

STUDY OF LIPID PROFILE IN CHRONIC RENAL FAILURE

Mudunuri Sita Rama Lakshmi¹, Yeedi Ramya Subhashini², K. S. R. Swami³

¹Post Graduate, Department of General Medicine, Konaseema Institute of Medical Sciences, Amalapuram.

²Post Graduate, Department of General Medicine, Konaseema Institute of Medical Sciences, Amalapuram.

³Professor, Department of General Medicine, Konaseema Institute of Medical Sciences, Amalapuram.

ABSTRACT

BACKGROUND

Chronic kidney disease (CKD) is an irreversible deterioration of renal function, which results from diminished effective functioning of renal tissue. Ensuing impairment of excretory, metabolic and endocrine functions of the kidney leads to the development of clinical syndrome of uraemia. Dyslipidaemia is found in many patients that lead to cardiovascular complication.

MATERIAL AND METHOD

Cases of chronic kidney disease admitted in the medical wards of Konaseema Institute of Medical Sciences from June 2013 to October 2015 were taken for study.

RESULT

Total cholesterol value in controls and CKD patients are 185.2 ± 24.51 and 187 ± 43.5 mg/dL respectively. Triglyceride values in patients and controls were 174 ± 60.7 and 97 ± 17 mg/dL respectively. HDL values in chronic kidney disease patients are decreased compared to controls, 36 ± 5.1 and 48.8 ± 10.3 mg/dL. VLDL- Significant increase in VLDL was found in CKD patients as compared to controls, 34.88 ± 12.15 and 19.3 ± 3.49 respectively.

LDL values were almost similar in both CKD patients and control group, 116.49 ± 38.34 and 116.8 ± 26.78 respectively.

CONCLUSION

Lipid abnormalities in chronic kidney disease accelerates the progression of the renal failure and predisposes to atherosclerosis, it is worthwhile detecting and treating hyperlipidaemia in chronic kidney disease patients.

KEYWORDS

Lipid profile, Chronic renal failure, Cardiovascular disease.

HOW TO CITE THIS ARTICLE: Lakshmi MSR, Subhashini YR, Swami KSR. Study of lipid profile in chronic renal failure. J. Evid. Based Med. Healthc. 2016; 3(32), 1508-1515. DOI: 10.18410/jebmh/2016/341

INTRODUCTION: Chronic kidney disease (CKD) is an irreversible deterioration of renal function, which results from diminished effective functioning of renal tissue. Ensuing impairment of excretory, metabolic and endocrine functions of the kidney leads to the development of clinical syndrome of uraemia.

Cardiovascular disease is a major cause of morbidity and mortality among patients with chronic kidney disease^{1,2,3,4,5}. Thus, although some patients with CKD will ultimately develop end stage renal disease (ESRD), most patients with CKD will die of CVD before dialysis becomes necessary.^{6,7}

The growing recognition that dyslipidaemia is a major risk factor for coronary heart disease has prompted interest in the identification and management of abnormalities in plasma lipids and lipoproteins. In chronic renal failure, the most prevalent lipid disorders are hypertriglyceridaemia and decreased HDL Concentration. LDL levels are usually normal

or marginally increased.^{8,9,10,11,12} Also there are reports available regarding accelerated atherosclerosis in chronic kidney disease due to altered lipid metabolism. In recent years, the levels of high-density lipoproteins have gained importance in view of the fact that increasing reports are available incriminating decreased HDL levels as one of risk factors for cardiovascular disease

MATERIAL AND METHOD: Cases of chronic Kidney disease admitted in the medical wards of Konaseema Institute of Medical Sciences from June 2013 to October 2015 were taken for study.

Inclusion Criteria: Patients of chronic kidney disease.

Diagnostic criteria for chronic kidney disease.

1. Clinical signs and symptoms of uraemia.
2. The presence of chronic kidney disease was established based on presence of kidney damage and level of kidney function (GFR). Markers of kidney damage included abnormalities in the composition of blood (elevated blood urea, serum creatinine) or urine or abnormalities in imaging tests (ultrasonogram).
3. Ultrasonographic evidence of bilateral shrunken kidney/loss of corticomedullary differentiation.

