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A COMPARATIVE STUDY ON ENDOTHELIAL DYSFUNCTION MARKER AND CVD MARKER IN CRF, HD (PRE & POST) AND CONTROLS

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ABSTRACT

CRF refers to an irreversible decay of renal capacity, which traditionally creates over a time of years. At first, it is show just as a biochemical irregularity. In the long run loss of excretory, metabolic and endocrine elements of the kidneys prompts the improvement of the clinical side effects and indications of renal disappointment, which are alluded to as uremia. The aim of our study is comparison Endothelial Dysfunction Marker and CVD Marker in CRF, HD (Pre &Post) and Controls. The present study was conducted on a patient group comprising of 50 patients diagnosed with CRF, 50 were pre hemodialysis, 50 were post hemodialysis (MHD). This patient group was compared to a healthy group including 100 controls. All the study participants were admitted in the department of Medicine [Nephrology department] at "Fathima Institute of Medical Sciences", Kadapa. Andhra Pradesh. India. The obtained serum samples were used for biochemical analysis for estimating nitric acid and homocystine. Cardiovascular diseases are common in patients with CKD, and CKD is a major risk factor for CV diseases. In our study increased nitric acid levels were seen in pre HD, Post HD and CRF patients. In our study increased Hcy levels were seen in pre HD, Post HD patients.

Keywords: Nitric acid, Homocystine, CVD, chronic kidney disease, Endothelial Dysfunction

INTRODUCTION

Renal failure term is used primarily to denote failure of the excretory functions of the kidneys leading to retention of nitrogenous waste products of metabolism. Various other aspects of renal function may fail at the same time, including the regulation of fluid and electrolyte status and the endocrine function of the kidney, a wide range of clinical manifestations may therefore occur **[1]**. An imbalance in favor of pro-oxidants can lead to the oxidation of macromolecules resulting in tissue injury **[2]**. CRF refers to an irreversible decay of renal capacity, which traditionally creates over a time of years. At first, it is show just as a biochemical irregularity. In the long run loss of excretory, metabolic and endocrine elements of the kidneys prompts the improvement of the clinical side effects and indications of renal disappointment, which are alluded to as uremia **[3]**.

Mortality from cardiovascular disease in renal failure patients is three and a half times that of an age matched healthy population. Heart disease accounts for more than 50% of the deaths in uremic patients. Anemia and hypertension commonly seen in patients before they begin dialysis, contribute to left ventricular hypertrophy and congestive heart failure. Secondary hyperparathyroidism can lead to metastatic calcification in the myocardium, cardiac valves, and arteries **[4]**. Accelerated atherogenesis is responsible for the high prevalence of coronary artery disease in this population and the high rate of recurrent coronary artery stenosis after angioplasty. Pericarditis can occur in uremic patients before they start dialysis and in patients who are already undergoing dialysis. Initiation of dialysis or intensifying dialytic therapy usually results in resolution of pericarditis in patients who are receiving inadequate dialysis. Pericarditis occurring in the setting of adequate dialysis may not respond to a further increase in dialysis therapy and may require surgical drainage or the use of nonsteroidal anti-inflammatory agents **[5]**.

Endothelial dysfunction is thought to be a key event in the development of atherosclerosis and is shown to be of significance in vascular events including stroke and heart attacks **[6]**. Inability of arteries and arterioles to dilate fully in response to an appropriate stimulus, intra-arterial administration of various vasoactive agents, localised heating of the skin and temporary arterial occlusion by inflating a blood pressure cuff to high pressures, a clinical reason for intracoronory,

catheterization These techniques are thought to stimulate the endothelium to release nitric oxide (NO) and possibly some other agents, which diffuse into the surrounding vascular smooth muscle causing vasodilation **[7]**.

