ORIGINAL RESEARCH ARTICLE

A CLINICAL STUDY OF DYSLIPIDEMIA IN PATIENTS OF CHRONIC KIDNEY DISEASE

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Abstract: Cardiovascular disease is a major cause of morbidity and mortality in patients with Chronic Kidney Disease (CKD). Patients with CKD exhibit significant alterations in lipoprotein metabolism, which in their most advanced form may result in the development of severe dyslipidemia which can lead to increased risk of cardiovascular complications. The K/DOQI (Kidney Disease Outcomes Quality Initiative) guidelines suggest that all CKD patients should therefore be evaluated for dyslipidemia. Hence this study was undertaken to know the prevalence of dyslipidemia in CKD patients and thereby assess the impending risk of cardiovascular morbidity in these patients. This study was carried out in 100 CKD patients and it was observed that the mean values of Triglycerides (TG), Total Cholesterol (TC), Low Density Lipoprotein Cholesterol (LDL-C), Very Low Density Lipoprotein Cholesterol (VLDL-C), TC/HDL, LDL/HDL were increased and High Density Lipoprotein Cholesterol (HDL-C) was decreased in the patients under study. There was also statistically significant increase in the values of TG, TC, VLDL-C and TC/HDL with the progression of the disease (stage of CKD). Early recognition of dyslipidemia in CKD patients and maintenance of desired lipid levels either through diet or lipid lowering drugs can be helpful in decreasing the risk of cardiovascular complications in these patients.

Key Words: Chronic Kidney Disease, dyslipidemia, cardiovascular disease, TG, TC, LDL-C, VLDL-C, HDL-C.

INTRODUCTION

Chronic Kidney Disease (CKD) is a silent epidemic of the 21st century. Its occurrence is not only confined to the developed countries; it is universal. It has been estimated that the age-adjusted incidence rate of End Stage Renal Disease (ESRD) in India is around 229 per million population[1], and more than one lakh new patients enter renal replacement programs annually in India[2].

CKD leads to disturbances in the function of virtually every organ system. However, it is well documented that Cardiovascular Disease (CVD) is a major cause of morbidity and mortality in patients with CKD. Even mild chronic renal dysfunction contributes actively to the development of CVD, so the American Heart Association has recommended that these patients should be classified in the highest risk group for developing cardiovascular events. In patients who finally advance to ESRD and especially in those on dialysis, the prevalence of coronary heart disease is 40% and CVD mortality is 10 to 30 times higher than in the general population of the same gender, age and race[3].

Patients with CKD are subjected to accelerated atherosclerosis leading to increased cardiovascular complications. Several factors contribute to atherogenesis, most notable among which is dyslipidemia. Patients with impaired renal function exhibit significant alterations in lipoprotein metabolism, which involve all lipoprotein classes and show variations depending on the degree of renal impairment, etiology of primary disease, presence of

nephrotic syndrome and the method of dialysis i.e. hemodialysis (HD) or peritoneal dialysis (PD). Dyslipidemia seen in CKD is characterized by high triglyceride and low high-density lipoprotein (HDL) cholesterol levels, accumulation of remnant particles, predominance of low-density lipoprotein (LDL) particles, and increased levels of lipoprotein a {Lp(a)}[4].

Dyslipidemia has also been hypothesized to cause kidney damage and to play an important role in the progression of renal failure[5]. Dyslipidemia may damage glomerular capillary endothelial and mesangial cells as well as podocytes. Mesangial cells express receptors for LDL and oxidized LDL, which upon activation induce mesangial cell proliferation, increase mesangial matrix deposition, and enhance the production of chemokines such as macrophage chemo-attractant protein-1, cytokines such as interleukin 6, or growth factors. Macrophage chemo-attractant protein-1 enhances the recruitment of macrophages, which can infiltrate the glomerulus and become foam cells that release cytokines causing damage to endothelial cells, mesangial cells and podocytes.

The aim of this study was to determine the prevalence and pattern of dyslipidemia in patients of CKD and to correlate findings of dyslipidemia with the severity of disease (stage of CKD).