Financial or Other, Competing Interest: None.

Submission 28-03-2016, Peer Review 02-04-2016,

Acceptance 11-04-2016, Published 21-04-2016.

Corresponding Author:

Dr. Mudunuri Sita Rama Lakshmi,

Post Graduate, Department of General Medicine,

Konaseema Institute of Medical Sciences, Amalapuram.

E-mail: anand_kims@yahoo.co.in

DOI: 10.18410/jebmh/2016/341

Exclusion Criteria:

1. Patients with diabetes mellitus.
2. Patients with ischaemic heart disease.
3. Patients who have undergone coronary artery bypass graft.
4. Patients on lipid lowering drugs.
5. Patients with history of alcohol consumption and smoking.

All the selected patients were subjected to detailed history and complete physical examination and data collected was noted in a pre-designed proforma.

The control group was formed by 30 healthy persons, which was age and sex matched to the study group.

Clinical Criteria for Chronic Kidney Disease: The clinical signs and symptoms are not absolute and are nonspecific, occur only in the later stage and are not dependable.

The features of uraemia include fatigue, lethargy, somnolence, headache, muscular irritabilities, muscle cramps, asterixis, myoclonus, seizures, coma.

Cardiovascular and pulmonary disturbances in the form of congestive cardiac failure, pulmonary oedema, pericarditis and arrhythmias.

Haematological disturbances in the form of anaemia, bleeding diathesis. Gastrointestinal disturbances in the form of anorexia, nausea and vomiting, gastroenteritis, peptic ulcer, gastrointestinal bleeding.

Features of hyperparathyroidism in the form of osteomalacia, bone pains, fractures and osteomalacic myopathy and gait disturbance.

Clinical evidence of pallor, oedema, Hypertension, pleuritis, pericarditis, CCF, pulmonary oedema, hypertensive retinopathy changes were noted.

Urine output below 500 mL was considered as oliguria¹³ and more than 3000 mL was considered as polyuria.¹⁴

Urine Findings: Proteinuria was considered as present when the heat test showed a definite cloud which did not get dissolved on addition of glacial acetic acid. Urine pus cells more than 2-3 per HPF was considered abnormal.

Biochemical Findings: The presence of chronic kidney disease was established based on presence of kidney

damage and level of kidney function (GFR). Markers of kidney damage included abnormalities in the composition of blood (elevated blood urea, serum creatinine) or urine or abnormalities in imaging tests (ultrasonogram).

Study of lipid profile by enzymatic method by using autoanalyser.

Serum Triglyceride Estimation¹⁵: This was determined by the fully enzymatic U-V method.

Serum total Cholesterol Estimation: This test was done on a method based on Liebermann-Burchard reaction calorimetric method.

HDL Cholesterol Estimation: This involves two steps. Precipitation and cholesterol estimation of the HDL fraction by a modification of the method described by Burstein et al.

LDL-Cholesterol Estimation: LDL cholesterol is calculated by using a standard WHO approved formula based on total cholesterol, triglyceride and HDL-cholesterol values;

$\text{LDL-cholesterol} = \text{Total cholesterol} - \frac{\text{Triglyceride}}{5} \rightarrow \text{HDL}$ $\frac{\text{Triglyceride}}{5}$ Indicate the cholesterol in VLDL fraction and was used only when triglyceride levels are below 400mg/100ml

VLDL-Cholesterol: VLDL is the primary triglyceride-carrying form in the fasting state, its concentration can be approximated by dividing the amount of plasma triglyceride by 5 (based on the above triglyceride to cholesterol ratio of VLDL).

Age and sex distribution of serum lipids and lipoproteins were calculated with standard deviation. These values were compared using Student's t test (unpaired) to calculate statistical significance.

RESULT: Thirty patients of chronic kidney disease and 30 normal subjects (controls) were taken for present study. Lipid levels like TC, TG, LDL and VLDL were estimated for both controls and CKD patients and were compared.

