

Comparative Study between Usage of Arterial Blood Gases versus Calculated Pulmonary Contusion Volume in Estimating Prognosis and Potential Complications of Pulmonary Contusion after Blunt Chest Trauma

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ABSTRACT

Background: Pulmonary contusion (PC) is a frequent and potentially fatal complication associated with blunt thoracic trauma.

Aim of the work: The goal of this research was to evaluate a comparative analysis of the prognostic value of ABG parameters versus CT-calculated PC volume in predicting complications (pneumonia & ARDS) following blunt chest trauma.

Patients and Methods: A prospective comparative study was executed on 50 participants with blunt chest trauma. All participants underwent ABG analysis and chest CT to assess PC volume.

Results: As regard ABG in lung contusion cases, PH, PCO₂ cannot predict pneumonia but can significantly predict ARDS. Hco₃ cannot predict pneumonia nor ARDS. Spo₂, PaO₂, P/F, Aa DO₂, Aa DO₂ augmentation, PaO₂ deficit, a/A O₂ tension ratio can significantly predict pneumonia and ARDS. Both ABG abnormalities and increased contusion volume were associated with higher complication rates. However, CT-based contusion volume demonstrated superior predictive accuracy, particularly for pneumonia and ARDS. Specific cutoff values (>0.19 for pneumonia and >0.429 for ARDS) showed high sensitivity, specificity, and negative predictive value, outperforming individual ABG-derived indices.

Conclusions: While both ABG analysis and CT imaging provide important prognostic information, CT-calculated PC volume proved to be a more reliable predictor of complications following blunt chest trauma.

Keywords: Arterial Blood Gases, Calculated Pulmonary Contusion Volume, Prognosis, Blunt Chest Trauma.

INTRODUCTION

Chest trauma continues to represent a significant clinical challenge and is generally categorized into penetrating and blunt Thoracic trauma that has a mortality of 25%. Blunt chest trauma is commonly attributable to various mechanisms. First is direct blow leading to rib fracture with underlying lung injury. Second is chest compression between bony structures. Third is sudden deceleration as in falling from height accidents. Fourth is blast injury ^[1].

These mechanisms disrupt the alveolar capillaries, resulting in the accumulation of blood and other bodily fluids within the lung tissue, which impairs gas exchange and consequently induces hypoxia. Additionally, this process is associated with the initiation of inflammatory response mediated by the innate immune system following trauma ^[2].

Blunt chest trauma can lead to pulmonary contusion (PC), pneumothorax or hemothorax. PCs are the most common one and have various clinical types ^[3].

The diagnosis of PC is based on several entities. Clinically, the patient shows respiratory distress with rapid shallow breathing. Radiologically, PC was shown as peripheral, non-anatomical ground glass, nodular opacities or consolidation. Laboratory, arterial blood gases (ABG) showed hypoxemia and hypercapnia because PC causes an increase in shunting areas in the lung leading to ventilation perfusion mismatch ^[4].

Routine ABG analysis is recommended for the assessment of base deficit and metabolic acidosis in

trauma patients who, according to initial history and physical examination, are assessed to be at risk for severe injury ^[5].

Computed tomography (CT) represents the preferred diagnostic modality for detecting and quantifying pleural cavity volume in patients with chest trauma. Previous research has highlighted the prognostic value of a novel CT-based volume index in poly-trauma individuals with PC. Precise assessment of the contusion volume index is instrumental in predicting individuals at elevated risk of adverse events as pneumonia, respiratory failure, and acute respiratory distress syndrome (ARDS), conditions that often necessitate mechanical ventilation ^[6].

These findings underscore the clinical significance of contusion volume for identifying patients at increased risk of clinical deterioration.

Accordingly, our study aimed to relate the utility of ABG analysis with that of calculated PC volume in predicting prognosis and potential complications, specifically pneumonia and acute ARDS, following blunt chest trauma.

PATIENTS AND METHODS

This prospective comparative clinical study was executed on 50 individuals aged above 18 years old, both sexes, who were presented with lung contusion resulting from blunt chest trauma and patients with lung contusion with hemothorax, pneumothorax or hemopneumothorax after lung expansion with chest

tube. The study was executed from November 2023 to May 2025.

Ethical approval:

The study was authorized from the Ethical Committee of Tanta University Hospitals, Tanta, Egypt (approval code: 36264MS403/11/23). Informed documented consent was gathered from the participants, adhering to the Helsinki University declaration guidelines.

Exclusion criteria: included patients who had lung contusion due to penetrating chest trauma, patients requiring mechanical ventilation for other causes (e.g. low Glasgow coma scale < 8), patients who required emergency surgery, previous lung pathological condition (pneumonia, tuberculosis, bronchiectasis or chronic obstructive pulmonary disease) or previous lung resection surgeries, hemorrhagic shock and fractured dislocated ribs penetrating the lung parenchyma.