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MATERIALS AND METHODS

This study was carried out in 100 cases diagnosed as having CKD, admitted in Medical wards/Nephrology department of Guru Nanak Dev Hospital attached to Government Medical College, Amritsar, after obtaining their informed consent. Diagnosis of CKD was established on the basis of clinical, biochemical and radiological (ultrasonography) findings. Detailed history was taken from each patient with special reference to cardiovascular symptoms.

Routine investigations like hemoglobin, total leucocyte count (TLC), differential leucocyte count (DLC), urine complete, 24hr urinary protein, fasting blood sugar, blood urea, serum creatinine, and electrocardiogram (ECG) were done. Fasting lipid profile was obtained in all the cases selected.

Inclusion criteria:
Diagnosed patients of CKD.

Exclusion criteria:
1. Patients with diabetes mellitus.
2. Patients on lipid lowering drugs.
3. Patients with history of alcohol consumption or smoking.
4. Patients with liver disease.
5. Kidney transplanted patients.

Patients selected for the study were kept fasting for 10-12 hours and about 10 ml of blood was drawn for fasting lipid profile. Total cholesterol and triglycerides in the plasma were measured enzymatically and then the cholesterol in the supernatant was measured after precipitation of Apo-B containing lipoprotein to determine the HDL cholesterol. LDL cholesterol was estimated by using the Friedewald Formula.

LDL-C = Total cholesterol - [HDL-C + (Triglycerides/5)]

VLDL-C was estimated by dividing the plasma triglycerides by 5 reflecting the ratio of cholesterol to triglyceride in VLDL particles.

Analysis of the data: Data collected was statistically analyzed for its significance. p value was determined to evaluate the level of significance. If p value was less than 0.05, observation was considered significant. Lipid classification was done according to NCEP - ATP III Guidelines:

Risk factor	
LDL-C (mg/dl)	**Goal**
< 100	Optimal
100-129	Near or above optimal
130-159	Borderline High
160-189	High
>190	Very high
Total cholesterol (mg/dl)	

<200	Desirable
200-239	Borderline high
>240	High
HDL-C (mg/dl)	
<40	Low
≥60	High
Triglycerides (mg/dl)	
<150	Normal
150-199	Borderline high
200-499	High
>500	Very high

RESULTS

Among the 100 patients studied, 4 were in stage 1 CKD, 9 were in stage 2 while 8, 23 and 56 patients were in stage 3, 4 and 5 respectively. The mean age was 42.50 ± 17.02, 43.33 ± 16.25, 46.75 ± 16.21, 46.00 ± 16.19 and 44.30 ± 20.54 years in CKD stage 1, 2, 3, 4 and 5 respectively. The mean age of total patients enrolled in this study was 44.73 ± 18.50 yrs. There was no statistically significant difference between the mean age of patients in different CKD stages (p value >0.05) and thus were comparable. Out of 100 patients, 57 were males and 43 were females. The mean value of blood urea in study patients was 107.61 ± 55.10 mg% and the mean value of serum creatinine was 6.57 ± 4.69 mg%.

Total cholesterol in patients under study averaged 205.33 ± 27.02 mg%. Mean value of Triglyceride in these patients was 250.91 ± 56.15 mg%, that of LDL-C was 120.29 ± 21.99 mg%. Mean HDL-C was 34.86 ± 3.99 mg% and mean VLDL was 50.18 ± 11.23 mg%. Mean of TC/HDL ratio in these patients was 5.95 ± 0.96 and that of LDL/HDL ratio was 3.49 ± 0.73 (Table 1, Chart 1)

Table 1: Lipid Profile in CKD patients

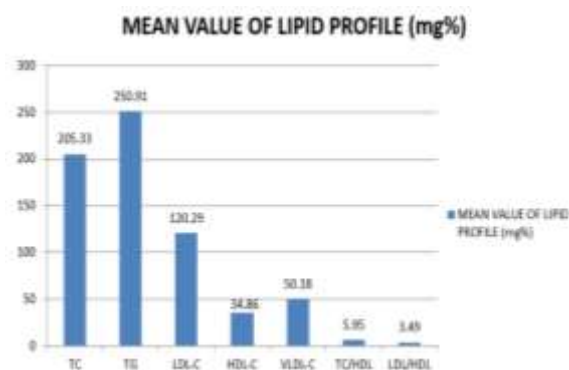| LIPID PROFILE	Range	MEAN ± SD (mg/dl)
Total cholesterol	120.0-286.0	205.33 ± 27.02
Triglyceride	110.0-380.0	250.91 ± 56.15
LDL-C	50.8-178.0	120.29 ± 21.99
HDL-C	26.0-42.0	34.86 ± 3.99
VLDL-C	22.0-76.0	50.18 ± 11.23
TC/HDL	3.16-8.52	5.95 ± 0.96
LDL/HDL	1.34-5.47	3.49 ± 0.73

