Complications of Pulmonary Tuberculosis

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1 Introduction

Tuberculosis is an ancient disease which continues to be a major health problem in most developing countries. In developed countries, it is gaining importance with the emergence of HIV pandemic. Pulmonary tuberculosis (TB) is caused by the bacteria *Mycobacterium tuberculosis*. According to the defenses of the host and virulence of the organism tuberculosis can occur in the lungs or extrapulmonary organs. Various forms of sequelae and complications may result from primary and post-primary pulmonary tuberculosis in both treated and untreated patients (Kim *et al.*, 2001). *Mycobacterium tuberculosis* can affect almost every organ in the body. Major manifestations of the disease usually occur in the lung. Pulmonary tuberculosis can manifest for the first time as a complication (SatyaSri, 2009). A variety of complications can occur in pulmonary tuberculosis. They can be categorized as follows: (a) Parenchymal lesions which include thin walled cavity (open negative syndrome), aspergilloma, end stage lung destruction and scar carcinoma. (b) Airway lesions which include tuberculous laryngitis, bronchiectasis, tracheobronchial stenosis, anthracofibrosis and broncholithiasis. (c) Vascular lesions such as Rasmussen aneurysm. (d) Pleural lesions which include dry pleurisy, pleural effusion, empyema, bronchopleural fistula and pneumothorax (Kim *et al.*, 2001). (e) General complications include cor pulmonale, secondary amyloidosis and chronic respiratory failure (see Table 1). This chapter describes the pathogenesis, clinical manifestations, diagnostic criteria and management of complications of pulmonary tuberculosis.

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Table 1: Complications of Pulmonary Tuberculosis
2 Parenchymal lesions

2.1 Open Negative Syndrome

Cavitation is a hallmark of pulmonary tuberculosis. Residual thin walled cavities may be seen in both active and inactive disease. The cavity may disappear after antituberculous chemotherapy. Occasionally the wall becomes thin and remains as an air filled cystic space even after a bacteriological cure, when it is called as open negative syndrome. Tuberculous cavities heal by two processes, open and closed depending on the status of the draining bronchus. In the open form, the wall of the cavity becomes free of tubercle bacilli with chemotherapy and wall undergoes fibrosis with subsequent epithelialization. The lumen of the draining bronchus usually remains patent (Hermel & Gershon-Cohen, 1954).

These are thin walled cavities with epithelialization extending from bronchioles. The wall may be smooth and the thickness varies from 1 cm to less than 1mm. These cavities may be associated with complications such as haemoptysis, secondary infection with pyogenic organisms resulting in a lung abscess. Aspergilloma can form in a cavity. Rupture of the cavity can give rise to a spontaneous pneumothorax. Rarely relapse of tuberculosis can occur. In the closed type, the draining bronchus gets occluded and the cavity undergoes atelectasis and scar formation (Hermel & Gershon-Cohen, 1954).

2.2 Aspergilloma

The most common cause of human aspergillosis and pulmonary aspergilloma is Aspergillus fumigatus. Published data indicate that saprophytic colonization of the lung that has been destroyed by tuberculosis, sarcoidosis, bronchiectasis, lung abscess & neoplasms lead to intracavitary aspergilloma. Of these tuberculosis is the most common. Most patients are asymptomatic. Symptomatic patients present with fever, cough and haemoptysis. Haemoptysis could be life threatening. The source of bleeding is usually the bronchial blood vessels. Dyspnea, malaise and weight loss may be there due to the underlying pulmonary disease. Physical examination may be normal or signs of the underlying lung disease may be present. The diagnosis is based on the characteristic radiological finding. Chest radiography reveals a mass in a pre-
existing cavity with a crescent of air outlining the mass called as ‘air crescent sign’ (Felson, 1988), seen usually in the upper lobe as shown in the image. CT scan images provide better definition of the mass within a cavity.

