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Review Article

'Destroyed lung' as Post Tuberculosis Sequel: A Preventable Stigma of 'disease of concern' of Millennium!

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Abstract

Pulmonary Tuberculosis is caused by bacilli mycobacterium tuberculosis and known to infect human race since the dawn of history and archaeological evidence has traced its association in neolithic times. Tuberculosis is the most common infectious lung diseases in India with significant mortality and morbidity. Tuberculosis can cause diverse thoracic presentations ranging from nodules, consolidations & cavitation, mediastinal adenopathy, pleural effusion to diffuse endobronchial disease presenting like bronchial asthma. Due to diverse presentations, diagnosis is many times delayed due to lack of suspicion by treating general physicians and rational treatment many not offered in time. In spite of awareness by government organizations and considered as 'global health issue of concern' by World Health organization, tuberculosis is still considered as social stigma. Destroyed lung is described in literature and known complication of pulmonary tuberculosis. Destroyed lung is defined as combination of pleural and parenchymal lung destruction with cavitation, bronchiectasis, loss of lung volume and mediastinal herniation to diseased side. In this case series, we have reported two cases with history of pulmonary tuberculosis in past and received adequate anti-tuberculosis treatment. Both were having residual chronic lung disease and symptoms causing significant impact on quality of life with recurrent hospitalization, hospital visits and cost of care. One patient has history of delayed diagnosis and ATT was started after maximum lung destruction due to tuberculous process has already occurred. In this patient tuberculosis was cured but residual lung damage or sequel presenting as destroyed lung. In second case, tuberculosis was diagnosed in adequate time but patient has defaulted due to adverse events of ATT and he has taken medications as per his own tolerance. Neither adherence nor compliance was acceptable in second case and resulted into partially treated case of pulmonary tuberculosis. Ongoing lung destruction inn second case would be cause for destroyed lung in absence of irrational medicines in today's era of good quality ATT. Destroyed lung is preventable with early diagnosis, prompt evaluation with microscopy and nucleic acid amplification tests and treatment with universally available, acceptable and affordable free ATT as National guidelines. Destroyed lung is having significant impact on quality of life and health expenditure and considered as 'radiological stigma' of Tuberculosis.

Keywords: Pulmonary Tuberculosis, radiological stigma, ATT, destroyed lung, sequel.

INTRODUCTION

Tuberculosis (TB) is a chronic infectious disease that severely affects the health of millions of people each year and is a major public health problem worldwide [1]. TB-related stigma has become a formidable challenge for TB prevention and control [2]. However, there is also a growing awareness of the need to address the stigma related to TB, a major social problem [3]. TB-related stigma has been identified as a major obstacle to patients seeking medical care and completing a full course of treatment [4].

Pulmonary tuberculosis is caused by Mycobacterium tuberculosis when droplet nuclei laden with bacilli are inhaled. In accordance with the virulence of the organism and the defenses of the host, tuberculosis can occur in the lungs and in extrapulmonary organs. A variety of sequelae and complications can occur in the pulmonary and

extrapulmonary portions of the thorax in treated or untreated patients. These can be categorized as follows: (a) parenchymal lesions, which include tuberculoma, thin-walled cavity, cicatrization, end-stage lung destruction, aspergilloma, and bronchogenic carcinoma; (b) airway lesions, which include bronchiectasis, tracheobronchial stenosis, and broncholithiasis; (c) vascular lesions, which include pulmonary or bronchial arteritis and thrombosis, bronchial artery dilatation, and Rasmussen aneurysm; (d) mediastinal lesions, which include lymph node calcification and extranodal extension, esophagomediastinal or esophagobronchial fistula, constrictive pericarditis, and fibrosing mediastinitis; (e) pleural lesions, which include chronic empyema, fibrothorax, bronchopleural fistula, and pneumothorax; and (f) chest wall lesions, which include rib tuberculosis, tuberculous spondylitis, and malignancy associated with chronic empyema. These varieties of radiologic manifestations can mimic other disease entities. Therefore, recognition and understanding of the radiologic manifestations of the thoracic sequelae and complications of tuberculosis are important to facilitate diagnosis [5].

