DOI: https://doi.org/10.53350/pjmhs211593087

ORIGINAL ARTICLE

A Study of Lipid Profile in Chronic Kidney Disease Patients

MUHAMMAD RIZWAN[1], MUHAMMAD TAHA LODHI[2], ASIM MAQSOOD[3], TAHIR MUKHTAR SAYED[4]
[1,2]*House Officer, Shaikh Zayed Hospital, Lahore*
[3]*Senior Registrar Medicine, Avicenna Medical College and Hospital, Lahore*
[4]*Associate Professor of Medicine, Fauji Foundation Medical College, Rawalpindi*
Corresponding author: Muhammad Rizwan, Email: mirzarizwan32@gmail.com, Cell: +0343 0810281

ABSTRACT

Background and Aim: Chronic kidney disease patients are more likely to develop cardiovascular diseases caused by atherosclerosis accelerated rate and variety of other factors, of which they exhibit the abnormality of lipid profile atherogonic characteristics. The current study aim was to investigate the lipid profile abnormalities pattern in non-diabetic chronic kidney disease patients and to evaluate the association between the lipid profile alteration extent and renal impairment degree.

Methods: This cross-sectional study was carried out on 118 chronic disease patients in the Department of Nephrology, Shaikh Zayed Hospital Lahore during the period, from August 2020 to May 2021. All the patients were carefully chosen based on their eligibility criteria. A history was taken, clinical investigation was performed, and biochemical tests were conducted. Blood was drawn for lipid profile analysis after 9 hours abstaining. The Institutional Ethical Committee approved the study and informed consent was taken from each individual. Chronic Kidney Disease as defined by the KDOQI Criteria Kidney damage for three months, defined as functional kidney abnormalities with or without decreased GFR, manifested by either: Pathological abnormalities; kidney damage markers such as changes in blood or urine composition, or abnormalities in imaging tests. GFR of less than 60 mL/min/1.73m2 for 3 months, with or without kidney damage. SPSS version 20 was used for data analysis.

Results: The study included 118 patients, 79 (66.9%) of whom were males and 39 (33.1%) were females. The mean age of patients was 49.46 + 9.35 years with an age range of 28 to 78 years. Stage 5CKD patients were 23 who underwent dialysis. Chronic kidney disease patients had lower HDL and higher levels of triglyceride whereas, with chronic kidney disease stage progression, the HDL and TGL levels increased. In both stages 4 and 5 CKD, there is a positive correlation between triglyceride levels and serum phosphorous and TGL and calcium had inverse correlation. Dialysis patients' lipid profiles do not differ from those of non-dialysis patients.

Conclusion: Our study found that patients with non-diabetic CKD have high triglyceride levels, low HDL, and had LDL and total cholesterol unchanged levels. As the CKD stage advances and GFR declines, triglyceride increases, and HDL decreases. TGL and serum phosphorous had a positive association in chronic kidney disease stage 4 and 5 whereas TGL and serum calcium had an inverse correlation in stages 4 and 5. Moreover, no significant difference between dialysed and non-dialysed chronic kidney disease patients' lipid profiles was observed.

Keywords: Chronic Kidney Disease; Lipid Profile; Lipid Profile Abnormalities

INTRODUCTION

Chronic kidney disease patients are more likely to develop cardiovascular diseases caused by atherosclerosis accelerated rate and variety of other factors, of which they exhibit the abnormality of lipid profile atherogonic characteristics [1]. Cardiovascular disease is a leading cause of increased mortality rate among chronic kidney disease (CKD) patients. The incidence of dyslipidemia is higher in CKD patients compared to the general population. Cardiovascular disease is a frequent risk factor for CKD patient mortality in the majority of cases compared to renal stage disease. All the patients should be assessed for dyslipidemias as per guidelines of KDOQI (Kidney Disease Outcomes Quality Initiative) [2]. For risk identification and treatment, triglycerides, LDL, high-density lipoprotein cholesterol, and complete lipid profile with fasting should be determined. The ideal values of triglyceride sand LDL is <150 and <100 respectively. The risk for cardiovascular events can be reduced with the initiation of hostile therapeutic intervention and highly risky categories should be assigned to CKD patients. Kidney disease progression rate was found accelerated due to hypercholesterolemia while studying the cardiovascular studies on animal models variety [3].

