

Can venous blood gas values be used instead of arterial blood gas values in respiratory alkalosis?

[Respiratuvar alkalozu olan hastalarda venöz kan gazı değerleri arteriyel kan gazı değerleri yerine kullanılabilir mi?]

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ABSTRACT

Objective: The increase in alveolar ventilation causes disposal of large amounts of carbon dioxide from the respiratory system resulting in hypocapnia and respiratory alkalosis. Hypocapnia reduces cerebral blood flow, alkalosis leads to a leftward shift in oxygen-hemoglobin dissociation curve causing reduced oxygen delivery to tissues. Therefore, immediate diagnosis and close monitoring of respiratory alkalosis is necessary in emergency situations. In this study, the comparison of arterial and venous blood gas parameters of patients with respiratory alkalosis, and the evaluation of the usability of venous blood gas instead of arterial blood gas in patients with respiratory alkalosis were aimed.

Methods: Ninety patients with respiratory alkalosis were enrolled in this study prospectively. Arterial and venous blood gas samples of patients enrolled in the study were drawn simultaneously in room air without administering any treatment after admitting to the emergency department.

Results: The correlation between the results of pH, partial pressure of carbon dioxide (pCO₂), bicarbonate (HCO₃) and base excess (BE) (respectively, r= 0.764, r= 0.839, r= 0.843, r= 0.883) in arterial and venous blood gas samples were statistically significant (p< 0.001). Patients included in the study were divided into three groups according to the values of arterial oxygen pressure (PaO₂) of blood gas samples. PaO₂ was >80 mmHg in 23 patients (25.6%), between 60-80 mmHg in 29 (32.2%) patients and <60 mmHg in 38 (42.2%) patients. A statistically significant correlation (r= 0.540, p<0.001) detected only between arterial and venous blood gas samples of patients with PaO₂ <60mmHg and O₂ saturation <90% among these three groups.

Conclusion: In the follow-up of patients with respiratory alkalosis, if O₂ saturation is ≥90%; pH, pCO₂ and HCO₃ of venous blood gas samples can be used instead of arterial blood gas samples. If patient's O₂ saturation <90%; pH, pCO₂, HCO₃ and also pO₂ values in venous blood gas sample can be used instead of arterial blood gas samples.

Key Words: Arterial blood gas, venous blood gas, respiratory alkalosis, hypocapnia

Conflict of Interest: The authors declare that there is no conflict of interest.

ÖZET

Amaç: Alveolar ventilasyon artışı, solunum sisteminden fazla miktarda karbondioksit atılmasına neden olur, sonuçta hipokapni ve respiratuvar alkaloz gelişir. Hipokapni beyin kan akımını azaltır, alkoloz oksijen-hemoglobin çözünme eğrisinin sola kaymasına neden olarak, dokuya oksijen sunumunu azaltır. Bu yüzden acil durumlarda respiratuvar alkalozun hemen tanınması ve yakın takibi gerekir. Bu çalışmada respiratuvar alkalozu olan hastalarda arteriyel ve venöz kan gazı parametrelerinin karşılaştırılması ve respiratuvar alkalozu olan hastalarda venöz kan gazının arteriyel kan gazı yerine kullanılabilirliğinin değerlendirilmesi amaçlandı.

Metod: Çalışmaya respiratuvar alkalozu olan 90 hasta prospektif olarak alındı. Çalışmaya alınan hastaların arteriyel ve venöz kan gazı örnekleri hastaların acil servise kabulü ile birlikte herhangi bir tedavi uygulanmadan oda havasında eş zamanlı olarak alındı.

