CAUSES AND OUTCOME OF MODERATE TO MASSIVE HEMOPTYSIS AMONG ADMITTED PATIENTS FROM RAJIV GANDHI GOVERNMENT GENERAL HOSPITAL IN CHENNAI

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CHENNAI

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

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MAY 2020

BONAFIDE CERTIFICATE

This is to certify that the dissertation titled **"CAUSES AND OUTCOME OF MODERATE TO MASSIVE HEMOPTYSIS AMONG ADMITTED PATIENTS FROM RAJIV GANDHI GOVERNMENT GENERAL HOSPITAL IN CHENNAI**" is the bonafide work done by **Dr. UMAPATHI S** during his **M.D (Respiratory Medicine)** course in the academic years **2018-2020**, at the Institute of Thoracic Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai. This work has not previously formed the basis for the award of any degree.

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DECLARATION BY THE GUIDE

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I, **Dr. UMAPATHI S** solemnly declare that the dissertation titled **"CAUSES AND OUTCOME OF MODERATE TO MASSIVE HEMOPTYSIS AMONG ADMITTED PATIENTS FROM RAJIV GANDHI GOVERNMENT GENERAL HOSPITAL IN CHENNAI"** is a bonafide work done by me at Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai-3 from December 2018 to June 2019 under the guidance and supervision of the Director Prof. Dr.A.Mahilmaran, M.D., D.T.C.D., Head of Department, Institute of Thoracic Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai.

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CONTENTS

INTRODUCTION

INTRODUCTION

The word ‘hemoptysis’ originated from Greek word ‘haima’ which means ‘blood’ and ‘ptysis’ which means ‘spitting’.[1]

It is the symptom per se and not the disease but alarms and frightens the patient that they had serious illness. It adds psychological and economical burden to the patient.[2]

Hemoptysis is defined as coughing out of blood from the lung parenchyma or tracheobronchial airway as a result of pulmonary or bronchial hemorrhage.[1–3] It can arises in the tracheobronchial tree from glottis to alveoli.[4]

Involvement of bronchial artery is responsible for majority of cases and pulmonary artery is the cause in <10% of the cases.[4] Bleeding from bronchial arteries are severe and massive due to high systemic pressure than pulmonary artery bleeding.

Hemoptysis ranges from blood streaking of expectorated sputum to frank blood without sputum.[4]

It was classified into mild, moderate, severe and massive according to amount and rate of bleeding.[4]

The effective treatment of hemoptysis depends upon identification of the etiology and localization of the bleeding site.[2] It requires further evaluation to investigate the diseases that causing it.[5]

Common site of hemoptysis are tracheobronchial tree, pulmonary parenchyma and pulmonary vasculature.[4]

Causes of hemoptysis[6]

Tracheobronchial origin:

Neoplasm (bronchogenic carcinoma, endobronchial metastasis, bronchial carcinoid, Kaposi's sarcoma), Bronchiectasis, Broncholithiasis, Foreign body and Trauma to airways.

Pulmonary parenchymal origin:

Tuberculosis, Mycetoma (fungal ball), Lung abscess, Pneumonia, Goodpasture's syndrome, Pulmonary hemosiderosis, Granulomatosis with polyangitis (formerely wegener's), Lupus pneumonitis, Lung contusion

Pulmonary vascular origin:

Pulmonary thromboembolism and Arteriovenous malformation.

Causes of hemoptysis varies from one geographical area to another area and time of study.[5]

Pulmonary tuberculosis is the commonest cause of hemoptysis in developing countries like India, but in the developed countries bronchitis, bronchiectasis and bronchogenic carcinoma are the top most causes.[5]

Massive hemoptysis into airway is life threatening situation to patient because asphyxiation will happen if tracheobronchial tree is flooded with blood.[7] Blood in the airways and alveolar spaces disturbs the gas exchange. The rate of bleeding in to airway and it's effect on alveolar gas exchange determines the need for urgent management. Death from hemoptysis will be sudden and results from major airway occlusion (asphyxia) rather than exsanguination.

The reported mortality rate was 80% for massive hemoptysis and 7% to 30% for mild to moderate hemoptysis.[4]

Morbidity and mortality in hemoptysis depends on not only the volume of blood expectorated but also rate of bleeding, ability of the patient to clear it from the airways and extent of underlying lung diseases.[8]

Treatment of specific cause could control hemoptysis permanently. So identification of cause is needed, which is the primary objective of this study. By comparing the outcome of hemoptysis management, we can assess the efficiency of each procedure and deleterious effect of each disease, which is the secondary objective of this study.