![Image of chest x-ray and CT scan]

*Figure 2: Chest X-ray PA and CECT Thorax showing right upper lobe aspergilloma.*

Laboratory abnormalities are uncommon. Aspergillus precipitin antibody test for IgG are usually positive in Aspergilloma (Harman, 2012). Treatment is considered when patients develop symptoms, usually presenting as haemoptysis. Bronchial artery embolization may be used for patients with massive haemoptysis. Prolonged oral itraconazole may provide partial or complete resolution of aspergillomas in 60% of patients. Itraconazole has a high tissue penetration (Kousha *et al.*, 2011). In a small number of patients intracavitary therapy with Amphotericin B has also been used with some success (Klein *et al.*, 1993). Surgical resection is curative and may be considered for patients with recurrent haemoptysis if the patient has adequate pulmonary reserve.

### 2.3 Destroyed Lung

Unilateral destruction of the lung due to tuberculosis has been a recognized entity. Rajasekaran *et al.* analyzed patients with unilateral lung destruction and found pulmonary tuberculosis as the cause in 83.3% of patients (Rajasekaran *et al.*, 1999). It may occur after primary disease or re-infection. Cicatization atelectasis is a common finding after postprimary tuberculosis. Marked fibrotic response is seen in up to 40% of patients with post primary tuberculosis (Kim *et al.*, 2001). Reduced lung volume, cavities, bronchiectasis and fibrosis are the predominant findings in destroyed lungs. Fibrotic response manifests as retraction of the hilum and mediastinal shift towards the fibrotic lung. Opposite lung will show compensatory hyperinflation. These patients might seek medical help for the first time for diagnosis or after completion of treatment or referred as non-responders due to treatment failure.
2.4 Scar Carcinoma

The development of lung cancer in a pulmonary scar was first described by Friedrich as scar or cicatricial cancer of the lung. Scar cancers are associated with high incidence of adenocarcinoma (Ashizawa et al., 2004). Ying U et al studied the relationship between lung cancer risks following detection of pulmonary scarring and found that baseline diagnosis of pulmonary scarring was associated during subsequent follow up with an increased risk of lung cancer (Yu et al., 2008). Lung cancer occurring in pulmonary tuberculosis patients after completing treatment are mistaken for relapse. Although the pathogenesis is unclear uncontrolled epithelial hyperplasia in relation to fibrosis is considered as a possible mechanism. Other possibility is the concentration of carcinogens by scars (McDonnell & Long, 1981). Cigarette smoking contributes to the lung cancer risk associated with scarring.
3 Vascular Lesions

3.1 Haemoptysis

Haemoptysis is defined as expectoration of blood originating from the lungs or tracheo-bronchial tree (Jean-Baptiste, 2005). Haemoptysis is a common symptom of pulmonary tuberculosis (Sharma, 2009). Haemoptysis is most commonly associated with cavitary lesions. Changes in the vasculature of tuberculous lungs are implicated in haemoptysis. Fritz Valdemar Rasmussen, a Danish physician described the aneurysmal dilatation of pulmonary vessels in the wall of tuberculous cavity as the cause for haemoptysis (Rasmussen, 1868). Calmette in 1923 mentioned that branches of pulmonary artery which traverse the wall of tuberculous cavities contain small pedunculated “aneurysms of Rasmussen”. Wood & Miller showed that the wall of tuberculous cavities have dilated bronchial arteries (Wood & Miller, 1938).

Haemoptysis may occur in the active stage of tuberculosis or as late sequelae, due to aneurysmal rupture or secondary to bronchiectasis. Aspergillomas can form in pre-existing cavitary lesions and lead to haemoptysis. Erosion of a blood vessel by a calcified lesion can also lead to haemoptysis. Definition of massive Haemoptysis is not clear. It can range from 100 milliliters per day to 1000 milliliters over a few days (Lordan, 2003). Massive haemoptysis can be fatal and lead to asphyxiation. Majority of patients with massive haemoptysis have a poor pulmonary reserve and are not fit for any surgical procedures. The lungs have a dual blood supply. The pulmonary arterial circulation is a low pressure system and bronchial arteries, a high pressure system. The source of massive haemoptysis is most commonly the bronchial circulation (90%) and pulmonary circulation constitutes only 5% (Remy et al., 1992). Patients with mild haemoptysis need a conservative approach. Treatment options available for patients with massive haemoptysis are endobronchial tamponade, single or double lumen intubation, bronchial artery embolization, or surgery (Lordan, 2003). Presently, the main life-saving modality used to control bleeding in massive
haemoptysis is angiography and bronchial artery embolization (Sopko et al., 2011). Shin et al. have retrospectively analyzed the outcomes of bronchial artery embolization for the treatment of haemoptysis in patients with pulmonary tuberculosis and reported 163 patients out of 169 (96.4%) had significant clinical improvement after the procedure (Shin et al., 2011).