All patients were underwent to complete history taking, laboratory tests [ABG ^[7]] and radiological investigations [Chest X-ray (CXR) and CT chest].

• ABG in room air, PO₂, PCO₂, Oxygen saturation and combined ABG.

Combined ABG calculations:

Alveolar-arterial O₂ gradient (AaDO₂) and its augmentation. The calculation of AaDO₂ was equal to the following: $AaDO_2 = 150 - 1.25PaCO_2 - PaO_2$. In our study, AaDO₂ augmentation calculation was executed along with the corresponding equation: $AaDO_2 \text{ augmentation} = AaDO_2 - \text{estimated normal gradient (ENG)}$. The ENG was determined via the following equation $(Age/4) + 4$. The P/F is the ratio between the PaO₂ and the fraction of oxygen determined in the inspiration phase. The determination of the estimated normal PaO₂ was performed by the following equation: $100 - Age/3$. In addition, The PaO₂ Deficit = estimated normal PaO₂ - ABG analysis of PaO₂. Additionally, The a A O₂ tension ratio refers to the ratio between the O₂ partial pressure of oxygen in arterial blood and alveolar PO₂, normally it ranges between 0.75 and ^[8].

• PC volume was calculated on CT chest as follows:

Total contusion volume/ total lung volume × 100 = %

$a \times b \times c / A \times B \times C \times 100 = \%$

Where: a was the maximum diameter of the contusion tissue perpendicular to the ground on the coronal plane. b denoted the maximum vertical dimension of the contusion area in the axial view.

c expressed the maximum horizontal dimension of the contusion area in the axial view.

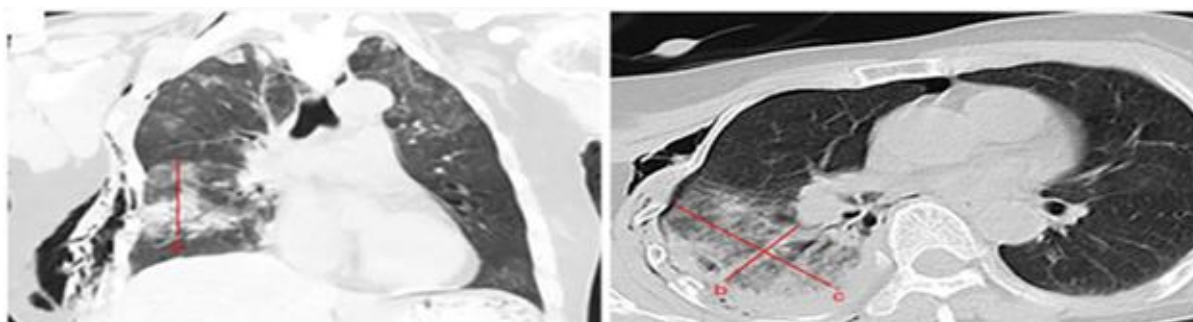


Figure 1 Measurement of PC volume in CT chest.

A: was defined as the length measured in the coronal plane from the superior aspect of the thorax at the level of the carina and the highest point of the ipsilateral diaphragmatic dome.

B: was defined as the vertical length from the posterior and anterior chest walls at the level of the carina on the axial cross-section.

C: was defined as the horizontal length from the lateral tracheal wall and the contralateral chest wall at the level of the carina on the axial cross-section.

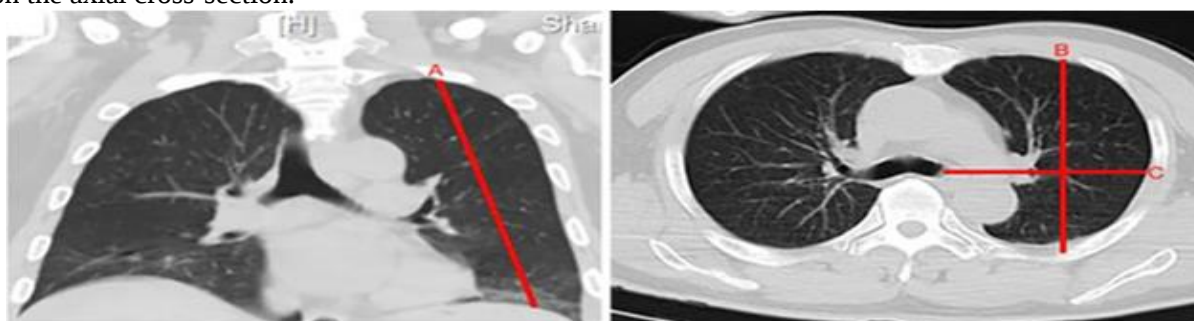


Figure 2 Measurement of lung volume in CT chest.