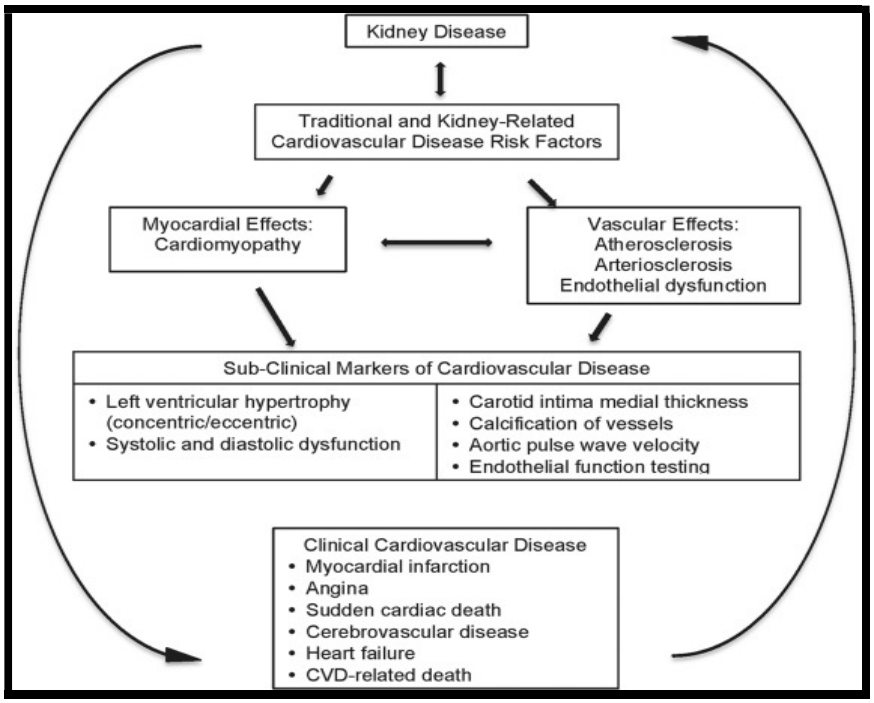

Figure 1: Cardiovascular disease risk with kidney disease

Nitric oxide (NO) is one of the main factors involved in the anti-atherosclerotic effects of the endothelium, 2 and chronic renal failure (CRF) has been associated with impaired NO bioavailability in the absence of concomitant risk factors, **[8]** even in children. However, the finding of a reduced NO bioavailability as demonstrated by functional studies does not provide insight into the mechanisms causing endothelial dysfunction, because reduced bioavailability can be the result of decreased NO production, increased NO degradation, or both **[9]**.

MATERIAL AND METHODS

All the Patients were admitted in the department of Medicine [Nephrology department] at "Fathima Institute of Medical Sciences", Kadapa. Andhra Pradesh. India. Present study were divided into four groups.

- Group-I: healthy controls - 100.
- Group-II –CRF Patients - 50.
- Group-III: CRF patients on haemodialysis (Pre HD) -50.
- Group-IV: CRF patients on haemodialysis (Post HD) - 50.

The exclusion criteria were patients with no clinical or laboratory evidence of diabetes mellitus, liver diseases, lupus nephritis, acute illness, respiratory diseases. None of the patients had history of antioxidant drugs supplementation.

The study was conducted after obtaining Institutional Ethical Committee clearance and informed consent was obtained from the study participants. Five ml of whole blood samples were drawn into plain tubes to obtain serum samples after centrifugation at 4000rpm for 10 minutes. The obtained serum samples were used for biochemical analysis for estimating nitric oxide, oxi-LDL and Homocysteine levels.

Statistical Analysis

All the values were expressed as mean and standard deviation (mean ± SD). The statistical analysis were done by using one-way analysis of variance (ANOVA) using SPSS for windows version 11.5 (SPSS, Inc., Chicago). A p-value of <0.001 was considered to be statistically significant.

RESULTS

Laboratory data showed a significant change in the levels of biochemical parameters in CRF, pre and post hemodialysis groups in comparison to normal controls **(Table 1)**. All the data was presented as mean and standard deviation. P value of < 0.001 was considered statistically Significant.

