# EFFECT OF A SINGLE DIALYSIS SESSION ON PLASMA LP(A) LEVELS IN PATIENTS ON MAINTENANCE HEMODIALYSIS

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## BACKGROUND & OBJECTIVE

Cardiovascular disease (CVD) accounts for significant morbidity and mortality in patients with chronic kidney disease (CKD). Besides the higher prevalence of traditional risk factors, several uremia-related factors may play a role in accelerated atherosclerosis, such as elevated levels of lipoprotein (a) (Lp(a)). Lp(a)consists of an LDL-like particle and the specific apolipoprotein(a) (apo(a)), which is covalently bound to the apoB of the LDL like particle. The effect of maintenance hemodialysis (MHD) on Lp(a) levels is not well understood. The present work was carried out to study the Effect of a single Dialysis session on plasma Lp(a) levels in End Stage Renal Disease (ESRD) patients on maintenance hemodialysis. Hourly concentrations of Lp(a) in plasma were measured as well.

## PATIENTS & METHODS

27 ESRD patients on MHD were assigned from Nephrology unit, SVIMS, Tirupati (A.P). Exclusion criteria include smoking and active infection. The patients were under HD since three years. The HD program consisted of four hour dialysis sessions, three times a week. Dialysis was done with polysulfone membrane using bicarbonate dialysate with flow rates of 500ml/min and 200-250 ml/min respectively for dialysate and blood. Anticoagulation was done with 2000 IU of heparin at the start of dialysis followed by continuous administration of 500-1000 IU per hour.

### Sampling & Analysis

Pre HD, hourly and post HD blood samples were collected from the arterial end of the dialyzer. Plasma was obtained by centrifugation at 3000 rpm for 15 minutes. Separated plasma was either analyzed immediately or stored at -80$^0$C for further analysis. Plasma Lp(a) levels were measured by standard methods using commercial kits on Beckman CX9 auto analyzer. Values obtained were corrected for hemoconcentration. The data were presented as mean ± SEM. Statistical significance of the changes was obtained by using analysis of variance for repeated measures under generalized linear model. Statistical significance was established at a p value of .05 or less. Statistical analysis was done using SPSS for windows version 16.0.

## RESULTS

Results of analysis of variance for repeated measures after correction for hemoconcentration where necessary revealed a decrease in Lp(a) (p=0.022) and triglycerides (p=0.001) levels and no change in cholesterol (p=0.48) levels. Hourly plasma concentrations of Lp(a) before and after correcting for hemoconcentration were presented in Table:1.

## DISCUSSION

High Lp(a) predicts risk of early atherosclerosis similar to high LDL, but in advanced atherosclerosis, Lp(a) is an independent risk factor not dependent on LDL. Apo (a) contains domains that are very similar to plasminogen (PLG). Lp(a) accumulates in the vessel wall and inhibits binding of PLG to the cell surface, reducing plasmin generation which increases clotting. This inhibition of PLG by Lp(a) also promotes proliferation of smooth muscle cells. These features of Lp(a) generate clots and atherosclerosis. In addition, because of LDL cholesterol content, Lp-a contributes to atherosclerosis.

Elevated Lp(a) in HD patients has been reported earlier but its role in the development of vascular complications has not yet totally proven. Rather, there is controversy concerning the effect of HD on plasma levels of Lp(a). Dialysis membrane and heparin can influence lipid parameters. However, there are limited and debatable data concerning lipid alterations during a single hemodialysis session. Moreover, the role of hemoconcentration after every hemodialysis session confuses the real effect of the heparin on lipid profile.

In our study we found no change in plasma Lp(a) levels as such after completion of HD. However significant (p=0.022) decrease was observed after correcting for hemoconcentration.
Our findings of decrease in plasma Lp(a) is answered by the clearing effect of heparin on lipids like HDL and LDL lipoproteins. Administration of heparin during HD had pronounced clearing effect for Lp(a). Although studies show an association of Lp(a) and CVD, Lp(a) cannot yet be regarded as a conventional risk factor for CVD. Association of Lp(a) and cardiovascular disease in HD patients is rather complicated, that warrant further research.

## CONCLUSION

Single HD session lowers Lp(a) levels which might be due to the administration of heparin producing clearing effect on plasma Lp(a) during dialysis. Furthermore, decrease in Lp(a) levels may have a beneficial effect on cardiovascular morbidity and mortality

**Table:1: Hourly concentrations of Lp(a) in plasma of HD patients**

| | Pre HD | 1 hour | 2 hour | 3 hour | Post HD | Change (p value) |
|---|---|---|---|---|---|---|
| **Lp(a) - mg/dL** | 21.2 ±2.7 | 22.3 ±2.9 | 22.1 ±3.06 | 22.1 ±2.9 | 20.6 ±2.7 | → (0.14) |
| **Lp(a) - (mg/g Albumin) corrected for hemoconcentration** | 7.41±0.9 | 7.89±1.0 | 8.30±1.1 | 7.81±0.6 | 7.0±0.9 | ↓ (0.02) |