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Research Article

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ARTERIAL BLOOD GAS ANALYSIS IN ACUTE AND CHRONIC BRONCHIAL ASTHMA

[†]PADMAVATHI. K , [††]SUMANGALI.P, [†††]SUBASH .Y.E

[†@]Assistant professor, Department of Physiology, Siddhartha Medical College, Gunadala, Vijayawada-520 008 (A.P.) India

[††]Assistant Professor. Department of physiology, Siddhartha medical College, Vijayawada

[†††]Professor, Department of physiology, Siddhartha medical college Vijayawada

@Email: suma_penumalla@yahoo.com

Padmavathi. K

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ABSTRACT

Arterial blood gas sampling is a very important investigation in the assessment of a patient's acid-base status and oxygenation in acute and chronic bronchial asthma. It is the gold standard test which measures amounts of oxygen and carbon dioxide as well as the pH of the blood while it also evaluates the effective delivery of oxygen to the blood by the lungs and effective elimination of the carbon dioxide by the lungs. Arterial blood gas analysis is done using a Blood gas analyzer with the blood sample (2 ml heparinized) drawn from the radial or ulnar arteries. The results are obtained within 125 seconds. The present study conducted on 40 patients aims at assessing the blood gas status in individuals with acute and chronic bronchial asthma. The cumulative analysis of all the three parameters considered in this study show a significant variation in pH between acute and chronic asthma with a P value <0.001, a significant variation in PaCO2 with a P value <0.001 showing a tendency to hypercapnoea in severe acute asthma and with a tendency towards hypocapnoea in chronic bronchial asthma. There are very few studies on chronic bronchial asthma. A comparison between severe acute and chronic bronchial asthma showed a variable pH in acute cases depending on the severity of the airways obstruction, showing cases of both respiratory acidosis and alkalosis. While in chronic bronchial asthma the pH was within a narrow range with no cases of acidosis and a moderate number of cases showing respiratory alkalosis.

Key words: Arterial Blood Gas Analysis, Acute Bronchial Asthma, Chronic Bronchial Asthma.

INTRODUCTION

Arterial blood gas sampling represents a very superior investigation to assess a patient's acid-base status. The evaluation of oxidation and acid base status of any critically ill patient is crucial to their proper management. In contrast to the detailed understanding of the pathological change observed in episodes of broncho constriction in bronchial asthma, little is known of the physiological functions during such episodes. The physician caring for the patient is to rely on clinical impression to assess the severity of the episode. The risk of death in severe acute attack of asthma and the trend towards vigorous and complex treatment makes it necessary to have more reliable methods of assessment, one of which is arterial blood gas analysis (9). This study is undertaken to observe the P^H PaO_2 and PaCO2 in patients with acute and chronic bronchial asthma at the time of admission to the hospital. During the early stages of severe asthmatic attack the arterial $PaCo_2$ is below normal and a rise to normal levels together with increasing hypoxemia is an ominous sign (6). The arterial pH usually simply reflects the ventilation. The net effect of a markedly decreased arterial

PaO_2 with increased PaCO2 denoting impending respiratory failure (2, 6). Arterial blood gas analysis (ABG) is the gold standard test which measures the amounts of O_2 and Co_2 in the blood as well as the acidity (pH) of the blood. An ABG analysis evaluates how effectively the lungs are delivering O_2 to the blood and how efficiently they are eliminating Co_2 from it. The test also indicates how well the lungs and kidneys are interacting to maintain normal blood pH (4). Blood gas studies are usually done to assess respiratory disease and other conditions that may affect the lungs. In addition the acid base component of the test provides information on kidney function (4). ABG is performed on blood from an artery. It measures PaO_2, PaCO2, pH, HCO_3, and O_2ct (O_2 saturation). ABG is fundamentally recommended if the physician suspects significant O_2 – Co_2 exchange aberration or acid-base imbalance. Therefore, in suspected patients, measurement of ABG tensions plays a very crucial role. Other indications for ABG are: To manage patients in ICU, to guide therapy in patients in ICU's, and to determine prognosis in clinically ill patients (4).

