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Pathology

A STUDY OF ARTERIAL BLOOD GAS ANALYSIS IN COPD PATIENTS AND IT'S IMPLICATION IN PREDICTING THE OUTCOME

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ABSTRACT The following prospective study was conducted in 70 known cases of COPD in the out patient and in patient department of pathology in association with department of medicine and LPS institute of cardiology Kanpur. Total 70 patients of COPD were picked up at random from urban as well as rural areas. 40 were of stable COPD and 30 were in exacerbation. Exacerbation of chronic obstructive pulmonary disease (COPD) is defined as an event in the natural course of the disease that is characterized by a change in the patient's baseline dyspnea, cough, or sputum beyond day-to-day variability and sufficient to warrant a change in management. In the present study arterial blood gas analysis was done in copd patients and acid base abnormality was found

. ABG sampling happens to be only reliable determination of ventilation success as reflected by CO_2 content . It constitutes a more precise measure of successful gas exchange and oxygenation. It also provides valuable information on the acid base balance at a specific point in the course of a patient's illness . It plays an important role in the management of Chronic Obstructive Pulmonary Disease (COPD). These analyses are relevant in the diagnosis of chronic respiratory failure and in long-term, continuous oxygen therapy monitoring.

KEYWORDS : COPD , Cigarette smoking, Respiratory acidosis

Introduction

COPD is one of the major causes of chronic morbidity and mortality worldwide. It is the fourth leading cause of death in the world.[1], and further increases in its prevalence and mortality can be predicted in the coming decades .[2,3] COPD is a pulmonary disease with significant extrapulmonary effects that may contribute to the severity of illness. Its pulmonary component is characterized by airflow limitation that is not fully reversible[4], usually is progressive and can lead to respiratory failure.[1]Systemic manifestations and co-morbidities in COPD are body weight loss, skeletal muscle wasting, cachexia, osteoporosis, right heart failure etc[5,6] Co-morbidities are common for people with COPD because organ systems work differently when they do not receive enough oxygen.Cigarette smoking is the most common risk factor for COPD, although in many countries, various kinds of air pollution have also been identified as COPD risk factors .[1,4,7]

Pathophysiology of copd

The chronic airflow limitation of COPD is caused by a mixture of small airway disease (obstructive bronchiolitis) and parenchymal destruction (emphysema), the relative contributions of which vary from person to person .[1] Chronic inflammation causes structural changes and narrowing of small airways. Destruction of the lung parenchyma leads to the loss of alveolar attachments to the small airways and decreases lung elastic recoil; these changes diminish the ability of the airways to remain open during expiration.[8,9,10,11] So in COPD inflammation causes small airway disease (airway inflammation, airway remodelling) and parenchymal destruction that all lead to airflow limitation.[9,10,11] Because of these anatomic changes in pulmonary tissue and consecutive airflow limitation respiratory failure can develop. For the body to function normally, ventilation must meet the metabolic demand of body tissue. Thus, metabolic tissue consumption of oxygen must be equal to the oxygen taken up in the blood from alveolar gas. Or, metabolic tissue production of carbon dioxide must be equal to the amount of carbon dioxide blown off at the alveoli. Respiratory failure is defined as a PaO2< 60 mmHg (8kPa) and is divided into type I and type II depending on PaCO2.

Type I of respiratory failure: PaCO2< 45 mmHg (6kPa)–normal or low; PaO2 is low (hypoxemia); this represents a ventilation/perfusion mismatch; we talk about partial respiratory insufficiency.

Type II of respiratory failure: PaCO2> 45 mmHg (6kPa), PaO2< 60 mmHg (8kPa). Both PaO2 and PaCO2 indicate that lungs are not well ventilated; we talk about a global or hypoventilation respiratory insufficiency.

The normal arterial pH lies in narrow range: 7.35-7.45 ([H^+] range 45-35 mmol/L). An acid -base disturbance arises when arterial pH lies outside that range. If pH is less than 7.35 an acidosis is present, if pH is greater than 7.45 the alkalosis is present. Alkalosis can be caused by either of: a fall in PaCO2 or rise in $HCO3^-$ When the primary change is in CO2 we name the disturbance respiratory, and when primary change is in bicarbonate, we name the disturbance metabolic.

