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Original Research Article

Study of lipid profile in diabetic and non-diabetic chronic kidney disease patients on haemodialysis: a prospective comparative study from a sub Himalayan region in North India

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ABSTRACT

Background: Diabetes mellitus (DM) is a major cause of chronic kidney disease (CKD) leading to diabetic kidney disease (DKD). Several studies have observed lipid profile abnormalities in non-diabetic CKD patients with and without haemodialysis. Our study aims to reveal lipid profile abnormalities both in DKD and non-diabetic CKD patients on haemodialysis.
Methods: A prospective comparative study included 50 DKD and 50 non-diabetic CKD patients on haemodialysis and 50 controls after fulfilling the inclusion and exclusion criteria. The demographic and biochemical, including lipid profile parameters data of all subjects was collected and statistically analysed. $p<0.05$ was considered as statically significant.
Results: A total of 100 study patients, 50 DKD and 50 non-diabetic CKD patients, both on haemodialysis revealed significant dyslipidaemia when compared to controls. Total cholesterol in DKD patients on haemodialysis when compared to controls (177.5±80.5 versus 146.5±31.8 mg/dl) was significantly elevated (p=0.01). Low density lipoprotein (LDL) in DKD patients when compared to controls (94.1±43.3 versus 76.3±26.3 mg/dl) was also significantly elevated (p=0.01). Triglyceride levels in both DKD and non-diabetic CKD patients on haemodialysis in comparison to controls (213.8±182.1 and 169.2±132.3 versus 109.2±28.9 mg/dl respectively) were significantly elevated (p=0.0002 and p=0.003 respectively). Similarly, very low-density lipoprotein (VLDL) levels in both DKD and non-diabetic CKD patients were also significantly elevated when compared to controls (p=0.002 and p=0.003 respectively) whereas high density lipoprotein (HDL) was significantly reduced.
Conclusion: Both DKD and non-diabetic CKD patients on haemodialysis revealed significant dyslipidaemia, a major cause of increased risk for cardiovascular diseases necessitating early treatment with statins.

Keywords: Chronic kidney disease, Dyslipidaemia, Haemodialysis, Lipid profile, Cardiovascular diseases

INTRODUCTION

Chronic kidney disease (CKD) is becoming a major health problem worldwide leading to increased morbidity and mortality. Dyslipidemia is associated with progression of CKD leading to increased risk of cardiovascular diseases (CVD) and mortality.[1] Functional unit of kidney is the nephron comprising of glomerulus and Bowman's capsule mainly involved in excretion of waste products of metabolism of human body.[2,3] The Kidney disease outcome quality initiative (K/DOQI) of the National kidney foundation (NKF) defines CKD as either kidney damage or a decreased glomerular filtration rate (eGFR) <60 ml/min/1.73 m^2 for 3 or more months.[4] Decreasing eGFR defines CKD stages (1 to 5) leading to End stage renal disease (ESRD) or Chronic renal failure (CRF) with

