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Prevalence And Pattern of Dyslipidemia in Children And Adolescent on Regular Hemodialysis at Zagazig University Hospitals

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Abstract: Background: Dyslipidemia, a known risk factor for atherosclerosis, is frequent among both adults and children with chronic kidney disease (CKD). In addition, there is evidence to suggest that dyslipidemia contributes to the initiation and progression of CKD itself.

Aim: To asses the frequency of dyslipidemia in children and adolscent with end stage renal disease (ESRD) on regular hemodialysis.

Patients and Methods: Our study is a cross – sectional study included 61 patients with agesranging from 4-17 years who were diagnosed with end-stage renal disease (chronic kidney disease stage V with eGFR less than 15 ml/min/1.73m2 by modified Schwartz equation and on regular hemodialysis.

Results: we found total cholesterol was high (>=200) in 18 (29.5 %) patients, was borderline (170 - 199) in 12 (19.7%) patients and was acceptable (<170) in 31 (50.8%) patients. The frequency of abnormally high levels of total serum cholesterol, triglycerides, and low-density were found to be 29.5%, 75.4% and 16.4% respectively. HDL ranged from 14.9 to 62.9 mg/dL with a mean value (± SD) of 37.59 (±8.68) mg/dL. 10(16.4 %) patients were acceptable (>45) ,11 (18%) patients were borderline (40 - 45), and 40 (65.6 %) patients were low (<40). LDL ranged from 54.8 to 166.7 mg/dL with a mean value (± SD) of 99.61 (±29.66) mg/dL. 39(63.9 %) patients were acceptable (<110) ,12 (19.7%) patients were borderline (110 – 129) and 10 (16.4 %) patients were high (>=130). VLDL ranged from 11.3 to 90.1mg/dL with a mean value (± SD) of 37.50 (±15.99) mg/dL. 21 (34.4%) patients were normal, and 40 (65.6%) patients were abnormal. Triglycerides, VLDL-C, and HDL levels were observed to be lower during maintenance hemodialysis.

Conclusion: High percentage of examined children had high lipid profile (29.5 %) of the populations, The frequency of abnormally high levels of total serum cholesterol, triglycerides, and low-density were found to be 29.5%, 75.4% and 16.4% respectively. Triglycerides, VLDL-C, and HDL levels were observed to be lower during maintenance hemodialysis.

Keywords: *Prevalence, Dyslipidemia, Children, Regular Hemodialysis*

1. Introduction

Chronic kidney disease is characterized by an irreversible deterioration of renal function that gradually progresses to end-stage renal disease (ESRD). Chronic kidney disease has emerged as a serious public health problem. Data from the United States Renal Data System (USRDS) show that incidence of kidney failure is rising among adults and is commonly associated

with poor outcomes and high cost. Moreover, in the past 2 decades, the incidence of chronic kidney disease in children has steadily increased, with poor and ethnic minority children disproportionately affected(**1).**

Dyslipidemias are defined as a group of lipoprotein abnormalities that can result in any of the following lipid abnormalities(**2):** Elevated total cholesterol (TCz, Elevated low-density lipoprotein-cholesterol (LDL-C), Elevated non-high-density lipoprotein cholesterol (HDL-C), Elevated triglycerides (TG), Decreased HDL-C.

Hypertriglyceridemia is considered as the hallmark of uremic dyslipidemia and is a consequence of accumulation of triglycerides and triglyceriderich lipoproteins (TRL) in CKD due to increased production and impaired catabolism and increased apolipoprotein C-III levels inducing an increase in triglyceride levels. An increased expression of apolip protein C-III, (inhibitor of lipoprotein lipase (LPL) that breaks down triglycerides), has been reported in vascular endothelial cells of skeletal muscle blood vessels and other cells that utilize fatty acids for their energy consumption (**3).**

Serum lipid levels vary depending on age, puberty and gender. They are slightly lower in girls than the boys while young adults with CKD have at least 10-fold higher risk of CVD mortality compared to the general population. The underlying glomerular renal pathology and duration of proteinuria have also been reported to influence the type of dyslipidemia in pediatric and adult patients with CKD (**4).**

Aim: To detect the frequency of dyslipidemia in patients with end stage renal disease (ESRD) at Zagazig university hospitals.

Patients and Methods

This cross sectional study was conducted in the pediatrics Nephro-dialysis unit children hospital university, With IRB number #9739/31-8-2022. During the study period (6 months from October 2023 to April 2024) , 61 cases on regular hemodialysis was included as a comprehensive sample aged from 4-17 years who were diagnosed with end-stage renal disease (chronic kidney disease stage V with eGFR less than 15 ml/min/1.73m^2 by modified Schwartz equation and on regular hemodialysis. Informed consent was taken from every patient or his parents or caregivers.

Inclusion Criteria: Aged up to 18 years, who were dagnosed with end stage renal disease (chronic kidney disease grade V with eGFR less than 15 ml/min/1.73m2 by modified Schwartz equation and on regular hemodialysis.

Exclusion Criteria: cases with any Associated malignancy, Previous transplantation, Dialysis within 3 months, Age more than 18 years.