Pneumothorax was considered recoverable lung volume, not contused tissue. For patients with multiple contusion foci, each was separately calculated and summed using Yang's index ^[9].

Data collected after admission:

Follow-up using CXR and ABG after 48 hours and 5 days. CT chest after 5 days. Duration of mechanical ventilation (in hours), if present. Length of ICU stay (in days), if present. Detection of any potential complications (pneumonia, respiratory failure).

Pneumonia was diagnosed by the following ^[10]: Clinically: exacerbation of profound symptoms with cough, expectoration or fever $\geq 38^{\circ}\text{C}$. Laboratory: elevated C-reactive protein (CRP), total leucocyte count (TLC) or positive sputum culture. Radiologically: development of new homogeneous opacity on top of lung contusion in CT chest.

Respiratory failure (adult - ARDS) was diagnosed by the following ^[11]: Clinically: exacerbation of profound respiratory symptoms. Laboratory: ABG (P/F

ratio ≤ 300 or $\text{PO}_2 \leq 60$ mmHg). Radiologically: bilateral or unilateral pulmonary infiltrates seen on CXR.

Statistical analysis

It was performed by SPSS v26 (IBM Inc., Chicago, IL, USA). Shapiro Wilks test and histograms were applied to assess the normality of the distribution of data. Quantitative parametric data were shown as mean and standard deviation (SD). Quantitative non-parametric data were shown as median and inter-quartile range (IQR). Qualitative variables were shown as frequency and percentage. A two tailed P-value < 0.05 was significant.

RESULTS

Demographic data, trauma, clinical & laboratory data and complications were presented in **Table 1**.

Table 1: Demographic data, trauma, clinical & laboratory data and complications of the studied patients

N=50		
Age (years)		31.4 $\pm$ 15.86
Sex	Male	45 (90%)
	Female	5 (10%)
Weight (Kg)		81.6 $\pm$ 15.35
Height (cm)		174.9 $\pm$ 8.52
BMI (Kg/m ²)		26.5 $\pm$ 3.88
Special habits	Smoker	19 (38%)
	Ex-smoker	5 (10%)
Comorbidities	Hypertension	6 (12%)
	Diabetes mellitus	3 (6%)
	Hepatic diseases	1 (2%)
	CKD	1 (2%)
Ejection Fraction		65.1 $\pm$ 4.55
Trauma		
Mode of trauma	RTA	37 (74%)
	FFH	9 (18%)
	Runover	2 (4%)
	Fight	2 (4%)
Time pass (h)		3.5 $\pm$ 2.57
Blood pressure (mmHg)	Systolic	111.2 $\pm$ 13.5
	Diastolic	70.6 $\pm$ 10.58
Clinical data		
Heart rate (bpm)		92.5 $\pm$ 11.35
Respiratory rate (breath/min)		21.9 $\pm$ 5.35
Temperature ($^{\circ}\text{C}$)		37.4 $\pm$ 0.66
Oxygen Saturation (%)		93 $\pm$ 4.71
GCS		14.6 $\pm$ 1.01
Laboratory investigations		
Hemoglobin (g/dL)		12.3 $\pm$ 2.28
Platelet ($10^9/\text{L}$)		220.1 $\pm$ 7.86
TLC (cells/ μL)		13.9 $\pm$ 3.15
CRP (mg/dL)		25.5(12-66)
Sputum Culture and Sensitivity	Positive	3 (6%)
	Negative	47(94%)
Complications	Pneumonia	9 (18%)
	ARDS	2 (4%)

Data are shown as mean $\pm$ SD or frequency (%). BMI: Body mass index, CKD: Chronic kidney disease, RTA: Road traffic accident, FFH: Fall from height, GCS: Glasgow coma scale, ARDS: Acute respiratory distress syndrome.

Radiological investigations, management of trauma, days of ICU and MV were presented in **Table 2**.

Table 2: Radiological investigations, management of trauma, days of ICU and MV of the studied patients

		N=50
CXR		
Hemothorax		13 (26%)
Right		6 (12%)
Left		7 (14%)
Pneumothorax		17 (34%)
Right		6 (12%)
Left		11 (22%)
Surgical emphysema		15 (30%)
Right		5 (10%)
Left		10 (20%)
CT chest		
Contusion volume (%)		16 ± 14.69
Hemothorax		18 (36%)
Right		6 (12%)
Left		12 (24%)
Pneumothorax		24 (48%)
Right		11 (22%)
Left		13 (26%)
Surgical emphysema		16 (32%)
Right		5 (10%)
Left		11 (22%)
3D (fracture)	Sternum	1 (2%)
	Right	11 (22%)
	Left	18 (36%)
Dexamethasone consumption		23 (46%)
Furosemide consumption		17 (34%)
Oxygen support		49 (98%)
Circulatory Support		2 (4%)
Balance	Euvolemic	26 (52%)
	Negative	24 (48%)
ICT need		19 (38%)
Right		8 (16%)
Left		11 (22%)
ICU stay (patients)		21 (42%)
ICU stay (days)		6(5 – 10)
Mechanical ventilation (patients)		7 (14%)
Mechanical ventilation (h)		50(30 – 411)

Data are shown as mean ± SD or frequency (%) or Median (IQR). CXR: Chest X-ray, CT: Computed tomography, ICT: Intercostal tube, ICU: Intensive care unit, MV: Mechanical ventilation.