a level of eGFR <15 ml/min.[5,6] Different types of etiological profiles involved in pathogenesis lead to development of uremic state in CKD.[7,8] Diabetic kidney disease (DKD) develops in approximately 40% of diabetic patients and is a leading cause of CKD worldwide.[9] DKD also termed as CKD due to diabetes, both type 1 and 2 is defined as persisting severely elevated albuminuria of >300 mg/ 24 hours (or >200 µg/min) or an urinary albumin to creatinine ratio (ACR) of >300 mg/gm with concurrent presence of diabetic retinopathy and absence of signs of other form of renal disease.[10] National cholesterol education programme (NCEP) defines dyslipidemia as hypercholesterolemia ≥200 mg/dl of serum total cholesterol (TC), hypertriglyceridemia ≥150 mg/dl of serum triglyceride (TG), reduced high density lipoprotein cholesterol (HDL-C) <40 mg/dl for men and <50 mg/dl for women, elevated low density lipoprotein cholesterol (LDL-C) ≥130 mg/dl and high TC to HDL-C ratio of ≥4.5.[11] Normal plasma lipids and lipoprotein metabolism is critical for cellular cholesterol homeostasis and protection against atherosclerosis, renal disease and other complication.[12] ESRD results in dysregulation of several key enzymes and receptors involved in metabolism of lipoproteins particularly HDL-C and TG rich lipoproteins in CKD and DKD patients.[13,14] The major changes include decrease in lecithin cholesterol acyl-transferase (LCAT), lipoprotein lipase (LPL), hepatic lipase, acyl-coA diacylglycerol acyl-transferase (DGAT) and LDL receptor-related protein (LRP), and increase in cholesterol ester transfer protein (CETP) and acyl-CoA cholesterol acyl-transferase (ACAT). Studies have reported significant dyslipidemias in non-diabetic CKD patients on hemodialysis.[15-17] Rifai et al reported elevated TG, LDL-C, TC to HDL-C ratio and reduced HDL-C in CKD patients on hemodialysis in comparison to controls.[15] Mekki et al found that intermittent and long term dialysis fail to correct dyslipidemia generated by CRF.[16] A study from India observed hypertriglyceridemia, elevated VLDL-C and lipoprotein-a and decreased HDL-C levels.[17] There is increased lipid peroxidation and oxidative stress due to increased reactive oxygen species (ROS) and decreased superoxide dismutase (SOD) in CKD patients on hemodialysis.[17,18] Ashish et al found significantly higher dyslipidemia in peritoneal dialysis (PD) than hemodialysis.[19] Herzog et al found poor survival in acute myocardial infarction(MI) and Karnik et al observed cardiac arrest and sudden cardiac death in CKD patients on hemodialysis.[20,21] Ganta et al reported that dyslipidemia in CKD accelerates the progression of cardiovascular disease and increased mortality.[22] Both traditional and nontraditional risk factors and hypertriglyceridemia as an independent risk factor predict coronary heart disease in CKD.[23,24] To reduce cardiovascular mortality due to dyslipidemia in CKD patients, early management with statins has been recommended by several studies.[25-27] Rhee et al recommended to follow guidelines and risk assessment before start of treatment of dyslipidemia.[28] Several studies have observed significant dyslipidemias as a major risk factor for CVD in non-diabetic CKD patients on hemodialysis and on conservative therapy. Our study aims to observe dyslipidemia both in DKD and non-diabetic CKD patients on hemodialysis with higher incidence and prevalence, especially in DKD.

METHODS

The study was conducted from June 2019 to May 2020 over a period of one year in a regional hospital catering CKD patient with high incidence and prevalence in sub Himalayan region of North India. A total of 100 study patients included 50 diabetic and 50 non-diabetic CKD patients on hemodialysis and were compared to 50 healthy individuals (controls).

Inclusion criteria

The inclusion criteria for the study was as follows: patients aged 18 years and above of both sexes; patients fulfilling the criteria of having diabetes mellitus both type 1 and type 2, albuminuria of >300 mg/ 24 hours or 200 µg/min or ACR of >300 mg/gm with concurrent presence of diabetic retinopathy with absence of signs of other form of renal disease and/or radiologically enlarged kidneys, allocated to diabetic CKD patients; patients of CKD without evidence of diabetes, allocated to non-diabetic CKD patients; both diabetic and non-diabetic CKD patients of ESRD with glomerular filtration rate (eGFR of <15 ml/min) on hemodialysis for a duration of more than 6 months and; age and sex matched to study group, the healthy individuals having normal renal function, allocated to controls, were included in the study.

Exclusion criteria

The exclusion criteria for the study was as follows: patients aged <18 years; patients taking statins, seriously ill patients and; non-consenting patients and controls were excluded from the study.

Approval from Institutional Ethical Committee was obtained. All eligible study patients and controls were thoroughly investigated and lipid profile including TC, TG, HDL-C, LDL-C and VLDL-C were done by enzymatic method using Merilyser auto quat 100 machine. NCEP guidelines were used for definition of dyslipidemia as having TC levels ≥200 mg/dl, TG levels ≥150 mg/dl HDL-C <40 mg/dl for men and <50 mg/dl for women and LDL-C ≥130 mg/dl.