Complete history was taken from parents or care givers of children, Complete physical examination included Blood pressure measurement, Body temperature measurement, Heart and respiratory rates. Signs of (Pallor, Cyanosis, Jaundice, and Lymph node enlargement) were assessed.

The laboratory investigations included routine tests and the lipid profile (fasting 12 hours)

Blood was drawn into a serum separator tube and allowed to clot at room temperature for 30 min before centrifugation and serum separation. Serum was shipped to the Zagazig University hospital labs by next day mail or (if the next day was not a working laboratory day) refrigerated at 4 °C and shipped the next available day. All lipid measurements were automated (Cobas 8000, chemistry c702, enzymatic, colorimetic method).

Serum TG and TC measurement followed routine enzymatic methods; HDL–C was analyzed by the Bayer (Siemens Diagnostics, Deerfield, IL, USA) clinical method for ADVIA 2400 direct HDL–C (D-HDL) V 1.00.00 technique. Briefly, this involves detection of HDL–C following selective elimination of cholesterol from chylomicron and very-low-density lipoprotein remnants from the detection reactions.51 As older techniques have slightly different performances in subjects with CKD, it should be noted that CKD-specific validation of this method was lacking.

However, this homogenous HDL–C technique was not subject to interference by TG <1700 mg/dl or anemia, and was commercially certified via comparison protocols against multiple other HDL–C techniques. In all uses, analyzer calibration was confirmed by known standards. Non-HDL–C was calculated as the difference between TC and HDL–C.

The following cut points were used to define the presence of dyslipidemia: TG>130 mg/dl, HDL–C<40 mg/dl, and non-HDL–C>160 mg/dl. These cut points were based on: (a) normative NHANES data available for children aged 12 years and older, (b) normative data of the Lipid Research Clinic data set, and (c) levels frequently considered atherogenic in adults.

For the majority of study participants, direct measurement of GFR was accomplished by the plasma disappearance of iohexol (Ominipaque, GE Healthcare, Princeton, NJ, USA) method (iGFR), as detailed previously. In the event iGFR was not available, GFR was estimated using a published estimating equation developed within this study population. In this paper, the pooled grouping of iGFR and estimated GFR was referred to as GFR. Total urine protein and urine creatinine were measured in a first morning urine sample and expressed as the Up/c in units of mg/mg. Details of the techniques used to measure Up/c in CKiD have been published. Proteinuria was categorized as normal (Up/c<0.2), mild (0.2≤Up/c<1.0), moderate (1.0≤Up/c<2.0), or nephrotic (Up/c≥2.0).

Nonlaboratory data were obtained concomitantly using standardized forms and physical exam. Data collected and used in the current analysis include age, sex, race, ethnicity, height, weight, and primary CKD diagnosis. BMI was calculated as weight/height2 (kg/m2). Age- and gender-specific BMI percentiles were calculated using 2000 Centers for Disease Control and Prevention standard growth charts for United States children.Overweight was defined as a BMI between the 85 and 95th percentiles, obesity as a BMI >95th percentile. CKD diagnosis was classified as glomerular or nonglomerular; a more detailed description of this classification for specific diagnoses was published.

Analysis was restricted to individuals with known age, sex, race, GFR, and CKD diagnosis, as well as complete lipid measures. Excluded from the analysis were children receiving lipid-lowering medication and those who were known to have been nonfasting at the time the blood sample was obtained.

Patients was be required to observe overnight fast for a minimum of 12 hours for analysis of various types of serum lipid levels. Levels of total serum cholesterol, serum triglycerides, high density lipoprotein (HDL-C), low density lipoprotein (LDL-C) and very low density lipoprotein (VLDL-C) was be measured. The levels of serum lipids was be classified into various subgroups. Method Enzymatic colorimetric assays Instrument Cobas 8000 (Roche Diagnostics ;Germany) .

Statistical analysis

The collected data was revised, coded, tabulated and introduced to a PC using Statistical package for Social Science (SPSS 26). Data was presented and suitable analysis was done according to the type of data obtained for each parameter. Mean, Standard deviation (± SD) and range for parametric numerical data, while Median and Interquartile range (IQR) for non-parametric numerical data. Frequency and percentage of non-numerical data.

Results

Basic Sociodemographic data and present history of illness, comorbidities are illustrated in Table 1.

Table 1: Sociodemographic data and present history of illness of the studied group.

		Mean or N	SD or %	Median (IQR)	Range
Age (in years)		9.79	3.35	8 (8 - 12)	(4 - 17)
Weight (Kg)		26.66	9.82	22 (20 - 35)	(12 - 54)
Duration of haemodialysis (years)		3.19	2.66	2.1 (1.9 - 2.3)	(0.4 - 12)
Sex	Male	34	55.7%		
	Female	27	44.3%		
Causes of CKD	Unknown	11	18.0%		
	Obstructive uropathy	22	36.0%		
	a. Undetermined	10	16.4%		
	b. Hydronephrosis	4	6.6%		
	c. Reflux	5	8.2%		
	d. Neurogenic bladder	3	4.9%		
	SLE	8	13.1%		
	UTI	5	8.2%		
	Nephrotic syndrome	11	18.0%		
	RPGN	2	3.3%		
	HUS	1	1.6%		
	Solitary kidney	1	1.6%		
Blood pressure	SBP	125.05	7.19	124 (120 - 131)	(114 - 141)
	DBP	81.70	7.87	81 (75 - 88)	(67 - 96)
No. of HTN drugs	No	15	24.6%		
	One drug	37	60.7%		
	Two drugs	9	14.8%		
Cardiac complications (HF-Infective endocarditis-Arrhythmia-pericardial effusion - Cardiomegaly)	No	23	37.7%		
	Yes	38	62.3%		

HUS:Hemolytic uremic syndrome, RPGN:Rapidly progressive glomerulonephritis, SLE:Systemic lupus erythematosus, UTI: Urinary tract infection, HTN: Hypertension.

