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Case Report

Massive pulmonary thromboembolism in high altitude area versus high altitude pulmonary oedema

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Case report

A 47-year-old male patient presented with sudden onset cough, tightness in chest and breathlessness on Day 2 of stage I acclimatization. It was associated with headache and giddiness with presyncope. There was no antecedent history of productive cough, fever, chest pain, diaphoresis, orthopnea and paroxysmal nocturnal dyspnoea (PND). Clinically patient was normotensive, had tachycardia, tachypnea and low oxygen saturation (80–85%)@6 l/min O₂ inhalation. On auscultation patient had left sided inferior axillary fine end inspiratory crackles with bronchial breath sounds. Electrocardiography (ECG) was initially normal and subsequently after 12 h showed features of RV strain. Chest radiograph showed non-homogenous opacities in left mid zone. Patient was managed as a case of HAAPE with oxygen inhalation, calcium channel blocker and diuretics. However, the patient became hemodynamically unstable with falling BP, persistent tachycardia, tachypnea, and low oxygen saturation. Patient was air evacuated to this hospital for further management.

At arrival, the patient was in shock with tachypnea, tachycardia, and SpO₂: 70%@6 l/min O₂ inhalation. Cyanosis present, JVP was raised. Auscultation revealed fine end inspiratory crackles in left inferior axillary region with bronchial breath sounds. D dimer was raised 0.7 mg/L (<0.3 mg/L). Repeat radiograph revealed attenuated descending pulmonary arteries bilaterally with consolidation left upper and mid zone (as depicted by CT topogram in Fig. 1).

Introduction

Pulmonary thromboembolism (PTE) is a common complication of thrombosis among troops living in high altitude areas¹ (HAA). In a high altitude scenario, PTE is very often missed clinically as other possible diagnosis like high altitude pulmonary oedema (HAPE) takes priority as a cause of acute onset breathlessness. Recently a patient with acute onset breathlessness during first stage of acclimatization was air evacuated to this hospital from high altitude area as a case of HAPE.

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Fig. 1 – CT topogram showing sudden attenuation of right (solid white arrow) and left (open white arrow) descending pulmonary arteries. Consolidation suggesting large pulmonary infarct left upper and mid zone (black quad arrow).

CT pulmonary angiogram revealed dilated right ventricle (RV) with abnormal left convexity of interventricular (IV) septum, dilated pulmonary arteries with filling defects extending into peripheral branches and consolidation left upper lobe (LUL) suggestive of acute PTE involving both right and left pulmonary arteries extending into secondary and tertiary branches with segmental infarct LUL (Figs. 1-4).

Patient was diagnosed as a case of acute massive PTE with RV dysfunction and lung injury and thrombolysed with Alteplase followed by anticoagulants. Vital parameters improved in 3 h and the patient became ambulant over 12 h. Subsequently, the patient was referred to a tertiary centre for further evaluation and management by haematologist and cardiologist.

Fig. 2 – CT pulmonary angiogram: central intraluminal filling defect in right pulmonary artery (white quad arrow). An eccentric filling defect in upper lobar branch of left pulmonary artery (white arrow). A large pulmonary infarct anterior segment of left upper lobe (white open arrow).

Fig. 3 – CT pulmonary angiogram: dilated right ventricle (black quad arrow) with abnormal left sided convexity of IV septum (white arrow).

Fig. 4 – CT pulmonary angiogram: dilated right pulmonary artery (black quad arrow) with saddle thrombus at bifurcation (white arrow). A large pulmonary infarct left upper lobe (white open arrow).

Discussion

PTE and HAA are close differentials among troops posted in HAA with common clinical features of cough, chest pain, acute onset breathlessness, tachycardia, tachypnea and RV strain pattern on ECG. Our patient further challenged the suspicion of PTE by developing symptoms on second day of induction to high altitude area with absence of risk factors or clinical features for deep venous thrombosis (DVT) or any prior history of DVT.

Possibility of PTE became more obvious, only when patient did not improve after being deinducted from HAA; making the diagnosis of HAA less likely. On revisiting the history, the patient told that he had been lying on his bed after the lunch when he had woken up with recurrent bouts of dry cough making him breathless. This history further made possibility of HAA less likely, wherein breathlessness usually starts during some physical activity/exertion. Subsequently D dimer was found to be raised and diagnosis of Massive PTE was confirmed by CT angiogram showing thrombus in both left and

right pulmonary arteries with features of RV dysfunction as well as lung injury (infarct). Patient was deteriorating hemodynamically with rapidly rising tachycardia, falling BP and hypoxia supporting the indication for emergency thrombolysis as a life saving measure.

Distinction between HAPE and PTE though very challenging in high altitude set-up, is very important for favourable patient outcome. Studies have documented; clinical assessment in combination with D-dimer assay, LDH and AST may be helpful in differentiating PTE from HAPE at high altitude where imaging facilities are not available.² However when available, CT pulmonary angiography is now gold standard in diagnosis of PTE with its high degree of resolution even a small peripheral emboli located in peripheral branches could be evaluated and hence it has obviated the need for conventional invasive contrast pulmonary angiography.³

Early evacuation to higher medical echelons for confirmation of diagnosis is important as presumptive management of a suspected case of HAPE by Calcium channel blockers and diuretics could further complicate a case of PTE by aggravating hypotension and promoting thrombus formation by increasing viscosity of blood. On the contrary, presumptive management of suspected PTE by anticoagulation/thrombolysis is of no therapeutic benefit in a case of HAPE and has got added risk of bleeding complications.

Conclusion

Although clinical differentiation between PTE and HAPE in high altitude area is very difficult, but if due to logistic

constraints holding of patient is unavoidable then repeated history taking, serial examinations, serial ECG and biochemical investigations may be of help for reconsidering the close differentials and accordingly modify the line of management. If the patient is not responding or is deteriorating despite the given presumptive management then imaging modalities like CT pulmonary angiography may be helpful in confirming the etiological diagnosis and bringing out better treatment outcome in terms of morbidity and mortality.

Conflicts of interest

The authors have none to declare.

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