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IP Indian Journal of Immunology and Respiratory Medicine

Journal homepage: https://www.ijirm.org/

Perspective

Destroyed lung syndrome

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ARTICLE INFO

Article history:
Received 01-02-2023
Accepted 04-04-2023
Available online 03-05-2023

Total or extensive destruction of the lung with a compromise in lung function is known as a destroyed lung.[1,2] Further, a destroyed lung is described as an amalgamation of pleural and parenchymal lung destruction with cavitation, bronchiectasis, loss of lung volume, and mediastinal herniation to the diseased side.[3] It is commonly diagnosed in radiological studies and is usually distinguished from other similar conditions by a noticeably reduced ventilation-to-perfusion ratio.[4] It is usually a sequela of recurrent or chronic lung infections like tuberculosis however it may result from bronchiectasis, aspergillosis, emphysema, and pneumonia.[4,5] Further, hypoplastic lung, pulmonary actinomycosis, pulmonary gangrene, and infection by nontuberculous mycobacteria also lead to destroyed lung syndrome.[6] Isolated reports of destroyed lung syndrome post-scoliosis correction with Harrington rod instrumentation and fusion for idiopathic scoliosis are also available.[7]

The destroyed lung is commonly reported in the male gender and has a predilection for the left lung[8–10]. The components of a destroyed lung include pulmonary cavitation, loss of lung volume, cystic bronchiectasis, pleuroparenchymal fibrosis, unilateral near complete lung parenchymal anomalies with combinations of the above findings, contralateral lung parenchymal compensatory hyperinflation manifested as emphysema, and pull of contralateral lung and mediastinal structures to diseased side radiologically termed as mediastinal herniation.[3] The clinical features are mostly chronic but acute presentations are also documented.[6] The patient may present with purulent (yellow-colored) expectoration, chronic fever, dyspnea, chest tightness, and may be complicated with episodes of recurrent hemoptysis.[6,11]

Diagnosis could be established by chest radiograph, CT chest, bronchography, and ventilation-perfusion ratio scan.[2] Chest radiographs in the affected lung show, scattered opacity with numerous cavities or a large single cavity.[1] Histopathologically, the destroyed lung ends up in extensive fibrosis.[6] In cases with left lung tuberculosis parenchymal destruction is comparatively higher which could be due to the layout and anatomy of the left bronchus, which is longer, narrower, and more horizontal ultimately worsening the voiding of secretions eventually resulting in obstruction.[6,8]

A destroyed lung could be detrimental to the health of the patient, as it could lead to complications that are often irreversible like respiratory insufficiency, massive hemoptysis, empyema, secondary fungal infections, septicemia, and left-right shunt.[1,2] The left-right shunt could result in pulmonary hypertension and respiratory failure even in the presence of a normal contralateral lung.[6] Due to the destruction of the affected lung, the contralateral lungs might show hyperinflation.[2]

Moreover, this condition results in changes in the thoracic cavity such as loss of the ipsilateral lung

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https://doi.org/10.18231/j.ijirm.2023.002

parenchyma which is predominantly replaced by fibrosis, hyperinflation of the contralateral lung, contraction and retraction of the hemithorax with reduced intercostal spaces, a lift of the ipsilateral hemidiaphragm, significant volume loss, and ipsilateral mediastinal shift[6]. In a study by Diego et al. decreased diameter of pulmonary arteries and veins with hypertrophy of ribs and thickening of extrapleural fat was reported in every single study subject.[8]

The diagnosis of a destroyed lung is plain sailing if a previous history of tuberculosis is available.[8] Differentials include some types of mycosis, total lung collapse, chronic pleural disease, previous lung surgery, and pulmonary agenesis/hypoplasia.[8]

There are no formal treatment guidelines for destroyed lung syndrome.[12–14] Medical management involves symptomatic treatment with the use of long-acting muscarinic antagonists or long-acting beta-2 agonists plus inhaled corticosteroids ultimately resulting in bronchodilatory effects[14]. Yum et al. inferred that the tiotropium had a therapeutic effect on tuberculous destroyed lungs.[12,13] In a recent multi-center, double-blind clinical trial, indacaterol was reported to significantly increase forced expiratory volume in 1 second (FEV1) and improved dyspnea compared with placebo.[15] Even though, a high-risk procedure, pneumonectomy could be indicated in the management of destroyed lungs to either resolve or prevent complications.[2]

Destruction of a lung due to pulmonary tuberculosis is widely documented in the literature.[8] It is commonly seen in the concluding phase of progressive disease or disease reactivation.[8] Destroyed lung, especially in unison with bronchiectasis, is effortlessly colonized by bacteria and fungi thereby resulting in substantial morbidity and mortality linked to tuberculosis[8]. In Africa and Asia, around 15.4% of tuberculosis cases are coinfected with Aspergillus or non-tuberculous mycobacteria, with Aspergillus coinfection markedly increasing the mortality risk.[11] In countries that are endemic to tuberculosis, the destroyed lung syndrome is a major issue.[11] The severity and socioeconomic burden of the destroyed lungs in countries with high tuberculosis burden is expected to be high, however, there has been no substantial investigation or large-scale data for the same.[12] Although there is a paucity of literature related to the prevalence and incidence of the destroyed lung in tuberculosis still this is a major contributor to the patient's quality of life.[11,12]

To summarize, destroyed lung syndrome is a permanent complication of lung secondary to chronic infections. Generally, tuberculosis is inactive in these cases but superinfections with nontuberculous bacteria may develop. In underprivileged countries, with a high load of tuberculosis, it is imperative that all post tuberculosis cases must be followed up with diagnostic tools to evaluate functional status (spirometry, plethysmography, and DLCO determination), arterial blood gases, capacity to perform an exercise, investigations for lesions (chest X-ray and CT chest) and efforts should be made at the grassroots level for timely diagnosis and management of such cases.

Conflicts of Interest

None declared

Acknowledgments

None.

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Cite this article: Yadav S, Rawal G. Destroyed lung syndrome. *IP Indian J Immunol Respir Med* 2023;8(1):4-5.