Tuberculosis and pulmonary hypertension: Commentary

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Pulmonary hypertension (PH) is a hemodynamic and pathophysiological condition, defined as an increase in mean pulmonary arterial pressure >25 mm Hg at rest as assessed by right heart catheterization (RHC).[1] According to the Dana Point (2008) classification of PH,[2] Group 1 PH is known as pulmonary arterial hypertension (PAH), which includes idiopathic PAH (IPAH), heritable, drug-induced and associated with PAH groups. Overall, PAH is a rare disease. Although worldwide prevalence rates are not known, overall prevalence in European countries has been reported as 15–50 cases per million population.[3] The other Groups 2–5 of PH are comparatively more common, although data are lacking. PH due to pulmonary causes, which are clubbed together as Group 3, is one of the most common causes of PH and eventually cor pulmonale. The diseases included in this group are chronic obstructive pulmonary disease, interstitial lung diseases, obstructive sleep apnea, combined emphysema and fibrosis, chronic pulmonary thromboembolism and high altitude residence.[2]

Pulmonary tuberculosis (TB) has not been cited as a cause for the development of PH in western literature. In India, on the other hand, being a high burden country, it is not unusual to find patients who have been treated for pulmonary TB to present with features of right heart failure. The possible causes for the development of PH in these patients are the destruction of vascular bed due to parenchymal abnormalities, vasculitis, and endarteritis, leading to reduced cross-sectional area of the pulmonary vasculature.[4,5] The common presentation of these patients include dyspnea out of proportion to their radiological picture, desaturation at even mild exertion and sometimes as overt heart failure with pedal edema, raised jugular venous pressure and tender hepatomegaly. Indian data on the prevalence of PH in patients with pulmonary TB is limited.[6-8] It is prudent to undertake such studies, as it will be helpful in understanding the exact pathophysiology and timely intervention can be done before the development of PH, as it portends a poor prognosis.

In this issue of the journal, Bhattacharya *et al*. reported PH in patients with tuberculosis.[9] This study has its drawbacks. The number of subjects in the study is too little to generalize the results in a population. Second, it has not been mentioned whether these patients are sputum smear positive at the time of study or have been previously treated for pulmonary TB. Not using RHC for the diagnosis of PH is another shortcoming. Furthermore, functional assessment of patients should have been done through a more composite scoring system than CAT, for proper analysis.

Despite this, such studies need to be encouraged at all tertiary care centers with facilities for RHC and other relevant investigations so that nation-wide data can be collected regarding the existence of PH in treated cases of pulmonary TB. These patients present with worsening dyspnea and are incorrectly diagnosed as relapse of TB and are started on anti-TB treatment or are prescribed inhaled bronchodilators without performing spirometry or documenting airflow obstruction. It is imperative to avoid such mismanagement. Furthermore, adequate and timely management of pulmonary TB would prevent the development of PH and ultimately cor pulmonale, which significantly reduces the quality of life as well as shortens survival.

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