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A STUDY OF ARTERIAL BLOOD GASES WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE IN A TERTIARY CARE HOSPITAL

Pulmonary Medicine

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ABSTRACT

Chronic obstructive pulmonary disease (COPD) is a major public health problem. Diseases of the respiratory system affect main lung function i. e. homeostasis of gases. Pathology in any organ of body is associated with its functional compromise. This functional compromise measurement reflects type and severity of that pathology, and its outcome. Similarly measurement of blood oxygen, carbon dioxide and pH may help in assessing type and severity of respiratory pathologies. So this prospective study was aimed to correlate arterial blood gas analysis with severity and outcome of COPD. 47 patients of COPD were selected randomly from OPD. During the treatment of patient periodically ABG was tested to assess the management. Pearson correlation test was done to correlate PaO2, Pa CO2 with spirometry parameters. We found that in COPD patients chronic respiratory acidosis i.e. persistent hypercapnia was common finding. In COPD patients with hypercapnia, alveolar-arterial gradient was not reliable indicator of hypoxaemia. Patients of COPD with cor-pulmonalae are more hypoxaemic and hypercapnic than patients without cor-pulmonalae. In COPD patients there is significant positive correlation of PaO2 with % FEV1 of predicted, and significant negative correlation of PaCO2 with %FEV1 of predicted.

KEYWORDS

COPD, PaO2, PaCO2, pH value, arterial blood gases

INTRODUCTION:

Chronic obstructive pulmonary disease (COPD) is a major public health problem. Epidemiologic data projection for 2020 indicates that COPD will be the third leading cause of death worldwide and the fifth leading cause of disability.[1] Even COPD is top most cause of death in non-communicable diseases in Maharashtra state in year 2008.[2] Even though the overall prognosis of COPD patients is improved, the mortality rate remains high and mostly acid-base disorders occurring in these subjects can affect the outcome.

It is clear that lung diseases affect gas exchange and body's acid-base status. The respiratory system can act quickly to compensate for metabolic disturbance. The renal system cannot act as quickly for compensation to take place in respiratory disease. Change in bicarbonates is characteristic of chronic lung disease rather than in acute.

The fundamental function of the respiratory system is to provide the correct homeostasis of arterial blood gases like O_2, CO_2, and arterial pH.[3] Main function homeostasis of lung gases is affected by diseases of the respiratory system. Whenever any organ in body is having pathology, it is associated with functional compromise. This functional compromise measurement reflects type and severity of pathology, and its outcome. Similarly measurement of blood oxygen, carbon dioxide and pH may help in assessing type and severity of respiratory pathologies. So this study was aimed to correlate arterial blood gas analysis with severity and outcome of COPD.

MATERIAL AND METHODS:

This prospective study was conducted in the department of pulmonary medicine and Intensive respiratory care unit (IRCU) of a tertiary referral and teaching hospital after approval by the institutional ethics committee. All adult patients presented with COPD were screened after clinical examination and accordingly shifted to either pulmonary medicine ward or IRCU. Patients with bleeding or clotting disorder and those having age < 13years were excluded from study. Total 47 patients of COPD were selected randomly. Then after written informed consent of patient first ABG sample was collected. Results were considered as ABG at time of admission. After this with initial necessary management of patient, detail clinical history and clinical examination was done. All necessary investigations were done, in order to confirm the diagnosis of patient. During the treatment of patient periodically ABG was tested to assess the management as per need. At time of discharge last ABG was tested. In patients who died during treatment, last ABG test done during management of patient considered as last ABG. All the data collected in Microsoft office excel sheet.

Procedure of Collection of Arterial Blood Sample:[4,5]

For collection of arterial blood sample radial artery at wrist was selected as it is near to surface, relatively easy to palpate and stabilize, and usually has good collateral supply from the ulnar arteries. This was confirmed by a modified Allen's test. Local anesthesia was given over area of collection. Then under aseptic precaution arterial blood was collected in heparin flushed syringe with use of 22 – 20 gauze needle. At least 3ml of blood was collected to avoid dilution effect from heparin, as too much heparin is possible source of error in ABG analysis. Any sample with more than fine air bubble was discarded, as it lowers arterial carbon dioxide pressure and increases arterial oxygen pressure level. Samples were analyzed quickly, as delay may lower the pH. Forced expiratory volume in one second (FEV1) and forced vital capacity (FVC) were measured with a portable spirometer. The patients were first shown how to make a forced expiration. After one or two training manoeuvres without the apparatus, they made a series of 2–4 forced expirations. The best values were kept for analysis.

