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A comparative study on arterial blood gas analysis among COPD patients and non-COPD patients

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***Abstract*---**Background: Chronic obstructive pulmonary disease (COPD) was confused with so many diseases but as the knowledge and medical skill advance gradually a dramatic change could be observed about all diseases. Cigarette smoking is a leading cause of COPD. Objective: The study principally aims to compare arterial blood gas analysis among patients having chronic obstructive pulmonary disease and healthy control cases. Methodology: A cross sectional study was conducted in 200 patients attended a department with 100 COPD patients and 100 healthy patients. Based on inclusion and exclusion criteria patients were recruited. All patients were under go through ABG analysis. Informed consent was obtained from each patient. P value less than 0.05 considered statistically significant. Results: The results of present study indicated that there were significantly greater alteration in the blood gases and acid base chemistry, particularly PaO_2, $PaCO_2$ in patients who show evidence of various complications mentioned earlier. Conclusion: It has thus been inferred that although COPD patients had pH within the normal range the majority of them had low normal values. This study concluded that all parameters of ABG were statistically significantly deranged in chronic bronchitis, emphysema and chronic bronchitis with emphysema patients.

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Keywords---chronic obstructive pulmonary disease, Smoking, Arterial blood gas analysis, haematology, Blood chemistry.

Introduction

Chronic obstructive pulmonary disease (COPD) might have been a disease since the origin of human being.[1] COPD is one of the most important and leading causes of increased morbidity and mortality in world including India. It includes chronic bronchitis and emphysema.[2-3] Cigarette smoking is the most commonly involved. Prolonged cigarette smoking impairs ciliary movement, inhibits function of alveolar macrophages, and leads to hypertrophy and hyperplasia of mucus-secreting glands. In addition to these chronic effects it is probable that smoke causes polymorphonuclear leukocytes to release proteolytic enzymes acutely. Inhaled cigarette smoke can produce an acute increase in airways resistance due to vagally mediated smooth-muscle constriction.[4] The increasing urbanization, industrialization and adoption of smoking by third world like our country apart from atmospheric pollution due to Indian way of cooking are the various causes of increasing incidence of COPD in our country.[5,6]

COPD patients are a heterogeneous group of patients who present with cough, (with or without sputum) and dyspnea (characteristically persistent) and generally follow a progressively downhill course punctuated by acute infection, bronchoconstriction or respiratory failure.[7] The clinical signs may not be diagnostic but may include features of over inflation, quiet breath sounds and associated reduction of force expiratory volume in first second (FEV_1) with obstructive pattern.[7,8]

The Arterial blood gas analysis (ABG) test is used to evaluate respiratory disease. It is used to determine the effectiveness of oxygen therapy.[9-11] The acid-base component of the test also gives information on how well the kidneys are functioning. So, we can predict early changes of respiratory failure with the help of ABG analysis. So that we can effectively use most appropriate therapy for the management of COPD. [10-11]

Arterial blood gas (ABG) analysis is useful in evaluation of clinical condition of critically ill patients however arterial puncture may cause many complications in these patients.[12] Hypoxemia and hypercarbia being present more commonly in those with more severe grades of obstruction.[13] Carbon dioxide levels are in addition also depend upon the sensitivity to it in different individuals and that factors other than airways obstruction play a role in the production of hypercarbia.[14] It is a useful test for assessment of oxygenation and acid base status of COPD patients.[15]

Keeping these facts into consideration this study was carried out to see the derangement of ABG in COPD patients. The study principally aims to compare arterial blood gas analysis among patients having chronic obstructive pulmonary disease and healthy control cases.

Material and Method

This study was conducted in a tertiary hospital situated in central India. The sample size was 200 patients with 100 patients in case arm (patients already diagnosed for Chronic obstructive pulmonary disease (COPD)) and 100 patients in control arm (not having COPD). The cases and control were matched for age and sex variable. The diagnosis of COPD was made according to the Gold Criteria.[16] The inclusion criteria for cases were as follow: -

Clinical Criteria

a. History of productive cough for minimum of 3 months a year for at least 2 years.
b. Symptoms and signs pertaining to the diseases of airway and or lung parenchyma.
c. Various grades of dyspnoea.
   Dyspnea that is: Progressive (worsens over time)
   Persistent (present every day)
   Worse on exercise
   Worse during respiratory infections
d. History of exposure to risk factors: Tobacco smoke (including popular local preparations).