Figure 6: Chest X-ray PA view showing bilateral cavitary lesions.

4 Airway Lesions

4.1 Tuberculous Laryngitis

Laryngeal tuberculosis is usually a complication of pulmonary tuberculosis. The sputum positive rate is 90-95% in patients with active pulmonary tuberculosis with tuberculous laryngitis. Clinical features include hoarseness of voice, odynophagia, cough and dysphagia. The larynx becomes infected either by a direct contact from pulmonary tuberculosis or by a lymphatic/ hematogenous spread from sites other than lungs. It can manifest as edema, hyperemia, ulcerative lesion, nodule or an exophytic mass. Most common site is posterior part of larynx. Laryngeal TB is uncommon in developed countries and authors from developed countries feel that it should be considered as a differential diagnosis in any laryngeal disease in particular laryngeal carcinoma. (Smulders et al., 2009). Antituberculous drugs are the primary treatment for laryngeal tuberculosis. Complications of laryngeal tuberculosis if not treated early can result in subglottic stenosis and vocal cord paralysis.

4.2 Bronchiectasis

Bronchiectasis is an abnormal and permanent dilatation of bronchi. Tuberculosis is a major cause of bronchiectasis worldwide. Bronchiectasis may be present in various stages of pulmonary tuberculosis. Bronchiectasis is seen in 30% -60% of patients suffering from active post primary tuberculosis. Various factors may cause bronchiectasis in pulmonary tuberculosis, mainly atelectasis and pulmonary fibrosis. Bronchial stenosis as a consequence of tuberculous inflammation and scarring of the bronchi can lead to
retention of secretions. When this is followed by bacterial infection it can lead to destruction and dilatation of the airways.

Compression of the bronchi by enlarged lymph nodes produces consequences similar to intraluminal obstruction. Bronchiectasis may follow as sequelae of healing of pulmonary tuberculosis. Fibrosis of lung parenchyma can lead to bronchial dilatation. Parenchymal retraction due to fibrosis can also cause bronchial dilatation and bronchiectasis. Three types of bronchiectasis have been described, cylindrical bronchiectasis wherein the involved bronchi appear uniformly dilated, varicose bronchiectasis, where the affected bronchi have a beaded pattern of dilatation resembling varicose veins, saccular (cystic) bronchiectasis which shows ballooned appearance of the bronchi ending in blind sacs.

In adults tuberculosis usually occurs in the upper lobes (Crofton, 2000). Other infections often involve dependent parts of the lungs. When upper lobes are involved secretions are drained and superinfection with pyogenic organisms does not usually occur. This is known as dry bronchiectasis or bronchiectasis sicca. Bronchiectasis in the apical and posterior segments of the upper lobe is highly suggestive of tuberculous etiology (Kim et al., 2001). The main complaints are persistent or recurrent cough, purulent sputum, haemoptysis, dyspnea, wheezing, fatigue, fever and failure to thrive. Haemoptysis is a common symptom in 50 – 70% of cases of bronchiectasis and is more common in dry bronchiectasis. On physical examination, clubbing may be present. Coarse crepitation and rhonchi may be heard over the area of bronchiectasis.

Chest radiograph may show nonspecific findings. The definitive diagnosis is made by thoracic CT. HRCT has replaced lipiodol bronchography to establish the presence, severity and distribution of bronchiectasis. HRCT has only 2% false negative and a 1% false positive rate (Young et al., 1991). It is a noninvasive investigation.