Bulgular: Arter ve venöz kan gazı örneklerinde pH, parsiyel karbondioksit basıncı (pCO₂), bikarbonat (HCO₃) ve baz fazlalığı (BE) sonuçları arasında (sırasıyla r= 0.764, r= 0.839, r= 0.843, r= 0.883) istatistiksel olarak anlamlı korelasyon saptandı (p<0.001). Çalışmaya alınan hastalar arter kan gazı örneklerindeki PaO₂ değerlerine göre üç gruba ayrıldı. Bu üç gruptan sadece pO₂ 60mmHg olan (O₂ sat <90%) hastaların arteriyel ve venöz kan gazı örnekleri arasında istatistiksel olarak anlamlı korelasyonu saptandı (r= 0.540, p<0.001).

Sonuç: Respiratuvar alkalozu olan hastaların takibinde O₂ sat ≥ 90 ise venöz kan gazı örneğindeki pH, pCO₂, HCO₃ ve BE arter kan gazı örneği yerine kullanılabilir. Hastanın O₂ saturasyonu <90% ise venöz kan gazı örneğindeki pH, pCO₂, HCO₃ ve BE ile birlikte pO₂ düzeyi de arter kan gazı örneği yerine kullanılabilir.

Anahtar Kelimeler: Arteriyel kan gazı, venöz kan gazı, respiratuvar alkaloz, hipokapni

Çıkar Çatışması: Yazarlar arasında çıkar çatışması bulunmamaktadır.

Introduction

The increase in alveolar ventilation causes disposal of large amounts of carbon dioxide (CO_2) from the respiratory system resulting in hypocapnia and respiratory alkalosis. Hypocapnia reduces cerebral blood flow; alkalosis leads to a leftward shift in oxygen-hemoglobin dissociation curve causing reduced oxygen delivery to tissues. Mild respiratory alkalosis may be considered to be harmless and may not need to intervene. On the other hand, in case of severe respiratory alkalosis, direct relationship was found between degree of hypocapnia and poor results. Therefore, immediate diagnosis and close monitoring of respiratory alkalosis is necessary in emergency situations [1,2].

In clinical practice, arterial blood samples are used to evaluate many metabolic and respiratory statuses. However, arterial puncture is often painful, and local hematoma, infection, arterial injury, hemorrhage, aneurysm, embolism or thrombosis may occur [3]. On the other hand; venous blood sampling is easier, the pain is less, it can be drawn along with blood for other tests [3-5]. Therefore, many studies have investigated the use of venous blood gas instead of arterial blood gas. In these studies; significant correlation between arterial and venous pH and bicarbonate (HCO_3) values in case of metabolic acidosis and it was stated that venous blood gas samples had showed the correct degree of acidosis [5-11]. In patients with terminal respiratory failure and respiratory acidosis, it was stated that venous pH could be used instead of arterial pH and venous partial pressure of carbon dioxide (pCO_2) was sufficient to monitor hypercarbia [3,12-15].

Patients with respiratory alkalosis were frequently encountered in clinical practice, and repeated blood gas samples are needed during the follow-up of these patients. Making many times arterial punctures occur severe distress for patients and their physicians. There have not been made any adequate studies regarding the availability of the usage of venous blood gas values instead of arterial blood gas analysis for the clinical management of patients with respiratory alkalosis.

In this study, the comparison of arterial and venous blood gas parameters of patients with respiratory alkalosis, and the evaluation of the usability of venous blood gas instead of arterial blood gas in patients with respiratory alkalosis were aimed.

Materials and Methods

This prospective study was conducted at Antalya Training and Research Hospital, Department of Emergency Medicine after approval by the local ethics committee. Patients with respiratory rate > 24 /minute and diagnosed respiratory alkalosis in arterial blood gas samples were included in this study. Metabolic acidosis, metabolic alkalosis, respiratory acidosis and mixed acid-base disorders were excluded from the study. Patients

admitted to the emergency department due to poisoning and patients with diabetes, kidney disease, liver disease and chronic lung disease were excluded. Arterial and venous blood gas samples of patients enrolled in the study were drawn simultaneously in room air without administering any treatment after admitting to the emergency department. Venous blood samples were drawn from peripheral veins opened for intravenous therapy and arterial blood samples were drawn from radial artery with heparin washed sterile syringes. Samples were delivered to the laboratory with ice within 10 minutes. Samples were evaluated with AVL compact 3[®] blood gas analyzers.

