HIGH ALTITUDE PULMONARY OEDEMA - AN EXPERIENCE IN EASTERN HIMALAYA

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ABSTRACT

Three hundred and five cases of high altitude pulmonary oedema (HAPO) hospitalised in eastern Himalayan region have been analyzed. Incidence of HAPO was 5.5 per cent. Eighty per cent cases occurred during latter half of the year. Fifty six per cent of cases belonged to the third decade of life. HAPO cases occurred most commonly between the height of 2740 m to 5960 m. Eighty three per cent cases developed symptoms within 72 hours of induction to high altitude and 65.9 per cent suffered from the illness despite complete acclimatization. Breathlessness, headache and cough were the commonest symptoms. Tachycardia and tachypnoea was present in all cases. Twenty five per cent cases showed various ECG abnormalities. Mortality rate was 0.98 per cent.

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KEYWORDS: Altitude; ECG abnormalities; Cerebral oedema; Pulmonary oedema.

Introduction

High altitude pulmonary oedema (HAPO) is the commonest medical emergency seen at high altitude (2740 m and above) and has been known for over a century. Mountaineers having heart failure, pneumonia of lungs, and double pneumonia have been recognized, but in fact they were all cases of HAPO. While Houston [1] described the first case of HAPO in 1960, early sixties saw the beginning of a deluge of HAPO cases. The Indian experience began in 1962 during the Indo-China conflict, resulting in two series of 101 and 332 cases of HAPO from Western Himalaya [2,3]. Two reports from Eastern Himalaya reviewed 85 and 55 cases respectively in early 80s [4,5]. The present study pertains to a series of 305 cases of HAPO in Eastern Himalaya.

Material and Methods

The study was carried out at a hospital located at 1830 m (6000 feet). The hospital received patients from a wide area of mountainous territory ranging from 2740 m to 6000 m. Most of the patients had lived all their lives in plains (lowlanders) and were inducted to high altitude for short durations. Hospital admissions of these individuals over a period of 3 years (1988-1990) have been included. The following criteria were taken as a

basis for the diagnosis of pulmonary oedema: cough, breathlessness at rest, chest discomfort and presence of pulmonary crepitations. Myocardial infarction and other causes of pulmonary oedema were excluded clinically and electrocardiographically. The diagnosis was confirmed radiologically in all cases. Chest radiography was repeated every 3rd day till complete radiological clearance was seen. The following investigations were done - haemoglobin (Hb), total and differential white cell count, urinalysis, chest radiography and ECG.

Observations

Incidence - High altitude pulmonary oedema (HAPO) was the most common life-threatening emergency seen, constituting about 5.5 per cent of all admissions in the medical ward of the hospital.

Month of onset - Onset of pulmonary oedema in various months of the year is depicted in Table 1. Most of the cases of HAPO occurred in the latter half of the year. Maximum cases (56) were seen in the month of October.

Age and sex - The age of the patients varied between 20 to 56 years. The maximum number of cases 171 (56.1%) were in third decade. While 91 cases were in age group of 31 to 40 years, 42 patients were in their fifth decade of life.

Altitude of onset - The lowest altitude where a

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TABLE 1
Month of onset of high altitude pulmonary oedema

Year	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec	Total
1988	2	2	6	5	1	4	5	9	17	16	15	4	86
1989	2	1	2	16	2	17	6	12	7	35	17	3	120
1990	4	0	5	2	10	23	17	9	5	5	4	15	99
Total	8	3	13	23	13	44	28	30	29	56	36	22	305

TABLE 2
Height of occurrence of HAPO

Height	2740 - 3050m	3050 - 3660m	3660 - 4270m	4270 - 4880m	4880 - 6000m
No of cases	33 (10.8%)	84 (27.5%)	63 (20.6%)	65 (21.3%)	60 (19.7%)

case of HAPO occurred was 2740 m and the maximum was at 5960 m. Majority of the cases (61.6%) occurred at a height beyond 3660 m (Table 2).

Induction to high altitude - All the 305 cases were divided into groups as per their induction to high altitude. Fresh inductees (entering high altitude for the first time) comprised 181 (59.3%) cases. Patients who had spent several months at high altitude and were re-entering the area after a varying period of stay in the plains were called the re-inductees and comprised of 120 cases (39.3%).

