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Comparative study between arterial blood gases and venous blood gases in Acute Cardiogenic Pulmonary Edema

Sadek Gamal-Aldin Sadek[1,*], mustafa Mohamed Nawar[2], Salah Mohamed EL-Tahan[2] and mohamed ibrahim lotfy[2]

[1]Department Emergency, Faculty of Medicine, Alexandria University, Egypt
[2]Department Cardiology and Angiology, Alexandria University, Egypt

E-mail: *mrsadek.alenani@gmail.com*

ABSTRACT

Acute cardiogenic pulmonary oedema (ACPE) is a critical situation with simultaneous respiratory & circulatory failure. Measuring arterial blood gases at presentation help to assess the severity of the process, giving information about the degree of tissue hypoperfusion (PH, HCO_3, base excess) and respiratory failure (PCO_2, PO_2, SaO_2) which may establish indications for specific therapies, like inotropic support or mechanical ventilation. Arterial blood gases ABG is essential in management of critically ill patients. However it requires invasive and sometimes painful techniques that are not risk free for patients or hospital staff. Alternatively venous blood gases have shown a good correlation with arterial blood gases in many clinical situations and specifically in diabetic ketoacidosis, exacerbation of chronic respiratory failure, uremic acidosis, or multiple trauma. Furthermore, in some clinical scenarios with severe circulatory failure like hemorhagic shock or while performing cardiopulmonary resuscitation, venous values have shown to reflect perfusions more accurately than arterial ones. The value of peripheral blood gases in patients with acute pulmonary oedema has not been well studied and this our scope in this issue.

Key words : Emergency, acute pulmonary edema, blood gases, oxygen saturation.

INTRODUCTION

Pulmonary edema refers to leakage and extravasation of fluid from pulmonary vasculature in the interstitium and alveoli of the lungs (Dargie and McMurray, 1994). Nieminen et al.,(2005) divided pulmonary edema to two fundamentally different types occur in humans; cardiogenic pulmonary edema “also termed hydrostatic or heamodynamic edema” and non-cardiogenic pulmonary edema “also known as acute lung injury, acute respiratory distress syndrome or increased permeability pulmonary edema”. Although they have distinct causes, cardiogenic and non-cardiogenic pulmonary edema may be difficult to distinguish because of their similar clinical manifestations. Pulmonary capillary blood and alveolar gases are separated by alveolar-capillary membrane (endothelial barrier), which consist of the three anatomically different layers: the capillary endothelium, the alveolar endothelium and the interstitial space in-between. Yancy et al.,(2013) stated that the fluid balance and exchange normally occur between the vascular bed and interstitium and is governed by the Frank- Starling law. According to, the main forces acting on the barrier between the intravascular and the extravascular spaces are the hydrostatic pressures and the protein oncotic pressure. Hydrostatic pressure pushes fluid out of the space while the oncotic pressure has the opposite

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effect keeping fluid in the space. Dargie and McMurray (1994) mentioned that in the normal lungs, fluid moves continuously outward from the vascular to the interstitial space according to the net difference between hydrostatic and oncotic pressure, as well to the permeability of the capillary membrane. Acute cardiogenic pulmonary edema (ACPE) is the result of an imbalance in fluid resorption and accumulation in the pulmonary interstitium and alveolar space that can be described by a sequence of three stages, progressing from pulmonary venous enlargement to interstitial edema to alveolar edema.

Swedberg et al., (2005) supported that ACPE is a critical situation with simultaneous respiratory & circulatory failure. Measuring arterial blood gases at presentation help to assess the severity of the process, giving information about the degree of tissue hypoperfusion (pH, HCO_3, base excess) and respiratory failure (PCO_2, PO_2, SaO_2) which may establish indications for specific therapies, like inotropic support or mechanical ventilation.

Arterial blood gases ABG is essential in management of critically ill patients. However it requires invasive and sometimes painful techniques that are not risk free for patients or hospital staff (Ak et al., (2006). Alternatively venous blood gases have shown a good correlation with arterial blood gases in many clinical situations and specifically in diabetic ketoacidosis and exacerbation of chronic respiratory failure (Paine et al., 1961). Furthermore, in some clinical scenarios with severe circulatory failure like hemorhagic shock or while performing cardiopulmonary resuscitation, venous values have shown to reflect perfusions more accurately than arterial ones. The value of peripheral blood gases in patients with acute pulmonary oedema has not been well studied and this our scope in this issue.