Statistical analysis

The data was entered in excel sheet and analyzed statistically using Epi-info version 7. Chi square (χ^2) test for comparison of categorical variables and student 't' test for comparison of means were used. For all comparison, of $p<0.05$ was considered as statistically significant.

RESULTS

A total of 100 study patients, 50 DKD and 50 non-diabetic CKD patients revealed mean age of 59.4±10.0 and 45.4±13.4 years with male to female ratio of 2.3:1 and

1.9:1 respectively as compared to 50 controls revealing mean age of 54.80±13.3 years with male to female ratio of 1.1:1. Both DKD and non-diabetic CKD patients on hemodialysis had ESRD (eGFR<15 ml/min) revealing significantly increased blood urea nitrogen (BUN) and serum creatine in comparison to controls (Table 1).

Table 1: Demographic and diagnostic parameters of diabetic and non-diabetic CKD patients on haemodialysis and controls.

Parameters		Diabetic CKD (n=50)	Non diabetic CKD (n=50)	Control (n=50)	p value	
Age (years)	Male	59.7±10.7	44.3±13.6	55.5±14.2	0.21	0.003**
	Female	58.5±8.3	47.7±12.6	53.9±12.0	0.18	0.13
	Total	59.4±10.0	45.4±13.4	54.80±13.3	0.06	0.0008**
Sex N (%)	Male	35 (70%)	33 (66%)	27 (54%)	0.15	0.16
	Female	15 (30%)	17 (34%)	23 (46%)	0.15	0.16
Blood urea nitrogen (mg/dl)		83.46±36.3	76.4±37.6	20.8±4.3	0.002*	0.01*
Serum creatine (mg/dl)		8.7±3.1	11.5±10.6	0.7±0.09	<0.001*	<0.001*
Fasting blood sugar (mg/dl)		146.26±23.74	99±16.29	93.76±13.36	0.0001*	0.08
HBA1C (%)		7.28±1.20	5.47±0.52	5.48±0.85	2.3	1.8
Albumin creatinine ratio (mg/gm)		2469.3±1809.8	139.2±94.3	-	0.00001†	
Diabetic retinopathy		+	-	-	-	
Duration of diabetes (years)		10.24±4.97	-	-	-	

p<0.05; *significant (diabetic CKD versus controls), **significant (non-diabetic CKD vs controls) and †significant (diabetic CKD versus non-diabetic CKD). CKD; chronic kidney disease.

Table 2: Serum lipid profile (TC, TG, HDL, LDL, VLDL) in diabetic and non-diabetic CKD patients on hemodialysis and controls.

Group	TC (mg/dl)	TG (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	VLDL (mg/dl)
Diabetic CKD (n=50)	177.5±80.5	213.8±182.1	38.7±14.0	94.1±43.3	42.6±36.5
Non-diabetic CKD (n=50)	158.3±72.5	169.2±132.3	41.9±10.0	82.4±47.4	33.8±26.4
Control (n=50)	146.5±31.8	109.2±28.9	48.3±5.6	76.3±26.3	21.8±5.7
P value	0.01*	0.0002*	0.00003*	0.01*	0.002*
	0.3	0.003**	0.0001**	0.4	0.003**

p<0.05; *significant (diabetic CKD versus controls) and **significant (non-diabetic CKD versus controls). CKD; chronic kidney disease, TC; total cholesterol, TG; triglycerides, HDL; high density lipoproteins, LDL; low density lipoprotein, VLDL; very low-density lipoprotein.

Table 3: Comparison of serum lipid profile (TC, TG, HDL, LDL, VLDL) in diabetic and non-diabetic CKD patients on hemodialysis.