Secondary dyslipidemia was experienced in chronic kidney disease patients that resemble atherogenic dyslipidemia seen in insulin-resistant patients. This is distinguished by small dense LDL particles, elevated VLDL, lower cholesterol (HDL), and elevated serum triglycerides. A significant atherogenic potential characterized these particles with triglyceride-rich Apolipoprotein B comprised of complex lipoproteins [4]. The proportion of increased cholesterol values (>240 mg/dl) in nephrotic syndrome CKD, general population, and CKD without nephrotic syndrome were 90%, 20%, and 30% respectively [5-7]. The prevalence of elevated -triglycerides (>200 mg/dl) in nephrotic syndrome CKD, general population, and CKD without nephrotic syndrome were 60%, 15%, and 40%respectively [8, 9]. CKD patients generally have lower concentrations of plasma HDL cholesterol than non-uremic individuals; additionally, the HDL sub-fractions distribution diverges. The esterification of free cholesterol and thus the conversion of HDL3 to HDL2 are reduced in uremia due to the low apo-AIV level and decreased lecithin cholesterol acyltransferase activity [10].

Hypercholesterolemia is more common in patients receiving continuous ambulatory peritoneal dialysis than in those receiving hemodialysis, most likely due to glucose absorption from the dialysate and peritoneal protein loss of

5-15 g/day [11]. Hypertriglyceridemia is the most common dyslipidemia in chronic kidney disease. Triglyceride clearance is reduced, which can lead to hypertriglyceridemia, due to changes in the composition of circulating triglycerides (which become enriched with apolipoprotein C-III) and, possibly later, reductions in the activity of lipoprotein lipase and hepatic triglyceride lipase, which are involved in triglyceride clearance. The hypertriglyceridemia alternate possible mechanism is the retention of blood inhibitor lipoprotein lipase in chronic kidney disease patients such as pre-beta-high density lipoprotein (HDL) [12].

MATERIALS AND METHODS

This cross-sectional study was carried out on 118 chronic disease patients in the department of Nephrology Shaikh Zayed Hospital, Lahore during the period, from August 2020 to May 2021. All the patients were carefully chosen based on their eligibility criteria. The history was taken, clinical investigation was performed, and biochemical tests were conducted. Blood was drawn for lipid profile analysis after 9 hours abstaining. The Institutional Ethical Committee approved the study and informed consent was taken from each individual. Chronic Kidney Disease as defined by the KDOQI Criteria Kidney damage for three months, defined as functional kidney abnormalities with or without decreased GFR, manifested by either: Pathological abnormalities; kidney damage markers such as changes in blood or urine composition, or abnormalities in imaging tests. GFR of less than 60 mL/min/1.73m2 for 3 months, with or without kidney damage.

The various stages of chronic kidney disease as per (KDOQI) are as follows; Stage 0, 1, 2, 3, 4, and 5 had GRF >90(With Risk Factors For CKD), >90(With Demonstrated Kidney Damage), 60-89, 30-59, 15-29, and <15 respectively. Chronic kidney disease was diagnosed in our study using clinical examination, serological abnormalities, and biochemical analysis. All the patients who met the inclusion criteria of chronic kidney disease were enrolled whereas patients of chronic liver disease, diabetes, proteinuria, hypolipidemic drugs users, and hypothyroidism were excluded. A detailed history and clinical examination were conducted, as well as routine haematological and biochemical investigations such as total leukocyte count, ESR, hemoglobin, platelet, differential count, blood urea, serum calcium, serum creatinine, magnesium, phosphorous, and uric acid. USG scanning was utilized for renal echo texture and size. Data analysis was done using SPSS version 20. Quantitative variables such as age, lipid profile values were expressed as frequency and percentage. Lipid profile association with biochemical parameters and clinical features were analyzed.