The duration of reinductee at plains varied between 18 to 408 days. Four patients (1.3%) developed HAPO on induction to higher altitude even after a stay at high altitude for 4-6 months.

Acclimatisation and precipitating causes - All inductees to high altitude travelled by road from plains, and underwent acclimatization as prescribed. Two hundred one patients (65.9%) developed HAPO, despite complete acclimatisation. While 84 cases (21%) who could not complete acclimatisation developed HAPO, 20 cases (6.6%) developed the illness while undergoing acclimatisation at various stages.

Eleven (3.6%) patients had some underlying illness before developing HAPO. While 7 cases (2.2%) had respiratory infection, 2 patients had malarial parasite and one patient each had viral hepatitis and acute gastritis.

Onset of illness - The onset of HAPO in majority of cases was sudden with cough, pain chest and

dyspnoea. A full-fledged picture of HAPO was visible within a few hours. While 70 cases (23%) had symptoms within 6 hours, the number increased to 177 (58.0%) within 24 hours and 253 (83.0%) cases developed HAPO within 72 hours of arrival. Four patients already in high altitude, developed HAPO within 96 hours after being sent to higher altitude.

Symptoms and signs - Table 3 shows symptoms and signs in HAPO cases. When seen for the first time, most of the patients (94.8%) were conscious. Only 3 patients were in deep coma.

Investigations - Haemoglobin was between 12 g/dL to 16 g/dL in most of the cases. Only 10 cases had Hb value of over 16 g/dL. The total leucocytic count ranged between 7200 to 19600/cumm. One hundred and twenty patients (39.3%) had counts of over 15000/cumm, while a total of 242 cases (73.3%) had counts over 12000/cumm. In most of these patients leucocytosis was polymorphonuclear variety, with no change in other cell components. Urinalysis was normal in all except 22 cases (7.2%) who showed mild proteinuria. Leucocytosis and proteinuria reverted to normal with recovery from HAPO in all cases. Chest radiography was confirmatory of the diagnosis of HAPO. To begin with right-sided non-homogenous opacity over mid zone was the commonest which gradually progressed to involve lower zone also. Twelve cases (3.9%) showed only left-sided unilateral opacities and 20 cases (6.6%) had unilateral right sided opacities till recovery. Rest of the cases had bilateral fluffy opacities of

TABLE 3

Symptoms and signs of HAPO

Symptoms	Frequency (%)		Signs	Frequency (%)	
Headache	(280)	91.8	Tachycardia	(305)	100.0
Cough	(278)	91.4	Pulse 80-100/min	(64)	20.9
Breathlessness	(277)	90.8	100-120/min	(188)	61.6
Frothy-expectoration	(210)	68.9	120 - 140/min	(57)	15.4
			> 140/min	(6)	1.9
Pain chest	(173)	56.7	Tachypnoea	(305)	100.0
Haemoptysis	(125)	40.9	Resp 20-30/min	(133)	43.6
Fever	(120)	39.4	> 30/min	(171)	56.3
Lack of sleep	(112)	36.7	Blood Pressure	—	—
Vomiting	(104)	34.4	Systolic > 140 mmHg	(24)	7.9
Loss of appetite	(44)	14.3	Diastolic < 60 mmHg	(26)	8.5
Giddiness	(43)	14.1	Diastolic > 100 mmHg	(12)	3.8
Bodyache	(40)	13.1	Fever	(144)	47.2
Weakness	(40)	13.1	Cyanosis	(40)	13.1
Palpitation	(30)	9.8	Mental confusion	(13)	4.3
Epigastric pain	(16)	5.2	Coarse crepts	(305)	100.0
Oliguria	(14)	4.6	Only right side crepts	(22)	7.2
Dyspepsia	(12)	3.9	Rhonchi	(6)	2.0
Nausea	(11)	3.6	Loud P2	(189)	62.0

various densities. The radiological clearance of opacities was seen in 3-5 days except 6 cases who recovered in 20 to 32 days.

