

## The Impact of hemodialysis and continuous peritoneal dialysis on lipoprotein(a) concentration and Apolipoprotein(a) Phenotypes in Patients with End Stage Renal Disease

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**Abstract: Back ground:** Elevated the concentration of lipoprotein(a) [lp(a)] and apolipoprotein(a) [apo(a)] phenotypes are an independent risk factor for coronary heart disease (CHD). **Aim:** investigation of lp(a) levels and apo(a) phenotypes in relation to coronary heart disease (CHD) and the effect of dialysis on serum lipoprotein(a) concentration according to apo(a) phenotype in patients with end-stage renal disease (ESRD). **Patients and methods:** This is a case control study among patients attending hospitals and medical dialysis centers in Khartoum capital of the Sudan in March 2011. There were 60 normal healthy subjects (controls), 34 hemodialysis (HD) patients and 29 continuous peritoneal dialysis (CPD) patients. Lipid profiles, Lp(a), and apo(a) phenotypes were determined. **Results:** The frequencies of the subjects with apo(a) phenotypes of high molecular weight (HMW) only, including S4, or S5 or null type were 96.3% of control, 98.2% of HD patients, and 93% of CPD patients. The frequent apo(a) phenotypes in patients consisted of S4, S4S5, S5, and S5S5 isoforms. There were significant different when compared mean levels of Lp(a) concentration between HD and CPD patients and controls [36±8.0 mg/dL; 38±9.0 mg/dL and 18±2.0 mg/dL respectively P<0.05]. The levels of total cholesterol and LDL cholesterol and ApoB levels were higher in CPD patients compared to HD patients. The levels of serum albumin was significantly higher in controls than in HD and CPD patients [4.4±0.3g/L; 4.0±0.4g/L 3.2±0.7g/dL respectively, P<0.05]. **Conclusion:** In (ESRD) patients the levels Lp(a) was increased mainly in CPD patients compared to HD patients and control subjects for common apo(a) phenotypes, which may contribute to the frequent cardiovascular. [Ahmed Mohammed Ahmed, Bader Eldien H. Elabid, Abdelrahman Abdelwahab Rhmtallah, Hussein Yousif Eledum, Akram H. Awadalla Hisham Ali Waggiallah. **The Impact of hemodialysis and continuous peritoneal dialysis on lipoprotein(a) concentration and Apolipoprotein(a) Phenotypes in Patients with End Stage Renal Disease.** *J Am Sci* 2014;10(8):40-45]. (ISSN: 1545-1003). <http://www.jofamericanscience.org>. 5

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### 1. Introduction

Lipoprotein(a), a low-density lipoprotein like particle in which apolipoprotein(a) [apo(a)] is linked to ApoB100 by a disulfide bridge (Berg, 1963), but has a specific apolipoprotein, called apolipoprotein(a) [apo(a)], linked by a disulfide bond to apolipoprotein B100 of LDL (Gazzaruso *et al.* 1999). Apolipoprotein(a) is characterized by a high degree of genetic polymorphism, with many isoforms in plasma (Ilyas Kamboh *et al.* 1991) and (Marcovina *et al.* 1993). Lipoprotein(a) plasma concentrations are largely determined by apo(a) gene, and they are inversely related to the molecular weight (MW) of apo(a) isoforms. Many studies have shown that Lp(a) levels are closely associated with premature coronary heart disease (CHD) (Dahlen *et al.* 1986) and (Rhoads

*et al.* 1986). Apolipoprotein(a) [apo(a)] is linked to ApoB100 by a disulfide bridge, is elevated in end-stage renal disease (ESRD) patients with atherosclerotic cardiovascular disease. The serum Lp(a) levels vary widely, ranging from less than 1 mg/dL to greater than 100 mg/dL, and are influenced by genetically determined apo(a) polymorphism, that is, there is an inverse relationship between serum Lp(a) concentration and the size of the apo(a) isoform (Kim *et al.* 1997). Lp(a) level was significantly elevated in patients with renal failure under dialysis when compared with normal healthy subjects (Kronenberg *et al.* 1995), (Dieplinger *et al.* 1993) and (Hirata *et al.* 1993). Patients with ESRD have a greatly elevated risk for atherosclerotic cardiovascular disease. This increased risk is only partially explained