Age Group	Total no. of cases	Percentage	Male	Percentage	Female	Percentage
<20	2	6.6	1	50	1	50
21-30	8	26.6	4	50	4	50
31-40	5	16.6	5	100	0	0
41-50	5	16.6	4	80	1	20
51 – 60	5	16.6	2	40	3	60
61-70	4	13.3	4	100	0	0
>70	1	3.3	1	100	0	0
Total	30		21		9	

Table 1: Age and sex distribution among CKD patients

In the present study, 30 patients of CKD were included, out of which 21 patients (70%) were male and 9 patients (30%) were females. On decade wise grouping, we found maximum number of patients between 21-30 years (26.6%)

The mean age for the total number of patients was 42.7, the mean age for male patients was 45.3, the mean age for female patients was 36.6, male to female ratio in the study group was 2.3:1

Groups	Blood urea	Serum creatinine
Controls (n=30)	15.7±5.3	0.82±0.33
Patients n=30	135.23±41.64	10.96±4.73
t-value*	14.235	10.681
Significance	<0.001	<0.001

Table 2: Biochemical data in controls and CKD patients. (Mean±SD) mg/dL

*Student's t-test (unpaired) P<0.001 highly significant.

Groups	Controls n=30	Patients n=30	t – value*	Significance
Total cholesterol	185.2±24.51	187±43.5	0.1838	Ns > 0.05
Triglycerides	97±17	174±60.7	6.14	< 0.001 H.S
HDLc	48.8±10.3	36±5.1	5.990	< 0.001 H.S.
LDLc	116.8± 26.78	116.49±38.34	0.03407	N S
VLDLc	19.3±3.49	34.88±12.15	6.194	< 0.001 H.S
HDL/TC	0.28±0.07	0.2002±0.0478	5.003	< 0.001 H.S

Table 3: Biochemical (lipid profile) data in controls and CKD patients (Mean±SD)mg/dL

*Students t-test (unpaired) P< 0.001 highly significant NS not significant P(>0.05).

Total cholesterol value in controls and CKD patients are 185.2±24.51 and 187±43.5 mg/dL respectively (P>0.05). However, this difference was not significant.

Triglyceride values in patients and controls were 174±60.7 and 97±17 mg/dL respectively. Triglycerides values in patients of CKD were significantly high compared to controls and this is statistically highly significant (P<0.001).

HDL values in chronic kidney disease patients are decreased compared to controls, 36±5.1 and 48.8±10.3 mg/dL respectively (P<0.001). This was also statistically highly significant.

VLDL- Significant increase in VLDL were found in CKD patients as compared to controls, 34.88±12.15 and 19.3±3.49 respectively. This was statistically highly significant. (P<0.001).

LDL values were almost similar in both CKD patients and control group, 116.49±38.34 and 116.8±26.78 respectively. This difference was not significant (P<0.001).

HDL/TC-There is significant reduction in HDL/ TC ratio in patients as compared to controls, 0.20±0.053 and 0.28±0.07 respectively. This was statistically highly significant (P<0.001).

Groups	Conservative treatment n=17	Dialysis n=13	t - value	p value
Total cholesterol	192.529±50.17	180.385±32.66	0.7595	N.S
Triglycerides	155.176 ±47	199.01±70	2.052	Sig P<0.05
HDLc	36.1±5.76	35.7±5.20	0.1964	N.S
LDLc	125.38±44.3	104.87±25.10	1.492	N.S
VLDLc	31.27±9.52	39.82±14	1.991	N.S
HDL/TC	0.20±0.053	0.202±0.0405	0.1130	N.S

Table 4: Biochemical data (lipid profile) in chronic kidney disease patients on conservative treatment and haemodialysis. (Mean ± SD)mg/dL

Mean total cholesterol in patients on conservative treatment was 192.529±50.17 mg/dL and for patients on Haemodialysis it was 180.38±32.66 and this difference was statistically not significant (P>0.05).

TG- mean triglycerides in patients of CKD on conservative treatment and haemodialysis group are 155.176±47 and 199.01±70 mg/dL respectively. This difference was statistically significant (P<0.05).

HDLc: Mean HDL value in conservative treatment group and those on haemodialysis are 36.1 ± 5.76 mg/dL and 35.7 ± 5.20 respectively and this difference was statistically not significant.

LDL: Mean LDL value in CKD patients on conservative group and haemodialysis group are 125.38 ± 44.3 mg/dL and 104.87 ± 25.10 respectively and this difference was statistically not significant.