RESULTS

The study included 118 patients, 79 (66.9%) of whom were males and 39 (33.1%) were females. The mean age of patients was 49.46 + 9.35 years with an age range of 28 to 78 years. Stage 5CKD patients were 23 who underwent dialysis. Chronic kidney disease patients had lower HDL and higher levels of triglyceride whereas, with chronic kidney disease stage progression, the HDL and TGL levels increased. In both stages 4and 5 CKD, there is a positive correlation between triglyceride levels and serum phosphorous and TGL and calcium had inverse correlation. Dialysis patients' lipid profiles do not differ from those of non-dialysis patients. Figure-1 demonstrate gender distribution among 118 CKD patients. The correlation of GFR and TGL, GFR and HDL, and GFR and LDL are shown in Table 1, 2, and 3 respectively. Correlation between HDL stage group and HDL and CKD group stage as shown in Figure-2. The prevalence of dialysis and non-dialysis CKD patients are illustrated in Figure-3.

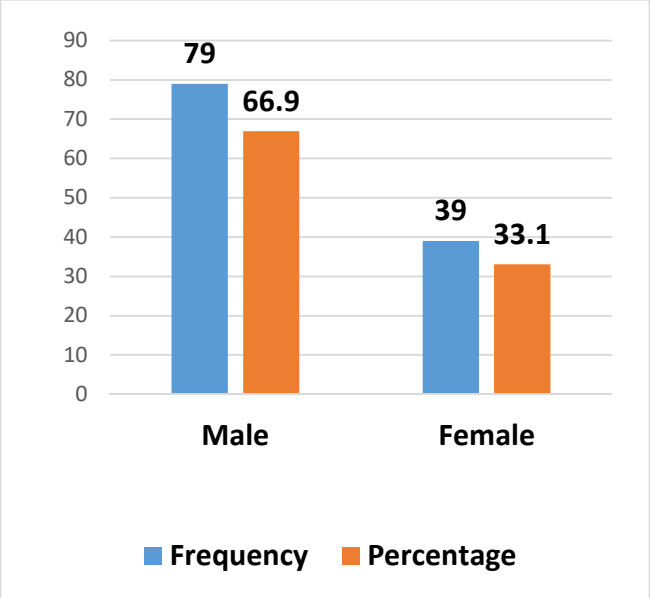

Figure-1 Gender distribution (n=118)

Table-1 Association between GRF and TGL (non-dialysis group n=95)

		GFR	TGL
GFR	Pearson Correlation Sig. (2-tailed) N	1 95	0.19 0.39 95
TGL	Pearson Correlation Sig. (2-tailed) N	0.19 0.39 95	 95

Table-2 Association between GRF and HDL (non-dialysis group n=95)

		GFR	HDL
GFR	Pearson Correlation Sig. (2-tailed) N	1 95	0.56 0.00 95
HDL	Pearson Correlation Sig. (2-tailed) N	0.56 0.00 95	 95

Table-3 Association between GRF and LDL (non-dialysis group n=95)

		GFR	LDL
GFR	Pearson Correlation Sig. (2-tailed) N	1 95	0.031 0.74 95
LDL	Pearson Correlation Sig. (2-tailed) N	0.031 0.74 95	 95