MATERIAL AND METHODS

The study is an observational prospective cohort study. The study enrolled 20 patients with acute cardiogenic pulmonary oedema who were admitted to Emergency Department of the main University Hospital of Alexandria and Nasser Institute Hospital between the period from December 2014 to June 2015. The study was approved by the Ethics Committee of the hospital, and informed consent was obtained from all the patient The patient must full fill the inclusion criteria which was: dyspnoea of sudden onset, signs of pulmonary oedema upon physical examination, congestionon chest radiograph and hypoxaemia, The study excluded patients with Immediate tracheal intubation at presentation Cardiogenic shock (systolic pressure<90), Severe chronic obstructive pulmonary disease, Chronic renal failure (serum creatinine>3.5 mg/dl), and Pneumonia. Patients received initial standard medical treatment with intravenous morphine (4 mg), furosemide (40 mg), and nitroglycerin and oxygen therapy either conventional (Ventimask) or up to 4 hours of noninvasive ventilation (NIV) with pressure support (average 15.2±2.4 cm^3H_2O) and positive end expiratory pressure (5 cm^3 H_2O). The initial FiO_2 was 0.5, which was increased, according to the response, up to 1 in patients treated with NIV or to 0.85−0.90 with a Monaghan reservoir mask in those assigned to conventional treatment. In addition to a peripheral venous line, arterial catheter was inserted for blood pressure monitoring and blood extractions. Arterial and peripheral venous samples were taken simultaneously during the first hours: on admission (0 minutes) and at 1, 2, 4, and 6 hours. Measurement of pH, PCO_2, bicarbonate, and oxygen saturation by co-oximetry (SaO_2 or SvO_2) was made for each sample, using an Instrumentation Laboratory Blood Gas Analyzer IL1620, which was located in the emergency laboratory. Blood samples were taken through a heparinized syringe and were carried from the ICU to the laboratory; pH, PCO_2, and PO_2 were directly measured, whereas actual plasma bicarbonate (HCO_3^-), SaO_2, and other additional parameters were derived mathematically. At the same intervals that blood samples were taken, simultaneous measurement of oxygen saturation by pulse oximetry (SpO_2) was obtained by with standard finger sensors. Because intubation was an end point of the study, measurements taken after intubation were not registered. The number of patients was enough for the purpose of the study. Data was analysed using the Pearson correlation coefficient. Receiver operating characteristic (ROC) curves were plotted to set the best value that identified acidosis (arterial pH<7.35), either metabolic (arterial bicarbonate<21 mmHg) or respiratory (defined by hypercapnia: arterial PCO_2>45 mmHg), as well as alkalosis (arterial pH >7.45) and hypocapnia (arterial pCO_2<36 mmHg).The limit range for these definitions was taken from our institutional laboratory. In order to establish a simple empiric formula to be used in clinical from venous samples, adding a coefficient to each of the venous values. These coefficients were calculated by rounding the mean of the differences between arterial and venous values to the nearest exact decimal value. The agreement of these 'empirically calculated arterial values' and the two measurements of oxygen saturation were tested using the bias plot (Bland−Altman) method. General data was analysed using IBM SPSS Statistics version 19, whereas the Bland−Altman test was analysed with MedCalc trial software (Mariakerke, Belgium).

RESULTS

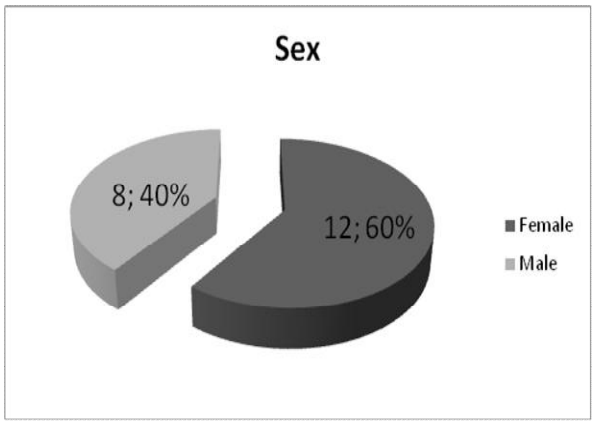
The study enrolled 20 patients with acute cardiogenic pulmonary oedema who were admitted to Emergency Department of the main University Hospital of Alexandria and Nasser Institute Hospital between the period from December 2014 to June

2015. A total of 100 pairs of samples were obtained. Average arterial and venous blood gases with their correlation coefficients, on admission and for the whole series, are shown in the following table and figures.

Patients Demographics and Co-morbid conditions

The study included 20 patients where females represented 60% and males represented 40% as shown in figure (1).

Figure-1. Patients Demographics



Data in Table 1 shows that most of the patients arrived to the hospital presented with severe systemic hypertension about 60% of patients.