by traditional risk factors associated with ESRD, prompting interest in novel atherosclerotic cardiovascular disease risk factors, such as lipoprotein(a) [Lp(a)], levels of which are elevated in ESRD (Craig *et al.* 2005). (Bloembergen *et al.* 1995) showed that myocardial infarction and cerebrovascular disease, were significantly higher in CPD treated patients, compared with hemodialysis (HD) treated patients. These phenomena may be explained in part by the more atherogenic lipoprotein profile, including Lp(a) levels, in CPD patients than HD patients (Kim *et al.* 1997) and (Hirata *et al.* 1993). The present study evaluate the distribution pattern of apo(a) phenotypes in patients and the effect of dialysis modality on Lp(a) level in each commonly found apo(a) phenotype.

## 2. Materials and Methods

Twenty nine patients on CPD (17 men, 12 women, age  $55 \pm 4$  (mean  $\pm$  SD) years), 34 patients on maintenance HD (20 men, 14 women, age  $52 \pm 3$  (mean  $\pm$  SD) years), and 60 healthy controls (35 men, 25 women, mean age  $43 \pm 4$  (mean  $\pm$  SD) years) were included. All were Sudanese, they which attended to renal dialysis centers in Khartoum state. Patients with liver disease or those taking corticosteroids or lipid-lowering drugs were excluded from this study.

Sudan is a large country with 30 million inhabitants. CPD was started in 1968, while HD was started in 1973. At present, there are only 16 HD machines serving 56 patients in two centers in Sudan (Suliman *et al.* 1995).

CPD duration ranged from 1 to 40 months. Renal diseases of CPD patients included diabetic nephropathy ( $n = 9$ ) and chronic glomerulonephritis ( $n = 5$ ). The dialysis procedure was performed with four daily exchanges of 2 L of standard dialysis solution. All CPD patients were free of peritonitis and other infections at the time of the study and in the four preceding weeks.

The patients had been treated with HD for 20 months ago. Renal diseases of HD patients included diabetic nephropathy ( $n = 8$ ) and chronic glomerulonephritis ( $n = 6$ ). HD had been performed 4 hours per session, two or three times per week, all using cellulose acetate hollow-fiber dialyzers with acetate or bicarbonate-containing dialysate. Normal controls were attended as co-patients. Persons who had cardiovascular, cerebrovascular, renal, or endocrine disease were excluded by history and physical examination.

### Methods

Fasting blood samples were obtained from patients both group and control group. For HD patients, this was performed before initiation of a HD session, prior to heparin administration. Blood urea

nitrogen (BUN) was measured by urease method, creatinine by the Jaffe method, and albumin by the bromocresol green method, using Roche/ Hitachi 902 full-automated analyzer, (Tokyo, Japan). Residual renal function was estimated by the average of the residual urea and creatinine clearances. Serum pre albumin was measured using laser nephelometry. Serum cholesterol and triglyceride (TG) were measured by enzymatic methods using Roche/ Hitachi 902 full-automated analyzer. High-density lipoprotein cholesterol (HDL-C) was analyzed enzymatically after precipitation of other lipoproteins with heparin and MnCl<sub>2</sub>. Low density lipoprotein cholesterol (LDL-C) level was calculated by the Friedewald formula ( $LDL-C = \text{total cholesterol} - HDL-C - TG/5$ ) (Friedewald *et al.* 1972). Measurement of Lipoprotein(a): by Roche/ Hitachi 902 full-automated analyzer, (wave length 570 nm). The principle of estimation: Lp(a) in sample or standard cause agglutination of the latex particles coated with anti-Lp(a) antibodies. The agglutination is proportional to the Lp(a) concentration in the sample and can be measured by turbidimetry.

