



A Prospective Study of Arterial Blood Gases in Bronchial Asthma in a Tertiary Care Hospital

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Abstract

Asthma is serious global health problem affecting all age groups with increasing prevalence in many countries. This main function of gas exchange and thus acid-base balance is affected by diseases of the lung. So it is expected that asthma also lead to respiratory acid-base disorders. In addition, the resulting hypoxemia can reduce tissue oxygenation causing lactic acidosis. Also chronic hypocapnia can lead to compensatory reduction in plasma bicarbonate resulting in metabolic acidosis. Thus functional compromise measurement reflects severity of that pathology and its outcome. Similarly measurement of blood oxygen, carbon dioxide and pH may help in assessing severity of respiratory pathology. So this prospective study was aimed to study arterial blood gas analysis with severity and outcome of asthma. 43 patients of asthma were selected randomly. During treatment of patient periodical ABG was tested to assess the management. We found that in asthma patient respiratory alkalosis i.e. hypocapnia (48.83%) was most common acid base abnormality followed by normal ABG (46.5%). Respiratory acidosis i.e. hypercapnia was seen only in 4.65%.patients. Hypercapnia and metabolic acidosis in asthma was associated with severity of disease. Significant fall in PaO₂ and increase in alveolar-arterial gradient is seen in very severe asthma.

Keywords: Bronchial asthma, PaO₂, PaCO₂, pH value, arterial blood gases.

Introduction

Asthma is serious global health problem affecting all age groups. Epidemiologic data suggest that its prevalence is increasing in many countries. Even though there is availability of effective therapies, international surveys show suboptimal asthma control in many countries. The respiratory system is very quick for compensation in case of metabolic disturbance. But the renal system acts very slowly to compensate to respiratory diseases.

So change in plasma bicarbonate is characteristic of chronic lung disease rather than acute. The function of the respiratory system is to maintain homeostasis of blood gases like O₂, CO₂ as well as arterial pH.¹ This main function of gas exchange and thus acid-base balance is affected by diseases of the lung. So it is expected that asthma will also lead to respiratory acid-base disorders. In addition, the resulting hypoxemia can reduce tissue oxygenation and thus anaerobic metabolism

causing lactic acidosis. Additionally, chronic hypocapnia can lead to compensatory reduction in plasma bicarbonate concentration resulting in metabolic acidosis. Thus functional compromise measurement reflects type and severity of pathology and its outcome. Similarly measurement of blood O₂, CO₂ and pH may help in assessing severity of respiratory pathologies. So we have studied arterial blood gas values with severity and outcome of asthma.

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 asthma 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 < 13 years were excluded from study. Total 43 patients of asthma were selected randomly. Diagnosis of asthma, severity of disease and management was done as per GINA guideline for asthma.²

Management: Management of asthma was done as per standard protocol with bronchodilators, antibiotics, inhalational or systemic steroids as per severity and standard guidelines, oxygen supplementation as per need, mechanical ventilation for patients of respiratory failure.

Result

Table 1: ABG Interpretation in Patients of Asthma at time of Diagnosis

Acid-Base Interpretation	No of Patients (n)	Percentage (%)
Normal	20	46.51%
Respiratory alkalosis	20	46.51%
Mixed acidosis	02	4.65%
Respiratory alkalosis with metabolic acidosis	01	2.17%
Total	43	100%

Table 2: ABG Interpretation in Asthma Patients on Discharge

Acid-Base Interpretation	No of Patients (n)	Percentage (%)
Normal	39	90.7%
Respiratory alkalosis	04	09.3%
Total	43	100%

First ABG sample at time of admission was collected after taking written informed consent of patient. Detailed history, clinical examination and initial necessary management of patient were done. All necessary investigations were done to confirm the diagnosis. During the treatment periodical ABG testing was done to assess the management as per need. At time of discharge last ABG was tested. All the data collected in Microsoft office excel sheet.

Procedure of Collection of Arterial Blood Sample:^{3,4}

Radial artery was selected for collection of arterial blood sample as it is relatively easy to palpate and stabilize with good collateral blood supply from the ulnar artery. Then with aseptic precaution under local anaesthesia at least 3ml of arterial blood was collected in heparin flushed syringe. Sample was analysed quickly as delay can 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. The best values were kept for analysis.

Statistical Methods

Values are expressed as mean \pm standard deviation. Comparisons were done by using Unpaired T Test. Statistical significance was accepted at the 95% confidence level (p<0.05).

Table 3: ABG Parameter in Patients of Asthma (ABG Sample Collected when patient was Breathing `Room Air) {n=43}:

ABG parameter	pH Mean (SD)	PaCO ₂ Mean (SD)	PaO ₂ Mean (SD)	HCO ₃ ⁻ Mean (SD)	PAaO ₂ Mean (SD)
On Admission	7.44 ±(0.04)	34.11 ±(4.46)	72.18 ±(10.75)	22.45 ±(1.45)	36.59 ±(11.63)
While Discharge	7.41 ±(0.02)	38.3 ±(2.32)	81.85 ±(4.99)	23.4 ±(1.21)	21.94 ±(6.19)

Table 4: Acid-Base Abnormality and Severity of Asthma

	Normal	Respiratory alkalosis	Respiratory acidosis	Metabolic acidosis
Mild asthma	06	04	-	-
Moderate asthma	13	07	-	-
Severe asthma	01	09	02	03