Table-1. Clinical presentation of the patients

Clinical presentation	Frequency	Percentage %
ST elevation myocardiac infarction	4	20
Cardiomyopathy	2	10
Valve disease	2	10
Severe systemic hypertension	12	60

Respiratory disease was the highest co-morbid condition with (50%) while chronic kidney disease was the second common co-morbid condition with (40%) and liver disease was the last co-morbid one with (20 %) as shown in figure (2)

About the risk factors: Hypertension was the highest one with nineteen (95%) patients. while diabetes mellitus was the second risk factor with sixteen (80%) patients mainly type II IDDM (75%). Coronary artery disease represented with eleven (55%) patients. Valve disease represented with eight (40%) patients, and the last risk factor was smoking where eight (40%) patients were smokers, as shown in figure (3).

Figure-2. Co-morbid conditions.

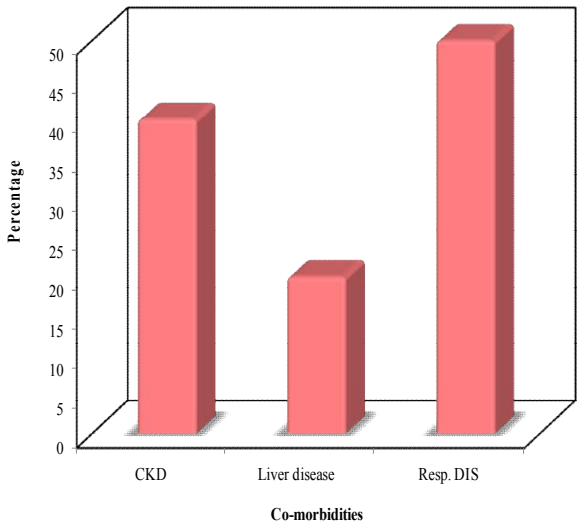
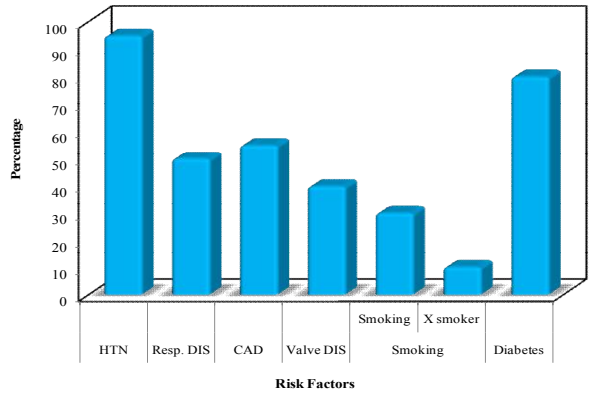


Figure-3. Risk factors.



A total of 100 pairs of samples were obtained. Average arterial and venous blood gases with their correlation co efficient, on admission, 1st hour, 2nd hour,4th hour and 6th hours after admission along the whole series for the pH, Bicarbonate(HCO_3), carbon dioxide(PCO_2) and oxygen saturations (PSO_2) were recorded, as shown in figures (4, 6 and 8). Oxygen saturation reached normal values in the first hour, while pH needed several hours to normalize.

On admission, the incidence of arterial acidosis was 30%, alkalosis 70%, hypercapnia 70%, and hypocapnia 30%. ROC curves were plotted to set the venous value that best identified arterial acidosis, either respiratory or metabolic. The range for these definitions was previously reported in the methods. When the whole series was considered (Figure 4), the cutoff (mean±SE) for acidosis was a venous pH of 7.31, with sensitivity 83.3%, specificity 100%, and test accuracy 95%, as shown in figure 5.

Figure-4. Comparison between arterial and venous gases according to pH

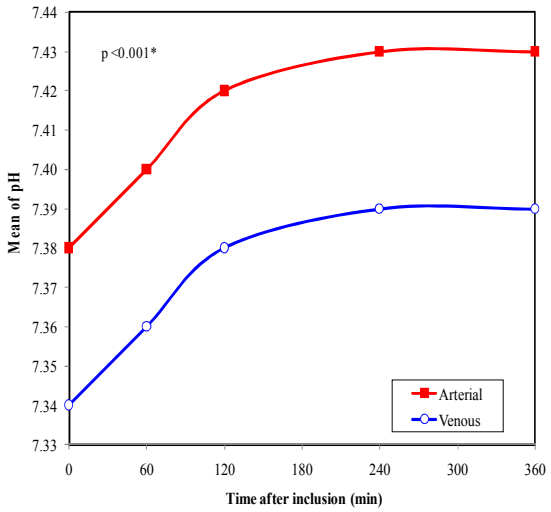


Figure-5. ROC curve of venous pH for identification of arterial acidosis (PH<7.35).

