

Relationship between arterial oxygen tension and mortality of patients in intensive care unit on mechanical ventilation support

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ABSTRACT

BACKGROUND: Although there are studies demonstrating hyperoxia may be an independent risk factor for increased mortality and morbidity, this issue remains unclear. Our research then aimed to examine the relationship between arterial oxygen tension, arterial carbon dioxide tension, and in-hospital mortality of critically ill patients in intensive care unit (ICU).

METHODS: After obtaining ethics committee approval, we analyzed a retrospective data of patients over the age of 18 who survived at least 24 hours in the ICU on mechanical ventilatory support between year 2008 and 2012. The demographic properties, mechanical ventilation, and blood gas parameters were studied. We defined hyperoxia group as PaO₂ value of ≥120 mmHg and normoxemia group as PaO₂ of 60–120 mmHg. Patients with PaCO₂ value <30 mmHg were determined to have hypocapnia, those with 30–50 mmHg normocapnia, and those with >50 mmHg hypercapnia.

RESULTS: Between 2008 and 2012, a total of 7689 patients were admitted to the ICU. Of 450 patients meeting the inclusion criteria of the study, 263 (58.4%) were male and 187 (41.6%) were female. Normoxia was observed in 232 (51.5%) patients and hyperoxia in 218 (48.5%) patients. The mean PaO₂ was 16.2 kPa (121.50 mmHg), and FiO₂ was 60%. 254 (56%) of the patients had died during the five-year period. There was no statistically significant difference in mortality between PaO₂ levels and PCO₂ levels (p>0.05). According to the classification of PaO₂ and FiO₂, there was no statistically significant difference in mortality (p>0.05) among patients. In addition, no statistically significant difference was found between the survival rates according to PCO₂ classification (p=0.602, p>0.05).

CONCLUSION: There was no significant association between mortality and oxygen and carbon dioxide of patients in ICU on mechanical ventilatory support.

Keywords: Hyperoxia; intensive care unit; mechanical ventilation; normoxia; oxygen toxicity.

INTRODUCTION

Oxygen is one of the most frequently used therapeutic agents in the clinical settings worldwide. However, healthcare professionals, even in developed countries, have paid little attention with respect to its indications for potential use, dosage, concentrations required for administration, duration of administration, side effects, and toxicity.^[1] It was toward the end of the 19th century that the use of oxygen became widespread in the treatment of diseases. Paul Bert and Lorrain Smith^[2]

reported that oxygen at high concentrations could lead to toxicity. As shown in several animal experiments and a limited number of human studies, the administration of high doses of oxygen can cause deleterious systemic effects on other organ systems, such as the lungs, brain, and heart.^[3–6] Although there are studies which have shown that hyperoxia increased morbidity and mortality,^[7] there are also studies highlighting its benefits.^[8–10] Currently, oxygen administered to patients is

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considered as a “drug”.^[11] Despite there appear studies on the exposure time and concentration of oxygen in the body, no clear guidelines have emerged yet on this subject.^[12,13]

The administration of oxygen therapy to critically ill patients is a complex process with many barriers, and it has been shown that the majority of applications have been made without sufficient evidence.^[14–16] Studying the role of oxygen toxicity in patients who are on mechanical ventilation is even more of a challenge. In a retrospective study of 50 intensive care units (ICUs), de Jonge et al.^[17] examined the relationship between mortality and arterial oxygen pressure in patients on mechanical ventilation support, and high mortality rates were observed in patients with high FiO_2 . They found that both low and high PaO_2 increased mortality rates in the first 24 hours in ICU.^[17] In a recent similar study by Eastwood et al.^[18] hypoxia increased mortality, but no finding was reported suggesting that hyperoxia increased mortality.

The primary aim of our study was to explore the relationship between arterial oxygen tension and mortality of patients in the intensive care unit on mechanical ventilation support, high or low PaO_2 in the first 24 hours being associated with mortality. Then we examine the relationship between arterial carbon dioxide tension and in-hospital mortality of patients requiring mechanical ventilation support.

