

# Pattern Of Lipid Profile In Dialysis Naive Chronic Kidney Disease Patients From Ilorin, Nigeria

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## Abstract

**Background:** The burden of CKD is increasing rapidly worldwide and has become a major health issue. Majority of these patients die from CVD before progression to ESRD. Studies have shown that more than 50% of deaths in CKD patients are attributable to cardiovascular events. Lipid disorders are recognized risk factors for CVD and progression of renal diseases of varied aetiologies. There is paucity of information on the prevalence and pattern of lipids in CKD patients in our environment. It is for these reasons that this study was designed to determine serum lipid profile in dialysis naive CKD patients in UITH Ilorin, Nigeria. **Method:** It was a descriptive cross sectional study that involved 120 CKD patients and sixty age and sex matched controls. The patients were recruited consecutively from nephrology clinics and adult medical wards, while controls were sourced from hospital staff and patient's relatives. A venous blood sample was obtained from each patient and control after an overnight fast for lipid profile and creatinine determination. The lipid fractions were analysed using standard methods. Data was analyzed with SPSS version 16. P- value <0.05 was considered significant. **Results:** The mean+ SEM of total cholesterol (10.5+3.6 mmol/l) and triglyceride (3.4+1.5 mmol/l) in the patients were significantly higher when compared with that of the controls (5.4+0.5 mmol/l and 1.7+0.2mmol/l) respectively,  $p<0.05$ . The leading cause of CKD was chronic glomerulonephritis. Similarly, there were significant differences in HDL, LDL and LDL/HDL ratio between the study group and controls (0.6+0.4, 0.9+0.5, and 2.1+2.6 versus 1.2+0.3, 1.4+0.4 and 1.2+0.1 mmol/l) respectively,  $p<0.05$ . However, both study group and control had total cholesterol as the most common dyslipidaemia. **Conclusion:** The study shows that dyslipidaemia is common among our dialysis naïve CKD patients, most especially in those with chronic glomerulonephritis. Our findings underscore the need for early assessment of these patients for lipid abnormalities as prompt treatment may prevent cardiovascular events and retard the progression of kidney disease.

## INTRODUCTION

The prevalence of chronic kidney disease (CKD) is increasing rapidly worldwide, and has become a major health issue<sup>1</sup>. The sensitization and awareness programme of annual World Kidney Day which began in March 2006 has sent a clear message to health care givers and general public on the burden of CKD<sup>2</sup>. In the United States, 9.6% of non-institutionalized adults are estimated to have CKD<sup>3,4</sup>. Studies from Europe, Australia, and Asia alluded to the high prevalence of CKD<sup>5-8</sup>. In Nigeria, the actual prevalence rate of this disease is not known, but hospital based studies show that it accounts for 2-8% of all admissions<sup>9,10</sup>.

Majority of patients with CKD are more likely to die from cardiovascular disease (CVD) rather than develop end stage

kidney failure<sup>11</sup>. Several studies have revealed that more than 50% of all deaths in CKD patients are attributable to cardiovascular diseases<sup>12,13,14</sup>. This underscores the need to look for risk factors associated with it. Lipid disorders are common among CKD patients and are recognized risk factors for CVD in CKD<sup>15,16</sup>. Lipid abnormalities also play pivotal role in the initiation and progression of glomerular and tubulo-interstitial diseases<sup>16-19</sup>.

In Nigeria, studies have shown that dyslipidaemia is common among diabetic and non-diabetic population<sup>20-22</sup>. There is paucity of data on the prevalence and pattern of lipids in CKD patients in our environment. It is for these reasons that this study was designed to determine the pattern of serum lipid profile in dialysis naive CKD patients

in University of Ilorin Teaching Hospital, Ilorin, Nigeria.

## **MATERIAL AND METHODS**

It was a descriptive cross sectional study involving CKD patients, with age and sex matched controls. The control subjects were recruited from healthy hospital staff and patient relatives. Informed consent was obtained from both patients and controls with approval given by the ethical and research committee of our hospital before commencement of the study. The patients were recruited from nephrology clinic and medical wards of the University of Ilorin Teaching Hospital, Ilorin, Nigeria. The inclusion criteria were newly diagnosed and known CKD patients already on conservative management. Exclusion criteria included the following: diabetes mellitus, obesity, significant cigarette smoking and alcohol consumption, use of lipid lowering medication and those on renal replacement therapy.

