE OF SERUM FETUIN-A PROTEIN AS INDICATOR OF CARDIOVASCULAR COMPLICATIONS IN PATIENTS WITH CHRONIC RENAL FAILURE ON HEMODIALYSIS

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ABSTRACT

Background: Chronic kidney disease (CKD) is a progressive condition leading to end-stage renal disease (ESRD), in which hemodialysis remains the most widely used therapeutic modality. Cardiovascular disease is a leading cause of morbidity and mortality in CKD and ESRD patients. Fetuin-A, a hepatic glycoprotein, has been implicated in vascular calcification, bone metabolism regulation, and insulin resistance. Reduced serum Fetuin-A levels have been associated with vascular calcification and cardiovascular complications in patients with metabolic and renal disorders. **Aim:** To evaluate serum Fetuin-A as a potential early indicator of cardiovascular risk in patients with chronic renal failure undergoing hemodialysis. **Patients and Methods:** This prospective, hospital-based study was conducted at Al-Kadhimiya Teaching Hospital between April and August 2021. Eighty participants were enrolled and divided into three groups: Group I (25 patients on hemodialysis ≥3 years), Group II (25 patients on hemodialysis <3 years), and Group III (30 apparently healthy controls). Serum Fetuin-A, total cholesterol, blood urea, and serum creatinine were measured in all participants. Blood pressure and clinical variables were also assessed. **Results:** Blood pressure was significantly lower in patients with ≥3 years on dialysis compared to those with <3 years ($p < 0.01$). Fetuin-A levels were markedly reduced in long-term dialysis patients. Serum Fetuin-A showed negative correlations with age, dialysis duration, systolic and diastolic blood pressure, blood urea, creatinine, and cholesterol. **Conclusion:** Serum Fetuin-A levels are significantly decreased in hemodialysis patients, particularly with longer dialysis duration, and may serve as an early biomarker for cardiovascular risk in this population.

KEYWORDS: Serum, Fetuin-A, protein, Cardiovascular, Complications, Chronic, Renal Failure, Hemodialysis.

INTRODUCTION

Chronic kidney disease (CKD) is defined as a progressive and irreversible decline in renal function, often evolving over years from asymptomatic biochemical abnormalities to end-stage renal disease (ESRD), when renal replacement therapy becomes mandatory for survival.[1] CKD is associated not only with progressive uremia but also with a markedly increased risk of cardiovascular disease (CVD), which accounts for the majority of morbidity and mortality in this population.[2] Although estimated glomerular filtration rate (eGFR) and albuminuria remain the cornerstone markers for CKD prognosis[3,4], novel biomarkers are increasingly investigated to improve risk stratification and clarify underlying mechanisms.[5,6] The global burden of CKD is substantial, with an estimated prevalence of ~10% worldwide.[7] Diabetes mellitus and hypertension are recognized as the leading causes, followed by chronic glomerulonephritis, polycystic kidney disease, and other secondary etiologies.[8,9] The risk of CKD is influenced by age, ethnicity, and genetic factors; for instance, APOL1 variants markedly increase susceptibility to ESRD in African ancestry populations.[10,11] The natural course of CKD involves progressive nephron loss, maladaptive hyperfiltration, proteinuria, and interstitial fibrosis, culminating in renal failure.[12] Prognosis is highly dependent on comorbidities such as diabetes, obesity, and

dyslipidemia.[13,14] Hemodialysis remains the most widely used therapy for ESRD, achieved through vascular access and extracorporeal solute clearance.[1] Despite its efficacy in prolonging survival, hemodialysis is associated with multiple complications, including hypotension, arrhythmias, sepsis, and accelerated vascular calcification.[15] Indeed, vascular calcification and arterial stiffness are central mediators of CVD in CKD patients, traditionally attributed to disordered calcium-phosphate metabolism but now recognized as active, regulated processes involving cytokines and serum proteins.[16] Among these proteins, Fetuin-A (α2-Heremans–Schmid glycoprotein, AHSG) has emerged as a critical systemic inhibitor of ectopic calcification. Fetuin-A is a 64-kDa glycoprotein synthesized by the liver and secreted into circulation.[17] It binds calcium and phosphate, forming soluble complexes that prevent pathological precipitation in soft tissues.[18] Experimental and clinical studies show that Fetuin-A deficiency promotes vascular and myocardial calcification, endothelial dysfunction, and higher mortality in hemodialysis patients.[19] Furthermore, low Fetuin-A levels are associated with peripheral arterial disease in type 2 diabetes and inversely correlated with coronary artery calcification.[20] Beyond mineral metabolism, Fetuin-A modulates insulin sensitivity and lipid handling, linking it to metabolic and cardiovascular pathways.[21] Recent evidence suggests that Fetuin-A may protect cardiomyocytes from glucose toxicity and myocardial fibrosis, highlighting its broader cardiovascular role.[22] Thus, reduced serum Fetuin-A concentrations in CKD and ESRD patients may serve as an early biomarker of cardiovascular risk, potentially improving prognostication and guiding preventive strategies. The aim of this study is to evaluate serum Fetuin-A Protein as an early indicator of risk factor of cardiovascular complication in patients with chronic Renal Failure on hemodialysis.

