Negative Effects of Nephrotic Syndrome on Pulmonary Function Test Parameters

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Abstract: Nephrotic syndrome is a non-specific kidney disorder characterized by hypo proteinurias, hypo-albuminemia, and edema. Edema termed as excess amount of accumulation of fluid in the interstitial space. In nephrotic syndrome there is an increased risk of edema in the pleural cavity. This decreases the efficacy of pulmonary system in the patients. The aim of the study is to analyze the pulmonary function test parameters among specified nephrotic syndrome patients who are on stage 4 end stage renal failure. Totally 50 subjects were participated in our study among them 25 patients was evaluated with nephrotic syndrome. Among them 16 were male subjects and 9 were female subjects. The age group of whole 25 patients was 40 ± 13 (Group I), 25 subjects were served as control group. Age group was between 42 ± 8 and both the genders were included in our study (Group II). FVC (% pred), FEV (% Pred), FEV1 / FVC, (% Pred), MVV (% Pred), PEFR (25-75%). There was a decreased in Pulmonary functions parameters such as FVC, FEV1, FEV1 / FVC, MVV, PEFR in Nephrotic Syndrome patients (Group I) when compared with control group (Group II). Patients who are on end stage chronic renal failure stage IV with Nephrotic syndrome showed a decreased in pulmonary function test parameters. The reason behind this is due to ascites and edema throughout the body. Ascites inflates the diaphragm to move upward which reduces the respiratory endurance among patients.

Keywords: Nephrotic syndrome, Pleural cavity, Pulmonary function test parameters

INTRODUCTION

Many possible pulmonary complications of renal failure have been described in past studies. Nephrotic syndrome is a non-specific kidney disorder characterized by hypoalbuminemia, abdominal ascites, edema, and hyperlipidemia, decreased coagulation status among chronic renal failure. Nephrotic syndrome is characterized by an increase in permeability of the capillary walls of the glomerulus leading to the presence of high levels of protein passing from blood through urine. The cause for hypo coagulation is due to damage to the glomeruli that alters the capacity to filter the substances transported in the blood. The severity of the damage caused to the kidneys can vary and leads to a lot of complications in other organs and systems [1]. Major of the nephrotic syndrome affects the kidney by decreasing the pore size of podocytes. Although it allows cells proteins to pass through it, by contrast in nephrotic syndrome red blood cells pass through the pores causing hematuria [2].

Pathophysiology of nephrotic syndrome

The renal glomerulus filters the blood that reaches the kidney. It is formed of capillaries with small pores that allow small molecules to pass through that have a molecular weight of less than 40,000 Daltons, but not larger macromolecules such as proteins. In nephrotic syndrome, the glomeruli are affected by an inflammation that allows proteins such as albumin, antithrombin or the immunoglobulin to pass through the cell membrane and appear in urine [4]. Albumin is the main protein in the blood that is able to maintain oncotic pressure, which prevents the leakage of fluid into the extracellular medium and the subsequent formation of edema. As a response to hypoproteinemia the liver commences a compensatory mechanism involving the synthesis of proteins, such as alpha-2 macroglobulin and lipoproteins. This affects the pulmonary system by increasing the inflammatory mediated responses in the blood along with increases in inflammatory markers. Nephrotic syndrome virtually affects the pulmonary system by causing pulmonary edema and pleural effusion attributed to fluid over load and increases the pulmonary capillary permeability. Rarer complications include pulmonary fibrosis, pulmonary hypertension, haemosiderosis and pleural fibrosis. Apart from the complications immunosuppressive drugs causes increases the risk of infection and inflammation which decreases the lung compliance. Uremia also has a major role in reducing
the efficiency of pulmonary system efficiency by blocking the major arteries of circulation [5].

MATERIALS AND METHODS

The study was conducted in Vinayaka mission university karikal in year 2010. Totally 50 subjects were participated in our study among them 25 patients was evaluated with nephrotic syndrome 16 were male subjects and 9 were female subjects the age group of whole 25 patients were 40 ± 13 (Group I). 25 subjects were served as control group. Age group was between 42± 8 and both the genders were included in our study (Group II). Control group subjects were free from renal diseases. Basic renal function parameters with evidence based analysis were done. The purpose of the study was explained to the institutional ethical committee and clearances were obtained.

Recording of pulmonary function parameters

Totally 50 subjects were participated in our study. All the subjects were instructed about the procedure. All the parameters were recorded by proper instructions, followed by demonstration. Totally three trails were recorded off that three trails least value is taken for statistical analysis. All the parameters were recorded in sitting position with erect spine posture. Each trail was recorded with the time gap of 2-5 minutes interval. Pulmonary function parameters analyzed are FVC (% pred), FEV1/FVC, (% Pred), MVV (% Pred), PEFR (%(25-75%)).