Chart 1: Lipid Profile in CKD patients

The mean value of triglycerides in total patients was 250.91 ± 56.15 mg%. In CKD stage 1, 2, 3, 4 and 5, the mean values of TG were 188.00 ± 50.68, 219.00 ± 37.66, 216.88 ± 31.94, 232.91 ± 58.90 and 272.79 ± 50.65 mg% respectively. The TG levels increased as the stage of CKD progressed except in stage 3 in which the level was less than that of stage 2 (Chart 2). This increase in TG levels with advancing CKD stage was statistically highly significant (p value <0.001). Among the 100 patients studied, 3 (3%) had normal TG values (<150 mg/dl) while 97 (97%) had elevated TG values. 17 patients (17%) had TG in borderline high range (150-199 mg/dl) range and 80 patients (80%) had TG in high (200-499 mg/dl) range.
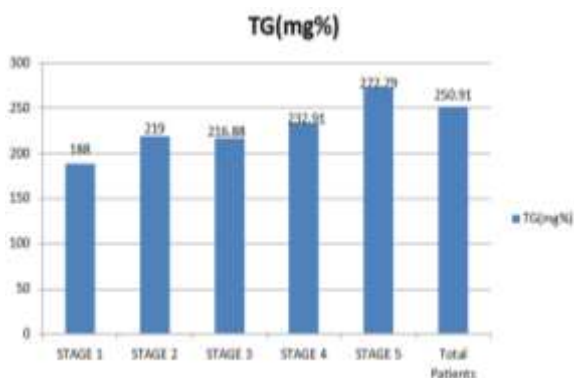

Chart 2: Serum Triglyceride levels among patients in different stages of CKD

The mean value of total cholesterol in study patients was 205.33 ± 27.02 mg%. In CKD stage 1, 2, 3, 4 and 5, the mean values of TC were 175.00 ± 37.61, 192.00 ± 25.02, 206.25 ± 23.92, 203.09 ± 26.08 and 210.43 ± 25.99 mg% respectively. The TC levels increased as the stage of disease progressed except in stage 4 in which the level was less than that of stage 3 (Chart 3). This increase in TC values was statistically significant (p value <0.05). Out of 100 cases of CKD in present study, 39 patients (39%) had TC <200 mg/dl (desirable range) and 61 (61%) had elevated TC values. 53 patients (53%) had TC between 200- 239 mg% (borderline high) and 8 (8%) had TC >240mg% (high).
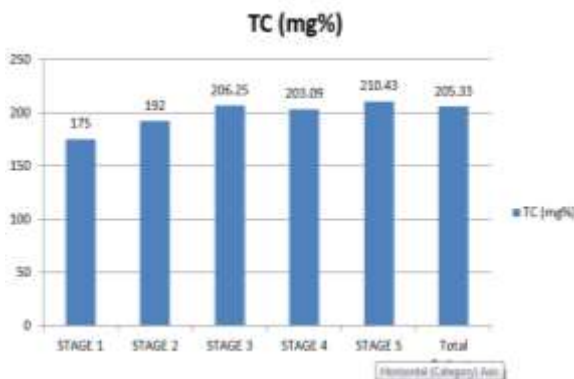