Age, gender, vital signs, oxygen saturation, diagnosis, treatment data of each patient were recorded. The pH, partial pressure of oxygen (pO_2), partial pressure of carbon dioxide (pCO_2), bicarbonate (HCO_3) and base excess (BE) results of arterial and venous blood gas samples were recorded.

SPSS 17 statistical software was used for statistical analysis. In statistical analysis; firstly, Kolmogorov Smirnov analysis performed to evaluate whether distribution of values are normal or not. Logarithm of all values were made to fit a normal distribution. It showed normal distribution ($p > 0.05$), and parametric tests were used for this reason. Statistical evaluation of the results of pH, pO_2 , pCO_2 , HCO_3 and BE were performed via Pearson correlation analysis and linear regression analysis.

Results

Totally 90 patients with respiratory alkalosis were enrolled in this study. Of the patients 46 (51.2%) were male and 44 (48.8%) were female. The average age of patients was 39 ± 15 years (range= 21-72). Respiratory rate was 30 ± 4 /minute (range= 25-42). Diagnoses of the patients are given in Table 1.

Patients included in the study were divided into three groups according to the values of pO_2 of arterial blood gas samples (PaO_2). PaO_2 was > 80 mmHg in 23 patients (25.6%), between 60 and 80 mmHg in 29 (32.2%) patients, and < 60 mmHg in 38 (42.2%) patients. A statistically significant correlation between arterial and venous blood gas samples of patients with only $\text{PaO}_2 < 60$ mmHg and O_2 saturation $< 90\%$ was detected among these three groups (Table 2). A significant relationship between arterial and venous pH, pCO_2 , HCO_3 and BE values were found with Pearson Correlation and r-values were $r = 0.839$, $r = 0.764$, $r = 0.843$, $r = 0.883$ respectively with a 95% confidence interval ($p < 0.001$) (Table 3). As strong relationship were found between arterial and venous pH, pCO_2 , HCO_3 values, and a 95% confidence interval equations were created to calculate arterial values from venous values. To do this, we created Bland Altman plots. Bland Altman bias plots of the average of the arterial and venous measurements, and the difference between them are

Table 1. Diagnoses of study patients

Disease	Number (n)	%
Acute pulmonary edema	21	23.3
Pneumonia	23	25.6
Pulmonary embolism	8	8.9
Traumatic brain injury	6	6.7
Spontaneous pneumothorax	4	4.4
Pregnant	1	1.1
Thoracic trauma	7	7.8
Anxiety	8	8.9
ARDS	1	1.1
Asthma	11	12.2
Total	90	100

Table 2. Correlation between PaO₂ values in arterial and venous blood gases.

	Arterial (mean±SD)	Venous (mean±SD)	r=	p	O ₂ saturation (mean±SD) (min-max)
PaO ₂ <60mmHg (n=38)	51.33 ±8.57	29.34±9.76	0.540**	p<0.001	82±11 (35-90)
PaO ₂ = between 60 and 80 mmHg (n= 28)	70.91±4.14	28.62±6.64	0.309	p= 0.110	94±2 (91-98)
PaO ₂ >80mmHg (n= 20)	95.17±9.39	36.49±10.01	-0.182	p= 0.417	98±2 (100-96)

** . Correlation is significant at the 0.01 level.

Table 3. Pearson correlation analysis, 95% confidence limits

	r=	Confidence limits
pCO ₂	0.764**	0.569-0.798
pH	0.839**	0.722-0.952
HCO ₃	0.843**	0.920-1.198
BE	0.883**	0.772-0.968

