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Hypoxemia in Polytrauma Patients Requiring Intensive Care Unit: Incidence, Causes, and Impact Outcome

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1. Abstract

1.1. Purpose: to study the incidence, the time of development, the etiologies, and the impact outcome of post-traumatic hypoxemia in the intensive care unit.

1.2. Methods: Prospective study in the ICU of a university hospital over a period of 10 months. All patients admitted for polytrauma and suffering from hypoxemia were included in this study.

1.3. Results: During the study period, 110 trauma patients were admitted into our ICU, among whom 83 patients (77.3%) developed hypoxemia on ICU admission and/or during ICU stay, and they were included in this study. The mean age (±SD) was 34±18 years. Seventy-eight patients (94%) needed sedation and mechanical ventilation on ICU admission. The mean time elapsed from the ICU admission to the development of hypoxemia was 2 ± 1.8 days. The three most common causes of hypoxemia were pulmonary contusion in 34 patients (41%), nosocomial pneumonia in 24 (29%), and acute pulmonary embolism in 23 (28%). The mean ICU stay was 20 ± 15 days. The mortality rate in the ICU at 28 days was 19.3%. Multivariate analysis showed that independent risk factors associated with mortality were age **(OR: 1.08)**, SOFA score calculated on the day of hypoxemia **(OR: 1.86)**, and high value of Blood Glucose on the day of hypoxemia **(OR: 1.64)**.

1.4. Conclusion: Post-traumatic hypoxemia is frequently observed in severe trauma patients requiring ICU admission. The causes of hypoxemia are usually multiple and associated. The presence of hypoxemia is associated with a high mortality rate.

2. Introduction

In the intensive care unit, hypoxemia, although frequently observed with an incidence exceeding 50% in patients in the medical-surgical category [1], is rarely studied. Indeed, few studies have focused on the prevalence of hypoxemia, its causes, and its prognostic significance in patients needing admission to the intensive care unit, according to an examination of the literature. [2–5]. In polytrauma patients, post-traumatic pulmonary lesions remain a major concern for resuscitators because of the hypoxemia that they can induce. Indeed, the causes leading to respiratory failure are multiple and can occur with a variable delay after the trauma. Despite its multiple causes and its negative prognostic impact, the incidence of post-traumatic hypoxemia in intensive care units has rarely been studied. The reported incidence of this complication varies, according to the few published studies, from 38 to 69% [2–5]. Management must be carried out as soon as possible and take into consideration the different etiologies involved in the development of this hypoxemia. Indeed, poor prognosis and a high mortality rate can be linked to any diagnostic and/or treatment delay. Finally, this management must combine symptomatic treatment and etiological treatment. In Tunisia, and to the best of our knowledge, no research on this topic has been published concerning the intensive care unit.. Thus, it seemed interesting to us to carry out this study. The primary endpoint was to study the incidence of post-traumatic hypoxemia and to study the time of development

of this complication. The secondary endpoint was to study the etiologies and the impact outcome of post-traumatic hypoxemia.

3. Methods

We conducted a prospective study in the ICU of a university hospital over a period of 10 months (From March 1, 2021, to December 31, 2021). There is no conflict of interest related to the research described above. This study was approved by the local institutional review board, and the requirement for written informed consent was waived by the ethics committee. Our department is a 22-bed medical-surgical ICU in a teaching hospital of 510 beds that serves as a first-line medical center for an urban population of two million inhabitants. In our city, severe trauma patients were usually admitted directly from the scene of the accident. On arrival at the hospital, all patients were examined and scored according to the Glasgow coma scale (GCS) and underwent computed cerebral tomography (CT) and/or whole-body CT scan as soon as feasible.