Management may be medical or surgical. Medical management is with antibiotics and chest physiotherapy. If the lesion is localized to a resectable area and the medical management fails, elective surgery is indicated.
4.3 Broncholithiasis

The presence of calcified lymph node masses within the bronchi is referred as broncholithiasis (Fishman et al., 2008). This is an uncommon complication of pulmonary tuberculosis (Ann Leung, 1999). Broncholithiasis is caused commonly by erosion and extrusion of a calcified adjacent lymph node into the bronchial lumen. Symptoms may include cough, haemoptysis, lithoptysis or symptoms related to bronchial obstruction (Seo et al., 2002). Chest radiography often does not show the calcification within the bronchus. CT usually provides useful information (Seo et al., 2002). Radiologic findings include calcified peribronchial nodes. In addition to this there may be segmental or lobar atelectasis, obstructive pneumonitis and rarely focal hyperinflation (Seo et al., 2002). If a broncholith is free completely within the bronchus, it can be removed bronchoscopically. Most of the symptomatic broncholiths should be removed at thoracotomy by lobectomy or segmentectomy (Fishman et al., 2008).

4.4 Tracheobronchial stenosis

The most common cause of benign tracheobronchial stenosis is endobronchial tuberculosis in Asian countries (Ryu et al., 2006). Endobronchial tuberculosis is caused either by direct inoculation of the bacilli from the lung parenchymal lesions or by infiltration of the airway by bacilli from adjacent mediastinal lymph nodes (Wan et al., 2002). Despite adequate anti-tuberculosis treatment endobronchial tuberculosis can result in major airway obstruction from stenosis. Studies have found predominance in the female sex (Low et al., 2004). Significant bronchial stenosis of major bronchi is rare (Fishman et al., 2008). Left main bronchus is the common site of stenosis. This could be because the left main stem bronchus is easily compressed by the arch of the aorta and lymph node TB is also more often noted on the left side (Low et al., 2004). CT scans show concentric narrowing of the lumen, uniform thickening of the wall and long bronchial segment involvement (Kim et al., 2001). Flexible bronchoscopy is the most useful modality for diagnosis and assessment of tracheobronchial stenosis (Hoheisel et al., 1994). Minimally invasive endoscopic techniques such as balloon dilation, Nd YAG laser therapy and tracheo-bronchial stent placement are used to establish patency (Verma, 2006). The use of silicone stents could provide an effective means of managing PTTS patients (Ryu et al., 2006). In subjects not responding to interventional bronchoscopic treatment surgical resection may be indicated (Tetikkurt, 2008).

4.5 Anthracofibrosis

Anthracofibrosis is bronchial stenosis due to local mucosal fibrosis that also presents anthracotic pigment in the mucosa. The exact cause has not been well defined. There is a frequent association with tuberculosis and exposure to smoke from biofuel or biomass combustion (Julio et al., 2012). It is a bronchoscopic finding. Hwang J. et al have studied the frequency of anthracofibrosis in foreign born Pulmonary TB patients in Canada. According to them patients from the Indian subcontinent were more likely to have anthracofibrosis compared to patients from other Asian countries. Most of the patients present with cough, sputum and dyspnea. CT and bronchoscopy has to be done to exclude bronchogenic carcinoma if chest radiographs show segmental or lobar atelectasis (Kim et al., 2000).
5 Pleural Lesions

5.1 Pleural Effusion

Inflammation of the pleura is called as pleurisy and when fluid accumulates in the pleural cavity, it is called as pleural effusion. Tuberculous pleural effusion remains as the leading inflammatory pleural disease in countries where tuberculosis is more common. Although tuberculosis is considered as a chronic illness, tuberculous pleuritis can present as an acute illness of less than 1 week duration. In USA, 2% of all effusions are attributed to tuberculosis (Fishman, 2008). In children, effusion may occur as part of primary tuberculosis. It also occurs with increased frequency in middle-aged and elderly people. Pleural effusion usually occurs 3-6 months after the primary infection (Wallgren, 1948). It occurs when the subpleural parenchymal focus or a caseating lymph node ruptures into the pleural space. Delayed hypersensitivity plays an important role in tuberculous pleural effusion. Direct contiguous spread to the pleura or haematogenous spread can also lead to pleural effusion (SatyaSri, 2009). The usual presenting symptoms are non-productive cough, fever, pleuritic chest pain and breathlessness in most patients. Patients with chronic infection present frequently with weight loss, malaise and dyspnea. Physical findings reveal dullness on percussion and absence of breath sounds. Tuberculous pleural effusions are usually unilateral and in approximately one-third of patients co-existing parenchymal disease is seen radiologically. Pleural effusions generally appear as dense homogeneous opacities. Chest radiograph will show blunting of the costophrenic angle with small quantities of free pleural fluid. Ultrasonography of the chest detects smaller effusions. Tuberculous pleural effusion is often unilateral and occupies about one-third to half of the hemithorax. Radiologically, it appears as a lateral opacity concave medially extending upwards in the axilla with the base obscuring the hemidiaphragm. It can also present rarely as massive pleural effusion obscuring the whole hemithorax and displacing the mediastinum to the opposite side. Sub-pulmonic effusions may present with fluid collection below the diaphragm in some patients.