** . Correlation is significant at the 0.01 level.

depicted in Figures (Figures 1-5). The bias plot for each of the variables showed excellent agreement with 95% limits of agreement being in acceptably narrow range in case of all the parameters. The mean bias in pH was 0.05 (95% limits of agreement = 0.04 to 0.2); bias in pCO₂ was -7.7 (95% limits of agreement = -15.6 to 0.1), and in bicarbonate levels -0.4 (95% limits of agreement = -2.29 to 2.1). Although the arterial and venous pO₂<60mmHg were significantly correlated with each other (r= 0.540,

p< 0.001), the bias between the two was -22 (95% limits of agreement = 4.6 to 39.4). Accordingly, Arterial pH= venous pH x 0.83 + 0.14, Arterial pCO₂ = venous pCO₂ x 0.68 + 0.39, Arterial HCO₃ = venous HCO₃ x 0.09 + 1.06, PaO₂= venous pO₂ x 0.39 + 1.13 (for patients PaO₂<60 mmHg and O₂ saturation <90%) (Table 4). The arterial and venous values for BE were very close, and it has not been created a healthy formula for BE (Figure 5).

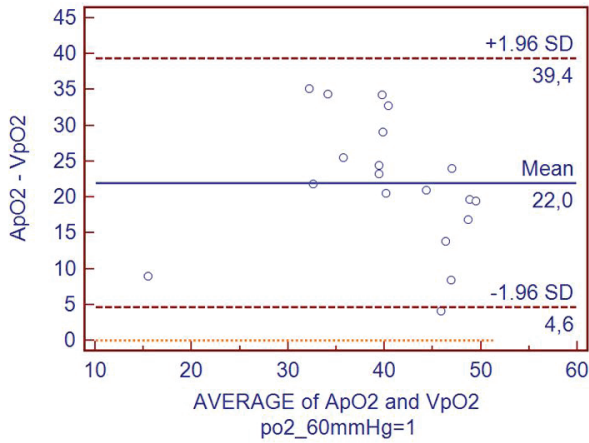


Figure 1. Bland Altman plots of arterial and venous pO₂ (Average vs. Difference)

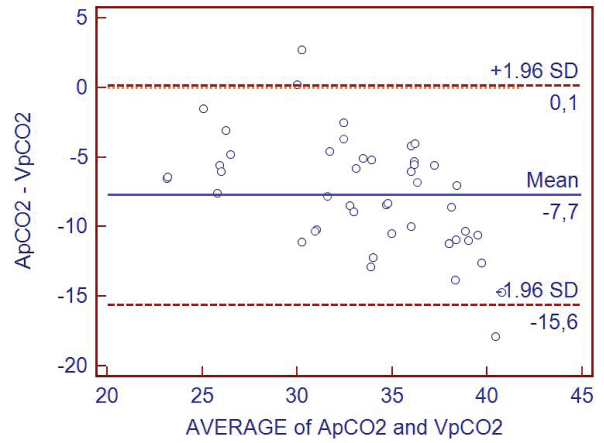


Figure 2. Bland Altman plots of Arterial pCO₂ (ApCO₂) and venous pCO₂ (VpCO₂) (Average vs. Difference)

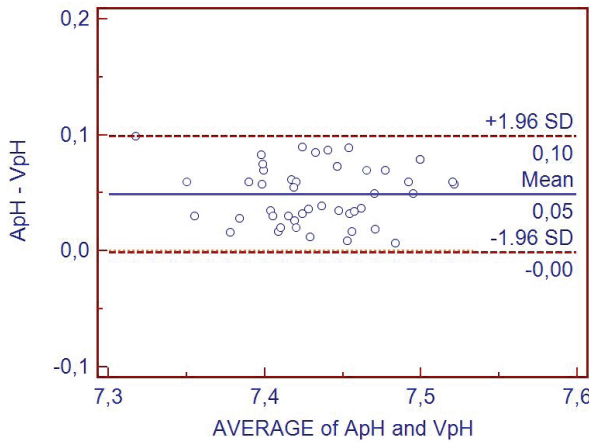


Figure 3. Bland Altman plots of arterial pH (ApH) and venous Ph (VpH) (Average versus Difference)