MATERIALS AND METHODS

Ethical Approval

The study was granted approval by the Ethics Committee of İstanbul University Medical Faculty (Registry Url: 2011/1790-790 in accordance with the Code of Ethics of the World Medical Association-Declaration of Helsinki).

Inclusion Criteria

Our ICU is a tertiary level unit with a total of 17 beds, comprising four isolation rooms and 13 beds in the open area. The inclusion criteria were patients aged over 18 years who received mechanical ventilator support from the time of admission and had arterial blood gas analyses in the ICU. Patients with severe burns or following cardiac surgery were not included in our study.^[17] We have also excluded those with incomplete records of blood gas values, APACHE II score with diagnosis on admission, or SOFA score.

Data Collection

From the patient records, the demographic characteristics (age, gender), date of admittance to ICU, length of stay in ICU, diagnosis causing ICU admittance, comorbidities, and blood gas values (provided it was arterial blood gas) within the first 48 hours in ICU were noted on a protocol form prepared with 6-hour sections. The values were examined from the blood gas values at 6-hour intervals, including the PaO_2 (arterial oxygen pressure), PaCO_2 (arterial carbon diox-

ide pressure), mechanical ventilation mode, PEEP (positive end-expiratory pressure), PAP (peak airway pressure), MAP (mean airway pressure), FiO_2 (inspired oxygen concentration), number of sections where FiO_2 decreased or increased, and the $\text{PaO}_2/\text{FiO}_2$ ratio.

To explore the relationship between arterial oxygen pressures and mortality, the arterial blood gas selection model described in the study by Eastwood et al.^[18] was used. Based on this model, if the FiO_2 value is at least and more than 0.5, the PaO_2 value was taken as the value at the time of the highest alveolar arterial gradient (A-a), and if the FiO_2 value was <0.5 , the worst PaO_2 value was taken. In case the FiO_2 values taken in the first 24 hours were both less than 0.5 and at least and more than 0.5, then the blood gas parameter was used to define as the “worst” PaO_2 . Following data preparation protocols, the patients were separated into two groups according to the PaO_2 values. Group 1 was the normoxemic group with a PaO_2 value of 60–120 mmHg and Group 2 was the hyperoxemic group with a PaO_2 value of ≥ 120 mmHg.

Hypocapnia, normocapnia, and hypercapnia were established by taking the mean value of the PCO_2 parameter throughout the follow-up period and examining these values. Patients with PaCO_2 value <30 mmHg were determined to have hypocapnia, those with 30–50 mmHg normocapnia, and those with >50 mmHg hypercapnia.

Statistical Analysis

The statistical analyses of the study findings were made by the Number Cruncher Statistical System 2007 and Power Analysis and Sample Size 2008 Statistical Software (Utah, USA) programs. Descriptive statistical methods such as the mean value, standard deviation [SD], median, and rate were used. In the comparison of quantitative data between two groups showing normal distribution of the parameters, the Student's t-test was applied, and for parameters not showing normal distribution, the Mann-Whitney U-test was employed. For comparison of qualitative data, the Continuity Correction (Yates) chi-square test and the Pearson chi-square test were used. The Kaplan-Meier analysis and the log rank test were applied to perform survival analysis. The results were evaluated with a 95% confidence interval. A value of $p < 0.05$ was accepted as statistically significant.

RESULTS

450 out of 7689 patients admitted to the ICU between 2008 and 2012 who met the inclusion criteria were recruited, comprising 263 (58.4%) males and 187 (41.6%) females. The mean age of the patients was 59.60 ± 16.96 years (range: 18–93 years).

The general characteristics of the patients according to mortality are shown in Table I. The mean age of the nonsurvivor patients was observed to be statistically significantly higher than

