Original Research Article

Study of serum malondialdehyde levels in chronic renal failure Patients: A hospital based study in Govt. general hospital, Anantapuramu, Andhra Pradesh

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ABSTRACT
Chronic Kidney disease is a recognized public health problem with a huge social and economical impact on the individual, family, society and country. Renal failure is a systemic disease and induces a slow and progressive decline of kidney function aggravated by various factors such as diabetes, infections, autoimmune diseases and toxic chemicals.

Aim: The study was aimed to estimate malondialdehyde, urea and creatinine levels in Chronic renal failure (CRF) patients.

Materials and Methods: The study was conducted in the department of Biochemistry, Government medical college, Anantapuramu, Andhra Pradesh. Study subjects were divided into 2 groups: Group - 1: healthy controls (35) with age group 35-65 years, Group - 2: CRF patients (35) age group 35-65 years. Blood Sample was analyzed for plasma glucose, Blood urea, serum creatinine and serum Malondialdehyde.

Results: The mean and standard deviation of Serum Malondialdehyde, blood urea and serum Creatinine in Chronic renal failure patients (Group-2) was higher compared to controls (Group-1) \((p < 0.0001)\).

Significant positive correlation was observed between serum Malondialdehyde and serum creatinine. A positive correlation was observed between serum Malondialdehyde and blood urea \((r= 0.40576)\).

Significant positive correlation existed between Malondialdehyde and serum creatinine \((r=0.46832)\).

Conclusion: In our study on chronic renal failure patients and age matched healthy controls, the renal parameters – urea, creatinine and BUN were elevated. In addition Malondialdehyde was found to be significantly elevated showing a pro oxidant status in CRF patients

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1. Introduction
Chronic Kidney disease is a recognized public health problem with a huge social and economical impact on the individual, family, society and country. Chronic renal failure is a syndrome characterized by progressive and irreversible deterioration of renal function due to slow destruction of renal parenchyma, eventually terminating in death when sufficient nephrons have been damaged. Chronic kidney disease is a growing problem that affects approximately 12% of the adult population. Major risk factors of CKD are diabetes mellitus, hypertension, glomerulonephritis, urinary tract infection, autoimmune diseases, kidney stones and toxic effects of drugs like Non steroidal anti-inflammatory drugs, Vancomycin, polymyxins, Sulfadiazine, Cisplatin etc.

Blood urea and serum creatinine, are good indicators of a functioning status of kidney and increase in the serum indicates kidney dysfunction. Urea is the end product of protein metabolism. The concentration of urea generally increases as the age advances. Blood urea is increased in all forms of kidney diseases. Blood urea nitrogen is an index of nitrogenous end products of protein and amino acid catabolism and is an indirect measurement of renal function that measures the amount of urea nitrogen in blood and is directly related to protein metabolism and excretory function of kidney. Creatinine is a excretory end waste product formed from creatine phosphate and is dependent

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on total muscle mass of the body. It is not affected by diet, age, or exercise. Creatinine level more than 1.5 mg/dl indicates impairment of renal function. Malondialdehyde is a 3 carbon low molecular weight aldehyde that is produced from free radical attack on polyunsaturated fatty acids (PUFA). The estimation of serum Malondialdehyde (MDA) is used to assess oxidative stress, and free radical damage to the body. The present study aimed to estimate malondialdehyde, urea and creatinine levels in chronic renal failure (CRF) patients.

2. Materials and Methods

The study has been approved by institutional ethical committee. Consent was obtained from the subjects whose sample was collected after explaining the purpose of study. The study comprised of total 70 subjects in the age group of 35 - 65 years, among which 35 were Chronic Renal Failure patients and 35 were age and sex matched healthy subjects who constituted the control group. Cases suffering from other chronic diseases with abnormal renal functions were excluded from the study.

Blood samples were collected at Government general hospital, Anantapuramu, Andhra Pradesh. Five ml of fasting venous blood samples were collected in plain vacutainer tubes in the morning after an overnight fast. Blood samples were centrifuged and serum was used for the estimation urea (DAM method), Creatinine (Jaffe’s method), and plasma glucose (GOD-POD) by using commercially available kits on semi auto analyzer in clinical biochemistry lab. Serum Malondialdehyde was estimated (TBARS method) colorimetrically. Blood urea nitrogen (BUN) was calculated by using the formula: BUN= Blood urea/2.14.

2.1. Statistical analysis

In data analysis, comparison of parameters between two groups were done by using unpaired t-test. All the data are expressed in mean and Standard deviation (SD). For the statistical significance, student’s t-test was performed using Graph pad software. Test of probability less than <0.05 was considered as significant.