Table 1 Shows the mean ± SD of Endothelial Dysfunction Marker and CVD Marker Homocysteine in CRF, HD (Pre &Post) and Controls. In our study a significant increase of NO values were seen in pre & post HD and CRF when compared with controls. Increased NO levels attributed to the blood membrane interactions platelet membrane interactions increases cytokine levels due to general inflammatory cells produced by HD. increased NO levels in post HD induction of iNOS by cytokines resulting in transcription of mRNA for iNOS takes at least 2hrs and the following protein synthesis every layer. Decreased NO levels: After dialysis lower NO values due to its elimination during dialysis session. Prevailing over its production and attributed it to loss of L – argenine in the dialysate. In our study a significant increase of Hcy values were seen in pre & post HD and CRF when compared with controls.

Graph of all the investigations of this study were plotted by using the healthy controls - 100. CRF Patients -50.CRF patients on haemodialysis (Pre HD) -50.CRF patients on haemodialysis (Post HD) - 50. Individual values of all investigations done in CRF Pre and Post HD and in control subjects.

Table 1: Comparison of Endothelial Dysfunction Marker and CVD Marker in CRF, HD (Pre &Post) and Controls

S. No.	Parameter	Control	Pre HD	Post HD	CRF	P value
1	NO (µmol/lit)	54.50±3.18	89.15±7.13	81.36±6.67	83.23±7.78	<0.001
2	Hcy (µmol/lit)	10.88±0.77	19.35±1.42	10.84±0.76	19.18±1.46	<0.001