PH, PCO₂ and HCO₃ were insignificantly different between day 0 and (after 2 and 5 days). PO₂, Spo₂ and P/F were insignificantly different between day 0 and after 2 days and were significantly higher after 5 days than in day 0 (P<0.05). Aa DO₂ and PaO₂ deficit were insignificantly different between day 0 and after 2 days and were significantly lower after 5 days than in day 0 (P<0.05). Aa DO₂ augmentation was significantly lower (after 2 and 5 days) than in day 0 (P <0.05). a/A O₂ tension ratio was significantly higher (after 2 and 5 days) than in day 0 (P <0.05) **Table 3**.

Table 3: ABG of the studied patients on room air

	Day 0	After 2 days	After 5 days
pH	7.4 ± 0.09	7.4 ± 0.1	7.5 ± 0.08
P-value		0.637	0.870
PCO ₂ (mmHg)	35 ± 7.63	35.5 ± 5.51	34.1 ± 5.21
P-value		0.697	0.382
PO ₂ (mmHg)	68.7 ± 18.4	74.4 ± 22.13	75.1 ± 12.23
P-value		0.103	0.008*
HCO ₃ (mmol/L)	24.2 ± 4.18	24.5 ± 5.04	25.6 ± 5.09
P-value		0.668	0.115
SpO ₂ (%)	91 ± 14.14	92.5 ± 11.21	95.1 ± 4.66
P-value		0.261	0.016*
P/F	322.7 ± 91.42	336.3 ± 77.93	357.7 ± 58.95
P-value		0.213	0.002*
Aa DO ₂ (mmHg)	41.5 ± 18.89	36.2 ± 15.14	33 ± 14.07
P-value		0.058	0.003*
Aa DO ₂ augmentation (mmHg)	29.7 ± 18.12	23.6 ± 13.83	21.2 ± 13.76
P-value		0.031*	0.003*
PaO ₂ deficit (mmHg)	21.7 ± 17.55	19.1 ± 15.65	14.5 ± 11.7
P-value		0.250	0.002*
a/A O ₂ tension ratio	0.6 ± 0.17	0.7 ± 0.15	0.7 ± 0.12
P-value		0.353*	0.01*

Data are shown as mean ± SD. * Significant P-value <0.05. PCO₂: Partial pressure of carbon dioxide, PO₂: Partial pressure of oxygen, HCO₃: Bicarbonate, SpO₂: Oxygen saturation P/F: PO₂/FiO₂, Aa DO₂: Alveolar-arterial oxygen difference, a/A O₂: tension ratio: arterial/Alveolar oxygen tension ratio.

Complications were insignificantly different between patients who received dexamethasone and furosemide and patients who did not received both drugs. Complications were insignificantly different between euvoletic balanced and negative balanced patients. Complications were significantly higher in positive sputum culture and sensitivity than negative ones (P<0.05) **Table 4.**

Table 4: Relation between complication and (dexamethasone and furosemide), and (balance and sputum culture and sensitivity) of the studied patients

Dexamethasone consumption			
	Yes (n=23)	No (n=27)	P-value
Pneumonia	4 (17.39%)	5 (18.52%)	0.959
ARDS	2 (8.7%)	0 (0%)	0.207
Furosemide consumption			
	Yes (n=17)	No (n=33)	
Pneumonia	2 (11.76%)	7 (21.21%)	0.699
ARDS	1 (5.88%)	1 (3.03%)	1
Balance			
	Euvoletic (n=26)	Negative (n=24)	
Pneumonia	2 (7.69%)	7 (29.17%)	0.069
ARDS	0 (0%)	2 (8.33%)	0.225
Sputum Culture and Sensitivity			
	Positive (n=3)	Negative (n=47)	
Pneumonia	3 (100%)	6 (12.77%)	0.004*
ARDS	2 (66.67%)	0 (0%)	0.002*

Data are shown as Frequency (%). * Significant P-value <0.05. ARDS: Acute respiratory distress syndrome.

