ORIGINAL ARTICLE

Effect of Long Term Exposure to Cotton Dust on Blood Pressure in Textile Mill Workers

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ABSTRACT

Background: Industrial pollution is a major occupational problem in developing countries. With rapid industrialization, the cotton dust induced lung diseases are poised to become a global health problem. The cotton dust penetrates the lungs of exposed persons and acts as a pro-inflammatory component which damages the structure of pulmonary endothelium. The endothelial damage results in shedding of angiotensin converting enzyme and as a result, angiotensin II also decreases.

Aim: To determine and correlate blood pressure and serum angiotensin II levels with duration of exposure to cotton dust in textile mill workers.

Methods: This study was conducted in a textile mill of Faisalabad. Eighty four conveniently selected workers participated in the research study who had been working in cotton industry for last 3-10 years. Data collection was done via questionnaire, blood pressure was measurement manually and serum angiotensin II levels were estimated via ELISA.

Results: There was no significant difference of systolic B.P between the three groups, control group, group II and group III. Serum angiotensin II levels were significantly lower in group II and group III as compared to that in control group.

Conclusion: Exposure to cotton dust damages the pulmonary endothelium and ultimately decreases the levels of serum angiotensin II in exposed persons.

Keywords: Cotton dust, Airway hyper responsiveness, Endothelial dysfunction.

INTRODUCTION

Air pollution is a major occupational problem in various industries. Workers of the industries involved in processing of cotton, especially manufacturers of yarn, thread and fabric are mostly exposed to cotton dust[1]. Harvested cotton consists of a mixture of leaves, bracts, stems, fiber, bacteria, fungal spores, pollen grains and endotoxins[2]. The workers are at the risk of many acute and chronic complications such as acute respiratory distress syndrome, byssinosis, chronic allergies and chronic obstructive pulmonary diseases (chronic bronchitis and asthma), all due to exposure to a dusty work site[3]. WHO World Health Report 2002, estimated that air pollutants are associated with a mortality rate of about 1% for respiratory tract infections[4].

In human beings, these invisible cotton dust particles enter into the alveoli of the lung through inhalation in dusty environment and accumulate in the lymph. These ultra fine particulate matter induce apoptosis and necrosis of macrophages and respiratory epithelial cells and hence the air way reactivity increases[1,4]. These particles produce proinflammatory mediators such as leukotriene B4, interleukin-8 and tumor necrosis factor-alpha, which have the ability to elicit a local inflammatory response in the lung tissues and thus results in endothelial damage[5].

Pulmonary endothelium is an active organ having numerous physiological, immunological, and metabolic functions. It represents a dynamic interface between flowing blood and vessel walls, and also produces a variety of factors that regulate the blood flow[6]. Due to the high vascularisation, the lungs are the major site for production of circulating angiotensin converting enzyme (ACE) and pulmonary endothelium is its rich source[7]. The surface of the capillary endothelium also acts as a main site for the conversion of hormone angiotensin I (ANG I) to angiotensin II (ANG II) by ACE through renin angiotensin system[8].

As kidneys are the main long term regulators of blood pressure through renin angiotensin system .The angiotensin II controls the human arterial pressure through various mechanism like arterial vasoconstriction, sodium reabsorption and by releasing aldosterone[9,10].

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Angiotensin converting enzyme is expressed mainly by pulmonary capillaries endothelium as compared with only 10%-20% in other organs, so the endothelial damage results in ACE shedding. In addition, the shear stress also suppresses ACE gene expression which results in down regulation of ACE receptors[11].

METHODOLOGY

It was a correlation study, conducted in a textile mill of Faisalabad. Ninety five mill workers were approached through convenient sampling, to participate in this research study from which 84 were selected based on inclusion and exclusion criteria aged 18-40 years. They were divided into three groups (n=26 for control group, n=29 for two study groups) depending on their exposure to cotton dust.