COPD Diagnosis, severity of disease, and management is done as per GOLD guideline for COPD.[6] Management COPD was done as per standard protocol with bronchodilators, antibiotics, inhalational or systemic steroids as per severity and standard guidelines, mucolytic, low flow oxygen supplementation as per need, mechanical ventilation for patients of respiratory failure.

STATISTICAL METHODS:

Values are expressed as mean ± standard deviation. Correlations were done between PaO_2, $PaCO_2$ and spirometry parameters by Pearson correlation test. Statistical significance was accepted at the 95% confidence level ($p<0.05$).

RESULT:

Table no 1: ABG Interpretation in patients of COPD at time of admission

Acid-Base Interpretation	No of Patients (n)	Percentage (%)
Normal	16	34%
Respiratory acidosis	29	61.7%
Mixed acidosis	02	4.3%
TOTAL	47	100%

Table no 2: ABG interpretation of patients of COPD at the time of discharge

Acid-Base Interpretation	No of Patients (n)	Percentage (%)
Normal	32	68.1%
Respiratory acidosis	07	14.9%
Mixed acidosis	02	4.3%
Metabolic alkalosis with respiratory acidosis	06	12.8%
TOTAL	47	100%

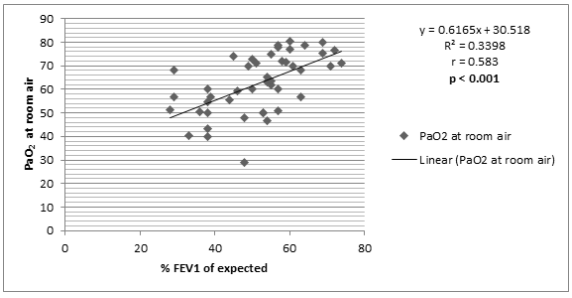
Table no 3: ABG parameter in patients of COPD (ABG sample collected when patient was breathing at room air:

ABG parameter	pH Mean (SD)	$PaCO_2$ Mean (SD)	PaO_2 Mean (SD)	HCO_3^- Mean (SD)	$PAaO_2$ Mean (SD)
On Admission	7.35 ± 0.04	50.73 ±11.00	62.29±12.59	27.17±3.96	26.57±8.52
While Discharge	7.40 ± 0.02	43.86 ± 3.68	71.76±7.96	26.38±3.34	25.34±5.96

Table no 4: comparison of COPD with cor-pulmonalae patients with COPD without cor-pulmonalae patients.

	Mean pH	Mean PaO2	Mean PaCO2
Without cor-pulmonalae (38)	7.36 ±(0.04)	65.2 ±(10.96)	47.35 ±(7.42)
With Cor-pulmonalae (9)	7.32 ±(0.04)	45.24 ±(7.94)	67.02 ±(8.95)

Scatter diagram 1: Correlation of PaO2 value with %FEV1 of expected in patients of COPD {n=45}.



Scatter diagram 2: Correlation of PaCO2 value with %FEV1 of expected in patients of COPD {n=45}.

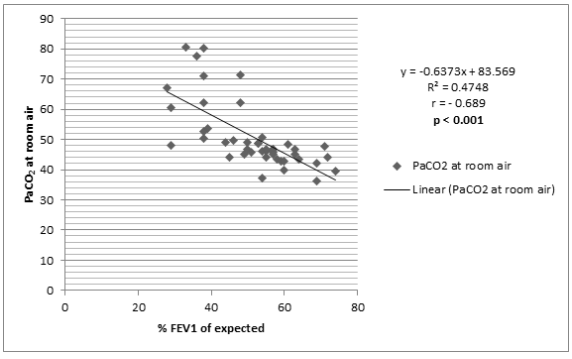


Table no 5 : Effect of NPPV on ABG parameter of 11 patients of COPD, who required and improved with NPPV.

