HYPERVENTILATORY CAPACITY – A PREDICTOR OF ALTITUDE SICKNESS

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Background: The aim of the study was to document link between hyperventilatory capacity and risk for developing acute mountain sickness (AMS). **Methods:** This study was carried out at Karakorum Mountain ranges (Northern areas of Pakistan) from March till July 2004. 54 healthy male subjects were enrolled in this study. Arterial oxygen saturation (SpO_2) of the subjects was measured by the pulse oximeter at rest and after 1 minute of voluntary hyperventilation at an altitude of 2833 meters. Symptoms of acute mountain sickness (AMS) were recorded on a questionnaire by using the Lake Louise consensus scoring system 24 hours after ascent to high altitude. **Results:** Mean pre hyperventilation oxygen saturation (SpO_2) was 94.07 $\pm$ 0.26% whereas SpO_2 after 01 minute of hyperventilation was 98.61 $\pm$ 0.14% that was significantly increased ($p<0.001$). The mean increase in percent oxygen saturation of hemoglobin after one minute of hyperventilation (hyperventilatory capacity) for the study group was 4.61 $\pm$ 0.24 % while the mean symptom score was 2.06 $\pm$ 0.26. It was noted that 19 (35.2%) subjects did not develop AMS whereas 34(63.0%) subjects had mild AMS and only one subject developed moderate AMS. There was no case of severe AMS. The data reveals significant ($P<0.01$) association between hyperventilatory capacity and development of the symptoms of AMS ($r= -0.664$). It is evident that individuals with greater hyperventilatory capacity manifest less number of symptoms of mountain sickness. **Conclusion:** It is concluded that post hyperventilation increase in oxygen saturation at lower altitude may help to predict the susceptibility of subjects to develop high altitude sickness.

Keywords: Acute Mountain Sickness, Hyperventilatory Capacity, Hypoxia

INTRODUCTION

The quest to predict the development of acute mountain sickness (AMS) in individuals ascending to high altitude is still unresolved. It is not uncommon to anticipate the development of altitude illness in travelers ascending to altitudes higher than 2500 meters even though many of them will develop mild and self limiting mountain sickness that may not require any medical intervention. However, altitude illness may rarely progress to more severe forms which can be life threatening. The Acute mountain sickness is a clinical syndrome comprising of headache, nausea, vomiting and lack of sleep.[1] The precise pathophysiology of AMS is not known, however, the primary insult delivered by the high altitude is hypoxia which results in decrease in oxygen tension in lungs and in blood.[2] Consequently hypoxemia develops that has been considered as the crucial mechanism for the development of AMS. The residents of high altitude usually perform physically better than the lowlanders on exposure to high altitude particularly on exertion[3] because of genetic adaptation during development.[4]

Acute adaptation to high altitude is primarily manifested by an increase in respiratory rate which basically helps to improve oxygen saturation of the blood. In some studies attempt has been made to establish a link between hyperventilatory capacity and altitude sickness.[4,5] Currently no measurement reliably predicts susceptibility to AMS before ascent to high altitude. However, it has been documented that SpO_2 is unrelated to AMS at moderate altitudes of around 3000 meters[6] whereas at greater altitude >4000 meters SpO_2 seems to be useful in detecting subjects who are highly susceptible to AMS.[7] Therefore, an effort has been made to see whether levels of post hyperventilation SpO_2 below 3000 meters altitude might instead predict future development of AMS during ascend to higher altitudes.

MATERIAL AND METHODS

Fifty four healthy male subjects participated in this study who were randomly selected from the individuals who had recently arrived at 2833 meters altitude and had no previous experience of ascent to high altitude. Fifty three subjects were residents of altitude less than 1000 meters, while one subject was native of Gilgit, (altitude 1700 meters) northern areas of Pakistan. Their age ranged between 21 and 39 years (Mean age 30.91 $\pm$ 0.72 years). After obtaining informed consent we carried out their clinical examination. All of them were physically fit and not suffering from any acute or chronic disease.

Acclimatization: The subjects traveled by road from Rawalpindi (515 meters) to an altitude 2500 meters (Skardu) in three days where they stayed for 5days. Subsequently they ascended to an altitude of 2833 meters in one day and stayed there for 10 days. The subjects then ascended to their respective altitudes.

