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ORIGINAL ARTICLE

Incidence of excess oxygen use in critically ill children and its impact on in-hospital mortality.

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ABSTRACT… Objective: To evaluate excess oxygen incidence and the prognostic value of Cumulative Excess Oxygen Exposure (CEOE), by using non-invasive SpO2, for in-hospital mortality in a Pakistani Pediatric intensive care unit (PICU). **Study Design:** Prospective observational cohort study. **Setting:** Department of Pediatrics and Child Health, Liaquat National Hospital, Karachi, Pakistan. **Period:** May 2024 to April 2025. **Methods:** Using non-probability consecutive sampling, this study included all children aged 1 month to 15 years admitted in PICU and requiring invasive or non-invasive respiratory support within the first 24 hours of the admission. Children with cyanotic heart diseases and who had a stay of <24hrs were excluded. CEOE was calculated as the mean hourly FiO2 exceeding 0.21, for the hours in which corresponding SpO2 was ≥95%, over the first 24 hours of respiratory support. Primary outcome was CEOE, secondary was in-hospital mortality. **Results:** Among 197 children for analysis, 98.5% experienced excess oxygen exposure of varying severity. In-hospital mortality was 10.7%. Median CEOE was 29.00 (IQR 19.08-40.87). CEOE of >39.46 showed 71.45% sensitivity and 77.8% specificity. CEOE significantly predicted mortality (AUC 0.754; 95% CI 0.687-0.812; p<0.0001). Higher CEOE was significantly associated with invasive mechanical ventilation (IMV), prolonged PICU stay, mortality and Multi-Organ Dysfunction Syndrome (MODS) (all p<0.01). Shorter PICU stay (adjusted OR 0.202; p=0.017) and High-flow nasal cannula (HFNC) (adjusted OR 0.123; p<0.001) use were independently protective against high CEOE. **Conclusion:** Nearly all critically ill children experienced excess oxygen exposure. CEOE, a potentially modifiable risk factor, predicts in-hospital mortality. This highlights the significance of oxygen titration, practicable in resource-limited settings like ours by using non-invasive SpO2.

Key words: Cumulative Excess Oxygen Exposure (CEOE), Critically Ill Children, In-hospital Mortality, Oxygen Therapy, Pediatric Intensive Care Unit (PICU), Pakistan.

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INTRODUCTION

Oxygen therapy is crucial in the PICU for correcting tissue hypoxia and for stabilization of critically ill children. However, it is often considered a low-risk, readily available therapy, leading to its inadequate scrutiny of dosing and duration. The therapeutic window of oxygen therapy is narrow. A growing body of evidence from adult literature and now from pediatrics as well suggests that the relationship between oxygenation and outcomes is U-shaped (polynomial), indicating that both hypoxemia and hyperoxemia are linked with increased morbidity and mortality.[1,2] Hyperoxemia induces injury at cellular level through oxidative stress producing reactive oxygen species (ROS), leading to cell inflammation and death, and endothelial dysfunction via vasoconstriction, collectively leading to multi-organ failure.[3] Although current clinical practice guidelines recommend titrating oxygen to achieve adequate rather than supranormal oxygen saturation levels, oxygen overuse still remains common, probably due to clinician's concerns of hypoxia.[4]

In children, research on hyperoxemia was previously restricted to pre-term neonates, showing its link to chronic conditions like retinopathy of prematurity.[5] For critically ill children beyond the neonatal period, evidence is emerging but less definitive. According to recent meta-analyses and systematic reviews, arterial hyperoxemia (defined as PaO2 ≥300 torr), is associated with increased mortality but there is considerable diversity present, regarding its definition, timing and method of measurement and patient population.[2,6] Most pediatric studies relied on PaO2, which are invasive and may not capture the cumulative burden of oxygen.[7] Non-invasive SpO2 can be used instead and can provide easy and continuous monitoring on hourly basis.[8]

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The concept of CEOE, using continuously available parameters like FiO2 and SpO2, offers a breakthrough in pediatric critical care as it acts as an independent predictor of mortality and MODS in mechanically ventilated children, proven by Balcarcel et al. and Geva et al. in their studies.[8,9]