Materials and Methods

Selection Criteria

A "Medica" easy blood gas analyzer was used and patients with a known history of bronchial asthma presenting with all the usual features were subjected to the test. Children, pregnant women, smokers and patients suffering from other respiratory disorders were excluded from the study. The sample size was limited to 20 cases of severe acute asthma and 20 cases of chronic bronchial asthma.

Procedure and Technique

An informed consent was first obtained from the patient after carefully explaining the procedure in detail. The radial and ulnar arteries were palpated and Allen's test (12) was performed. The patient was placed in a supine position and a rolled towel was placed under the wrist to maintain hyperextension. A 2 CC syringe with 24 G needle was flushed with Heparin (25,000 IU in 5 ml). This was used to pierce the skin at an angle of 45° and then the radial artery was entered into. 2 CC blood was drawn from the radial artery and pressure was applied over the site of puncture for 2-5 minutes to prevent the formation of a hematoma. Blood sample was analyzed using the "Medica" easy blood gas analyzer. The instrument gives the values of pH, $PaCO_2$ and PaO_2. Results are typed within 125 seconds. The risk to the patient is very minimal. Extreme care was taken while drawing and during disposal of the sample of blood. As far as the reference values were concerned the range for pH was between 7.35 to 7.45, for PaO_2 80 to 100 mm Hg and for $PaCO_2$ 35 to 45 mm Hg (2, 11).

Results and Data Analysis

Arterial blood gases are one of the first tests to be done on any critically ill patient because the results give useful information about Oxygenation and ventilation, both of which are seriously disturbed in bronchial asthma especially in severe acute asthma. In the present study the levels of pH, PaO_2 and $PaCO_2$ were studied in 2 groups of patients with severe acute and chronic asthma.

Table 1 and 3**.** In the severe acute asthma group consisting of 20 patients 8 were males and 12 were females. Age of the patients ranged between 25 and 79 years with most of the patients aged above the age of 60 years. This shows that there is increased incidence of severe acute asthma in the <40 and >70 age group where as the incidence is moderate in the 40-60 age group. Moreover the number of females presenting to the hospital with severe acute asthma is slightly more when compared to the number of males.

TABLE 1
SEVERE ACUTE ASTHMA – AGE DISTRIBUTION

AGE	NO. OF PATIENTS	PERCENTAGE
<40	5	25
40-49	3	15
50-59	3	15
60-69	4	20
>70	5	25
TOTAL	**20**	**100**

TABLE 2
CHRONIC ASTHMA – AGE DISTRIBUTION

AGE	NO.OF PATIENTS	PERCENTAGE
<40	1	5
40-49	6	30
50-59	4	20
60-69	7	35
>70	2	10
TOTAL	**20**	**100**

TABLE 3
SEX DISTRIBUTION IN BOTH SEVERE ACUTE AND CHRONIC ASTHMA

TYPE OF ASTHMA	MALE	PERCENTAGE	FEMALE	PERCEN	TOTAL
SEVERE ACUTE ASTHMA	8	40	12	60	100
CHRONIC ASTHMA	10	50	10	50	100

Table 2 and 3 This comprises the chronic bronchial asthma group showing increased incidence in the 40-70 age groups where as the incidence is low in the <40 and >70 age group which is in sharp contrast to the age distribution of severe acute asthma. Moreover, the incidence is equal among males and females.

TABLE 3
SEX DISTRIBUTION IN BOTH SEVERE ACUTE AND CHRONIC ASTHMA

TYPE OF ASTHMA	MALE	PERCENTAGE	FEMALE	PERCENTAGE	TOTAL
SEVERE ACUTE ASTHMA	8	40	12	60	100
CHRONIC ASTHMA	10	50	10	50	100

Table 4 and 5 The number of patients presenting with respiratory acidosis was higher (45%) than those presenting with respiratory alkalosis (20%) in severe acute asthma where as in the chronic bronchial asthma group patients presented either with respiratory alkalosis (55%) or with a normal acid base status (45%). There were practically no cases presenting with respiratory acidosis.