![Figure 8: Chest X-ray PA view showing massive left pleural effusion.](image)

A tuberculous pleural effusion is usually a serous exudate with high protein level (>4 gm/ml). The fluid is defined as an exudate if it fulfils one of the Light’s criteria such as pleural/serum ratio of total protein >0.5, pleural/serum ratio of LDH >0.6, or LDH greater than 2/3 of the upper limit of serum value. Adenosine deaminase (ADA) level greater than 70 IU/L has been shown to be highly sensitive and spe-
cific for the diagnosis of tuberculous pleural effusion. Increased ADA levels have also been found in malignancy or empyema. Low glucose (<60 mg/ml) and low pH (<7.30) are found in approximately 20% of patients (SatyaSri, 2009). Analysis of the pleural fluid for differential cell count shows predominance of lymphocytes in tuberculous pleural effusion. Pleural fluid smear is positive for acid fast bacilli in less than 10% of patients (SatyaSri, 2009). Culture of pleural fluid is often negative for tubercle bacilli. Chances of positive culture increases in proportion to the quantity of fluid sent to the laboratory. Pleural biopsies show granulomas in about two-thirds of patients. Pleural aspiration and pleural biopsy using Abrams punch can be done in one sitting. Treatment involves pleural fluid aspiration and administration of anti-tuberculosis drugs. Therapeutic aspiration of pleural fluid will relieve symptoms of dyspnea. Aspiration should be done relatively slowly. Rapid removal of large quantity of pleural fluid may result in unilateral pulmonary edema due to increased microvascular permeability in the expanded lung.

5.1.1 Empyema

An empyema is pus in the pleural cavity (Light, 2006). In the developing world, tuberculosis remains as the common cause of empyema. Studies done in India have shown tuberculous empyema as the common cause of empyema (Kundu, 2010). Tuberculous empyema occurs usually as a result of the rupture of a subpleural caseous focus into the pleural cavity. Rarely, hematogenous spread from the involved thoracic lymph nodes or from a sub diaphragmatic focus can cause empyema. The presenting clinical symptoms are fever, cough with expectoration, chest pain and breathlessness. On examination, patients might have digital clubbing due to chronic secondary infection by pyogenic organisms. Tenderness of the chest wall is seen in some of the patients. Radiological investigation shows a dense homogeneous opacity of the hemithorax. Broncho-pleural fistula is a common complication in empyemas of tubercular origin, when there is a delay in treatment. Development of bronchopleural fistula is characterized by expectoration of a large quantity of sputum and volume depends on the position of the patient.

If the patient develops bronchopleural fistula, it can present as pyopneumothorax. Pus aspirated from the pleural cavity can be positive for acid fast bacilli. Secondary infection by staph, streptococcus, pseudomonas and K. pneumoniae are common. If tuberculous empyema is not treated properly, it may spontaneously perforate the pleura and extend through fascial planes and collect in the chest wall. This condition is known as ‘empyema necessitates’. It is an unusual complication of empyema. Other sites of extension of empyema include the paravertebral soft tissue and retroperitoneum. Rarely, pus can track down posterior to the diaphragm and will accumulate in the groin or lumbar region (Behera, 2010). Chest x-ray shows a soft tissue mass in the chest wall with or without bony destruction in empyema necessitates. Contrast enhanced CT is helpful in identifying the direct communication between the pleural and the chest wall collection. Management consists of appropriate antibiotics and anti-tubercular treatment. Repeated aspirations are attempted in the initial stage, but intercostal tube drainage is required to remove the pus, since the chances of developing pleural thickening are more with empyema. In some patients surgical debridement of the pleura with physical removal of loculation, fibrous septae and thick exudate is required. Minimal access surgery using video-assisted thoracoscopy techniques have succeeded in achieving equivalent results to open thoracotomy in the past few years (Wait et al., 2007). Surgical procedures like thoracoaplasty and decortication are required in some cases. Decortication via thoracotomy is the standard treatment method for chronic empyema. Decortication is an elective surgical procedure, in which fibrous wall of the empyema cavity, the cortex or rind is stripped off the visceral and parietal pleura. This procedure allows the expansion of the underlying lung (Katariya et al, 1998). Some patients may require thoracoaplasty.
5.2 Spontaneous Pneumothorax