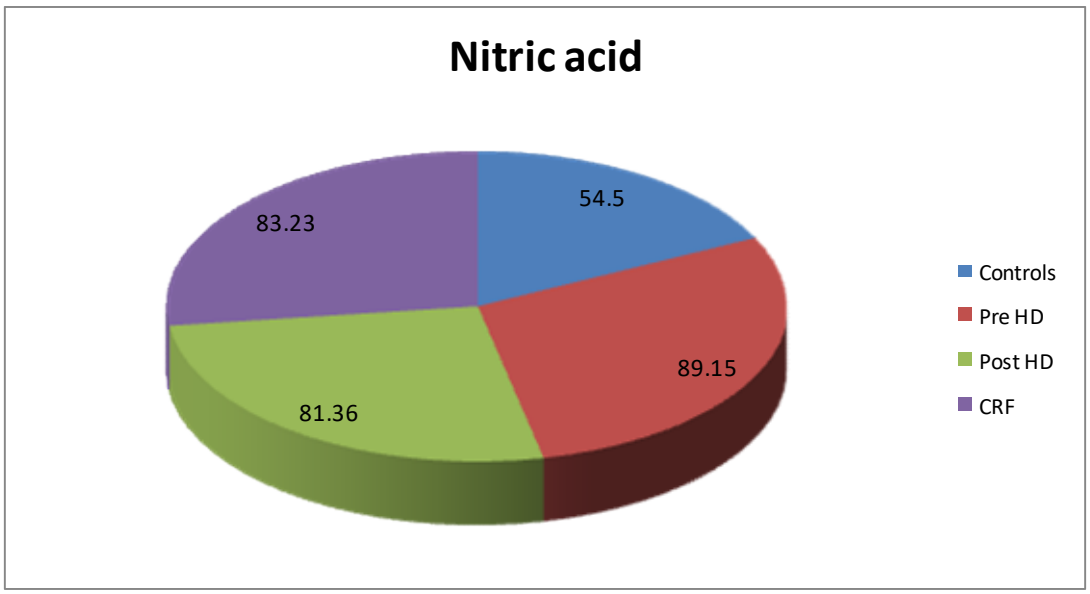

Figure 2: Shows Nitric acid levels in Controls, Pre HD, Post HD and CRF

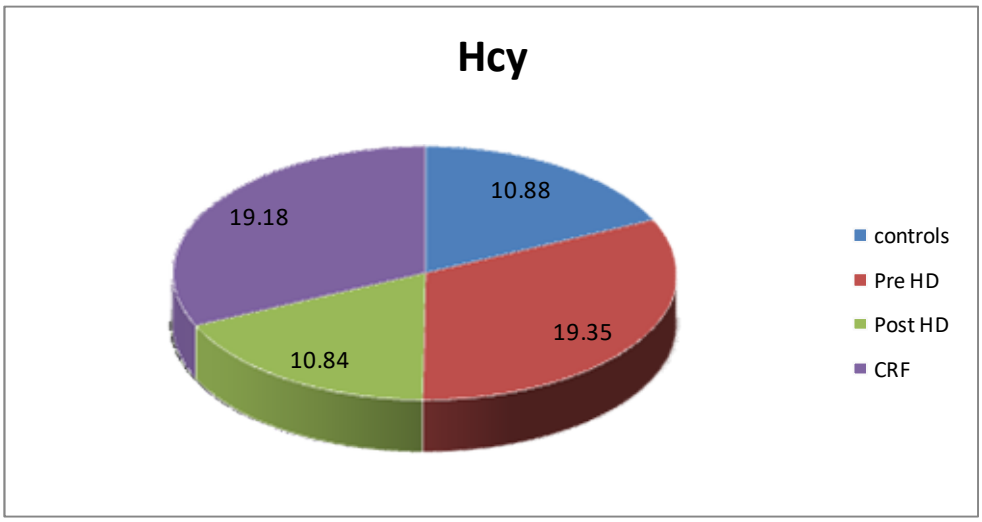

Figure 3. Shows Homocystine levels in Controls, Pre HD, Post HD and CRF

DISCUSSIONS

In the present study shows the mean ± SD of Endothelial Dysfunction Marker and CVD Marker Homocysteine in CRF, HD (Pre &Post) and Controls. In our study a significant increase of NO values were seen in pre & post HD and CRF when compared with controls. Increased NO levels attributed to the blood membrane interactions platelet membrane interactions increases cytokine

levels due to general inflammatory cells produced by HD. increased NO levels in post HD induction of iNOS by cytokines resulting in transcription of mRNA for iNOS takes at least 2hrs and the following protein synthesis every layer. Decreased NO levels: After dialysis lower NO values due to its elimination during dialysis session. In our study a significant increase of Hcy values were seen in pre & post HD and CRF when compared with controls.

Cross et al. also showed (using brachial artery studies based on flow mediated vasodilatation/hyperaemia) that dialysis improves ED vascular reactivity, whereas the EID responses remained unchanged. At the same time, reductions in plasma concentrations of ADMA and homocysteine were recorded **[10]**.

Miyazaki *et al*. **[11]** and Kosch *et al*. **[12]**, demonstrated an acute worsening of the brachial artery ED vasoreactivity, following dialysis with a cellulosic membrane. This was related to a significant increase in the oxidative stress caused by a bio incompatible membrane, and was abrogated by using a vitamin E-coated HD membrane or a poly sulphone membrane. These studies have generally demonstrated relatively small changes in endothelium dependent (NO mediated) vasodilatation and an enhancement in endothelium independent vasodilatation at the end of HD **[11-13]**.

CONCLUSION

Cardiovascular diseases are common in patients with CKD, and CKD is a major risk factor for CV diseases. In our study increased nitric acid levels were seen in pre HD, Post HD and CRF patients. In our study increased Hcy levels were seen in pre HD, Post HD patients. Many traditional and nontraditional risk factors also contribute to CV diseases. In this manner, it is critical for doctors to perceive the patients in danger and execute early counteractive action and treatment procedures.

Conflict of Interest: Nil

Source of Funding: Self/Diagnostic kits are provided by institution as on complimentary basis for research.

Ethical Clearance No: No.IEC/C:62/2016

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