TABLE 4
INCIDENCE OF ACID BASE STATUS IN SEVERE ACUTE ASTHMA

ACID BASE STATUS	NO. OF PATIENTS	PERCENTAGE
Normal	7	35
Respiratory Acidosis	9	45
Respiratory Alkalosis	4	20

TABLE 5

INCIDENCE OF ACID BASE STATUS IN CHRONIC ASTHMA

ACID BASE STATUS	NO. OF PATIENTS	PERCENTAGE
Normal	9	45
Respiratory Acidosis	Nil	0
Respiratory Alkalosis	11	55

Table 6 The incidence of hypoxemia is almost equal in the 2 groups

TABLE 6
DISTRIBUTION OF PaO2 IN BOTH TYPES OF ASTHMA

SEVERE ACUTE ASTHMA		**CHRONIC ASTHMA**	
PaO2	No. of Pts	PaO2	No. of Pts
Normal	9	Normal	8
PaO2↓ Hypoxaemia	11	PaO2↓ Hypoxaemia	12

Table 7 The incidence of Hypercapnoea is almost double in the severe acute asthma group with 9 patients presenting with an increased $PaCO_2$ where as only 4

patients presented with a decreased $PaCO_2$. In the chronic bronchial asthma group 11 patients out of the total 20 presented with a decreased $PaCO_2$ where as there was no patient with an increased $PaCO_2$ showing that the incidence of hypercapnoea is extremely rare in the chronic bronchial asthma group.

TABLE 7
DISTRIBUTION OF PaCO2 IN BOTH TYPES OF ASTHMA

SEVERE ACUTE ASTHMA		CHRONIC ASTHMA	
PaCO2	No. of Pts	PaCO2	No. of Pts
Normal PaCO2	7	Normal PaCO2	9
Hypercapnoea ↑ PaCO2	9	Hypercapnoea ↑ PaCO2	Nil
Hypocapnoea ↓	4	Hypocapnoea ↓	11

RELATIONSHIP OF $PaCO_2$ AND pH IN SEVERE ACUTE ASTHMA

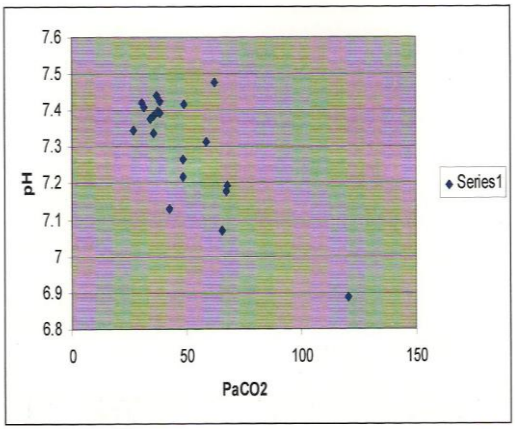


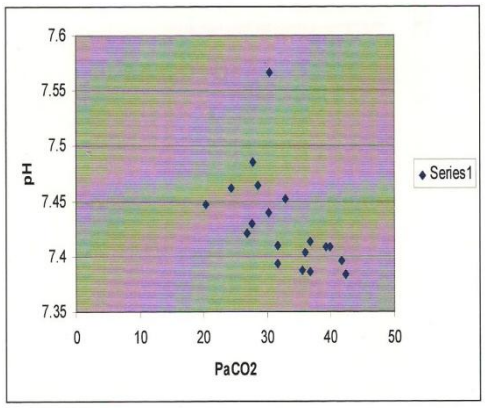
Table 8 The cumulative analysis of all the 3 parameters considered in this study show that there is a significant variation in pH between acute and chronic asthma with a P value less than 0.001. At the same time there is also a similar significant variation in $PaCO_2$ with a P value less than 0.001 with a tendency to hypercapnoea (respiratory acidosis) in severe acute asthma and a tendency towards hypocapnoea (respiratory alkalosis) in chronic bronchial asthma.

