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Dynamic ECG Changes in a Patient with High Altitude Pulmonary Edema

Taruna Ahuja[1], Himadri Shekhar Dey[2], Sachin Dutt[3], Danesh Kumar Dewangan[4]

[1]Physician, MH Kargil (Corresponding Author)

[2, 3]Medical Officer, MH Kargil

[4]Commanding Officer, MH Kargil

Abstract: ***High Altitude Pulmonary Edema (HAPE) is a severe and potentially fatal condition affecting travellers and military personnel at high elevations. It primarily results from hypoxic pulmonary vasoconstriction leading to increased pulmonary artery pressure. This case report describes a 30 - year - old male who developed HAPE after a rapid ascent to 3500m and subsequent descent to 2676m. Despite clinical improvement with oxygen therapy and medication, dynamic electrocardiogram (ECG) changes suggestive of right ventricular strain were observed. The findings highlight the potential cardiac involvement in HAPE, which can complicate diagnosis and necessitate extensive cardiac evaluations. Understanding such ECG changes can aid clinicians in distinguishing HAPE from other cardiac emergencies, ensuring timely and appropriate management while avoiding unnecessary resource utilization.***

Keywords: High Altitude Pulmonary Edema, ECG changes, right ventricular strain, pulmonary hypertension, hypoxic vasoconstriction

1. Introduction

Travelers to high - altitude areas have increased exponentially over the past few years. Many major armed forces in the world occupy areas that are well above sea level. Medical complications in travellers to high altitude areas are well documented and can be potentially life - threatening if not diagnosed early and promptly treated. The three most common forms of high altitude - related illnesses are acute mountain sickness (AMS), high altitude cerebral edema (HACE), and high altitude pulmonary edema (HAPE) [1].

High Altitude Pulmonary Edema is a severe form of altitude - related illness and if left untreated may even be fatal. The diagnosis of HAPE is mostly clinical. Patients present with a history of fatigue, dyspnea and cough with exertion within three to five days of travel to a high - altitude region [2]. Objective clinical criteria may also assist in the diagnosis of HAPE and include tachycardia (heart rate of more than 95 bpm), tachypnoea (respiratory rate more than 21 per minute) and SpO2 less than 86% while breathing room air [3]. Other clinical findings are varied and include loss of stamina, dyspnea, dry cough with exertion, dyspnea at rest, rales, cyanosis, cough, and pink, frothy sputum, etc [4].

Multiple risk factors have been described for the occurrence of HAPE and include rate and height of ascent, male sex, use of sleep medication, excessive salt ingestion, genetic polymorphisms, increased pulmonary vascular reactivity and patent foramen ovale.

High Altitude exposure is well known to be associated with ECG changes [4, 5]. It is hypothesized that hypoxic pulmonary vasoconstriction leads to a rise in pulmonary artery pressure and causes morphological changes on the electrocardiogram. These changes include right axis deviation, right bundle branch block, and changes to P and T wave amplitudes. These are commonly found on ascent and resolve only after return to lower altitudes. Although atrial and ventricular ectopic activity is also common, tachyarrhythmias are rare in healthy individuals. In individuals with cardiac disease, the hypoxic environment can be hazardous, exposing individuals to ischemia and risk of sudden cardiac death [6].

Although ECG changes are known to occur in HAPE, these changes have not been adequately or specifically described in the literature. Moreover, no report describes dynamic ECG changes in HAPE patients.

2. Case Presentation

In this report, we describe the case of a 30 - year - old male with no prior known comorbidities. He was asymptomatic till presentation. He arrived at an altitude of 3500m (11, 483ft) by air from sea level followed by descent to 2676m (8780ft) on the same day by road for acclimatization. He was involved in routine daily physical activity at this height and was not involved in height gain or rigorous exercise. Two days later, he developed generalized weakness and a feeling of extreme lethargy. He took over - the - counter medications for the same with which he had minimal relief. That same night, he woke up from his sleep due to breathlessness which was associated with dry cough. Breathlessness was described as a sensation of air hunger. He reported to our hospital for the same.