Case summary 1:

42-year-old male, farmer by occupation, ex-alcoholic, normotensive, non-diabetic, referred to our center by family physician for recurrent cough, sputum production and shortness of breath.

Further clinical details-

1. Cough-for last five years dry, intermittent, seasonal exacerbations, diurnal variation with more during early morning and night time, more during sleeping position on right side and associated with copious amount of greenish, yellow and white sputum production.
2. Sputum production for five years, more during night and sleeping on right side. He was having more than a glass of sputum production during night time for five years. He also mentioned yellowish sputum production with foul smell and associated with breathlessness intermittently once in while with deterioration of health. He narrated that his sputum quality and content changes after medical treatment in outdoor or indoor units with family physicians or general physicians.
3. Shortness of breath on exertion for five years. He narrated that his shortness of breath was grade I according to SJRQ and gradually progressed to grade II and III over five years. His symptoms were showing improvement with inhaled bronchodilators and oral medicines including antibiotics for sputum production.
4. Loss of appetite and weight loss over period of three years
5. Weakness and myalgia with fatigability for two years
6. Fever-for 4 months, intermittent, low to moderate grade without chills and rigors associated with minimal body ache and headache. He was treated as case of enteric fever for 2 months by family physician and later one month as bronchial asthma without laboratory workup documentation.

He has brought chest Xray done three months before and shown large fibrocavity in right lung in upper and midzone with changes of fibrosis and bronchiectasis in right lower zone and shift of upper and lower mediastinum towards right side. [Image 1] We have also noted changes of pleuroparenchymal fibrosis in right thoracic cavity. Upper mediastinal shift documented with shift of trachea to right side, and lower mediastinal shift documented with shift of heart to right side (Image 1). Pleuroparenchymal fibrosis also confirmed as loss of thoracic volume in right side and compensatory hyperinflation left side.

Clinical examination documented as-

Restless, dry oral mucosa, cyanosis, pallor, Clubbing (Image 2)

Heart rate-92/min Respiratory rate: 25/bpm, BP-80/60 mmhg

PsO2: 91% @ room air resting and 85% @room air during routine walk

Respiratory system examination revealed- Vesicular breath sounds normal left side & amphoric breathing right side in right mammary and interscapular area. Adventitious sounds as crepitation's heard over right lower axillary and intrascapular area.

Cardiovascular, gastrointestinal & Nervous systems were normal.

We have assessed past records of hospitalization and he narrated chronic constitutional symptoms six years before and received treatment in accordance to symptoms towards enteric fever, jaundice and bronchial asthma with bronchitis. He was treated empirically by family physician and resulted into delay in final diagnosis and actual workup of tuberculosis was done very late after advancement of disease.

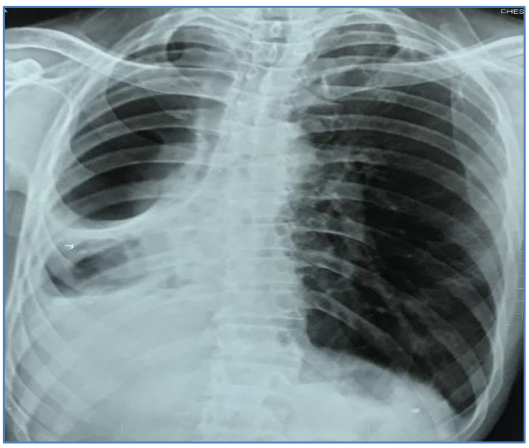

Image 1: Chest x-ray PA showing large fibrocavity in right lung with upper and lower mediastinal shift to right side

Image 2: Showing clubbing

He was diagnosed with advanced pulmonary tuberculosis five years before with sputum microbiological workup shown acid fast bacilli in sputum with CBNAAT positive for MTB genome and negative rifampicin resistance and received treatment for one year of ATT i.e., anti-tuberculosis treatment (6 months RNTCP and 6 months ATT at private hospital). He was declared bacteriologically cured after completion of treatment. His chest Xray before and after completion of treatment was not available but patient disclosed that his symptoms were persistent even after completion of treatment and his treating physician mentioned residual lung disease after treatment.

