Case Report

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Temporary hypoxemia at high altitude in an intensive care unit physician

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Abstract

A 42-year-old pediatric intensive care unit physician traveled to Nepal and took a helicopter trip to Everest Base Camp. The helicopter reached an altitude of 5500 m during flight and descended at different destinations with varying altitudes. At Hotel Everest View at 3820 m, his oxygen saturation was 79%. He had mild tachypnea and deep breathing but was able to walk around, jump, and take photographs. He returned to Kathmandu (altitude, 1324 m) without using any supplemental oxygen during the entire trip. Based on calculations with the alveolar gas equation, he observed that he and his fellow passengers probably had hypoxemia during the trip. In summary, temporary hypoxemia associated with high altitude in healthy individuals without cardiorespiratory compromise may not require oxygen therapy. In contrast, intensive care unit patients who have respiratory failure may have similar oxygen saturation levels but may require oxygen therapy and mechanical ventilation. The oxygen saturation level must be interpreted in consideration of the clinical scenario before deciding about the need for oxygen therapy.

Keywords

Oxygen saturation, respiratory physiology, respiratory failure

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Introduction

Hypoxia is an acute life-threatening condition defined as decreased oxygen delivery to the tissues, whereas hypoxemia refers to low oxygen level in the blood. The terms hypoxia and hypoxemia frequently are used interchangeably in clinical practice, but the difference between these conditions may be understood by considering altitude physiology. Hypoxemia at high altitude can be offset by increased cardiac output (stroke volume × heart rate) to prevent hypoxia, whereas uncompensated hypoxemia can trigger hypoxia in patients with respiratory failure.

It is important to understand oxygen delivery in terms of supply and demand in different situations. Typical oxygen delivery is fourfold greater than use, as the body typically extracts only 25% of delivered oxygen.[1] In the hospital, the prompt use of oxygen therapy to treat hypoxia is an established practice. However, we cannot bring unlimited supplemental oxygen to locations at high altitudes because of limitations imposed by equipment weight. Therefore, using oxygen to normalize asymptomatic low SaO_2 in healthy adults can be practically impossible at high altitudes.

A pediatric intensive care unit (PICU) physician traveled to a high-altitude location and experienced hypoxemia (low SaO_2) that did not require oxygen therapy. This was in contrast with his experiences in the PICU with patients who require urgent oxygen therapy for life-threatening hypoxia and impending respiratory failure. The purpose of this case report is to alert physicians about the distinction between hypoxemia observed at high altitude versus hypoxia in intensive care unit patients.

Case report

A 42-year-old PICU physician traveled to Nepal and took a helicopter trip to Everest Base Camp. He was born in Nepal, lived for many years at altitudes of 1020 to 1350 m, had never previously traveled to Everest Base Camp, and lived in US cities at sea level during the past 15 years. He traveled together with two US tourists whom he had met during the trip. They embarked at Kathmandu Airport and landed at Lukla Airport within 1 h to offload reserve fuel, enabling

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Table 1. Alveolar and arterial partial pressure of carbon dioxide and oxygen versus altitude, calculated with the alveolar gas equation.[a]

Stop no.	Location	Altitude (m)	PB[b] (mm Hg (hPa))	$PACO_2$ (mm Hg)	PAO_2 (mm Hg)[c]
–	Sea level	0	760 [1013]	40	100
0	Kathmandu Airport, Bagmati	1324	650 [867]	40	77
1	Lukla Airport, Solukhumbu	2845	475 [633]	30	52
2	Pheriche, Solukhumbu[d]	4200	400 [533]	30	37
3	Kala Patthar, Everest Base Camp[e]	5420	375 [500]	30	31
4	Hotel Everest View, Syangboche, Solukhumbu[f]	3820	450 [600]	30	47

FIO_2: fraction of inspired oxygen; $PACO_2$: alveolar partial pressure of carbon dioxide; $PaCO_2$: arterial partial pressure of carbon dioxide; PAO_2: alveolar partial pressure of oxygen; PB: barometric pressure; PH_2O: saturated vapor pressure of water at body temperature; RQ: respiratory quotient.