Material and methods

The study was conducted in 70 known cases of COPD in the out patient and in patient department of pathology in association with department of medicine and LPS institute of cardiology Kanpur. Both rural and urban populations were included in the study out of which 40 were of stable COPD and 30 were in exacerbation. In each patient the duration of illness was recorded so is sex, age ,residential background and data(duration and number of pack years) of smoking habits.Patients were examined clinically and radiologically with a view to establish diagnosis and condition. They were subjected to spirometry, TLC,DLC, Fasting and Post prandial blood sugar, and serum creatinine, SGPT, Serum Bilirubin. Other investigations if required were also done. Condition of peripheral pulses and veins were then recorded.A resting pulse oximetry was performed immediately before taking samples for the study. Patients were explained the arterial blood gas (ABG) sampling procedure , with particular attention to the associated risks and benefits. Written consent was taken before the procedure The arterial punctures were done by a 24 gauze needle with a 2 ml syringe containing 0.1 ml sodium heparinate as an anticoagulant. All the samples were immediately analyzed in the blood gas analyzer(cobas b 121) and results were obtained subsequently.

Results

According to this study Male female ratio of Chronic Obstructive Pulmonary Disease was found to be 3:2. Maximum number of patients were in 46-55 years age group(42.86%) followed by 56-65 years of age group(34.29%). The mean age of study population of COPD was found to be 53.86+-7.80 years . Incidence of COPD is increasing among females as well. Diagnosed COPD appears to be more prevalent in men than women, whether expressed in terms of mortality or hospital separation data. This is usually attributed to the historically higher rates of cigarette smoking among men and their greater likelihood of exposure to significant occupational respiratory irritants. Our study has shown that 54.29% of COPD patients were residents of urban areas and 45.71% of patients came from rural areas.This can be attributed to increased air pollution in urban areas along with smoking habits. Incidence of COPD was higher in females of rural areas(57.15%).Whereas incidence of COPD amongst male was higher in urban areas. Most of the males(80.95%) were smokers and few(14.29%) were ex smokers.However, none of the female patient of

COPD was current smoker. Out of 42 COPD patients who were either current or ex-smokers, majority of the patient had 10-20 pack years(85.72%) followed by 20-30 pack years(9.52%) and 4.76% of patients smoked less than10 pack years. Smoking is by far the most important risk factor associated with functional decline in COPD .

Mean pH amongst stable COPD patients was 7.431 in the arterial blood samples with a range of 7.339-7.495 . These values lie within normal range of 7.350 to 7.450. Values less than 7.350 indicate acidosis whereas values higher than 7.450 indicate alkalosis. Mean ph amongst COPD exacerbation phase was found to be 7.248 within a range of 7.148-7.340.Mean PCO_2 in stable COPD patients was found to be 48.9mm Hg within a range of 34.5-58.2 mm Hg. This value is slightly higher for normal values of 35-45 mm Hg. Similarly mean PCO_2 in exacerbation was found to be 69.7mm Hg indicating hypercapnea. Respiratory acidosis due to hypercapnia is a common and severe complication observed in patients with chronic obstructive pulmonary disease in advanced phase. Development of acidosis worsens the prognosis and is associated with higher mortality rate.Mean PO_2 in stable COPD was found to be 71.96 indicating mild hypoxemia as normal values of PO_2 range from 80-100 mm Hg. Mean PO_2 in exacerbation was found to be 55.0 which indicate severe hypoxemia and the patient is having respiratory insufficiency. These patients presented with acute respiratory distress .

Mean HCO_3 levels in stable COPD patients was found to be 30.8 mmol/L in stable COPD patients . This raised bicarbonate levels are as a result of renal compensation to maintain near normal Ph. Mean HCO_3 levels in exacerbation patients was found to be 40.78mmol/L. Bicarbonate is produced by the kidneys and acts as a buffer to maintain a normal pH. The normal range for bicarbonate is 22 – 26mmol/l.The kidney plays a pivotal role in acid base balance.They excrete H^+ ions in exchange of HCO_3^- whenever H^+ rises above normal. Thus near normal pH is maintained in stable COPD patients. However in exacerbation COPD patients , H^+ ions are overloaded to such a extent that renal compensation is not able to maintain normal/near normal arterial pH.These patients are thus having respiratory acidosis inspite of increase in bicarbonate ions.