Case summary 2:

49-year-old female, farmer by occupation, no addiction history, normotensive, non-diabetic, referred to our center for recurrent hospitalization due to worsening of existing chronic lung disease of 12 years duration.

Further clinical details-

He was having recurrent cough, fever and shortness of breath for 12 years before and having history of ATT for one year with diagnosis of sputum positive pulmonary tuberculosis. He was treated with standard protocol at government center. He was a defaulter of ATT management and took only four months of ATT in overall duration of one year. He disclosed the issue for noncompliance and nonadherence to ATT was thrice weekly government protocol as per National guidelines which was very hot tablets and had recurrent nauseas-vomiting & fever which resulted into once weekly, one seven tablet blister & one table daily for seven days in spite of one blister in a day. He is very genuine in his illness, diagnosis and serious regarding his illness, but due to lack of counselling he followed protocol.

He has brought chest X-ray done one months before and shown small fibrocavity with cystic changes in right lung in lower zone with changes of fibrosis and bronchiectasis in right upper and midzone and shift of upper and lower mediastinum towards right side (Image 3). We have also noted changes of pleuroparenchymal fibrosis in right thoracic cavity. Upper mediastinal shift documented with shift of trachea to right side, and lower mediastinal shift documented with shift of heart to right side (Image 3). Pleuroparenchymal fibrosis also confirmed as loss of thoracic volume in right side and compensatory hyperinflation left side.

Clinical examination documented as-

Thin built, cachexic, pallor present, no cyanosis and no clubbing

Heart rate-102/min Respiratory rate:21/bpm, BP-100/60 mmhg

PsO2: 96% @ room air resting and 93% @room air during routine walk

Respiratory system examination revealed- Vesicular breath sounds heard bilateral lung fields. Adventitious sounds as crepitation's and rhonchi heard over right side of thorax in all zones.

Cardiovascular, gastrointestinal & Nervous systems were normal.

We have advised sputum examination and which is negative for acid fast bacilli and CBNAAT (cartridge based nucleic acid amplification test) were negative for MTB genome. We have advised HRCT thorax for assessment of underlying lung parenchymal involvement including pulmonary vasculature due to lung parenchymal destruction and chronic hypoxia.

HRCT Thorax suggestive of- (Images 4-6)

1. Moderate sized caviatary disease in right lower lobe with pericavitary cystic opacities without air fluid level. Cavity with cystic bronchiectasis in right lower lobe. Right thoracic cavity is significantly decreased in size with pleuroparenchymal fibrosis as seen in sagittal section (Image 4).
2. Right lung volume loss with shift of midline towards right side with complete anterior mediastinal herniations towards right side. Cystic bronchiectasis documented middle lobe, medial basal and posterior basal segments of right lower lobe. Compensatory hyperinflation of left lung and enlargement of airspaces in left upper and lower lobes in such a way that left lung also herniated in right thoracic cavity. Mediastinal structures such as trachea, bronchi, pulmonary vasculature, heart and liver has been completely shifted to right side, i.e., total mediastinal shift with herniation (Image 5).
3. Cystic bronchiectasis in right upper & middle lobe and right lung volume loss with shift of midline towards right side with complete anterior mediastinal herniations towards right

side with pleuroparenchymal fibrosis as seen in coronal section. Compensatory hyperinflation of left lung and enlargement of airspaces in left upper lobe & lingula in such a way that left lung also herniated in right thoracic cavity. Mediastinal structures such as trachea, bronchi, pulmonary vasculature has been completely shifted to right side, i.e., total mediastinal shift with herniation. All bronchial cut sections such as main stem at carina, right and left main stem bronchi seen herniated to right side (Image 6).