He gave no history of chest pain, palpitations, diaphoresis, or syncope. He had never had a similar episode in the past. There was no history of taking any medication, alternative medicines, or sleeping pills. He was a non - smoker and did not consume alcohol. There was no history of any infections or chronic illness in the past. He gave no family history of cardiovascular diseases, hypertension, or chronic infections.

General Examination revealed him to be afebrile with tachycardia (HR of 124/min, regular). His blood pressure was

116/74mmHg. He was noticed to have pulse oxygen saturation (SpO2) of 76% on room air. His respiratory rate was 32 beats/min. There was no pallor, cyanosis, or pedal edema and his JVP was not elevated.

Examination of the respiratory system revealed scattered crackles in the right infra - axillary and inter - scapular regions. Normal vesicular breath sounds were heard in all other areas. Examination of the cardiovascular system and the rest of the systemic examination was normal.

He underwent Point of Care Ultrasound (POCUS) in the form of lung ultrasound and 2D Echo which were normal. His initial ECG was found to be unremarkable.

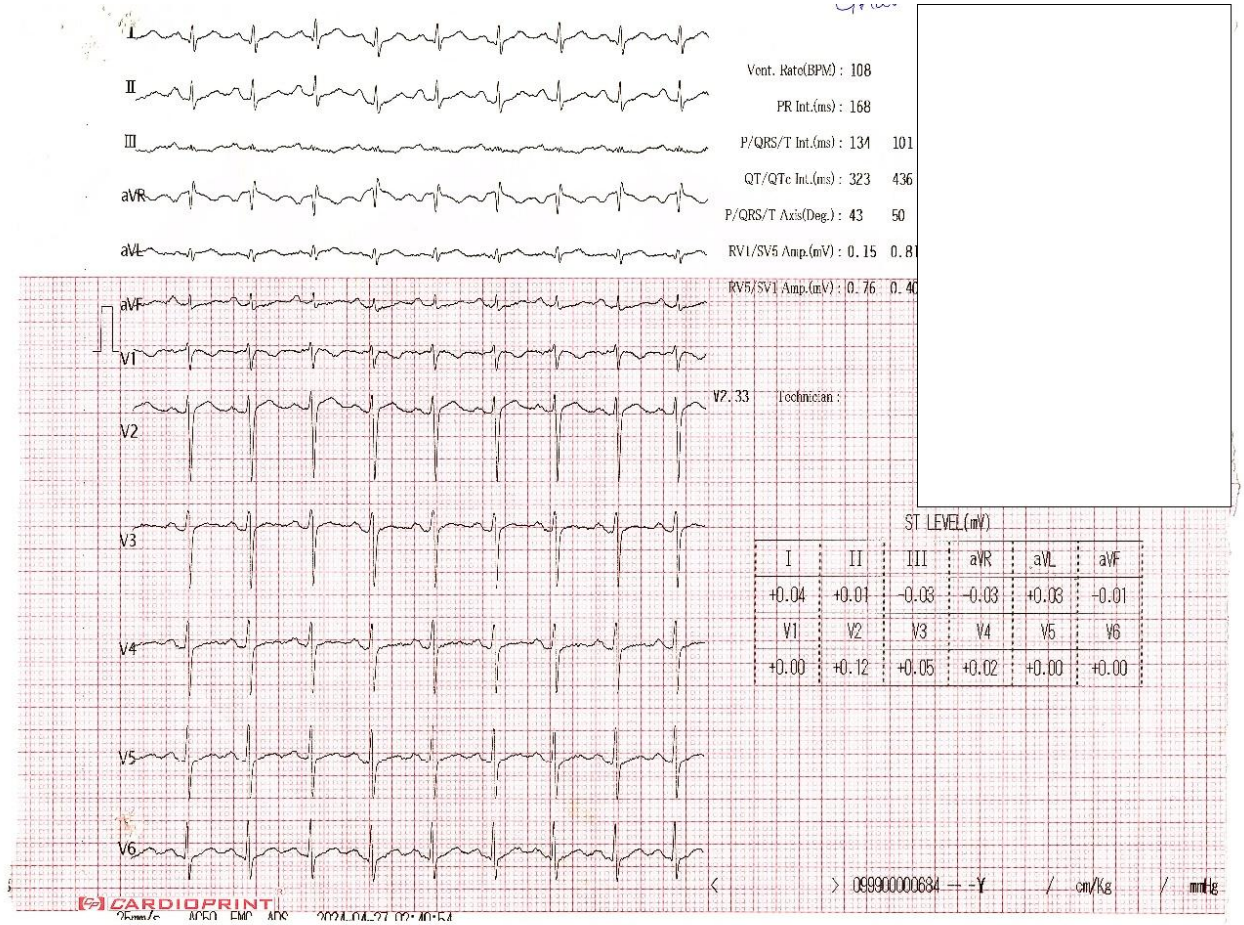

Figure 1: Initial ECG showing normal sinus rhythm, normal ECG

Chest X - Ray revealed non - homogenous, fluffy, alveolar opacities with no evidence of pulmonary arterial hypertension. The opacities mainly involved the right middle zone.