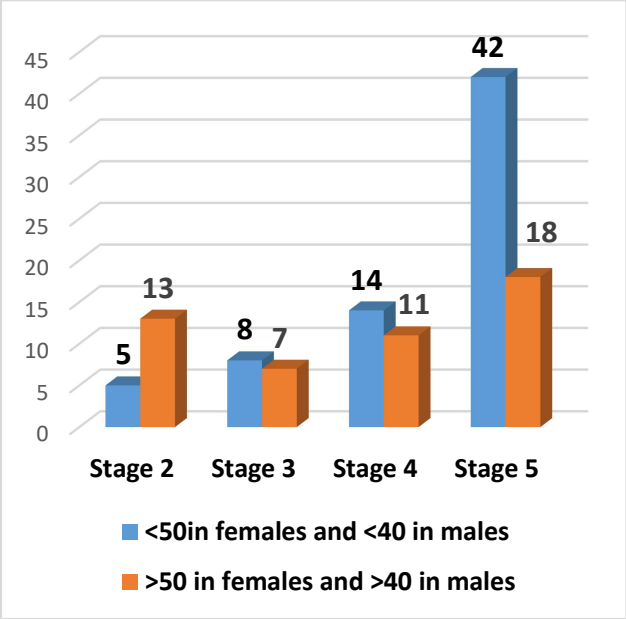

Figure-2 Correlation between HDL stage group and HDL and CKD group stage

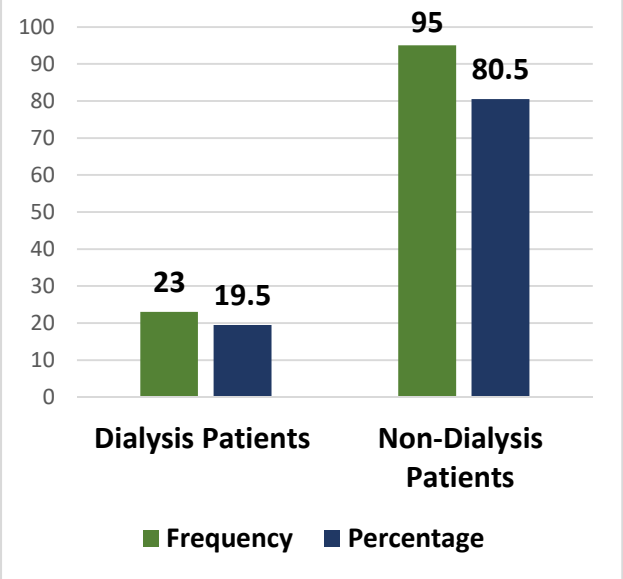

Figure-3 Prevalence of Dialysis and Non-Dialysis Patients (n=118)

DISCUSSION

We investigated the serum lipid profile in CKD patients in the present study. The study included 118 patients, of which 79 (66.9%) were males and 39 (33.1%) were females. The mean age of patients was 49.46 + 9.35 years with an age range of 28 to 78 years. Stage 5CKD patients were 23 who underwent dialysis. A significant inverse relation between GFR and levels of serum triglyceride was found among non-dialysis patients. With the increasing CKD stage, higher values of glyceride were observed in a growing number of patients, nonetheless, this is statistically insignificant. However, in CKD stage 5 growing number of patients had abnormal triglyceride when equated with patients of non-stage 5 which was statistically significant with a 0.026 level of significance. In our study, we found that CKD patients with the nephrotic syndrome had higher triglyceride levels (>150) which resemble another study conducted by Rogacev et al. [13] reported that 3.40% of patients had TGL >200.

Pavlakou et al. [14] found the incidence of hyper triglycerideamia among the Indian population was 29.5%. According to Kasper et al [15] and Pandya et al [16], hypertriglyceridemia is the utmost common abnormality of lipid profile detected in CKD patients. Previous studies on CKD patients' lipid profiles evaluated the individual lipid abnormalities pervasiveness rather than the dyslipidaemia overall prevalence among population. However, this occurrence is high when associated with the pediatrics CKD population having inclusive occurrence of 45%. [17]. The latter study's lower prevalence might be due to dyslipidaemia definition, as well as the study was carried out in a pediatrics age patients [18].

Female CKD patient's had significantly higher dyslipidaemia than male subjects. The dyslipidaemia dominancy in CKD female patients might be caused by low HDL <50mg/dl cutoff compared to male <40mg/dl in males. Furthermore, oestrogen to be defensive against dyslipidaemia by HDL-C levels increase in females, is typically low CKD female patients [19]. In contrast to previous studies [20, 21], chronic kidney disease and dyslipidemia had a significant association because there was a significant increase in the occurrence of TG with deteriorating GFR and abridged HDL-C, so dyslipidemia primary assessment and management should be encouraged in CKD patients to impede the progression and prevention of ESRD and cardiovascular disease.

The cardiovascular events could be shown in patients sensitive AIP than other ratios [22]. It is a better predictor of cardiovascular risk than previously used lipid parameters because of HDL-C fractional esterification rate determinant [23]. The high AIP occurrence CKD patients was 64.8%, which was considerably advanced than the control group's 33.8%. AIP is a strong predictor of myocardial infarction [24]. Extraordinary AIP was more common than distinct lipid abnormalities. This emphasizes the importance of atherogenic risk evaluating among CKD patients utilizing various lipid constituents rather than seeing in isolation [25].