VLDL: Mean VLDL values for conservative and haemodialysis group are 31.27 ± 9.525 mg/dL and 39.82 ± 14 mg/dL respectively and this difference was statistically not significant.

HDL/TC: The difference in mean values of HDL/TC in both conservative treatment and haemodialysis group was also statistically not significant.

Lipid patients mg/dL	Male (n=21) Mean±SD	Female (n=9) Mean±SD	't'	p-value
Total cholesterol	184.47±46.8	193.7±33.57	0.533	N.S
Triglyceride	175.38±55.03	171.44±72.16	0.1637	N.S
HDL	35.3±5.45	37.2±3.94	0.9416	N.S
LDL	114.01±41.72	122.27±28.01	0.5412	N.S
VLDL	35.13±11.03	34.29±14.43	0.1743	N.S

Table 5: Biochemical (lipid profile) data among male and female CKD patients

On comparing mean values of TC, TG, HDL, LDL, VLDL values between male and female patients, there was increase in TG and decrease in HDL in male patients and there was increase in TC and LDL in female patients. However, these differences were statistically not significant ($p > 0.05$).

ECG changes	Total	Percentage	Male	Percentage	Female	Percentage
Normal	19	63.3	12	57.1	7	77.7
LVH	6	20	5	83.3	1	11.1
Hyperkalaemia	5	16.6	4	80	1	11.1
Total	30		21		9	

Table 6: Electrocardiographic Changes

In this table, it is shown that out of 30 patients, 19 patients had normal ECG (63.3%). 6 patients had LVH (20%), 5 male (83.3%) and 1 female patient respectively. 5 patients (16.6%) had hyperkalaemia, 4 male (80%) and 1 female patient (11.1%).

Lipid abnormalities:

Total Cholesterol mg/dL	Male	Percentage	Female	Percentage	Total	Percentage
Desirable <200	14	66.6	6	66.6	20	66.6
Borderline high 200-239	4	19.05	2	22.2	6	20
High >240	3	14.3	1	11.11	4	13.3
Total	21				30	

Table 7: Total cholesterol levels among CKD patients

Table 7 shows difference in total cholesterol values among CKD patients. 20 patients (66.6%) had total cholesterol <200 mg/dL (desirable range) and 10 patients had abnormal value, among them 6 (20%) patients had borderline high (200- 239) and 4 patients had high values (>240) (13.3%).

S. Triglycerides mg/dL	Male	%	Female	%	Total	%
<150	6	28.5	5	55.5	11	36.6
Borderline high (150-199)	9	42.8	01	11.1	10	33.3
High (200-499)	6	28.5	3	33.3	9	30.0
Very high >500	0	0	0	0	0	0
	21		9		30	

Table 8: Serum Triglycerides levels among CKD patients

Table 8 shows difference in serum TG values among CKD patients. 11 patients (36.6%) had normal TG values (<150 mg/dL) 19 patients (33%) had elevated TG values, among them 10 patients (33%) had TG in borderline high (150-199) range and 9 patients (30%) had TG in high (200-499) range. This was statistically significant ($P < 0.05$).

Serum HDL-C HDL-C	Male	%	Female	%	Total	%
<40	18	85.7	7	77.7	25	83.3
41-50	3	14.3	2	22.2	5	16.6
51-60	0	0	0	0	0	0
Total	21		9		30	

Table 9: Serum HDL cholesterol levels among CKD patients

Table 9 shows difference in serum HDL levels among CKD patients.

25 patients (83.3%) had HDL values <40 mg/dL, 5 patients (16.6%) had HDL >40 mg/dL, 83% of patients of study group had HDL < 40 mg/dL and this was statistically significant ($P < 0.001$)

S.LDL-C mg/dL	Male	Percentage	Female	Percentage	Total	%
<100	9	42.8	2	22.2	11	36.66
Near optimal (101-129)	6	28.5	4	44.4	10	33.3
Borderline high (130-159)	3	14.2	2	22.2	5	16.66
High (160-189)	2	9.5	1	11.1	3	10
Very high >190	1	4.7	0	0	1	3.3
Total	21				30	

Table 10: Serum LDL cholesterol levels among CKD patients

11 patients (36.66%) had normal LDL levels (<100), 10 patients (33.33%) had near optimal LDL levels (101-129).