Table 2 and Figures (1:6) shows that Cholesterol was high (>=200) in 18 (29.5 %) patients , was borderline (170 - 199) in 12 (19.7%) patients and was acceptable (<170) in 31 (50.8%) patients, high plasma total cholesterol levels were associated with increased mortality, as observed in the general population. To determine the frequency and pattern of dyslipidemia in pediatric patients with ESRD, a study on 61 patients with mean age 9.79(± 3.35)years and male to female ratio 1.2:1. The frequency of abnormally high levels of total serum cholesterol, triglycerides, and low-density were found to be 29.5%, 75.4% and 16.4% respectively. HDL ranged from 14.9 to 62.9 mg/dL with a mean value (± SD) of 37.59 (±8.68) mg/dL. 10(16.4 %) patients were acceptable (>45) ,11 (18%) patients were borderline (40 - 45), and 40 (65.6 %) patients were high (<40).LDL ranged from 54.8 to 166.7 mg/dL with a mean value (± SD) of 99.61 (±29.66) mg/dL. 39(63.9 %) patients were acceptable (<110) ,12 (19.7%) patients were borderline (110 – 129) and 10 (16.4 %) patients were high (>=130). VLDL ranged from 11.3 to 90.1mg/dL with a mean value (± SD) of 37.50 (±15.99) mg/dL. 21 (34.4%) patients were normal, and 40 (65.6%) patients were abnormal.Triglycerides, VLDL-C, and HDL levels were observed to be higher during maintenance hemodialysis. Cholesterol/HDL ratio ranged from 2.64 to 9.03 with a mean value (± SD) of 4.8 (±1.28). 7(11.5 %) patients were desirable (<3.4), 29 (47.5%) patients were borderline (3.4 - 5) and 25 (41%) patients were high (>5).

Table 2: laboratory investigations of the studied group.

	Mean or N	SD or %	Median (IQR)	Range
Hb	9.70	1.07	9.6 (9.1 - 10.2)	(7.6 - 12.9)
PLT	245.71	116.92	216 (187 - 277)	(50.2 - 886)
WbCs	6.66	2.02	6.7 (5.2 - 7.4)	(3.2 - 14.5)
Alb	4.21	0.43	4.17 (4.07 - 4.41)	(2.91 - 5.5)
Urea	48.47	15.92	49.7 (40.5 - 55.95)	(4.06 - 92.2)
Creat	7.45	6.73	6.92 (5.47 - 8.87)	(0.93 - 55.4)
PTH	388.56	1155.86	216 (186.5 - 277)	(10.1 - 9212)
Ca	8.78	0.92	8.8 (8.07 - 9.4)	(5.77 - 10.5)
PO4	4.97	1.42	4.99 (4.05 - 5.6)	(2.17 - 9.81)
Iron	93.53	40.27	82.1 (59.1 - 112.3)	(32.3 - 198.7)
Ferritin	727.74	809.08	457.2 (204.2 - 963.2)	(9.16 - 4281)
Na	135.70	2.71	136 (134 - 137)	(125 - 141)
K	5.41	0.86	5.3 (4.8 - 5.8)	(3.6 - 8.6)
CRP	6.86	9.75	4.13 (1.25 - 6.85)	(0.52 - 46.1)

Hb:Hemoglobin,PLT: Platelets,WbCs: White blood cells, ,Alb: Albumin,Create: Creatinine,PTH:Parathyroid hormone,CRP:C-reactive protein.

Table 3: Lipid profile of the studied group.

Cholesterol		173.59	38.65	159.1 (144.6 - 201.1)	(113.4 - 252.1)
Cholesterol	Acceptable (<170)	31	50.8%		
	Borderline (170 - 199)	12	19.7%		
	High (>=200)	18	29.5%		
TG		187.69	79.95	186.4 (121.2 - 249.5)	(56.8 - 450.5)
TG	Acceptable	3	4.9%		
	Borderline	12	19.7%		
	High	46	75.4%		
HDL		37.59	8.68	37.4 (32.7 - 41.2)	(14.9 - 62.9)
HDL	Acceptable (>45)	10	16.4%		
	Borderline (40 - 45)	11	18.0%		
	Low (<40)	40	65.6%		
LDL		99.61	29.66	92.3 (78.5 - 117)	(54.8 - 166.7)
LDL	Acceptable (<110)	39	63.9%		
	Borderline (110 - 129)	12	19.7%		
	High (>=130)	10	16.4%		
VLDL		37.50	15.99	37.2 (24.2 - 49.9)	(11.3 - 90.1)
VLDL (2 - 30)	Normal	21	34.4%		
	Abnormal	40	65.6%		
Cholesterol/HDL ratio		4.8	1.28	4.56 (3.86 – 5.45)	(2.64 – 9.03)
Cholesterol/HDL ratio	Desirable (<3.4)	7	11.5%		
	Borderline (3.4 - 5)	29	47.5%		
	High (>5)	25	41.0%		

TG: Triglyceride. HDL: high density lipoprotein. LDL: Low density lipoprotein. V LDL: Very low density lipoprotein.