Doing this study in our centre we could be familiar in treating hemoptysis and to choose intervention procedures based on need and condition of the patient. So this study is essential for the treatment of patients with hemoptysis in current situation and near future.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Hemoptysis is a serious problem by causing disturbance in alveolar gas exchange and hypovolemic shock secondary to acute massive blood loss.[3] Outcome of hemoptysis management was different in each area due to wide spectrum of causative factors and variation of it's prevalence.[2] So through analysis of hemoptysis symptom is essential to prevent or reduce mortality due to it. It is essential to evaluate the frequency of each causes in our hospital and to find whether any changes happened by comparing reports from other areas of our country or world. By analysing merits and limitations of each management procedure we can decide which one among them was superior and early institution of such management without delay we can save so many lives. Classification of the cause based on severity of bleeding we can decide whether hospitalisation is needed or not.[9] The causes of hemoptysis is diagnosed by combination of history, through clinical examination, chest radiography, CT chest scan, fibreoptic bronchoscopy, microbiology, serology and histology.[3]

Pulmonary vascular system

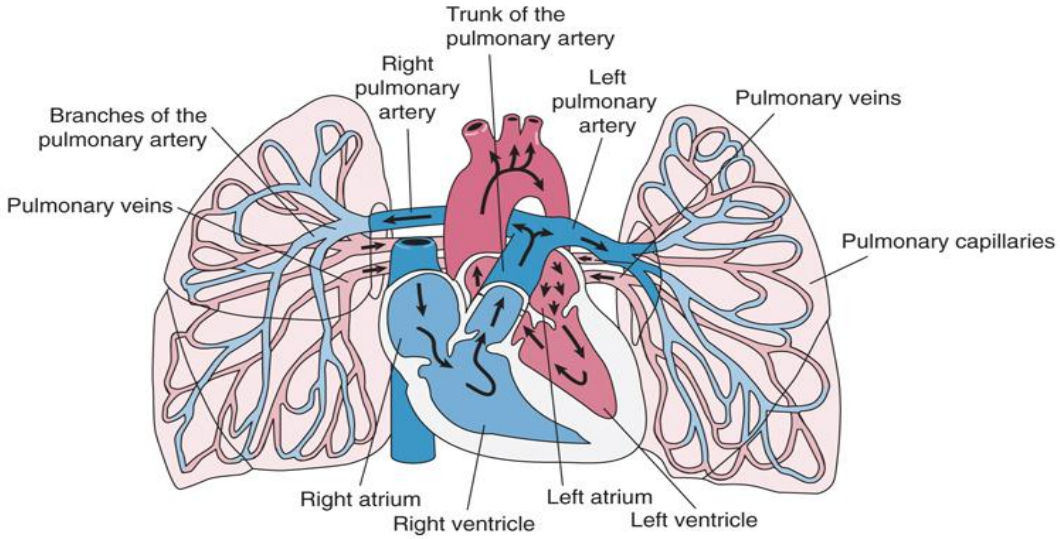
Arterial supply:[10]

Bronchial arteries supply nutrition to the bronchial tree and pulmonary tissue. These are small arteries that varies in size, number and origin.

Usually on right side one bronchial artery which arises either from 3rd posterior intercostal artery or from upper left bronchial artery.

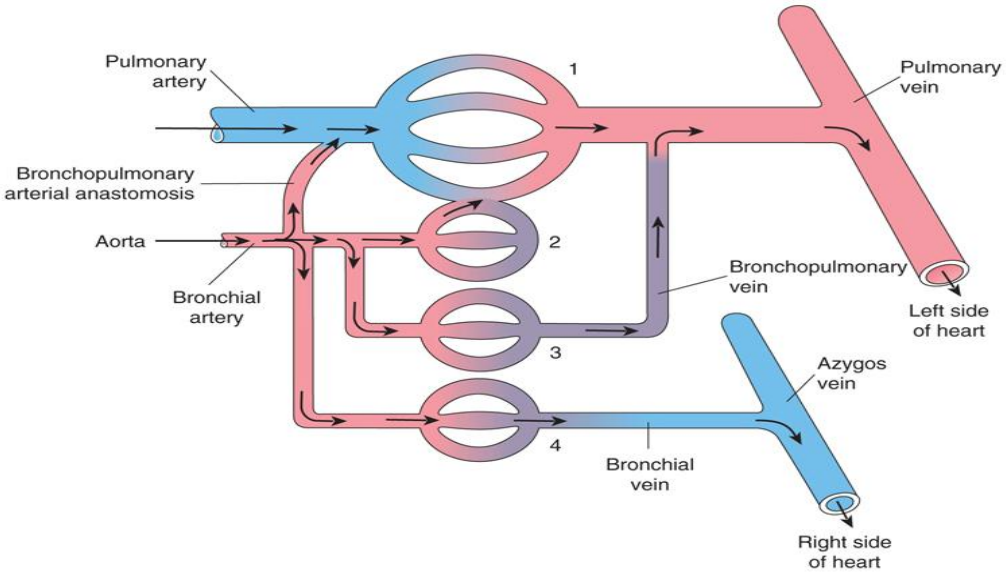
On left side there are two bronchial arteries. Both arises from the descending thoracic aorta. Upper one at the level of opposite to T5 vertebra and lower one just below the left main bronchus.