Statin utilization has been shown to decrease cardiovascular risk and slow the CKD progression [26, 27]. According to Lee Y et al, [28] weight loss significantly reduces blood pressure and various lipid constituents as an atherogenic risk factors [29] so consistent workout and lifestyle changes intended will also supplement the lipid-dropping prescriptions impacts. Dyslipidaemia is a risk factor for common cardiovascular in CKD patients, particularly females and the elderly. Low HDL-C, high TG, and atherogenic risk, tend to worsen as renal function deteriorates and lipid abnormalities. Overall, there is no abnormalities difference in lipid profile between dialyzed and non-dialyzed patients. Latiwesh al [30] and Musso et al [31] show that the liquid profile abnormalities of dialyzed and non-dialyzed patients are nearly identical. According to Wheeler D.C, renal replacement therapy does not correct abnormalities in lipoprotein metabolism that are associated with renal failure.

CONCLUSION

Our study found that patients with non-diabetic CKD have high triglyceride levels, low HDL, and had LDL and total cholesterol unchanged levels. As the CKD stage advances and GFR declines, triglyceride increases, and HDL decreases. TGL and serum phosphorous had a positive association in chronic kidney disease stage 4 and 5 whereas TGL and serum calcium had an inverse correlation in stages 4 and 5. Moreover, no significant difference between dialysed and non-dialysed chronic kidney disease patients' lipid profiles was observed.