Chart 3: Serum Cholesterol levels among patients in different stages of CKD

The mean LDL-C in total patients was 120.29 ± 21.99 mg%. In CKD stage 1, 2, 3, 4 and 5, the mean values of LDL-C were 101.40 ± 33.89, 112.53 ± 26.10, 127.62 ± 20.05, 121.29 ± 15.88 and 121.42 ± 22.60mg% respectively. The LDL-C levels increased as the stage of disease progressed except in stage 4 and stage 5 in which the levels were less than that of stage 3 (Chart 4). However, the increase in LDL-C values with advancing CKD stage was not statistically significant (p value >0.05). Among 100 patients studied, 19 (19%) had optimal LDL levels (<100mg%), 47 (47%) had near optimal LDL levels (100-129 mg%), 31 (31%) had borderline high LDL levels and 3 (3%) had high LDL levels.
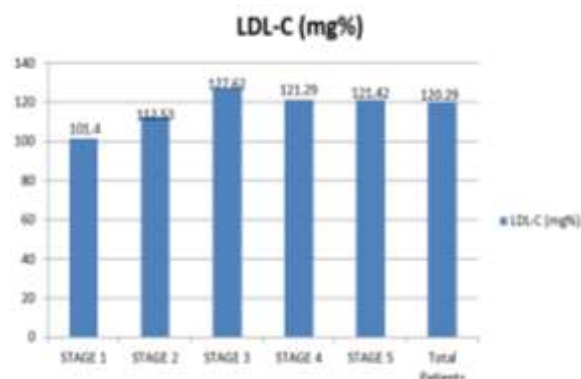

Chart 4: Serum LDL-C levels among patients in different stages of CKD

The mean HDL-C in total patients was 34.86 ± 3.99 mg%. In CKD stage 1, 2, 3, 4 and 5, the mean values of HDL-C were 36.00 ± 2.83, 35.67 ± 3.71, 35.25 ± 3.06, 35.22 ± 3.92 and 34.45 ± 4.28 mg% respectively. The HDL-C levels decreased as the stage of CKD progressed (Chart 5) but the decrease was not statistically significant (p value >0.05). Out of 100 patients studied, 87 patients (87%) had low HDL values <40 mg/dl and 13 patients (13%) had HDL values >40mg/dl.
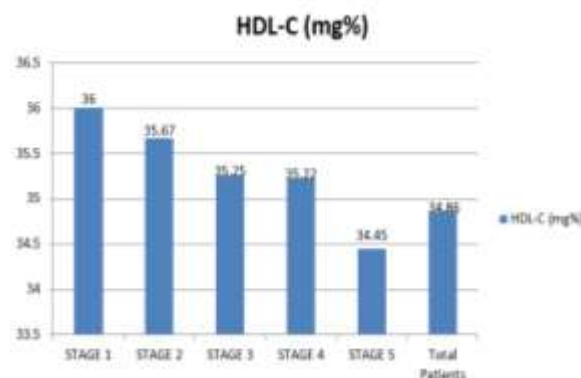

Chart 5: Serum HDL-C levels among patients in different stages of CKD

The mean value of VLDL-C in total patients studied was 50.18 ± 11.23 mg%. In stage 1, 2, 3, 4 and 5 the mean VLDL-C levels were 37.60 ± 10.14, 43.80 ± 7.53, 43.38 ± 6.39, 46.58 ± 11.78 and 54.56 ± 10.13 mg%

respectively. The VLDL-C levels increased as the stage of disease progressed except in stage 3 in which the level was less than that of stage 2 (Chart 6). The increase in VLDL-C values with progression of CKD was statistically highly significant (p value <0.001).
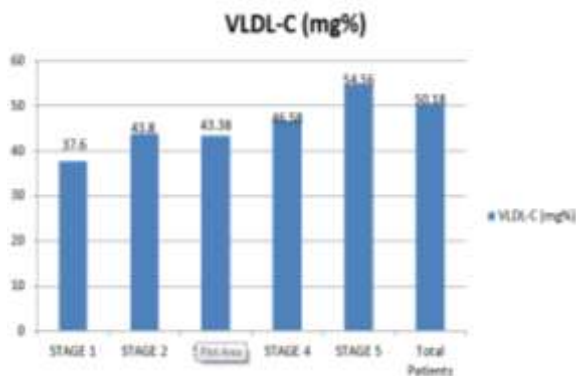

Chart 6: Serum VLDL-C levels among patients in different stages of CKD

The mean value of ratio of TC/HDL in total patients was 5.95 ± 0.96. In CKD stage 1, 2, 3, 4 and 5, the mean values were 4.90 ± 1.17, 5.39 ± 0.54, 5.85 ± 0.48, 5.81 ± 0.85 and 6.19 ± 1.02 respectively. The TC/HDL ratio increased as the stage of disease progressed except in stage 4 in which the ratio was less than that of stage 3. This increase in TC/HDL ratio as the stage of CKD progressed was statistically significant (p value <0.05).