Table 1. General characteristics of patients according to mortality

	Mortality		p
	Survivors (n=195)	Non-survivors (n=254)	
Age (years), mean±SD	57.70±18.04	61.05±15.99	¹ 0.042*
Intensive care unit stay (days), mean±SD (median)	17.31±14.6 (13)	13.99±15.28 (9)	² 0.001**
APACHE, Mean±SD (median)	18.38±6.39 (18)	24.61±5.99 (24)	² 0.001**
Simplified Acute Physiology Score II, mean±SD	39.44±13.09	52.24±15.88	¹ 0.001**
Total mechanical ventilatory time (days), mean±SD (median)	10.7±9.71 (8)	13.01±12.78 (9)	² 0.048*
Renal replacement therapy (days), mean±SD (median)	0.87±2.73 (0.8)	2.05±3.98 (1.5)	² 0.001**
Inotrope (days), mean±SD (median)	2.62±3.36 (2)	6.19±5.33 (5)	² 0.001**
Male, n (%)	111 (56.9)	151 (59.4)	³ 0.590
Female, n (%)	84 (43.1)	103 (40.6)	

¹Student t-test; ²Mann-Whitney U test; ³Pearson Ki-Kare test; *p<0.05; **p<0.01. SD: Standard deviation.

Table 2. The diagnoses of the patients on admission to ICU according to in-hospital mortality

	Mortality				p
	Survivors (n=195)		Non-survivors (n=254)		
	n	%	n	%	
Cardiovascular	68	34.9	87	34.4	³ 0.915
Diabetes mellitus	38	19.5	61	24.0	³ 0.251
Hypertension	62	31.8	87	34.3	³ 0.584
Respiratory	30	15.4	41	16.2	³ 0.814
Renal	21	10.8	33	13.0	³ 0.473
Gastrointestinal	5	2.6	20	7.9	³ 0.026*
Malignancy	30	30	48	18.9	³ 0.001**
Hematological	6	3.1	35	13.8	⁴ 0.001**
Neurological	20	10.3	25	9.8	⁴ 1.000
Other	9	4.6	7	2.8	⁴ 0.426

³Pearson Ki-Kare test; ⁴Yates Continuity Correctin test; *p<0.05; **p<0.01. ICU: Intensive care unit.

that of the survivors (p<0.05). It was found that the length of stay in ICU of the survivor group was statistically significantly longer than that of the nonsurvivor group (p<0.01). The total duration of mechanical ventilation, the renal replacement therapy (RRT) days, and the duration of vasopressor agent administration were also higher in the nonsurvivor group than the survivor group. The APACHE II scores and the SAPS II levels were statistically significantly higher in the nonsurvivor group than in the survivor group (p<0.01).

No statistically significant relationship was found between mortality and the place from where the patient was admitted to ICU. The exitus rate was statistically significantly higher in

Table 3. The blood gas parameters of the patients according to mortality

	Mortality		p		
	Survivors (n=195)			Non-survivors (n=254)	
	Mean±SD			Mean±SD	
PaO ₂	122.07±59.37		115.92±46.89		¹ 0.221
FiO ₂	0.59±0.19		0.60±0.18		¹ 0.741
PaO ₂ /FiO ₂	218.82±97.81		213.67±98.90		¹ 0.583

¹Student t-test. SD: Standard deviation.

Table 4. The evaluation of mortality according to PaO₂ classifications

Mortality	PaO ₂				p
	Normoxia (n=231)		Hyperoxia (n=218)		
	n	%	n	%	
Survivors	102	44.2	93	42.7	0.749
Non-survivors	129	55.8	125	57.3	

Pearson chi-squared test.

the group transferred from clinics within the same hospital (p<0.01), and the discharge rate was determined to be higher in the group transferred postoperatively from the operating theater (p<0.01).

The diagnoses of the patients on admission to ICU according to mortality are shown in Table 2. No statistically significant difference was determined between the admission diagnoses

Table 5. The evaluation of mortality according to PaO₂ classifications

	PaO ₂		P
	Normoxia (n=232)	Hyperoxia (n=218)	
Age (years) (mean±SD)	60.03±15.71	59.14±1.26	¹ 0.581
Intensive care unit stay (days), mean±SD (median)	15.65±14.36 (11)	15.17±15.78 (10)	² 0.189
APACHE, Mean±SD (median)	22.22±6.95 (22)	21.58±6.82 (21)	² 0.347
Simplified Acute Physiology Score II, mean±SD	46.54±15.48	46.81±16.59	¹ 0.857
Total mechanical ventilatory time (days), mean±SD (median)	12.55±12.71 (9)	11.4±10.24 (8)	² 0.371
Renal replacement therapy (days), mean±SD (median)	1.37±3.36 (0)	1.71±3.71 (0)	² 0.078
Inotrope (days), mean±SD (median)	4.39±4.86 (3)	4.89±4.94 (4)	² 0.188
Male, n (%)	140 (60.3)	123 (56.4)	³ 0.399
Female, n (%)	92 (39.7)	95 (43.6)	

¹Student t-test; ²Mann-Whitney U test; ³Pearson Chi-squared test. SD: Standard deviation.