PH was insignificantly different in day 0 and after 5 days between pneumonia and non-pneumonia groups and was significantly elevated after 2 days in non-pneumonia group than pneumonia group ($P=0.006$). PCO_2 was insignificantly different in day 0 and after 5 days between pneumonia and non-pneumonia groups and was significantly lower after 2 days in non-pneumonia group than pneumonia group ($P=0.025$). PO_2 was insignificantly different after 2 days between pneumonia and non-pneumonia groups and was significantly lower in day 0 and after 5 days in non-pneumonia group than pneumonia group ($P<0.05$).

HCO_3 was insignificantly different in day 0 and (after 2 and 5 days) between pneumonia and non-pneumonia groups. SpO_2 and P/F were significantly higher in day 0 and (after 2 and 5 days) <<in non-pneumonia group than pneumonia group ($P<0.05$).

Aa DO_2 , Aa DO_2 augmentation and Pa O_2 deficit were significantly lower in day 0 and (after 2 and 5 days) in non-pneumonia group than pneumonia group ($P<0.05$). a/A O_2 tension ratio was significantly elevated in non-pneumonia group than pneumonia group ($P<0.05$) **Table 5**.

Table 5: Relation between Pneumonia and ABG of the studied patients

		Pneumonia (n=9)	Non pneumonia (n=41)	P-value
PH	Day 0	7.41±0.15	7.45±0.07	0.229
	After 2 days	7.36±0.14	7.46±0.08	0.006*
	After 5 days	7.49±0.09	7.47±0.08	0.524
PCO₂ (mmHg)	Day 0	38.91±9.6	34.17±6.98	0.092
	After 2 days	39.16±5.55	34.67±5.22	0.025*
	After 5 days	34.74±5.77	33.95±5.15	0.684
PO₂ (mmHg)	Day 0	53.32±26.86	72.02±14.35	0.005*
	After 2 days	63.66±25.43	76.71±20.95	0.110
	After 5 days	64.62±14.81	77.37±10.46	0.004*
HCO₃ (mmol/L)	Day 0	24.67±4.4	24.06±4.18	0.698
	After 2 days	22.89±6.1	24.84±4.79	0.298
	After 5 days	27.3±5.83	25.19±4.91	0.264
SpO₂ (%)	Day 0	75.74±27.23	94.4±5.7	<0.001*
	After 2 days	83.77±23.3	94.38±4.95	0.009*
	After 5 days	91.02±8.69	95.97±2.65	0.003*
P/F	Day 0	228.72±124.92	343.28±68.52	<0.001*
	After 2 days	274.38±93.06	349.93±68.23	0.007*
	After 5 days	306.78±72.57	368.9±49.91	0.003*
Aa DO₂ (mmHg)	Day 0	56.39±23.19	38.26±16.4	0.008*
	After 2 days	45.45±14.01	34.15±14.76	0.041*
	After 5 days	43.66±13.66	30.72±13.2	0.011*
Aa DO₂ augmentation (mmHg)	Day 0	44±20.32	26.53±16.22	0.007*
	After 2 days	33.06±13.4	21.55±13.19	0.022*
	After 5 days	31.27±11.88	18.98±13.25	0.014*
PaO₂ deficit (mmHg)	Day 0	40.44±22.79	17.65±13.32	<0.001*
	After 2 days	31.46±18.02	16.38±13.9	0.007*
	After 5 days	24.41±12.79	12.31±10.39	0.004*
a/A O₂ tension ratio	Day 0	0.5±0.23	0.67±0.13	0.003*
	After 2 days	0.56±0.17	0.68±0.13	0.017*
	After 5 days	0.6±0.14	0.72±0.11	0.006*

Data are shown as mean ± SD. * Significant P-value <0.05. PCO_2 : Partial pressure of carbon dioxide, PO_2 : Partial pressure of oxygen, HCO_3 : Bicarbonate, SpO_2 : Oxygen saturation P/F: PO_2/FiO_2 , Aa DO_2 : Alveolar-arterial oxygen difference, a/A O_2 tension ratio: arterial/Alveolar oxygen tension ratio.

PH was significantly higher in day 0 in non-ARDS group than ARDS group ($P<0.05$) and was insignificantly different after 2 and 5 days between ARDS and non- ARDS groups. PCO_2 was significantly lower in day 0 and after 2 days in non- ARDS group than ARDS group ($P<0.05$) and was insignificantly different after 5 days between ARDS and non- ARDS groups. PO_2 , SpO_2 , P/F and Pa O_2 deficit was significantly higher in day 0 and (after 2 and 5 days) in non- ARDS group than ARDS group ($P<0.05$). HCO_3 was insignificantly different in day 0 and (after 2 and 5 days) between ARDS and non- ARDS groups. Aa DO_2 , Aa DO_2 augmentation and Pa O_2 deficit were insignificantly different in day 0 between ARDS and non- ARDS groups and were significantly lower after 2 and 5 days in non- ARDS group than ARDS group ($P<0.05$). a/A O_2 tension ratio was significantly elevated in non-ARDS group than ARDS group ($P<0.05$), **Table 6**.

