

“SUBCLINICAL HIGH ALTITUDE PULMONARY EDEMA (HAPO): AN ESTABLISHED CLINICAL ENTITY.”

Sports Science

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ABSTRACT

Introduction: High Altitude pulmonary edema (HAPO) is a serious and early sign of mountain sickness, which develops in individuals who rapidly ascend to an altitude above 3000 meters. Troops including aircrew and support ground staff, when deployed at high altitudes are often air transported and hence are at a risk of developing HAPO. The incidence of HAPO diagnosed clinically is 1.5-5.5%.¹ However, the incidence of HAPO rises sharply when chest radiography is used as a screening tool in these subjects who are clinically asymptomatic.

Purpose of study: The study intends to focus on identifying personnel at risk and screen them for same using a chest radiograph.

Material and methods: A total of 50 young healthy troops on arrival at a high-altitude base (3300 m) were selected for the study. All of them were examined immediately on arrival and on day 3. The clinical assessment was made using Lake lousie questionnaire, ECG, echocardiography, pulmonary function study and hematological investigations followed by chest x-ray.

Results: None of the patients developed a clinical evidence of HAPO but all patients had pulmonary parameters consistent with process of acclimatization. Two of these patients developed a subclinical form of HAPO and they were screened using chest x-rays. Peri-hilar haziness, perivascular cuffing & interstitial thickening were very well appreciated. Echocardiography revealed a higher pulmonary arterial pressure. These patients on follow-up x-rays were consistent with HAPO.

Conclusion: The study indicates that subclinical form of HAPO is actually important to identify to prevent complications and also to aggressively treat it. Chest X-rays provide a cheap and easily available modality for detecting the same.

KEYWORDS

Exercise Physiology, pulmonary edema, Sub-clinical, High-altitude, Low-lander.

Introduction:

On ascent to high altitude the low land resident experiences a number of physiological and biochemical responses in an attempt to compensate for the decrease in atmospheric partial pressure of oxygen. It is a well-known fact that individuals who ascend rapidly to altitudes above 3000 meters develop various symptoms and signs. In most people, this condition is more in the nature of inconvenience rather than an illness.¹ But in a minority of cases it rapidly escalates to high altitude pulmonary edema, a serious life-threatening condition. The incidence of HAPO depends on the method used to detect the condition and rises sharply when chest radiography is used as a screening tool in these subjects who are clinically asymptomatic. This study is an attempt to discover the sub-clinical form of pulmonary edema in asymptomatic subjects acclimatizing to high altitude.

Material & methods

Prospective trial conducted at High Altitude Medical Research Centre (HAMRC) included 50 soldiers (sea level residents) without previous HA exposure volunteered as subjects. None of the included patients had a history of any previous hospitalization, chronic medication intake or any recent illness.

All patients were assessed on next day to their arrival (Day 1) and after 72 hours again (Day 3). The ambient temperature varied from 5 degrees to 25 degrees. The Lake-Lousie questionnaire was used to diagnose acute mountain sickness.² Subjects scoring less than three in lake lousie consensus score i.e. symptoms not amounting to be classified as Acute mountain sickness (AMS), were included in the study group. Vital charting along with basic hematological examination was done along with ECG, PFT, Chest X-ray & Echocardiography was done.

Results:

The mean age of study group was 23.3 years, 2.1 years, mean height 166.55.4 cm and a mean weight of 56.24.7 kg. Vitals did not change significantly on day 1 but there was a marked decrease in heart rate and marginal reduction in mean respiratory rate on day 3. The patients with a subclinical HAPO had a higher oral temperature as compared to the cohort but finding was statistically insignificant. AMS scores using Lake-lousie questionnaire were significantly greater on day

1 (mean=4.76) and reduced on day 3 (mean=2.82). SAO₂ was significantly elevated on day 3 ($p < 0.002$) in comparison to day 1. Seven patients had markedly high lake lousie scores (mean=6.13) and their chest radiographs revealed a thickened pleura, perivascular cuffing, peri-hilar zone haziness and thickened interstitium on day three. Patients with positive findings and clinical respiratory distress with deterioration of SAO₂, less than 80 (5%) were hospitalized and managed as HAPE. All routine hematological examination was within normal limits.

PFT showed no significant difference between day 1 and day 3 with respect to FVC, FEV₁, FEV_{25%-75%}. There was a significant improvement in flow rates on day 3. Acceptable data for PFT could only be obtained in 38 (76%) patients. No patient showed any evidence of Right axis deviation and/or T wave inversion on day 1 whereas 3 subjects (6%) had right axis deviation on day 3. There were 5 patients showing a wide QRS complex on day 3. The PFT revealed an increase in the FVC, FEV₁, MMF on day 3 in comparison to day 1 but the difference was insignificant. Four patients did not improve on the foresaid parameters, this was probably due to obstructive dysfunction. Pulmonary arterial pressure was measured using echocardiography. The parameters recorded were mean pre-ejection period (PEP), Mean acceleration time (MAT) and mean right ventricular ejection time (RVET). PEP/AT ratios were calculated. Four patients (8%) showed a significant increase in pulmonary arterial pressure.