Information obtained from both the patients and controls include age, height, weight, blood pressure and body mass index calculated using the formula, ( $BMI = Wt (kg)/Ht (m^2)$ ). Weight in kilograms and height in metres were obtained using weighing scale and standiometer respectively. The waist circumference was taken as the midpoint between the ribcage and iliac crest, while the hip circumference was taken as the maximal circumference around the buttocks posteriorly and pubic symphysis anteriorly<sup>23</sup>.

Ten mls of venous blood was drawn aseptically from the antecubital fossa of every patient and control after an overnight fast for lipid profile and creatinine determination. The serum total cholesterol and high density lipoprotein cholesterol (HDL) were analyzed using cholesterol oxidase method, triglyceride assessment was by glycerol kinase method while low density lipoprotein cholesterol (LDL) was obtained using Friedwald formular. Dyslipidaemia was defined using the European Atherosclerosis Society<sup>24</sup> except hypertriglyceridaemia that was defined on the basis of the local value for Nigerians<sup>25</sup> because it differs significantly from the European values. The stages of CKD were determined from calculated GFR, using Cockcroft-Gaunt formular<sup>26</sup>.

Data analysis was done using statistical soft ware SPSS version 16. Comparison of means was done using student t-test and comparison of proportion was by chi-square test. The level of statistical significant was taken as  $p < 0.05$ .

## **RESULTS**

One hundred and eighty individuals were recruited in this

study (120 CKD, 60 controls). Chronic glomerulonephritis (50%) was the leading cause of CKD, followed by hypertension (44.2%), sickle cell disease (3.3%) and adult polycystic kidney disease (2.5%). Table 1 shows the demographic, clinical and laboratory parameters of study subjects. The ratio of patients to controls was 2:1, with mean age of  $41.4 \pm 16.1$  years. The weight of patients were greater than that of the controls ( $63.7 \pm 11.9$  kg versus  $52.9 \pm 10.5$  kg),  $p < 0.05$ . Similarly comparing the body mass index, waist circumference and waist hip ratio between patients and the controls showed significant difference, with  $p < 0.05$ . The mean estimated GFR was significantly lower in patients ( $34 \pm 19.5$  ml/min) than that of the controls ( $87.5 \pm 29.8$  ml/min),  $p < 0.05$ . Both the systolic ( $153.2 \pm 29.7$  mmHg) and diastolic ( $87.2 \pm 14.5$  mmHg) blood pressure in patients were significantly higher when compared to that of the controls ( $116.7 \pm 8.8$  mmHg and  $77.3 \pm 4.5$  mmHg respectively),  $p < 0.05$ .

Table 2 shows the lipid profile pattern in the CKD patients and the controls. The mean of total cholesterol ( $10.5 \pm 3.6$  mmol/L) and triglyceride ( $3.4 \pm 1.5$  mmol/L) were significantly higher when compared with that of controls ( $5.4 \pm 0.5$  mmol/L and  $1.7 \pm 0.2$  mmol/L) respectively,  $p < 0.05$ . Similarly, there were significant differences in HDL, LDL and LDL/HDL ratio between the CKD patients and the controls ( $0.60 \pm 0.4$ ,  $0.9 \pm 0.5$ , and  $2.1 \pm 2.6$  versus  $1.2 \pm 0.3$ ,  $1.4 \pm 0.4$  and  $1.2 \pm 0.1$  mmol/L) respectively,  $p < 0.05$ .

Table 3 shows the prevalence of dyslipidaemia from lipid profile components in the study population. The prevalence of dyslipidaemia was: total cholesterol (90.8%), triglyceride (81.7%) and HDL (75.8%). However in the control group, the commonest dyslipidaemia was still total cholesterol (66.7%), while both triglyceride and HDL were 33.3%.

**Figure 1**

Table 1: Demographic, clinical and laboratory parameters of the study subjects and controls.

Variables	CKD (N = 120)	Controls (N=60)	P – Value
Male	63	32	
Female	57	28	
Age (years)	41.6±16.1	32.5±12.6	0.001
Weight (kg)	63.7±11.9	52.9±10.5	0.001
Waist circumference (cm)	84.3±16.2	74.4±5.1	0.001
Waist-hip ratio	1.1±0.1	0.8±0.1	0.001
BMI (kg/m <sup>2</sup> )	22.2±5.3	18.4±5.1	0.001
Systolic BP (mmHg)	153±29.7	116.7±8.8	0.001
Diastolic BP (mmHg)	87.2±14.5	77.3±4.5	0.001
Creatinine (umol/L)	671.9±80.1	68.8±18.8	0.001
Estimated GFR (ml/min)	34±19.5	99.5±19.5	0.001
Urea (mmol/L)	32.2±2.2	4.4±1.9	0.001
Sodium (mmol/L)	130.4±0.6	137±0.3	0.001
Potassium (mmol/L)	4.00±0.1	3.4±0.2	0.001
Bicarbonate (mmol/L)	20.2±0.3	22.8±0.3	0.001

**Figure 2**

Table 2: Lipid profile pattern in study subjects and controls.