METHOD

This prospective hospital-based study was carried out at Al-Kadhimiya Teaching Hospital between April 1 and August 1, 2021, and included 80 participants. The study population was divided into three groups: Group I, 25 adults with end-stage renal disease (ESRD) on hemodialysis for ≥3 years (mean age 47.6, range 25–71 years); Group II, 25 adults on dialysis for <3 years (mean age 44.2, range 28–64 years); and Group III, 30 apparently healthy adults without personal or family history of CKD serving as controls (mean age 38.1, range 25–66 years). **Inclusion criteria** were adults with ESRD undergoing hemodialysis and negative viral screens for hepatitis B, hepatitis C, and HIV. **Exclusion criteria** included diabetic nephropathy and peritoneal dialysis. Clinical diagnosis of CKD was based on biochemical parameters and imaging studies. Baseline data collected included age, gender, weight, height, blood pressure, and body mass index (BMI), in addition to laboratory values. **Sample collection:** Following informed consent, 5 mL of venous blood was obtained from each participant, allowed to clot, centrifuged at 3000 rpm for 20 minutes, and serum aliquots were stored at –20°C until analysis. **Laboratory assays:** Serum Fetuin-A was measured using an ELISA kit (HUMAN, Germany) based on the biotin double antibody sandwich technique, with an assay range of 10–4000 mg/L. Blood urea was determined enzymatically by urease and glutamate dehydrogenase, while creatinine was assayed by an enzymatic method using creatininase and sarcosine oxidase. Total cholesterol was measured by enzymatic hydrolysis and oxidation followed by colorimetric detection. **Anthropometry:** Height and weight were measured, and BMI was calculated as weight (kg)/height (m^2). **Ethics:** Approval was obtained from the University of Baghdad, College of Medicine, Department of Clinical Biochemistry, and the Iraqi Board for Medical Specializations. Oral informed consent was secured from all participants. **Statistical analysis:** Data were analyzed using SPSS v20. Continuous variables were expressed as mean ± SD or median (IQR). Group differences were assessed using ANOVA or Kruskal–Wallis test as appropriate. Correlations were examined with Pearson's test. A p-value <0.05 was considered statistically significant.

RESULTS

Eighty samples were included in this case-control study. The mean age for the patients was 47.0 ± 12.1 years old and for the control group was 43.1 ± 11.7 years. A 46% of the cases were males and 54% were females in comparison to 60% males and 40% females in the control group (Table 1). The mean dialysis sessions duration in the patients with CKD was 3.5 ± 2.7 years. When comparing cases to control groups, a statistically significant difference was found concerning systolic blood pressure, diastolic blood pressure, blood urea, serum creatinine, and serum cholesterol. In regard to fetuin-A, the median (IQR) was 229.6 (193.8 - 333.5) mg/L in the cases group and 372.8 (264.1 - 783.1) mg/L in the control group and difference was significant (P-value = 0.007).

Table 1: Description of study parameters in cases and control groups and the statistical difference between them.