Measurement of Apolipoprotein A1: by Roche/ Hitachi 902 full-automated analyzer, (wave length 570 nm). Apolipoprotein A-I in the sample precipitates in the presence of anti-human apolipoprotein A-I antibodies. The light scattering of the antigen-antibody complexes is proportional to the apolipoprotein A-I concentration and can be measured by turbidimetry. Measurement of Apolipoprotein B: by Roche/ Hitachi 902 full-automated analyzer, (wave length 570 nm). Apolipoprotein B in the sample precipitates in the presence of anti-human apolipoprotein B antibodies. The light scattering of the antigen-antibody complexes is proportional to the apolipoprotein B concentration and can be measured by turbidimetry.

### Apo(a) isoform determination

Measurement of apo(a) LMW isoforms: by immunoblotting technique, Briefly, after an electrophoretic run performed on 1% Sodium Dodecyl Sulfate-Polly Acrelamide Gel Electrophoresis (SDS-PAGE), The separated proteins were transferred in nitrocellulose membrane (Bio-Rad, Segrate, Italy) by a capillary blotting technique and tested with a polyclonal antihuman Lp(a) antiserum from rabbit (DAKO, Glostrup, Denmark). A peroxidase-conjugated goat antirabbit (DAKO) was used as the second antibody. Lastly, the membrane was immersed in a developing solution. The reduced specimens were applied to SDS-PAGE (polyacrylamide gel electrophoresis) in a 15% gradient polyacrylamide gel. The separated proteins were transferred to a nitrocellulose membrane and then incubated with sheep antihuman Lp(a) as the first antibody and rabbit anti sheep IgG(Fc)-alkaline phosphate conjugate as

the second antibody. Subsequent treatment with substrate revealed the Lp(a) bands. The apo(a) phenotype standard consisted of B, SI, S3, S4, and S5 isoforms whose kringle IV numbers were 14,19, 23,27, and 35, respectively. Recently (Utermann 1995) suggested that the standardization of apo(a) isoforms be achieved according to their kringle IV repeat number. This categorizes apo(a) isoforms into F, B, SI, S2, S3, S4, and S5, of which the number of kringle IV repeats are 11-13,14-16,17-19, 20-22, 23-25, 26-28, and 29-42; the KpnI allele numbers are 1-3,4-6, 7-9,10-12, 13-15, and 16-32, respectively. The low-molecular weight (LMW) group included all phenotypes with at least one of the isoforms F, B, SI, or S2, which correspond to the number of kringle IV repeats 11 to 22. The high-molecular weight (HMW) group was comprised of subjects with only S3, S4, or S5 isoforms or with the null type. The number of kringle IV repeats is 23-42. This classification was applied in this study.

### Statistical Analysis

Statistical analysis was done by using SPSS version 19. Student's t-test or one-way analysis of variance (ANOVA) with multiple comparisons (Scheffe test) was used to compare biochemical parameters. The difference of Lp(a) phenotype was evaluated by 2x2 table chi-square test. P<0.05 or P<0.01 was considered statistically significant. Lp(a) serum concentration was performed by nonparametric test using SAS program.

### 3. Results

Comparison of clinical and biochemical characteristics of HD and CPD patients and control

group, were shown in Table 1. Blood urea nitrogen and creatinine levels of CPD patients were lower than those of HD patients. A significant difference was found in serum albumin level among controls, HD, and CPD patients showed albumin level significant lower when compared with controls ( $4.4\pm 0.3$ ), HD patients ( $4.0\pm 0.4$ ), and CPD patients ( $3.2\pm 0.7$ ) g/dL. The serum prealbumin concentration was not different between HD and CPD patients. The serum cholesterol level was significantly different among the three groups; the highest level was found in CPD patients, where the lowest level was seen in control group (P <0.05). The serum TG level was significantly higher in CPD patients than in HD patients and control. The serum HDL-C level was significantly lower in CPD and HD patients than in control. The serum LDL-C level was significantly different among the three groups; highest in CPD patients and lowest in HD patients (P <0.05). The serum ApoA1 level was significantly lower in CPD and HD patients than in the control group. The serum ApoB level was significantly different among the three groups: highest in CPD patients, lowest in HD patients (P <0.05). The serum Lp(a) concentration was different among controls and HD and CPD (controls  $18 \pm 2$  mg/dL, HD  $36 \pm 8$  mg/dL, CPD  $38 \pm 9$  mg/dL, P <0.001). There was no correlation between serum Lp(a) level and age or duration of dialysis in both CPD and HD groups.