Table 6: Relation between ARDS and ABG of the studied patients

		ARDS (n=2)	Non-ARDS (n=48)	P-value
pH	Day 0	7.23±0.25	7.46±0.07	<0.001*
	After 2 days	7.33±0.21	7.44±0.09	0.091
	After 5 days	7.5±0.09	7.47±0.08	0.700
PCO ₂ (mmHg)	Day 0	51.5±13.58	34.34±6.69	0.001*
	After 2 days	44.4±7.64	35.11±5.18	0.018*
	After 5 days	38.7±0.42	33.9±5.23	0.206
PO ₂ (mmHg)	Day 0	26.3±17.39	70.42±16.34	<0.001*
	After 2 days	34.45±24.68	76.02±20.66	0.008*
	After 5 days	46.8±11.03	76.25±10.86	<0.001*
HCO ₃ (mmol/L)	Day 0	21.75±6.43	24.27±4.13	0.409
	After 2 days	23.85±7	24.51±5.04	0.858
	After 5 days	30.55±6.01	25.36±5.01	0.160
SpO ₂ (%)	Day 0	45.1±39.89	92.95±9.01	<0.001*
	After 2 days	55.75±46.32	94±5.06	<0.001*
	After 5 days	80.5±14.85	95.68±2.93	<0.001*
P/F	Day 0	125.5±82.73	330.87±82.73	0.001*
	After 2 days	164±117.38	343.51±68.72	<0.001*
	After 5 days	218.5±58.69	363.52±51.87	<0.001*
Aa DO ₂ (mmHg)	Day 0	61.61±2.81	40.69±18.82	0.126
	After 2 days	59.83±15.45	35.2±14.46	0.023*
	After 5 days	57.63±9.09	32.03±13.33	0.01*
Aa DO ₂ augmentation (mmHg)	Day 0	49.11±4.22	28.87±18.03	0.123
	After 2 days	47.33±14.04	22.63±13.05	0.012*
	After 5 days	45.13±7.67	20.19±13.06	0.011*
PaO ₂ deficit (mmHg)	Day 0	60.77±13.25	20.12±15.8	<0.001*
	After 2 days	54.22±22.8	17.63±13.77	<0.001*
	After 5 days	42.77±10.42	13.31±10.24	<0.001*
a/A O ₂ tension ratio	Day 0	0.29±0.13	0.65±0.15	0.001*
	After 2 days	0.35±0.22	0.67±0.13	0.001*
	After 5 days	0.45±0.11	0.71±0.11	0.002*

Data are shown as mean ± SD. * Significant P-value <0.05. PCO₂: Partial pressure of carbon dioxide, PO₂: Partial pressure of oxygen, HCO₃: Bicarbonate, SpO₂: Oxygen saturation P/F: PO₂/FiO₂, Aa DO₂: Alveolar-arterial oxygen difference, a/A O₂ tension ratio: arterial/Alveolar oxygen tension ratio.

Contusion volume can significantly predict pneumonia at cut-off >0.19 while PH cannot predict it. PCO₂ cannot predict pneumonia while Po₂ can significantly predict it at cut-off ≤68.8. Hco₃ cannot predict Pneumonia while Spo₂ can significantly predict it at cut-off ≤94.4. P/F can significantly predict pneumonia at cut-off ≤328 while Aa DO₂ can significantly predict it at cut-off >58.5. Aa DO₂ augmentation can significantly predict pneumonia at cut-off >24 while PaO₂ deficit can significantly predict it at cut-off >23. a/A O₂ tension ratio can significantly predict pneumonia at cut-off ≤0.66 (Table 7).

Table 7: Role of contusion volume and ABG in prediction of pneumonia among the studied patients

	Cut-off	Sensitivity	Specificity	PPV	NPV	AUC	P-value
Contusion volume	>0.193	%100	%85.37	%60	%100	0.943	<0.001*
PH	≤7.46	%55.56	%46.34	%18.5	%82.6	0.523	0.845
PCO₂	>32.9	%66.67	%46.34	%21.4	%86.4	0.660	0.114
Po₂	≤68.8	%66.67	%60.98	%27.3	%89.3	0.729	*0.043
Hco₃	>25.7	%66.67	%73.17	%35.3	%90.9	0.580	0.474
Spo₂	≤94.4	%66.67	%68.29	%31.6	%90.3	0.757	*0.011
P/F	≤328	%77.78	%60.98	%30.4	%92.6	0.786	*0.009
DO₂ Aa	>58.5	%66.67	%92.68	%66.7	%92.7	0.748	0.025*
Aa DO₂ Augmentation	>24	%77.78	%60.98	%30.4	%92.6	0.767	0.009*
PaO₂ deficit	>23.1	%77.78	%73.17	%38.9	%93.7	0.791	0.008*
a A O₂ tension ratio	≤0.66	%77.78	%60.98	%30.4	%92.6	0.745	0.019*

PPV: positive predictive value, NPV: negative predictive value, AUC: area under the curve.