Characteristics	Cases, N = 50[1]	Control, N = 30[1]	P-value[2]
Age, years	47.0 ± 12.1	43.1 ± 11.7	0.15
Sex			
Male	23 (46%)	18 (60%)	0.2
Female	27 (54%)	12 (40%)	
Systolic BP, mmHg	141.6 ± 26.1	111.05 ± 15.78	**0.006**
Diastolic BP, mmHg	81.8 ± 13.7	75.03 ± 9.67	**0.01**
BMI, kg/m^2	25.2 ± 4.8	26.5 ± 2.6	0.11
Blood urea, mg/dL	147.9 ± 39.8	24.8 ± 6.0	**<0.001**
Serum creatinine, mg/dL	9.9 ± 2.8	0.7 ± 0.2	**<0.001**
Serum cholesterol, mg/dL	182.9 ± 39.8	156.4 ± 38.6	**0.005**
Serum Fetuin-A, mg/L	229.6 (193.8 - 333.5)	372.8 (264.1 - 783.1)	**0.007**

[1]Mean ± SD; n (%); Median (IQR)
[2]Welch Two Sample t-test; Fisher's exact test; Pearson's Chi-squared test; Wilcoxon rank sum test

Those with less than 3 years of dialysis and those with more than 3 years of dialysis were compared with the control group in table 2 and 3. It was found that blood pressure, renal function test, serum cholesterol and especially serum fetuin- A values were significantly different when compared with the control group.

Table 2: Blood pressure and blood biomarkers comparison between those with less than 3 years of dialysis and the control group.

Characteristics	< 3 years, N = 25[1]	Control, N = 30[1]	P-value[2]
Systolic BP, mmHg	151.1 ± 23.1	111.05 ± 15.78	**0.006**
Diastolic BP, mmHg	87.6 ± 13.2	75.03 ± 9.67	**0.008**
BMI, kg/m^2	24.8 ± 4.4	26.5 ± 2.6	0.13
Blood urea, mg/dL	148.7 ± 38.5	24.8 ± 6.0	**<0.001**
Serum creatinine, mg/dL	9.6 ± 2.6	0.7 ± 0.2	**<0.001**
Serum cholesterol, mg/dL	174.5 ± 37.5	156.4 ± 38.6	**0.01**
Serum Fetuin-A, mg/L	261.7 (207 - 540.6)	372.8 (264.1 - 783.1)	**0.02**

[1]Mean ± SD; Median (IQR)
[2]Welch Two Sample t-test; Wilcoxon rank sum test

Table 3: Blood pressure and blood biomarkers comparison between those with more than 3 years of dialysis and the control group.

Characteristics	≥ 3 years, N = 25[1]	Control, N = 30[1]	P-value[2]
Systolic BP, mmHg	132.0 ± 25.9	111.05 ± 15.78	**0.008**
Diastolic BP, mmHg	76.1 ± 11.9	75.03 ± 9.67	**0.002**
BMI, kg/m^2	25.5 ± 5.2	26.5 ± 2.6	0.5
Blood urea, mg/dL	147.1 ± 41.9	24.8 ± 6.0	**<0.001**
Serum creatinine, mg/dL	10.2 ± 2.9	0.7 ± 0.2	**<0.001**
Serum cholesterol, mg/dL	198.2 ± 40.4	156.4 ± 38.6	**0.01**
Serum Fetuin-A, mg/L	199.2 (191.3 - 235.9)	372.8 (264.1 - 783.1)	**0.004**

[1]Mean ± SD; Median (IQR)
[2]Welch Two Sample t-test; Wilcoxon rank sum test

In patients with < 3 years and those with ≥ 3 years of dialysis duration (Table 4), it was found that blood pressure was significantly lower in those with ≥ 3 years than those with < 3 years (P-value <0.01). Also, fetuin-A level was much lower in those with ≥ 3 years' duration with a median of 199.2 compared to 261.7 (P- value = 0.002).

Table 4: Description of study parameters in regard to dialysis session duration (less than 3 years and more than 3 years duration)			
Characteristics	**< 3 years, N = 25[1]**	**≥ 3 years, N = 25[1]**	**P-value[2]**
Age, years	46.5 ± 10.1	47.5 ± 14.0	0.8
Sex			
Male	15 (60%)	12 (48%)	0.4
Female	10 (40%)	13 (52%)	
Systolic BP, mmHg	151.1 ± 23.1	132.0 ± 25.9	**0.008**
Diastolic BP, mmHg	87.6 ± 13.2	76.1 ± 11.9	**0.002**
BMI, kg/m^2	24.8 ± 4.4	25.5 ± 5.2	0.6
Blood urea, mg/dL	148.7 ± 38.5	147.1 ± 41.9	0.9
Serum creatinine, mg/dL	9.6 ± 2.6	10.2 ± 2.9	0.5
Serum cholesterol, mg/dL	174.5 ± 37.5	198.2 ± 40.4	0.7
Serum Fetuin-A, mg/L	261.7 (207 - 540.6)	199.2 (191.3 - 235.9)	**0.002**
[1]Mean ± SD; n (%); Median (IQR) [2]Welch Two Sample t-test; Fisher's exact test; Pearson's Chi-squared test; Wilcoxon rank sum test			

In table 5, sex was compared in both cases and controls regarding serum fetuin-A values. No significant difference observed in both males and females.