Variables	CKD (N-120)	Control (N-60)	P – Value
Total cholesterol (mmol/L)	10.5±3.6	5.4±0.5	0.001
Triglycerides (mmol/L)	3.4±1.5	1.7±0.2	0.001
HDL (mmol/L)	0.6±0.4	1.2±0.3	0.001
LDL (mmol/L)	0.9±0.5	1.4±0.4	0.001
LDL/HDL ratio	2.1±2.6	1.2±0.1	0.001

**Figure 3**

Table 3: Prevalence of dyslipidaemia in the study subjects and controls.

Variables	CKD (N-120)	Control (N-60)	P – Value
Total cholesterol (>5.2mmol/L)	109 (90.8%)	40(66.7%)	0.001
Triglycerides (> 1.75mmol/L)	98 (81.7%)	20 (33.3%)	0.001
HDL (< 0.9mmol/L)	91 (75.8%)	20 (33.3%)	0.001
LDL (> 3.5mmol/L)	Nil	Nil	

## DISCUSSION

Our study shows that the peak age range was between 20 and 50 years with a percentage of 82%, while those above 50 year accounted for 18%. This is in accord with earlier studies<sup>26,27</sup> in Nigeria that reported the peak incidence of CKD to be between the third and fourth decades. In advanced countries, the prevalence of CKD increases with advancing age<sup>28,29</sup> and the peak incidence is found in 7<sup>th</sup> and 8<sup>th</sup> decades. The reason for disparity in peak age range among CKD patients from developed countries and our study population may be related to genetics, sociocultural factors, access to diagnostic tools, therapeutic modalities and the pattern of diseases causing CKD<sup>30-36</sup>.

A comparative study clearly showed that primary glomerular disease is more common in Blacks than Whites and over 80% of Blacks with CGN are below 40years of age<sup>35</sup>. The relatively young age at which these patients with CKD due to CGN present in tropical developing countries has been reported by several authors<sup>26,27,34-36</sup>. The reason for the foregoing is linked with variety of infective agents implicated in aetiology of CGN, which are present in endemic proportions in the tropics<sup>37-42</sup>.

Patients with CKD were found to have higher BMI as compared to the controls. These observations are in keeping with findings from other studies<sup>43-46</sup>, which showed that weight increase is a risk factor of CKD. The mean values of both systolic and diastolic blood pressure were higher in study subjects as compared to that of the controls. This was not surprising as hypertension was the second leading cause of CKD in this study and is in accord with reports from other studies<sup>47-49</sup>. However, results of studies on end stage renal disease (ESRD) secondary to hypertension should generally be interpreted with caution as it is usually very difficult to tell whether hypertension or insidious renal disease was the primary cause of ESRD. The initial pathological process may elude definition as ESRD is the common end point of long standing renal disease of varied aetiologies. One is often satisfied with a presumptive diagnosis of hypertensive nephrosclerosis in a setting of long standing hypertension, left ventricular hypertrophy, aortic unfolding, hypertensive retinopathy, microscopic haematuria and moderate proteinuria without past history of nephrotic syndrome. The presence of hypertension in these patients could accelerate the development of dyslipidaemia and progression of CKD<sup>50</sup>.

The mean values of urea (32.2±2.2mmol/L), creatinine (671.9±80.1umol/L) and estimated glomerular filtration rate

(eGFR) ( $34 \pm 19.5$  ml/min), shows that majority of our patients presented in advanced stage of the disease. It is pertinent to note that many of our patients with advanced CKD were managed conservatively because they could not afford the cost of renal replacement therapy (RRT) as there is no RRT subsidy in Nigeria. The prognosis of advanced CKD in most Sub-Saharan Africa is still very poor due to late presentation/referral and inability to pay for treatment<sup>51</sup>. Studies from other developing world have shown that most cases of CKD presents in advanced stages as compared to findings from developed countries<sup>26-34</sup>.