Group	TC (mg/dl)	TG (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	VLDL (mg/dl)
Diabetic CKD (n=50)	177.5±80.5	213.8±182.1	38.7±14.0	94.1±43.3	42.6±36.5
Non-diabetic CKD (n=50)	158.3±72.5	169.2±132.3	41.9±10.0	82.4±47.4	33.8±26.4
p value	0.2	0.1	0.0001**	0.2	0.1

p<0.05; **significant (diabetic CKD versus non-diabetic CKD). CKD; chronic kidney disease, TC; total cholesterol, TG; triglycerides, HDL; high density lipoproteins, LDL; low density lipoprotein, VLDL; very low-density lipoprotein.

DKD patients with 10.24±4.97 years of mean duration of having diabetes in comparison to non-diabetic CKD patients revealed significantly increased ACR (p<0.001) with concurrent non-proliferative diabetic retinopathy (NPDR) which fully met out the diagnostic criteria of DKD (Table 1 and Figure 1).

TC in DKD patients in comparison to controls (177.5±80.5 mg/dl versus 146.5±31.8 mg/dl) was significantly elevated (p=0.01) but not significantly elevated in non-diabetic CKD patients in comparison to controls (p=0.3). Similarly, LDL-C in DKD patients in comparison to controls was also significantly elevated (p=0.01) but not significantly elevated in non-diabetic CKD patients in comparison to

controls (p=0.4). HDL-C in both DKD and non-diabetic CKD patients in comparison to controls (38.7±14.0 and 41.9±10.0 versus 48.3±5.6 mg/dl respectively) was significantly decreased (p<0.001). TG levels in both DKD and non-diabetic CKD patients in comparison to controls (213.8±182.1 and 169.2±132.3 versus 109.2±28.9 mg/dl respectively) were significantly elevated (p=0.0002 and 0.003 respectively). Similarly, VLDL-C levels in both DKD and non-diabetic CKD patients in comparison to controls were also significantly elevated (p=0.002 and 0.003 respectively). Comparative results of lipid profile abnormalities in study patients in comparison to controls are revealed by p value for significance (Table 2) and also depicted graphically (Figure 2).

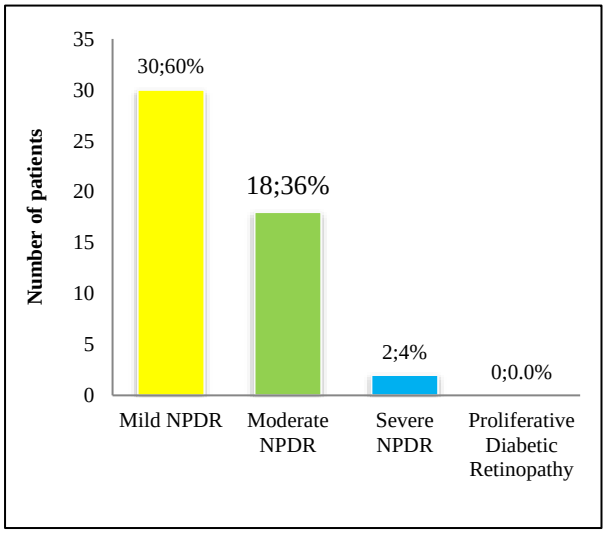

Figure 1: Proliferative and non-proliferative diabetic retinopathy (NPDR) in diabetic CKD patients.

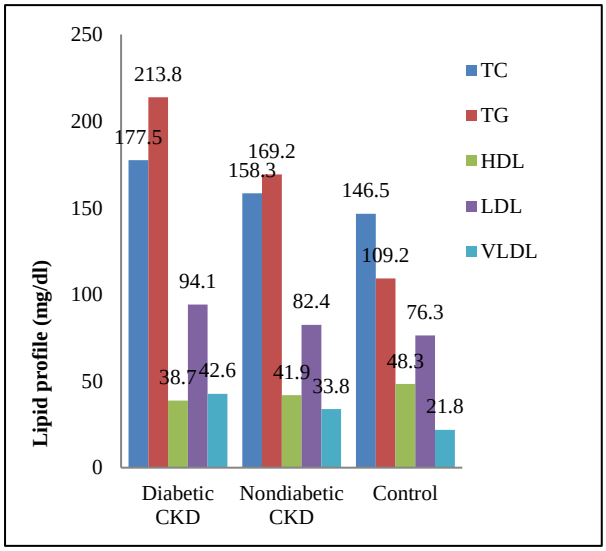

Figure 2: Comparison of lipid profile in study and control groups.