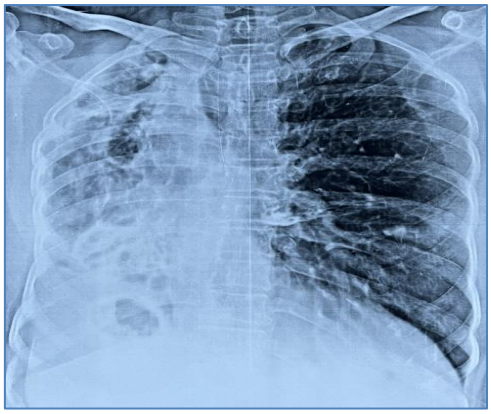

Image 3: Chest X-ray PA view showing bronchitis, Fibrosis and mediastinal shift right lung

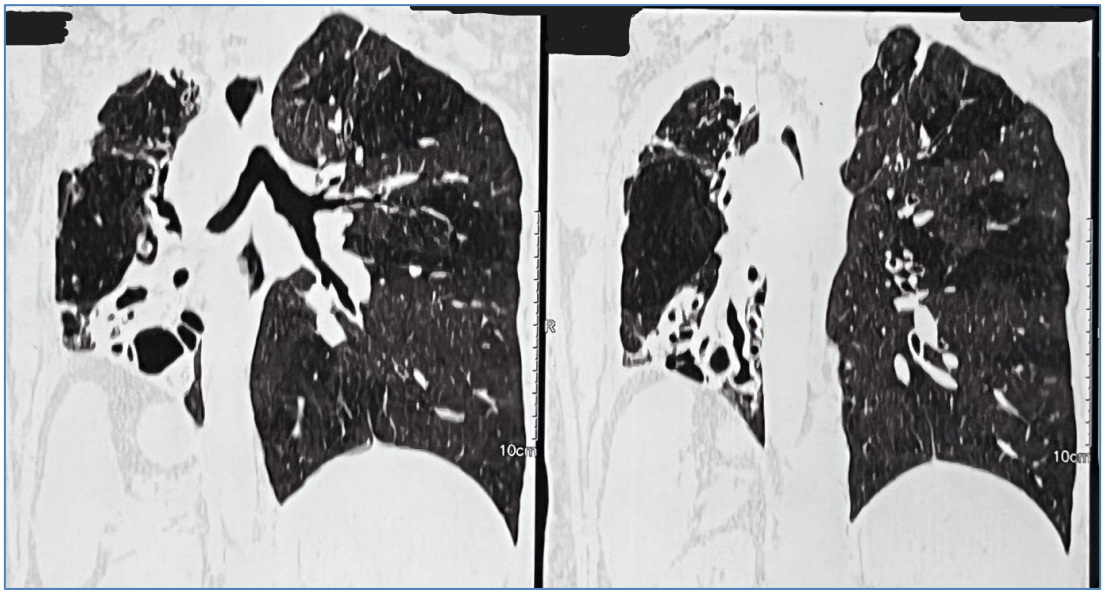

Image 4: HRCT thorax showing cavity with cystic bronchiectasis in right lower lobe