Our study, revealed that the mean  $\pm$  SEM of all the lipid profile studied were significantly higher than those of the controls. These findings are in keeping with earlier reports<sup>15,16,46,52-56</sup>. The high prevalence of lipid abnormalities in our study may be related to high incidence of chronic glomerulonephritis in our environment<sup>9,10,35,36</sup>. It underscores the importance of evaluating CKD patients for lipid disorders as experimental evidence supports the hypothesis that lipids contribute directly to glomerulosclerosis and tubulointerstitial injury and that correction of lipid abnormalities associated with renal disease will slow the progression of chronic kidney disease<sup>57,58</sup>. Studies demonstrated strong clinical evidence that both elevation and quality of plasma LDL is associated with atherosclerosis in accelerating CKD<sup>52,53</sup>. The LDL/HDL ratio has been shown to be a better index of coronary artery disease risk than either LDL or HDL alone<sup>53-55</sup>. The ratio was increased in our CKD patients which imposes the burden of atherosclerosis. The observation in this study, that dyslipidaemia occurred in all the components of lipid profile, especially in the total cholesterol (90.8%) is in contrast with earlier reports in which hypertriglyceridaemia was the most common lipid abnormality<sup>16,18,59,60</sup>.

Magnitude of CKD and the scarcity of existing treatment modalities for such patients in our environment which they can hardly afford, means that effort should be geared toward preventive measures and early treatment in order to curtail future ESRD epidemic. An aggressive proactive approach is required in the management of CKD and this will include the control of dyslipidaemia, hypertension, infections, diabetes mellitus, smoking and alcohol consumption. The need to intensify effort on health education and screening of general public for CKD and dyslipidaemia cannot be over emphasized. In conclusion, our study has shown that dyslipidaemia is common among our dialysis naïve CKD patients, most especially in those with chronic

glomerulonephritis. This underscores the need for early assessment of these patients for lipid abnormalities as prompt treatment may prevent cardiovascular events and retard the progression of chronic kidney disease.

## References

1. El Nahas AM, Bello AK. Chronic Kidney disease: The global challenge. *Lancet* 2005; 365:331-340.
2. Levey AS, Andreoli SP, DuBose T et al. Chronic kidney disease: common, harmful and treatable – World Kidney Day 2007. *Am J Kidney Dis.* 2007; 49:175-179.
3. Coresh J, Byrd-Holt D, Astor BC et al. Chronic kidney disease awareness, prevalence, and trends among US adults, 1999 to 2000. *J. Am Soc Nephrol.* 2005; 16: 180-188.
4. Stevens LA, Coresh J, Greene T, Levey AS. Assessing kidney function – measured and estimated glomerular filtration rate. *N Engl J Med* 2006; 354:2473-2483.
5. de Zeeuw D, Hillege HL, de Jung PE. The kidney, a cardiovascular risk marker and a new target for therapy. *Kidney Int. Supple* 2005; 98:25-29.
6. Chen J, Wildman RP, Go D et al. Prevalence of decreased kidney function in Chinese adults aged 35 to 74 years. *Kidney Int.* 2005; 68: 2839-2845.
7. Hallan SI, Coresh J, Astor BC et al. International comparison of the relationship of chronic kidney disease prevalence and ESRD risk. *J Am Soc Nephrol.* 2006; 17:2275-2284.
8. Chadban SJ, Briganti EM, Kerr PG et al. Prevalence of kidney damage in Australian adults: The Aus Diab kidney study. *J. Am Soc Nephrol.* 2003; 14:131-138.
9. Akinsola A, Odesanmi WO, Ogunniyi JO, Ladipo GOA. Diseases causing renal failure in Nigeria. A prospective study of 100 consecutive cases. *African J. Med. Sci.* 1989; 18:131-137.
10. Oyediran AB, Akinkugbe OO. Chronic renal failure in Nigeria. *Trop. Geog. Med.* 1970; 22:41-44.
11. Keith D, Nicholls G, Guillion C et al. Longitudinal follow-up and outcomes among a population with chronic kidney disease in a large managed care organization. *Arch Intern med* 2004; 164: 659-663.
12. ANZ Data Registry Report. Adelaide, South Australia: Australian Kidney Foundation; 1998.
13. Rohm DD. Is atherosclerosis accelerated in haemodialysis patients? *Int. J. Artg. Organs.* 1992; 15:323-326.
14. Charnwy DI, Walton DF, Cheung AK. Atherosclerosis in chronic renal failure. *Curr. Opin. Nephrol. Hyper tens.* 1993; 2:876-882.
15. Massey ZA, Kaminski BH. Hyperlipidaemia and its management in renal disease. *Curr. Opin. Nephrol. Hyper tens.* 1996; 5:141-146.
16. Attman PO, Alaupovic P. Lipid abnormalities in chronic renal insufficiency. *Kid. Intern.* 1991; 39:16-23.
17. Moorhead JF, Chan MK, El-Nahas M, Varghese Z. Lipid nephrotoxicity in chronic progressive glomerular and tubulointerstitial disease. *Lancet* 1982; 1:1309-1311.
18. Keane WF, Mulcahy WS, Kaminski BL, Kim O'Donnell MP. Hyperlipidaemia and progressive renal disease. *Kid. Intern.* 1991; 39:41-48.
19. Moorhead JF. Lipids and progressive Kidney disease. *Kid. Intern.* 1991; 39:35-40.
20. Igwe CU, Duru LA, Ukwamedua H, Kharacha C. Prevalence of Hyperlipidaemia amongst insulin-dependent and non-insulin dependent diabetes mellitus in Delta state Nigeria. *Trop. Doct.* 2007; 37:120-121.
21. Anumah FE, Bakari AG, Bello-Sanni F, Dyslipidaemia