Mean SO_2 in stable COPD group was found to be 88.9% within a range of 84.2-93.1. Mean SO_2 in exacerbation group was 83.0% within a range of 68.2-86.1, whereas mean SO_2 in cor pulmonale group was 76.4% within a range of 58.9-79.6% . Patients of COPD exacerbation and cor pulmonale had low oxygen saturation and presented with acute dyspnoea.The above acid base findings were consistent with those found in most of the studies in the literature .

Discussion

In the present study arterial blood gas analysis was done in copd patients and acid base abnormality was found among the two groups viz. stable patients and in exacerbation. Arterial blood gas analyses play an important role in the management of Chronic Obstructive Pulmonary Disease (COPD). These analyses are relevant in the diagnosis of chronic respiratory failure and in long-term, continuous oxygen therapy monitoring. Arterial blood gas measurements can be performed in a clinical laboratory or near the patient as Point-of-Care Testing (POCT). Assessment of blood gas values play an important role in the evaluation of clinical condition and making decision regarding treatment options. It is a very useful tool to obtain information about oxygenation ,ventilation and acid base status of the body.

Male female ratio of Chronic Obstructive Pulmonary Disease was found to be 3:2. Maximum number of patients were in 46-55 years age group(42.86%) followed by 56-65 years of age group(34.29%). The mean age of study population of COPD was found to be 53.86+-7.80 years . Incidence of COPD is increasing among females as well. This is usually attributed to the historically higher rates of cigarette smoking among men and their greater likelihood of exposure to significant occupational respiratory irritants.

According to this study 54.29% of COPD patients were residents of urban areas and 45.71% of patients came from rural areas. Incidence of COPD was higher in females of rural areas(57.15%).Whereas incidence of COPD amongst male was higher in urban areas. Most of the males(80.95%) were smokers and few(14.29%) were ex smokers.However, none of the female patient of COPD was current smoker. Out of 42 COPD patients who were either current or ex-smokers, majority of the patient had 10-20 pack years(85.72%) followed by 20-30 pack years(9.52%) and 4.76% of patients smoked less than10 pack years. Smoking is by far the most important risk factor associated with functional decline in COPD . As well as having more respiratory symptoms, smokers also have a greater annual rate of lung function decline, as assessed by change in FEV_1, and are at greater risk of premature mortality due to COPD than nonsmokers. Stopping smoking can prevent the onset of disability and reduce the rate of functional decline and the risk of premature mortality . Irrespective of such considerations, stopping smoking has positive benefits on functional decline in individuals with COPD. Exposure to occupational dusts and chemicals and air pollution are also important risk factors and can cause COPD independently of tobacco smoke . Such exposure results in inflammation, a key factor in the pathogenesis of COPD. Chronic inflammation throughout the airways, parenchyma, and pulmonary vasculature are hallmarks of the disease process and lead to the pathologic changes characteristic of COPD. According to global initiative for chronic obstructive lung diseases(GOLD)2017 criteria,out of 70 patients ,40(57.13%)were having moderate COPD and 2 (2.84%) patients were having very severe COPD. Maximum number of patients were having their COPD illness between 5-10 years(52 out of 70, 74.28%).Mean duration of COPD diagnosis among study population was 9.37+-4.66 years.Out of total 70 patients 40(57.14%) were of stable COPD and 30(42.86%) were in exacerbation at the time of presentation.