Pulmonary artery carries deoxygenated blood to the lungs and oxygenated blood from lung is returned to heart via pulmonary veins.



From Hicks GH: Cardiopulmonary anatomy and physiology, Philadelphia, 2000, WB Saunders.

Precapillary anastomosis is seen between bronchial and pulmonary arteries.



From Hicks GH: Cardiopulmonary anatomy and physiology, Philadelphia, 2000, WB Saunders.

Venous drainage:[10]

Major part of venous blood from lung was drained by pulmonary veins. Bronchial veins carry venous blood from the first two divisions of bronchi. Each lung was drained by 2 bronchial veins. Right bronchial vein drains into azygos vein. Left bronchial vein drains into either superior intercostal vein or hemiazygos vein.

History point of view hematemesis mimics hemoptysis and was couldn't be differentiated by patients many of the times. As a clinician we can differentiate it by following additional details.

Difference between hemoptysis and hemetemesis

	Hemoptysis	**Hematemesis**
History	Cough precedes hemoptysis	Nausea and vomiting precede hematemesis
Colour	Bright red	Dark brown due to acid hematin
Froth	Present due to admixture of air	Absent
Gross appearance	Liquid or clotted	Coffee ground
Microscopic appearance	Mixed with macrophage and neutrophil	Mixed with food particles
pH	Alkaline	Acidic
Melena	Absent	Present
Past history	Respiratory disease	Peptic ulcer disease
Diagnosis	Bronchoscopy	Gastroscopy

Types of Hemoptysis

Frank hemoptysis:

- It is expectoration of blood only. Frank hemoptysis daily is suggestive of bronchogenic carcinoma.

Spurious hemoptysis:

- Hemoptysis originates above the level of larynx from upper respiratory tract.

Pseudo hemoptysis:

- It is due to a pigment prodigiosin produced by gram-negative organism - Serratia marcescens.

Endemic hemoptysis:

- Due to infection with Paragonimus westermani (lung fluke).

Hemoptysis in suppurative lung disease:

- Large quantity of foul smelling sputum with blood suggests suppurative lung disease.

Cryptogenic Hemoptysis

- When the cause remains unknown after thorough workup

Catamenial hemoptysis

- Occurrence of hemoptysis during menses. It is caused by intrathoracic endometriosis[11].

Classification of hemoptysis:

Classification depending upon quantity and rate of bleeding. It was not completely agreed in literature and varies widely from one author to another author.

Amirana et al[12] defined massive hemoptysis as expectoration of 100 ml of blood per day but Corey et al[13] defined Expectoration of >1000 ml of blood per day was massive hemoptysis.

Hemoptysis classification of Bhalla et al[4], Nawal et al[2] and Fidan et al[14] in their study taken as mild (<30ml/day), moderate (31 – 100 ml/day), severe (100-600 ml/day) and massive (>600 ml/ day or >100 ml/day with respiratory or hemodynamic compromise) to find out the cause.

Prasad et al[15], Singh et al[5], and Patel et al[7] were divided hemoptysis into mild (<100 ml/day), moderate (100 – 400 ml/day) and severe (>400 ml/day). Single bout of 150 ml blood expectoration was considered as massive by Ronald win B et al[3] and he classified hemoptysis further in to mild (<100 ml/day), moderate (100-400 ml/day) and massive (>400 ml/day or single bout of 150 ml).

Das et al[1] arbitrarily classify hemoptysis in to mild (<30 ml), moderate (30-200 ml) and severe (>200 ml) per day during their study. Hemoptysis was mild (<100 ml/day), moderate (100-300 ml/day) and severe (>300 ml/day) by Rachakonda et al[6].

Classification of hemoptysis was not uniform as evidence by the few above mentioned studies. We followed the classification by Bhalla et al[4] as their study was the most recent one.