Occupational dusts and chemicals
Smoke from home cooking and heating fuel Spirometry Criteria for COPD detection was Spirometry evidence of airway obstruction and FEV_1 < 70% of predicted normal or FEV_1/ FVC < 0.6. The exclusion criteria for the cases were Patients with past or present diagnosis of asthma, Patients with history of seasonal or episodic dyspnea or wheezing and Patients with associated specific lung conditions such as pulmonary tuberculosis, bronchiectasis, lung abscess, bronchial carcinoma, tropical eosinophilia or pulmonary fibrosis. A detailed history including demographic data, detailed medical history, thorough general and physical examination and detailed hematological and radiological investigation was recorded. Electrocardiography, Pulmonary function test and Arterial Blood Gas Analysis was performed for all patients. The informed consent was obtained from all patients. Confidentiality of patients was maintained at each and every point. The data was entered in to Microsoft excel and analyzed using statistical software epi-info (freeware). The unpaired t test for parametric analysis and Mann-Whitney U test for non parametric analysis was applied for calculation of p value. P value less than 0.05 was considered as statistically significant.

Result & Observation

The incidence of COPD is higher in 50-59 years age group (n=37) followed by 60-69 age group (n=28) in both males and females. Male predominance was observed in COPD. (Table 1). It was observed that dyspnea on exertion was present in 100% of COPD patient followed by cough with sputum 65%. All patients of chronic bronchitis and chronic bronchitis with emphysema had cough with sputum while only 10% of emphysema patient had cough with scanty sputum. This table shows physical signs in 100 patients of COPD Crepitations in chest was most commonly observed physical sign found in 94% patients followed by

neck veins and Tachypnoea which were observed in 70% cases. Cyanosis was observed in 39% cases, signs of encephalopathy in 24% hepatomegaly in 20% cases.

It was observed that Hb% and TRBC was increased in 40% and 30% cases. While TLC, DLC were normal in all patients. It was observed that 50% of the patients were having x-ray findings suggestive of chronic bronchitis, 40% of emphysema and 10% were having mixed finding. X-ray finding were present in variable proportion in individual patient. It was observed that majority of the patients had poor progression of R wave and low voltage QRS complexes. P pulmonale was present in 59% and right ventricular hypertrophy was present in 36%. When pulmonary function test was done in all COPD patients, It was observed that FEV_1 and FEV_1/FVC ratio were on the lower side in chronic bronchitis and chronic bronchitis with emphysema patients as compared to emphysema patients, thus indicating more severe airway obstruction in these group. Type 1 – Respiratory Failure was present in 16%, 62.5%, 0% and type II Respiratory Failure was present in 76%, 7.5%, 100%, of chronic bronchitis, emphysema and chronic bronchitis with emphysema patients respectively. (Table 2-3)

When detailed analysis was done in COPD patients, It was observed that the values of PCO_2, PO_2 and SaO_2 were more effected in those patient in whom duration of illness was 5 year or more than 5 year. The difference was highly significant. ($p < 0.01$) The values of pH, PaO_2, BE, SBE, & SaO_2 were more deranged in those patients in whom duration of smoking was more than 20 years. (p value <0.01) It was observed that ABG values had direct correlation with smoking index all the ABG parameter except SBC were affected more with increasing smoking index the difference was highly significant ($p < 0.01$). The difference in pH and BE was significant with p value < 0.05. As regards other aspects of smoking habits it was observed that almost all the parameters were significantly altered in the sub-group of COPD patients who used to smoke more than 20 pieces per day for more than 20 years. It was observed that there were significantly higher alterations in PaO_2, $PaCO_2$ and SaO_2 values in patients with chronic cor pulmonale. (Table 4,5)

In present study, cyanosis was found in 39 cases it was observed that change in pH, PCO_2, PaO_2, BE, SBE and SaO_2 was highly significant in patient with central cyanosis. (p value < 0.01). It was observed that changes in PCO2, PaO2, SaO2 was statistically deranged in cor-pulmonale patient. The difference in other parameters of ABG was not statistically significant between 2 groups. (Table 6)
It was observed that PCO_2, PaO_2, TCO_2, BE SBE, SBC and SaO_2 were statistically changed in patient with respiratory failure $p < 0.01$. The difference of pH, HCO3 between two group was not statistically significant (p value > 0.05). Further it was seen that the patients who had cyanosis and/or respiratory failure showed significantly higher alterations in the pH, $PaCO_2$, PaO_2, SaO_2, BE and SBE values. Incidence of both types of respiratory failure was higher in > 5 years duration of illness. In the present study type I respiratory failure was present in 33% COPD patients (n=25 emphysema, n=8 chronic bronchitis) and type II failure was present in 51% COPD patients (n=38 chronic bronchitis, n=3 emphysema, n=10 chronic bronchitis with emphysema).