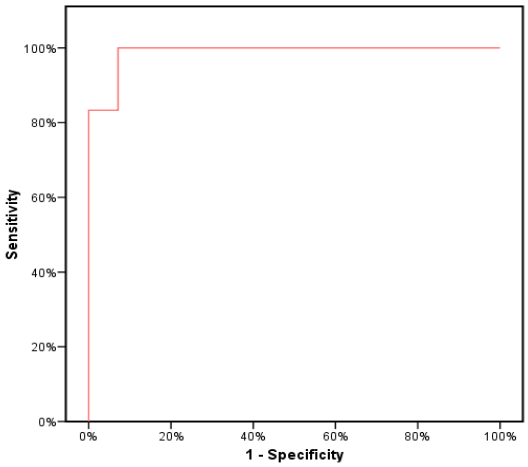


Figure-6. Comparison between arterial and venous gases according to PCO_2.

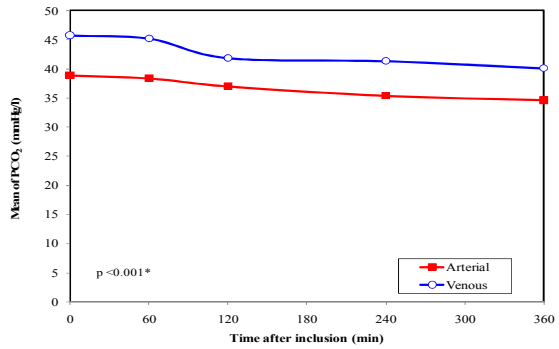


Figure-7. ROC curve of venous CO_2 for identification of arterial respiratory acidosis. ($PaCO_2$>46).

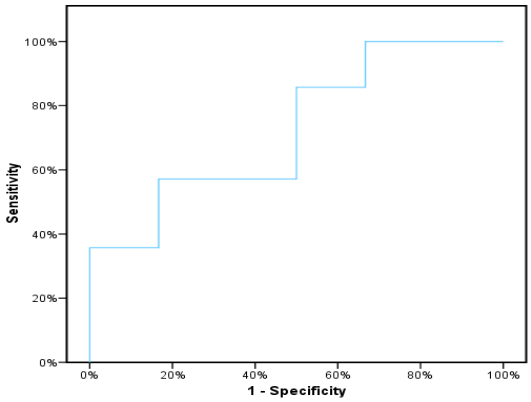
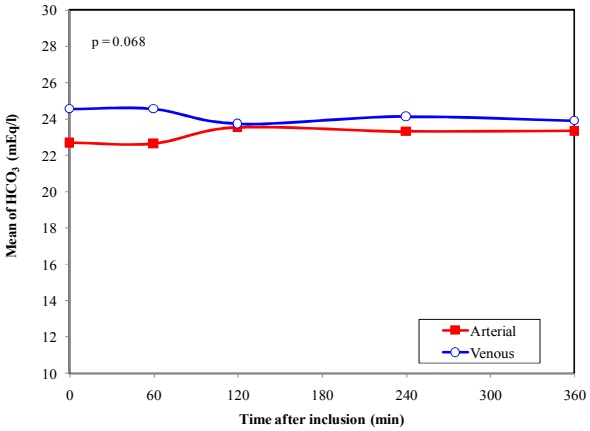


Figure-8. Comparison between arterial and venous blood gases according to HCO_3.



The cutoff for hypercapnia was a venous pCO_2 of 47.8±0.32 mmHg, with sensitivity 57.1%, specificity 83.33%, and test accuracy 65% as shown in figure (6 and 7).

The cutoff for metabolic acidosis was venous bicarbonate of 21±0.03mEq/l, with sensitivity 90%, specificity 80%, and test accuracy 85% as shown in figure (9).

The bias plot for agreement (Bland−Altman) before the empirical adjustement for venous and arterial blood gases showed a mean bias for pH of −0.04 (95% CI -0.03 to 0.12) , for CO_2 +6 mmHg (−17 to 5.1 mmHg), and for bicarbonate +1.1 mEq/l (-8 to 5.8 mEq/l). as shown in figures (10,11,12).

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Figure-9. ROC curve of venous bicarbonate for identification of arterial metabolic acidosis (atrerial bicarbonate <21 mEq/L).