TABLE 8
CUMULATIVE ANALYSIS OF PaO_2, $PaCO_2$ & pH IN SEVERE ACUTE & CHRONIC ASTHMA

VARIABLES	ACUTE	CHR-ONIC	P VALUE
pH mean	7.31	7.428	<0.001 S
S.D	0.1499	0.0433	
$PaCO_2$ mean	48.91	32.85	<0.001 S
S.D	21.227	5.88	
PaO_2 mean	73	70	>0.001NS
S.D	15.9	15.98	

S = Significant NS = Not Significant

RELATIONSHIP OF $PaCO_2$ AND pH IN CHRONIC ASTHMA



DISCUSSION

Arterial blood gases are one of the first tests done on any critically ill patient because the results give useful information about: 1) Oxygenation and 2) Ventilation, both of which are seriously disturbed in bronchial asthma, especially in severe acute asthma (2). The incidence morbidity and mortality of asthma are increasing and its fundamental cause is still unknown despite intensive research (2). In the present study the levels of pH, $PaCO_2$, and PaO_2 were studied in 2 groups of patients with severe acute and chronic bronchial asthma. In the severe acute asthma group consisting of 20 patients, 12 females presented with severe acute asthma while the rest, that is 8 were male individuals. Age of the patients ranged between 25 years and 79 years with 9 patients above the

age of 60 years. 8 patients had normal PaO_2, whereas 12 patients showed hypoxemia, and severe hypoxemia in 3 patients with a PaO_2 less than 60 mm Hg. The pH was less in 7 patients where as it was within normal limits in 13 patients. The $PaCO_2$ showed clear cut respiratory acidosis in 6 cases with $PaCO_2$ above 45 mm Hg and pH less than 7.35. The $PaCO_2$ showed an increase in 3 cases but in these the pH was normal indicating probable compensation. The $PaCO_2$ showed a decrease in 4 cases indicating respiratory alkalosis but with normal pH. The progression of arterial blood gas disorders that occurs in severe acute asthma is not a uniform process. In the early stage the usual manifestations are hypocapnoea (6) due to hyperventilation and hypoxemia due to ventilation/ perfusion (VA/Q) inequality (mismatch). The statement that in a patient with severe acute asthma, there may be initial hyperventilation with decreased $PaCO_2$ that is respiratory alkalosis may appear contradictory. This can be explained as follows: Even in patients with severe airways obstruction the increase in airway resistance is not uniform throughout, so that inspired air is directed preferentially to areas in the lung with the lowest flow resistance. These areas may occupy less than 25% of the lung volume yet may receive over 80% of the inspired air and act as if they ventilate at twice the rate of normal lung. Thus a relatively small number of alveolar units hyperventilate (in relation to their perfusion) giving rise to an increase in ventilation perfusion ratio, hypocapnoea and respiratory alkalosis. The rest of the alveoli would receive less of the inspired air with a fall in the ventilation perfusion ratio which results in hypoxemia. This hypoxemia cannot be compensated for by the few hyperventilating units so that an overall fall in PaO_2 occurs. However the $PaCO_2$ may vary. It will be low if there are a sufficient number of hyperventilating units that wash off excess CO_2. It will be normal if there is a balance between hyperventila1ting and hypoventilating units. It will be raised when the overall ventilation perfusion ratio of the whole lung is clearly reduced (13). In the present study the 4 cases showing decreased $PaCO_2$ are examples of the early stage of severe acute asthma (6). Out of the 4 cases 3 are associated with severe hypoxemia. This confirms that the hyperventilating units in these cases could wash off the excess CO_2 but could not compensate for the severe hypoxemia, because of the sigmoid shape of the oxygen dissociation curve. The clear cut 6 cases of respiratory acidosis are the typical cases in which the condition has worsened and where the overall ventilation perfusion ratio of the whole lung is clearly reduced. The PaO_2 was normal in 7 cases. These represent instances, where a balance was established between the hyperventilating and hypoventilating units of the lung. In the study conducted by F.E.Udwadia (10) on 17 patients, 8 had normal $PaCO_2$ (47%), 4 had hypercapnoea (23%) and hypocapnoea was observed in 5 patients (29%), where as in our study hypercapnia was seen in 9 patients out of 20 (45%) and hypocapnoea in 4 patients (20%). In his study all patients showed severe hypoxemia, where as in the present study only 60% showed hypoxaemia with 3 cases out of 20 showing severe hypoxaemia. In Udwadia's (10) study mean pH was 7.41 ± 0.02 with 5 patients out of 17 (29%) showing respiratory acidaemia and 4 patients (23%) showing respiratory alkalaemia. In our study mean pH was 7.31± 0.149 with 7 cases out of 20 (35%) showing respiratory acidaemia. Eventhough 4 cases showed hypocapnia these were not associated with a raised pH so there were no clear cut cases of respiratory alkalemia. In the study done by Dr.John C.Varghese (7) on 42 patients, 21 patients (50%) showed hyper-carbia, 9 patients showed hypocarbia (21%) and normocarbia was observed in 12 patients (28%). This study showed close correlation with our study. In the study done by Odhiambo J.A. and Chwala R.D (9) on 40 patients, marked degrees of hypoxaemia (mean PaO_2 of 60 mmHg) hypocapnia (mean $PaCO_2$ of 34 mm Hg) with apparently normal pH (mean 7.384) were documented in majority of the patients. Because the $PaCO_2$ in this study was most frequently normal or low even in the more severe phases of asthma, the authors observed that elevated $PaCO_2$ therefore indicates very severe disease state that calls for vigorous therapy. They further stated that assessing the patient by blood gas level and pH appears crucial as it alerts the clinician to potentially explosive situations. In the chronic bronchial asthma group of 20 patients, 6 patients had normal arterial O_2 tension. In 14 patients the PaO_2 was less than normal with 5 out of these showing severe hypoxemia of less than 60 mm Hg. The pH was normal in 16 patients and in 4 it was elevated showing alkalaemia. The $PaCO_2$ was normal in 9 patients where as it was decreased in the rest of the patients that is 11 cases with no cases showing elevated $PaCO_2$. There are very few studies on chronic bronchial asthma. In a study conducted by Cochrane, J.G. Prior and CB Wolf (1) on 29 patients with chronic asthma the pH ranged around 7.46, $PaCO_2$ was decreased in 14 cases (48%) and PaO_2 was decreased in 16 cases (55%). In our study the pH ranged between 7.38 to 7.48 with only one case showing an elevated p^H of 7.56, the $PaCO_2$ was