Table 6. The standardized mortality rate according to PaO₂ and FiO₂ classifications in nonsurvivor group

Non-survivors		SMR		P
		Mean	SD	
PaO ₂ (mmHg)	60–80	0.69	0.16	0.170
	80–100	0.61	0.18	
	100–120	0.67	0.18	
	≥120	0.62	0.20	
FiO ₂	0.21–0.40	0.59	0.19	0.367
	0.41–0.60	0.64	0.19	
	0.61–0.80	0.65	0.20	
	0.81–1.0	0.77	0.12	

Kruskal Wallis test. SMR: Standardized mortality ratio according to SAPS II; SD: Standard deviation.

and mortality rate ($p>0.05$) except in patients admitted for gastrointestinal, malignancy, and hematological diagnoses ($p<0.05$).

The evaluation of the worst values and the mean values throughout the follow-up period of the blood gas parameters of the patients according to mortality is shown in Table 3. In the first 24 hours in ICU, the mean PaO₂ was determined as 16.2 kPa (122.44±31.8 mmHg) and mean FiO₂ as 60%. The PaO₂ was 115.92±46 mmHg in the nonsurvivor group and the PaO₂ was 122.07±59.37 mmHg in the survivor group. Furthermore, no significant difference was found between the two groups.

The evaluation of mortality according to PaO₂ classification ($p>0.05$) is presented in Table 4. It has been shown that there is no statistically significant difference between the survivor and nonsurvivor groups in respect of mortality.

No statistically significant difference was also seen in the length of stay in ICU, APACHE score, SAPS II, total duration of mechanical ventilation and RRT day, inotropic agent day, or gender in accordance with the PaO₂ classification ($p>0.05$) (Table 5).

The survival rates of the normoxia and hyperoxia groups were evaluated using the log rank test. It can be noticed that there was no statistically significant difference between the two groups ($p=0.506$, $p>0.05$) (Fig. 1).

No statistically significant difference was observed between the standardized mortality rates (SMRs) according to PaO₂ classification ($p>0.05$) and FiO₂ classification ($p>0.05$) (Table 6).

Hypocapnia (PCO₂ <30 mmHg) was determined in 36 (8%) patients, normocapnia (PCO₂ 30–50 mmHg) in 354 (78.7%), and hypercapnia (PCO₂ >50 mmHg) in 60 (13.3%) patients. It further revealed no statistically significant difference in mortality between patients as per the PCO₂ classifications ($p>0.05$) (Table 7).

When the survival rates of the hypocapnia, normocapnia, and hypercapnia groups were assessed with the log rank test, they have also shown no statistically significant difference ($p=0.602$, $p>0.05$) (Fig. 2).

DISCUSSION

The aim of this study was to examine the relationship between mortality and the arterial oxygen pressure and arterial carbon dioxide pressure values measured during the conducted follow-up of patients on mechanical ventilator support in ICU. From the results of this study, the following conclusions were reached:

1. No significant relationship was determined between in-hospital mortality and the arterial oxygen pressure values of the first and second 24 hours.