In control group the subjects having less than one month exposure to cotton dust were included. In group II, subjects having 3-5 years exposure to cotton dust and in group III subjects having exposure to cotton dust for 5-10 years were included. Consent was taken from all the subjects included in the study. History and examination of the subjects were recorded on questionnaire proforma. Blood pressure of the subjects was recorded in sitting position by using mercury sphygmomanometer, three readings were taken and mean was calculated. Blood samples were taken under aseptic measures and serum angiotensin II levels were estimated by using ELISA technique. All ethical consideration and confidential protection of the individuals was specially observed. The collected data was analyzed by using Statistical Package for Social Sciences (SPSS) version 17.0. All the quantitative variables were presented as ± SEM (Standard error mean). Pearson correlation was applied to observe correlations between blood pressure, serum angiotensin II and duration of cotton dust exposure. One way ANOVA was applied to observe the group mean differences. Student t-test was applied to find out the differences between two groups. A *p*-value of <0.05 was considered as statistically significant.

RESULTS

In Table 1: Comparison of blood pressure (systolic and diastolic) and serum angiotensin II in control group and group II subjects is given. The difference between the control group and group II subjects for both these variables is non-significant (p >0.05).The difference between the control group and group II for serum angiotensin II (IU) is found highly significant (p- value < 0.001).

Table 2: Comparison of blood pressure (systolic and diastolic) and angiotensin II in control group and group III subjects is shown. For this variable the difference between the two groups is not significant (p-value 0.171). The difference between the two groups for diastolic blood pressure is statistically significant (p-value < 0.05). The difference between the two groups for angiotensin II (IU) is statistically significant (p-value < 0.001).

Table 3: In case of systolic blood pressure (mmHg) there is no significant difference seen between the subjects of group II and III (p >0.05), but for diastolic blood pressure (mmHg) a significant difference is present (p <0.05). The difference between the two groups for angiotensin II (IU) is statistically significant (p-value < 0.001). Fig 1 shows the correlation between diastolic blood pressure(mmHg) and serum angiotensin II (IU) for group III subjects, the r value is -0.476 showing a negative correlation but statistically significant as p <0.05

Table 1: Comparison of blood pressure and serum angiotensin II in control group and group II subjects

Parameter	Control group	Group II	t-value	*p*-value
Systolic blood pressure (mmHg)	123.46 ±1.13	123.62 ± 0.89	-0.11	0.912†
Diastolic blood pressure (mmHg)	76.53 ± 1.14	79.14± 1.03	-1.70	0.094†
Serum Angiotensin II (IU)	39.12 ± 0.87	32.69 ± 1.62	3.38	<0.001**

Table 2: Comparison of blood pressure and serum angiotensin II in control group and group III subjects.

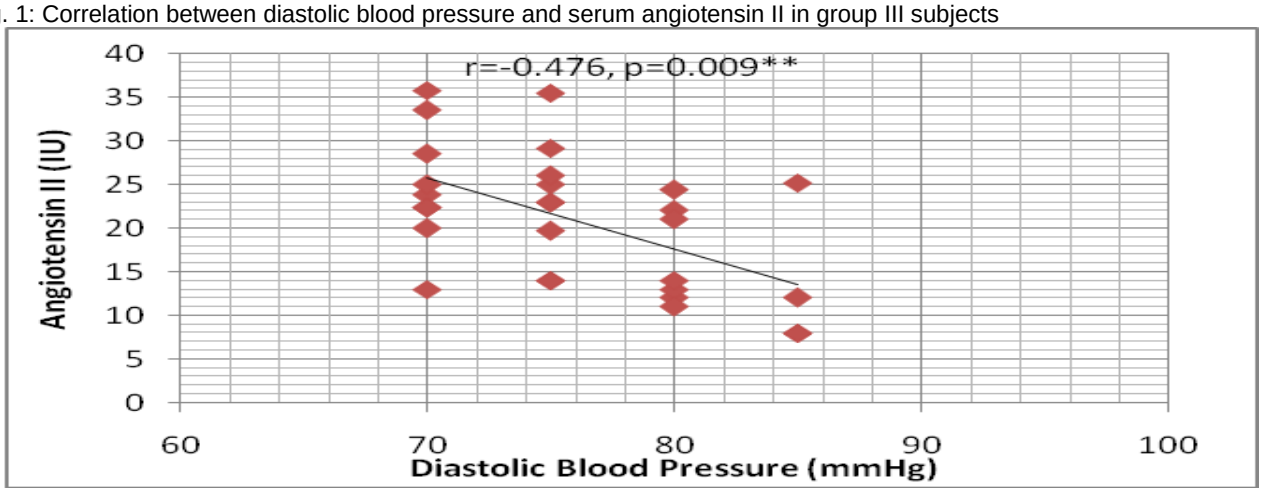
Parameter	Control group	Group III	t-value	*p*-value
Systolic blood pressure (mmHg)	123.46 ± 1.13	121.21 ± 1.15	1.40	0.171†
Diastolic blood pressure (mmHg)	76.53 ±1.14	76.55± 0.93	1.40	0.040*
Serum Angiotensin II (IU)	39.12 ± 0.87	20.83 ± 1.44	10.54	<0.001**