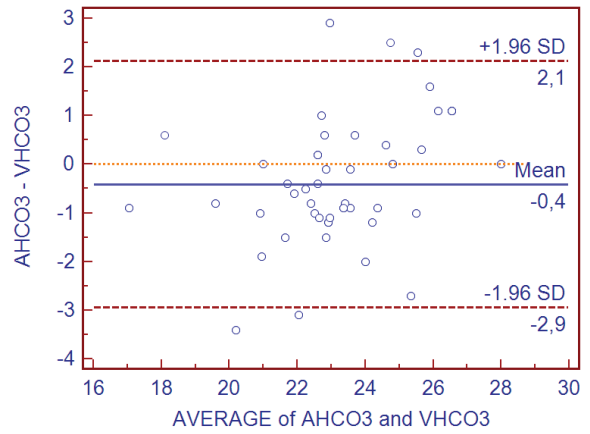


Figure 4. Bland Altman plots of arterial HCO₃⁻ (AHCO₃) and venous HCO₃⁻ (VHCO₃) values (Average vs. Difference)

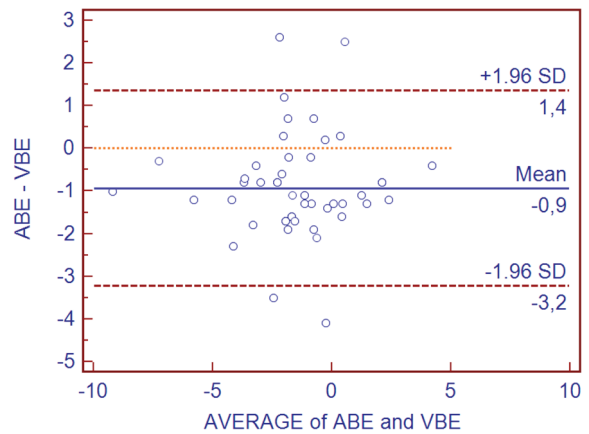


Figure 5. Bland Altman plots of arterial BE (ABE) and venous BE (VBE) (Average versus Difference)

Discussion

The increase in alveolar ventilation causes disposal of large amounts of CO₂ from the respiratory system resulting in hypocapnia and respiratory alkalosis. Respiratory alkalosis is a common acid-base disorder in clinical practice. It may occur as the result of an illness or can happen accidentally. For example, it may occur in patients undergoing mechanical ventilation without proper ventilation or with extracorporeal membrane oxygenation. Recently, respiratory alkalosis or hypocapnia is applied in the treatment of intracranial hypertension and pulmonary hypertension in children [16].

In previous studies, a direct relationship between respiratory alkalosis and the degree of hypocapnia and poor results was found in critically ill patients. For example, hypocapnia after cardiac arrest was detected

Table 4. Calculation of arterial blood gas samples using venous blood gas samples

	Regression formula
PaO ₂ <60mmHg	Venous pO ₂ x 0.39 + 1.13
Arterial pCO ₂	Venous pCO ₂ x 0.68 + 0.39
Arterial pH	Venous pH x 0.83 + 0.14
Arterial HCO ₃	Venous HCO ₃ x 0.09 + 1.06

as an independent marker for poor neurologic function [17,18]. An independent relationship between duration of hypocapnia and poor functional results and symptomatic vasospasm was found in aneurismatic subarachnoid hemorrhage [19]. In addition, maternal hypocapnia due to hyperventilation in pregnant women at birth was shown to impair placental transport of oxygen [20]. Respiratory alkalosis due to primary hypocapnia was found to reduce tissue oxygen distribution clinically significantly by causing a leftward shift in oxygen-hemoglobin dissociation curve and impair tissue oxygenation [16]. Therefore, hypocapnia and respiratory alkalosis caused by it have to be diagnosed and intervened early. At the same time close monitoring of patients with repeated blood gas analysis should be done.