- in persons with type 2 diabetes mellitus in Kaduna. *Int. J diabetes and metabolism* 2007; 15:9-13.
22. Agbola-Abu CF, Onabolu A. Plasma lipid level in patients attending Igbinedion Hospital and Medical Research Centre, Okada, Edo state. *Nigeria. Nig. Med. J.* 2000; 38:1-5.
23. Bray GA. Obesity: Basic consideration and clinical approaches. *Dis. Mon.* 1989; 35:449-537.
24. European Atherosclerosis society. International task force for prevention of coronary heart disease, scientific background and new clinical guidelines. *Nut. Metab. Cardiovascular Dis.* 1992; 2:113-156.
25. Jarikre AE, Ofogba CJ, Emuveyan EE. Reference values for the nutritional indices in urbanized adult Nigerians living in the Lagos area. *Journal of clinical practice.* 1998; 1:22-25.
26. Alebiosu CO, Ayodele OO, Abbas A, Olutoyin IA. Chronic renal failure at the Olabisi Onabanjo University Teaching Hospital, Sagamu, Nigeria. *African Health Sciences* 2006; 6:132-138.
27. Akinsola A, Sanusi AA, Adelekun TA, Arogundade FA. Magnitude of the problem of chronic renal failure in Nigerians. *African Journal of Nephrology* 2004;8:24-26.
28. Feest TG, Mistry CD, Grimes DS, Mallick NP. Incidence of advanced chronic renal failure and the need for end-stage renal replacement treatment. *BMJ* 1990; 301:897-900.
29. McGowan MG Prevalence of advanced renal failure in Northern Ireland. *BMJ* 1990; 301:900-903.
30. Pandreigh DM, Hewitt's LF, MacDougall AI, et al. Survey of chronic renal failure in Scotland. *Lancet* 1972; ii 304-307.
31. Stewart JH, McCarthy SW, Storey BG, et al. Diseases causing end stage renal disease. *N. Eng. J. Med.* 1975; 309:1276-1279
32. Rostand SG, Kirk Rustsky EA, Pale BA. Racial differences in incidence and treatment of ESRD. *N. Eng. J. Med.* 1982; 306: 1276-1279.
33. Perneger TV, Brancati FL, Walton PK, et al. End stage renal disease due to diabetes mellitus. *Ann. Intern. Med.* 1994; 121: 912-919.
34. Hutt, MSR, Wing AJ. Renal failure in the tropics. *Britt Med. Bull.* 1971; 27: 122-127.
35. Chijioke A, Adeniyi AB, End stage renal disease: Racial differences. *Orient J. Med.* 2003; 15 24-31.
36. Adu D, Anim- Addo Y, Foli AK, et al. The nephrotic syndrome in Ghana: Clinical and pathological aspects. *Quart J. Med.* 1981; 50: 297-306.
37. Whittle HC, Abdullahi MT, Fakunle F. Scabies pyodema and nephritis in Zaria, Nigeria. *Trans Roy. Soc. Trop. Med. Hyg.* 1973; 67: 349
38. Hendrikse RG, Adeniyi A, Erdington GM et al. Quartan malaria nephropathy. *Lancet* 1972; i: 1142-1149
39. Andrade ZA, Andrade SG, Scaliguisky M. Renal changes in patients with hepatosplenic schistosomiasis, *Am. J. Trop. Med. Hyg.* 1971; 20: 77-80.
40. Shwe T. Immune complexes in glomeruli of patients with leprosy. *Leprosy review* 1972; 42: 282-289.
41. Ginsburg BE, Wassermann J, Huldt G, et al. A case of glomerulonephritis with acute toxoplasmosis. *Brit Med. J.* 1974; iii: 664-665.
42. Pulley VKG, Kirsch E, Kurtzman NA. Glomerulonephritis associated with filarial loasis. *J. AM. Med. Assoc.* 1973; 225: 179.