Pulse oximetry: All the subjects were seated comfortably .The procedure of pulse oximetry was explained to all the subjects in the language they understood and was demonstrated physically. Prior to pulse oximetry measurement, the oximeter was calibrated, the index finger was cleaned with alcohol swab and the reading was carried out with the subject at rest. Oxygen saturation of blood (SpO_2) was measured (in the evening), by using the portable pulse oximeter after the subject had rested for 30 minutes at room temperature of 20 degrees Celsius. The pulse oximetry was rechecked after 01 minute of hyperventilation in each subject who were asked to breathe deep and fast for 01 minute while they were seated comfortably.

Altitude ascended: The subjects were then randomly assigned to various altitudes for acute ascent. Mean altitude to which they ascended was 5768 meter (range 5166 - 6500 meters.). The data was stored and analyzed on the spss 11 version.

AMS scoring: Symptoms were recorded 24 hours after ascent on a questionnaire by using Lake Louise consensus scoring system[1]. In this system ,five symptom categories (headache, gastrointestinal upset, fatigue, dizziness, and sleep disturbance) are assessed on a score scale 0-3 where 0 for no symptom and 1, 2,3, for mild, moderate and severe symptoms. The magnitude of AMS was assessed on the following scale:-

1. No AMS 0
2. Mild AMS 1-5
3. Moderate AMS 6-9
4. Severe AMS >10

RESULTS

Fifty four subjects were enrolled in this study and their parameters are given in (table-1). 19 (35.2 %) subjects did not develop AMS while 34 (63.0 %) subjects developed mild AMS and only one subject developed moderate AMS.

The data was analyzed on the basis of hyperventilatory capacity of the subjects. Mean hyperventilatory capacity for the study group was 4.61 $\pm$ 0.244 % that was the difference between mean oxygen saturation of the subjects before and after 1 minute of hyperventilation ($p<0.001$). Mean symptom score for the study group was 2.06 $\pm$ 0.26.There was significant inverse correlation between hyperventilatory capacity and symptom score ($r= -0.664$, $p < 0.01$) as presented in figure 1.

Three groups were made on the basis of percentage rise in oxygen saturation after 1 minute hyperventilation with the difference of 03% in each group (table-2). The group of subjects (n=17) who had the lowest increase in oxygen saturation (0-3%) developed the maximum symptoms score (mean 3.71$\pm$0.40). The group of individuals (n=5) with the highest increase in oxygen saturation (7-9%) manifested the lowest symptoms score (mean 20$\pm$0.20).

Mean altitude ascended was 5771.41$\pm$71.93 meters. Sixteen subjects ascended 6500 meters, 12 subjects 6000 meters, 12 subjects 5500 meters while 14 subjects ascended 5166 meters (table-3). There was direct correlation between symptoms score and altitude ascended ($r= 0.301$, $p = 0.027$) as highlighted in figure-2. The group (n=16) who ascended to the maximum altitude (6500meters) had the maximum number of cases of mild AMS and less number of cases of no AMS. This group had the highest increase in hyperventilatory capacity (mean 4.78$\pm$0.60) with the mean symptoms score of 2.71$\pm$0.44. The subjects (n=14) who ascended to the altitude of 5166 meters, had the maximum number (n=10) of cases of no AMS and their symptoms score was the lowest (mean 2.00$\pm$0.40) as presented in table-3.

There was only one subject (native of Gilgit) who had more than 10 % increase in oxygen saturation and remained symptoms free. One subject had 8 % increases in oxygen saturation after 1 minute of hyperventilation but developed moderate symptoms of AMS and was the exception to our results.

