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Case Report

# High Altitude Pulmonary Edema with Pulmonary Embolism in High Altitude Traveller – A Case Report

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## Abstract

High Altitude Pulmonary Edema (HAPE) often occurs 2-5 days after ascent to altitudes >2500m. In this report, we discuss a case where a 33-year-old Chinese lady was initially diagnosed to have HAPE after developing severe respiratory symptoms during high altitude ascent. She later demonstrated to have Deep Vein Thrombosis (DVT) and Pulmonary Embolism (PE) after her symptoms persisted despite HAPE treatment. We speculate whether the presence of subacute thrombo-emboli led to HAPE, or if PE presented independently and concurrently with HAPE in the patient. While HAPE is commonly diagnosed, PE is less commonly suspected and can result in life-threatening implications in high altitude travellers if left untreated. HAPE and PE have vastly different managements despite similar clinical presentations. Due to the high fatality of PE, it is important that ED physicians are aware of PE as a differential when high altitude travellers with features of HAPE present to the ED.

## Introduction

High Altitude Pulmonary Edema (HAPE) is characterized by cough, dyspnoea, and reduced exercise performance, and often occurs 2-5 days after ascent to altitudes >2500m [1]. At high altitudes, alveolar hypoxia causes intrapulmonary vasoconstriction to optimize ventilation/ perfusion matching [1]. Rapid ascent without proper acclimation to high altitude hypoxia can rupture the alveolar-capillary barrier and inhibit alveolar fluid clearance, resulting in pulmonary edema [1]. A patient with persistent dyspnoea after high altitude mountaineering was found to have Deep Vein Thrombosis (DVT) and Pulmonary Embolism (PE) after an initial diagnosis of HAPE. In this report, we propose that PE can coexist with HAPE in patients after high altitude ascension.

## Case Report

A 33-year-old Chinese lady with a past medical history of high altitude cerebral edema three years prior, attempted to climb the Himalayas. She began her climb at 3000m and ascended at a pace of 1000m daily. On the second day, the patient developed cough and dyspnoea that was progressively worsening. She complained of dizziness, headache and lethargy. She received supplemental oxygen therapy before land evacuation the following day. At the hospital in Nepal, blood tests and a Chest X-Ray (CXR) were done, in which asymmetrical infiltrates were seen bilaterally (Figure 1). The patient was diagnosed with HAPE and treated with acetazolamide and sildenafil. Subsequently, she developed right calf tenderness with haemoptysis. There was no prior trauma to her right leg. Against medical advice, the patient discharged herself to continue treatment in Singapore.



Figure 1: CXR in Nepal showing asymmetrical infiltrates bilaterally

The patient presented at our Emergency Department (ED) in Singapore with a four-day history of right calf tenderness that was associated with dizziness, dyspnoea and palpitations. On examination, she was comfortable at rest with no respiratory distress. She was afebrile, mildly hypotensive (100/67 mmHg), tachypnoeic (24bpm) with a regular pulse rate (47bpm). There were vesicular breath sounds with equal air entry on auscultation. Calves were supple with equal circumference bilaterally (36cm). The patient was not taking any hormonal medication. There was no family history of hypercoagulability.

Laboratory investigations disclosed the following: haemoglobin 12.4g/dl; white blood cells 7x10<sup>9</sup>/L; platelet 221x10<sup>9</sup>/L; capillary blood glucose 5mmol/L; sodium 139mmol/L; potassium 3.6mmol/L; urea 4.1mmol/L; creatinine 63µmol/L. Arterial blood gas at room air showed: pH 7.43; pCO<sub>2</sub> 33; pO<sub>2</sub> 94; HCO<sub>3</sub> 22; base excess -2; SaO<sub>2</sub> 98%; lactate 0.44. CXR findings were normal (Figure 2). ECG conducted did not reveal any changes indicative of PE such as sinus tachycardia or S1Q3T3 (Figure 3). A Computed Tomography Pulmonary Angiogram (CTPA) was conducted after elevated D-dimer levels (2.97) were noted. Filling defects were seen in the right and left lower lobe anterior basal segmental pulmonary arteries. Scattered ground-glass opacities were also recognized, suggesting the presence of PE (Figure 4). A Doppler ultrasound of her right calf revealed thrombosis in the gastrocnemius veins. The patient was started on subcutaneous enoxaparin in the ED and was discharged with oral rivaroxaban three days later.