43. Bonnet F, Defrele C, Sassolas A, et al. Excessive body weight as a new independent risk factor for clinical and pathological progression in primary IgA nephropathy. *Am J Kidney Dis.* 2001; 37:225-234.
44. Isezuo SA. The Metabolic Syndrome: Review of Current Concepts. *Nigeria postgraduate medical journal* 2003; 3:247-255.
45. Morales E, Valero MA, Leon M, Hernandez E, Prague M. Beneficial effects of weight loss in overweight patients with chronic proteinuric nephropathies. *Am J Kidney Dis.* 2003; 41:319-327
46. Mshelia DS, Buratai LB, Mamza YP. Lipid profile in pre-dialysis chronic kidney disease patients attending University of Maiduguri Teaching Hospital, Nigeria. *Nigerian Journal of Clinical Practice.* 2002; 2:173-178.
47. Bosan IB. Chronic kidney disease in Nigeria: Primary care physicians must intervene earlier. *Nigerian Medical Practitioner* 2006; 49: 18-23
48. Bosan IB. Recommendations for early diagnosis of chronic kidney disease. *Annals of African Medicine* 2007; 16: 130-136.1983;
49. Ojogwu LI, Ana CO. Renal failure and hypertension in tropical Africa – A predialysis experience from Nigeria. *E. Afr. Med. J.*1983; 60: 478-484.
50. Taylor OG, Oyediran OABO, Bamboye AE, Afolabi BM, Osuntokun BO. Profile of some risk factors for coronary heart disease in a developing country: Nigeria. *Afr. J. Med. Med. Sci.* 1996; 25:341-346.
51. Khmer V. End stage renal disease in developing countries. *Am. J. Kid disease* 2002; 62: 350-362.
52. Snidermant A, Cianflone K, Kuitovich PO, Hutchinson T, Barre P, Prichard S. Hyperlipoproteinaemia: the major dyslipidaemia in patients with chronic renal failure treated with chronic ambulatory peritonea dialysis. *Atherosclerosis.* 1987; 65:257-264.
53. Kennedy R, Case C, Fatti R, Johnson D, Isabel N, Marwick TH. Does renal failure cause an Atherosclerotic milieu in patients with end-stage renal disease? *Ann J. Med.* 2001; 110:198-204.
54. Mshelia DS. Role of free radicals in pathogenesis of diabetic nephropathy. *Ann. Afr. Med.* 2004; 3:55-62.
55. Mshelia DS, Pindiga HU. Dyslipidaemia, Lipid oxidation and free radicals in diabetic nephropathy: an overview. *Highland Med Res. J.* 2004; 2:1-7.
56. Mshelia DS, Kadiri S, Osifo BOA. Antioxidant vitamins in patients with chronic glomerulonephritis. *J. Life Environ. Sci.* 2004; 6: 386-390.
57. Keane WF, Kasiske BL, O'Dunnell MP et al. The role of altered lipid metabolism in the progression of renal disease: Experimental evidence. *AM.J.Kid.Dis.*1991;17:38-42
58. Moorhead JF, Wheeler DC, Varghese Z. Glomerular structures and lipids in progressive renal disease. *AM.J.Med.*1989;87(suppl. 5N):12N-20N.
59. Lipinska I, Curewick V. The value of measuring percentage of high-density lipoprotein in assessing risk of cardiovascular disease. *Arch. Intern. Med.* 1982; 142:469-472.
60. Atman P, Alaupovic P, Gustafson A. Serum apolipoprotein profile of patients with chronic renal failure. *Kid. Intern.* 1997; 32:368-375.

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