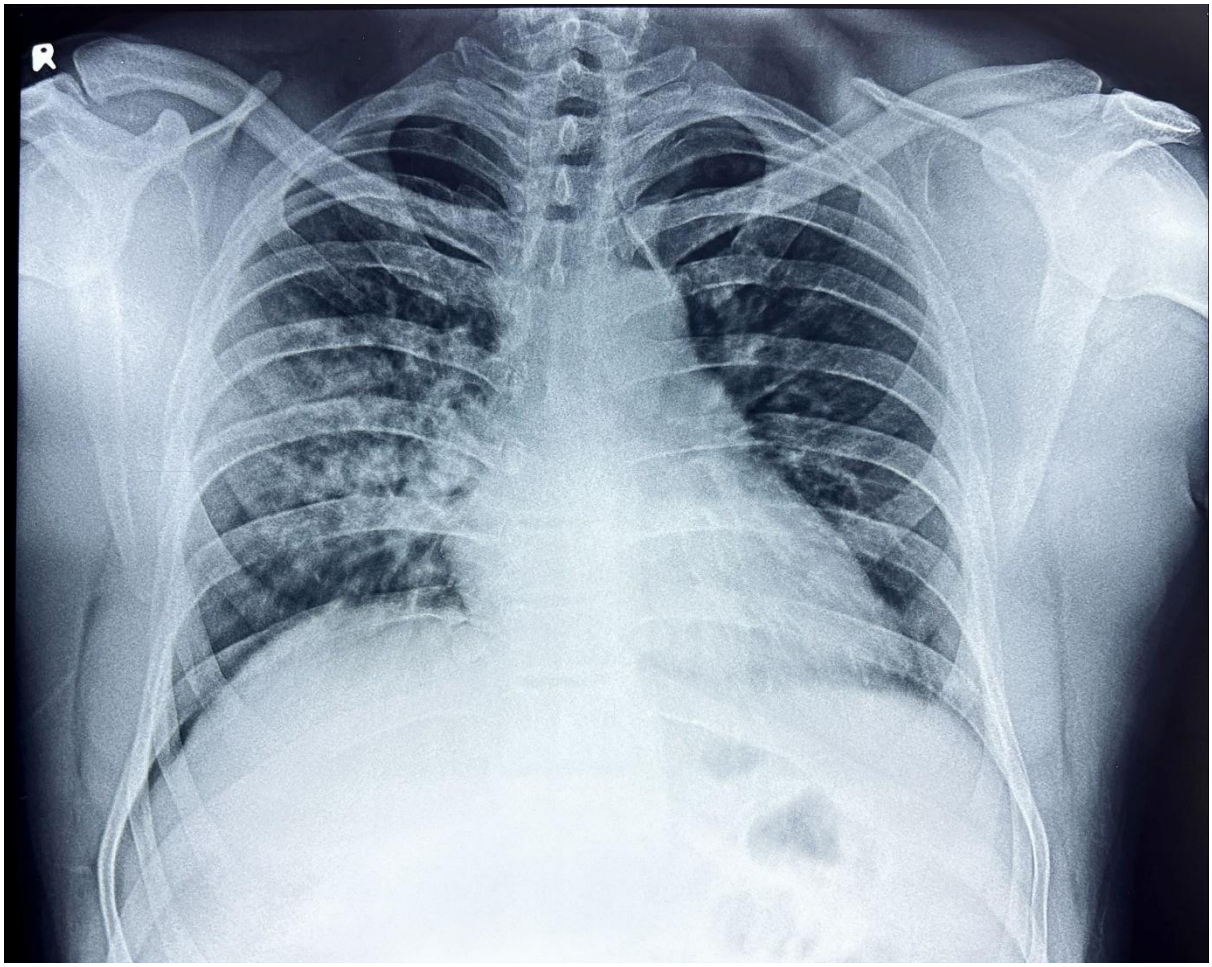

Figure 2: Chest X - Ray showing bilateral pulmonary edema

His initial investigations are given in Table 1

Table 1: Baseline and cardiac investigations

Investigation	Value	Reference Range
Hb (g/L)	16.2	>14
TLC (/cumm)	10, 500	4000 - 11000
DLC (%)	P83 L14	
Platelet (/cumm)	1.76 x 10^3	>1.5 x 10^3
PT (Control/ Test)	14/16 s	
INR	1.1	
Urea (mg/dL)	37	15 - 40
Creatinine (mg/dL)	1.1	0.6 - 1.3
Uric Acid (mg/dL)	4.3	2 - 7
Sodium (mEq/L)	146.4	135 - 155
Potassium (mEq/L)	3.6	3.5 - 5.5
Total Bilirubin (mg/dL)	1.2	0.3 - 1.2
Direct Bilirubin (mg/dL)	0.2	
AST (IU/L)	52	<40
ALT (IU/L)	65	<40
Qualitative Troponin I	Negative	
CPK - MB (IU/L)	52	<25
nt - pro BNP (pg/mL)	410	<130

Based on clinical and radiological findings in the background of a recent gain in altitude, he was diagnosed as a case of Re - Entry High Altitude Pulmonary Edema (Lake Louis Definition for HAPE). Other differential diagnoses were Pulmonary Thromboembolism (unlikely with alveolar opacities on x - ray), pneumonia (unlikely in the absence of fever), cardiogenic pulmonary edema secondary to ACS (unlikely with no chest pain, a normal ECG, normal 2D Echocardiogram and negative cardiac enzymes).

He was started on oxygen therapy to maintain SpO2 >90% and was started on Tab Nifedipine SR 20mg thrice a day along with supportive care as per prevalent treatment protocols. He was followed up to rule out other differentials and observed for the resolution of symptoms.

Follow up ECG findings

On follow up (12 hourly ECG and 48 hourly chest x - ray) he was found to develop dynamic ECG changes in the form t wave inversions in anterior chest leads suggestive of Right Ventricular Strain.

On day two of hospitalization, he improved symptomatically with reduction of tachycardia and tachypnoea and improvement in pulse oxygen saturation. However, he continued to develop new ECG changes and began to show t wave inversions in leads V1 - V4. The ECG changes further evolved during the next two days and the presence of right ventricular strain became more prominent.