Discussion

The present study was conducted in the tertiary care center located in central India, to study blood gases and acid base chemistry of COPD patients (consisting of chronic bronchitis, bronchitis and emphysema) and compare their results with healthy controls. The mean age of cases was 56.37 ± 6.99 and mean age of control were 54.87 ± 7.14. Majority of participants in case and control arm were male. The similar representation was found in the study in various parts of world.[17-23]

Dyspnea on exertion was present in 100 % of COPD patients followed by cough with sputum in 65%. All patients of chronic bronchitis and chronic bronchitis with emphysema has cough with sputum while only 10% of emphysema patient had cough with scanty sputum. It was observed that 50% of the patients were having x-ray findings suggestive of chronic bronchitis, 40% of emphysema and 10% were having mixed finding.

It was observed that FEV_1 (mean) & FEV_1 / FVC (mean) were on lower side in all COPD patients. FEV_1 mean was 45.20%, 58.57% , 37.9% in chronic bronchitis, emphysema, chronic bronchitis with emphysema respectively and FEV_1/ FVC (mean) was 49.17% 62.28% 47.84% respectively in above mentioned group being more lower in chronic bronchitis and chronic bronchitis with emphysema as compare to emphysema alone thus indicating more severe air way obstruction in these group. The findings were fairly consistent with earlier reports on COPD.[24, 25]

It was observed in present study that 80% of control had pH between 7.34-7.45. 60% of emphysema had normal pH value while in 50% of chronic bronchitis and 60% chronic bronchitis with emphysema, it was below 7.34. All participants of control group had PaO_2 within the normal range (75-100 mmHg) while chronic bronchitis 80%, emphysema 50%, chronic bronchitis with emphysema 90% below normal range. In the previous studies it was found that significant hypoxia existed in these patients.[27, 28]

All controls had $PaCO_2$ within normal limits. 86% of emphysema had $PaCO_2$ within normal limit. Only 14% had $PaCO_2$ between 46-55 while in majority of chronic bronchitis (60%, n=30) and chronic bronchitis with emphysema (90%, n=9) had $PaCO_2$ above normal limit. Studies done by various authors described that a state of hypoxemia remains both in emphysema and chronic bronchitis but also mentioned that emphysema patients tend to have lower PaCO2 than bronchitis patient at similar values of FEV1.[20]

This study mentioned that majority of the control had HCO_3 within normal limit while it was below 20 mEq/Lt in 56% (n=28) of chronic bronchitis and 55% (n=22) of emphysema, 60%(n=6) of chronic bronchitis with emphysema patients. In their study, Gao L et al[29] observed severe grades of acidosis. Occasional patients showed gross acidosis as evident by low pH and HCO_3 values. These observations indicate that in patients with chronic airway obstruction the pH is being maintained within normal range although on a lower side. These patients are said to be in chronic respiratory failure in a compensated state but when acute respiratory failure is precipitated by an inter-current illness the decompensation

manifests in the form of fall in pH and inability of the Milieu Interior to bring pH to normal by conserving base. These patients show gross disturbances of acid base equilibrium in the form of low pH, low HCO_3, and high base deficit values. In COPD group, 90% cases had SaO_2 values below the normal range of 95-98% out of this 50% chronic bronchitis had SaO_2 within 75-95% range, 28% within 52-73 range and 12% cases within the lower range of 30-51%. The similar finding was observed in studies done by Trask CH et al.[27]

The results of present study indicated that there were significantly greater alteration in the blood gases and acid base chemistry (particularly PaO_2, $PaCO_2$ in patients who show evidence of various complications mentioned earlier). The study done by Leach RM et al[28] found that changes in PaCO2 as well as PaO2 influence neuroendocrine function. These two parameters of ABG (PaO_2 and $PaCO_2$) frequently deranged in patients with cor pulmonale and type II respiratory failure.