In LMIC like Pakistan, where resources are limited and disease burden is high, titrating oxygen therapy is pivotal not only for clinical outcomes but also for resource stewardship. Local data on oxygen practices and its impact is scarce. In our previous retrospective study Naz et al., we found that almost all critically ill children were exposed to excess oxygen, though there was no significant association found with MODS and mortality.[10] However, due to limitations of retrospective design, we structured this prospective study to further analyze the incidence of excess oxygen use and the predictive accuracy of CEOE for in-hospital mortality among the children admitted to a tertiary care PICU in Karachi, Pakistan.

METHODS

We conducted a prospective observational cohort study in a 12 bed PICU of Liaquat National Hospital, one of the major tertiary care centers in Karachi, Pakistan, from May 2024 to April 2025 after the ethical approval from the hospital's Ethical Review Committee (ERC-0950-2023-LNH-ERC). The waiver of informed consent was granted as the study only required nursing chart reviews and lab investigations.

A non-probability consecutive sampling method was used, enrolling all eligible children. A sample size of 196 was calculated, using an open-Epi based calculator, based on 95% confidence interval, hyperoxemia incidence of 50.3%[10] and absolute precision of 75%. We add 4 dropouts, making the final sample size of 200.

All children aged 1 month to 15 years old, admitted in our PICU, requiring HFNC or MV as an oxygen support during the 1st 24 hours of admission, during the defined timeline of one year, were eligible for enrollment. Exclusion criteria included: 1) Children with cyanotic heart diseases, 2) Children who had a PICU stay of <24hours, 3) Children having SpO2/FiO2 data missing for >10%.

Demographic data (age, gender), co-morbidities, primary diagnosis, type of respiratory support, length of PICU stay, development of MODS and in-hospital mortality were recorded. The Pediatric Organ Dysfunction Information Update Mandate (PODIUM) was used to define MODS.[11]

We adopted the Balcarcel et al. approach for data processing in our study8. Hourly FiO2/SpO2 values were extracted from nursing charts. Only 1st 24 hours data of oxygen therapy was used as the oxidative stress, due to ROS, is most conspicuous in this time period. If FiO2/SpO2 was not recorded for a given hour, the most recent value was then used and mean value was used if multiple values were recorded within the same hour. SpO2 threshold of >95% is considered healthy in normal children12,13. Excess oxygen exposure was identified when FiO2 exceeded 0.21 while SpO2 remained ≥95%. Patients with hypoxemia, defined as SpO2 of <90% for consecutive ≥2 hours at any point during the 1st 24 hours of oxygen therapy, were isolated to a separate hypoxemia group as hypoxemia is likely a significant confounder of outcomes. Our primary outcome was CEOE. CEOE was defined as the mean hourly FiO2 above 0.21 for the hours in which corresponding SpO2 was ≥95% and then divided by 24. For example, if a child receives FiO2 of 0.6 for 24 hours with constant SpO2 of ≥95%, then the CEOE would be 39 [60-21= 39 => (39x24)/24 = 39].

Data was analyzed using IBM SPSS Statistics v27 and MedCalc v20. Normality was checked by the Shapiro wilk test. Median and quartiles (1st quartile-3rd quartile) were calculated for quantitative variables whereas frequency and percentages were reported for qualitative variables. The predictive accuracy was determined by the Receiver Operating Characteristic (ROC) curve analysis. The optimal cut-off value was identified by the Youden index. Chi-square/fisher exact tests were applied to determine the association between qualitative variables (e.g., CEOE groups vs. clinical outcomes). Odds were calculated by binary logistic regression. Adjusted odds were calculated for variables significant on univariate binary logistics regression. P-value ≤ 0.05