Table 5: Gender matched comparison of serum fetuin‑A levels (mg/L) in the study groups.			
Characteristics	**Male[1]**	**Female[1]**	**P-value[2]**
Cases	235.9 (199.7 - 303.3)	218.1 (191.7 - 347.6)	0.6
Control	355.9 (375.6 - 931.1)	301.3 (283.7 - 531.3)	0.09
[1]Median (IQR) **[2]Wilcoxon rank sum exact test**			

Table 6 illustrate the age matching of serum fetuin‑A levels (mg/L) in the study groups. When comparing cases and control groups, those with less than 30 years of age showing a significantly difference among the two group (P-value = 0.04). the same was true for those 30-50 years and those > 50 years of age, P-value = 0.01 and 0.002, respectively.

Table 6: Age matched analysis of serum fetuin‑A levels (mg/L) in the study groups.			
Characteristics	**Cases, N = 50[1]**	**Control, N = 30[1]**	**P-value[2]**
< 30 years	206.2 (192.5 - 259.6)	246.9 (208.4 - 323.6)	0.04
30-50 years	152.1 (139.4 - 161.1)	270.7 (180.3 - 907.6)	0.01
> 50 years	177.0 (167.6 - 186.7)	252.1 (201.0 - 781.6)	0.002
[1]Median (IQR) [2]Wilcoxon rank sum exact test			

Pearson correlation analysis was conducted to study the association between serum fetuin-A and other study parameters. Serum fetuin-A was negatively correlated with age, dialysis session duration, systolic blood pressure, diastolic blood pressure, blood urea (R = -0.43), serum creatinine (R = -0.38), and serum cholesterol (R = -0.067), as shown in table 7, 8 and fig 1.

Table 7: Correlation analysis between fetuin-A, age, blood pressure, BMI, and blood biomarkers.		
Characteristics	**Correlation Coefficient (R)**	**P-value***
Age, years	-0.11	0.31
Session duration, years	-0.46	**0.0006**
Systolic BP, mmHg	-0.28	**0.04**
Diastolic BP, mmHg	-0.38	**0.006**
BMI, kg/m^2	-0.08	0.42
Blood urea, mg/dL	-0.43	**0.021**

Serum creatinine, mg/dL	-0.38	**0.042**
Serum cholesterol, mg/dL	-0.067	**0.05**
*Pearson's product-moment correlation		

Table 8: cutoff point of Fetuin-A protein to predict Risk factor of CV complication.

Characteristics	Cut-off point	Sensitivity	Specificity	AUC
Serum Fetuin	401.45	60%	95%	0.81
*Youden method				