Table 4: Lipid profile in studied group according to underlying etiology

	Cholesterol			TG			HDL			LDL			VLDL	
	Acceptable (<170)	Borderline (170 - 199)	High (>=200)	Acceptable	Borderline	High	Acceptable (>45)	Borderline (40-45)	High (<40)	Acceptable (<110)	Borderline (110-129)	High (>=130)	normal	Abnormal
Unknown (10)	2 (20%)	2 (20%)	6 (60%)	1 (10%)	1 (10%)	8 (80%)	2(20%)	2(20%)	6 (60%)	2(20%)	4 (40%)	4 (40%)	3 (30%)	7(70%)
Atrophic kidney (1)	0 (0%)	1 (100%)	0 (0%)	0 (0%)	0 (0%)	1 (100%)	0 (0%)	0 (0%)	I (100%)	0(0%)	I (100%)	0(0%)	o (0%)	I (100%)
Autoimmune (5)	3 (60%)	1 (20%)	1 (20%)	0 (0%)	1 (20%)	4 (80%)	I (20%)	(40%)	2 (40%)	(100%)	0 (0%)	0 (0%)	I (20%)	4 (80%)
HUS (1)	0 (0%)	0 (0%)	1 (100%)	0 (0%)	0 (0%)	1 (100%)	0 (0%)	0 (0%)	1 (100%)	0(0%)	0 (0%)	(100%)	o (0%)	I (100%)
Hydronephrosis (4)	2 (50%)	1 (25%)	1 (25%)	1 (25%)	0 (0%)	3 (75%)	0 (0%)	(25%)	(75%)	2(50%)	I (25%)	I (25%)	2(50%)	2 (50%)
Nephrotic syndrome (11)	8 (72.7%)	1 (9.1%)	2 (18.2%)	0 (0%)	2 (18.2%)	9 (81.8%)	0 (0%)	2 (18.2%)	9 (81.8%)	(72.7%)	3 (27.3%)	0 (0%)	3 (27.3%)	8 (72.7%)
neurogenic bladder (3)	1 (33.3%)	1 (33.3%)	1 (33.3%)	0 (0%)	0 (0%)	3 (100%)	0 (0%)	I (33.3%)	2 (66.7%)	2(66.7%)	0(0%)	I (33.3%)	I (33.3%)	2 (66.7%)
obstructive uropathy (10)	6 (60%)	1 (10%)	3 (30%)	1 (10%)	1 (10%)	8 (80%)	1(10%)	I (10%)	8(80%)	7(70%)	I (10%)	2(20%)	2 (20%)	8 (80%)
Pyelonephritis (4)	2 (50%)	2 (50%)	0 (0%)	0 (0%)	2 (50%)	2 (50%)	3(75%)	I (25%)	0(0%)	4(100%)	0(0%)	0(0%)	2(50%)	2(50%)
Reflux (5)	2 (40%)	0 (0%)	3 (60%)	0 (0%)	1 (20%)	4 (80%)	2(40%)	0 (0%)	3(60%)	2(40%)	(40%)	I (20%)	I (20%)	4 (80%)
RPGN (2)	2 (100%)	0 (0%)	0 (0%)	0 (0%)	1 (50%)	1 (50%)	0 (0%)	0 (0%)	2(100%)	2 (100%)	0(0%)	0(0%)	2(100%)	0 (0%)
SLE (3)	3 (100%)	0 (0%)	0 (0%)	0 (0%)	2 (66.7%)	1 (33.3%)	0(0%)	0 (0%)	3(100%)	3(100%)	0 (0%)	0(0%)	3(100%)	0 (0%)
Solitary kidney (1)	0 (0%)	1 (100%)	0 (0%)	0 (0%)	0 (0%)	1 (100%)	0(0%)	(100%)	0(0%)	I (100%)	0 (0%)	0(0%)	0 (0%)	(100%)
UTI (1)	0 (0%)	1 (100%)	0 (0%)	0 (0%)	1 (100%)	0 (0%)	(100%)	0 (0%)	0(0%)	I (100%)	0 (0%)	0(0%)	(100%)	0 (0%)

Data is presented as frequency (%).HUS: Hemolytic uremic syndrome, RPGN: Rapidly progressive glomerulonephritis, SLE: Systemic lupus erythematosus, UTI: Urinary tract infection