Contusion volume can significantly predict ARDS at cut-off > 0.42 while PH can significantly predict it (P <0.001 and AUC = 0.875 at cut-off ≤7.41. PCO₂ can significantly predict ARDS at cut-off >41 while Po₂ can significantly predict it at cut-off ≤38.6. Hco₃ cannot significantly predict ARDS while Spo₂ can significantly predict it at cut-off ≤73.3. P/F can significantly predict ARDS at cut-off ≤184 while Aa DO₂ can significantly predict it at cut-off >58.5. Aa DO₂ augmentation can significantly predict ARDS at cut-off >43.7 while PaO₂ deficit can significantly predict it at cut-off >50.84 and a/A O₂ tension ratio can significantly predict it at cut-off ≤0.38 (**Table 8**).

Table 8: Role of contusion volume and ABG in prediction of ARDS among the studied patients

	Cut-off	Sensitivity	Specificity	PPV	NPV	AUC	P-value
Contusion volume	>0.429	%100	%97.92	%66.7	%100	0.990	<0.001*
PH	≤7.41	%100	%72.92	%8.3	%97.4	0.875	<0.001*
PCO₂	>41	%100	%87.50	%25	%100	0.938	<0.001*
Po₂	≤38.6	100%	%97.92	%50	%97.9	0.990	<0.001*
Hco₃	≤17.2	%50	%97.92	%50	%97.9	0.630	0.603
Spo₂	≤73.3	%100	%97.92	%66.7	%100	0.757	*<0.001
P/F	≤184	%100	%95.83	%50	%100	0.979	<0.001*
DO₂ Aa	>58.5	%100	%85.42	%22.2	%100	0.875	<0.001*
Aa DO₂ augmentation	>43.7	%100	%83.33	%20	%100	0.865	<0.001*
PaO₂ deficit	>50.84	%100	%93.75	%40	%100	0.969	<0.001*
a/A O₂ tension ratio	≤0.38	%100	%93.75	%40	%100	0.969	<0.001*

PPV: positive predictive value, NPV: negative predictive value, AUC: area under the curve.

DISCUSSION

Chest trauma is a significant leading contributor to morbidity, disability, and mortality on a global scale, representing a critical component of traumatic injuries [12]. In the current study among 50 patients, CXR hemothorax was observed in 13 (26%) participants; 6 (12%) participants on the right side and 7 (14%) participants on the left side. Similarly, Zeiler *et al.* [13] revealed that chest radiograph demonstrating a massive left-sided hemothorax secondary to blunt trauma Galal *et al* [14] sustained in a motor vehicle collision.

In the same context about CT findings, the author reported that chest wall damages, as rib and clavicle fractures or chest wall hematomas, were identified in 17 patients (34%) by CT. When analyzing the distribution of all thoracic injuries, including parenchymal, pleural, and mediastinal lesions, 21 patients (42%) had bilateral involvement, 17 (34%) had isolated right-sided injuries, and 12 (24%) had left-sided injuries.

In our study, PH, PCO₂, HCO₃ were insignificantly different between day 0 and (after 2 and 5 days). This matches typically Karmy-Jones and Jurkovich [15] that show early PC presentation, where oxygenation is impaired, but ventilation is relatively preserved unless severe.

This was supported by Gwely *et al.* [17] who found that HCO₃ was insignificantly different between short and long hospital stay. However, pH was significantly lower with longer hospital stay in chest trauma patients, while PCO₂ and Aa DO₂ were significantly higher with longer hospital stay. Aa DO₂ augmentation was

insignificantly different between short and long hospital stay.

In our study, PO₂, SPO₂ and P/F were insignificantly different between day 0 and after 2 days and were significantly higher after 5 days than in day 0. This pattern aligns with studies like: Mahmood *et al.* [16] who found P/F ratio inversely correlated with contusion volume. Easter [17] showed that oxygenation often worsens in the first 24–48 hrs and begins to improve by day 4–5.

In our study, Aa DO₂ and PaO₂ deficit were insignificantly different between in day 0 and after 2 days and were significantly lower after 5 days than in day 0. This tracks with Miller *et al.* [18] and Cohn *et al.* [19] who studied the natural course of contusion: peak hypoxia around 48 hrs, followed by gradual resolution over 5–7 days.