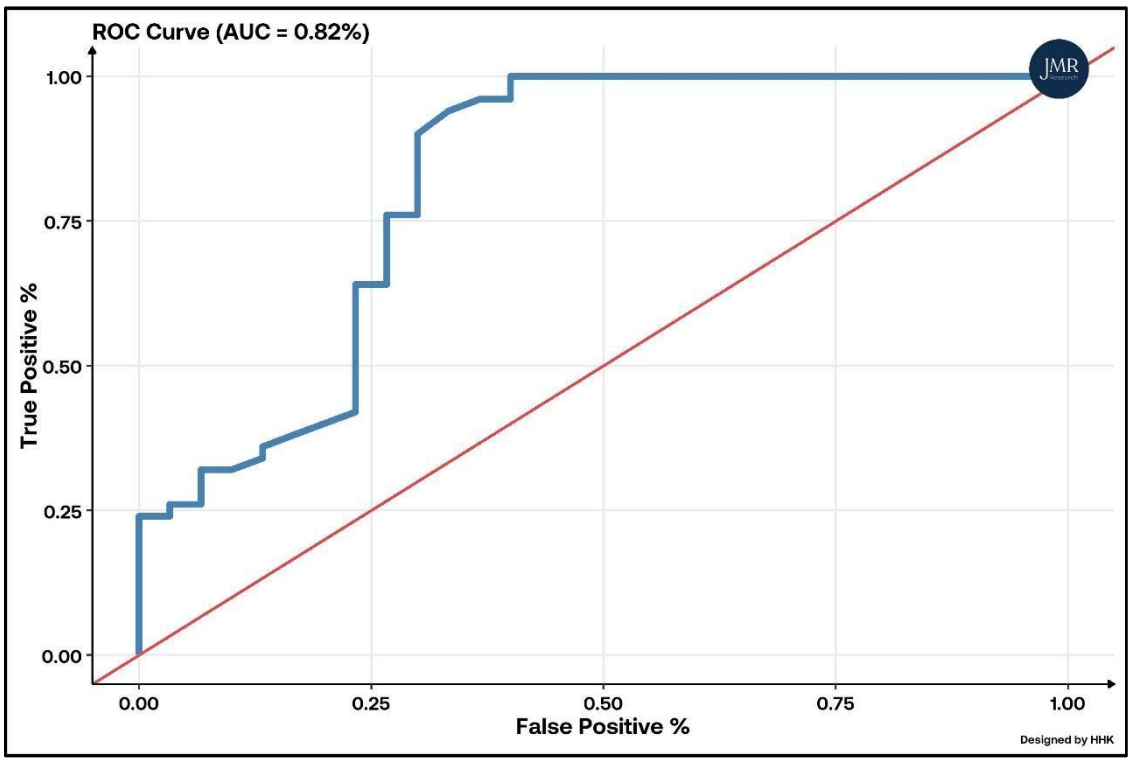

Figure 1: Prevalence of a parameter.

DISCUSSION

This study investigated serum Fetuin-A levels and their association with cardiovascular risk markers in patients with chronic kidney disease (CKD) undergoing hemodialysis, compared with healthy controls. The findings demonstrate that patients with established renal failure exhibit significantly higher blood pressure, urea, creatinine, and cholesterol levels, coupled with markedly reduced serum Fetuin-A concentrations. These alterations were consistent irrespective of dialysis duration, highlighting the potential of Fetuin-A as an early biomarker of cardiovascular risk in CKD. The demographic characteristics of our patients, including mean age (47.0 ± 12.1 years) and gender distribution (46% males, 54% females), were comparable to Iraqi and Indian studies[23,24] lower than Iranian cohorts.[25] This suggests regional variability in CKD populations, potentially reflecting differences in underlying etiologies, lifestyle, and healthcare access. Importantly, our study found no significant effect of age, gender, or body mass index (BMI) on Fetuin-A levels, a result consistent with Deepa et al[24], and Smith et al.[26] Blood pressure was significantly higher among CKD patients compared to controls, a finding supported by Makulska et al[27], who reported similar elevations in both systolic and diastolic readings. The link between renal failure and systemic hypertension is well established, as impaired renal sodium handling and activation of the renin–angiotensin system contributes to persistent hypertension, which in turn accelerates vascular damage and cardiovascular complications.[27] Interestingly, patients on long-term dialysis (≥3 years) had lower blood pressure than those with shorter dialysis duration, which may reflect improved fluid balance and better adherence to dietary and pharmacological interventions Consistent with previous studies[24,27,28], our results revealed significantly elevated serum urea, creatinine, and cholesterol levels in CKD patients. Dyslipidemia is common in renal failure due to impaired lipoprotein metabolism and contributes to atherosclerotic risk.[28] While Deepa et al[24] and Makulska et al[27] also reported increased triglyceride levels, this parameter was not evaluated in our cohort. The observed biochemical abnormalities underscore the systemic metabolic disturbances of CKD and their role in cardiovascular morbidity. The central finding of our study was the significantly lower serum Fetuin-A levels in CKD patients compared with controls (229.6 vs 372.8 mg/L,