Frequency distribution of apo(a) phenotype are summarized in Table 2. The frequencies of the HMW group were 96.3% (77/80) of control, 98.2% (56/57) of HD patients, and 93% (40/43) of CPD patients; (not statistically significant difference).

Table 1. Clinical and biochemical characteristics of HD and CPD patients compared to control group.

	Control	HD	CPD
Number of subjects	60	34	29
BUN (mg/dL)		$86 \pm 21$	$56 \pm 27^2$
SCr (mg/dL)		$12.3 \pm 3.5$	$7.9 \pm 3.7^2$
Prealbumin (mg/dL)		$35.4 \pm 10.7$	$32.6 \pm 9.1$
Albumin (g/dL)	$4.4 \pm 0.3^1$	$4.0 \pm 0.4^1$	$3.2 \pm 0.7^{1,2}$
Cholesterol (mg/dL)	$117 \pm 33$	$160 \pm 40^1$	$199 \pm 85^{1,2}$
Triglyceride (mg/dL)	$126 \pm 24$	$141 \pm 33$	$141 \pm 39^1$
LDL-C (mg/dL)	$50 \pm 12$	$139 \pm 11^1$	$160 \pm 12^{1,2}$
HDL-C (mg/dL)	$102 \pm 11$	$91 \pm 16$	$89 \pm 13^2$
Apolipoprotein A1 (mg/dL)	$136 \pm 25$	$116 \pm 25^1$	$113 \pm 29^1$
Apolipoprotein B (mg/dL)	$104 \pm 30$	$89 \pm 33^1$	$126 \pm 62^{1,2}$
Lipoprotein(a) (mg/dL)	$18 \pm 2^1$	$36 \pm 8^1$	$38 \pm 9^1$

HD: Haemodialysis, CPD: Continuous peritoneal dialysis, BUN: Blood urea nitrogen, SCr: Serum creatinine, HDL-C: High density lipoprotein cholesterol, LDL-C: Low density lipoprotein cholesterol, Apo A1: Apolipoprotein A1, Apo B: Apolipoprotein B, <sup>1</sup>: P ≤0.05 compared with control group, <sup>2</sup>: P ≤0.05 compared with HD patients.

Table 2. Frequency distribution of Apo(a) phenotypes in study groups.

	Control		HD		CPD	
	Fr	%	Fr	%	Fr	%
F	1	1.3	0	0	0	0
S4	8	10	11	19.6	6	14
S4S4	3	3.7	6	10.8	0	0
S4S5	14	17.6	7	12.5	9	21
S5	32	40	19	33.9	20	46.5
S5S5	16	20	12	21.4	5	11.5
Null	3	3.7	1	1.8	0	0
LMW <sup>1</sup>	3	3.7	1	1.8	3	7
HMW <sup>1</sup>	77	96.3	56	98.2	40	93
Total	80	100	57	100	43	100

### Fr: Frequency

Lp(a) concentrations according to apo(a) phenotype, only frequent phenotypes are summarized in Table 3. For the S4 and S isoforms, serum Lp(a) was significantly different among the three groups, highest in CPD patients and lowest in normal controls. For the S4S5 and S5S5 isoforms, serum Lp(a) of CPD patients was significantly higher than that of HD patients and normal controls.

Table 3. concentration of lipoprotein(a) according to apo(a) phenotype.

	Control	HD	CPD
S4	17	26 <sup>1</sup>	41 <sup>1,2</sup>
No. of subjects	18	15	8
S4S5	26	39	522
No. of subjects	27	14	15
S5	16	22 <sup>1</sup>	30 <sup>1,2</sup>
No. of subjects	56	27	34
S5S5	22	19	43 <sup>1,2</sup>
No. of subjects	23	17	9

<sup>1</sup>: P ≤0.05 significant compared with control group, <sup>2</sup>: P ≤0.05 compared with HD