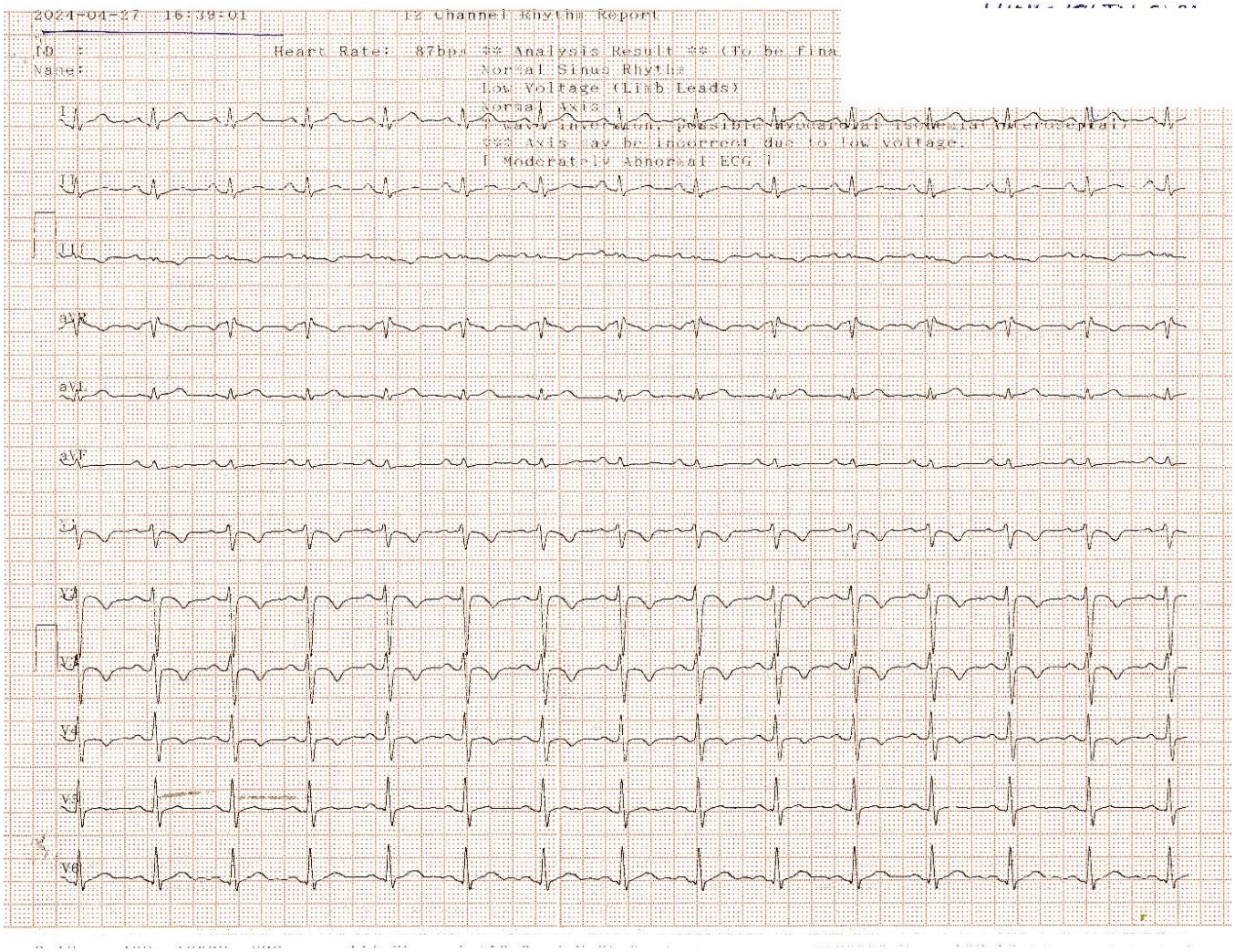

Figure 3: Follow up ECG showing RV Strain

Other changes indicative of right ventricular hypertrophy like RAD without RBBB, p wave abnormalities, etc were not present in the ECGs. Moreover, the ECG changes were not associated with an increase of cardiac enzyme levels or worsening X - Rays. Serial x - rays revealed gradual subsidization of the opacities congruent with clinical findings.

He was further observed and routine ECGs were taken over the next two days. On the fourth day of presentation, dynamic changes were again noticed in the form of resolution of the t wave inversions, initially in V2 - V4 and subsequently in all affected leads.