Viewing from another angle it can safely be stated that gross alterations in acid base chemistry herald the onset of various complication. Thus a continuous watch over these parameters can help in the better management of these patients and averting the various fatal complications which ultimately ensue. This being a tertiary hospital the patients of COPD admitted over here were very chronic and severe cases of COPD therefore it is obvious that most of them had various complications of COPD like cor-pulmonale, CCF and respiratory failure. As such the patients of COPD are so much used to the physical problem that until it is beyond their control to combat the problem which they face because of COPD which is usually seen in above mentioned conditions and as well as AECB.

Conclusion

In present study all the controls had $PaCO_2$ with in normal limits. Majority of emphysema patients had $PaCO_2$ with in normal limit while majority of chronic bronchitis had $PaCO_2$ above the normal limit. SaO_2 values were also found to be on a significantly lower side in COPD group. There was difference of the mean values for SaO_2 between controls and COPD group being lower in the later. There was significant difference of mean pH values between control and COPD groups. It is a well-known fact that chronic respiratory failure is associated with respiratory acidosis although in a compensated state. In COPD patients HCO_3 level were significantly low as compared to control group. Mean values for SBC were significantly low in the COPD group. It was observed that PaO_2, $PaCO_2$ and SaO_2 values were more altered with the increasing duration of illness.

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Table 1: Showing age and sex-wise distribution of cases and controls

		Number Cases (N=100)	**Number Control (N=100)**
Age groups (in years)	20-29	**1**	**2**
	30-39	**5**	**6**
	40-49	**22**	**23**
	50-59	**37**	**35**
	60-69	**28**	**25**
	70-79	**7**	**09**
Sex	Male	**92**	**90**
	Female	**08**	**10**

Table No. 2: showing finding of Arterial blood gas analysis among cases and controls

		Chronic Bronchitis	Emphysema	Chronic bronchitis with emphysema	Control
Ph	**below 7.34**	50%	30%	60%	16%
	7.34-7.45	46%	60%	40%	80%
	> 7.45	4%	10%	**0**	4%
PaO_2 (75-100 mmHg)	**below 65**	54%	**0**	**70%**	**0**
	65-74	26%	50%	**20%**	**0**
	75-84	8%	20%	**10%**	24%
	85-95	2%	**20%**	**0**	28%
	>95	10%	**10%**	**0**	48%
$PaCO_2$ (35-45 mmHg)	**35-45**	**40%**	86%	**10%**	100%
	46-55	**50%**	14%	**30%**	
	56-65	**10%**	0	**60%**	
	>65	**0**	0		
HCO3 (20-26 mEq/Lt)	**<20**	**56%**	55%	**60%**	12%
	20-26	**44%**	45%	**40%**	88%
	14-20	**60%**	55%	**70%**	4%
TCO2 (21-27 mmol/Lt.)	**21-27**	**40%**	45%	**30%**	92%
	> 27				4%
	+2.5 to -2.5	**32%**	40%	**30%**	100%
BE (-2.4 to +2.3 mmol/lit.)	**-2.6 to -7.5**	**44%**	45%	**50%**	
	-7.6 to -12.5	**22%**	15%	**20%**	
	-12.6 and above	**2%**			
SBC (22-26	**12-16**	**6%**		**20%**	8%

mEq/Lt.)	**17-21**	**82%**	80%	**80%**	92%
	22-26	**12%**	20%		
SaO2 (95-98%)	**30-51**	**12%**	10%	**30%**	8%
	52-73	**28%**	20%	**40%**	92%
	74-95	**50%**	60%	**30%**	
	>95	**10%**	10%		

Table No. 3: Showing comparison of ABG finding among cases and controls

Blood gases	COPD		Control		p
	Mean	SD	Mean	SD	
pH	7.34	2.86	7.39	2.35	<0.05
$PaCO_2$	49.87	11.91	37.96	3.23	<0.01
PaO_2	64.51	17.10	93.95	8.63	<0.01
HCO_3	19.75	1.85	21.63	2.57	<0.01
TCO_2	20.47	1.95	23.74	1.86	<0.01
BE	-5.20	3.51	-2.28	2.69	<0.01
SBE	-5.25	3.35	-2.31	2.06	<0.01
SBC	19.98	2.29	20.99	2.208	<0.05
SaO_2	76.67	17.75	93.07	4.39	<0.01