[a]No arterial blood gas measurements or data about PH_2O were available. It was assumed that $PACO_2$ was 30 mm Hg during the hypoxic hyperventilatory state, and $PACO_2$ was assumed identical to $PaCO_2$. PH_2O was assumed constant. In normal healthy lungs with low alveolar-arterial gradient, PAO_2 was assumed equal to PaO_2.

[b]PB was calculated as reported previously.[2,3]

[c]PAO_2 was calculated using the alveolar gas equation: $PAO_2 = FIO_2 \times (PB - PH_2O) - (PACO_2/RQ)$, where $FIO_2 = 0.21$; PB varied with altitude; $PH_2O = 47$ mm Hg, and RQ = 0.8.

[d]Stayed for 30 min.

[e]Stayed for 15 min.

[f]Place where maximum time was spent and oxygen saturation was measured with pulse oximetry. Ventilatory drive is normally driven by $PaCO_2$. Hypoxia is not a usual stimulant for the respiratory center. However, in a hypobaric oxygen environment at altitudes ca. 3000 m and higher, the hypoxia-induced reflex tachypneic response is prominent and leads to protective hypocarbia.[4]

them to continue higher. Various altitudes and anticipated $PaCO_2$ and PaO_2 are described (Table 1).[2–4] Supplemental oxygen was used by the pilot for the duration of the flight, as required by local federal aviation policies, but was optional for passengers. They reached a maximum altitude of 5500 m in the air, but none of the passengers used supplemental oxygen or had altitude-related symptoms. The helicopter landed at higher altitude stops at Pheriche for 30 min and Everest Base Camp for 15 min.

They flew back down for a 3-h stop at Hotel Everest View, which had basic supplies for SaO_2 monitoring and a limited oxygen supply that was reserved for symptomatic visitors with low SaO_2 ($\leqslant$70%). The pulse oximeter that was used for academic interest showed that SaO_2 ranged between subjects from 79% to 90% and heart rate from 75 to 129 bpm (Table 2). The passengers were told by the local Sherpa, who was similar in age, that his normal SaO_2 was 90%, which is at the 50th percentile for acclimatized local people at that altitude.[5] The PICU physician had mild tachypnea and deep breathing but was able to walk around, jump, and take photographs, and he was told that his symptoms were typical for this trip duration and altitude. The other two passengers also were asymptomatic except for minimal tachypnea and deep breathing. They were advised that supplemental oxygen might have been required to prevent further desaturation during an overnight stay,[6] but they returned to Kathmandu after 6 h as initially planned.

Discussion

Based on the calculations with the alveolar gas equation, the PICU physician observed that the passengers probably had hypoxemia during the trip, especially at Everest Base Camp (Table 1). The low SaO_2 level experienced during the trip at high altitude was surprising to the PICU physician because it was comparable to levels that may indicate the need for respiratory support in PICU patients who have respiratory failure. When he returned to the United States and shared his experience with his PICU coworkers, they were equally surprised and unfamiliar with the distinctions between hypoxemia at high altitude versus life-threatening hypoxia. Therefore, this case may be useful in alerting clinicians about the varied responses indicated for low SaO_2 levels observed in altitude physiology versus respiratory failure.

In noncardiac PICU patients, the SaO_2 typically is maintained at >90% by giving supplemental oxygen.[7] Although the decision in the PICU to initiate mechanical ventilation is multifactorial, noncardiac shunt patients with respiratory distress or failure typically undergo endotracheal intubation and ventilation when SaO_2 is 80% to 90%, especially when SaO_2 does not increase with supplemental oxygen and noninvasive ventilation. An SaO_2 of 80% typically signifies acute, progressive, and potentially life-threatening respiratory failure in PICU patients.