9 patients (29.9%) had elevated LDL.

21 patients (70%) had normal and near optimal LDL levels and only 9 patients (30%) had elevated LDL (>130 mg/dL). This was statistically not significant.

DISCUSSION: The results of the study on the lipid profile in patients with chronic kidney disease show that there are significant alterations in the lipid profiles of these patients as compared to controls.

Triglycerides: In this study, triglycerides were markedly elevated compared to control group and it was statistically highly significant (P values <0.001).

Vaziri et al¹⁶ stated that hypertriglyceridemia is the most common plasma lipid abnormality in patients of chronic kidney disease.

The cause for hypertriglyceridemia in chronic kidney disease patients has not been clearly delineated. Available data derived from kinetic studies have demonstrated that reduced catabolism of triglycerides is the predominant defect due to deficiency of lipoprotein lipase^{16,17,18} or hepatic triglyceride lipase or both.

Reasons for decrease in activity of these enzymes are possibly due to;

- Presence of circulatory inhibitor of lipolytic enzymes in the serum.
- Changes in apoprotein concentrations which can effect lipoprotein lipase activity.
- Insulin resistance seen in renal insufficiency.¹⁶
- Alteration of lipoprotein substrate.¹⁸

Thomas Quaschnig et al¹⁹ reported that combined hyperlipidaemia (elevated cholesterol and triglycerides) with low HDL cholesterol reflects more atherogenic condition.

HDL Cholesterol: There was decrease in HDL cholesterol seen in patients compared to controls, which was statistically significant ($P < 0.001$).

P. O. Attman et al²⁰ found decrease in plasma HDL cholesterol concentration in patients with CKD. It was also reported that decreased HDL was associated with decrease in both the fractional catabolic rate and the total synthetic rate of ApoA1, HDL. The slow fractional catabolic rate of ApoA1 in patients with chronic kidney disease could be a primary event resulting from a decrease in synthesis or secretion of ApoA1.

Total Cholesterol: There was marginal increase of serum total cholesterol in chronic kidney disease patients, compared to controls but this was not statistically significant P (>0.05). P.O. Attman et al²⁰ in their study showed no significant change in levels of total cholesterol.

Thomas Quaschnig et al¹⁹ reported combined hyperlipidaemia (elevated total cholesterol and triglycerides) in their study.

VLDL: There is significant raise in VLDL levels in chronic kidney disease patients compared to controls ($P < 0.001$).

LDL: There was no significant difference in LDL levels in patients as compared to controls. In uraemia LDL lipoproteins are qualitatively altered. Marion Morena et al²¹ reported that there was increase in small, dense LDL sub-fractions in haemodialysis patients. Hypertriglyceridaemia observed in haemodialysis patients results from a reduced lipolysis of TG-rich VLDL that leads to the accumulation of

partially metabolised remnant lipoproteins (IDL and TG-rich LDL). This lipoprotein catabolism impairment is usually associated with reduced levels of HDL affecting reverse cholesterol transport. Such defect in atherogenic lipoprotein catabolism may predispose to the formation of small dense LDL particles, which appear to be more sensitive to ex vivo oxidation.

HDL/TC: There was significant reduction in HDL/TC ratio in patients as compared to controls, this was statistically highly significant, ($P < 0.001$).

The results of comparative study of lipid profile in chronic kidney disease patients on conservative management and haemodialysis: Total cholesterol levels were decreased in patients on haemodialysis (HD) as compared to patients treated by conservative line, but this difference was statistically not significant ($P > 0.05$).

HDL levels were marginally low in patients of HD compared to conservatively treated patients but this was also statistically not significant.

VLDL in HD group was modestly increased compared to conservative group but this was also statistically not significant ($P > 0.05$).

LDL values were modestly low in patient treated with Haemodialysis as compared to patients treated conservatively however this difference was statistically not significant.

There was significant increase in triglycerides in patients treated with haemodialysis compared to patients on conservative treatment ($P < 0.05$). Increased serum triglyceride levels have been well documented by Alam et al.