Spontaneous pneumothorax is a well known complication in cavitary tuberculosis. Pneumothorax is defined as the presence of gas in the pleural space (Loscalzo, 2010). The term Pneumothorax was first coined by Itard, a student of Laennec in 1803 and Laennec himself described the clinical picture of pneumothorax in 1819 (Kaya et al., 2009). Pneumothorax may be primary or secondary. Secondary pneumothorax occurs in persons with significant underlying pulmonary disease. Pneumothorax complicating pulmonary tuberculosis can occur at any age (Lichter & Gwynne, 1971). In countries where tuberculosis is a common problem, pulmonary tuberculosis remains the commonest cause of secondary spontaneous pneumothorax (SSP). According to Gupta et al., the commonest cause of spontaneous secondary pneumothorax in India is pulmonary tuberculosis (Gupta et al., 2006). In tuberculosis, the rupture of the sub pleural focus and rupture of a cavity are the common causes for pneumothorax. Other causes are rupture of a bleb or bulla which has developed adjacent to fibrosis.

All the patients will have chest pain and dyspnea. The clinical symptoms are dependent on the degree of collapse of the underlying lung. Cough is also a common symptom. Patients with tension pneumothorax will have acute onset of chest pain and dyspnea. On examination diminished movements of chest and reduced vocal fremitus are found on the affected side. Trachea deviates away from the affected side. The affected side may also be hyper resonant on percussion with diminished or absent breath sounds. Hypotension, tachycardia, hypoxia, jugular venous distension may be present. Radiography of the chest shows hyper-translucent area without bronchovascular markings on the affected side with the lung being compressed towards the hilum.

![Chest X-ray PA view showing Pyopneumothorax on left side.](image)

**Figure 9:** Chest X-ray PA view showing Pyopneumothorax on left side.

In tension pneumothorax the mediastinum will be displaced to the opposite side. Pneumothorax secondary to tuberculosis is frequently associated with untreated empyema, when it is called as pyopneumothorax. Pneumothorax is uncommon in miliary tuberculosis, and thus only few cases have been reported (Khan et al., 2011; Arya et al., 2011).
Management of pneumothorax consists of conservative approach and active management. Both ACCP and BTS guidelines recommend treatment based on the severity of symptoms and the degree of collapse of the underlying lung as determined by chest radiographs (Baumann et al., 2001). Conservative approach is indicated if patient has minimal symptoms and volume of pneumothorax is very small i.e. less than 20% of the hemithoracic volume. The principle of this approach is based on natural tendency for the gas to get absorbed. The absorption is hastened by giving 100% supplementary oxygen. Patients with pre-existing lung disease are unable to tolerate pneumothorax in which case active management is indicated. Active management consists of simple aspiration or intercostal tube drainage. Aspiration is less likely to be successful in patients with SSP. Blanco et al. studied pneumothorax in active pulmonary tuberculosis. Patients with pulmonary tuberculosis showed a lesser and slower response to catheter aspiration in their study (Blanco-Perez et al., 1998). It can be considered in patients with small pneumothoraces, who are symptomatic in order to avoid chest tube drainage. Otherwise, underwater seal chest tube drainage which allows gradual re-expansion of the lung is recommended. In case of persistent air leak or recurrent pneumothorax surgical intervention is recommended. Surgical intervention consists of limited thoracotomy with pleurectomy or pleural abrasion. Chemical pleurodesis by using sclerosing agents is indicated in patients with recurrent pneumothorax who are unwilling or unable to undergo surgery. Over the past years, widespread use of less invasive video-assisted thoracic surgery (VATS) has been more commonly used in the management of pneumothorax (Ng, 2006).