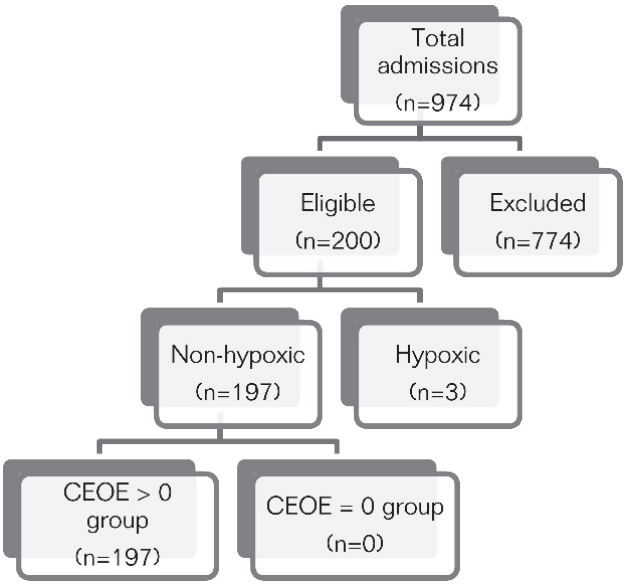
were considered as significant.

RESULTS

Out of 974 children admitted in our PICU from May 2024 till April 2025, a total of 200 children who met the inclusion criteria were enrolled. Out of these 200 children, 3 were further excluded for the data analysis due to hypoxia - resulting in a total of 197 children included in the final analysis. No child had a CEOE =0 and all 197 children (98.5%) had excess oxygen exposure of varying severity (Figure-1).

FIGURE-1

Participant distribution



The cohort had a male predominance 63.5% (125) with a median age of 2.0 years (IQR 0.72-5.0); 78.2% (154) were ≤5 years old. The most common primary diagnosis was respiratory illness 65% (128), followed by surgical/trauma patients 11.2% (22) and neurological diseases 9.1% (18). HFNC was the most common respiratory support used in 61.9% (122) of children. The median length of PICU stay was 5 days (IQR 4.0-7.0), with 58.4% (115) of children hospitalized for 4-7 days. The overall in-hospital mortality rate was 10.7% (21) and MODS developed in 21.8% (43) of children. The median CEOE was found to be 29.00 (IQR 19.08-40.87) with 53.8% (106) having a CEOE of ≤29.0 (Table-I).

Our ROC curve analysis showed that CEOE was a significant predictor of in-hospital mortality. The Youden Index identified an optimal cut-off value of >39.46, with a sensitivity of 71.43% and specificity of 77.84%. The Area Under the Curve (AUC) was 0.754 (95% CI: 0.687 to 0.812; $p < 0.0001$) (Figure-2).

TABLE-I

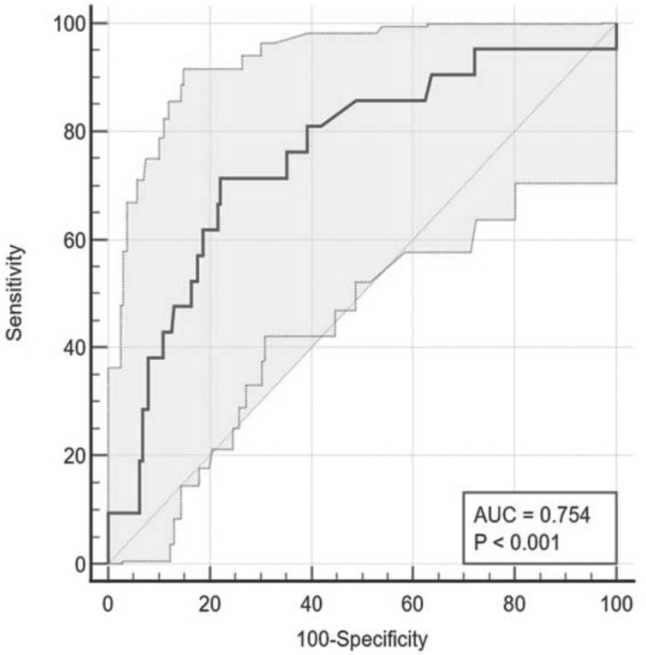
Demographic and clinical profile of study participants (n=197)