Table 7. Characteristics of patients according to PCO₂ classifications

	PCO ₂			P
	Hypocapnia (n=36)	Normocapnia (n=354)	Hypercapnia (n=60)	
Age (years), mean±SD	63.34±12.89	59.27±17.51	59.15±15.64	⁵ 0.330
ICU stay (days), mean±SD (median)	13.58±11.49 (10)	15.32±14 (11)	17.1±21.63 (10)	⁶ 0.774
APACHE, mean±SD (median)	22.56±7.39 (23)	21.79±6.76 (21)	22.25±7.42 (22)	⁶ 0.661
SAPS II, mean±SD	49.11±15.77 (45.5)	46.18±15.88 (45)	48.1±16.96 (48.5)	⁵ 0.440
Total MV Time (days), mean±SD (median)	9.83±6.81 (8)	11.95±11.13 (9)	13.55±15.72 (9)	⁶ 0.764
RR (days), mean±SD (median)	2.58±5.39 (0)	1.45±3.37 (0)	1.4±3.03 (0)	⁶ 0.201
Inotrope (days), mean±SD (median)	5.22±4.68 (4)	4.61±4.83 (4)	4.42±5.43 (3)	⁶ 0.408
Male, n (%)	27 (75.0)	203 (57.3)	33 (55.0)	³ 0.104
Female, n (%)	9 (25.0)	151 (42.7)	27 (45.0)	
Survivors, n (%)	13 (37.1)	158 (44.6)	24 (40.0)	³ 0.589
Non-survivors, n (%)	22 (62.9)	196 (55.4)	36 (60.0)	

⁵Oneway ANOVA test; ⁶Kruskal Wallis test; ³Pearson Ki-Kare test.

ICU: Intensive care unit; SAPS II: Simplified Acute Physiology Score; MV: Mechanical ventilatory; RR: Renal replacement therapy; SD: Standard deviation.

2. No significant relationship was determined between in-hospital mortality and the arterial carbon dioxide values.

There have been conflicting reports on the subject of the relationship between hyperoxia and mortality in studies which have examined the relationship between arterial oxygen values and mortality in patients on mechanical ventilator support.^[17-20] However, our study has obtained similar results with Eastwood et al.^[18] concerning the relationship between oxygen pressure and mortality in patients on mechanical ventilator support. In their study, while there was a significant relationship between hypoxia and mortality, no relationship was established between hyperoxia and mortality. The methodology utilized by Eastwood et al.^[18] found a correlation between the PaO₂ values on admittance to ICU and the peak PaO₂ values in the first 24 hours. In our study, the same methodology was used in determining the worst PaO₂ value in the first 24 hours. Despite similar statistical results in the two studies of mean

PaO₂ (15.1 vs. 16.2 kPa) and FiO₂ values (62% vs. 60%), a significant difference was found between the mortality values (26% vs. 56%). The reason for this difference could be that in the Eastwood et al. study, there were a greater number of patients being monitored in the ICU following elective surgery and the mortality of this patient group is known to be low. The SAPS II score evaluating the severity of the disease was determined to be higher (46.7 vs. 43.1) in our study compared to that of Eastwood et al. A similar study carried out by de Jonge^[17] revealed that mortality increased with high FiO₂, low PaO₂, and high PaO₂ values in the first 24 hours in ICU. The common ground between our study and that of de Jonge et al. was the determination of high FiO₂ (50.4% vs. 60%) and high PaO₂ (13.1 kPa vs. 16.2 kPa) in the first 24 hours. It has been indicated that there was a significant difference in the mortality rates of patients (23% vs. 56%) and there may be several reasons for this difference. The results could have been affected by the different methodologies used in determining the worst PaO₂

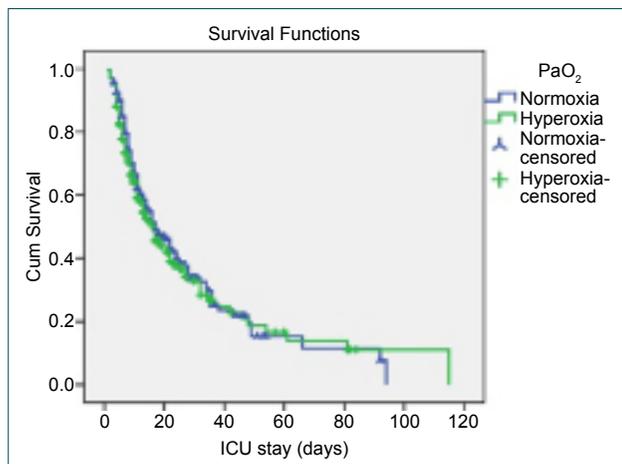


Figure 1. The survival rates of the normoxia and hyperoxia groups.