Our study concerns all patients suffering from head trauma and/or polytrauma during the study period. The adapted definition of a polytrauma patient is an injured person who has sustained multiple injuries, at least one of which may cause significant disability and may be life-threatening, and/or when the mechanism or the violence of the trauma suggests that such lesions exist. In this study, we included all patients admitted for head trauma and/or polytrauma and suffering from hypoxemia. Hypoxemia was defined as oxygen saturation measured by pulse oximetry (SPO2) <92% in room air. In patients receiving mechanical ventilation, arterial hypoxemia was defined as arterial oxygen tension (PaO2)/fractional inspired oxygen (FiO2) ratio (PaO2/FIO2 ratio) less than 300. FiO2 for patients with spontaneous ventilation was calculated using the following formula: FiO2= Oxygen flow O2×4 + 21%.

The exclusion criteria concerned patients less than 16 years of age and/or those who were discharged from the ICU within 48 hours. Patients who died within 48 hours after ICU admission were also excluded.

Acute respiratory distress syndrome (ARDS) was defined according to the Berlin classification [6]. According to these guidelines, ARDS was classified as mild (200< PaO2/FiO2 ≤300 mm Hg), moderate (100< PaO2/FiO2 ≤200 mm Hg), or severe (PaO2/FiO2 ≤100 mmHg) [6].

The mean time elapsed from the onset of hypoxemia to admission was recorded for each patient. Hypoxemia is classified as “immediate” if it is diagnosed on ICU admission, “early” if it is diagnosed within 48 hrs of admission, and “Late” if it was developed later than the 3rd day after ICU admission.

All epidemiological, clinical, biological, and radiological parameters and the related therapeutic measures were registered on admission and during the ICU stay.

Biochemical parameters measured on admission and during the ICU stay were also recorded, such as arterial blood gases and acid-base status (pH, PaO2/FiO2 ratio, and HCO 3), hemoglobin concentration, serum glucose and sodium levels, blood urea, and platelet counts. The use of preventive anticoagulant agents, the delay of development of hypoxemia, and the clinical manifestations associated with the hypoxemia were also recorded for each patient. Injury severity score (ISS) [7], simplified acute physiology score (SAPS II) [8], Sequential Organ Failure Assessment (SOFA) score [9], and ICU stay (LOS) were also calculated for all patients.

For each patient, the cause of hypoxemia was identified based on clinical examination and the analysis of Chest X-ray and/or Computed tomography (CT) of the chest.

Finally, all trauma patients admitted to our ICU during the study period were classified into two groups. The first group included all trauma patients with confirmed hypoxemia. The second group included Hypoxemia-free patients. The last group is not included in our study.

3.1. Statistical Analysis

The Chi-square test is used to assess subgroups (survival and death), and categorical data that are reported as proportions. Subgroups are assessed using the Student t-test, and continuous variables are reported as means (± SD).Risk factors are evaluated in univariate analysis and by multivariate analysis by a multiple logistic stepwise regression procedure. Odds ratios are estimated from the b coefficients obtained, with respective 95% confidence intervals (CI 95%).

4. Results

From March 1, 2021, to December 31, 2021, the total number of trauma patients admitted into our ICU was 110, among whom 83 patients (77.3%) developed hypoxemia on ICU admission and/or during ICU stay, and they were included in this study (Figure 1). The causes of ICU admission were: isolated traumatic head injury (TBI) in 17 patients (21%) and multiple- trauma in 66 (79%). Among the patients included in our study, 61 patients (74%) had a traumatic brain injury (TBI), 55 patients (66%) had a chest trauma, 21 (29%) had an abdominal trauma, and 15 (18%) had long-bone fractures.

There were 70 males (84%) and 13 females (16%). The mean age (±SD) was 34±18 years (median: 31 years) (Figure 2). Seventy-eight patients (94%) needed sedation, oro-tracheal intubation, and mechanical ventilation on ICU admission. Arterial blood gases done on ICU admission showed that 50 patients (60.4%) had hypoxemia with PaO2/FiO2 ratio< 300. Table 1 shows the clinical characteristics of the study group at admission.

