Level of antioxidant vitamins in children suffering from pneumonia

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Abstract

Oxidant antioxidant balance is essential for the normal lung function. Both an increased oxidants and/or decreased antioxidants may reverse the physiologic oxidant antioxidant balance in favour of oxidant leading to lung injury. The aim of present study was to examine the levels of vitamin E & C in children who were suffering from pneumonia. 40 pneumonia children and 40 controls matched with age were analyzed for the study. We observed that highly significant decrease in the concentrations of vitamin E & vitamin C was seen in children with pneumonia compared to controls (p<0.001) respectively. Also there was increased ratio of vitamin E & vitamin C in patients with pneumonia as compared to the controls.

Key words: Antioxidants, Vitamin E, Vitamin C, Pneumonia
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Introduction

The most widespread and fatal of all acute disease, pneumonia is now ‘captain of the men of death’ [1]. The most common cause of illness in children & one of the most common indications for imaging in children are the respiratory tract infections. Primarily each year an estimated 4 million children in developing countries die from pneumonia [2].

Pneumonia is a serious infection or inflammation in which the air sacs fill with pus and other liquids [3]. Viruses such as influenza virus, respiratory syncytial virus, are responsible for 45% of the episodes of pneumonia identified in hospitalized children in Dallas. But there are several non-infectious causes also, but are not limited to aspiration of food, or gastric acid, foreign bodies, hydro-carbons and lipid substances, hypersensitivity reactions and drugs or radiation induced pneumonitis [4].

Materials and Methods

The present study was conducted on patients from pediatric OPD in the general hospital, Sangli & Government medical college & hospital, Miraj. The age limit in study group ranges from 5 to 15 years. The chest radiograph by the pediatrician confirmed pneumonia. Total of 40 pneumonia patients were included in the study and 40 individuals of the same age limit in the control group. The informed consent was obtained from patient’s parents & approved by the ethical committee of institution.

The sample collection of patients has been excluded after 24 hours of admission. The patients with pneumonia who had human immunodeficiency virus (HIV) infection, tuberculosis, and fungal infection were excluded from the study. Blood samples were withdrawn by using 5 ml disposable syringe and needle. Blood samples were collected into plain bulb for vitamin E & in heparin bulb for vitamin C. The samples in plain bulb were allowed to clot for half an hour followed by centrifugation for 15 min at 2000 rpm.

The vitamin E was measured by Baker & Frank Myhod-1968. In this method, serum vitamin E (tocopherol) can be measured by their reduction of ferric to ferrous ions which then form a red complex with α,α’-dipyridyl. Tocopherol
and carotene were first extracted into xylene and absorbance was read at 460 nm to measure carotene. A correction for carotene was made after adding ferric chloride and was read at 520 nm. The plasma vitamin C was measured by DNPH method [6] in which (ascorbic acid) vitamin C was oxidized & further converted to diketogulonic acid in strong acid solution. It further forms a diphosphorylhydrazone by reaction with 2,4-dinitrophenyl hydrazone. Hydrazole dissolves in strong sulphuric acid solution to produce red colored complex measured spectrophotometrically. This method measures both ascorbic acid & dihydroascorbic acid. However, as very little dihydroascorbic acid exists in blood, so it provides substantially the same results.

All the values obtained were expressed as mean ± SD. Student 't' test was applied to compare the difference in means between control and study groups. The difference were considered as highly significant if 'p' value was <0.001.

Results

The results shown in the Table 1 indicated a highly significant difference between the means of vitamin E and vitamin C between study group and control group.

The levels of vitamin E and vitamin C were found to be low in the study group (patients) as compared to control group which are highly significant. Also the ratio of vita-min E to vitamin C was higher in patients with pneumo-nia than the control group as shown in Table 2.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Patients (study group)</th>
<th>Control group</th>
<th>Level of significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin E (mean ± SD)</td>
<td>5.43±2.07</td>
<td>13.53±1.1</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>Vitamin C (mean ± SD)</td>
<td>0.40±0.12</td>
<td>1.18±0.27</td>
<td>P&lt;0.001</td>
</tr>
</tbody>
</table>

Table 2. The ratio of vitamin E to vitamin C in controls an patients from Pneumonia.