Discussion:

HAPE is a well-known entity which requires emergent management. This usually occurs in severe form typically in subjects gaining heights rapidly. There is always a higher chance in air-inductees than road inductees. This has been observed earlier by many workers. Reports from the literature suggest that in soldiers stationed at high altitudes, trekkers, climbers and residents of high altitude returning home after a period of stay at low altitude the incidence of lung edema is 1.5-5.5%.

However, since the clinical signs and symptoms are the late manifestation of an increased accumulation of fluid in lungs, it is suggested that there is a greater incidence of HAPO than suggested by literature which is of a severity insufficient to cause symptoms. Hultgren and Multicorena investigated 97 residents of La Oroya in

central Peru by means of questionnaire and concluded that the incidence including mild cases was much higher (6.1%).^{3,4} Scoggin et al were of the view that the subclinical form of pulmonary edema was at least three times as common as the well-defined fully developed form.⁵

The incidence of HAPO rises sharply when the sub clinical cases of diagnosed by radiography. When Vock et. al studied chest radiographs of subjects, without history of a previous attack of HAPO within a day of ascent to an altitude of 4550m he found that the incidence to be 12.5%. Gray measured the washout of a single breath of nitrogen in 17 subjects during the exposure to about 4500m.⁶ There was significant increase in the slope of the plateau in these subjects. This indicated that exposure to HA produced an intra-regional maldistribution of ventilation, which could have been due to an increase in the volume of interstitial fluid. The definition of sub-clinical HAPO is the presence of a disease. The subclinical is the presence of a disease without manifestation of symptoms and may be in the early stage of evolution of the disease. The sub-clinical disease does not have any manifestation or symptoms and does not warrant treatment. It has been further mentioned by Vock et al that when the radiological examination for pulmonary edema was carried out in subjects who had a previous attack of subclinical HAPO, the incidence of lung edema increased to as high as 66%.⁶ This study is an attempt to discover the sub-clinical form of pulmonary edema in asymptomatic subjects acclimatizing to high altitude.

Chest X-ray, PFT and Echocardiography form the most important non-invasive modalities with minimal radiation exposure. Increased pulmonary pressure is a possible precursor of lung edema. It is known that the exposure to hypoxia results in increase in pulmonary arterial pressure consequent to hypoxic vasoconstriction in pulmonary vascular bed. Echocardiography & Chest X-ray is used to indirectly assess this rise in pulmonary arterial pressure. The reduction in Lake-Louis on day three in majority of patients revealed acclimatization changes whereas the patient with increasing lake lous scores were managed on protocol of HAPE. Hematological examination revealed an increased hematocrit & hemoglobin. The possible reason to which is haemo-concentration and in part also due to dehydration. The change when followed for two-week time was found to result in statistically significant difference in both values. Arterial oxygen saturation values were significantly greater on day 3 in comparison to day 1. These improvement in AMS scores and oxygen saturation were probably a result of cardiovascular and respiratory adjustments. Mean heart rate was higher on day 1 as compared to day 3. However, it was statistically not significant and likely due to increased sympathetic tone in response to hypoxic stress.

Obstruction can involve small airways contributed by reduced compliance of lungs because of hypoxic pulmonary arterial hypertension, which subsequently leads to interstitial edema. Previous studies have demonstrated similar findings due to increase in airway resistance or reduction in elastic recoil of lung due to presence of edema fluid in bronchial wall.

ECG changes in these patients have been variable and inconsistent. The changes seen may include T wave inversions, right axis deviation, wide QRS complexes and features of right sided overload. Our patients demonstrated a wide QRS complex in 5 cases possibly due to increased workload on the right ventricle. No patients demonstrated a T wave inversion.

Pulmonary arterial pressure was assessed by two-dimensional echocardiography and pulsed Doppler in four subjects. Four subjects had marked increase in the pulmonary arterial pressure on day 1 as well as day 3. It has demonstrated that echocardiographic evidence of pulmonary arterial hypertension can be found in individuals. Echocardiography gives a two-dimensional indirect measure of the PAP and so it is not a reliable method for obtaining accurate values, but it is a reliable indicator during hypoxic stress. Radiographic evidence of edematous pulmonary interstitium is the first sign of interstitial edema and is quite sensitive. The total radiation exposure during the study to one subject was about 0.1 rads. This was well under the defined acceptable limit of 5 rads/year/individual.

The major limitation of the study was the small cohort and a small follow up. Larger multi-centric trials with long term follow up might help us to better know the exercise physiology and its effect on the

course of HAPO. Extrapolation of results to normal athletes may not be a true representation of the population.

Conclusion:

The study indicates that subclinical form of HAPO is actually important to identify to prevent complications and also to aggressively treat it. Chest X-rays provide a cheap and easily available modality for detecting the same.

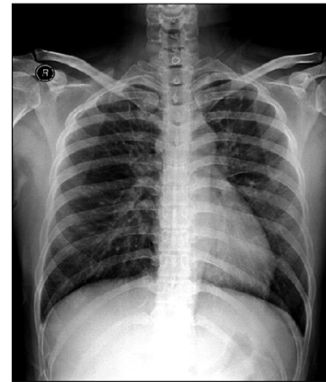


Figure 1: Chest radiograph of Case 1 with sub-clinical HAPO.



Figure 2: Chest radiograph of Case 2 with sub-clinical HAPO.



Figure 3: Chest radiograph of Case 3 with sub-clinical HAPO.



Figure 4: Chest radiograph of Case 4 with sub-clinical HAPO.

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