Allergic bronchopulmonary aspergillosis causing bendopnea

Sir,

A 55-year-old female presented with a complaint of dyspnea that worsened during activities involving bending forward such as wearing shoes and sweeping the floor for the last 3 months. She had been suffering with severe asthma since the age of 35 years and was diagnosed to have bilateral lower lobe cystic bronchiectasis 9 years ago. Investigations had revealed elevated total IgE levels (2300 U/L; normal <100 U/L), peripheral blood eosinophilia (1800 cells/µL), and raised Aspergillus fumigatus specific IgE (5.2 kUA/L; normal <0.35 kUA/L). Chest radiograph [Figure 1] showed bilateral middle and lower zone cystic opacities. High-resolution computed tomography of the chest [Figure 2] showed extensive areas of cystic bronchiectasis involving bilateral lower lobes. A diagnosis of severe asthma complicated by allergic bronchopulmonary aspergillosis (ABPA) was made. She was treated with oral steroids for 6 months that were subsequently tapered. Around 7 years ago, she experienced an exacerbation, which was managed with itraconazole (400 mg/day) and steroids. She developed steroid-induced myopathy and steroids were reduced and stopped. Over the next year, she developed worsening asthma symptoms with four exacerbations requiring hospitalization. She received omalizumab injections once every 4 weeks (300 mg s.c.) and achieved disease control for the next 1 year; thereafter anti-IgE was stopped. The asthma had been relatively well controlled with a combination of inhaled long-acting beta-agonists and inhaled steroids, oral montelukast, and oral theophylline. She had also been using domiciliary oxygen therapy for last 5 years along with other medications. On level ground, she was able to walk for 75–100 m at her own pace.

On examination, her weight was 52 kg with a body mass index of 21 kg/m². There was clubbing, central cyanosis, heart rate was 89 beats/min, BP was 122/70 mmHg, and respiratory rate was 25 breaths/min. Pulse oximetric saturation was 84% while breathing room air. There was no pedal edema, and jugular venous pressure was normal. Auscultation revealed bilateral coarse crackles in interscapular and infrascapular regions with few polyphonic rhonchi. Cardiovascular examination was normal. Arterial blood gas analysis showed hypoxemia and chronic Type 2 respiratory failure (PaO<sub>2</sub> 46 mmHg, PaCO<sub>2</sub> 45 mmHg, pH 7.46, HCO<sub>3</sub> 27 mEq/L). Echocardiography findings were suggestive of normal valves and normal left ventricular function. There were features of Cor pulmonale with moderate tricuspid regurgitation and moderate pulmonary hypertension (right ventricular systolic pressure...
58 mmHg). There were no features of relapse of ABPA on immunological workup that included total IgE levels and peripheral blood eosinophil count.

In view of the predominant complaint of bendopnea, a repeat echocardiography was performed which revealed the same findings (moderate tricuspid regurgitation, right ventricular systolic pressure 50 mmHg, left ventricular ejection fraction 65%, and no diastolic dysfunction). Serum NT-ProBNP was 154 ng/mL and 166 ng/mL on two different occasions (normal <300 ng/mL). She was reassured about the presence of the new symptom and was advised to avoid activities that precipitate bendopnea.

The term “bendopnea” was coined by Thibodeau et al. for describing the symptom of dyspnea on bending forward. They defined bendopnea as dyspnea occurring within 30 s of activities involving bending forward such as putting on shoes and socks.[1] Bendopnea was observed in 29 of the 102 (28%) patients of systolic heart failure in their study. Hemodynamic measurements by right heart catheterization documented the mechanism as further elevation of left-sided filling pressures on bending in those with preexisting advanced systolic heart failure (left ventricular ejection fraction <40%) with a reduced cardiac index.[1]

To the best of our knowledge, this novel symptom has never been reported in respiratory diseases. We report the occurrence of bendopnea in this lady with advanced ABPA and bilateral bronchiectasis.[2] Bendopnea, in this case, is likely due to bilateral bronchiectasis affecting lower lobes and elevated right ventricular pressures. In the absence of overt heart failure, bendopnea in advanced respiratory diseases may be hypothesized to occur due to falling vital capacity on bending forward with subsequent worsening of ventilation-perfusion mismatch.

In conclusion, bendopnea can be seen in patients with advanced respiratory disease and should be considered as an indicator of severe cardiorespiratory compromise.

**Financial support and sponsorship**
Nil.

**Conflicts of interest**
There are no conflicts of interest.

**Ajay Handa, Ritesh Agarwal**
Department of Medicine and Pulmonary Medicine, Command Hospital (Air Force), Bengaluru, Karnataka, 1Department of Pulmonary Medicine, Postgraduate Institute of Medical Education and Research, Chandigarh, India
E-mail: agarwal.ritesh@outlook.in

**REFERENCES**
1. Thibodeau JT, Turer AT, Gualano SK, Ayers CR, Velez-Martinez M, Mishkin JD, et al. Characterization of a novel symptom of advanced heart failure: Bendopnea. JACC Heart Fail 2014;2:24‑31.
2. Agarwal R, Chakrabarti A, Shah A, Gupta D, Meis JF, Guleria R, et al. Allergic bronchopulmonary aspergillosis: Review of literature and proposal of new diagnostic and classification criteria. Clin Exp Allergy 2013;43:850‑73.

This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 3.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.