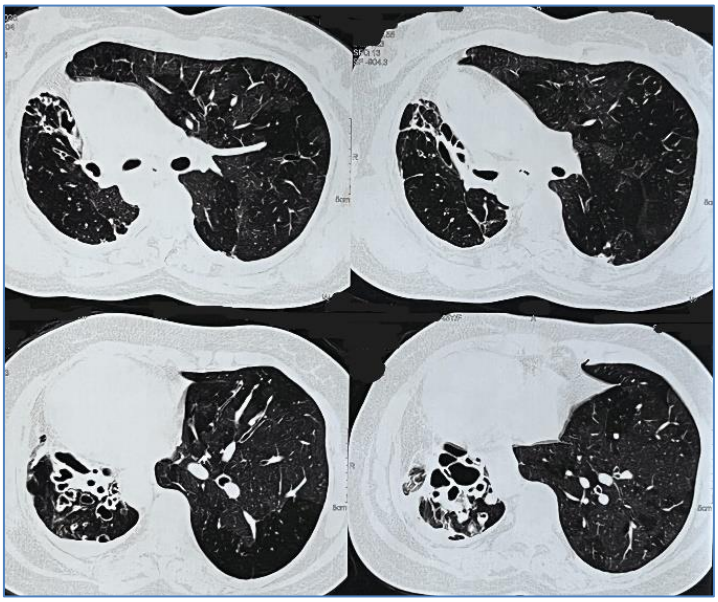

Image 5: HRCT thorax suggestive of Right lung volume loss with shift of midline towards right side with complete anterior mediastinal herniations towards right side

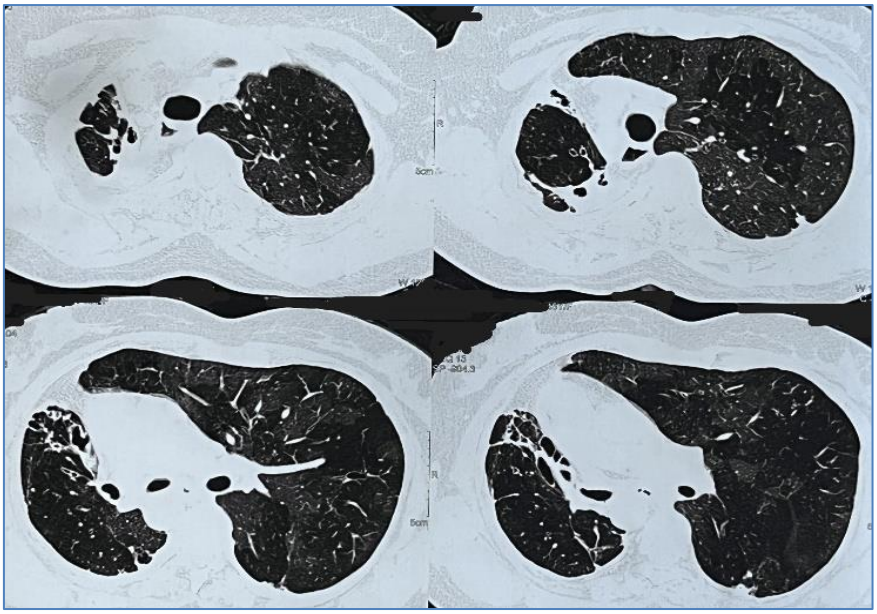

Image 6: HRCT thorax showing all bronchial cut sections such as main stem at carina, right and left main stem bronchi seen herniated to right side

During hospitalization, we have started supportive care till final reports came with intravenous fluids and beta-lactum antibiotics. Sputum bacterial culture was done to identify chronic bacterial colonization as pseudomonas and staphylococcus in first patient and klebsiella in second patient and offered antibiotics according to culture sensitivity. After 10 days of antibiotics both cases were discharged on oral medicines as a hospital protocol-

- Dry powder inhaler of salmeterol plus fluticasone 250 microgram two times
- Dry powder inhaler of Tiotropium 18 microgram one time
- Tablet acebrophylline 200 mg bed time daily
- Tablet N-acetyl cysteine 600 mg one time daily in glass of water.
- Postural drainage for sputum clearance and chest physiotherapy and breathing exercises with incentive spirometry.
- High protein diet and regular walk for maximum tolerance.
- Influenza vaccination annually
- Pneumococcal vaccine every five years.