Important Clinical Features of Patients with Hemoptysis		
Category	**Feature**	**Disorder**
History	Cigarette smoking	Bronchogenic carcinoma
	Risk factors for aspiration	Lung abscess, foreign body aspiration
	Recent chest trauma or invasive procedure	Traumatic/iatrogenic lung injury
	Previously diagnosed malignancy	Metastatic malignancy
Symptom	Purulent sputum	Bronchitis, Bronchiectasis, Pneumonia, lung abscess
	Paroxysmal nocturnal dyspnea, orthopnea	Mitral stenosis, Left ventricular failure
	Pleuritic chest pain	Pneumonia, Pulmonary embolism
	Fever	Pneumonia, lung abscess
	Weight loss	Tuberculosis, Lung abscess, Bronchogenic carcinoma, other malignancies
Sign	Bronchial breath sounds, egophony	Pneumonia
	Decrease in breath sounds, wheezing	Bronchogenic carcinoma, broncholithiasis, foreign body
	Coarse leathery crackles, rhonchi	Bronchiectasis, bronchitis
	Pleural rub	Pneumonia, pulmonary embolism
	S3 gallop	Left ventricular failure
	Diastolic murmur	Mitral stenosis

Thorough physical examination can give some clues in the identification of cause. Neck examination showing lymphadenopathy may give a clue that the cause is tuberculosis or malignancy (lung cancer or lymphoma).

Pulmonary Tuberculosis

The incidence of hemoptysis in patients of pulmonary tuberculosis is ranges from 30%-35%.[16] More than 60% of hemoptysis were caused by pulmonary tuberculosis in india.[1–7,15,17] Hemoptysis does not indicate that the TB is active. Hemoptysis may occur as initial manifestation of active TB or during course of treatment or even after disease is apparently cured.[15] Hemoptysis from TB could be streaky or massive and becomes life-threatening. Wall of a TB cavities becomes atrophic by inflammation and necrosis. Increased pressure leads in to weakening of the cavity wall, blood vessels dilatation and formation of Rasmussen's aneurysms. During coughing and strenuous exercise further increase in pressure leads into rupture of these blood vessels and results in hemoptysis. The vessel walls can be eroded directly either by endarteritis, or vasculitis secondary to TB. Occasionally allergic response to Mycobacterium tuberculosis can gives rise to hemoptysis by producing damages in the wall of the blood vessel. Bleeding from bronchial granuloma can result in hemoptysis. Aneurysmal dilatation of blood vessels and accentuated bronchopulmonary communications are present in these granulomas. In this setting, bronchial blood vessels which are under systemic pressure could be the source of bleeding. All the above factors should be taken into consideration while treating hemoptysis in patients with TB.[16]

Causes of hemoptysis in pulmonary TB[16]

Rupture of Rasmussen's aneurysm
Bleeding from cavity wall Scar carcinoma
TB endobronchitis Broncholith, cavernolith (erosion of calcified lesion into airway)
Aspergilloma Post tuberculous bronchiectasis Direct erosion of blood vessels by granulomatous inflammation

In most of the situation restoring fluid balance and hemodynamic condition of the patient by bed rest, sedatives, and resuscitative measures will controls the bleeding.[9] Patients with massive hemoptysis (> 600 mL of blood / 24 hours) may prone to unstable hemodynamic state and often require blood transfusion. Broad spectrum antibiotics are needed to cover super-added secondary bacterial infection. Anti-TB treatment is indicated only if patient had active disease. If the bleeding is massive, and recurrent, high resolution computed tomography (HRCT) and fibreoptic bronchoscopy should be done to localise site of bleeding.[18] Patients with massive hemoptysis will need angiography and bronchial artery embolization. Bronchial artery embolization (BAE) is an excellent and relatively safe procedure in management of hemoptysis caused by pulmonary TB. The risk of recurrent bleeding after BAE in pulmonary TB is high, if patients having destroyed lung, diabetes mellitus, chronic liver disease, fungal ball, raised pre procedure C-reactive protein or using anticoagulants and/or antiplatelet agents. Rarely surgical excision of the diseased site may be needed.[16]

Aspergilloma (Fungal ball or Mycetoma)

Incidence is calculated by approximate number of aspergilloma cases occurred per year. Jean-francois regnard et al[19] reported 4.3 cases per year in 2000 but it is high in study by CK Park et al[20] who reported 8.4 cases per year in 2002.

It was the cause in 3% of hemoptysis in north east india.[1] 17% of incidence was seen in a resurvey conducted in great Britain.[21] Bhalla et al[4] found one patient having fungal ball radiologically among the 64 hemoptysis cases in a tertiary care centre of north India.

Fungal hyphae that colonises and grows in a lung cavity is known as mycetoma.[16] Although other fungi like Fusarium and Zygomycetes may produce the formation of a fungal ball, Aspergillus fumigatus, is the most common causative agent of aspergilloma.[16] It is a saprophyte present over the decaying and dead tissues.[22] Aspergilloma is composed of necrotic mass of matted fungal hyphae, inflammatory cells and fibrin. It is usually mobile within the cavity hence antifungal agents have no successful role in it's outcome.[22] Aspergillus invades pre-existing lung cavities formed by pulmonary tuberculosis, histoplasmosis, bronchiectasis, sarcoidosis, bullae, neoplasms, pulmonary infarct, chronic lung abscess, bronchogenic cyst, asbestos, and cavitating malignant diseases. Of the above conditions pulmonary tuberculosis was most frequent. The natural history of aspergilloma is highly variable and it may remain stable, increases in size or resolve spontaneously. In the early developmental phase, the fungus ball grows inside the lung cavity contains both living and dead fungi. If the local conditions

favours death, the fungus ball undergo liquefaction and will be expectorated out. Calcification occurs rarely. Hemoptysis is the most common presentation in such cases and it's estimated frequency varies from 5% - 90%.[16] The amount of blood expectoration may be scanty to massive. Bleeding usually originates from the systemic circulation via bronchial arteries.[16] Hemoptysis is usually recurrent and sometimes leads in to fatal due to massive bleeding.[22]