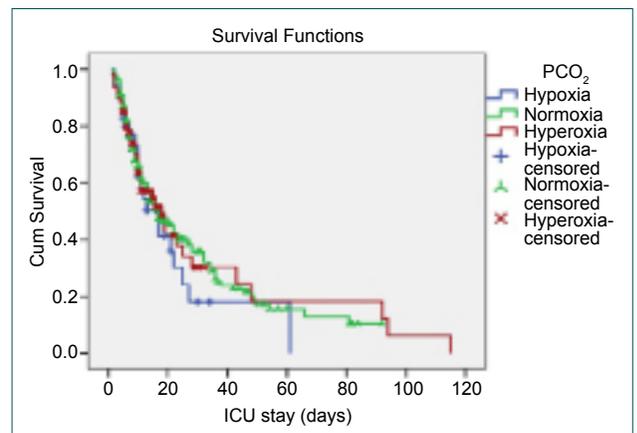


Figure 2. The survival rates of the hypocapnia, normocapnia, and hypercapnia groups.

value in the first 24 hours (the highest alveolar arterial gradient vs. the worst PaO₂/FiO₂ ratio), differences in diagnoses on admittance, differences in ICU management, and the fact that the latter study was conducted in 50 different centers and there may have been differences in practices in the ICUs.

Other studies have shown that the majority of patients on mechanical ventilatory support in the ICUs are administered a large amount of oxygen and the ventilator settings are not adjusted according to hyperoxemic levels.^[4,21] In a recent prospective, randomized, controlled study of critical patient groups on mechanical ventilatory support in ICU, Suzuki et al.^[20] compared conventional and conservative oxygen treatment. They reported that restrictive oxygen therapy that aimed to keep the SpO₂ value between 90% and 92% would not have any major clinical or physiological side effects. Although the mean PaO₂ values of the conservative group were statistically lower than those of the conventional group (83 mmHg [71–97 mmHg] vs. 107 mmHg [94–131 mmHg]), no difference was determined in terms of mortality rates. However, Girardis et al.^[19] reported lower mortality rates in the conservative group (mean PaO₂: 87 mmHg [79–97 mmHg]) than the conventional group (mean PaO₂: 102 mmHg [88–116 mmHg]) in a similar study. Comparing these studies with our study, the mean PaO₂ values and mortality rates in our study were notably higher. This difference may be attributed to the detrimental effects of hyperoxia.

In the current study, no statistically significant relationship was determined between mortality and the hypocapnia, normocapnia, and hypercapnia groups classified according to the arterial carbon dioxide values. A recent retrospective study of a large series of patients monitored in ICU with mechanical ventilation support demonstrated that hypocapnia led to poor clinical results.^[22,23] A work by Del Castillo et al.^[24] which examined the results of pediatric patients after cardiac arrest revealed that mortality has increased in hypocapnia and hypercapnia groups. In a meta-analysis of traumatic brain injury patients, Roberts et al.^[25] reported that hypocapnia and hypercapnia were reasons for poor clinical outcomes. In our study, the reasons for the different results could have been that the patients were admitted to ICU for several reasons and the study methodology was different. Hence, there is a need for further studies of larger patient groups to investigate the relationship between partial carbon dioxide values and mortality.

The most important limitation of this retrospective study was the difficulty in determining a causal relationship between oxygenation and in-hospital mortality. No clear statements have been made related to the limit values and standard categorization values, which are the most important determining factors in the relationship between arterial partial oxygen pressures and hypoxia, normoxia, and hyperoxia classifications. Hyperoxia has been described as PaO₂ >300 mmHg and hypoxia as <60 mmHg^[24,26,27] in some studies, but Brenner et al.^[28] defined hyperoxia as PaO₂ >200 mmHg and hypoxia as <60 mmHg. de Graaff et al.^[4] and Eastwood et al.^[18] also defined hyperoxia as