	n (%)
Gender	
Male	125(63.5)
Female	72(36.5)
Age(years)∫	2.00(0.72-5.00)
Age group	
≤5 years	154(78.2)
>5 years	43(21.8)
Co-morbidities	
Yes	69(35)
No	128(65)
Diagnosis	
CNS	18(9.1)
CVS	5(2.5)
GIT	3(1.5)
Respiratory	128(65)
Sepsis	15(7.6)
Surgical/Trauma	22(11.2)
Other	6(3)
Respiratory Support	
High-flow nasal cannula (HFNC)	122(61.9)
Invasive mechanical ventilation (IMV)	75(38.1)
Length of stay(days) ∫	5.00(4.00-7.00)
Stay group	
≤3 days	35(17.8)
4-7 days	115(58.4)
>7 days	47(23.9)
Multiorgan dysfunction syndrome	
Yes	43(21.8)
No	154(78.2)
In-Hospital mortality	
Yes	21(10.7)
No	176(89.3)
Cumulative Excess Oxygen Exposure (CEOE) ∫	29.00(19.08-40.87)
CEOE Group	
≤ 29.0	106(53.8)
> 29.0	91(46.2)

∫Median (1st Quartile-3rd Quartile)

FIGURE-2

Receiver operating curve for CEOE in predicting in-hospital mortality



Youden index J	0.4927
Associated criterion	>39.46
Sensitivity	71.43
Specificity	77.84
Area under the ROC curve (AUC)	0.754
95% Confidence interval [b]	0.687 - 0.812
Significance level P (Area=0.5)	<0.0001

Patients were divided into 2 separate groups according to the ROC-derived CEOE cut-off (≤39.46 vs. >39.46) for the bivariate analysis using Chi-square/Fisher exact test. There were strong and significant associations found with a CEOE >39.46 with a primary diagnosis other than respiratory ($p < 0.001$), use of IMV rather than HFNC ($p < 0.001$), longer PICU stay of >7 days ($p = 0.002$), development of MODS ($p < 0.001$) and in-hospital mortality ($p < 0.001$). Though, no significant association was found between CEOE >39.46 with age group, gender and co-morbidities (Table-II).

TABLE-II

Association of cumulative excess oxygen exposure (as per cut-off values) with demographic and clinical profile of study participants

	Cumulative Excess Oxygen Exposure n (%)		P-Value
	≤39.46 143 (72.6%)	>39.46 54 (27.4%)	
Gender			
Male	93(65)	32(59.3)	0.453
Female	50(35)	22(40.7)	
Age group			
≤5 years	116(81.1)	38(70.4)	0.103
>5 years	27(18.9)	16(29.6)	
Co-morbidities			
Yes	49(34.3)	20(37)	0.716
No	94(65.7)	34(63)	
Diagnosis			
CNS	8(5.6)	10(18.5)	<0.001*
CVS	2(1.4)	3(5.6)	
GIT	0(0)	3(5.6)	
Respiratory	110(76.9)	18(33.3)	
Sepsis	7(4.9)	8(14.8)	
Surgical/Trauma	12(8.4)	10(18.5)	
Other	4(2.8)	2(3.7)	
Respiratory Support			
High-flow nasal cannula (HFNC)	110(76.9)	12(22.2)	<0.001*
Invasive mechanical ventilation	33(23.1)	42(77.8)	
Length of stay			
≤3 days	30(21)	5(9.3)	0.002*
4-7 days	88(61.5)	27(50)	
>7 days	25(17.5)	22(40.7)	
Multi-Organ dysfunction syndrome (MODS)			
Yes	18(12.6)	25(46.3)	<0.001*
No	125(87.4)	29(53.7)	
In-Hospital mortality			
Yes	6(4.2)	15(27.8)	<0.001*
No	137(95.8)	39(72.2)	

Chi-square/fisher exact test were applied.
p-value≤0.05 considered as significant.
*Significant at 0.05 levels.