3. Results

In the present study, the mean value of blood urea and serum creatinine, blood urea nitrogen and serum Malondialdehyde levels in chronic renal failure (Group-2) patients were significantly elevated when compared to controls (Group-1) as shown in Table 1.

Serum Malondialdehyde (MDA) showed significant positive correlation with Serum creatinine (r= 0.46832), blood urea (r =0.40576) and blood urea nitrogen (r= 0.40568) as shown in Table 2.

4. Discussion

Chronic kidney disease (CKD) is a progressive reduction in renal function and is becoming a major health problem. By comparing the values of study group with control group it was found that serum creatinine values (6.36±0.54) were found to be much higher than control group (0.9±0.18). Our findings were consistent with the works of other investigators (D Pandya et al 2016, Nisha R et al 2017, SN Sridhar AV et al. Blood urea levels are quite sensitive indicators of renal disease, becoming elevated when renal function drops 25-50% of normal. Generally urea accumulation in blood of kidney failure patients arise from the degradation of food and tissues such as muscle. The high levels of urea in blood leads the body very sick unless remove d from the blood streams by kidneys. In the present study, the mean value of serum blood urea nitrogen levels in chronic renal failure patients is high when compared to healthy controls. In the present study, the mean value of serum Malondialdehyde (MDA) in chronic renal failure (CRF) patients is significantly high compared to healthy controls. The obtained result was consistent previous studies by Devecchi AF et al 2009, Rusu CC et al 2016, SN Sridhar AV et al 2018. Significant positive correlation existed between serum Malondialdehyde and serum creatinine (r =0.46832) and also between serum Malondialdehyde and blood urea (0.40576) as shown in Table 2. Free radical induced lipid peroxidative tissue damage has been found to coexist in the pathogenesis of chronic renal failure. Oxidative stress results from the imbalance between oxidative and anti-oxidative mechanisms with increased levels of pro-oxidant and depletion of antioxidants, leading to tissue damage. Reactive oxygen species (ROS) can react with polyunsaturated fatty acids producing lipid hydroperoxides. Malondialdehyde (MDA) is used as a biochemical marker for the assessment of lipid peroxidation. In the present study, the mean blood urea nitrogen levels in chronic renal failure patients are higher compared to healthy controls. Our study findings
Table 1: Comparison of biochemical parameters between chronic renal failure (Group-2) and controls (Group-1)

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Group 1 (n=35)</th>
<th>Group 2 (CRF patients) (n=35)</th>
<th>t -value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting plasma glucose (mg/dl)</td>
<td>92±7.2</td>
<td>165.4±40.2</td>
<td>10.63</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Post prandial plasma glucose (mg/dl)</td>
<td>124.5±9.9</td>
<td>195. 9±50.07</td>
<td>8.287</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Blood urea (mg/dl)</td>
<td>27.9±3.1</td>
<td>87.54±15.9</td>
<td>21.76</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Serum creatinine (mg/dl)</td>
<td>0.91±0.18</td>
<td>6.36±0.54</td>
<td>62.6</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Blood urea nitrogen (mg/dl)</td>
<td>13.0±1.4</td>
<td>40.8±7.4</td>
<td>21.83</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Serum Malondialdehyde (µ mol/L)</td>
<td>1.29±0.2</td>
<td>4.26±1.04</td>
<td>25.75</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

Table 2: Correlation of Malondialdehyde with serum creatinine, blood urea and blood urea nitrogen

<table>
<thead>
<tr>
<th>Parameters</th>
<th>r-value</th>
<th>t-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MDA with serum creatinine</td>
<td>0.46832</td>
<td>11.112</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>MDA with blood urea</td>
<td>0.40576</td>
<td>30.933</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>MDA with blood urea nitrogen</td>
<td>0.40568</td>
<td>28.997</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

are similar with previous studies of Kamal A et al 2014,17 Saki M et al 2019.11 In CKD, declining kidney function which is characterized by chronic elevation of Blood urea nitrogen (BUN) is pathological state that promotes the formation of isocyanate. In fact it was reported that the average plasma concentration of cyanate, which was 45 mmol/L in healthy individuals, increased up to 141mmol/L in patients before dialysis. Accordingly, when kidney function declines accompanied by accumulation of urea, the burden of carbamylation naturally increases.11

5. Conclusion

In our study on chronic renal failure patients and age matched healthy controls, the renal parameters – urea, creatinine and BUN were elevated. In addition, Malondialdehyde was found to be significantly elevated showing a pro oxidant status in CRF patients. The authors recommend further studies in this context to evaluate anti oxidant status in CRF patients, to delay the progression of damage and thereby prolong life free of dialysis.

5.1. Acknowledgement

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6. Conflict of interest

None.

7. Source of funding

None.

References


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