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Comparative Study of Oxidative Stress and Antioxidant Enzymes in Post-Hemodialysis Patients and Healthy Controls

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ABSTRACT

Background: *Chronic kidney disease (CKD) is associated with increased inflammation. Oxidative stress (OS) arises when there is an imbalance between free radical production and antioxidant defense. Thiobarbituric acid-reactive substance (TBARS) is a marker of oxidative stress. In patients treated with regular hemodialysis, the activity of enzymatic and non-enzymatic antioxidative systems is reduced. Oxidative stress damage is further compounded in patients requiring dialysis therapy due to the bio-incompatibility of dialysis membranes in hemodialysis or solutions in peritoneal dialysis.*

Methodology: *The study includes total N 70; healthy controls (35) and post hemodialysis (HD) patients (35) with age and sex matched undergoing dialysis treatment in Hemodialysis unit of a tertiary care. The levels of oxidative stress markers such as GSSG/GSH, TBARS, Oxidized GSSG along with the activities of antioxidant enzymes Catalase, reduced glutathione (GSH), superoxide dismutase (SOD), Glutathione peroxidase (GPX) were analyzed by spectrophotometric method.*

Results: *The levels of TBARS, ratio of GSSG/GSH (0.6297±0.6178) and antioxidant enzymes SOD (48.594±14.540), GPX (226.257±179.811) significantly increased in post HD patients compared with control subjects. Similarly, the levels of GSSG and antioxidant enzymes CAT, GSH were significantly decreased in post HD patients compared with control subjects.*

Conclusion: *In post HD patients the oxidative stress increased with markers such as TBARS and GSSG/GSH with a concomitant decrease in antioxidant systems such as GSH and catalase. The oxidative stress is due to the use of bio-incompatibility membrane during dialysis therapy which is reduced by the use of antioxidant coated membranes*

Keywords: *Oxidative stress (OS), TBARS-Thiobarbituric acid reactive substance, GSSG (Oxidized glutathione) GSH (reduced glutathione), CAT (catalase), GPX (Glutathione peroxidase)*

INTRODUCTION

Chronic kidney disease has arisen as a major health crisis worldwide due to its substantial association with high mortality and morbidity rates. Rise in the incidence of both diabetes and hypertension are known to be the major risk factors contributing to the development of CKD (1,2). In recent years, the burden due to CKD has been felt in terms of economy and health care systems in India as well, with a rising incidence of 12% - 21% in different regions of India (3).

CKD is associated with increased inflammation. (4) Reactive Oxygen Species (ROS) are free radicals which are constantly being produced in our body during various metabolic processes. When healthy, our body is endowed with the ability to deal with these attackers with the help of antioxidant mechanisms. Trouble starts brewing when the subtle balance tilts in favour of the free radicals. They gain an upper hand while the defence mechanism lags behind. Cellular antioxidants are overwhelmed by repeated oxidative insults. The production of ROS soon cascades leading to a scenario of tissue damage, cell death and disease. ROS cause tissue damage by a variety of different mechanisms which include DNA & protein damage, lipid peroxidation, stimulating release of pro-inflammatory cytokines, etc.(5)

This disturbance in the delicate equilibrium of pro-oxidants: Antioxidant in favour of the pro-oxidants is termed as oxidative stress (6). Oxidative stress results from increased production of reactive oxygen radicals or impairment of the antioxidant system (7). Oxidative stress arises when there is an imbalance between free radical production and antioxidant defense. As a result, certain biomolecules are oxidized, leading to structural and functional modifications of these molecules (8). Thiobarbituric acid-reactive substance (TBARS) is a marker of oxidative stress, which is a simple, inexpensive but less specific method to evaluate oxidative stress (9). It has been demonstrated that TBARS levels are elevated in coronary artery disease (10). In short, oxidative stress represents an imbalance between the production and manifestation of ROS and a biological system's ability to readily detoxify the reactive intermediates or to repair the resulting damage. Oxidative stress may be the sole cause of some disease but more often it weakens the immune system and makes the body vulnerable to diseases caused by other factors. It may also worsen existing conditions and slow down healing process (6).

In patients treated with regular hemodialysis, the activity of enzymatic and non-enzymatic antioxidative systems is reduced. The decreased activity of antioxidant enzymes (superoxide dizmutase, glutathione peroxidase) is due to reduced concentration of trace elements, such as selenium, copper and zinc. Concentration of trace elements is reduced due to insufficient input, but also increased loss during hemodialysis session (8). Because of the lack of vitamin C and vitamin E, the capacity of non-enzymatic antioxidative protection systems is reduced (11,12).