Arterial blood gas analysis is the gold standard to evaluate acid-base status of patients. However, arterial puncture can cause thrombosis, dissection, and local hematoma. Arterial puncture is also very painful, technical implementation is very difficult especially in the elderly and children and some experimentation may be necessary. However, venous blood gas sampling is easier and less painful. Also there is no risk of arterial hemorrhage, thrombosis, and dissection. Therefore, many studies investigating the usage of venous blood gases instead of arterial blood gas analysis have been conducted in metabolic and respiratory cases. Significant correlation was found between pH (r=0.969, r=0.979) and HCO₃ (r=0.954, r=0.995) values of hemodynamically stable diabetic ketoacidosis and uremic acidosis patients without respiratory failure [10,11,21]. Recently, arterial and venous blood gas samples were compared in patients with hypercapnia and respiratory acidosis due to chronic obstructive pulmonary disease (COPD). In some of these studies, correlation was found between levels of pH (r=0.934, r=0.828, r=0.826), Arterial pCO₂ (r=0.908, r=0.877, r=0.838) and HCO₃ (r=0.927, r=0.896) (p<0.001) [3,11,12,15]. In another study performed in patients with respiratory acidosis, correlation values were found as p<0.001 between arterial and venous blood gas samples regarding pH (r=0.864), pCO₂ (r=0.761), HCO₃ (r=0.749) and also including PaO₂ (r=0.702) [22]. In patients undergoing mechanical ventilation for acute respiratory failure in Intensive care unit, venous pH, pCO₂ and HCO₃ values were found to be able to be used instead of arterial blood gas samples [23].

A significant correlation was found between pCO₂ (p<0.001, r=0.764) and pH (p=0.002, r=0.836) levels of arterial and venous blood gas samples in this prospective study which is conducted to facilitate close follow-up of pCO₂ and pH values in patients with respiratory alkalosis. Also significant correlation was found between HCO₃ (p<0.001 r=0.843) and BE (p<0.001, r=0.883) values of these patients (Table 3).

In studies of patients with respiratory acidosis, no significant correlation was found between PaO₂ (r=0.252, r=0.599) and SO₂ (r=0.296, r=0.312) [3,4]. In another study of patients with respiratory acidosis, a significant correlation was found between PaO₂ (r=0.702, p<0.001) values [22]. In our study, pO₂ results were divided into three levels in order to evaluate the correlation between pO₂ levels of arterial and venous blood gas samples. No statistically significant correlation was found in groups of PaO₂>80mmHg and pO₂ between 60 and 80 mmHg. However, a statistically significant correlation was found in patients with PaO₂<60mmHg or moderate and severe hypoxia (r=0.540, p<0.001). Oxygen saturation value of these patients measured by pulse oximeter was calculated as 82 ± 11 (min=35 max=90) (Table 2). Only determination of the statistically correlation in patients with moderate and severe hypoxia could be due to inadequate compensatory mechanisms of the body under these values.

In this study, the venous measurements of pH, pCO₂, pO₂ and bicarbonate were highly correlated with their corresponding arterial measurements. Bland Altman plots demonstrated a high degree of agreement between the two corresponding sets of measurements with clinically acceptable differences. Accordingly, Arterial pH = Venous pH x 0.83 + 0.14, Arterial pCO₂ = Venous pCO₂ x 0.68 + 0.39, Arterial HCO₃ = Venous HCO₃ x 0.09 + 1.06, PaO₂ = Venous pO₂ x 0.39 + 1.13 (for patients PaO₂<60mmHg and O₂ saturation <90%) (Table 4).

As a result, respiratory alkalosis is an acid-base disorder that should be monitored closely. For follow-up of these patients, pH, pCO₂ and HCO₃ values of venous blood gas samples can be used instead of arterial blood gas samples if O₂ saturation is ≥ 90%. If O₂ saturation is <90%, pH, pCO₂ and HCO₃ together with pO₂ values of venous blood gas can be used instead of arterial blood gas samples.

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