REFERNCES

1. Noce A, Bocedi A, Campo M, Marrone G, Di Lauro M, Cattani G, Di Daniele N, Romani A. A pilot study of a natural food supplement as new possible therapeutic approach in chronic kidney disease patients. Pharmaceuticals. 2020 Jul;13(7):148.
2. Zheng HJ, Guo J, Wang Q, Wang L, Wang Y, Zhang F, Huang WJ, Zhang W, Liu WJ, Wang Y. Probiotics, prebiotics, and synbiotics for the improvement of metabolic profiles in patients with chronic kidney disease: A systematic review and meta-analysis of randomized controlled trials. Critical reviews in food science and nutrition. 2021 Feb 21;61(4):577-98.
3. Peng TC, Wang CC, Kao TW, Chan JY, Yang YH, Chang YW, et al: Relationship between hyperuricemia and lipid profiles in US adults. Biomed Res Int 2015; 2015: 127596.
4. Kuwabara M, Borghi C, Cicero AF, Hisatome I, Niwa K, Ohno M, Johnson RJ, Lanaspa MA: Elevated serum uric acid increases risks for developing high LDL cholesterol and hypertriglyceridemia: a five-year cohort study in Japan. Int J Cardiol 2018;pii:S0167-5273(17)37927-5.
5. Stone NJ, Robinson JG, Lichtenstein AH, Bairey Merz CN, Blum CB, Eckel RH, et al: 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am College Cardiol 2014; 63(25 Pt B):2889–2934.
6. Kuwabara M, Borghi C, Cicero AF, Hisatome I, Niwa K, Ohno M, Johnson RJ, Lanaspa MA: Elevated serum uric acid increases risks for developing high LDL cholesterol and hypertriglyceridemia: a five-year cohort study in Japan. Int J Cardiol 2018;pii:S0167-5273(17)37927-5.
7. Stone NJ, Robinson JG, Lichtenstein AH, Bairey Merz CN, Blum CB, Eckel RH, et al: 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am College Cardiol 2014; 63(25 Pt B):2889–2934.
8. Kuwabara M, Borghi C, Cicero AF, Hisatome I, Niwa K, Ohno M, Johnson RJ, Lanaspa MA: Elevated serum uric acid increases risks for developing high LDL cholesterol and hypertriglyceridemia: a five-year cohort study in Japan. Int J Cardiol 2018;pii:S0167-5273(17)37927-5.
9. Stone NJ, Robinson JG, Lichtenstein AH, Bairey Merz CN, Blum CB, Eckel RH, et al: 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am College Cardiol 2014; 63(25 Pt B):2889–2934.
10. Abujrad H, Mayne J, Ruzicka M, Cousins M, Raymond A, Cheesman J, et al: Chronic kidney disease on hemodialysis is associated with decreased serum PCSK9 levels. Atherosclerosis 2014; 233: 123–129.
11. Rogacev KS, Heine GH, Silbernagel G, Kleber ME, Seiler S, Emrich I, et al: PCSK9 plasma concentrations are independent of GFR and do not predict cardiovascular events in patients with decreased GFR. PLoS One 2016; 11:e0146920.
12. Pavlakou P, Liberopoulos E, Dounousi E, Elisaf M: PCSK9 in chronic kidney disease. Int Urol Nephrol 2017; 49: 1015–1024.
13. Rogacev KS, Heine GH, Silbernagel G, Kleber ME, Seiler S, Emrich I, et al: PCSK9 plasma concentrations are independent of GFR and do not predict cardiovascular events in patients with decreased GFR. PLoS One 2016; 11:e0146920.
14. Pavlakou P, Liberopoulos E, Dounousi E, Elisaf M: PCSK9 in chronic kidney disease. Int Urol Nephrol 2017; 49: 1015–1024.
15. Kasper DL, Fauci A, Hauser SL, Longo DL, Jameson JL, Loscalzo J. Harrisons Principles of Internal Medicine. 19th ed. USA: McGraw-Hill Education; 2015:1813.
16. Pandya V, Rao A, Chaudhary K. Lipid abnormalities in kidney disease and management strategies. World J Nephrol 2015;4:83-912.
17. Jha V, Garcia-Garcia G, Iseki K, Li Z, Naicker S, Plattner B, et al. Chronic kidney disease: Global dimension and perspectives. Lancet 2013;382:260-72.
18. Sun K, Lin D, Li F, et al.: Fatty liver index, albuminuria and the association with chronic kidney disease: a population-based study in China. BMJ Open. 2018, 8:1-9. 10.1136/bmjopen-2017-019097
19. Singh S, Pathak AK, Parappanavar NU: A study of fasting lipid profile in chronic kidney disease patients. Int J Res Med Sci. 2019, 7:2282-2285. 10.18203/2320-6012.ijrms20192513
20. Orlić L, Mikolasevic I, Bagic Z, Racki S, Stimac D, Milic S: Chronic kidney disease and nonalcoholic fatty liver disease-is there a link?. Gastroenterol Res Pract. 2014, 2014:1-6. 10.1155/2014/847539
21. Sun K, Lin D, Li F, et al.: Fatty liver index, albuminuria and the association with chronic kidney disease: a population-based study in China. BMJ Open. 2018, 8:1-9. 10.1136/bmjopen-2017-019097
22. Singh S, Pathak AK, Parappanavar NU: A study of fasting lipid profile in chronic kidney disease patients. Int J Res Med Sci. 2019, 7:2282-2285. 10.18203/2320-6012.ijrms20192513
23. Gluba-Brzozka A, Franczyk B, Rysz J: Cholesterol disturbances and the role of proper nutrition in CKD patients. Nutrients. 2019, 11:1-30. 10.3390/nu11112820
24. Florens N, Calzada C, Lyasko E, Juillard L, Soulage CO: Modified lipids and lipoproteins in chronic kidney disease: a new class of uremic toxins. Toxins. 2016, 8:1-27. 10.3390/toxins8120376
25. Bulbul MC, Dagel T, Afsar B, et al.: Disorders of lipid metabolism in chronic kidney disease. Blood Purif. 2018, 46:144-152. 10.1159/000488816
26. Mikolasevic I, Milic S, Wensveen TT, et al.: Nonalcoholic fatty liver disease-a multisystem disease?. World J Gastroenterol. 2016, 22:9488-9505. 10.3748/wjg.v22.i43.9488
27. Marcuccilli M, Chonchol M: NAFLD and chronic kidney disease. Int J Mol Sci. 2016, 17:1-15. 10.3390/ijms17040562
28. Lee Y, Park S, Lee S, et al.: Lipid profiles and risk of major adverse cardiovascular events in CKD and diabetes: a nationwide population-based study. PLoS ONE. 2020, 15:1-14. 10.1371/journal.pone.0231328
29. Jang HR, Kang D, Sinn DH, et al.: Nonalcoholic fatty liver disease accelerates kidney function decline in patients with chronic kidney disease: a cohort study. Sci Rep. 2018, 8:1-9. 10.1038/s41598-018-23014-0
30. Latiwesh OB, Younis MYG, Shakila S, et al.: Hepatic enzymes changes in chronic kidney disease patients- a need for modified reference values. J Evolution Med Dent Sci. 2018, 7:1949-1954. 10.14260/jemds/2018/439
31. Musso G, Cassader M, Cohney S, et al.: Fatty liver and chronic kidney disease: novel mechanistic insights and therapeutic opportunities. Diabetes Care. 2016, 39:1830-1845. 10.2337/dc15-1182