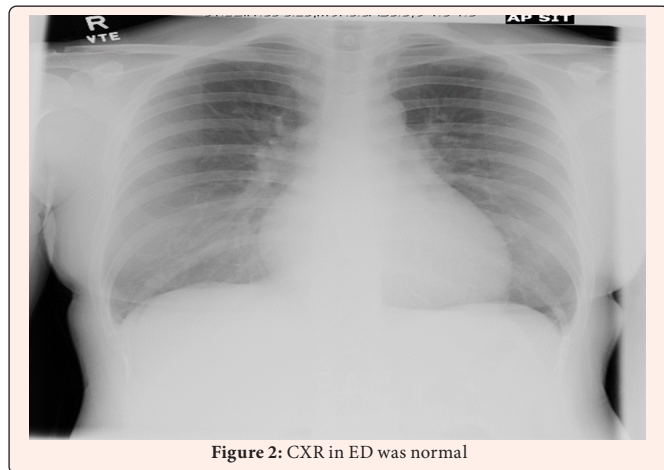


Figure 2: CXR in ED was normal

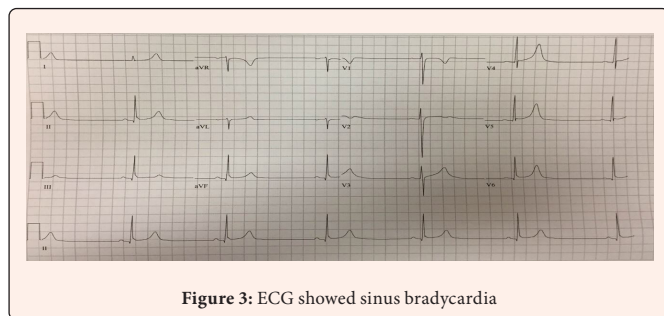


Figure 3: ECG showed sinus bradycardia

At high altitudes, factors such as hypoxia, dehydration, physical trauma, extremely low temperatures and bradycardia could contribute to hypercoagulability, circulatory stasis and vascular damage [2]. In addition, raised D-dimer concentration and activated protein C resistance indicate activation of coagulation during ascent [2]. As such, ascension to high altitudes can result in thromboembolic phenomena such as DVT, PE, thrombophlebitis and stroke [2]. In PE, increased intrapulmonary pressure in patent pulmonary arteries can lead to extravasation of fluid into the alveoli [3]. Similarly, the presence of proinflammatory cytokines, thromboxane and other inflammatory mediators can also lead to alveoli edema [3]. In this patient, we speculate whether the presence of subacute thrombo-emboli led to HAPE, or if they occurred independently. It is imperative for ED physicians to be aware that PE can also occur in patients with recent high-altitude travel. The Well's criteria evaluate the risk of PE in patients by taking into consideration the signs and symptoms of DVT, recent

surgery/ immobilization and presence of malignancy [4]. Based on the Well's criteria, the patient obtained one point, indicating low risk of PE with a 1.3% prevalence [4]. These criteria were not useful in accurately identifying the risk of PE in the patient. D-dimer is a fibrin degradation product, which is produced from the proteolysis of cross-linked fibrin [5]. D-dimer screening has high sensitivity but low specificity in the diagnosis of DVT/ PE [4,5]. Furthermore, ascent to high altitude has been suggested to raise D-dimer levels [2]. D-dimer assays should only be conducted to rule out PE when there is high clinical suspicion or poor response to HAPE treatment [5]. CTPA is recommended when patients presenting with HAPE symptoms do not improve with treatment or worsen with descent [5]. Both HAPE and PE have similar clinical presentations but vastly different managements.

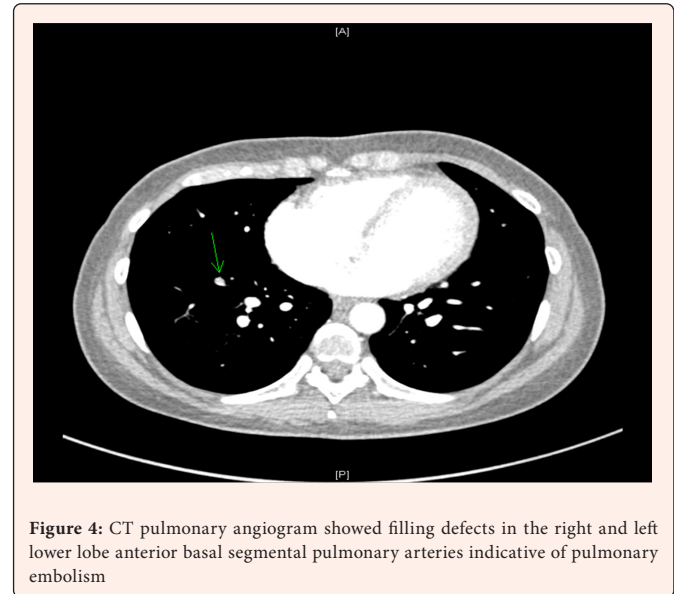


Figure 4: CT pulmonary angiogram showed filling defects in the right and left lower lobe anterior basal segmental pulmonary arteries indicative of pulmonary embolism

### Conclusion

Delayed or missed diagnoses of PE can have life-threatening implications. It is important for ED physicians to consider PE as a differential when patients with features of HAPE respond poorly to treatment.

**Competing interests:** None

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