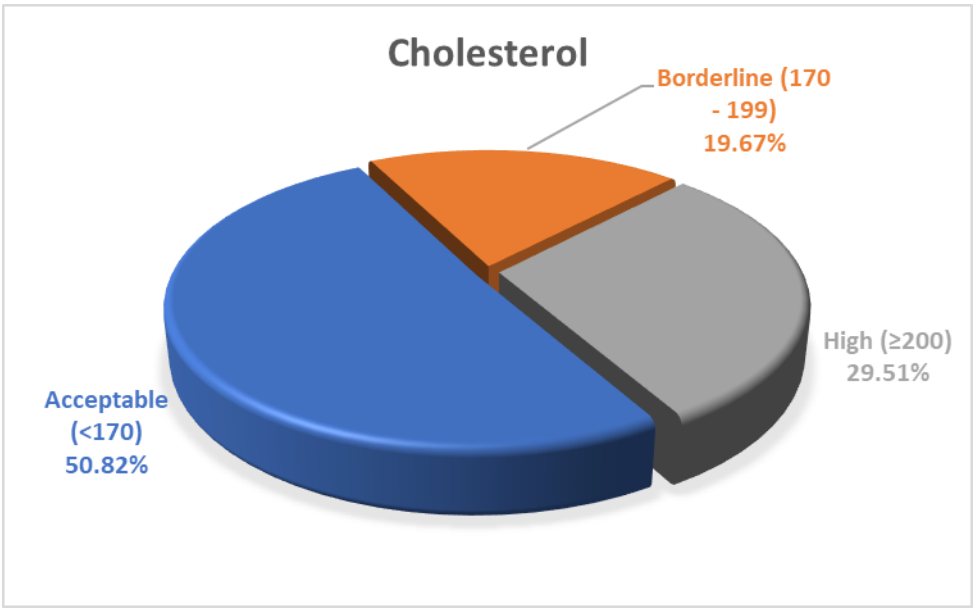

Figure 1: Cholesterol of the studied patients

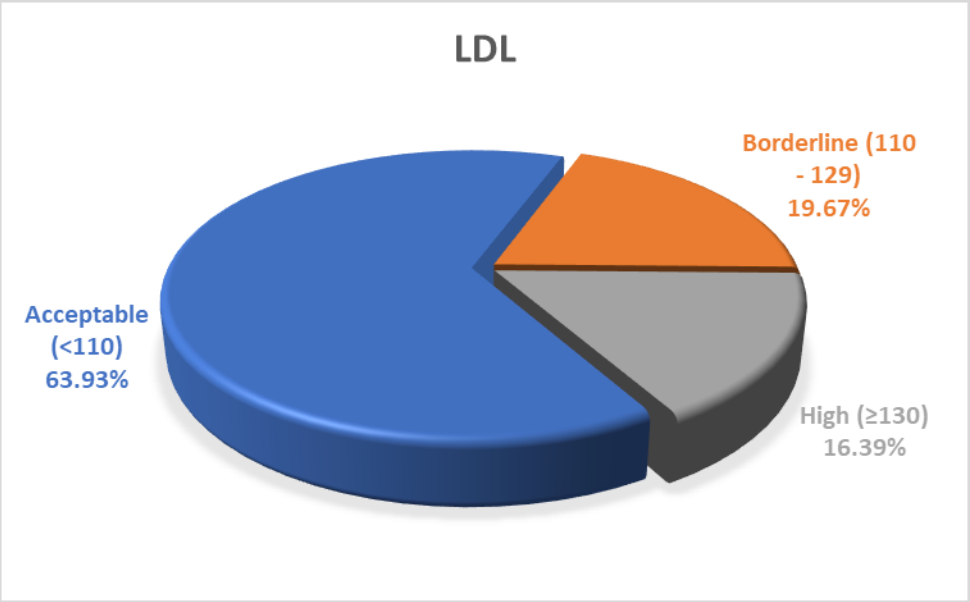

Figure 2: TG of the studied patients

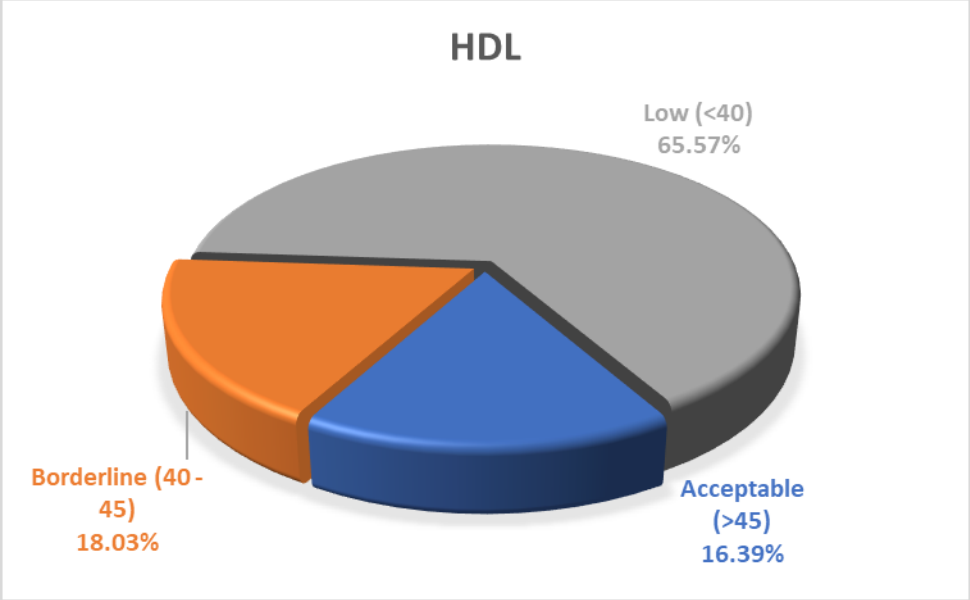

Figure 3: HDL of the studied patients

Figure 4: LDL of the studied patients

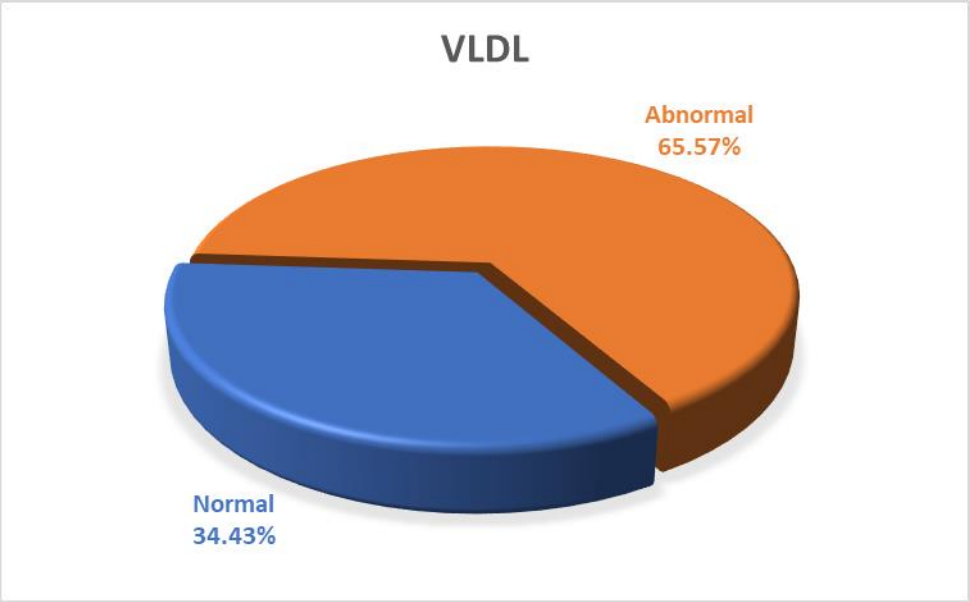

Figure 5: VLDL of the studied patients

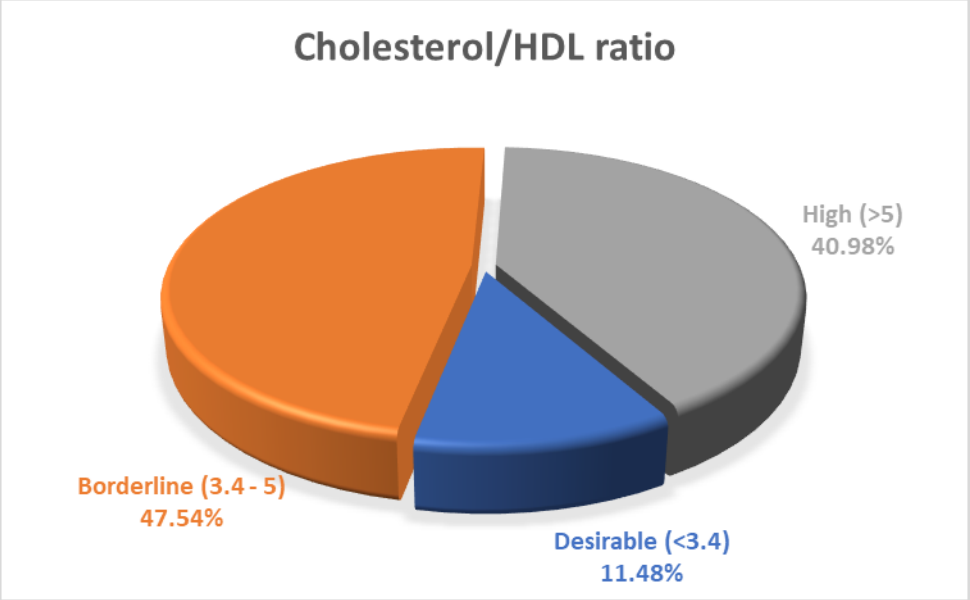

Figure 6: Cholesterol/HDL ratio of the studied patients

Discussion

Chronic kidney disease (CKD) is a life-long condition associated with substantial morbidity and premature death due to complications from a progressive decrease in kidney function. The incidence and prevalence of all stages of CKD in children continue to increase worldwide. Between 2000 and 2008, the kidney replacement therapy incidence rate in those aged 0-19 years increased by 5.9% to 15 per million population, highlighting the importance of CKD research in children**(5)**.

CKD is an important condition in children. Cross-sectional analysis of baseline CKD data has revealed valuable information that better defines the prevalence of comorbid conditions and associated risk factors, including hypertension, LVH, dyslipidemia, anemia, poor growth, and abnormal neurocognitive development, that accompany CKD**(6,7)**.

