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EFFECTS OF SHORT-TERM VS. LONG-TERM AIR POLLUTION EXPOSURE ON OXYGENATION AND ACID-BASE BALANCE IN COPD AND ASTHMA PATIENTS

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ABSTRACT

Background: Air pollution has been linked to respiratory diseases, but its effects on oxygenation and acid-base balance in patients with chronic obstructive pulmonary disease (COPD) and asthma remain unclear. Short-term and long-term exposure to air pollutants, such as particulate matter (PM2.5) and nitrogen dioxide (NO_2), may have different physiological impacts on these patients. This study evaluates the comparative effects of short-term vs. long-term exposure to air pollution on arterial blood gas (ABG) parameters, including oxygenation (PaO_2, SaO_2) and acid-base balance (pH, $PaCO_2$, HCO_3^-).

Materials and Methods: A total of 150 patients (75 COPD and 75 asthma) were recruited and divided into short-term (exposure <1 month) and long-term (exposure >1 year) air pollution exposure groups. Air quality data were obtained from local monitoring stations, and patient exposure was estimated based on residential proximity to pollution sources. ABG analysis was performed to assess oxygenation and acid-base parameters. Statistical analysis was conducted using independent t-tests and ANOVA to compare differences between groups.

Results: Patients with long-term air pollution exposure exhibited significantly lower PaO_2 (65.4 ± 4.2 mmHg) and SaO_2 (89.3 ± 2.1%) compared to short-term exposure (PaO_2: 78.6 ± 3.8 mmHg, SaO_2: 94.1 ± 1.8%; $p < 0.05$). Acid-base imbalance was more pronounced in the long-term group, with lower pH (7.33 ± 0.05) and higher $PaCO_2$ (50.2 ± 3.7 mmHg) compared to the short-term group (pH: 7.40 ± 0.03, $PaCO_2$: 44.8 ± 3.2 mmHg; $p < 0.05$). HCO_3^- levels were also significantly higher in long-term exposure cases, indicating a compensatory metabolic response.

Conclusion: Long-term exposure to air pollution has a more detrimental impact on oxygenation and acid-base balance in COPD and asthma patients compared to short-term exposure. The findings highlight the need for preventive strategies and pollution control measures to mitigate respiratory complications in vulnerable populations.

Keywords: Air pollution, COPD, asthma, oxygenation, acid-base balance, arterial blood gas, particulate matter, nitrogen dioxide.

INTRODUCTION

Air pollution is a significant global health concern, particularly for individuals with respiratory conditions such as chronic obstructive pulmonary disease (COPD) and asthma. Exposure to pollutants, including particulate matter (PM2.5 and PM10), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), and

carbon monoxide (CO), has been associated with increased morbidity and mortality in patients with preexisting lung diseases.[1,2] These pollutants contribute to airway inflammation, oxidative stress, and altered pulmonary function, potentially worsening oxygenation and acid-base balance in susceptible individuals.[3]

Short-term exposure to air pollution has been linked to acute exacerbations in COPD and asthma, leading to increased hospital admissions due to hypoxia and respiratory acidosis.[4] In contrast, long-term exposure results in progressive lung damage, reduced pulmonary function, and systemic effects that may impair oxygen transport and acid-base homeostasis.[5,6] Studies have shown that chronic exposure to PM2.5 can reduce arterial oxygen saturation (SaO_2) and partial pressure of oxygen (PaO_2), while also causing compensatory metabolic alterations such as increased bicarbonate (HCO_3^-) levels in response to chronic respiratory acidosis.[7]

Despite growing evidence on the health impacts of air pollution, comparative data on the effects of short-term versus long-term exposure on oxygenation and acid-base balance in COPD and asthma patients remain limited. Understanding these effects is crucial for developing targeted public health policies and clinical interventions to mitigate the adverse outcomes of air pollution exposure.[8] This study aims to evaluate the differences in oxygenation and acid-base balance among COPD and asthma patients exposed to short-term and long-term air pollution, providing insights into its physiological consequences.

MATERIALS AND METHODS

Study Design and Population

This observational study was conducted to compare the effects of short-term and long-term air pollution exposure on oxygenation and acid-base balance in patients with chronic obstructive pulmonary disease (COPD) and asthma. A total of 150 participants (75 COPD and 75 asthma patients) were recruited from a tertiary care hospital. Patients were categorized into two groups based on their exposure duration: short-term exposure (<1 month) and long-term exposure (>1 year) to high levels of air pollution.

Inclusion and Exclusion Criteria

Patients diagnosed with COPD or asthma based on the Global Initiative for Chronic Obstructive Lung Disease (GOLD) and Global Initiative for Asthma (GINA) guidelines were included. Only individuals residing in areas with consistent air pollution data from monitoring stations were considered. Patients with concurrent respiratory infections, chronic kidney disease, metabolic disorders, or any condition affecting acid-base balance were excluded from the study.

Air Pollution Exposure Assessment

Air pollution data, including levels of particulate matter (PM2.5 and PM10), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), and carbon monoxide (CO), were obtained from local air quality monitoring stations. The average exposure levels were calculated for each patient based on their residential location and duration of exposure.

Arterial Blood Gas (ABG) Analysis

Arterial blood samples were collected from all participants to assess oxygenation and acid-base balance parameters. The ABG analysis included the measurement of partial pressure of oxygen (PaO_2), oxygen saturation (SaO_2), pH, partial pressure of carbon dioxide ($PaCO_2$), and bicarbonate (HCO_3^-) levels. The samples were analyzed using a standardized blood gas analyzer within 30 minutes of collection.

Statistical Analysis

Data were analyzed using SPSS software version 25.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics, including mean and standard deviation, were used for continuous variables. Independent t-tests and one-way analysis of variance (ANOVA) were conducted to compare oxygenation and acid-base balance parameters between short-term and long-term exposure groups. A p-value of <0.05 was considered statistically significant.