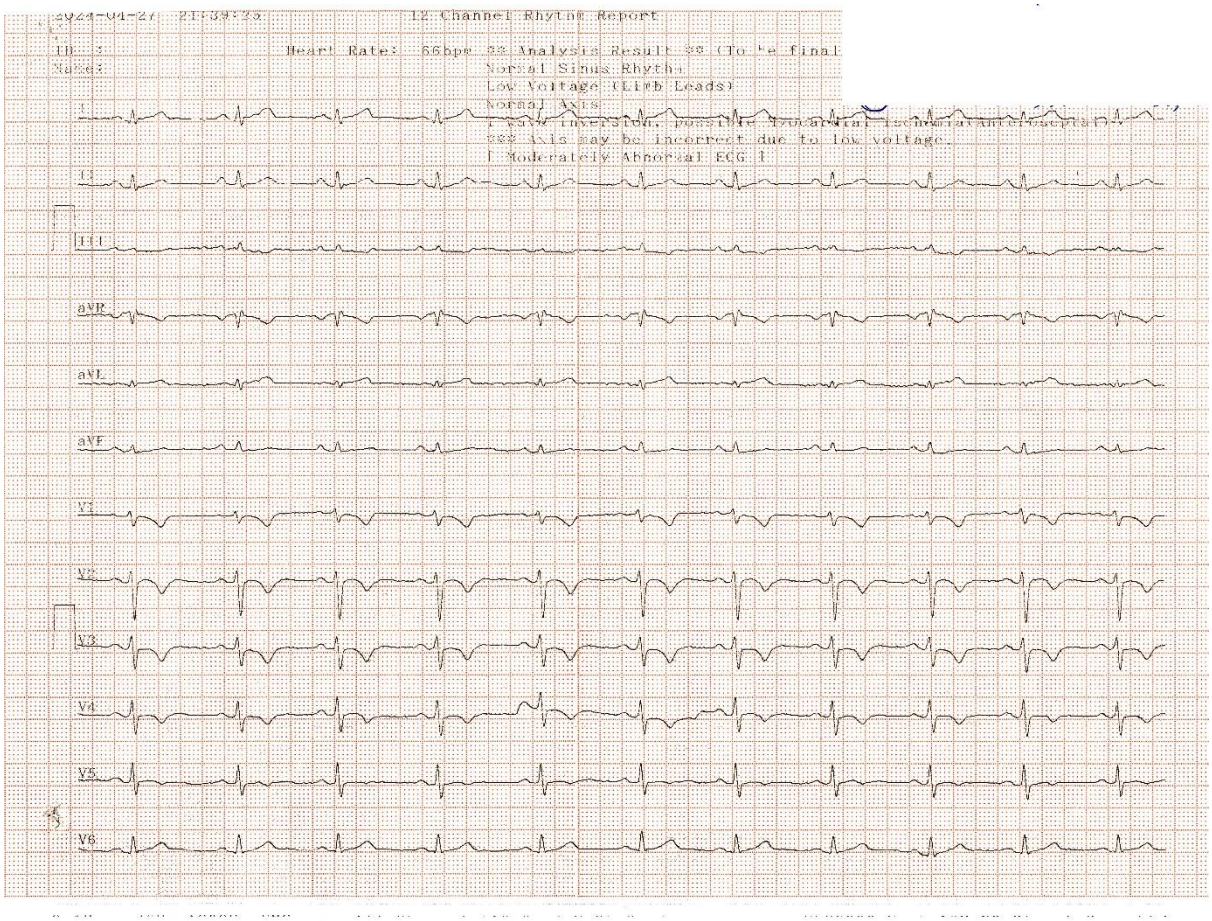

Figure 4: Follow up ECG after day 4 showing resolution of changes

These changes were noticed to evolve over the next two days following which the patient continued to have normal ECG findings till his discharge on the tenth day of presentation.

The dynamic ECG changes had prompted evaluation for other possible aetiologias. Pulmonary Embolism was ruled out by CT Pulmonary Angiography which did not reveal evidence of thrombosis or RV hypertrophy.2D Echo and cardiac biomarker negativity conclusively ruled out ACS.

The patient was gradually weaned off oxygen and was comfortable in room air by day five of admission. The dynamic ECG changes were attributed to transient increase in pulmonary capillary pressures.

To the best of our knowledge, this is the only report which describes dynamic ECG changes in a patient with high altitude pulmonary edema despite clinical improvement.

3. Discussion

In patients with HAPE, ECG changes have been noted in anterior chest leads and have been seen to mimic cardiac emergencies like ACS. The clinical setting of high altitude, extreme cold and the possibility of pulmonary arterial hypertension and pro - thrombotic complications like Pulmonary Embolism (PE) associated with similar symptoms and signs are considered confounding factors for the accurate diagnosis of HAPE. Moreover, cardiac biomarkers and D - dimer have also been seen to be elevated in HAPE patients. Systematic follow - up has been suggested for such patients to rule out cardiac abnormalities.