Among children with moderate CKD, dyslipidemia is common and independently associated with lower GFR and nephrotic-range proteinuria. In particular, a high prevalence of hypertriglyceridemia, increased non-HDL–C, reduced HDL–C, and combined dyslipidemias, particularly with lower GFR were reported. Nephrotic-range proteinuria is associated with increased TG and non-HDL–C, without significant effect on HDL–C**(8)**.

Children with CKD/ESRD exhibit various co-morbidities, including dyslipidemia. The prevalence of dyslipidemias in children with CKD and ESRD is high (39-65 %) but is significantly dependent on the cause and vintage of CKD (e.g., usually more common and severe with glomerular disease and proteinuria) and the stage of the disease **(5)**.

Risk for cardiovascular disease (CVD) is notably increased in children and adolescents with chronic kidney disease (CKD) and end-stage renal disease (ESRD) requiring renal replacement therapy. Cardiovascular (CV) mortality, specifically, is increased up to 1000-fold for pediatric patients with evolving stages of CKD and ESRD as compared to age-matched peers **(6)**.

The relative risk for CVD from dyslipidemia in children with CKD and ESRD compared to the general pediatric population is not known due to the short time frame of follow-up and the existence of other CVD risk factors (e.g., inflammation). However, the American Heart Association Expert Panel on Population and Prevention Science has concluded that prevention of CVD in high-risk pediatric patients is warranted due to the higher risk of developing disease as adults**(9)**.

Pediatric as well as adult patients with chronic kidney disease (CKD) are susceptible to cardiovascular disease (CVD) events, which increase their mortality. Dyslipidemia is thought to be one of the most important contributing risk factors for developing CVD**(10)**.

The lipoprotein and triglyceride (TG) concentration reflects lipid metabolism that is modulated by genetic and environmental factors. Dyslipidemia can result from an intrinsic, extrinsic, or a combination of genetic predisposition and external factors **(11)**.

Hypertriglyceridemia is considered as the hallmark of uremic dyslipidemia and is a consequence of accumulation of triglycerides and triglyceriderich lipoproteins (TRL) in CKD due to increased production and impaired catabolism and increased apolipoprotein C-III levels inducing an increase in triglyceride levels as detected by **Hruska et al., (12)**.

Indeed, there is strong evidence of reduced lipolysis of TRL due to the decreased activity of the major lipases, lipoprotein lipase (LPL) and hepatic triglyceride lipase (HTGL), that are mainly responsible for breaking down TG into free fatty acids. Increased production of TRL may also be secondary to reduced carbohydrate tolerance and enhanced hepatic VLDL synthesis. The reduced fractional catabolic rate is likely due to the decreased activity of two endothelium-associated lipases, LPL and HTGL

In the same context, the pathogenesis of dyslipidemia in CKD features various factors, including increased levels of triglycerides, triglyceride-rich lipoproteins, apolipoprotein C3 (ApoC-III), decreased levels of cholesterylester transfer protein and high-density lipoproteins, and aberrations in serum very low-density and intermediate-density lipoproteins. **(14)**

In addition, **Hager et al., (15)** revealed that HTG is present in early stages of renal disease and its origin is multifactorial, including impaired catabolism of VLDL and chylomicrons secondary to decreased lipoprotein lipase (LPL) activity. With the onset of uremia, inhibitors of LPL are increased, including apoC-III and pre-beta-HDL. A decrease in lecithin:cholesterol acyltransferase (LCAT), important for the maturation of HDL, and reduced expression of the apoA-I gene *APOA1*, the main apoprotein of HDL, have also been reported. These changes in gene expression and protein availability lead to alterations in two key HDL functions: 1) reverse cholesterol transport; and 2) anti-oxidation.

Our study is a cross – sectional study included 61 patients with agesranging from 4-17 years who were diagnosed with end-stage renal disease (chronic kidney disease stage V with eGFR less than 15 ml/min/1.73m2 by modified Schwartz equation and on regular hemodialysis .

In this study we determine the frequency and pattern of dyslipidemia in pediatric patients with ESRD, a study on 61 patients with mean age 9.79(± 3.35)years and male to female ratio 1.2:1. The frequency of abnormally

high levels of total serum cholesterol, triglycerides, and low-density were found to be 29.5%, 75.4% and 16.4% respectively.

In this study, we found Cholesterol was high (>=200) in 18 (29.5 %) patients , was borderline (170 - 199) in 12 (19.7%) patients and was acceptable (<170) in 31 (50.8%) patients, high plasma total cholesterol levels were associated with increased mortality, as observed in the general population.

However, **Kwan et al., (1)**in a study of 1167 HD patients found that among those with low plasma albumin levels, low plasma total cholesterol levels were also associated with increased all-cause mortality. This dichotomous relationship was confirmed in the Choices for Healthy Outcomes in Caring for ESRD (CHOICE) study, which showed a nonsignificant negative association of cardiovascular mortality with plasma total as well as non–HDL cholesterol levels in the presence of inflammation and/or malnutrition in contrast, there was a positive association between total and non–HDL cholesterol and mortality in the absence of inflammation or malnutrition. These observations are compatible with the hypothesis that the inverse association of total cholesterol levels with mortality in dialysis patients is mediated by the cholesterol-lowering effect of malnutrition and/or systemic inflammation and not due to a protective effect of high cholesterol concentrations.