Discussion & literature of review:

A third of the world's population is infected with Mycobacterium tuberculosis (MTB), and over 9 million new cases of tuberculosis (TB) are reported annually [1]. However, up to half of TB survivors have some form of persistent pulmonary dysfunction despite microbiologic cure. Pulmonary dysfunction, ranging from minor abnormalities to severe breathlessness, can increase the risk of death from respiratory causes [6-10].

Definitions for processes contributing to lung remodelling during pulmonary tuberculosis (TB) and pulmonary impairment after TB- [11]

1. Pulmonary cavitation- Process by which normal pulmonary tissue is obliterated, becoming gas-filled spaces or cavities in the lung. This process initially involves caseous necrosis of lipid pneumonia lesions, producing caseous pneumonia. During caseation, alveolar cells and septa are destroyed along with neighbouring vessels and bronchi. Cavities form when these regions of caseous pneumonia liquefy, fragment and are released upon coughing.
2. Pulmonary fibrosis- Results from long-term lung tissue injury that is characterised by excessive extracellular matrix deposition in the lung. Replacement of normal lung parenchyma with collagenous tissue results in architectural changes in the lung, such as thickening and stiffening of the lung walls.
3. Bronchiectasis- Manifests as irreversible bronchial dilatation and thickening of the bronchial wall. Elastic and muscular components of the bronchial wall are destroyed in bronchiectasis. Bronchial dilatation associated with bronchiectasis in TB may be due to multiple factors, including traction from surrounding tissue fibrosis, caseous necrosis that makes its way into the bronchi, and elevated luminal pressure due to coughing. Bronchiectasis can also predispose to recurrent exacerbations of purulent sputum production and possibly bacterial pneumonia in subsequent years.
4. Pulmonary impairment after TB- A broad term we use in this review to refer to lung

dysfunction that includes airflow obstruction, restrictive ventilatory defects and impaired gas exchange. Pulmonary impairment after TB is probably downstream of a wide variety of lung remodelling events, some of which are described above. Given the lung's considerable reserve, these structural changes may manifest as symptoms and pulmonary disability over a period of time.

Destroyed lung:

The expression of "destroyed lung" is, now, accepted to designate the large destructions of the lung, secondary to pulmonary and essentially infectious diseases, the cure of which is obtained but with important sequelae. The main cause remains tuberculosis, cured by chemotherapy. Some large pulmonary suppurations, treated by antibiotics, can lead to the same sequelae. These "destroyed lungs" can keep an asymptomatic form. But often, about ten years after the initial disease, they cause several troubles such as progressive dyspnea leading to irreversible respiratory insufficiency, repeated pulmonary infectious episodes and haemoptysis, the risk of which is increased by aspergillosis. The radiological aspect of these "destroyed lungs" is made of opacities with multiple cavities or with one unique large cavity.

Destroyed lung is defined as combination of pleural and parenchymal lung destruction with cavitation, bronchiectasis, loss of lung volume and mediastinal herniation to diseased side.

Components of destroyed lung-

1. Pulmonary Cavitations
2. Cystic bronchiectasis
3. Loss of lung volume
4. Pleuroparenchymal fibrosis
5. Unilateral near complete lung parenchymal abnormalities with combinations of above findings
6. Contralateral lung parenchymal compensatory hyperinflation manifested as emphysema
7. Pull of contralateral lung and mediastinal structures to diseased side radiologically documented as mediastinal herniation.

Mediators of lung damage and dysfunction in TB [11]

Targeted treatment of TB-associated lung impairment requires knowledge of the precise mechanisms of immune pathology. A major barrier to studying TB immunopathogenesis longitudinally in humans is that serial lung biopsies through disease progression and treatment, which could be used to determine local immune pathways involved in tissue injury, are nearly impossible to obtain. Nevertheless, human studies and data from animal models provide compelling evidence for the crucial role of the host's immune response to MTB in lung remodelling shown in Figure 1.