In the logistic regression analysis, factors which are associated with lower odds of high CEOE group (i.e. 39.46) included HFNC use (OR: 0.086, $p < 0.001$) and shorter PICU stay of ≤7 days (≤3 days= OR:

0.189, p = 0.003; 4-7 days = OR: 0.349, p = 0.004). Factors with higher odds included development of MODS (OR: 5.987, p < 0.001) and in-hospital mortality (OR: 8.782, p < 0.001). After adjusting for confounders, only HFNC use (adjusted Odd Ratio (aOR): 0.123, p < 0.001) and a short PICU stay of ≤3 days (aOR: 0.202, p = 0.017) remained significant. While, association between high CEOE >39.46 with development of MODS (aOR : 1.782, p = 0.247) and in-hospital mortality (aOR: 2.665, p = 0.123) became non-significant (Table-III).

DISCUSSION

This prospective cohort study, conducted in a PICU of Karachi, Pakistan found that nearly all critically ill children admitted in our PICU had excess oxygen exposure of varying severity and CEOE, quantified using hourly FiO2 and SpO2, was a significant predictor of in-hospital mortality. While emerging data has already established a dose-dependent and time-dependent relationship between hyperoxia and outcomes in critically ill children, most of them come from developed countries and they have largely relied on PaO2 to measure hyperoxemia.[2,6,14] Unlike adults, a major disadvantage of this strategy is the uncommon use of arterial lines in children and thus, unnecessary pricks for this investigation.

TABLE-III

Odds for cumulative excess oxygen exposure>39.46

	Un-adjusted		Adjusted	
	P-Value	**OR (95% CI)**	**P-Value**	**aOR (95% CI)**
Gender				
Male	0.453	0.782(0.411-1.487)		
Female		1.000		
Age group				
≤5 years	0.106	0.553(0.269-1.134)		
>5 years		1.000		
Co-morbidities				
Yes	0.716	1.128(0.588-2.165)		
No		1.000		
Diagnosis				
CNS	0.353	2.500(0.361-17.315)		
CVS	0.383	3.00(0.255-35.334)		
GIT	0.999	NA		
Respiratory	0.216	0.327(0.056-1.919)		
Sepsis	0.413	2.286(0.316-16.512)		
Surgical/Trauma	0.597	1.667(0.251-11.071)		
Other		1.000		
Respiratory Support				
High-flow nasal cannula (HFNC)	<0.001*	0.086(0.040-0.182)	<0.001*	0.123(0.055-0.276)
Invasive mechanical ventilation (IMV)		1.000		1.000
Length of stay				
≤3 days	0.003*	0.189(0.063-0.573)	0.017*	0.202(0.054-0.748)
4-7 days	0.004*	0.349(0.170-0.714)	0.226	0.578(0.238-1.403)
>7 days		1.000		1.000
Multi-Organ dysfunction syndrome MODS)				
Yes	<0.001*	5.987(2.890-12.400)	0.247	1.782(0.670-4.745)
No		1.000		1.000
In-Hospital mortality				
Yes	<0.001*	8.782(3.194-24.145)	0.123	2.665(0.767-9.259)
No		1.000		1.000

OR; Odds Ratio, aOR; adjusted Odds Ratio, CI; Confidence Interval

Binary logistic regression was applied. p-value ≤ 0.05 considered as significant. *Significant at 0.05 levels.

In contrast, our study demonstrates that excess oxygen exposure can be effectively measured using the readily available and non-invasive SpO2. Using SpO2 instead of PaO2, is a practical approach for resource-limited countries like ours. Though local data is emerging[10,15], our prospective design adds some strength.

Importantly, our SpO2-derived CEOE threshold of >39.46 demonstrates good sensitivity and specificity and thus, helps in identifying children at high risk of death. This is consistent with Balcarcel et al., Geva et al. and Panhwer et al., who also proved that high CEOE is associated with increased risk of MODS and mortality and can be measured using SpO2 instead of invasive PaO2.[8,9,15] Our study shows that the path to safer oxygen therapy does not require advanced technology, rather use of monitors which are already present at every bedside. This is congruent with the recent Oxy-PICU trial, which showed that conservative oxygenation (SpO2 of 88% - 92%) have improved outcomes, and also used this very same non–invasive parameter of SpO2.[16] Collectively, this evidence shows that CEOE is a quantifiable and modifiable risk factor and our SpO2-derived CEOE threshold offers a practical and achievable warning sign for clinicians.