No cardiac abnormality was detected radiologically in any case. Electrocardiographic (ECG) changes in 78 cases showed abnormalities. ECG was repeated every 72 hours till full recovery. The ECG abnormalities included sinus tachycardia, ST segment abnormality, symmetrical T wave inversion and right axis deviation. ST elevation of 2.5 mm was seen in two cases over anterolateral leads, while significant ST segment depression was seen in 5 cases over inferior and lateral wall leads. These changes reverted to normal in 3-9 days (average 5 days). A total of 65 patients (21.3%) showed T wave inversion. Of these, 10 cases had T inversion over limb leads only, 44 patients over chest leads only (usually V1-V4) while 11 cases had T inversion over both limb and chest leads. These changes reverted to normal in 12 to 76 days.

Response to therapy - Most of the cases (97.7%) were evacuated to the hospital by road, and only 7 cases were air evacuated. Response to evacuation to lower altitude was dramatic with clinical and radiological clearance occurring in 1-3

days. The recovery was usually in sequence of physical well-being, reduction in severity of symptoms, decrease in heart rate, lung clearance and radiological clearance. The average duration of hospitalization was 7.14 days with a range of 5-56 days. Of the 305 cases treated 7 cases were hospitalized with severe peripheral circulatory failure which responded well to vasopressor therapy. Three patient had associated high altitude cerebral oedema (HACO) and none of them could be saved giving a mortality rate of 0.98 per cent. Most of the cases were fit enough to resume duties after full recovery. Only 26 (8.5%) cases showing delayed recovery and prolonged ECG changes were not inducted back to high altitude area (HAA). Follow-up of rest of the cases inducted back to HAA showed that 3 of these cases suffered from ill effects of high altitude again.

Discussion

There are 3 major acute clinical manifestations of illness at high altitude - acute mountain sickness (AMS), high altitude pulmonary oedema (HAPO) and high altitude cerebral oedema (HACO) [6]. The prevalence of HAPO as reported by various

workers ranges from 0.24 per cent to 15 per cent [2,3,7] in both air and road inductees. The incidence in troops inducted by air was 0.33 per cent to 0.57 per cent [2,7] as compared to 0.07 per cent by road [7]. This is due to fact that there is sudden change in altitude and temperature on induction by air, predisposing the individual to HAPO. Another study has reported a cumulative incidence of 1.42 per 1000 inductions to high altitude in western Himalaya [8]. Incidence of HAPO from eastern Himalaya has not been recorded earlier [4,5]. The incidence of 5.5 per cent in this study is, where 97.7 per cent of the troops were inducted by road. The higher incidence could be due to physical fatigue of journey, exposure to cold and sudden physical exertion on arrival at high altitude [8,9]. Bhalwar [8] has observed that the main risk of developing HAPO due to physical exertion was during the first 24 hours of entry to high altitude. Majority of the cases (80%) were noticed during June to December. Similar seasonal variation has been noticed in one study [7], though not by others [2,5]. Majority (56.1%) of the cases were in their third decade of life, as observed by other workers also [2,5,7,10]. The preponderance of young population only reflects the exposed population, though no age seem to be exempt from developing HAPO. All the studies have a male bias due to the population studied and the sex ratio does not reflect the true incidence amongst female population. No caste or tribe predisposition was seen amongst the cases.

The altitude of onset of illness ranged from 2740 m to 5960 m. One case was reported from 2740 m which is beginning of high altitude by definition. No particular height was vulnerable (Table 2), though 61.6 per cent of the cases suffered from HAPO at the height beyond 3660 m. Incidence of 45-81 per cent of cases from a height of less than 3660 m has been reported [2,5,7]. The incidence of HAPO at extreme altitude of 2 per cent and 5 per cent was reported [2,7] as compared to 20.9 per cent in the present study. This gross variance could be due to the number of vulnerable population deployed at such heights. The occurrence of HAPO below 2750 m is extremely rare and raises the suspicion of pre-existing anomaly in the pulmonary circulation [11-13].

Fresh inductees to high altitude comprised ma-

jority (41.6%-75.0%) of the cases of HAPO [3,4,5,10], as also in the present study (59.3%). This is in gross variance with some earlier studies who found reinductees to form the majority (64.3% and 85%) of cases of HAPO [2,7], while Bhalwar [8] did not notice any association with inductee status.

The incidence of HAPO in persons staying at high altitude for a varying period of 4-6 months and moving to further higher altitude comprised 7.9 per cent of the cases [2] as compared to 1.3 per cent in the present study. In both the studies, almost half of these cases had severe physical exertion on arrival at higher altitude, while in others individuals susceptibility and extreme cold could be responsible [7,9].