Table 1: Shows the difference in pulmonary function test parameters in two groups. (Group I) Nephrotic syndrome patients. (Group II) control group subjects

<table>
<thead>
<tr>
<th>Sl. No.</th>
<th>Parameters</th>
<th>Group I Nephrotic syndrome patients</th>
<th>Group II control group subjects</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>FVC (% pred)</td>
<td>84.38±3.80</td>
<td>98.39±2.13</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>2.</td>
<td>FEV (% Pred)</td>
<td>89.17±6.34</td>
<td>93.81±13.2</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>3.</td>
<td>FEV / FVC, (% Pred)</td>
<td>103.31±1.39</td>
<td>105.42±0.008</td>
<td>0.33</td>
</tr>
<tr>
<td>4.</td>
<td>PEFR (25-75%)</td>
<td>91.78±5.33</td>
<td>116.81±6.83</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>5.</td>
<td>MVV (% Pred)</td>
<td>75.43±8.13</td>
<td>96.33±8.33</td>
<td>&lt;0.01*</td>
</tr>
</tbody>
</table>

p value <0.05 *considered as statstically significant.

The results of our study showed significant changes in pulmonary function test among the two groups. Group I Nephrotic syndrome patients, Group II control group subjects. FVC (% pred) in Group I Nephrotic syndrome patients was around 84.38±3.80 98.39±2.13 in Group II control group subjects. FVC (% pred) was decreased in Group II when compared to Group I. P Value <0.01* FEV (% Pred) in Group I Nephrotic syndrome patients was around89.17±6.34 93.81±13.2 in Group II control group subjects. FEV (% Pred) was very less when in Group II when compared to Group I P Value <0.01*. FEV / FVC, (% Pred) Group I Nephrotic syndrome patients was around103.31±1.39 105.42±0.008 in Group II control group subjects. P value <0.01*.PEFR (% Pred) in Group I Nephrotic syndrome patients was around91.78±5.33 116.81±6.83 in Group II control group subjects. PEFR (25-75%) in Group I Nephrotic syndrome patients was around84.38±8.13 96.33±8.33 in Group II control group subjects. MVV (25-75%) decreased in Group II when compared to Group I. P Value <0.01*.  

Statistical analysis

Student paired” t “test was used to analysis between the variables. Interpretations of data were done by SPSS software 16.0 version. The values are analyzed accordingly for the study.

DISCUSSION

In our study we analyzed 50 patients as two groups. Group-I included nephrotic syndrome patients and group II healthy control subjects who are free from renal diseases. Our main findings in the study is Nephrotic syndrome along with chronic renal failure decrease the pulmonary system efficiency by reducing FEV, FVC, FEV1/FVC, PEFR, MVV. The common pathological condition of the lungs in chronic renal failure with Nephrotic syndrome is usually pulmonary edema it is due to combination of fluid over load and abnormal permeability of the pulmonary micro circulation. Most of Our patients were free from overt edema; minor degrees of fluid retention, uremic pulmonary toxicity are likely to be multifactorial and may differ with different patterns of renal disease. A study conducted by Knudson RJ et al. in 1983 stated that chemical toxic factors due to uremia affects the lung functions. From the study they found that the pulmonary function parameters level was reduced in Nephrotic syndrome (chronic renal failure) patients [6]. The major cause for decrease in pulmonary function in Nephrotic syndrome patients is due to in taking of more amount of steroid therapy which causes sub-acutealveoli is, decrease the respiration muscle efficacy. This was proved by a study conducted by Koerts-de Lang E et al. in 2000 [7]. Neder JA et al. in1999 from their study they showed that pulmonary edema would be favored by increased vascular permeability, fluid over, load and low serum albumin concentration, causes ascites which inflates diaphragm abnormally causes
reduced in pulmonary function. Uremic pulmonary edema, and ascites may progress to interstitial fibrosis, this could be evidence four decreased in pulmonary function [8]. A study conducted by et al in Nidus et al. In 1969 stated that the reduction in FVC, FEV, FVC/FEV, MVV, and PEFR is due to decreased inspiratory muscle strength and its correlation with proportional loss and weakness of expiratory muscle strength. They suggested that the muscle strength parameters were the main component with the greatest influence on impairment of lung function in chronic renal failure patient [9]. Another study conducted by stated that the main cause of decrease in pulmonary function is due to long duration of corticosteroid therapy, recurrent infections in the respiratory passage, protein imbalance in the microcirculation interstitial fibrosis and calcification of lung parenchyma and bronchial free reduces the FVC, FEV, FEV1/FVC, and PEFR in chronic renal failure patients.Owens MW et al. in 1987 stated that decrease in the pulmonary function in renal failure is due to uremic myopathy which decreases the respiratory muscle mass, the other cause for decrease in respiratory muscle mass is due to oxidative metabolic stress, which decreases the muscle protein synthesis, which affects the calcium plasmatic concentration [10].

CONCLUSION

From our study we conclude that abnormalities of lung functions are common in both Nephrotic syndrome and in renal failure. Minimal dosage of steroid therapy drugs with correction of uremic may increase the pulmonary function in patients. Correction of ascites by albumin therapy may improve the anatomical position of the respiratory tract and inflation of diaphragm which may improve the pulmonary function efficiency in the Nephrotic syndrome population.

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REFERENCES