Mean pH amongst stable COPD patients were 7.431 in the arterial blood samples with a range of 7.339-7.495 . These values lie within normal range of 7.350 to 7.450. Values less than 7.350 indicate acidosis whereas values higher than 7.450 indicate alkalosis. Mean ph amongst COPD exacerbation phase was found to be 7.248 within a range of 7.148-7.340. pH is a logarithmic scale of the concentration of hydrogen ions in a solution. It is inversely proportional to the concentration of hydrogen ions.When a solution becomes more acidic the concentration of hydrogen ions increases and the pH falls.Normally the body's pH is closely controlled at between 7.35 – 7.45. This is achieved through buffering and excretion of acids. Buffers include plasma proteins and bicarbonate (extracellular) and proteins, phosphate and haemoglobin (intracellularly). Hydrogen ions are excreted via the kidney and carbon dioxide is excreted via the lungs.. Mean PCO_2 in stable COPD patients was found to be 48.9mm Hg within a range of 34.5-58.2 mm Hg. This value is slightly higher for normal values of 35-45 mm Hg. Similarly mean PCO_2 in exacerbation was found to be 69.7mm Hg indicating hypercapnea. Respiratory acidosis due to hypercapnia is a common and severe complication observed in patients with chronic obstructive pulmonary disease in advanced phase. Development of acidosis worsens the prognosis and is associated with higher mortality rate. Mechanisms of compensation consist of an increased Mean PCO_2 in stable COPD patients was found to be 48.9mm Hg within a range of 34.5-58.2 mm Hg. This value is slightly higher for normal values of 35-45 mm Hg. Similarly mean PCO_2 in exacerbation was found to be 69.7mm Hg , indicating hypercapnea. Respiratory acidosis due to hypercapnia is a common and severe complication observed in patients with chronic obstructive pulmonary disease in advanced phase. Development of acidosis worsens the prognosis and is associated with higher mortality rate. Mechanisms of compensation consist of an increased renal reabsorption of bicarbonate and increased excretion of H^+. Mean PO_2in stable COPD was found to be 71.96 indicating mild hypoxemia as normal values of PO_2 range from 80-100 mm Hg. Mean PO_2 in exacerbation was found to be 55.0 which indicate severe hypoxemia and the patient is having respiratory insufficiency. These patients presented with acute respiratory distress .

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Mean PO_2 in stable COPD was found to be 71.96 indicating mild hypoxemia as normal values of PO_2 range from 80-100 mm Hg. Mean PO_2in exacerbation and in cor pulmonale was found to be 55.0 and 52.4 which indicate severe hypoxemia and the patient is having respiratory insufficiency. These patients presented with acute respiratory distress .

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The above acid base findings are consistent with those found in most of the studies in the literature .***Kettel et al. (1971***)and ***Warren et al. (1961)*** reported an higher mortality rate in COPD patients with a pH value at admission below 7.23 and 7.26, respectively. ***Jeffrey et al(1992)***did a study on 139 COPD patients and concluded that arterial H^+concentration is an important prognostic factor for survival . Similar findings were reported by more recent papers confirming that a more severe acidosis worsens the outcome of COPD patients..***Varinder saini et al(1993)***studied 348 patients of chronic obstructive pulmonary diseases (COPD) for their acid base profie. 185 patients (53.1%) had simple disorders (respiratory acidosis—53%, respiratory alkalosis—25.4%, metabolic acidosis—11.3%, metabolic alkalosis—10.2%). Mixed disorders were present in 131 patients (34.9%) (respiratory acidosis and metabolic acidosis—75.2%, respiratory acidosis and metabolic acidosis—14%, metabolic acidosis and metabolic alkalosis—5.7%, metabolic alkalosis and respiratory alkalosis—4.9%). Hypoxemia without other acid base abnormalities was observed in early patients of COPD (42 patients—12%). Chronic respiratory acidosis was the most common finding in advanced cases of COPD (98%). An almost equal number of such patients had a mixed disorder of respiratory acidosis with metabolic alkalosis (91%)..***Fuso L et al(1995)***discussed in a prospective cohort study of 1,016 adult patients from five hospitals who were admitted for a COPD exacerbation with a PCO_2 value greater than 50 mm Hg, that survival was independently related to severity of the illness, body mass index, age, prior functional status, PaO2 , inspiratory oxygen fraction (FiO2), congestive cardiac failure, serum albumin, and the presence of cor pulmonale.

Conclusion

Arterial blood gas analysis is a simple procedure to determine gas exchange levels in the blood related to respiratory, metabolic, and renal function in COPD patients. Its ability to be performed at the patient's bedside, and its rapid analysis makes it an important tool to initiate and redirect the treatment of the patients, especially in patients who are critically ill. In this study out of 70 patients 42(60%) were males and 28(40%) were females. 54.29% of the patients were residents of urban areas and 45.71% resided in rural areas . Smoking was found to be an important risk factor for COPD. Most of the males (80.95%) were current smokers whereas 14.29% of males were ex smokers and 7.14% of females were ex smokers. Majority of the patients had 10-20 pack years.Cessation of smoking was found to be associated with a better outcome.COPD exacerbation patients presented with respiratory acidosis and hypercarbia. It was found to be associated with poorer outcome and longer hospital stay. Whereas COPD patients in stable phase were having near normal ABG values and better outcome with short hospital stay time.

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