<table>
<thead>
<tr>
<th>Study group</th>
<th>Vitamin E/ vitamin C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls</td>
<td>11.46</td>
</tr>
<tr>
<td>Patients</td>
<td>13.57</td>
</tr>
</tbody>
</table>

Discussion

The pulmonary system, as its direct contact with the ex-ternal environment is mainly exposed to a variety of nox-iou-s agents e.g. bacteria, which are the major cause of pneumonia. Phagocytic burst, an essential part of the de-fence system, results in large production of reactive oxy-gen species. [7] The involvement of oxygen free radicals has been associ-ated with a no. of pulmonary diseases such as acute respi-ratory distress syndrome (ARDS), bronchopneumonic dysplasia, emphysema, pneumocononiosis, hyperoxia, bleomycin toxicity, cystic fibrosis or bronchial asthma. The source of oxidants varies considerably for each spe-cific case.

Oxygen free radicals are by products of normal aerobic metabolism. They are inactivated by antioxidant mecha-nisms which includes enzymes such as superoxide dismu-tase, catalase, glutathione peroxidase & others such as albumin, uric acid, lactoferrin, β-carotene, vitamin C & vitamin E. [7]
Imbalance between oxidant and antioxidants establishes cellular damage and pathophysiological disorder. Increased generation of ROS in vivo can lead to the depletion of one or more antioxidants. Loss of individual anti-oxidants (e.g., ascorbate or glutathione) can be measured as an index of oxidative stress. [8]

During pneumonia, a massive influx of activated phagocytes into the lower airways is observed. These cells are the first line of defense against invading microorganisms. Polymorphonuclear neutrophils and macrophages kill these microorganisms by using ROS, lysosomal enzymes including proteinases and antibiotic proteins. Increased oxidative stress in blood has been reported in patients with pneumonia. [9]

In the present study we observed the significant fall in the levels of antioxidants vitamin E & vitamin C in pneumonia patients than in controls. These results are similar to those of Cemek, Caksen et al (2006) [10] They observed that the vitamin C and E levels were lower in the study group compared with control group. All antioxidant activities were decreased in acute pneumonia. The study demonstrated that oxidative stress was increased whereas enzymatic and non-enzymatic antioxidant activities were significantly lower in children with pneumonia.

Raghunath R Rai and Madhavi S. Phadke (2006) [11] also confirmed the fact that in different respiratory disorders the status of plasma oxidants and antioxidants vitamin C & vitamin E showed decreased levels than in controls. Similar results were reported by K. Katsoulis et al (2005) [7] and Suzy A. A. Comhair et al (2002) [12] The results of K. Katsoulis et al showed decreased serum total anti-oxidant status in patients with pneumonia, indicating the presence of oxidant/antioxidant imbalance, probably due to the increased oxidative load.

Earlier it was hypothesized that the alveolar space can recruit additional antioxidant activity from epithelial lining fluid [12]. The multiplicity of the antioxidant system available to the lung and their overlapping specific activities suggest that to maintain pulmonary cellular function, it is critically important for the lung to adequately control redox balance. Disequilibrium either through increased oxidant stress or decreased antioxidant resources can result in the lungs that culminate in cellular death and pul-monary dysfunction.

Thus all the results in the present study, when taken together suggested the lowered activity of vitamin E & vitamin C may be due to the oxidant-antioxidant imbalance and increased oxidative stress. As oxidant-antioxidant balance is essential for the normal lung function, the imbalance between oxidant-antioxidant may lead to lung injury. This disequilibrium may cause pathophysiological changes in the lung that culminate in the cellular death and pulmonary dysfunction either through increased oxidative stress or decreased antioxidant resources.

References

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