The cause of hemoptysis in aspergilloma were due to[16]

(i) mechanical friction of mycetoma

(ii) hemolytic endotoxin from Aspergillus

(iii) anticoagulant factor from Aspergillus

(iv) direct invasion of blood vessels overlying the cavity wall

(v) local vasculitis.

Aspergilloma may be a incidental radiological finding in patients who came for some other diseases. Fungus ball surrounded by crescent of air is called "air meniscus sign" or "air crescent sign" which is a characteristic radiological finding but not specific to mycetoma. It may also seen in hematoma or pus within a cavity and bronchogenic carcinoma. Fungus ball is mobile when the patient changed his position and is seen in fluoroscopy or CT chest in different position. Fungus ball may be visualised during bronchoscopy.

Surgery is indicated for recurrent and massive hemoptysis. Bleeding commonly results from systemic circulation occasionally stops on it's own. Peripherally placed mycetoma with chest wall invasion can erode intercostal

arteries. Bleeding from such large vessel could be fatal if not stopped immediately.[22]

The mortality rate was varying between 2% and 14%. Sputum culture confirms the diagnosis but is positive only in 50% of cases. Ig G specific antibody against aspergillus is positive in almost all cases.[16]

Bronchiectasis:

It's incidence varies from 4% to 35% in hemoptysis reported by various studies conducted in India.[1–7,17] It is defined as permanent dilatation of airways >2 mm in diameter from their normal state.[23] Bronchiectasis results from broad range of pathological process. Bronchial abnormality (cartilage deficiency in William campbell syndrome), impaired muco-ciliary clearance (cystic fibrosis), infections (recurrent childhood respiratory infections, tuberculosis and immune deficiency) and inflammatory diseases are the basic pathological process.[23]

As upper lobes were more commonly affected by TB, post tuberculous bronchiectasis also common at this site.[24] It is a "sicca" or "dry" type of bronchiectasis because secretions were drained by gravity. It usually presents as repeated episodes of secondary bacterial infection or severe hemoptysis.[23]

Most frequently isolated organisms in bronchiectasis were Haemophilus influenzae and Pseudomonas aeruginosa. Biofilm produced by Pseudomonas act as a barrier against host defence and antibiotics. It is a commonly found organism in bronchiectasis due to cystic fibrosis.[25]

Cough efficiency is decreased in bronchiectasis because to produce high forced expiratory flow it requires transient narrowing of airway. This narrowing does not occur in abnormally dilated airways.[23]

Chronic suppuration in airways leads in to complications. Chronic inflammation secondary to infection causes airway injury and bronchial dilatation. Chronic inflammation results in increased expression of angiogenic factors IL-8 and endothelin-1 leads in to remodelling of bronchial circulation by neovascularization and bronchial vessel hypertrophy. This leads in to life threatening and recurrent hemoptysis.[23]

Bronchiectasis is confirmed by presence of dilated bronchi in high resolution CT of chest.[18] Characteristic findings are dilated thick wall bronchi extending to the periphery of lung.[25]

Postural drainage and chest physiotherapy, muco-kinetics, anti-inflammatory agents, bronchodilators, antibiotics and sometimes surgery are needed to control hemoptysis.[26]

Pulmonary Vasculitis

It is a very rare cause of hemoptysis as shown by reports from national and international literature. Only few cases found till date as a cause of significant hemoptysis. Bhalla et al[4] found only one case caused by ANCA associated vasculitis and prasad et al[15] found one case by systemic lupus erythematosus in their study. Idiopathic causes contributes to 1-2% in study conducted by

Rachakonda et al[6] and Ronald win b et al[3] in our country. Undiagnosed causes contributes to 32% in a study conducted by Das et al[1] in north eastern India. So further evaluation of these unknown causes could give an actual incidence of vasculitis causing respiratory tract bleeding.