Oxidative stress damage is further compounded in patients requiring dialysis therapy due to the bio-incompatibility of dialysis membranes in HD or solutions in peritoneal dialysis (PD) (13,14). Oxidative stress is further exacerbated when these molecules accumulate in the context of renal dysfunction and thus provoke further inflammatory responses. Emerging clinical evidence has revealed that oxidative stress and inflammation correlate with adverse outcomes among CKD patients (15-17).

The effect that an HD session may have on OS is a controversial issue. For some authors, HD would aggravate OS due to activation of inflammatory cells caused by the use of bioincompatible membranes and net losses of soluble antioxidants in water (18-20) or by generation of free radicals (21, 22). The degree of inflammatory response is closely related to the incidence and mortality due to cardiovascular events in CKD patients (23). Susla et al conclude in their study that oxidative stress and chronic inflammation are important factors in the development of cardiovascular calcification in CKD patients and this plays a very important role in the functional activity of the endothelium (24). The resulting atherosclerosis increases the risk of cardiovascular complications in these patients (25,26). The exact role of inflammation as a trigger of oxidative stress remains poorly defined and it is not yet clear as to which is the initial culprit: inflammation or oxidative stress(27).

In the present study TBARS, Oxidized GSSG for oxidative stress markers and catalase (CAT), reduced GSH, Glutathione peroxidase (GPX), superoxide dismutase (SOD) for antioxidant markers are compared between post hemodialysis patients and healthy controls.

MATERIALS AND METHODS:

This retrospective study was carried out at Hemodialysis unit in a tertiary care centers at South India. Total participants (N 70) sample size; Thirty five (35) patients recruited were those aged 18 years to 75 years with sex matched population, who receive two or three hemodialysis sessions per week. Thirty five (35) healthy adults matched by age and sex served as a control group. All participants provided written informed consent. The protocol approved and cleared by institutional ethical and research committee.

The data included the patients name, age and gender, duration of maintenance hemodialysis, BMI, Blood pressure and smoking status. Inclusion criteria were on patients on maintenance conventional HD as a constant modality of renal replacement therapy (thrice weekly), chronic renal failure from any cause and

signed written consent to participate in the study. Exclusion criteria were smokers, diabetics and those on antioxidant supplement and lipid lowering medications.
5ml of venous blood was collected in serum vials from the HD dialysis patients after HD process and healthy controls. The analysis samples were divided into heparinized tubes, EDTA-containing tubes and tubes without anticoagulant. Plasma EDTA and serum were obtained by centrifugation at 1200g within 45min of venesection and stored at -80°C until assay
Post dialysis samples of patients and healthy controls were analyzed in the major laboratory for oxidative stress such as TBARS (Lipid Peroxidase LPO assay), Oxidized GSSG and antioxidant such as Catalase, reduced glutathione (GSH), superoxide dismutase (SOD), and Glutathione peroxidase (GPX) by spectrophotometric method . Ratio of GSSG/GSH calculated as a oxidative stress analysis.
All Statistical analysis in the study performed using SPSS for window version 20.0 and results summarized as a mean ± Standard deviation (SD) by Independent 't' test (comparing with two groups) at the significance level $p<0.05$

RESULTS:
From table 1; based on gender males are higher in frequency % than females. Based on age, Post HD patients (54.314±11.385) are significantly higher than controls.
From table2; oxidative stress marker TBARS (54.314±11.385), ratio of GSSG/GSH (0.6297±0.6178) significantly increased in post HD patients than controls. Similarly, GSSG (88.285±83.998) significantly decreased in post HD patients than controls. Antioxidant enzymes such as CAT (53.000±43.898), GSH (29.342±19.143) significantly decreased in post HD patients than controls. Similarly, SOD (48.594±14.540) and GPX (226.257±179.811) significantly increased in post HD patients.