Lipid profile (TC, TG, LDL-C and VLDL-C) revealed insignificantly elevated levels in DKD patients when compared to non-diabetic CKD patients (p>0.05; Table 3). Although mean lipid profile levels in DKD and non-diabetic CKD patients except for DKD patients revealing hypertriglyceridemia (213.8±182.1 versus 169.2±132.3 mg/dl), were in normal range but exhibited significant dyslipidemia when compared to controls (Table 2 and 3). However, HDL-C in DKD patients in comparison to non-diabetic CKD patients (38.7±14.0 versus 41.9±10.0 mg/dl) was significantly decreased (p<0.001).

DISCUSSION

One-year study was conducted in regional hospital catering patients of DKD and non-diabetic CKD with increased incidence and prevalence in periphery. This comparative study included 50 DKD and 50 non-diabetic CKD patients on hemodialysis with mean age of 59.4±10.0 and 45.4 ±13.4 years with male to female ratio of 2.3:1 and 1.9:1 respectively in comparison to 50 controls with mean age of 54.80±13.3 years and male to female ratio of 1.1:1. In a similar comparative study by Raju et al, 45 CKD patients on hemodialysis, 50 non-hemodialysis CKD patients and 50 controls had mean age of 45.24 ±11.03, 45.9±10.50 and 40.94±10.0 2 years with male to female ratio of 1.2:1, 1.4:1 and 1.5:1 respectively.[18] The present study revealed increased incidence of DKD and non-diabetic CKD in males. BUN and serum creatine levels were significantly increased in comparison to controls (8.7±3.1 and 11.5±10.6 versus 0.7±0.09 mg/dl respectively), as the DKD and non-diabetic CKD patients had ESRD (stage 5) and were on hemodialysis. These observations are closely related to study by Kularia et al where CKD patients with and without dialysis and controls revealed BUN and serum creatine of 9.66±2.98, 7.67±2.45 and 0.76±0.34 mg/dl respectively.[29] In the present study DKD patients revealed mean duration of 10.24±4.9 7 years of diabetes and high ACR in comparison to non-diabetic CKD patients (2469.3±1809.8 versus 139.2±94.3 mg/gm) with concurrent NPDR (Table 1). Wu et al reported more severe diabetic retinopathy and macular oedema.[30] In the present study, TC and LDL-C levels were significantly elevated in DKD patients on hemodialysis (p<0.01) but not significantly elevated in non-diabetic CKD patients(p>0.05) when compared to controls, although the mean levels were in normal range in both DKD and non-diabetic CKD patients but insignificantly higher in DKD patients (p>0.05). TG (213.8±182.1 mg/dl) resulting in hypertriglyceridemia and VLDL-C levels were significantly elevated and HDL-C was significantly reduced in DKD patients in comparison to controls (p<0.001; Table 2). These observations of dyslipidemia in DKD patients on hemodialysis is similar to a study by Maurya et al where they observed increased levels of both TC and LDL-C along with increased levels of TG and VLDL-C with reduced HDL-C levels in CKD patients on hemodialysis.[31] However Ashish et al found TC, LDL-C significantly higher and HDL-C significantly lower in CKD patients on PD than on hemodialysis.[19] The present study revealed significantly increased TG and VLDL-C (p=0.003) and significantly reduced HDL-C levels