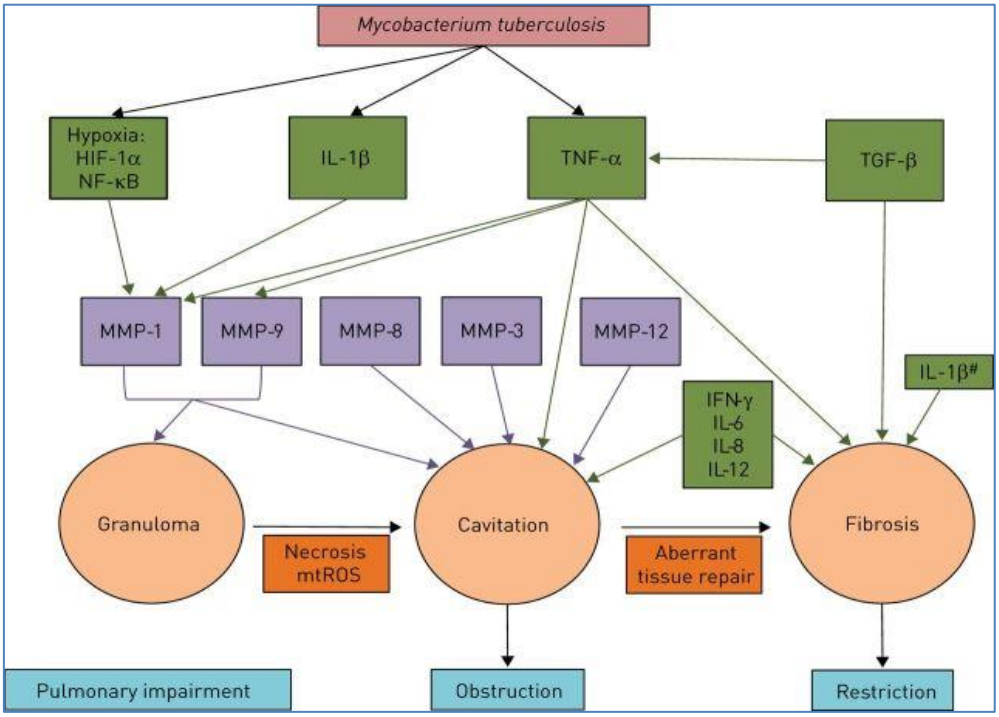

Figure 1: Immune mediators of tissue remodelling and lung function impairment in tuberculosis. (Matrix metalloproteinases (MMP) that promote granuloma and cavitation are depicted in purple. HIF: hypoxia inducible factor; NF: nuclear factor; IL: interleukin; TNF: tumour necrosis factor; TGF: transforming growth factor; IFN: interferon; mtROS: mitochondrial reactive oxygen species)

Lung destruction and bronchial tree anomalies [12]

Pulmonary tuberculosis infection leads to lung destruction through a process of cavitation, spread of disease to other areas and secondary development of fibrosis [13]. Lung destruction due to primary progressive TB is generally unilateral, with upper lobe predominance [13]. The presence of peribronchial or hilar lymph adenopathies, a common finding in primary infection that is occasionally accompanied by endobronchial injury, can give rise to bronchial obstruction. The lymph adenopathies may disappear, while the bronchial stenosis persists. Bronchial obstruction due to caseum, pus or an excess of mucus can lead to a secondary pyrogenous infection or lobar collapse, with resulting atelectasis due to fibrosis of a lobe (partial) or the entire lung. [12] Lung destruction due to TB reactivation presents differences relative to the primary disease. It consists of pulmonary dissemination and secondary fibrosis, and the hemithorax contralateral to the infection is often affected [13].