Morena Marion et al²¹ in their study stated that haemodialysis patients are exposed to several atherogenic factors resulting from qualitative and functional lipid abnormalities, including triglyceride rich particles, increased susceptibility to LDL oxidation and impairment of HDL protective effects.

Comparative study of lipid profiles in chronic kidney disease:

Lipids	Avasthi et al ²² Mean±SD	Present study Mean±SD	Controls Mean ±SD
TC	204±50.54	187±43.5	185±24.5
TG	181±21.8	174± 60.7	97±17

Table 11: Comparative study of lipid profiles in chronic renal failure with previous studies

Avasthi et al²² studied 20 patients of chronic renal failure. During investigation all the patient were placed on conservative treatment for renal failure. They reported mean triglyceride levels of 181±21.87 and total cholesterol of 204±50.54 mg/dL respectively. (Table 11)

Avasthi et al			Present study		
Male	Female	M:F	Male	Female	M: F
13	7	1.8:1	21	9	2.3:1

Table 12: Sex distribution

In study conducted by Avasthi et al male to female ratio was 1.8:1.

Group	S. M. Alam et al			Present study		
	Male	Female	Total	Male	Female	Total
Conservative	13	7	20	11	6	17
Haemodialysis	9	6	15	10	3	13

Table 13

Table 13 shows comparison between conservatively managed group and Haemodialysis group in study conducted by Alam et al with present study.

	John D. Bagdade et al ²³		Present study		
	Uraemia	H.D	Uraemia	H.D	Controls
Lipid					
T C	209±44	196±31	192.5±50.17	180.38±032.66	185.2±24.2
TG	209±91	210±127	155.176±47	199.01±70	97±17
HDLc	35±23	38±18	36.1±5.76	35.7±5.20	48.8±10.3

Table 14: Lipid profile data(mean ±SD)

John D. Bagdade et al²³ Studied lipid profile in 27 patients of CKD, 13 patients not on dialysis, 14 patients on dialysis who had stable chronic uraemia. The results of this study is shown in table 14. Triglycerides were found to be elevated in both non-dialysed and dialysed CKD patients and HDLc was found to be decreased (<40 mg/dL) in both dialysed and non-dialysed groups.

John D. Bagdade et al						Present study					
Conservative group			Haemodialysis group			Conservative group			Haemodialysis Group		
M	F	T	M	F	T	M	F	T	M	F	T
6	7	13	11	3	14	11	6	17	10	3	13

Table 15: Sex distribution

Table 15 shows comparison of number of patients in conservatively managed group and haemodialysis group in Bagdade et al with present.

Lipids	Altaf Basha et al ²⁴		Present study		
	CKD	Controls	Uraemia	H.D	Controls
TC	208±88.5	216.8±39.5	192.5±50.17	180.38±32.66	185.2±24.2
TG	171.08±99.25	108.3±27	155.176±47	199.01±70	97±17

Table 16: Lipid profile data (mean±SD)

Altaf Bash et al²⁴ studied twenty cases of CKD and 10 controls. Results are shown in table 16.

Present study on alterations of lipid profile in CKD was found to be well correlated with studies by previous investigators. In most of the studies, mean values of triglycerides and VLDL were found to be elevated significantly as compared to controls and mean values of HDL were decreased.

CONCLUSION: Significant reduction in HDL and HDL/total cholesterol ratio are the important predictive indices for the risk of developing coronary artery disease in all group of patients with chronic kidney disease. This may be major contributory factor for enhanced atherogenesis in these patients.

Finally, because the lipid abnormalities in chronic kidney disease accelerates the progression of the renal failure and predisposes to atherosclerosis it is worthwhile detecting and treating hyperlipidaemia in chronic kidney disease patients.