Pulmonary vasculitis is usually the manifestation of systemic disorder causing inflammation of vessels by immunological mechanisms.[27] The bronchial veins were closely associated with the bronchial arteries, although neither is commonly affected in pulmonary vasculitis. Capillaries are most commonly affected vessel in pulmonary vasculitis.[26]

Hallmark of diffuse alveolar hemorrhage is hemoptysis, but around 33% of patients were seen without hemoptysis. DAH is diagnosed by bronchoscopy while doing bronchoalveolar lavage. Serial aliquots of (30 to 60 mL) sterile saline are instilled and aspirated (for total 100 to 300 mL of volume) with the bronchoscope in wedge position. If serial aliquots of fluid showing increasingly hemorrhagic or a persistently bloody return, then a diagnosis of DAH is made. The finding of DAH is not absolute diagnostic of vasculitis. DAH could be caused by diseases associated with histopathologic finding of capillaritis (includes primary idiopathic and secondary vasculitis), diseases causing diffuse alveolar damage and bland hemorrhage. Capillaritis is always found in diffuse alveolar hemorrhage caused by ANCA associated vasculitis.[26]

Granulomatosis with polyangiitis (GPA):

Most common form of vasculitis involving the lung is GPA. It is defined by Chapel Hill Consensus Conference as necrotizing granulomatous inflammation of the respiratory tract, and necrotizing vasculitis of small-to medium-sized vessels.[27] GPA affects pulmonary parenchyma, bronchi, and rarely pleura. Parenchymal involvement may present as cough, chest pain, dyspnoea, or hemoptysis. About one-third of patients had no hemoptysis.[27] Patient may deteriorate rapidly and prone for respiratory failure, which had mortality rate of up to 50%. Pulmonary capillaritis is the cause for clinical presentation of alveolar hemorrhage. Neutrophils are the predominant inflammatory cells in this reaction. Fibrinoid necrosis of alveoli and vessel wall leads into destruction of underlying lung architecture. Necrotizing granulomatous inflammation of the lung parenchyma presents as nodules or mass lesions radiographically which may cavitate. Endobronchial disease may cause parenchymal collapse or post obstructive infection and present with cough, hemoptysis, wheezing or dyspnoea. It may be an incidental finding while doing bronchoscopy. [27]

Microscopic polyangiitis (MPA) :

Diffuse alveolar hemorrhage caused by inflammation of pulmonary capillaries is seen in 10-30% of patients. [27]

Eosinophilic granulomatosis with polyangiitis (EGPA):

Diffuse alveolar hemorrhage is rare in contrast to GPA and MPA.[27]

Behçet disease:

Respiratory manifestations of Behçet disease are cough, hemoptysis, chest pain, and dyspnea. Hemoptysis is most often massive and fatal. The vasculitis is immune complex mediated, and affects vessels of all sizes. Massive hemoptysis is the result of destruction of elastic lamina, bronchial erosion and arterial bronchial fistulae. The prognosis of pulmonary involvement is very poor. About one-third patients will die within 2 years from fatal pulmonary hemorrhage. Embolization therapy is used for prevention and treatment of hemorrhage caused by pulmonary artery aneurysms.[27]

Systemic lupus erythematosus (SLE):

Pulmonary capillaritis leads into diffuse alveolar hemorrhage is rare in SLE. Pulmonary capillaritis is immune complex mediated. The onset of diffuse alveolar hemorrhage is usually abrupt, and is rarely the first sign of SLE. The reported mortality by diffuse alveolar hemorrhage have wide variation between 0% and 90%.[27]

Anti–glomerular basement membrane disease:

Diffuse alveolar hemorrhage is common but requires additional injury by smoking for the development of the pulmonary manifestations. Definitive diagnosis made by documentation of linear anti-GBM deposits over the kidney or lung. [27]

5% to 15% of GPA and 30% to 50% of EGPA have cardiac involvement and carries high risk of mortality. So screening by cardiologist with electrocardiogram and echocardiography is required.

Definitive diagnosis require tissue biopsy from lung, skin or kidney. Specific pathological features are granulomatous inflammation and vascular necrosis. Immunofluorescence pattern on tissue are characteristic of particular vasculitis. Linear Ig A deposits are seen in goodpasture syndrome. Systemic lupus erythematosus shows irregular immunoglobulin and complement fixation.[26]

Lung malignancy

Hemoptysis was a first symptom reported by 6 to 25% of lung cancer patients. About 5% of lung cancer patients with hemoptysis having normal chest x-ray.[27] Massive hemoptysis in a malignancy is associated with 80% mortality in a study done by Jean-Baptiste et al[28].

Hemoptysis in a smoker or COPD should raise the possibility of lung cancer.[11] Exposure to arsenic, asbestos, nickel, chromium and ethers are other risk factors of hemoptysis.

Hemoptysis can present as blood streaking sputum for prolonged period of time before presentation to the physician because patient thought it due to smoking related bronchitis. Lung cancer may cause massive hemoptysis because highly vascular in nature.[26]

Benign or malignant tumors causes hemoptysis by superficial mucosal invasion, erosion of blood vessels or producing highly vascular metastasis[11]. Post obstructive pneumonitis also causes hemoptysis.