DISCUSSION
Worldwide, the incidence of end-stage renal disease (ESRD) in the elderly has risen in the past decades resulting in a rapidly growing number of older patients starting haemodialysis (28, 29). The present study of mean age in post HD is (54.314±11.385). The ageing phenomenon in the dialysis population is amplified by a more liberal acceptance of older patients on dialysis, better survival of dialysis patients and reduced access to transplantation for elderly patients. The elderly have a higher prevalence of comorbidities that increase the burden of dialysis, and a substantially higher mortality rate compared with younger counterparts (30, 31).
Women undergoing maintenance hemodialysis have substantially higher risks of hospitalization and 30-day readmission than men (32). In our study females 7(20%) were lower than males 28(80%). Furthermore, the survival advantage that women have over men in the general population is markedly diminished in hemodialysis patients(33,34).In fact, young women on dialysis (<45 years old) have a higher risk of mortality compared with men, mainly due to non-cardiovascular events(32).
The basic marker of oxidative stress is the level of TBARS – thiobarbituric acid reactive substances (most often dialdehydes). Their formation results from degradation by free radicals of polyunsaturated fatty acids present in lipids (35). Although much more elevated in patients undergoing HD(36,37) TBARS plasma levels are less elevated in conservatively-treated patients with renal failure(36,37). In our study TBARS (54.314±11.385) increased in post HD patients than controls.
Oxidative stress is high in patients treated in chronic hemodialysis (HD) program, as evidenced by increased lipid peroxidation, e.g. elevated malondialdehyde (MDA), while low antioxidant, e.g. decreased glutathione peroxidase levels(38-46) (35-43). In our present study glutathione peroxidase GPX (226.257±179.811) was increased in post HD patients than controls
Meenakshi Sreeram et al (4) found that MDA levels are lower in controls as compared to patients with CKD. As compared to early stages of CKD, later stages of CKD exhibit significantly higher levels of MDA, which implies that oxidative stress increases with disease progression. On the contrary, decreased MDA with HD has also been pointed out, and the work by Himmelfarb et al.(47,48) stands out, showing the beneficial effects of HD on major plasma amino-thiols (cysteine, homocysteine, cysteinyl glycine, and gluthathione), which are important markers of oxidation.
Oxidative stress is further exacerbated by HD treatment itself (49). Furthermore, level of MDA is significantly correlated with the severity of kidney dysfunction (50), as well as with the duration of HD program (46).

Biasioli et al. (51) studied the effect produced by several types of membranes used during hemodialysis and they observe a decrease in OS throughout HD with more biocompatible membranes; they also performed post-dialysis determinations showing an improvement of the different markers, and 30 minutes after completing HD with MDA values becoming similar to those pre-dialysis. Similarly, polysulphone membranes resulted in higher plasma levels of MDA and reduced GSH-Px activity and selenium plasma levels, compared with modified cellulose (hemophan) membrane in maintenance HD patients (52).

SOD and GPx are the enzymes of antioxidant defence in our body. As oxidative stress increases, the defence mechanism is overwhelmed by the repeated onslaught of the ROS, resulting in lower levels of these enzymes. It is also possible that persons with CKD have a lower concentration of antioxidant enzymes making them susceptible to the disease in the first place. These results are in agreement with other studies which also found higher MDA and decreased SOD and GPx values in HD patients as compared to controls (36, 40, 53-56).

All available studies in literature are unanimous in saying that MDA values increase with increasing oxidative stress. However, some studies report higher SOD values in HD patients as compared to controls (57, 58). The present study showed increased SOD (48.594±14.540) in Post HD patients than controls.

Kose et al (59) reported an increased erythrocyte glutathione peroxidase and superoxide dismutase activities in the postdialysis group when compared with predialysis group. But there was a decrease in the activity of these enzymes in post dialysis group when compared with control group. In our study both GPX and SOD increased in post HD patients than controls.

Increase in superoxide anion generation is due to the blood membrane interaction during dialysis. Increase in concentration due to increased synthesis is unlikely to occur during the short course of dialysis insult (<6 hrs.). The increase observed is mostly due to its increased activity in defense against superoxide radicals (60). This is in agreement with Rico et al (61) who have reported an increase in erythrocyte SOD activity. Some earlier studies found a decrease in erythrocyte SOD as a result of a dialysis session, which was attributed to the presence of an activating factor in uremic plasma (62).