5.3 Calcification

The term calcification refers to the deposition of the calcium salts in tissues. It may be limited or diffuse. Diffuse can be metastatic calcification where calcium deposits are found in normal tissues. In dystrophic calcification it occurs in injured lung tissues. Dystrophic calcifications are frequently seen in granulomatous infections like tuberculosis, histoplasmosis and coccidioidomycosis. Tuberculous lung lesions heal by calcification. The calcification can either be microscopic or macroscopic (Behra, 2010). In parenchymal disease calcification presents as discrete radio opacities. Sheet like calcification can be seen in pleural
disease. Tuberculous pleuritis leaves sequelae ranging from minimal pleural thickening to extensive calcification encompassing and restricting the lung (Choi et al., 2001). Occasionally calcified lymph nodes or concretions can get detached and erode through the bronchial walls or blood vessels causing massive haemoptysis. Extensive calcification can lead to respiratory failure or cor pulmonale.

![Figure 11: Chest X-ray PA view showing bilateral extensive calcification.](image)

![Figure 12: Chest X-ray PA view showing bilateral extensive pleural calcification.](image)

6 General Complications

6.1 Cor Pulmonale

Cor pulmonale is right ventricular dysfunction (enlargement) due to pulmonary hypertension secondary to diseases of the lung, bony thorax, lung ventilation or pulmonary circulation (Rajendran, 2004). Vir-
chow in nineteenth century found changes like right ventricular hypertrophy in autopsies of patients who died of pulmonary tuberculosis (Kapoor, 1994).

Bilateral and extensive tuberculosis can cause pulmonary hypertension due to extensive fibrosis which causes distortion of parenchyma. The basic underlying pathophysiology is increase in the pulmonary vascular resistance and pulmonary hypertension. Clinical diagnosis depends on right ventricular dysfunction, pulmonary hypertension and evidence of primary lung disease. Early treatment prevents this late complication of pulmonary tuberculosis.

6.2 Amyloidosis

Tuberculosis is the commonest underlying cause for renal amyloidosis in developing countries, whereas Rheumatoid arthritis is the most frequent underlying inflammatory disease in developed countries. Amyloidosis is caused by deposition of insoluble fibrillar proteins in various tissues which leads to organ dysfunction or failure. Amyloidosis commonly involves kidneys. Secondary renal amyloidosis is seen in disorders that usually includes chronic inflammatory disease or infectious diseases. Pro-inflammatory mediators /cytokines such as interleukin -1, tumor necrosis factor alpha, and interleukin 6 stimulate the serum amyloid A synthesis in liver and other sites. This accumulates in renal tissue. Proteinuria is the most consistent feature of renal amyloidosis. In patients with tuberculosis, presence of pedal edema, proteinuria and grossly abnormal kidneys on ultrasonography should be evaluated by renal biopsy for amyloidosis. The time interval between the onset of predisposing disease and first evidence of amyloidosis has shown variable results in different studies, varying from one to thirty years (Chug et al., 1981) or two months to seven years (Dixit et al., 2009). Secondary amyloidosis has now become a rarity as a complication of pulmonary tuberculosis due to availability of effective anti-tuberculosis treatment.

6.3 Chronic Respiratory Failure

Chronic respiratory failure may complicate pulmonary tuberculosis (Sharma, 2009). Tuberculosis continues to be a chronic infection with very high rates of morbidity and mortality. Delays in the diagnosis and treatment of tuberculosis can lead to pulmonary sequelae that are characterized by impairments in the bronchial and parenchymal structure. The structural changes include bronchovascular distortions, bronchiectasis, emphysema and fibrosis. This leads to greater ventilation perfusion (V/Q) mismatch (Behra, 2010). Tuberculosis destroyed lung term is used to describe the destructive lung parenchymal changes which occur over many years following pulmonary tuberculosis (WHO Report. 2008) Non-invasive ventilation has greatly improved the outlook for patients with previous tuberculosis who develop chronic respiratory failure (Shneerson, 2004). Acute respiratory failure can occur over a chronic respiratory failure due to respiratory infection with pyogenic organisms (SatyaSri, 2009).

7 Conclusion

Various forms of complications may result from pulmonary tuberculosis. It is important to be aware of the full spectrum of clinical and radiologic features of the sequelae to facilitate diagnosis and management of the complications of pulmonary tuberculosis.
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