BIBLIOGRAPHY:

1. Yamamoto S, Kon V. Mechanisms for increased cardiovascular disease in chronic kidney dysfunction. *Curr Opin Nephrol Hypertens* 2009;18(3):181-188.
2. Van Biesen W, De Bacquer D, Verbeke F, et al. The glomerular filtration rate in an apparently healthy population and its relation with cardiovascular mortality during 10 years. *Eur Heart J* 2007;28(4):478-83.
3. Baigent C, Burbury K, Wheeler D. Premature cardiovascular disease in chronic kidney disease. *Lancet* 2000;356(9224):147-152.
4. Sarnak MJ, Levey AS, Schoolwerth AC, et al. Kidney disease as a risk factor for development of cardiovascular disease: a statement from the American heart association councils on kidney in cardiovascular disease, high blood pressure research, clinical cardiology, and epidemiology and prevention. *Circulation* 2003;108(17):2154-2169.
5. Hallan SI, Coresh J, Astor BC, et al. International comparison of the chronic kidney disease prevalence and ESRD risk. *Jam Soc Nephro* 2006;17(8):2275-84.
6. Chertow GM, Chertow GM, Fan D, et al. Chronic kidney disease and the risks of death, cardiovascular events. *N Engl J Med* 2004;351(13):1296-1305.
7. Van der Zee S, Baber U, Elmariah S, et al. Cardiovascular risk factors in patients with chronic kidney disease. *Nat Rev Cardiol* 2009;6(9):580-589.
8. Tsimihodimos V, Dounousi E. Dyslipidaemia in chronic kidney disease an approach to pathogenesis and treatment. *Am J Nephrol* 2008;28(6):958-973.
9. Kaysen GA. Lipid and lipoprotein metabolism in chronic kidney disease. *J Ren Nutr* 2009;19(1):73-77.
10. Attman PO, Samuelsson O. Dyslipidaemia of kidney disease *Curr Opin Lipidol* 2009;20(4):293-299.
11. Vaziri ND, Moradi H. Mechanisms of dyslipidaemia of chronic kidney disease. *Haemodial Int* 2006;10(1):1-7.
12. Kwan BC, Kronenberg F. Lipoprotein metabolism and lipid management in chronic kidney disease. *J Am Soc Nephrol* 2007;18(4):1246-1261.
13. Aljama P. New insight in ESRD kidney *Int* 2002;80.
14. Karl Skorecki. Chronic kidney disease: In *Harrison's Principles of internal medicine*. McGraw Hill 2010;18th Edn.
15. Prabhakar Reddy E, Suchitra MM, Reddy V, et al. Dyslipidaemia: end Stage Renal Disease and Haemodialysis. *Journal of Nephrology* 2009;5(1):1540-2665.
16. Vaziri ND. Dyslipidaemia of chronic kidney disease: the nature, mechanisms, and consequences. *Am J Physiol Renal Physiol* 2006;290(2):F262-F272.
17. Charlesworth JA, Kriketos AD, Jones JE, et al. Insulin resistance and postprandial triglyceride levels in primary renal disease. *Metabolism* 2005;54(6):821-828.
18. Prinsen BH, de Sain-van der Velden MG, de Koning EJ, et al. Hypertriglyceridemia in patients with chronic kidney disease: possible mechanisms. *Kidney Int Suppl* 2003;84:S121-S124.
19. Quaschnig T, Krane V, Metzger T, et al. Abnormalities in uremic Lipoprotein Metabolism and it's impact on cardiovascular disease. *Am J kidney dis* 2001;38(4 Suppl 1):S14-S19.
20. Attman PO, Samuelsson O, Alaupovic P. Lipoprotein metabolism and renal failure. *Am J Kidney Dis* 1993;21(6):573-592.
21. Marion Morena, Jean-Paul Cristol, thierry Dantoine, et al. Protective effects of high-density lipoprotein against oxidative stress are impaired in haemodialysis patients. *Nephrol Dial Transplant* 2000;15(3):389-393.
22. Ridker PM, Genest J, Boekholdt SM, et al. HDL cholesterol and residual risk of first cardiovascular events after treatment with potent statin therapy: an analysis from the JUPITER trial. *Lancet* 2010;376(9738):333-339.

23. John D Bagdade, Albers JJ. Plasma high-density lipoprotein concentration in chronic haemodialysis and renal transplant patients. *N Engl J* 1977;296:1436-1439.
24. Vasilis Tsimihodimos, Dounousi E, Siamopoulos KC. Dyslipidaemia in chronic kidney disease: an approach to pathogenesis and treatment. *Am J Nephrol* 2008;28(6):958–973.