arterial oxygen pressure of >16 kPa or 120 mmHg. The international campaign for survival in sepsis and studies made on an ARDS study group recommended that in determining the clear arterial oxygen concentration values, the PaO₂ value of arterial oxygen pressures in the least inspiratory oxygen fraction should be between 55 and 80 mmHg or the arterial hemoglobin saturation should be kept between 88% and 95%.^[29] A similar definition was used in the current study and the hyperoxia group was defined to have pressure >120 mmHg. There are no standard categorizations related to oxygen values and no clear classification had affected the results of studies conducted. Our study was implemented in a single center with a limited number of patients. As previous similar studies have been made with larger patient series, it is difficult to determine the significant relationship between the two studies and to form an opinion of methods in the approach to hyperoxia in our country.

Furthermore, the total hospital stay (days) and markers of clinical conditions of the patients were not available and the reasons for ventilator adjustment were unknown. As the study was planned according to the first blood gas values only, it was not possible to come to a conclusion on how long the values remained at normoxic and hyperoxic levels and how much this affected mortality. Although no significant relationship was determined between mortality and the oxygen values of the subgroups according to the diagnoses of the patients, further studies are warranted to examine subgroups of more extensive patient series.

Conclusion

In conclusion, no significant relationship was determined between in-hospital mortality and the arterial oxygen pressure and arterial carbon dioxide pressure of patients on mechanical ventilation in ICU. In contrast to previous studies, the relationship between early hyperoxemia and mortality was unclear. As the current study was a retrospective, observational study, there is a need for further prospective, controlled research including national data collection with an organized recording system and interventional strategies to determine a causal relationship.

Conflict of interest: None declared.

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DENEYSEL ÇALIŞMA - ÖZET

Yoğun bakım ünitesinde mekanik ventilatör desteği ile takip edilen hastalarda arteriyel oksijen basıncı ile mortalite arasındaki ilişki

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AMAÇ: Hiperoksinin yoğun bakım ünitelerinde mekanik ventilasyon desteği ile takip edilen hastalarda mortalite ve morbiditeyi artıran bağımsız bir risk faktörü olduğuna dair farklı çalışmalar olmasına rağmen bu konu belirsizliğini korumaktadır.

GEREÇ VE YÖNTEM: Bu çalışma, etik komite onayı alındıktan sonra mekanik ventilatör desteği ile yoğun bakım ünitesine takip edilen 18 yaş üzerindeki 7689 hastanın ilk 24 saatteki verileri incelenerek yapılan geriye dönük, gözlemsel bir çalışmadır. Hastaların demografik özellikleri, mekanik ventilasyon ve kan gazı parametreleri analiz edildi. Hiperoksemiye PaO₂ ≥ 120 mmHg, normoksemiye PaO₂ 60–120 mmHg olarak tanımlandı. PaCO₂ değeri <30 mmHg olan hastalar hipokapnik, 30–50 mmHg olanlar normokapnik, >50 mmHg olanlar hiperkapnik olarak tanımlandı.

BULGULAR: Hastaların yaş ortalaması 59.60±16.96 (dağılım, 18–93) yıl idi. Ortalama PaO₂ değeri 16.2 kPa (121.50 mmHg) ve FiO₂ değeri %60 idi. İki yüz otuz iki hasta normoksi (%51.5) ve 218 hastada (%48.5) hiperoksi gözlemlendi. Hastaların mortalite oranı %56 idi. PaO₂ düzeyleri ve PCO₂ düzeyleri ile mortalite arasında istatistiksel olarak anlamlı fark bulunmadı (p>0.05). PaO₂ ve FiO₂ sınıflamasına göre mortalitede istatistiksel olarak anlamlı fark yoktu (p>0.05). PCO₂ sınıflamasına göre sağkalım oranları arasında istatistiksel olarak anlamlı fark bulunmadı (p=0.602, p>0.05).

TARTIŞMA: Yoğun bakım ünitesindeki mekanik ventilatör desteği ile takip edilen hastalarda oksijen ve karbondioksit değerleri ile mortalite arasında anlamlı ilişki bulunmadı.

Anahtar sözcükler: Hiperoksemi; mekanik ventilasyon; normoksemi; oksijen toksisitesi; yoğun bakım ünitesi.

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