Also, in this study, TG ranged from 56.8 to 450.5 mg/dL with a mean value (± SD) of 187.69 (±79.95) mg/dL., 46 (75.4 %) patients were high 12 (19.7%) patients were borderline, and 3(4.9 %) patients were acceptable. Triglyceride levels are higher due to decreased activity of lipoprotein lipase (LPL), which hydrolyzes triglycerides, as well as increased triglyceride synthesis in the liver from free fatty acids released from adipose tissue and muscles.

Chaudhry et al., (16) conducted a study on 138 patients with mean age 11.24 ± 2.37 years and male to female ratio 2.5:1. The frequency of abnormally high levels of total serum cholesterol, triglycerides, and low-density were found to be 21.7%, 84.8% and 19.6% respectively. Majority of pediatric patients with ESRD had suffered from dyslipidemia especially raised serum triglycerides. Timely identification of abnormal lipid levels and appropriate management is expected to help reduce cardiovascular morbidity and mortality associated with dyslipidemia in these pediatric patients.

Corresponding to the present study findings, HDL ranged from 14.9 to 62.9 mg/dL with a mean value (± SD) of 37.59 (±8.68) mg/dL. 10(16.4 %) patients were acceptable (>45) ,11 (18%) patients were borderline (40 - 45), and 40 (65.6 %) patients were low (<40).LDL ranged from 54.8 to 166.7 mg/dL with a mean value (± SD) of 99.61 (±29.66) mg/dL. 39(63.9 %) patients were acceptable (<110) ,12 (19.7%) patients were borderline (110 – 129) and 10 (16.4 %) patients were high (>=130).

The pattern of dyslipidemia seen in CKD is typically characterized by hypertriglyceridemia (HTG), decreased HDL-C, variable changes in LDL-C, increase in non-HDL-C, and an increase in small dense LDL-C as detected by **(17)**.

Cholesterol/HDL ratio ranged from 2.64 to 9.03 with a mean value (± SD) of 4.8 (±1.28). 7(11.5 %) patients were desirable (<3.4), 29 (47.5%) patients were borderline (3.4 - 5) and 25 (41%) patients were high (>5).

Also, Combined Dyslipidemia as defined by **(7)**is characterized by moderate to severe increased TG levels associated with low HDL levels (with increased non-HDL cholesterol) and is currently the most common form in pediatric age. It results from the epigenetic and environmental influence, that is, the combination of genetic variants and polymorphism effects with external stimuli (diabetes mellitus (DM), obesity, kidney disease, hypothyroidism, high-calorie diet, fats, or alcohol)

Ultimately, the resulting endothelial dysfunction, inflammation, and oxidative stress could be responsible for the association between HDL and increased mortality in CKD according to **Honda et al., (18)**. Another interesting concept was presented by **Florens et al., (19)** who showed that CKD-induced carbonylation of HDL was responsible for impaired platelet aggregation, further contributing to the etiology of increased cardiovascular events in the CKD population.

Moreover, **Ikewaki et al., (20)** suggested increased levels of both LDL and VLDL due to severely impaired catabolism of these particles. They also suggested that increased particle time in circulation leads to further modifications of apoB that will reduce recognition by LDL receptors causing rising level of LDLs. The reduced catabolism is also masked by decreased production.

In the current study, VLDL ranged from 11.3 to 90.1mg/dL with a mean value (± SD) of 37.50 (±15.99) mg/dL. 21 (34.4%) patients were normal, and 40 (65.6%) patients were abnormal.Triglycerides, VLDL-C, and HDL levels were observed to be higher during maintenance hemodialysis. **Shoji et al.,(21)** discovered the same changes when they investigated the role of heparin in the pathophysiology of hemodialysis-induced dyslipidemia.

Additionally, **Singh et al., (22)**reported thatESRD patients on continuous hemodialysis are more likely to develop dyslipidemia, which is characterised by hypertriglyceridemia, high VLDL, and decreased HDL values without regard to gender. Hemodialysis can successfully minimise the accumulation of nitrogenous waste products, however it cannot remove dyslipidemia caused by CRF.

Speer et al., (23) stated that the biosynthesis and the metabolism of lipoproteins are substantially altered in CKD. Lipoproteins from CKD patients are structurally modified by several mechanisms. Such modified lipoproteins promote inflammation, oxidative stress, impaired lipid transport, endothelial dysfunction, and atherogenesis. Thus, lipoproteins play an integral role in the development of CKD-associated CV complications. Lipid-lowering therapies are an important part in the management of CKD patients. However, results of clinical trials of lipid-lowering therapies from the general population can only be partially transferred to CKD patients due to altered metabolism, structure, and function of the lipoproteins.

Conclusion

The pattern of dyslipidemia is abnormally high levels of total serum cholesterol, triglycerides, and low-density were found to be 29.5%, 75.4% and 16.4% respectively. Triglycerides, VLDL-C, and HDL levels were observed to be lower during maintenance hemodialysis

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