Roxborough et al (63) found that after HD plasma GSH-Px activity increased and reached the value observed in the control group. Both teams thought that the decreased activity might be attributed, at least in part, to the inhibition caused by ligands or toxic agents of endogenous nature. These inhibitors are most likely removed by dialysis. Catalase is the other enzyme which can act on H2O2. Mimic-Oka J et al (43) found low catalase activity in hemodialysis patients. Sommerburgetal (64) demonstrated increase in catalase activity as a result of dialysis. In our study catalase (53.000±43.898) decreased in post HD patients than controls

Increase in formation of hydrogen peroxide as a result of dismutation of superoxide radicals by SOD causes increased activity of GPX & catalase. However, due to the difference in Km, the contribution of GPX & catalase in detoxification of H O is different. GPX acts at low hydrogen peroxide concentration whereas catalase plays a role when GPX pathway reaches saturation. An increase in catalase activity observed might be due to low levels of GPX observed in dialysis patients coupled with increased generation of hydrogen peroxide above the capacity of GPX (65-67).

Gluthathione is a tripeptidicthiol found in the inside of all animal cells and likely is the most important cellular antioxidant. Oxidized gluthathione (GSSG) is highly toxic to cells so that the organism tends to reduce GSSG to GSH through gluthathione reductasa. Thus, determining GSSG/GSH ratio is considered a reliable estimate of the degree of cellular oxidative stress (68,69). The present study showed decreased GSH and GSSG with increased GSSG/GSH ratio in post HD patients than controls.

In contrast, Ceballos-Picot and colleagues (69) reported increased GR activity in the erythrocyte of patients on hemodialysis. Superoxide dismutase, glutathione peroxidase and catalase, together with glutathione, form the main line of defense against ROS in erythrocytes. There was an increase in SOD activity (P = 0.001) as a result of dialysis session showed by prabakar reddy et al.(60)

The pentose phosphate pathway supplies NADPH required by GR to generate reduced Glutathione. It is known that this pathway is impaired in the uremic state leading to a decreased content of reduced Glutathione in erythrocytes which is required for GPX activity. The increased GR activity suggests that there may be a transient improvement in the pentose phosphate pathway because of improvement in the

uremic state due to dialysis. However, variations occur in the impairment of Pentose Phosphate Pathway and this possibly explains the difference observed in the enzyme activity, in different studies. Increased GR activity has been suggested to be a defensive mechanism by which glutathione availability is preserved (70).

Limitations of the study are the small sample size compared between post HD patients and controls.The study consists of only two markers of oxidative stress such as TBARS and GSSG. The study further to be analyzed with large sample size of pre-post HD and controls with additional markers of oxidative stress such as 8 hydroxy 2 deoxyguanosine, MDA (Malondialdehyde).

CONCLUSION:

In post HD patients the oxidative stress increased with markers such as TBARS and GSSG/GSH with decrease in antioxidant system markers such as GSH and catalase. The oxidative stress is due to the use of bio-incompatibility membrane during dialysis therapy (10, 11) which is reduced by the use of antioxidant coated membranes. (68)

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Table 1: Based on gender (frequency %) and age (mean±SD) between Post HD patients and healthy controls

SL. No	Contents		Post HD Patients (N=35)	Healthy controls (N=35)	TOTAL (N=70)
1	**Gender** (Frequency %)	Male	28 (80.0)	9 (25.7)	35 (100.0)
		Female	07 (20.0)	26 (74.3)	35 (100.0)
2	**Age**		(mean±SD)		t(p value)
			54.314±11.385	42.371±17.064	3.444 (0.001*)

*95% significance Level

Table 2: Comparison of Oxidative stress (TBARS, GSSG, GSSG/GSH) and Antioxidant (SOD, CAT, GPX)

SL .No	Parameters	Patients (mean±SD)	Healthy controls (mean±SD)	t (P-Value)
1	TBARS	54.314±11.385	42.371±17.064	9.607 (0.000*)
2	GSSG	88.285±83.998	359.714±64.829	15.134 (0.000*)
3	GSSG/GSH	0.6297±0.6178	0.1343±0.0715	4.712 (0.000*)
4	GSH	29.342±19.143	50.000±30.097	3.426 (0.001*)
5	SOD	48.594±14.540	6.721±4.826	16.169 (0.000*)

6	CAT	53.000±43.898	72.028±18.245	2.368 (0.021*)
7	GPX	226.257±179.811	188.571±44.068	1.204 (0.233)

*95% significance Level

TBARS-Thiobarbituric acid reactive substance, GSSG (Oxidized glutathione) GSH (reduced glutathione), CAT (catalase), GPX (Glutathione peroxidase)