All patients underwent a brain CT scan upon admission to the hospital. It was normal only in 22 patients (26.5%). The brain CT-scan results are shown in Table 2. On the other hand, a body CT scan was performed for 79 patients (95%). It showed a chest injury in 55 patients (66%), abdominal injury in 24 (29%), and

long-born fractures in 23 (28%). Pulmonary contusions represent the most observed injury (41%). Table 3 shows the chest wall and pulmonary injuries. On ICU admission, 78 patients (94%) needed mechanical ventilation, 49 (59%) developed shock requiring the use of vasopressors, 34 (41%) needed blood transfusion, and 27 (32.5%) underwent surgical intervention.

The mean time elapsed from the ICU admission to the development of hypoxemia was 2 ± 1.8 days (ranges 1-12 days) with a median at 1 day. Fifty patients (60.4%) developed hypoxemia on the first day of ICU admission, 8 (9.6%) within 48 hours, and 25 (30.2%) developed late hypoxemia (3rd day after ICU admission). Figure 3 shows the timing of the occurrence of hypoxemia in all population groups. Hypoxemia was associated with other organ failure in 60 patients (73%).

According to the PaO2/FiO2 ratio, Hypoxemia was classified as mild (200< PaO2/FiO2 ≤300 mm Hg) in 46 patients (56%), moderate (100< PaO2/FiO2 ≤200 mm Hg) in 32 (38%), and severe (PaO2/FiO2 ≤100 mmHg) in 5(6%).

The top three causes of respiratory failure among those experiencing acute hypoxic episodes were pulmonary contusion in 34 patients (41%), nosocomial pneumonia in 24 (29%), and acute pulmonary embolism in 23 (28%). The causes of early and later hypoxemia are detailed in Table 4. As shown in Table 4, the causes of early hypoxemia are dominated by pulmonary contusion (34%), pulmonary atelectasis (20.4%), and pulmonary embolism (18%). However, the most observed causes of late hypoxemia were nosocomial pneumonia (28%) and pulmonary embolism (9.6%) (Table 4).

The mean ICU stay was 20±15 days (range: 30-90 days) with a median time at 17 days. During their ICU stay, 78 (94%) patients needed invasive mechanical ventilation (mean time 12.9±10; median: 10 days). The mortality rate in the ICU at 28 days was 19.3% (16 patients). According to the PaO2/FiO2 ratio, the morality rate was at 6.5% (3 patients) in the mild hypoxemia group, 28% (9 patients) in the moderate hypoxemia group, and 80 % (4 patients) in the severe hypoxemia group.

Univariate analysis showed that factors associated with poor mortality were low PaO2/FiO2 ratio on ICU admission, hyperglycemia, and high severity score indexes on ICU admission and the day of hypoxemia diagnosis (Table 5).

Multivariate analysis showed that independent risk factors associated with mortality were age (p: 0.013; OR: 1.08; 95% CI: 1.01-1.15), SOFA score calculated on the day of hypoxemia (p: 0.008; OR: 1.86; 95% CI: 1.18-2.94), and high value of Blood Glucose on the day of hypoxemia (p: 0.004; OR: 1.64; 95% CI: 1.17-2.30).

Table 1: Patient characteristics at the time of intensive care unit admission

Parameters	Results (Total number 83) [median]
Age (years)	34±18 [31]
Sex M/F	70/13
SAPS II score	32 ± 12 [30]
ISS	22.6 ± 14.7 [20]
SOFA Score	8±3 [8]
APACHE II	14 ± 6.3[13]
GCS score	8 ± 4 [3-15]
Heart rate (beats/min)	90 ± 24 **[50-147]**
SBP (mmHg)	120 ± 22 **[53-184]**
Shock	**49(59%)**
Use of catecholamine	**49(59%)**
Use of mechanical ventilation	78 (93 %)
PaO_2/FiO_2 ratio	50 (60.4%)
Hypoxemia (PaO_2/FiO_2 < 300)	254 ± 116 (241)
Use of antibiotics (Yes/No)	24(29%)
Blood Transfusion	34 (41%)