decreased in 11 cases out of 20 (55%) showing a very close correlation with the above study. However decreased PaO_2 was observed in 14 cases out of 20 (70%) showing that the incidence of hypoxaemia was higher in the present study. When we make a comparison between the 2 groups we can see that the pH was variable in the acute cases and depending on the severity of the airway obstruction there were cases showing respiratory acidosis and respiratory alkalosis. Where as in chronic bronchial asthma the pH was within a very narrow range, there were no cases of acidosis and a moderate number of cases showing respiratory alkalosis. This is in accordance with the observation made by Marc.H Lavietes (8) that in an asthmatic patient whose airway obstruction is not severe hypocapnia is the rule.

CONCLUSION

Arterial blood gas analysis is an extremely useful investigation in assessing oxygenation, pH and $PaCO_2$ in severe acute asthma. This study demonstrated that the characteristic blood gas pattern found in severe asthma is that of hypoxaemia with respiratory acidosis in a moderate number of patients and hypoxaemia with respiratory alkalosis in the rest (i.e. among those with abnormal blood gas patterns). In the chronic bronchial asthma group the study shows that hypoxaemia with respiratory alkalosis was present in a moderate number of cases but none showed respiratory acidosis. The pH also in this group was maintained within normal limits of 7.38 to 7.49 indicating the stability of the clinical condition. This confirms that many patients with chronic bronchial asthma maintain remarkably stable arterial blood gas tensions.

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