Table No. 4: Showing the correlation of duration of illness and smoking with ABG values among COPD Patients

Blood gases	Duration of Illness			Duration of smoking		
	> 5 years	2-5 years	P value	> 20 years	< 20 years	P value
pH	7.34 ±0.076	7.34 ±0.06	>0.05	7.33±0.0637	7.37±0.07	<0.01
$PaCO_2$	55.17 ±13.08	44.88 ±6.52	<0.01	54.31±12.74	43.62±6.12	<0.01
PaO_2	57.76 ±15.66	72.13 ±15.51	<0.01	59.50±15.50	72.15±14.58	<0.01
HCO_3	19.75 ±1.35	19.72 ±2.30	>0.05	19.60±1.32	20.55±2.51	>0.05
TCO_2	20.34 ±1.78	20.44 ±2.30	>0.05	20.35±1.41	21.52±2.50	<0.01
BE	-5.27 ±3.38	-5.20 ±3.60	>0.05	-5.68±3.18	-3.82±3.74	<0.01
SBE	-5.41 ±3.41	-5.06 ±3.44	>0.05	-5.79±3.31	-3.74±2.91	<0.01
SBC	20.17 ±1.73	19.75 ±2.79	>0.05	19.85±1.95	20.74±2.85	>0.05
SaO_2	69.00 ±19.67	85.47 ±10.19	<0.01	71.27±18.30	85.75±9.95	<0.01

Table No. 5: Showing the correlation of ABG values with smoking habits (in terms of or day, Smoking index) and central cyanosis

Blood gases	pieces of cigarettes/bidi smoked			Central Cyanosis		
	> 21 pieces	< 20 pieces	P value	With cyanosis	Without cyanosis	P value
pH	7.33±0.07	7.36±0.065	<0.05	7.31±0.065	7.36±0.10	<0.01
$PaCO_2$	57.42±11.65	43.00±5.51	<0.01	58.73±13.54	44.51±6.86	<0.01
PaO_2	57.56±13.73	72.33±15.72	<0.01	53.44±17.00	71.40±13.37	<0.01
HCO_3	19.39±1.26	20.51±2.23	<0.01	19.47±1.52	19.90±2.20	<0.05

TCO_2	20.09±1.26	21.36±2.48	<0.01	20.21±1.64	20.63±2.11	<0.05
BE	-5.91±3.33	-4.07±3.27	<0.05	-6.62±3.29	-4.56±3.42	<0.01
SBE	-6.05±3.25	-3.97±2.95	<0.01	-6.74±3.27	-4.40±3.08	<0.01
SBC	19.83±1.87	20.57±2.74	>0.05	19.63±1.99	20.05±2.46	<0.05
SaO_2	67.32±17.81	86.15±9.52	>0.05	64.61±19.64	84.14±11.47	<0.01

Table No. 6: Showing the correlation of ABG values in COPD patients Chronic Cor pulmonale an respiratory failure

Blood gases	**Cor pulmonale**			**Respiratory failure**		
	(+)	**(-)**	**P value**	**(+)**	**(-)**	**P value**
pH	7.34±0.06	7.34±0.07	>0.05	7.33±0.076	7.33±0.06	> 0.05
$PaCO_2$	53.85±16.00	45.95±8.22	<0.01	61.69±9.26	41.77±4.81	>0.01
PaO_2	60.34±16.99	67.66±16.65	<0.05	53.30±11.60	71.21±17.18	<0.01
HCO_3	19.77±1.39	19.71±2.14	>0.05	19.39±1.30	19.96±2.12	> 0.05
TCO_2	20.53±1.65	20.43±2.16	>0.05	19.97±1.35	20.89±2.11	<0.01
BE	-5.31±3.00	-5.13±3.90	>0.05	-6.06±3.81	-4.74±3.23	<0.01
SBE	-5.47±3.02	-5.08±3.68	>0.05	-6.07±3.82	-4.64±3.02	<0.01
SBC	20.29±1.80	19.70±2.54	>0.05	19.80±1.91	20.86±2.56	<0.01
SaO_2	71.55±20.11	80.62±14.17	<0.05	62.02±16.45	86.44±10.35	<0.01