Table 3: Comparison of blood pressure (systolic and diastolic) and serum angiotensin II in group II and group III subjects ..

Parameter	Group II	Group III	t-value	*p*-value
Systolic blood pressure (mmHg)	123.62 ± 0.89	121.21 ± 1.15	1.65	0.104†
Diastolic blood pressure (mmHg)	79.14 ± 1.03	76.55 ± 0.93	3.01	0.004*
Serum Angiotensin II (IU)	32.69 ±1.62	20.83 ±1.44	5.46	<0.001**

*p < 0.05 significant **p < 0.001 highly significant †p > 0.05 non significant

Fig. 1: Correlation between diastolic blood pressure and serum angiotensin II in group III subjects



DISCUSSION

The cotton dust induced environmental agents, play a central role in the pathogenesis of chronic airway disease in textile workers[12]. Up till now many research studies have been done in different countries to find out the relationship between cotton dust pollution and its acute and chronic effects on human health[13].

As we know that endotoxins and particulate matter found in cotton dust initiate the inflammatory response in airways and releases different mediators which damage the lung parenchyma and pulmonary endothelium[14,4]. The release of vasoactive agent i.e., nitrous oxide enhances the inflammatory process which further damages the endothelial cells and leads to endothelial dysfunction[15]**.** As pulmonary endothelium is a rich source of membrane bound angiotensin converting enzyme, so any damage to endothelium results in reduction of angiotensin converting enzyme[11,16].

However the available data on the effects of cotton dust exposure on serum angiotensin converting enzyme and angiotensin II and its subsequent changes in arterial pressure is at scanty. Moreover, no study was found which studied all these parameters in the same subject and comparing the findings in subjects exposed to cotton dust for varying durations.

The present study was conducted to find out the effects of cotton dust exposure and its relationship with blood pressure in three different groups of subjects working in polluted environment of textile industry.

We found that the systolic and diastolic blood pressure in the control group and group II subjects were almost the same with a very little variation, standard errors were also similar in these groups which indicates similar variability in responses for these two variables.This observation shows that exposure to cotton dust for less than five years reflects no effect on blood pressure. While in group III the blood pressure was relatively on the lower side as compared to other two groups, a statistically significant difference in systolic and diastolic blood pressure is seen between control group and group III subjects, which was found relatively on lower side in group III subjects as compared to control group.Which shows that exposure of cotton dust pollution for five to ten years may cause endothelial damage and dysfunction which may lower the blood pressure in exposed persons.

We also studied serum angiotensin II levels in all the three groups, higher values of serum angiotensin II were found in control group, while its value decreased progressively in the subjects of group II as well as in group III. This shows that with cotton dust exposure the levels of serum angiotensin II were decreased which were lowest in those subjects who had exposure to pollution for more than five years. In all three groups a statistically significant difference is seen with respect to this variable.

No significant correlation between the blood pressure (systolic and diastolic) and serum angiotensin II is seen between control group and group II subjects. while a statistically significant correlation is found between control group and group III subjects, whichs shows that with exposure to cotton dust the pulmonary endothelium may get damage which may result in decrease levels of angiotensin converting enzymes, with the lower ACE levels the angiotensin II levels also decrease , with these physiological changes the blood pressure ultimately decreased in these subjects.

Limitations: Apart from cotton dust, many other factors such as noise pollution, smoking and obesity can also affect the blood pressure but because of our

limited resources these parameters were not included in the study.

CONCLUSION

Exposure to cotton dust pollution for a prolonged period (five to ten years) may damage the pulmonary endothelium which may reduce the activity of angiotensin converting enzyme, as a result the serum angiotensin II levels may also decrease which tends to lower the blood pressure in exposed persons.

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