($p<0.001$) in non-diabetic CKD patients when compared to controls. However, TC and LDL-C levels were not significantly elevated ($p>0.05$). These observations are similar to study from north India which revealed that increased TG, increased VLDL-C and reduced HDL-C levels rather than increased TC and LDL-C levels are responsible for increased cardiovascular complications in CKD patients.[32] Ahmad et al in a study in 2018 observed increased TC,TG, LDL-C and VLDL-C and reduced HDL-C levels in CKD patients with and without hemodialysis as high risk for development of dyslipidemia as hemodialysis effectively reduces the nitrogenous waste products but fails to clear dyslipidemia.[33] In hemodialysis there is more oxidative stress leading to dyslipidemia.[17,18] Protective effects of HDL-C against oxidative stress are impaired in hemodialysis patients.[34] The present study also observed increased prevalence of DKD in diabetic patients. A study from India also observed increased prevalence of CKD in type -2 diabetes mellitus.[35]

CONCLUSION

Both DKD and non-diabetic CKD on haemodialysis revealed significant dyslipidaemia with a difference of DKD patients revealing significantly elevated TC and LDL-C levels as well in comparison to controls and significantly reduced HDL-C in comparison to non-diabetic CKD patients leading to significant difference of dyslipidaemia also between DKD and non-diabetic CKD patients, a major cause of increased risk for CVD attributable to necessitating early treatment with statins. However, a larger study recruiting DKD patients with and without haemodialysis and controls needs to be conducted for better comparative analysis of dyslipidaemia in DKD.

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REFERENCES

1. Rajapurkar M, Dabhi M. Burden of disease – prevalence and incidence of renal disease in India. Clin Nephrol. 2011;74:9-12.
2. Madsen KM, Neilsen S, Tisher CC. Anatomy of the Kidney. In: Brenner BM, editors. Brenner and Rector's The Kidney. 7th edition. Philadelphia: Saunders. 2004;3-72.
3. Strong CG. Physiology of the human kidney. JAMA.1969;210(13):2399-400.
4. Persson F, Rossing P. Diagnosis of diabetic kidney disease: state of the art and future perspective. Kidney Int Suppl. 2018;8(1):2-7.
5. Lamb EJ, Tomson CRV, Roderick PJ. Estimating kidney function in adults using formulae. Ann Clin Biochem. 2005;42(5):321-45.
6. Agarwal R. Defining end-stage renal disease in clinical trials: A framework for adjudication. Nephrol Dial Transplant. 2016;31(6):864-7.
7. Kalmegh R, Idhol S. A retrospective study on chronic kidney disease and its etiological profile. J Med Res Prac. 2019;08(03):40-3.
8. Bricker NS, Bourgoignie J, Weber H, Schmidt RW, Slatopolsky E. Pathogenesis of the uremic state: a new perspective. Adv Nephrol Necker Hosp. 1972;2:263-76.
9. Alicic RZ, Rooney MT, Tuttle KR. Diabetic kidney disease: challenges, progress, and possibilities. Clin J Am Soc Nephrol. 2017;12(12):2032-45.
10. Gross JL, Azevedo MJ, Silveiro SP, Canani LH, Caramori ML, Zelmanovitz T. Diabetic nephropathy: diagnosis, prevention, and treatment. Diabetes Care. 2005;28(1):164-76.
11. Kuhar MB. Update on managing hypercholesterolemia. The new NCEP guidelines. AAOHN J. 2002;50(8):360-4.
12. Mehta V, Bhatt K. Lipids and its metabolism. J Cardiol Cardiovasc Ther. 2017;4(2):555-635.
13. Vaziri ND. Dyslipidemia of chronic renal failure : the nature, mechanisms, and potential consequences. Am J Physiol Renal Physiol. 2019;92868:262-72.
14. Kawanami D, Matoba K, Utsunomiya K. Dyslipidemia in diabetic nephropathy. Ren Replace Ther. 2016;2:1-9.
15. Rifai N, King ME, Sica DA. Effects of long-term haemodialysis on lipid, lipoprotein and apolipoprotein levels of black patients with chronic renal failure. Ann Clin Biochem. 1988;25(3):242-5.
16. Mekki K, Prost J, Remaoun M, Belleville J, Bouchenak M. Long term hemodialysis aggravates lipolytic activity reduction and very low density, low density lipoproteins composition in chronic renal failure patients. BMC CARDIOVASC DISOR. 2009;8:1-8.
17. Sharma H, Shah TJ, Gorasia JH, Baria DP. Lipid profile and lipoprotein(a) in chronic renal failure patients with and without hemodialysis. Int J Med Public Heal. 2012;2(4):28-31.
18. Raju DSSK, Lalita DL, Kiranmayi P. A study of lipid profile and lipid peroxidation in chronic kidney disease with special reference to hemodialysis. J Clin Res Bioeth. 2013;04(01):1-5.
19. Anjankar AP, Dharme PV, Anjankar VP. Study of comparative effect of hemodialysis and peritoneal dialysis on lipid profile of patients of chronic kidney disease. Asian J Biomed Pharmaceut Sci. 2014;04(36):30-4.
20. Herzog CA, Ma JZ, Collins AJ. Poor long term survival after acute myocardial infarction among patients on long-term dialysis is poor. N Engl J Med. 1998;339(12):799-805.
21. Karnik JA, Young BS, Lew NL, Herget M, Dubinsky C, Lazarus JM, et al. Cardiac arrest and sudden death in dialysis units. Kidney Int. 2001;60(1):350-7.
22. Ganta V, Yalamanchi RP, KC M, Sahu B, Raghvendar K, Anusha G, et al. A study of lipid profile in non-diabetic chronic kidney disease. Int J Adv Med. 2016;3(4):965-70.