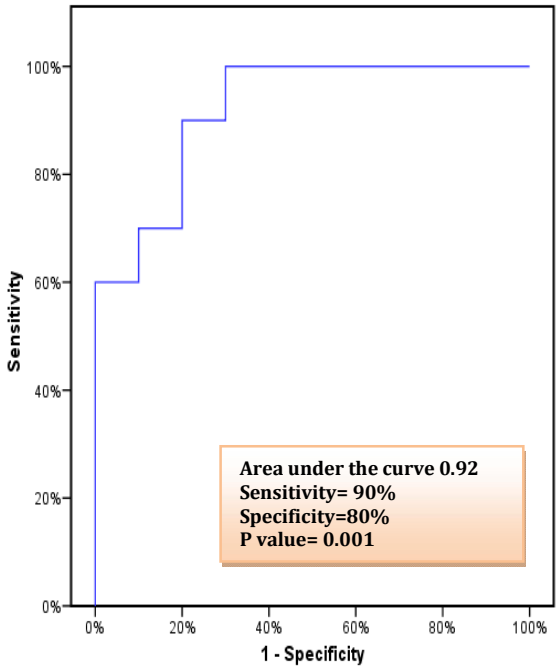


Figure-10. Bland-Altman Before the empirical adjustment Bias (Bland−Altman) plots for agreement of (A) venous and arterial pH(B) mean venous and arterial pH.
Mean difference 95% CI 0.04 0.03 to 0.12

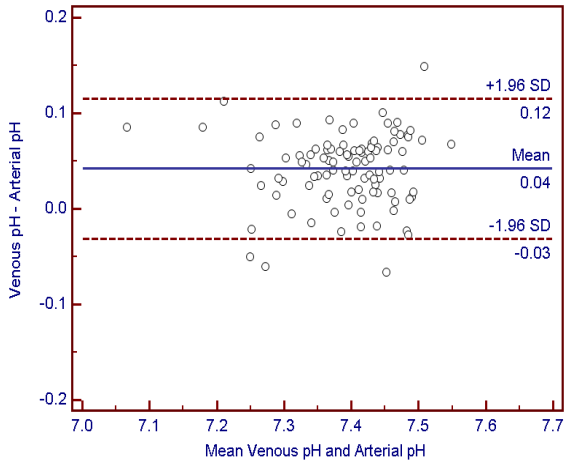


Figure-12. Bland-Altman Before the empirical adjustment Bias (Bland−Altman) plots for agreement of (A) venous and arterial pco2 (B) Mean venous and arterial pCO_2.
Mean difference 95% CI 6.0 -17.1 to 5.1

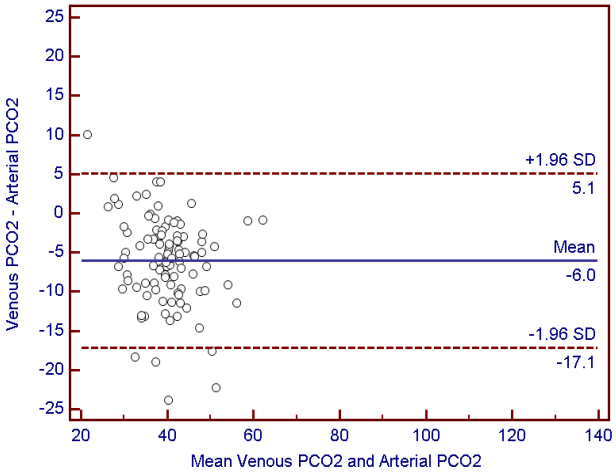


Figure-11. Bland –Altman Before the empirical adjustment Bias (Bland−Altman) plots for agreement of (A) venous and arterial bicarbonate
Mean difference 95% CI 1.1 -8.0 to 5.8

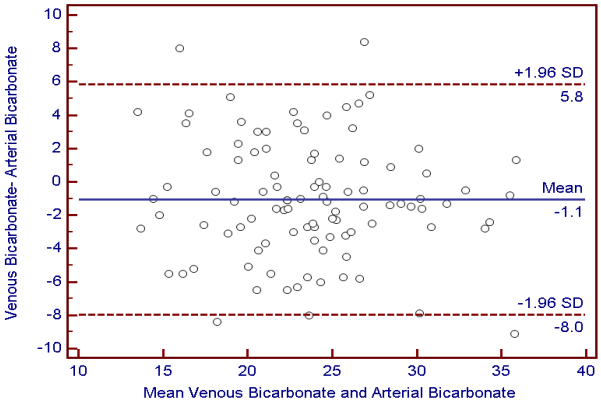
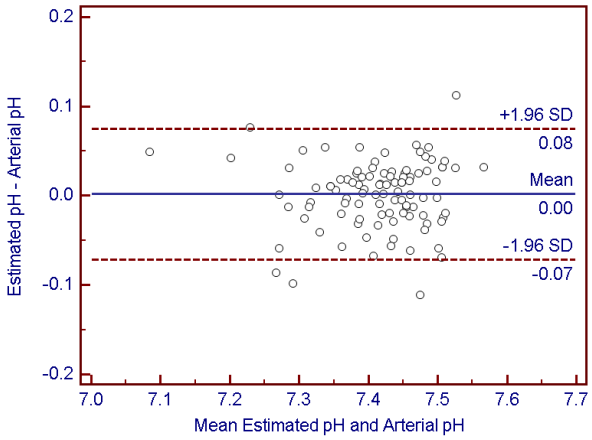