ABG parameter	pH Mean (SD)	$PaCO_2$ Mean (SD)	PaO_2 Mean (SD)	HCO_3^- Mean (SD)	$PAaO_2$ Mean (SD)
Before NPPV	7.32±(0.02)	63.05±(11.44)	46.91±(7.38)	31.18±(4.93)	27.15±(11.98)
After NPPV	7.42±(0.02)	47.45±(3.14)	62.58±(6.79)	30.11±(3.7)	30.22±(5.39)

DISCUSSION:

In present study, out of 47 patients of COPD, 31 66%) patients had respiratory acidosis. Only two patients had additional component of metabolic acidosis. At the time of admission, COPD patients had mean $PaCO_2$ value was 50.73 mmHg and mean PaO_2 value was 62.29 mmHg. This indicates that these patients had considerable hypoxaemia with hypercapnia. Also on discharge noticeable improvement was seen in PaO_2 value but associated changes were not observed in gradient. In COPD patient with hypercapnia level of hypoxemia does not correlate with gradient. These findings are similar with study of Gray BA and Blalock JM who calculated $PAaO_2$ from arterial blood gas measurement in 23 patients of COPD with hypercapnia and hypoxemia. They found gradient varied inversely with $PaCO_2$. So the gradient may be unreliable indicator of abnormal gas exchange in presence of hypercapnia in COPD patients.[7]

In present study, 9 patients had associated cor-pulmonalae in COPD. Their ABG parameter indicate they were more hypercapnic and hypoxaemic than patients without cor-pulmonalae. Their mean PaO_2 and mean $PaCO_2$ were 45.24 mmHg and 65.2mmHg respectively, while in patients without cor-pulmonalae mean PaO_2 and mean $PaCO_2$ were 65.2 mmHg and 47.35 mmHg respectively. These finding correlates with studies of Karadag et al [8] and Koh YI et al. [9] They concluded that alterations in pH and $PaCO_2$ (respiratory acidosis and hypercapnia) appear to have more prominent role in oedema formation in COPD patients.

In patients of COPD when PaO_2 was plotted against % FEV1, we got significant positive linear correlation (r=0.583) and if $PaCO_2$ was plotted against % FEV1, we got significant negative linear correlation (r= - 0.689). Delclaux B, et al [10] and Roberto R, et al [11] also got the similar result.

In the present study total 11 patients of COPD with respiratory failure improved with NPPV. With use of NPPV their mean pH improved from 7.32 to 7.42 with corresponding improvement in mean $PaCO_2$ from 63.05mmHg to 47.45mmHg. Hypoxemia also improved in the form of mean PaO_2 from 46.91 mmHg to 62.58mmHg, but mean gradient deteriorated from 27.15 mm Hg to 30.22 mm Hg. These findings correlate with study of Orlando Diaz et al [12], and George IA et al [13].

In patients of respiratory failure due to COPD, with use of NPPV their mean pH normalizes from 7.32 to 7.42 with corresponding correction of both hypercapnia and metabolic acidosis. Their mean PaO_2 value also improved from 46.91 mmHg to 62.58 mmHg but mean gradient deteriorated from 27.15 to 30.22.

NPPV was used by Orlando Diaz in 10 patients of respiratory failure in COPD. He observed moderate increase in PaO_2 (from 50± 6 to 57± 9 mm Hg; p< 0.05), and a fall in PaCO2 (from 66 ±10 to 59 ±10 mm Hg; p< 0.0001). When George A et al [87] used NPPV in patients of hypercapnic respiratory failure, mean pH improved from 7.25 to 7.39 and mean PaCO2 from 76.6 mm Hg to 51.9 mm Hg.[13]

CONCLUSION:

From our study results we conclude that in COPD patients chronic respiratory acidosis i.e.- persistent hypercapnia was common finding. In COPD patients with hypercapnia, alveolar-arterial gradient was not reliable indicator of hypoxaemia. Patients of COPD with cor-pulmonalae were more hypoxaemic and hypercapnic than patients without cor-pulmonalae. In COPD patients, there was significant positive correlation of PaO_2 with % FEV1 of predicted, and significant negative correlation of $PaCO_2$ with %FEV1 of predicted.

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