23. Muntner P, He J, Astor BC, Folsom AR, Coresh J. Traditional and nontraditional risk factors predict coronary heart disease in chronic kidney disease: results from the atherosclerosis risk in communities study. J Am Soc Nephrol. 2005;16(2):529-38.
24. Harchaoui KEL, Visser ME, Kastelein JJP, Stroes ES, Thie GMD. Triglycerides and cardiovascular risk. Curr Cardiol Rev. 2009;5:216-22.
25. Bhagwat R, Joshi SP, Salgia P, Sepaha A. Lipid abnormalities in chronic renal failure. Indian J Clin Biochem. 1997;12(1):81-5.
26. Weiner DE, Sarnak MJ. Managing dyslipidemia in chronic kidney disease. J Gen Intern Med. 2004;19(10):1045-52.
27. 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.
28. Rhee EJ, Kim HC, Kim JH, Lee EY, Kim BJ, Kim EM, et al. Guidelines for the management of dyslipidemia. Korean J Intern Med. 2019;34(4):723-71.
29. Kularia R, Sharma A, Acharya J. Dyslipidemia in chronic renal failure patients : a clinical study at tertiary care hospital , Bikaner. Int J Med Res Prof. 2018;4(3):147-9.
30. Wu L, Loaiza PF, Sauma J, Bogantes EH, Masis M. Classification of diabetic retinopathy and diabetic macular edema. World J Diabetes. 2013;4(6):290.
31. Maurya NK, Sengar NS, Arya P. Impact of hemodialysis on lipid profile among chronic renal failure patients (regular and non-regular haemodialysis). Pharma Innov J. 2018;7(4):363-5.
32. Phukan RR, Goswami RK. Unusual dyslipidemia in patients with chronic kidney diseases. J Clin Diagn Res. 2017;11(1):BC01-4.
33. Ahmad N, Agarwal S, Chauhan A, Khrime D, Varma A. A study of lipid profile in chronic renal failure patients in tertiary care hospital of Uttarakhand. Indian J Appl Res. 2018;1(1):51-2.
34. Morena M, Cristol JP, Dantoine T, Carbonneau MA, Descomps B, Canaud B. Protective effects of high-density lipoprotein against oxidative stress are impaired in haemodialysis patients. Nephrol Dial Transplant. 2000;15(3):389-95.
35. Prasannakumar M, Rajput R, Seshadri K, Talwalkar P, Agarwal P, Gokulnath G, et al. An observational, cross-sectional study to assess the prevalence of chronic kidney disease in type 2 diabetes patients in India (START -India). Indian J Endocrinol Metab. 2015;19(4):520-3.

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