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

Authors
Dr Mahavir Bagrecha¹, Dr Anand Yannawar², Dr Nitin Gaikwad³, Dr Sanjay Gabhale⁴
¹Assistant Professor, Dept of Pulmonary Medicine, Dr D Y Patil Medical College, Hospital and Research centre, Pune
²Assistant Professor, Dept of Pulmonary Medicine, Dr D Y Patil Medical College, Hospital and Research centre, Pune
³Professor, Dept of Pulmonary Medicine, Dr D Y Patil Medical College, Hospital and Research centre, Pune
⁴Senior Resident, Dept of Pulmonary Medicine, Dr D Y Patil Medical College, Hospital and Research centre, Pune
*Corresponding Author
Dr Anand Yannawar
Assistant Professor, Dept of Pulmonary Medicine, Dr D Y Patil Medical College, Hospital and Research centre, Pune

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 O_2, CO_2 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.

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 ± standard deviation. Comparisons were done by using Unpaired T Test. Statistical significance was accepted at the 95% confidence level (p<0.05).

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

| ABG Parameter | pH Mean (SD) | PaCO₂ Mean (SD) | PaO₂ Mean (SD) | HCO₃⁻ Mean (SD) | PAaO₂ 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

| 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:

| Severity of Asthma | Mild Asthma | Moderate Asthma | Severe Asthma |
|--------------------|-------------|-----------------|--------------|
| PaO₂               | 78.6 ± (5.77)| 75.74 ± (10.34)| 62.07± (10.75)|
| PA-aO₂             | 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 |
|-------------------|---------|---------|--------------|
| PaO₂ of mild asthma with moderate asthma | t₃₃=0.44 | p > 0.1 | NS           |
| PAaO₂ of mild asthma with moderate asthma | t₃₃=0.25 | p > 0.1 | NS           |
| PaO₂ of moderate asthma with severe asthma | t₃₁=2.35 | p<0.05 | Significant  |
| PAaO₂ of moderate asthma with severe asthma | t₃₁=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) | PaCO₂ Mean (SD) | PaO₂ Mean (SD) | HCO₃⁻ Mean (SD) | PAaO₂ 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 Lyons ⁶ 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
found patients of mild to moderate asthma had only mild hypoxemia without any significant difference in $\text{PaO}_2$ and $\text{PAAO}_2$. But patients of severe asthma had significant fall in $\text{PaO}_2$ with corresponding significant change in gradient. These findings correlate with study done by P.D. Wagner et al.\textsuperscript{8} P.D. Wagner et al found Patient with asthma maintain $\text{PaO}_2$ value at mild hypoxemic level until FEV1 reached about 40% of predicted value, then with little further airway obstruction causes significant fall in $\text{PaO}_2$ 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 $\text{PaO}_2$ 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\textsuperscript{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 $\text{PaCO}_2$ 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 $\text{PaO}_2$ 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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**References**
1. The 10 leading causes of death by broad income group (2008). World Health Organization (WHO), Fact sheet no 310; updated June 2011.
2. Global Strategy for Asthma Management and Prevention updated 2011; link- www.ginasthma.org
3. Bhome A.B.COPD in India: Iceberg or volcano? Journal Of Thoracic Disease. 2012; 4(3): 298-309.
4. Report of The American-European Consensus Conference On Acute Respiratory Distress Syndrome: Definition, Mechanism, Relevant Outcome, And Clinical Trial Coordination; Journal of critical care.1994; 9(1): 72 -81.
5. Moore T. Respiratory Assessment In Adults; Nursing Standard. 2007; 21: 48-56.quiz 58. 10.7748/ns2007.08.21.49.48.c4605.
6. Martin J. Tobin, Karl Yang. Breathing Pattern in Asthma. 1989.95 (1):1-2.
7. Richard C, John E, Newton C, Steven A: Chest. Acid-Base Disturbances in Acute Asthma. 1990;98(3): 651–655.
8. Wagner V G. Hedenstierna R. Rodriguez-Roisin. Gas Exchange, Expiratory Flow Obstruction And The
Clinical Spectrum Of Asthma: Eur Respir J. 1996, 9: 1278–1282.
9. Meduri GU, Cook TR, Turner RE, Cohen M, Leeper KV. Noninvasive Positive Pressure Ventilation In Status Asthmaticus: Chest. 1996,110(3):767-74.