CORRESPONDENCE

Interstitial Lung Disease at High Altitude

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Sir,

The clinical presentation of Interstitial Lung Disease(ILD) without acclimatization at high altitude is not studied. On review of the literature we could find only two studies which presented physiological changes in the body on exposure to simulated hypoxia.[1,2] We have a case of ILD who was accidently exposed to high altitude with presentation mimicking High Altitude Pulmonary Edema (HAPE), ACS/NSTEMI, ALVF and AECOPD. The diagnosis of ILD was not on the card during her stay in the high altitude. Once she returned back to India, we could have a retrospective study of the presentation.

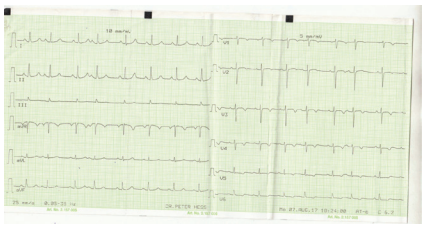

Fig. 1: ECG on 02.08.2017: NSR 70/min, long QTc negative T in III and Avf, V3-V5, slight ST elevation in V1-V3

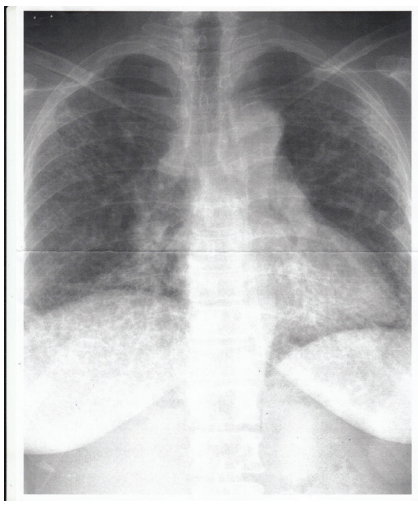

Fig. 2: Chest radiograph on 03.08.2017 showing medium sized heart and mediastinum, blurred right border of heart with bilateral prominent hili, congestion both upper lobes

A 60 Year old female went on a package tour to Europe on 27.07.2017. She arrived at Bern, Switzerland which is 11,333 ft. high above the sea level by train in the afternoon on 02.08.2017. She developed severe respiratory distress inside the coach and had to be rescued by Emergency Rescue Team (ERT) to an adjacent Primary Care Centre. On examination at the centre she was complaining of severe respiratory distress, right sided intermittent chest pain and discomfort and nausea. She had sore throat, stuffy nose and cold for the previous 2-3 days. There was no history of fever. She gave past history of similar respiratory distress 1 year back. No history of allergy. She was not a known diabetic or hypertensive. She was orthopnoeic with no cyanosis, pallor, jaundice, oedema or clubbing. JVP was normal. Heart rate 82/min., regular, BP 120/65, Resp. 25-30/min., Temp. 36.3C. Chest symmetrical expansion, vesicular breath sounds with basal rales. Heart sounds normal with regular rhythm, no pathological heart sounds. Peripheral pulses normal. No carotid bruit. Abdomen soft, non tender with normal bowel sounds. CNS conscious but apprehensive, no neurological deficit. Spine and joints normal. Skin warm with normal colour. Her SpO2-87%, ECG – normal sinus rhythm with rate 70/min., long QTc, negative T in III and aVF, V3-V5 and slight ST elevation in V1-V3 (Figure 1). Her hs- Troponin- I initially 57 later 109.4ng/L, CK237/L. Her Hb was 9.8 G/dl with microcytic hypochromic PBS. With the provisional diagnosis of ACS / NSTEMI she was administered O2 2L/hr, LMWH 5000 IU/IV, Prasugrel 60 mg., Aspirin 250 mg, Furosemide 40mg/IV. After 2 hours of treatment she was shifted to Bern Heart and Vascular Centre, University Association Bern Hospital, Bern where TTE was done on 03.08.2017 to find normal systolic LV function (LVEF 65%), diastolic dysfunction, normal RV function and no regional wall motion or valve abnormality. CAG with right heart catheterization excluded CAD, LVEF 65%, no PAH (mPAP 17mm of Hg). Chest radiographs on 03.08.2017 and 04.08.2017 showed medium sized heart and mediastinum, blurred right border of the heart with bilateral prominent hili, congestion both lower lobes and slightly in both upper lobes. Radiological D/D was interstitial transudation or pulmonary oedema or fibrosis, no relevant pleural effusion, no pneumothorax (Figure 2) D-dimer <500U. Initial diagnosis at the Primary Center was ALVF due to NSTEMI with supportive evidence of some changes in ECG and elevated hs-Troponin I and CPK. In the advanced centre acute coronary event was excluded with normal ECHO and CAG. Her PAP was normal and both the ventricular functions were normal. On 03.08.2017 she was continued with monitoring, O2 2L/hr, Aspirin 100mg, Prasugrel 10mg. A diagnosis of HAPE was considered because of her non-acclimatization and low SpO2. She was also considered for acute exacerbation of COPD and pre-existing pulmonary disease. She improved with SpO2 100% under 3L O2/hr, BP 138/77, Pulse 82/min, Resp 20-25/min and Temp 36.3 C. Discharged from the hospital on 04.08.2017. She returned back to India on 06.08.2017. On examination on 08.08.2017 she had mild dyspnoea, no cyanosis, Resp 20/min, BP 120/70, Pulse 88/min, Temp 98.4 F. Her chest had coarse rales in both the bases, SpO2 99%, Sr Creatinine0.9MG/D/, RBS 98mg/dl, HbA1C 6.1, Sr Cholesterol 169mg/dl, Sr Triglyceride 104mg/dl, HDL 44mg/dl, LDL 105mg/dl, ANA 2.88, PFT suggestive of moderate to severe restrictive lung disease. HRCT Thorax