HAPO was noticed in 65.9 per cent of fully acclimatised cases which is similar to the reported figure of 63.5 per cent from the same region [4], as compared to 11.9 per cent to 18.2 per cent [2,5]. While 73.6 per cent cases developed HAPO during acclimatisation [7], incidence of only 13.1 per cent was seen in this study, and 21.8 per cent by another [5]. The higher incidence [7] could be due to the fact that initial acclimatisation there was carried out at a much higher altitude (3380 m) on rapid ascent by air, as compared to 2740 m on slow induction by road [5]. However, HAPO has also been reported in the individuals moving from higher to lower altitude [3].

Seven cases (2.2%) gave history of preceding respiratory infection in the present study as compared to 7.8 per cent [7]. One hundred seventy seven cases (58.0%) developed HAPO within 24 hours of induction to high altitude as compared to 52.4 per cent to 61.8 per cent [4,7]. Seventy patients (22.9%) reported with symptoms of HAPO within 6 hours in the present study, which is similar to another observation [7]. Onset of HAPO within 72 hours of arrival at high altitude varies between 51.8 per cent to 90 per cent [2,4,5,7,8,10] as also 82.5 per cent in the present study. While no case developed HAPO after 72 hours in the present study, earlier observation are of great variance of 1 per cent to 37.3 per cent [3,5,7,10].

The first symptom was usually headache (91.8%), dry cough (91.4%) and progressive breathlessness (90.8%). Frothy pink expectoration

characteristic of HAPO was present in only 68.9 per cent cases, as compared to 5.5 per cent to 39 per cent [2,5,7,10]. Altered level of consciousness was seen in 12 (3.3%) case, while 3.6 per cent to 4.9 per cent by others [2,4,5,7]. Cyanosis was noticed in 13.1 per cent cases as compared to 5.5 per cent to 51.5 per cent [2,5,7]. Persistence of cyanosis suggests a poor prognosis. A total of 47.2 per cent patients were febrile on admission as compared to 20 per cent to 73.3 per cent [3,5,7]. Tachycardia was often disproportionate to the fever and tachypnea. Blood pressure (BP) recorded was usually normal or lower side of normal in all cases. With recovery, there was distinct rise of BP indicating improved myocardial efficiency. Persistence of hypotension was a bad prognostic indicator. While most of the cases had bilateral crepitations over the lungs, only 22 cases (7.2%) had unilateral crepitations at the time of hospitalization. Bronchospasm was present in only 6 cases (1.9%). Abnormal investigations were seen in form of leucocytosis (73.3%). This was neither related to clinical severity nor to evidence of infection. This observation [2,5,7] varies between 15 per cent to 78.3 per cent, while denied by other [3]. High Hb value (16 g/dL or more) observed in 10 cases could be related to polycythemia of high altitude. Presence of mild proteinuria seen in 7.2 per cent cases has not been recorded by any other study. However a specific study [10] reported high incidence of proteinuria to hypoxia of high altitude.

The nature, extent and density of radiological opacities is variable. Only 10.5 per cent cases showed unilateral lung opacities (right>left) as compared to 5.9 per cent [4]. The ECG changes as also observed earlier [2,3,5,7,15] were seen in 25.5 per cent cases. These changes were transient and reverted to normal in an average of 2 weeks.

Even in the presence of leucocytosis, antibiotics were not used and count came to normal with treatment of HAPO. Presence of altered sensorium and HAPO warranted use of cerebral decongestants. Out of the 305 cases treated, only three patients (0.98%) died as compared to 0 per cent to 7.2 per cent [2-5,7]. All these patients had associated HACO, which was confirmed histopathologically.

The pathophysiology of HAPO is unclear, but hypoxia induced raised pulmonary artery pressure (PAP) is a cardinal feature, whereas pulmonary capillary wedge pressure remains normal [16]. Persons who are prone to the condition have higher PAP at rest and in response to hypoxia, altitude and exercise than those who are resistant to it. Inhalation of nitric oxide is reported to be beneficial in subjects prone to HAPO by reducing PAP [17]. Oelz [18] and Bartsch [19] have demonstrated protective and therapeutic effects of lowering PAP with nifedipine without impairing performance in climbers prone to HAPO. This preventive aspect of this deadly disease requires further research and shows a faint ray of hope in man's fight against nature.

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