Bronchial adenomas are slow growing malignant tumour and present with occasional bleeding over many years. Hypercoagulable state induced by adeno carcinoma causes pulmonary embolism.

Endobronchial metastasis by breast cancer, colon cancer, kidney cancer, carcinoid tumors and melanoma causes hemoptysis. [29] Most frequent lung tumour causing hemoptysis in children is carcinoid. It presents as hemoptysis in 18% of carcinoids. Primary pulmonary mucoepidermoid carcinoma rarely causes hemoptysis. Germ Cell Tumours can cause hemoptysis by bronchial erosion.

Pneumonia

Pneumonia occupies 2 -10% of hemoptysis from studies conducted in various parts of our country.[4–7,15] Pneumonia is an infection caused by infective organisms affects gas exchanging unit of the lung. It develops when host defences are defeated by infective pathogens. Pneumonia developed outside the hospital is called community acquired pneumonia. To define clinically it require 2 or more of the following symptom and signs: tachypnea (respiratory rate > 20 breaths/minute), productive cough, purulent sputum, pleuritic type of chest pain, chills or rigors with chest radiographic evidence of new opacity.[23]

Inflammation and edema of the superficial mucosa by infective organisms leads into rupture of the superficial blood vessels and results in hemoptysis. Chronic pulmonary infection leads into enlargement of the bronchial arterial circulation. Hemoptysis is common even in patients with mild lung disease and indicates infection.[23]

Klebsiella pneumona causes pleuritic chest pain and red currant jelly sputum. Streptococcus pneumoniae cause rusty sputum mimics hemoptysis. Staphylococcus aureus causes hemoptysis by pulmonary infarct from septic emboli.[27]

Lung abscess

It was also a rare cause of moderate to massive hemoptysis in our country. Out of 72 hemoptysis patients only 2 cases of lung abscess found by Singh et al[5] at northern Madhya Pradesh of India. Another study by Prasad et al[15] found 2 cases of lung abscess out of 476 patients from a chest clinic in India. Rachakonda et al[6] found 4 cases out of 216 patients from their study at a tertiary care centre.

Lung abscess is defined as localised suppurative necrosis within lung parenchyma of usually >2 cm in size.[23] Micro-aspiration of oropharyngeal secretions during sleep was common in more than 50% of normal individuals.[26] Aspiration of infectious oropharyngeal content in a person with inadequate defence leads in to abscess formation is the basic mechanism for lung abscess. Patients with poor dentition and gingival diseases were more prone because they had salivary bacterial count of $>10^{11}$/ml.[23]

Body position at the time of aspiration and gravity are determining factors for the location of abscess.[30] Lung abscess are typically found in basal & apical segments of lower lobe and posterior segment of upper lobe.[23]

Lung abscess is either could be primary or secondary to other systemic conditions. Most of them were primary due to untreated aspiration pneumonia.[31] Primary abscess is mainly caused by infections and neoplasm. Anaerobes are the most common organism causeing lung abscess. Mixed bacterial flora found in majority of cases. Secondary abscess is one that is complicated by septic emboli from infective endocarditis or bronchial obstruction by aspirated foreign body.[23]

If symptoms were present for more than 4-6 weeks it is called chronic abscess. Chronic lung abscess has been complicated by life-threatening hemoptysis, metastatic abscess in brain and other sites, bronchopleural fistula, empyema necessitans and secondary amyloidosis, but these complications are rare.[25]

Sputum or bronchial was culture will confirm the causative agent. Specimen obtained by bronchoscopy is free from contamination of the upper airways. Specimen should be discarded if contains >10 squamous epithelial cells per low power field because it indicates excessive oropharyngeal contamination.[26] Chest radiograph shows typical appearance of thick wall cavity with air fluid level. In last few decades, improved treatment of pneumonia had declined the

incidence of lung abscess. Broad spectrum antibiotics not affected by β-lactamases are the mainstay of treatment.[23]

Important Radiographic Findings in Patients Who Have Hemoptysis	
Radiographic Finding	**Disorder(s)**
Atelectasis	Foreign body, Bronchogenic carcinoma or other Endobronchial neoplasm, Broncholithiasis
Nodule(s) or mass(es)	Bronchogenic carcinoma or other neoplasm, Granulomatosis with polyangitis, Fungal infection, Lung abscess
Air-space opacity	Pneumonia, Lung contusion, Diffuse alveolar hemorrhage,
Hilar/mediastinal adenopathy	Mycobacterial or fungal infection, Bronchogenic carcinoma or other neoplasm, sarcoidosis
Dilated peripheral airways	Bronchiectasis
Cavity/cavities	Mycobacterial or Fungal infection, Lung abscess, Bronchogenic carcinoma, Granulomatosis with polyangitis
Reticulonodular opacity	Sarcoidosis, lymphangitis carcinomatosa
Hilar/mediastinal calcification	Past Tuberculous or fungal infection, broncholithiasis