Figure-13. Bias (Bland−Altman) plots After this empirical adjustement for agreement of (A) estimated arterial and arterial pH (B) mean estimated arterial' and arterial pH.
Mean difference 95% CI 0.0 -0.07 to 0.08



As described in the methods, arterial blood gases were empirically estimated according to these differences, by adding 0.04 to venous pH, subtracting6 mmHg from venous CO_2, and subtracting 1.1 mEq/l from venous bicarbonate.

After this empirical adjustment, the Bland-Altman test showed greater agreement: mean bias difference for pH−0.002 (95% CI −0.07 to 0.08), for CO2 0.1 (−11.1 to 11.1), and for bicarbonate −0.01 (−6.9 to 6.9), as shown in Figures (13,14,15).

Figure-14 Bias (Bland−Altman) plots After this empirical adjustment for agreement of (A) estimated, arterial pCO_2 (B) mean estimated , arterial pCO_2.
Mean difference 95% CI; 0.0 -11.1 to 11.1

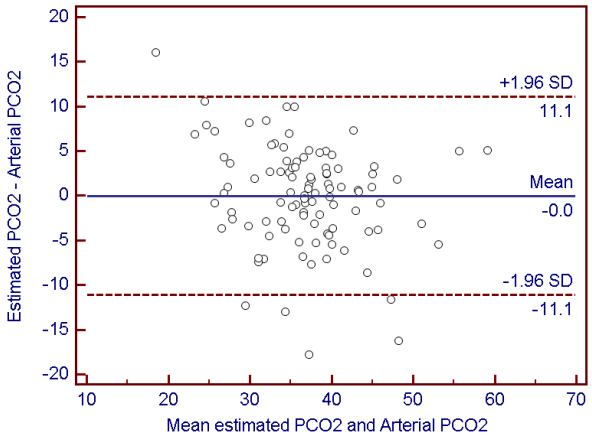
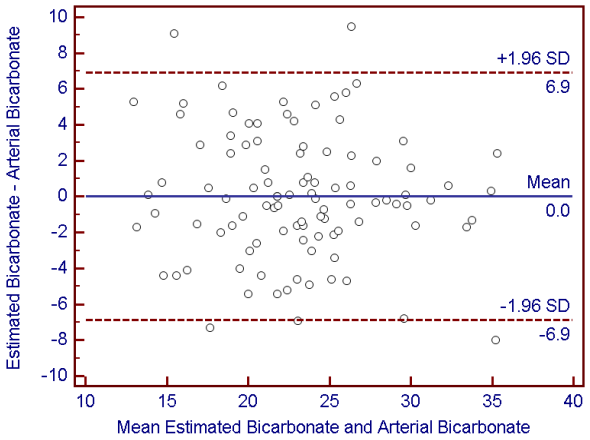
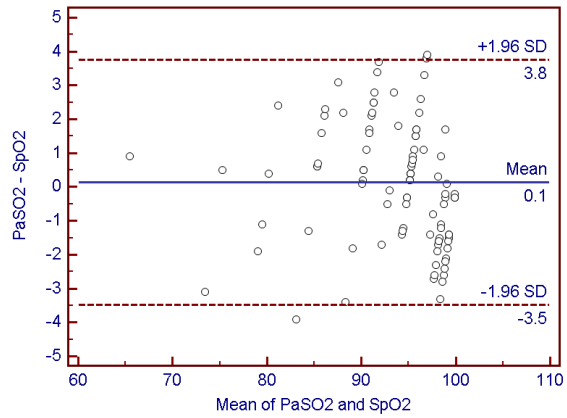


Figure-15. Bias (Bland-Altman) plots After this empirical adjustment for agreement of (A)estimated arterial' and arterial bicarbonate(B) mean estimated arterial' and arterial bicarbonate.
Mean difference 95% CI 0.0 -6.9 to 6.9



The Bland−Altman plot for SaO_2 and SpO_2 before the empirical adjustement is, showing a mean bias 0.1 (−3.5 to 3.8) as shown in figure (16)

Figure-16. Bland–Altman Before the empirical adjustment Bias (Bland−Altman) plots for agreement of Mean SaO_2 and SpO_2.
*SaO_2: oxygen saturation in arterial blood gases.
*SPO_2: oxygen saturation in pulse oximetry.
Mean difference 95% CI; 0.1 -3.5 to 3.8