Excess oxygen can cause oxidative stress leading to cell death, endothelial dysfunction, impairment of mitochondrial respiration and amplification of inflammatory pathways, all of which may collectively exacerbate organ failure and worse outcomes, as proven by previous studies.[17,18] Our study also shows a strong association of high CEOE with development of MODS and prolonged length of PICU stay. As Horvat et al. emphasized that in methodological discussion on observational research, confounding by indication is an inherent challenge when studying supportive therapies such as oxygen.[19] Though MODS was strongly associated with high CEOE in the initial analysis of our study but in adjusted model, its effect was attenuated, suggesting that MODS may not be just a confounding variable but likely a mediator in the causal pathway between excess oxygen exposure and mortality. In other words, we may directly mitigate the risk of triggering this deleterious chain of events by reducing cumulative exposure.

Interventional evidence in pediatrics remains scarce but informative. Randomized control trials comparing conservative and liberal oxygenation targets, including Oxy-PICU trial have shown the feasibility and safety of conservative oxygenation. Though, there were no differences in mortality between the 2 groups.[16] Emerging evidence of potential harm from excess oxygen along with no clear benefit from liberal oxygenation, suggests that the equilibrium has shifted towards the more judicious use of oxygen. Our study of cumulative exposure can be a practical and feasible framework for more future trials and quality improvement studies.

Our study has several strengths. First, unlike our retrospective study, this prospective design limits measurement bias and allows for real-time data collection. Second, easily available parameters of FiO2/SpO2 to calculate the CEOE, which are routinely monitored in all PICU, unlike invasive PaO2. Third, it focused on a resource-limited setting where data on excess oxygen is limited.

This study has several limitations. First, the single-center design may limit generalizability. Second, while we adjusted for key confounders, residual confounding is possible in any observational study. That is, higher FiO2 might indicate greater illness severity which was not fully captured. Third, we focused on the data of first 24 hours of oxygen support but as suggested by Geva et al. and Zhu Zhu et al., the impact of CEOE over a more extended period might have different implications.[9,17] Fourth, darker skin pigmentation might show less accurate SpO2, which is a relevant consideration in our population.[20] Lastly, the decision to use an SpO2 threshold of 95% to define "excess oxygen" reflects current guideline recommendation but warrants further investigation to define it properly.[21]

CONCLUSION

Our study proved that excess oxygen is not benign, rather it is linked to poor outcomes. CEOE (derived from SpO2) is a strong predictor of in-hospital mortality and it is also a modifiable risk factor. This showed that improving outcomes does not necessarily require advanced technologies, rather oxygen titration can be achieved by simple, cost-free and easily available intervention like SpO2. In

other words, our study highlights the significance of careful oxygen titration, supports existing guidelines recommendations, and strengthens the rationale for large randomized trials of conservative oxygen saturation. Treating oxygen as a drug which requires proper scrutiny like dosing, monitoring and reassessment might be a key to improve outcomes in PICU.

Ethical Approval

Ethical approval was obtained from the hospital's Ethical Review Committee of Liaquat National Hospital (ERC-0950-2023-LNH-ERC).

Patients' consent

The waiver of informed consent was granted as the study only required nursing chart reviews and lab investigations.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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AUTHORSHIP AND CONTRIBUTION DECLARATION	
1	**Rubab Naz:** Conceived & designed the study, collected data, interpret the data and wrote the manuscript
2	**Atika Sher:** Conceived the study, review it critically.
3	**Madiha Hussain:** Collected data, Interpret the data.
4	**Misbah Haroon:** Collected data, Interpret the data.
5	**Saba Hussain:** Collected data.
6	**Anwar-ul-Haque:** Analyzed the data, Interpret the data.