p=0.007). This observation aligns with the results of Hameed et al[23], Deepa et al[24], Haddad et al[25], Makulska et a[27], and Toprak et al[28], all of which demonstrated reduced Fetuin-A in CKD patients. Fetuin-A acts as a systemic inhibitor of ectopic calcification by forming soluble mineral complexes, thereby preventing vascular and myocardial calcification.[19,20] Its deficiency in CKD may therefore contribute to the high prevalence of arterial stiffness, endothelial dysfunction, and cardiovascular mortality in this population. Furthermore, we found that Fetuin-A levels decreased significantly with longer dialysis duration, consistent with Deepa et al[24] and Caglar et al.[29] Conversely, Haddad et al[25] reported no association between dialysis duration and Fetuin-A, likely due to shorter dialysis exposure in their cohorts. The progressive reduction of Fetuin-A may be explained by chronic inflammation, impaired hepatic synthesis, or urinary loss of the protein.[28] Chronic inflammation in ESRD, characterized by leukocyte dysfunction and persistent immune activation, is a key factor in lowering Fetuin-A and promoting vascular calcification.[28] Correlation analysis in our study demonstrated significant negative associations between Fetuin-A and dialysis duration, blood pressure, urea, creatinine, and cholesterol. These findings are in agreement with Makulska et al[27] and Deepa et al[24], who reported similar correlations with biochemical and cardiovascular parameters. Such associations strengthen the hypothesis that declining Fetuin-A levels may serve as a surrogate marker for both renal dysfunction and cardiovascular risk. Overall, the current evidence supports Fetuin-A as a promising prognostic biomarker in CKD and ESRD. Reduced levels are strongly linked to adverse metabolic and cardiovascular profiles, independent of demographic factors. Longitudinal studies are warranted to establish whether monitoring Fetuin-A could enhance risk stratification and guide therapeutic strategies aimed at mitigating cardiovascular complications in patients on chronic hemodialysis.

CONCLUSION

Serum Fetuin-A protein level is significantly low in patients with renal failure. Serum Fetuin-A Protein level is negatively correlated with duration of dialysis (in more than 3 years) systolic and diastolic blood pressure, blood urea, serum creatinine and serum cholesterol levels. Age and body mass index does not correlate with serum Fetuin-A protein levels Serum Fetuin-A protein could be a promising marker to predict cardiovascular complications in patients with renal failure undergoing hemodialysis.

REFERENCES

1. Conway B, Phelan PJ, Stewart GD. Nephrology and urology. In: Davidson's Principles and Practice of Medicine. 24th ed. Edinburgh: Elsevier; 2022; p. 588, 592, 594.
2. Bochud M. On the rationale of population screening for chronic kidney disease: a public health perspective. Public Health Rev, 2015; 36: 11. doi: 10.1186/s40985-015-0009-9.
3. Liu C, Chen H, Liu C, Fu C, Zhang H, Yang H, et al. Combined application of eGFR and albuminuria for the precise diagnosis of stage 2 and 3a CKD in the elderly. J Nephrol, 2014; 27(3): 289-97. doi: 10.1007/s40620-013-0011-6.
4. Wang J, Wang F, Liu S, Zhou M, Zhang L, Zhao M. Reduced kidney function, albuminuria, and risks for all-cause and cardiovascular mortality in China: a population-based cohort study. BMC Nephrol, 2017; 18: 188. doi: 10.1186/s12882-017-0603-9.
5. Mihai S, Codrici E, Popescu ID, Enciu AM, Rusu E, Zilisteanu D, et al. Proteomic biomarkers panel: new insights in chronic kidney disease. Dis Markers, 2016; 2016: 3185232. doi: 10.1155/2016/3185232.
6. Homsak E, Ekart R. ST2 as a novel prognostic marker in end-stage renal disease patients on hemodiafiltration. Clin Chim Acta, 2018; 477: 105-12. doi: 10.1016/j.cca.2017.12.006.
7. Ferguson TW, Tangri N, Rigatto C, Komenda P. Cost-effective treatment modalities for reducing morbidity associated with chronic kidney disease. Expert Rev Pharmacoecon Outcomes Res, 2015; 15(2): 243-52.
8. Kilbride H. Estimating glomerular filtration rate and the effects of acute kidney injury on progression of chronic kidney disease [MD thesis], 2015; 59-195.
9. Sharma M, Doley P, Das H. Etiological profile of chronic kidney disease: a single-center retrospective hospital-based study. Saudi J Kidney Dis Transpl, 2018; 29(2): 409.
10. Lerma EV, Sparks MA, Topf J. Nephrology secrets. 4th ed. Philadelphia: Elsevier Health Sciences, 2019 Feb 10.
11. Jameson J, Loscalzo J. Harrison's Nephrology and Acid-Base Disorders. 2nd ed. New York: McGraw-Hill Education, 2013.
12. Al-Taiee T, Al-Shammaa N. Effect of antidiuretic hormone (ADH) in kidney function on post-hemodialysis end stage renal failure disease (ESRD) Iraqi patients. Iraqi J Sci, 2018; 59(3): 1372-7.
13. Ingrasciotta Y, Sultana J, Giorgianni F, et al. Association of individual non-steroidal anti-inflammatory drugs and chronic kidney disease: a population-based case-control study. PLoS One, 2015; 10(4): e0122899.
14. Mikolasevic I, Zutelija M, Mavrinac V, Orlic L. Dyslipidemia in patients with chronic kidney disease: etiology and management. Int J Nephrol Renovasc Dis, 2017; 10: 35-45.
15. Jamaati BA, et al. Serum fetuin-A levels and its relationship with biochemical parameters in hemodialysis patients. J Renal Inj Prev, 2022; 11(1): e06.
16. Chertow GM, Raggi P, Chasan-Taber S, Bommer J, Holzer H, Burke SK. Determinants of progressive vascular calcification in haemodialysis patients. Nephrol Dial Transplant, 2004; 19(6): 1489-96.