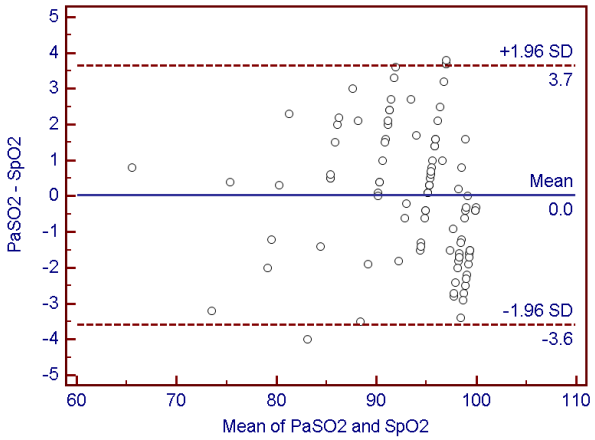


The Bland−Altman plot for SaO_2 and SpO_2 after the empirical adjustment is, showing a mean bias SaO_2 (95% CI-3.6 to 3.7) as shown in figure (17).
When applying this empirical adjustment in the identification of arterial hypercapnia on admission, venous blood samples only misclassified one patient (8%).

Compared to arterial gases, venous samples underestimated pH and overestimated pCO_2 and bicarbonate. Mean differences between venous and arterial were: pH −0.042, pCO_2 6 mmHg, and

bicarbonate 1.1 mEq/l. Correlation was lower for all parameters on admission, with the exception of bicarbonate and SpO_2 that remained substantially stable at all intervals. Oxygen saturation as well as arterial and venous blood gases showed a parallel course at all time points.

Figure-17. Bias (Bland-Altman) plots After this empirical adjustment for agreement of mean SaO_2 and SpO_2.
Mean difference 95% CI; 0.0 -3.6 to 3.7



DISCUSSION

Blood gas analysis has an important role in the assessment of patients with severe respiratory and metabolic disease, in particular for the accurate determination of pH (Treger et al., 2010). Arterial puncture is however often painful and carries the risk of complications such as local haematoma, infection and occlusion/embolisation of the artery with consequent ischaemic injury to the digits according to Walkey et al.,(2010) and Brandenburg and Dire (1998). Over the past several decades, a number of small studies have shown that pH can be accurately estimated from venous blood and "arterialised" venous blood. Gennis et al., (1985) reported that venous pH is almost identical clinical value to arterial pH. This is supported by a recent small study of patients with diabetic ketoacidosis that showed that venous blood could be substituted for arterial one in the assessment of acidosis (Brandenburg and Dire,1998).

Despite this evidence, arterial blood sampling remains the common method of determining acid-base status. There is few published studies that showed evidence regarding the accuracy of venous pH measurement in the population of emergency department (ED) patients requiring assessment of their acid-base status.

Our study aimed to determine the extent of correlation of arterial and venous pH with a view to identifying whether venous samples can be used as an alternative to arterial values in the clinical management of selected patients who presented with acute cardiogenic pulmonary edema in the ED with simultaneous recording of arterial oxygen saturation via pulse oximetry.

In our study we enrolled 20 patients presented with acute pulmonary edema to our ED and we excluded Immediate tracheal intubation at presentation patients who had cardiogenic shock (systolic pressure<90), severe chronic obstructive pulmonary disease, chronic renal failure (serum creatinine >3.5mg/dl), pneumonia and acute myocardiac infarction receiving reperfusion therapy. Every patient who needed intubation and invasive mechanically ventilated was also ex-rolled.

Masip et al., (2012) used the same exclusion criteria for 34 patients presented with acute pulmonary edema withdrawing 34 pairs of arterial and venous blood gases for 5 intervals which was grossly more than our study where we studied 5 pairs per patient that accounts for 100 pair of samples (200 samples) that was collectively more than Masip total number of valid samples after exclusion of intubated patients (178 samples).

Middleton et al., (2006) studied in 2006 agreement between arterial and central venous values for pH, bicarbonate, base excess, and lactate over 110 patients but without strict population specifications and although he found adequate correlation between the previously mentioned parameters.

Evaluation of the same topic of correlation between arterial and venous blood gases was fatherly done over different population groups like diabetic ketoacidosis, pulmonary units, general ICU population (Gennis et al.,1985).

Regarding arterial and venous blood gases mean parameters: The general mean for arterial pH was (7.41±0.06) and the mean venous pH was (7.37±0.06), the general mean for arterial PCO_2 was (36.77±5.34) and the mean venous PCO_2 was (402.78±6.09) and the general mean for arterial HCO_3 was (23.1±4.4) and the mean venous HCO_3 was (24.16±4.68).