APACHEII: Acute Physiology and Chronic Health Evaluation II; GCS: Glasgow Coma Scale; P/F ratio:the ratio of arterial oxygen partial pressure to fractional inspired oxygen; SAPSII: Simplified Acute Physiology Score II; SOFA: Sequential Organ Failure Assessment; SBP: systolic blood pressure

Table 2: Results of cerebral CT scan

CT Scan signs	Number	Percentage (%)
Meningeal hemorrhage	44	53
Cerebral contusion	44	53
Subdural hematoma	28	33.7
Extradural **hematoma**	21	25.3
Mass lesion	7	8.4
Brain herniation	1	1.2
Cerebral Trunk injury	3	3.6
Diffuse Axonal Injury	3	3.6
corpus callosum damage	2	2.4
Normal CT-scan	22	26.5

Table 3: Results of Chest CT scan on ICU admission

Type of lesion	Number (%)
Pulmonary contusion	34 (41%)
Rib fracture	26 (31.3%)
Pneumothorax	19 (22.9%)
Intra alveolar hemorrhage	14 (16.7%)
Hemothorax	11 (13.3%)
Inhalation	4

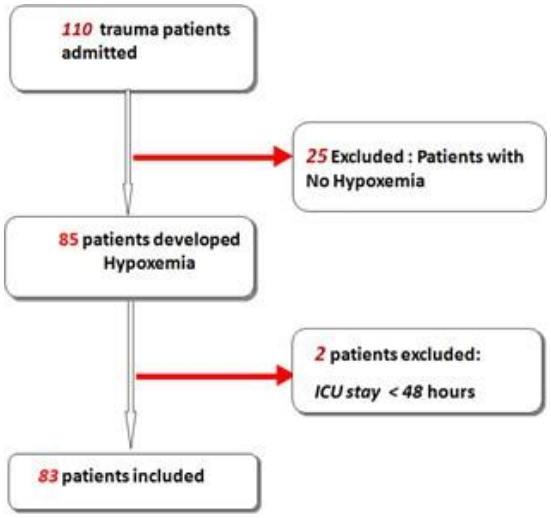

Figure 1: Study's flowchart.

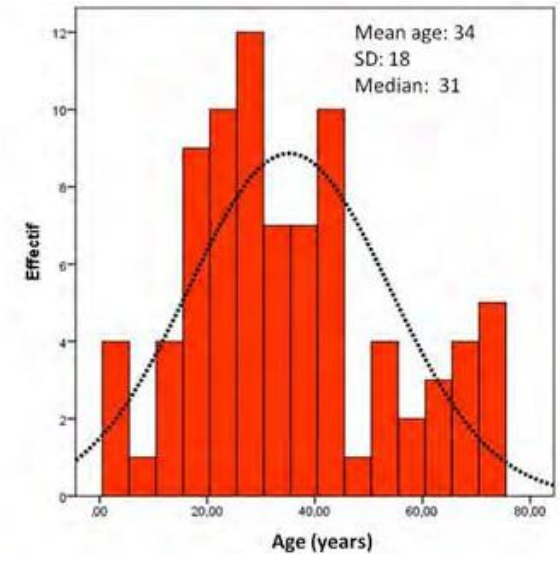

Figure 2: Distribution of patients according to their age

Table 4: The causes of hypoxemia in all population, early and later hypoxemia groups.

	Causes	Number (%)
All population groups	Pulmonary contusion	34 (41 %)
	Nosocomial Pneumonia	24 (29 %)
	Pulmonary embolism	23 (27.8 %)
	Pulmonary atelectasis	17 (20.4 %)
	Pneumothorax	15 (18 %)
	ARDS	14(17%)
	Neurogenic pulmonary edema	6 (7 %)
	community-acquired pneumonia	4 (4,8 %)
	Cardiogenic pulmonary edema	6 (7 %)
	Fat embolism	2 (2.4 %)
Early hypoxemia groups	Pulmonary contusion	34 (41 %)
	Pulmonary atelectasis	17 (20.4 %)
	Pulmonary embolism	15(18 %)
	Pneumothorax	14 (17 %)
	Community-acquired pneumonia	4 (4,8 %)
Later hypoxemia groups	Nosocomial Pneumonia	24 (28 %)
	ARDS	14(17%)
	Pulmonary embolism	8 (9.6 %)
	Neurogenic pulmonary edema	6(7%)
	Cardiogenic pulmonary edema	6 (7 %)
	fat embolism	2 (2.4 %)
	Pneumothorax	1 (1.2 %)
Number of causes of hypoxemia	1	46(55 %)
	2	26(31%)
	3	8 (9.6 %)
	4 or more	3(3.6%)