In contrast, the decrease in SaO_2 despite normal lung function in the travelers at high altitude was caused by diminished barometric pressure and associated compromised oxygen supply and was compensated by an increase in heart rate and resultant cardiac output.[8] During acclimatization to high altitude, increased cardiac output maintains oxygen delivery.[9] Interindividual variations in the responses to altitude may be due to age, obesity, genetics, physical activity history, and exercise capacity.[10,11] These factors may cause varied symptoms, depending on the efficiency of adaptation to the demands for increased oxygen delivery. The passenger who best tolerated the high-altitude exposure, as evidenced by the higher SaO_2 and lower heart rate, was a lean, muscular

Table 2. Characteristics of passengers on the trip to Everest Base Camp.[a]

	Passenger no.		
	1	2	3
Characteristic			
Age (years)	42	48	51
Weight (kg)	76	104	91
Height (m)	1.67	1.83	1.74
Body mass index (kg/m^2)	27	31	30
Lifestyle history			
Previous activity level	Regular activities	Intensive physical exercise Discontinued running due to knee problems 1 year ago	Played competitive basketball until age 40 years Consistent exercise routine until 2 years ago
Work	ICU physician (sedentary, bedside rounds)	Former army officer (30 years) Currently police officer	Sales manager (sedentary)
Current exercise regimen	Walking 3–4 h/week	Regular strength training and isometric exercise	Past 2 years: biking 1 h every other day, total 3 h/week
Cardiorespiratory issues	None	None	None
Recent COVID-19 infections	None	None	None
Living altitude (m)			
Previous	1200 m	Varied altitude; during army work, 1200 m	Most of life, 350 m
Current	Sea level (past 15 years)	Near sea level (past few years)	350 m
Oxygen saturation (SaO_2) (%)	79	90	81
Heart rate (bpm)	120	75	129

ICU: intensive care unit.
[a]All three passengers were men. Oxygen saturation and heart rate were measured at Hotel Everest View (altitude, 3820 m).

police officer who had a regular exercise regimen (⩾1 h daily) (Table 2).[12] It is possible that his greater exercise capacity provided cardiovascular reserve that helped maintain his higher SaO_2 level. Individuals with higher cardiorespiratory fitness levels may have lower postoperative morbidity and mortality, indicating that cardiorespiratory reserve in patients with high levels of fitness may attenuate associated cardiorespiratory burdens of surgery.[13] Obese individuals are considered less fit and respond poorly to hypoxic exposure and hypoxic exercise, experiencing increased autonomic nervous system response with higher heart rate and blood pressure, and subsequently, acute mountain sickness.[14]

The hypoxic hyperventilatory response at high altitudes is a protective mechanism to improve arterial oxygen levels (Table 1). At 8400 m (barometric pressure, 272 mm Hg (363 hPa)), arterial blood gas measurements without supplemental oxygen may show markedly low levels of arterial partial pressure of carbon dioxide ($PaCO_2$ 13 mm Hg) and oxygen (PaO_2 25 mm Hg).[3] At the summit of Mount Everest (8848 m; barometric pressure, 250 mm Hg (333 hPa)),[15] PaO_2 may be less than 25 mm Hg without supplemental oxygen and would be insufficient to sustain life except possibly for highly trained and adapted Sherpas. On commercial airplane flights with pressurized cabins (barometric pressure, 575 mm Hg (767 hPa); equivalent to 2400 m), passengers typically have PaO_2 levels of 60 mm Hg (80 hPa) and SaO_2 90%.[16] In contrast, helicopter cabins are not pressurized, creating the potential need for acute cardiovascular compensatory mechanisms.[17]

Conclusion

In summary, the experience of the travelers at high altitude underscores the importance of the science of altitude physiology. Hypoxemia associated with high altitude, in the absence of respiratory compromise, may not necessarily require oxygen therapy. Oxygen is an important lifesaving medicine, and it is important to optimize strategies about when and how to use it at high altitudes because of limitations in availability.

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Informed consent

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