The mean LDL/HDL ratio in total patients was 3.49 ± 0.73. In CKD stage 1, 2, 3, 4 and 5, the mean values were 2.86 ± 1.07, 3.14 ± 0.55, 3.62 ± 0.44, 3.48 ± 0.80 and 3.58 ± 0.73 respectively. The LDL/HDL ratio increased as the stage of disease progressed except in stage 4 and stage 5 in which the ratio were less than that of stage 3, however the increase in LDL/HDL ratio with progression of CKD stage was statistically not significant (p value >0.05).

DISCUSSION

The prevalence of clinical coronary heart disease in CKD patients is 40% and CVD mortality is 10 to 30 times higher than in the general population of the same gender, age and race[3]. Patients with CKD display a clinical picture of early atherosclerosis. Dyslipidemia has been established as a well-known risk factor for CVD in general population and it is noticeable that patients with CKD exhibit significant alterations in lipoprotein metabolism, which in their most advanced form may result in the development of severe dyslipidemia. The mean value of triglycerides in this study was found to be 250.91 ± 56.15 mg% and was increased (high) according to NCEP-ATP III guidelines for dyslipidemia. This result was in concordance with the work done by Bhagwat *et al.,* [6] which showed that hypertriglyceridemia was a fairly constant observation

in CKD patients. Similar results were also seen in studies conducted by Bhansali *et al.,*[7] Massy *et al.,*[8] and Ljufti *et al.,*[9]. In the present study the level of triglycerides increased as the stage of disease progressed and was statistically highly significant (p value <0.001) indicating that triglycerides level goes on increasing as the CKD worsens. This is in concordance with the studies conducted by Kaysen[10] and Peck *et al.,*[11].

Hypertriglyceridemia is one of the most common quantitative lipid abnormalities in patients with CKD. It occurs due to increased synthesis and/or diminished clearance from the circulation. The concentrations of triglyceride-rich lipoproteins (VLDL, chylomicrons and their remnants) start to increase in early stages of CKD and show the highest values in nephrotic syndrome and in dialysis patients, especially those who are treated with PD[12]. CKD is associated with insulin resistance which can promote hepatic VLDL production leading to increased plasma triglycerides. However, the predominant mechanism responsible for hypertriglyceridemia is defective metabolism of TG rich lipoproteins by lipoprotein lipase (LPL) and hepatic lipase. The decrease in enzyme activity may be due to down regulation of enzyme gene and presence of circulating inhibitors of lipolytic enzymes like pre βHDL in uremia and changes in concentration of apolipoprotein C-II (activator of LPL) and apolipoprotein C-III (inhibitor of LPL) with a decrease in apolipoprotein C-II/C-III ratio due to a disproportionate increase in plasma apolipoprotein C-III[13].

The mean value of total cholesterol in study patients was 205.33 ± 27.02 mg%. There was a marginal increase (borderline high) in values of total cholesterol according to NCEP-ATP III guidelines for dyslipidemia. This is in accordance with studies conducted by Attman *et al.,* [14] and Tsumura *et al.,* [15] which showed elevated levels of total cholesterol in CKD patients. It was also observed that the total cholesterol levels increased as the stage of CKD progressed, similar to the results obtained by Kasiske BL *et al.,* [16] in their study.

Hypercholesterolemia in CKD is due to associated proteinuria and renal insufficiency per se. Proteinuria leads to alteration in gene expression for HMG-CoA reductase which results in increased activity of this enzyme leading to hypercholesterolemia. It is also known that LDL receptor mediated cholesterol uptake plays an important role in cholesterol homeostasis. Patients with renal insufficiency alone or in combination with heavy proteinuria exhibit an acquired LDL receptor deficiency which plays a central role in the genesis of hypercholesterolemia in CKD. The proportion of small dense LDL particles, which are considered to be highly atherogenic, is also increased[17].

Mean LDL-C in total patients was 120.29 ± 21.99 mg% which was marginally increased (near or above optimal) according to NCEP-ATP III guidelines. This result was similar to the one obtained in the study by Bhagwat *et al.,* [6] where they found that LDL-C was increased in CKD patients which was not statistically significant. However Ljulfi Z[9] and Bagade *et al.,* [18] in their studies showed significant increase in LDL-C levels in CKD patients. In present study, LDL-C levels increased as the stage of CKD progressed but this was not statistically significant (p value >0.05) and was in concordance with the studies conducted by Kasiske *et al.,* [16] and Deighan *et al.,* [17].