Table 5: Factors associated to death in univariate analysis.

Variables		Mean±SD/Proportion (%)		P value
		Survivors	Deaths	
		(N: 67)	(N: 16)	
Demographic characteristics	Age	33.7 ± *18*	41.8±*20*	0.16
	Sex, M/F	56/11	14/2	1
Severity scores	GCS on admission	8±*3*	7±*4*	0.38
	SAPSII score on admission	30.7±*11.4*	39±*16*	0.03
	APACHEII score on admission	13.3±*6*	17.4±*6.7*	0.023
	SOFA score on admission	7.6±*2.6*	10±*2.8*	0.72
Clinical and biological parameters on ICU admission	Blood glucose on ICU admission	7.5±*2.5*	10±*4.8*	0.007
	PaO2/FiO2 ratio *on ICU admission*	269±*113*	185±*95*	0.01
	Shock on ICU admission	37(55%)	12(75%)	0.17
	Blood creatinine (µmol L–1)	74±*57*	87±*54*	0.4
	Blood Transfusion	23(34%)	11(68%)	0.02
	Surgical procedures	18(26%)	9(57%)	0.037

Injury assessment	Injury Severity Score	21±*13*	26±*18*	0.56
	Meningeal hemorrhage	32(47%)	12(75%)	0.057
	Cerebral contusion	35(52%)	9(56%)	1
	Subdural hematoma	23(34%)	5(31%)	1
	Pulmonary contusion	27(40%)	7(43%)	1
	Rib fracture	22(32%)	4(25%)	0.1
	Pneumothorax	18(26%)	1(6%)	0.1
biological parameters (DHD)	*PaO2/FiO2* ratio (DHD)	216 ±56	177±*71*	0.02
	Blood Glucose (mmol L–1) (DHD)	7±2	11±*5*	<0.001
Severity scores (DHD)	SAPSII score on admission	30.7±*11.4*	39±*16*	0.03
	SOFA score	8±2.5	11±*2.7*	0.001
Duration of mechanical ventilation (days)		13±10	9±*6*	0.16
ICU length of stay (days)		22±16	10±*7*	0.008

APACHEII: Acute Physiology and Chronic Health Evaluation II; GCS: Glasgow Coma Scale;P/F ratio:the ratio of arterial oxygen partial pressure to fractional inspired oxygen; SAPSII: Simplified Acute Physiology Score II; SOFA: Sequential Organ Failure Assessment; DHD: the day of hypoxemia diagnosis

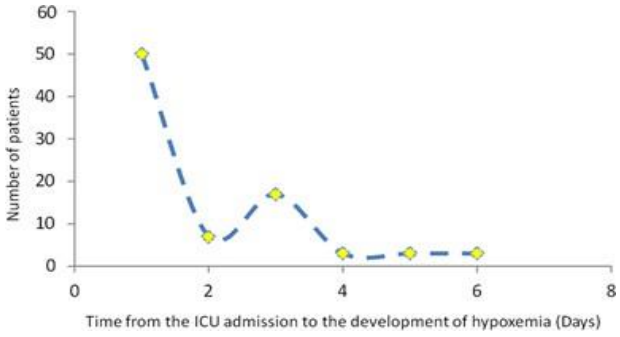

Figure 3: Elapsed Time from the ICU admission to the development of hypoxemia (Days)

5. Discussion

Our study confirms that post-traumatic hypoxemia is frequently observed. Moreover, depending on the time elapsed from the onset of hypoxemia to admission, an early occurrence (within 48 hrs. of admission) of this complication was observed in 58 patients (70%). The etiologies are multiple and differ according to the time of the onset of this complication. Finally, a poor outcome and a high death rate are linked to the emergence of this condition. Independent risk factors associated with mortality were age, SOFA score calculated on the day of hypoxemia, and high value of Blood Glucose on the day of hypoxemia.