17. Carracedo M, Witasp A, Qureshi AR, Laguna-Fernandez A, Brismar T, Stenvinkel P, et al. Chemerin inhibits vascular calcification through ChemR23 and is associated with lower coronary calcium in chronic kidney disease. J Intern Med, 2019; 286(4): 449-57.
18. Jahnen-Dechent W, Heiss A, Schafer C, Ketteler M. Fetuin-A regulation of calcified matrix metabolism. Circ Res, 2011; 108(12): 1494-509.
19. Ulutas O, Taskapan MC, Dogan A, Baysal T, Taskapan H. Vascular calcification is not related to serum fetuin-A and osteopontin levels in hemodialysis patients. Int Urol Nephrol, 2018; 50(1): 137-42.
20. Lichtenauer M, et al. Specifics of fetuin-A levels in distinct types of chronic heart failure. J Clin Lab Anal, 2018; 32(1): e22179.
21. Bourebaba L, Marycz K. Pathophysiological implications of fetuin-A glycoprotein in the development of metabolic disorders: a concise review. J Clin Med, 2019; 8(12): 2033.
22. Mori K, et al. Fetuin-A and the cardiovascular system. Kidney Int Suppl, 2012; (3): 87-8.
23. Hameed MN, Khudhair KA, Yasir I. Evaluation of serum level of cystatin C and fetuin-A in patients with chronic renal failure. Indian J Forensic Med Toxicol, 2021; 15(3): 2226-34.
24. Deepa P, Sasivathanam N. Serum fetuin-A in chronic kidney disease: a promising biomarker to predict cardiovascular risk. Int J Sci Study, 2016; 4(9): 138-43.
25. Haddad M, Tajbakhsh R, Farajollahi M, Qorbani M, Besharat S, et al. Association of serum fetuin-A and biochemical parameters in hemodialysis patients. Saudi J Kidney Dis Transpl, 2014; 25(4): 769-73.
26. Smith ER, Cai MM, McMahon LP, Pedagogos E, Toussaint ND, Brumby C, et al. Serum fetuin-A concentration and fetuin-A-containing calciprotein particles in patients with chronic inflammatory disease and renal failure. Nephrology (Carlton), 2013; 18(3): 215-21.
27. Makulska I, Szczepańska M, Drożdż D, Polak-Jonkisz D, Zwolińska D. The importance of fetuin-A in vascular calcification in children with chronic kidney disease. Adv Clin Exp Med, 2019; 28(4): 499-505.
28. Toprak AE, Gerin F, Erman H. Serum fetuin-A levels and association with hematological parameters in chronic kidney disease and hemodialysis patients. Turk J Biochem, 2019; 44(4): 517-23.
29. Caglar K, Yilmaz MI, Saglam M, Cakir E, Kilic S, Sonmez A, et al. Serum fetuin-A concentration and endothelial dysfunction in chronic kidney disease. Nephron Clin Pract, 2008; 108(3): c233-40.