The differential diagnosis of HAPE includes other conditions causing hypoxia and dyspnea like pneumonia, pulmonary embolism (PE), acute coronary syndrome, etc. PE and HAPE are close differentials with similar presentation and clinical background. Common features include cough, chest pain, acute onset breathlessness, tachycardia, tachypnoea and RV strain pattern on ECG [7]. PE should be considered in patients who do not improve or get worse with descent within 24 hours [8]. Other differential diagnoses include asthma, bronchitis, congestive cardiac failure, hyperventilation syndrome, myocardial infarction, pneumonia and pulmonary embolism [9]. Myocardial infarction is an important differential, and its recognition is potentially life - saving [10].

Management of HAPE is as per the Wilderness Medical Society guidelines and oxygen is the mainstay of treatment [1]. Studies have also found that the use of auto - PEEP with or without oxygen may be beneficial to patients [11]. Other modalities include the use of dexamethasone and nifedipine. It has also been seen that oxygen as a sole treatment is noninferior to use of oxygen along with medications [12]. Moreover, nifedipine likely provides no additional benefit in the management of HAPE [13].

HAPE patients may show varied ECG changes. The most common finding is sinus tachycardia. There may also be changes suggesting pulmonary arterial hypertension which

include right axis deviation, right bundle branch block, right ventricular hypertrophy (tall R wave over the right precordial leads) and right atrial enlargement (peaked P waves in leads II, V1, and V2). Hemodynamic measurements in such patients have revealed high pulmonary artery pressures and increased pulmonary vascular resistance as well as low to normal pulmonary capillary wedge pressures, cardiac output and systemic arterial blood pressure [14]. Similar findings are not generally seen in other altitude - related illnesses like AMS and HACE. Prolonged ECG abnormalities have also been noted in HAPE patients despite descent and no other cardiac abnormalities.

Chest radiographs in HAPE usually show a normal size heart, full pulmonary arteries and patchy infiltrates generally confined to the right middle and lower zones in mild cases and involve both lungs in severe cases [9]. However, there is no radiographic feature specific to HAPE [15].

Such ECG changes suggest that although HAPE is considered a non - cardiogenic form of pulmonary oedema and left ventricular filling pressures are preserved, there are occult associated effects on the heart demonstrated by an increase in cardiac - specific markers of ventricular pressure/volume overload including Brain Natriuretic Peptides (BNP) and cardiac troponins [cTn]) [16].

The pathophysiology behind cardiac involvement in HAPE has been studied using right heart catheterization and has shown that the edema is caused by an increased microvascular hydrostatic pressure in the presence of normal left atrial pressure, resulting in leakage of large - molecular - weight proteins and erythrocytes across the alveolar - capillary barrier in the absence of any evidence of inflammation [17].

While exaggerated pulmonary hypertension is most probably a prerequisite for HAPE, it may not always be a sufficiently strong causative factor in itself. Defective alveolar fluid clearance has also been proposed as a second important pathogenic mechanism [18].

Various contributing factors for pulmonary arterial hypertension include hypoxia - induced suppression of nitric oxide synthase enzymes, polycythemia caused by high altitude - related increased resistance to pulmonary blood flow, increased blood viscosity, etc. Pulmonary arterial hypertension, besides being a causative factor for HAPE also results in reduced exercise capacity, and right ventricular dysfunction and may facilitate the transition to chronic pulmonary arterial hypertension [19].

4. Conclusion

ECG Changes have been well documented in cases of High Altitude Pulmonary Edema. These changes are not unique to HAPE and are also seen in individuals ascending to high - altitude regions who do not suffer from the same. These changes mainly correspond to right ventricular dysfunction and are hypothesized to indicate pulmonary arterial hypertension. Although HAPE is defined as a non - cardiogenic form of pulmonary oedema, a strong body of evidence exists to suggest that associated cardiac dysfunction is more common than anticipated. Serial ECG changes as described in this report further help in describing associated cardiac dysfunction. Such changes complicate the diagnosis of HAPE and prompt expensive and exhaustive cardiac evaluation. In high - altitude areas, they also necessitate the use of significant resources to evacuate patients to a cardiac center which, more often than not is located far away.

Such reports are valuable in helping clinicians in interpreting unusual ECG changes in HAPE patients, enabling confident diagnosis and effective management as the primary treatment in HAPE is Oxygen which is available in most local medical centers in high altitude areas thereby avoiding unnecessary utilization of resources.

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