The reported incidence of post-traumatic hypoxemia varies, according to the few published studies, from 38 to 69% [2–5]. The severity of our patients' conditions may explain the high incidence which was observed in our study. In fact, of the 83 patients included, 66 (79%) had multiple trauma with TBI in 61 patients (74%), chest trauma in 55 patients (66%), and long-bone fractures in 15 (18%). Moreover, 78 patients (93%) needed sedation, oro-tracheal intubation, and mechanical ventilation on ICU admission.

Hypoxemia is common and potentially life-threatening in critically ill patients. However, the incidence, management, and outcomes of hypoxemia in patients requiring intensive care unit (ICU) admission are poorly known [2, 10]. Moreover, among patients suffering from hypoxemia, trauma-induced respiratory failure is a particular entity because it typically affects younger individuals, is frequently brought on by a lung contusion and inhalation, and is associated with instability of the rib cage [4, 11]. The presence of serial rib fractures with instability of the rib cage may represent an etiology of prolonged hypoventilation. In addition, patients with severe brain injury may have significant brain-lung interaction, which can set off a domino effect that worsens the patient's prognosis [12]. In fact, numerous conditions, including ventilator-associated pneumonia, post-traumatic pulmonary embolism, neurogenic pulmonary edema (NPE), and acute respiratory distress syndrome can be observed in this specific condition [12-16]. In ICU, the causes of hypoxemia are usually multiple, and associated causes are often identified [2]. In our study, 45% of included patients have more than two isolated causes of hypoxemia.

Our study is characterized by the high incidence (28%) of post-traumatic pulmonary embolism in comparison with other published data [14, 15, 17, 18]. Traumatic injuries are often associated with an increased risk of VTE events, which can be explained by the severity of the conditions of the patients included in our study and the use of a systematic screening protocol in our ICU, even in patients who are clinically asymptomatic.

In our practice, if the initial injuries did not progress on a control brain CT-scan (performed in 24 hours and at varying intervals thereafter based on clinical symptoms), we initiate preventive anticoagulation (with low molecular weight heparin) within 24 hours following ICU admission.

In our series, 78 (94%) patients required invasive mechanical ventilation (IMV). The high incidence of IMV used in our study is explained by the severity of our included population. In fact, Among the included patients, 61 patients (74%) had traumatic brain injury (TBI), 55 patients (66%) had chest trauma, and 21 (29%) had abdominal trauma. In our study, for all patients who needed IMV, we used a low tidal volume (6-8 ml/kg ideal body weight) and airway pressure (plateau pressure <30 cmH2O) as recommended [6].

The presence of hypoxemia is associated with a high mortality rate in many published data [2-4]. Moreover, this association is well-established for patients suffering from severe hypoxemia [2-4]. In our study, the mortality rate in the ICU at 28 days was 19.3%. According to the PaO2/FiO2 ratio, the morality rate was at 6.5% in mild hypoxemia groups, 28% in moderate hypoxemia groups, and 80% in severe hypoxemia groups. Our study confirms the same results published by other studies [2-4].

Finally, our study has at least two limitations. First, there are few patients included. Second, this research was based on a single-center data set. However, despite the above-mentioned limitations, we should acknowledge that this study is one of the rare studies analyzing the incidence, time of development, causes, and impact outcome of post-traumatic hypoxemia in severe trauma patients requiring ICU admission.

In conclusion, post-traumatic hypoxemia is frequently observed in severe trauma patients requiring ICU admission. The causes of hypoxemia are usually multiple and associated. The presence of hypoxemia is associated with a high mortality rate. Prevention is highly warranted.

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