In our study, Aa DO₂ augmentation was significantly lower (after 2 and 5 days) than in day 0.

This reflects reduced worsening of oxygenation over time, possibly with treatment (e.g., oxygen, conservative ventilation), suggesting that intrapulmonary shunting decreased, consistent with healing contused tissue.

In our study, a/A O₂ tension ratio was significantly higher (after 2 and 5 days) than on day 0.

which indicates better oxygen transfer efficiency, that reflects improved alveolar-capillary diffusion and decreasing edema which was supported by previous reports, like: Radermacher *et al.* [20] who showed improving gas exchange with supportive care in PC over 4–6 days.

According to our findings, complications were pneumonia in 9 (18%) patients and ARDS in 2 (4%) patients. Higher incidence of ARDS was recorded by **Mahmood et al.**^[16] who noted that the ARDS was present in 12.4% of participants. This difference could be attributed to exclusion of patients who intubated on low GCS and patients who are in hemorrhagic shock in the present study that led to exclusion of a significant number of massive lung contusion cases.

In the present study, PH, PCO₂ cannot predict pneumonia but can significantly predict ARDS. Hco₃ cannot predict pneumonia nor ARDS. Lung contusion volume, SpO₂, PaO₂, P/F, Aa DO₂, Aa DO₂ augmentation, PaO₂ deficit, a/A O₂ tension ratio can significantly predict pneumonia and ARDS. Aligning with our study, **Hapsari et al.**^[21] highlighted that the P/F ratio can predict mortality in participants with pneumonia.

In the same context, **Catozzi et al.**^[22] reported that severe ARDS associated with low oxygenation. Similarly, **Su et al.**^[23] found that acute ARDS is defined by impairment of the alveolar–capillary membrane barrier, resulting in hypoxemia, hypercapnia, and pulmonary edema. In the same context, **Bienvenu et al.**^[24] claimed that PaO₂ correlated independently with ARDS-related mortality. This was consistent with **Ngan**,^[25] who showed that increased AaDO₂ may serve as a valuable parameter for identifying participants predisposed to developing ARDS. In line with our data, **Scala et al.**^[26] highlighted that AaDO₂ predicted the occurrence of severe pneumonia. Also, **Ruan et al.**^[27] stated that the P/F ratio serves a critical function in delineating ARDS. Supporting to our study, **Sayed et al.**^[28] found that the individuals with intense lung contusions had an elevated probability of ARDS development. Regarding this, **Patel et al.**^[29] reported that P/F ratio is the most frequently applied method for evaluating the severity of hypoxic respiratory failure, particularly in cases of ARDS. Besides, **DesPrez et al.**^[30] found that oxygen saturation provides prognostic information and assessment of ARDS severity. Moreover, **Mahmood et al.**^[16] demonstrated that the quantification of PC volume may facilitate the detection of individuals at elevated probability for developing ARDS. Furthermore, **Wang et al.**^[31] showed that measurement of PC volume using three-dimensional CT is a feasible approach in emergency department settings and proves valuable for identifying patients at increased probability of developing ARDS. In addition, **Gattinoni et al.**^[32] declared that the decrease in PCO₂ acts as a deterrent of positive outcomes in patients with ARDS. Similarly, **Lee et al.**^[33] concluded that for forecasting pneumonia, the area under the receiver operating characteristic (ROC) curve for the PC volume ratio was 0.85 (95% CI: 0.76–0.95, $p = 0.008$), with an optimal cutoff value of 70.4%. Quantification of PC volume on initial CT facilitates the onset determination of chest trauma individuals at elevated probability of

developing late respiratory complications. Also, a study done by **Fischer et al.**^[34] stated that SPO₂ < 95% was found to be predictors of pneumonia. Further aligning with our study, **Çelik et al.**^[35] revealed that AaDO₂ sensitivity was found at 49.6% and specificity at 82.7% for the predicting of survival in pneumonia individuals.

The limitations of this study encompass its relatively small sample size and its single-center design. Moreover, the investigation primarily addressed in-hospital outcomes and short-term complications, without assessing long-term pulmonary function or post-discharge morbidity.

CONCLUSIONS

In the evaluation of outcomes following blunt chest trauma, CT-derived PC volume found to be a more robust prognostic tool than ABG analysis. Specifically, contusion volume was highly effective in predicting complications such as pneumonia and ARDS, with critical thresholds identified at >0.19 and >0.429, respectively, yielding strong diagnostic performance across sensitivity, specificity, and negative predictive value. Despite some ABG indices such as PO₂, SpO₂, P/F ratio, and Aa DO₂ offering moderate predictive relevance, parameters like pH, HCO₃, and PCO₂ demonstrated limited reliability. These findings position CT quantification of lung injury as a superior method for early risk stratification in chest trauma cases.

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