Although the posterior and apical segments of the upper lobes are the most common sites of pulmonary TB, with a similar incidence on both sides, lung destruction due to primary or post-primary disease most commonly occurs on the left side, in the same way as the fibrotic phase of endobronchial TB [14]. This finding was observed in 11 patients in our review (85%), in agreement with other retrospective studies in which left-side predominance ranged from 63% to 80% [15-17]. The explanation resides in the anatomy of the left pulmonary bronchus, which crosses a narrow anatomical space, the aorto-pulmonary window, and is longer and smaller in diameter than the main right bronchus [18]. These factors all favour bronchial collapse due to adjacent lymph adenopathy. In addition, the more horizontal course of the main left bronchus can have an effect on drainage of secretions, favouring obstruction. In our case series we have documented destroyed lung on right side.

Tuberculosis infection tends to be inactive in cases of lung destruction, making the diagnosis more difficult. The microbiological study of secretions often shows other microorganisms [13], which may be of bacterial origin, such as those described in our review and indicating processes of superinfection. The isolated microorganisms in our series (Pseudomonas aeruginosa, Streptococcus pneumoniae, and Haemophilus influenza) are consistent with the findings from a prospective study in hospitalised patients with acute COPD exacerbations [19] and similar to previous literature [20]. Similarly in our case series we have documented colonization due to similar microbiological patterns resulting into clinical and radiological worsening.

CONCLUSION

In our study we have documented case series with destroyed lung on right side of thoracic cavity. Patients presented with chronic respiratory symptoms and significantly affected quality of life with increased cost burden to control chronic respiratory illness in post tuberculosis care. Usually, these patients are having recurrent exacerbations and sputum culture showed evidence of colonization with polymicrobial flora and needs targeted antibiotic therapy to control and prevent further exacerbations. Lung function abnormalities would have both restrictive and obstructive mixed pattern due to pleuroparenchymal fibrosis on one side and compensatory hyperinflation on other side. Combination of inhaled medicines such as bronchodilators, inhaled corticosteroids and inhaled antimuscarinic agents with oral mucolytics and bronchodilators with short course of antibiotics is the protocolised approach during routine care of cases with destroyed lung.

Key learning points from this case report are:

1. Pulmonary tuberculosis has significant impact on mortality and morbidity if timely diagnosis is not done and rational treatment is not initiated. ‘First hit should be best hit’ in diagnosis and management of tuberculosis to prevent further complications which will affect entire life due to permanent lung damage or sequel due to partially treated tuberculosis.
2. Pulmonary tuberculosis can present with consolidation, cavitation, bronchitis, pleural effusion and mediastinal adenopathy.
3. All complications of pulmonary tuberculosis except bronchiectasis and fibrosis is irreversible and irreparable.
4. Destroyed lung is defined as combination of pleural and parenchymal lung destruction with cavitation, bronchiectasis, loss of lung volume and mediastinal herniation to diseased side.
5. Delayed diagnosis and late in initiation of ATT in cases with maximum lung destruction due to tuberculous process resulted in cure of TB but residual lung damage or sequel presenting as destroyed lung.
6. In spite of timely diagnosis and early start of ATT, destroyed lung is possible outcome and presenting as sequel in cases with noncompliance to medications. In second case, tuberculosis was diagnosed in adequate time but patient has defaulted due to adverse events of ATT and he has taken medications as per his own tolerance. Neither adherence nor compliance was acceptable in second case and resulted into partially treated case of pulmonary tuberculosis resulted into destroyed lung.
7. Ongoing lung destruction inn second case would be cause for destroyed lung in absence

of irrational medicines in today's era of good quality ATT.

8. Lung function impairment is known to occur after PTB irrespective of duration of treatment and outcome of disease.
9. Rational treatment as per National guidelines is must to prevent deadlier complication as 'radiological stigma' as 'destroyed lung'.
10. More awareness is required regarding symptoms, early diagnosis, benefits of rational treatment, cost effectiveness of treatment, free and universally available ATT as per NTEP and beneficial role of all these in preventing 'radiological stigma' and destroyed lung which has negative impact on overall outcome.

Conflicts of Interest: Nil

Research Funding: Nil

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