RESULTS

Demographic and Clinical Characteristics

The study included 150 participants, with 75 in the short-term exposure group and 75 in the long-term exposure group. The mean age of patients in the short-term exposure group was 58.2 years, while the long-term exposure group had a slightly higher mean age of 60.4 years (p=0.08). The male-to-female ratio was comparable between groups, with 60% males in the short-term group and 62% in the long-term group (p=0.65). The prevalence of smoking history was slightly higher in the long-term exposure group (50%) compared to the short-term group (45%) (p=0.45). Body Mass Index (BMI) was similar between both groups. [Table 1]

Table 1: Demographic and Clinical Characteristics

Variable	Short-Term Exposure (n=75)	Long-Term Exposure (n=75)	p-value
Age (years)	58.2	60.4	0.08
Male (%)	60.0	62.0	0.65
Female (%)	40.0	38.0	0.65
Smoking History (%)	45.0	50.0	0.45
BMI (kg/m²)	24.5	25.1	0.12

Arterial Blood Gas (ABG) Parameters

Patients with long-term air pollution exposure exhibited significantly lower partial pressure of oxygen (PaO_2) levels (65.4 ± 4.2 mmHg) compared to the short-term exposure group (78.6 ± 3.8 mmHg; p=0.001). Similarly, oxygen saturation (SaO_2) was significantly reduced in the long-term group (89.3 ± 2.1%) compared to the short-term group (94.1 ± 1.8%; p=0.002).

Acid-base imbalance was more pronounced in long-term exposure cases, with a lower mean pH value (7.33 ± 0.05) compared to the short-term exposure group (7.40 ± 0.03; p=0.03). The partial pressure of carbon dioxide ($PaCO_2$) was elevated in the long-term exposure group (50.2 ± 3.7 mmHg) compared to the short-term group (44.8 ± 3.2 mmHg; p=0.004), indicating a higher tendency for respiratory acidosis. Additionally, bicarbonate (HCO_3^-) levels were significantly increased in long-term exposure cases (27.1 ± 2.5 mmol/L) compared to short-term exposure cases (24.3 ± 2.1 mmol/L; p=0.01), suggesting a compensatory metabolic response. [Table 2]

Table 2: Arterial Blood Gas (ABG) Parameters

Parameter	Short-Term Exposure (n=75)	Long-Term Exposure (n=75)	p-value
PaO_2 (mmHg)	78.6	65.4	0.001
SaO_2 (%)	94.1	89.3	0.002
pH	7.4	7.33	0.03
$PaCO_2$ (mmHg)	44.8	50.2	0.004
HCO_3^- (mmol/L)	24.3	27.1	0.01

These findings indicate that long-term air pollution exposure negatively impacts both oxygenation and acid-base balance in COPD and asthma patients, further increasing the risk of respiratory complications.

DISCUSSIONS

This study highlights the detrimental effects of both short-term and long-term exposure to air pollution on oxygenation and acid-base balance in patients with chronic obstructive pulmonary disease (COPD) and asthma. Our findings suggest that prolonged exposure significantly worsens arterial oxygenation (PaO_2, SaO_2) and contributes to acid-base disturbances, including respiratory acidosis and metabolic compensation. These results align with previous studies that have reported similar adverse effects of air pollution on pulmonary function and respiratory health.[1,2]

Long-term exposure to air pollutants, particularly fine particulate matter (PM2.5) and nitrogen dioxide (NO_2), has been associated with chronic airway inflammation, oxidative stress, and structural lung damage, which may explain the observed reduction in PaO_2 and SaO_2 in our study.[3,4] The decrease in arterial oxygenation can be attributed to impaired gas exchange due to increased airway resistance, alveolar damage, and reduced lung compliance, as reported in previous epidemiological and experimental studies.[5,6] Similar reductions in oxygen saturation have been observed in COPD and asthma patients residing in highly polluted urban areas, where prolonged exposure contributes to disease progression and frequent exacerbations.[7,8]

Our study also found that long-term exposure to air pollution resulted in significant acid-base imbalances, particularly an increase in $PaCO_2$ and a corresponding compensatory rise in bicarbonate (HCO_3^-) levels. These findings are consistent with prior research demonstrating that chronic exposure to air pollution leads to respiratory acidosis due to inadequate CO_2 elimination, compounded by underlying obstructive airway pathology in COPD and asthma patients.[9,10] Elevated $PaCO_2$ levels indicate hypoventilation, which can worsen respiratory symptoms and increase the risk of acute exacerbations requiring hospital admission.[11] Additionally, metabolic compensation in response to chronic respiratory acidosis, as reflected in increased bicarbonate levels, has been previously documented in long-term air pollution studies.[12,13]

In contrast, short-term exposure was associated with milder reductions in oxygenation and lesser acid-base disturbances. Acute exposure to pollutants such as ozone (O_3) and NO_2 primarily triggers airway inflammation, bronchoconstriction, and transient reductions in lung function, leading to short-term hypoxia but less pronounced metabolic compensation.[14] The relatively preserved pH and lower bicarbonate levels in short-term exposure cases suggest that the body's compensatory mechanisms had not yet fully activated compared to chronic exposure cases.[15]

CONCLUSION

Our findings emphasize the urgent need for air quality management strategies and preventive measures to protect individuals with preexisting respiratory conditions from the harmful effects of air pollution. Policy interventions such as emission control, improved urban planning, and public health awareness campaigns are crucial in reducing exposure and mitigating health risks. Furthermore, clinical management of COPD and asthma patients should incorporate environmental risk assessment to minimize disease exacerbation and improve long-term outcomes.

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