When opposing Masip et al., (2012) study as he included similar studied population, the previously mentioned means of parameters were pH 7.35±0.09 in ABGs 7.32±0.09 in VBGs, CO_2 (mmHg) 46.1±11 in ABGs, 51.2±13.2 in VBGs and Bicarbonate (mEq/l) 25.3±4.8 in ABGs , 26.3±4.7 in VBGs.

In our study venous samples underestimated pH (mean difference −0.04) and overestimated CO_2 (+6 mmHg) and bicarbonate (+1.1 mEq/l). Conversely, SpO2 tended to underestimate SaO2 (mean±SD: 12.8 -12.3 to 37.9). Applying simple mathematical formulae based on these differences, arterial values were empirically calculated from venous samples, showing acceptable agreement in the Bland−Altman test.

Masip et al., (2012) in concordance to our former data when compared venous samples to arterial gases, and found that the mean differences between venous and arterial were: pH −0.028, pCO_2 5.1 mmHg, and bicarbonate 1 mEq/l. Correlation was lower for all parameters on admission, with the exception of bicarbonate and SpO_2 that remained substantially stable at all intervals.

The bias plot for agreement (Bland−Altman) for venous and arterial blood gases showed a mean bias for pH of −0.028 (95% CI −0.042 to 0.099), for CO_2 +5.1 mmHg (−18.4 to 8.3 mmHg), and for bicarbonate +1 mEq/l (−5.3 to 5.4 mEq/l). As described in the methods, arterial blood gases were empirically estimated according to these differences, by adding 0.03 to venous pH, subtracting 5 mmHg from venous CO_2, and subtracting 1 mEq/l from venous bicarbonate. After this empirical adjustment, the Bland-Altman test showed greater agreement in their study (Masip et al., 2012) .

On the other hand in our study there was significant cut-off value of acidosis which was PH 7.31 with sensitivity 83.8% and specificity 100% and test accuracy 95% (area under the curve of 1 and p value 0.0001). It showed a mean bias for agreement with Bland−Altman test for PH of- 0.04 (95% CI-0.03 to 0.12), Arterial blood gases were empirically estimated according to these differences, by adding 0.04 to venous PH.

There was also a significant cut off value of hypercapnea for a venous PCO_2 47.8 at which was on admission of 46% showed a sensitivity 57.1% and specificity 83.33% and test accuracy 65% (area under the curve of 0.72 with p value 0.117). It showed a mean bias for pco2 of 6 (95% CI-17.1 to 5.1), Arterial blood gases were empirically estimated according to these differences, by subtracting 6 from venous PCO_2. When applying this empirical adjustment in the identification of arterial hypercapnia on admission, venous blood samples only misclassified one patient (5%).

Lastly, the cut-off value of metabolic acidosis was venous bicarbonate of 21 mEq/l with sensitivity 90%, specificity 80%, and test accuracy 85%. (with area under the curve of 0.92 with p value 0.001). It showed a mean bias for HCO_3 of 1.1 (95% CI-8.0 to 5.8), Arterial blood gases were empirically estimated according to these differences, by subtracting 1.1 from venous HCO_3.

To our knowledge our study is the 2nd study that assesses the correlation between serial arterial and venous blood gases in the setting of acute cardiogenic pulmonary oedema, providing detailed information about the evolution of these parameters throughout the first hours. Masip et al., (2012) study in 2012 was the 1st one and previous studies have analysed the correlation in other clinical scenarios as Toftegaard et al., (2009) mentioned.

Lastly we can say However, since a venous line is generally inserted for blood analysis and intravenous treatment in all patients admitted for pulmonary oedema, the matter of course inclusion of venous blood gas testing would be worthwhile, and the results of this study support the use of venous samples, together with pulse-oximetry, as a useful approach to arterial blood gases in these patients.

CONCLUSION

In patients with cardiogenic pulmonary oedema, arterial blood gas disturbances may be estimated from peripheral venous samples. By monitoring SpO_2 simultaneously, arterial punctures could often be avoided. Venous blood gas samples can replace the arterial blood gas punctures in cases of acute cardiogenic pulmonary edema associated with monitoring SpO_2 simultaneously by pulse oximetry. Venous blood gas samples can't be used in cardiogenic shocked patients (systolic blood pressure <90), patients with acute myocardial infarction receiving reperfusion therapy, severe chronic obstructive pulmonary disease or chronic renal failure patients

Conflict of Interests:

Authors declare that there is no conflict of interests regarding the publication of this paper.

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