During lipolysis, TG are hydrolyzed to form LDL particles which contain mainly cholesterol ester. LDL particles are subsequently removed by the liver. In uremia there is decrease in catabolism of IDL and LDL leading to their increased plasma residence time. This is accompanied by further modification of the apo-B contained in these lipoproteins by oxidation, carbamylation, and glycation. These modifications lead to reduced recognition and binding of these lipoproteins to LDL receptors in the liver and hence further reduction in plasma clearance. This reduced catabolism, however, is masked by the decreased production of LDL, resulting in near normal plasma levels of LDL in patients of CKD[19].

Mean HDL-C in the present study was 34.86 ± 3.99 mg% and was decreased (low) according to NCEP-ATP III guidelines for dyslipidemia. This was in concordance with the results obtained by Bhagwat R[6], Massy *et al.,* [8] and Morena M *et al.,* [20]. In this study the HDL-C levels decreased as the stage of CKD progressed but the decrease was not statistically significant (p value >0.05) indicating that HDL-C levels does not worsen significantly as the disease progresses.

Patients with CKD generally have reduced plasma HDL-C because they usually exhibit decreased levels of apolipoproteins A-I and A-II (the main protein constituents of HDL)[21], diminished activity of Lecithin-cholesterol acyltransferase (LCAT; the enzyme responsible for the esterification of free cholesterol in HDL particles)[22], as well as increased activity of cholesteryl ester transfer protein (CETP) that facilitates the transfer of cholesterol esters from HDL to triglyceride-rich lipoproteins thus reducing the serum concentrations of HDL-C.

The mean value of VLDL-C in total patients studied was 50.18 ± 11.23 mg% and was increased. Similar results were obtained in the study conducted by Bagade *et al.,* [18]. VLDL-C level increased, as the stage of disease progressed and was statistically highly significant (p value <0.001).

CKD is associated with impaired clearance of VLDL and chylomicrons. This is due to dysregulation of Lipoprotein Lipase (LPL), hepatic lipase, VLDL receptor and impaired HDL metabolism leading to increased levels of VLDL-C. CKD is also associated with hepatic lipase deficiency, which appears to be caused by secondary hyperparathyroidism and dysregulation of cytosolic calcium leading to decreased VLDL clearance[23].

In the present study the mean value of ratio of TC/HDL was 5.95 ± 0.96 and was increased. Similar results were obtained in the studies done by Cheung *et al.,* [24] and Avram *et al.,* [25]. Also, TC/HDL ratio increased as the stage of disease progressed and was statistically significant. Increased TC/HDL-C ratio is indicative of atherogenic risk in ESRD patients. The mean value of ratio of LDL/HDL in total patients was also increased (3.49 ± 0.73), result concordant to the studies of Cheung *et al.,* [24] and Massy *et al.,* [8]. This ratio increased as the stage of disease progressed, however the increase was statistically not significant.

CONCLUSIONS

Patients with CKD are subjected to accelerated atherosclerosis leading to increased cardiovascular complications. Several factors contribute to atherogenesis, most notable among which is dyslipidemia. TG, TC, LDL-C, VLDL-C, TC/HDL, LDL/HDL increase and HDL-C decrease in CKD, however maximum derangement is seen in levels of TG, TC, VLDL-C, HDL-C and TC/HDL. Dyslipidemia in CKD tends to worsen as the disease (stage of CKD) progresses with statistically significant increase in the values of TG, TC, VLDL-C and TC/HDL from stage 1 to 5. LDL-C and LDL/HDL also increase with the progression of disease but less significantly than other lipid derangements. HDL-C on the other hand continues to decrease as the stage of CKD advances but the decrease is not alarming. This study confirms the presence of atherogenic lipid profile in patients of CKD which can lead to increased morbidity and mortality due to additional cardiovascular risks. Hence maintenance of desired lipid levels either through diet or early initiation of lipid lowering drugs can be helpful in decreasing the risk of cardiovascular complications in these patients.

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