Table-1: Mean parameters of the study group (n = 54)

Parameters	Mean	$\pm$ SEM
Age (years)	30.91	0.72
Pre hyperventilation oxygen saturation (%)	94.07%	0.26
Post hyperventilation oxygen saturation (%)	98.61%	0.14
Mean hyperventilatory capacity (%)	4.61%	0.244
Mean symptom score	2.06	0.26
Mean altitude ascended (meters)	5771.41	71.93

Table-2: Comparison of Ventilatory Capacity and Development of AMS (n=54)

% rise in oxygen capacity	No of cases	No AMS	Mild AMS	Moderate AMS	Mean symptom score ± SEM
0-3 %	17	01	15	01	3.71±0.40
4- 6%	32	14	18	0	1.47±0.27
7-9%	5	04	01	0	0.20±.20

Table-3: Altitude Ascended and Development of AMS Symptoms

Altitude (meters)	No. of Cases	No AMS	Mild AMS	Moderate AMS	Mean % rise in O_2	Mean symptom score± SEM
6500	16	4	9	1	4.78±0.60	2.71±0.44
6000	12	3	9	0	4.75±0.45	2.42±08
5500	12	6	6	0	4.75±0.60	1.83±0.63
5166	14	6	10	0	4.25±0.38	1.38±0.40

Fig.1: Association between hyperventilatory capacity and symptom score

Fig.2: Association between altitude ascended and symptom score

DISCUSSION

In normobaric conditions hyperventilation causes increase in percent saturation of haemoglobin in arterial blood (SpO_2). Nevertheless there is a variable increase in SpO_2 in different individuals on hyperventilation. We hypothesized that reduced increase in SpO_2 on hyperventilation might predict the predisposition of the development to acute mountain sickness (AMS).

This study analyzed the predictive value of percentage increase in oxygen saturation after one minute of voluntary hyperventilation.[8] The maximum symptom score (mean 3.71±0.58) was recorded in the study group (n=17) with the lowest range of increase in percent SpO_2, that is; 0-3% and the lowest AMS symptoms score (0.20±0.20) was recorded in the range of 7-9% of SpO_2 in our study (n=5). Hyperventilation is the first physiological adjustment that takes place in response to sudden exposure to hypoxia and this adaptive pulmonary mechanism[9] helps to improve the levels of PaO_2 and SpO_2. Therefore, the volunteers who manifest lower hyperventilatory capacity on exposure to high altitude are likely to suffer more from hypoxia. These individuals will therefore develop more symptoms of acute mountain sickness as in the present study.

The highest mean symptoms score (2.71±0.44) was recorded in the group of subjects (n-16) who ascended to the highest altitude of 6500 meters in our study. In spite of the fact that this group had the highest increase in hyperventilatory capacity (mean SpO_2=4.78+0.60), they still developed maximum number of cases of mild AMS, for unknown reasons. Since hypoxia is primarily responsible for the development of AMS, therefore hyperventilatory response on exposure to hypobaric hypoxia at 6500 meters may be disproportionate alongwith individual differences in tissue oxygenation that might partly explain the development of AMS.[10] We could not measure SpO_2 at 6500 meters altitude but decrease in ventilation due to the decrease in central chemoreceptor drive[9] could be the reason for the effects of hypoxia at that altitude and development of symptoms.

Lowest mean symptom score (1.38±0.44) was recorded in the group of subjects (n=14) that ascended to the altitude of 5166 meters. Their mean increase in SpO_2 on hyperventilation was 4.25±0.38% which was statistically very close to the subjects ascending to 6500 meter altitude. The hypoxic ventilatory response at 5166 meter altitude was probably adequate and transport of O_2 to the

tissues was optimum that saved the subjects to suffer from AMS.

One individual manifested 8 % increase in post hyperventilation oxygen saturation but still developed moderate symptoms of AMS. He was an exception to our study who despite having good hyperventilatory capacity developed moderate AMS. The possible reason could be that AMS susceptibility might change due to various factors such as rate of ascent or fluid intake.[11] It was later on found that he ascended too rapidly. Another individual had low resting oxygen to start with but had more than 10% rise in SpO_2 after 1 minute of hyperventilation. He was the resident of northern area who did not develop symptoms of AMS and did well at high altitude possibly due to genetic adaptation.[4] It is therefore evident that the subjects with greater hyperventilatory capacity perform better at high altitude than the subjects manifesting lower hyperventilatory capacity. Nevertheless, with exceptions one may be able to predict the vulnerability of individuals to develop AMS at high altitude on the basis of percent increase in SpO_2 after one minute of voluntary hyperventilation at lower altitude before ascent.

CONCLUSION

The study concludes that magnitude of post hyperventilation increase in oxygen saturation at lower altitude may help to predict the susceptibility of subjects to develop AMS.

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