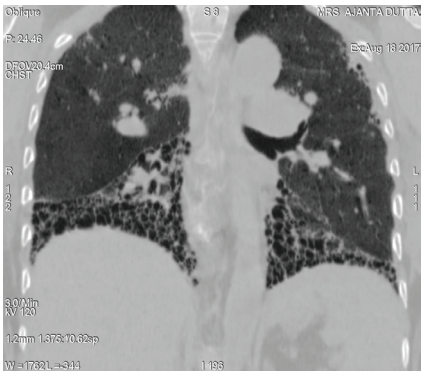

Fig. 3: HRCT thorax on 18.08.2017 suggestive of ILD with prominent bi-basal honey combing and pulmonary hypertension

(Figure 3) suggestive of ILD with predominant bi-basal honeycombing and pulmonary hypertension.

Exposure to low O2 tension at high altitude leads to a series of physiological events for acclimatization. In some cases sudden exposure to high altitude hypoxia can cause maladaptive responses called High Altitude Pulmonary Edema(HAPE), High Altitude Cerebral Edema (HACE) and Acute Mountain Sickness(AMS). Patients with underlying lung disease are at higher risk of complication in hypoxic environment and warrant careful evaluation. Only a few studies have addressed the effect of high altitude travel on patients with ILD. Seccombe et al[1] exposed a mixed group of ILD patients to normobaric hypoxia (FiO2:0.15) demonstrating increased dyspnoea and low PaO2 both on rest and exertion. This findings were corroborated with those of Christensen et al,[2] who reported that PaO2 decreased from mean of 78±12 mm of Hg at sea level to 49±8 mm of Hg at rest and 38±7 mm of Hg with mild exercise at a simulated altitude of 2438M. They also demonstrated that supplemented O2 at a rate of 2 L/min at rest and 4 L/min with exercise was sufficient to keep PaO2 above 50 mm of Hg in those patients.

No studies have examined changes in pulmonary hemodynamic in ILD patients at simulated or high altitude hypoxia. It is known, however that many of the patients develop PAH as complication of ILD which predispose them to HAPE or RV dysfunction at high altitude. Individuals with ILD should be evaluated at sea level with PFT and ABG before travelling to high altitude to determine the need for supplemental O2.

References

1. Seccombe LM, Kelly PT, Wong CK et al. Effect of simulated commercial flight on oxygenation in patients with interstitial lung disease and obstructive pulmonary disease. *Thorax* 2004; 59:966-970.
2. Christensen CC, Ryg MS, Refvem OK, et al. Effect of hypobaric hypoxia on blood gases in patient with restrictive lung disease. *Eur Respir J* 2002; 20:300-305.