Table 5: Hypoxemia and Alveolar-Arterial Gradient with Severity of Asthma at Time of Admission:

	Mild Asthma	Moderate Asthma	Severe Asthma
PaO ₂	78.6 ± (5.77)	75.74 ± (10.34)	62.07± (10.75)
PA-aO ₂	28.39 ± (8.13)	33.17 ± (9.93)	48.16 ± (11.63)

Table 6: Verifying Significance using Unpaired T Test, For Hypoxemia and Alveolar-Arterial Gradient in Relation to Severity:

Comparison groups	t value	p value	Significance
PaO ₂ of mild asthma with moderate asthma	t ₂₈ = 0.44	p > 0.1	NS
PAaO ₂ of mild asthma with moderate asthma	t ₂₈ = 0.25	p > 0.1	NS
PaO ₂ of moderate asthma with severe asthma	t ₃₁ =2.35	p<0.05	Significant
PAaO ₂ of moderate asthma with severe asthma	t ₃₁ =2.05	p<0.05	Significant

Table 7: Effect of NPPV on ABG Parameter of 2 Patients of Asthma 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	7.29 ±(0.01)	46.2 ±(1.42)	50.5 ±(2.12)	20.65 ±(0.64)	43.79 ±(0.42)
After NPPV	7.38 ±(0.0)	40.5 ±(0.71)	75 ±(4.24)	23.45 ±(0.35)	26.13 ±(3.39)

Discussion

Amongst 43 patients, 10 patients of mild asthma (mean FEV₁ - 82.15%) had mean PaO₂ and mean PA-aO₂ of 78.6 mmHg and 28.39 mmHg respectively. 20 patients of moderate asthma (FEV₁ - 65.55%) had mean PaO₂ and mean PA-aO₂ of 75.74 mmHg and 33.17 mmHg respectively. Remaining 13 patients of severe asthma (mean FEV₁ - 46%) had mean PaO₂ and mean PA-aO₂ of 62.07 mmHg and 48.16 mmHg respectively.

Out of 43 asthma patients, respiratory alkalosis i.e. hypocapnia was most common acid base abnormality found in 21 patients (48.83%).

Normal ABG seen in 20 patients (46.5%). Respiratory acidosis i.e. hypercapnia was seen only in two patients (4.65%). These finding correlates with study of McFadden and Lyons⁶ and Richard D et al.⁷ McFadden and Lyons found Resp. Alkalosis in 75.3%, Normal-15.8%, Resp. Acidosis -8.9%. Richard D et al found Resp. Alkalosis-47.6%, Normal-12.2%, Resp. Acidosis -10%.

Out of 43 patients of asthma, two patients of severe asthma had respiratory acidosis i.e. hypercapnia and three patients of severe asthma had component of metabolic acidosis. So hypercapnia and metabolic acidosis in asthma was

associated with severity of disease. These finding correlates with studies of McFadden and Lyons⁶ Richard D et al.⁷ McFadden and Lyons In asthma, hypercapnia occurred when FEV1 fell below 15% of predicted. Metabolic acidosis was more likely to occur in patients with evidence of more severe airflow obstruction.

Patients of mild to moderate asthma had only mild hypoxemia without any significant difference in PaO₂ and PAaO₂. But patients of severe asthma had significant fall in PaO₂ with corresponding significant change in gradient. These findings correlate with study done by P.D. Wagner et al.⁸ P.D. Wagner et al found Patient with asthma maintain PaO₂ value at mild hypoxemic level until FEV1 reached about 40% of predicted value, then with little further airway obstruction causes significant fall in PaO₂ and associated significant change in gradient.

Two patients were required NPPV for respiratory failure. With use of NPPV the mean pH normalized from 7.25 to 7.38 with correction of both hypercapnia and metabolic acidosis. Their mean PaO₂ value also improved from 50.5 mmHg to 75 mmHg with associated improvement in gradient. This finding correlates with study done by Meduri GU et al.⁹ Meduri GU et al found NPPV was used in 17 patients of respiratory failure in asthma. Their mean pH improved from 7.25 to 7.38, mean PaCO₂ from 65 mmHg to 45 mmHg with improvement in hypoxemia and gradient.

Conclusion

From our study results we conclude that various acid-base abnormalities have been observed in bronchial asthma. Acute respiratory alkalosis is most common acid-base abnormality in asthma patients as airway hyper-responsiveness leads to hyperventilation and chronic hypocapnia lead to increased renal bicarbonate loss. Isolated hypercapnia is rare in asthma patients but when present it is associated with severe asthma attacks with risk for intubation and mechanical ventilation. Finally, hypercapnia with metabolic acidosis due to lactic acidosis is rare but has also been

observed in asthma. Lactic acidosis may be due to reduced tissue oxygenation because of hypoxemia or increased oxygen demand due to increased workload of respiratory muscles. Significant fall in PaO₂ and increase in alveolar-arterial gradient is seen in very severe asthma. Patients of mild to moderate asthma maintain their blood oxygen status at low baseline level. Hence meticulous and careful evaluation of acid-base disturbances in asthma will be helpful for differential diagnosis and its treatment.

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