### 4. Discussions

The present study agree with (Kim *et al* 1997), in which showed that the majority of the Sudanese population has apo(a) of high-molecular weight. The frequencies of apo(a) phenotypes of high molecular weight only including S3, S4, or S5 or null type were 93% of control, 98% of HD patients, and 93% of CPD patients, which are much higher prevalence than that in the Caucasian population (71.1% in controls, 73.8% in HD patients, and 73.2% in CPD patients) (Kronenberg *et al.* 1995). This finding seems to be compatible with the observation of (Gaw *et al.* 1994) who showed that mean size of the apo(a) alleles in the Chinese population was significantly larger than that

in the Caucasian and African American populations. According to the above finding, respect to the populations of Sudan showed that the frequency of LMW phenotypes was more than 20% in healthy persons, which is much higher than that of this study (Kim *et al.* 1994) and (Abe *et al.* 1992). When we comparing our data to those of (Kim *et al.* 1994) the percentage of no band decreased from 7.3% to 2.0%, and that of double band increased from 38.5-46.4%. Serum Lp(a) level of CPD patients was higher than that of HD patients or normal controls in all frequent apo(a) phenotypes. In the study of a Caucasian population, (Kronenberg *et al.* 1995) also found that Lp(a) of CPD patients was significantly higher than that of HD patients. The higher serum Lp(a) concentration in CPD patients than in HD patients after adjustment of apo(a) phenotypes suggests that CPD modality leads to greater elevation of serum Lp(a) and this agree with (Kim *et al* 1997). Lp(a) level in CPD patients higher than in HD patients, the suggestion lower serum albumin concentration may lead to higher serum Lp(a) level in CPD patients also this finding agree with (Kim *et al* 1997). CPD patients have a lower serum albumin level than HD patients because they lose substantial amounts of protein into the dialysate fluid. (Kronenberg *et al.* 1995) found a significant inverse correlation between serum albumin and Lp(a) concentrations in the cross-sectional studies of HD and CPD patients. (Heimbürger *et al.* 1996) reported that CPD patients have significantly higher Lp(a) levels than HD patients, although serum albumin levels were similar. In the CPD patients, significant correlations were found between plasma Lp(a) level and 24-hour peritoneal and total clearance for albumin. There is a third possible mechanism. CPD patients in this study had higher residual renal function. In a recent report, loss of residual renal function in HD patients was associated with decreased lipoprotein(a) (Variath *et al.* 1996). Higher residual renal function in CPD patients might be associated with more urinary loss of some unidentified Lp(a)-inhibiting substance. Lower blood urea nitrogen and creatinine levels in CPD patients can be explained by higher residual renal function, because adequacy of dialysis was in an acceptable range and nutritional status measured by pre albumin was not different than that of HD patients. The Report of the National Heart, Lung, and Blood Institute Workshop on Lipoprotein(a) and Cardiovascular Disease, presented a brief review of treatment strategies to decrease Lp(a) levels, including niacin, ascorbic acid with l-lysine, estrogen, aspirin, statins, diet, and apheresis. Overall, Lp(a) level is difficult to lower, and most of the medications that do have an effect on Lp(a) levels also treat other atherogenic lipids. Sorting out the

beneficial effect of Lp(a)-lowering medications beyond the cardiovascular benefit of such medications will require carefully designed, randomized, clinical trials (Craig *et al.* 2005).

There were significantly increased of LDL-cholesterol and ApoB in CPD patients when compared to HD patients. Like Lp(a), may be in part explained by the lower serum albumin level in CPD patients. Inverse correlations between serum albumin and cholesterol, LDL cholesterol and ApoB in this study support this possibility. (Stenvinkel *et al.* 1996) showed that elevated LDL-ApoB production rate is highly correlated to the prevailing serum albumin levels in patients with nephrotic syndrome. It is possible that the underlying mechanisms causing these lipid abnormalities could be related. In support of this, (Shoji *et al.* 1992) found a correlation between Lp(a) and LDLcholesterol and ApoB, respectively, in CPD patients. These more atherogenic lipid profiles may be one of the possible causes of the more frequent cardiovascular events (Kronenberg *et al.* 1996).

In conclusion, In (ESRD) patients the levels Lp(a) was increased mainly in CPD patients compared to HD patients and control subjects for common apo(a) phenotypes, which may contribute to the frequent cardiovascular.

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