High-resolution CT of chest

High-resolution CT has become useful in the initial workup of hemoptysis. It is preferred if pulmonary parenchymal disease is suspected.[9] Its use with bronchoscopy gives a higher positive yield of diagnosis. Further studies are needed to find out it's role on hemoptysis and it's effect on patient management.[18]

Fibreoptic bronchoscopy:

Fibreoptic bronchoscopy should be done before bronchial artery embolization to establish the site of bleeding.[32] However in massive hemoptysis, localization is very difficult because blood is spread through entire bronchial tree by vigorous coughing.

If neoplasia is suspected, fiberoptic bronchoscopy is a preferred investigation. It diagnoses endobronchial lesions and directly visualise the bleeding site. It had advantage of taking tissue biopsy from visualised lesion and can do bronchial lavage and brushings for histopathological diagnosis.[33] Fiberoptic bronchoscopy can be used therapeutically to control bleeding. Rigid bronchoscopy is the preferred tool in cases of massive bleeding because it had greater suctioning and airway maintenance capability.[27]

Therapeutic role of bronchoscopy in hemoptysis:[27]

Fibreoptic bronchoscopy is valuable tool in hemoptysis for several reasons. Rigid bronchoscopy had advantage of removing large clots than fibreoptic bronchoscopy. Blood clots will impair the gas exchange if not removed.

1) Topical application of iced saline or epinephrine: it can be instilled through bronchoscope in to bleeding site. These agents stop bleeding by local vasoconstriction.
2) Balloon catheter can be placed endo-bronchially to tamponade bleeding and prevents contamination of proximal airway by facilitate clot formation.

3) Endobronchial blockers can be used to occlude the right or left main bronchus to stop bleeding.
4) Nd:YAG laser photocoagulation
5) Argon plasma coagulation
6) Endobronchial packing of oxidized regenerated cellulose isolates segmental or subsegmental bleeding site. This agent promote clot formation by fibrin polimerization.[27]

The overall goals of management in patient with hemoptysis are: to stop bleeding, prevention of aspiration, and treatment of the underlying cause.[11] In cases of massive hemoptysis, diagnosis and treatment must be started simultaneously. Maintenance of patent airway is vital because the primary cause for death is asphyxiation, not exsanguination. Assistance by a cardiothoracic surgeon is often helpful because emergency surgical intervention may be needed.[22]

Conservative management:[13]

Hemoptysis should be treated immediately on arrival to the hospital. The first priorities should be given to maintain airway patency, supplementation of oxygen and stabilisation of hemodynamic status by intravenous fluids administration.[5] Often patients can tell that from which side bleeding is coming. They should be placed like bleeding side down and should be given supplemental oxygen. If massive bleeding continues and the airway patency is compromised, the patient should be intubated and mechanically ventilated.[5]

Anti-tuberculous treatment: Before starting ATT primary drug resistance to isoniazid and rifampicin should be ruled out by using Line Probe Assay (LPA) and Catridge Based Nucleic Acid Amplification Test (CBNAAT).[34]

New pulmonary tuberculosis: as per the revised national tuberculosis control programme, intensive phase consists of 8 weeks of isoniazid, rifampicin, pyrazinamide and ethambutol in daily dosages as per five weight band categories. There will be no need for extension of intensive phase if sputum shows presence of acid-fast bacilli at the end of intensive phase. Continuation phase is 16 weeks consists of isoniazid, rifampicin, and ethambutol in daily dosages.[34]

Previously treated pulmonary tuberculosis: intensive phase will be of 12 weeks, where injection streptomycin will be stopped after 8 weeks and the remaining four drugs (isoniazid, rifampicin, pyrazinamide and ethambutol) in daily dosages as per the weight bands will be continued for another 4 weeks. There will be no need for extension of intensive phase. At the start of continuation phase pyrazinamide will be stopped while the rest of drugs - isoniazid, rifampicin, and ethambutol in daily dosages continued for another 20 weeks.[34]

From 19.12.2018 onwards there is no separate regimen for new and previously treated tuberculous patients as per RNTCP programme. All previously treated TB patients will also be initiated a standard first line anti TB regimen (2HRZE/4HRE) as prescribed for new TB patients with no injection streptomycin.

Antibiotics: to control of secondary bacterial infection

Coagulants:

Adrenochrome monosemicarbazone reduces capillary fragility and controls oozing from raw area. It also prevents micro-vessel bleeding. It is available in injection form for parenteral administration.[28]

Antifibrinolytic:

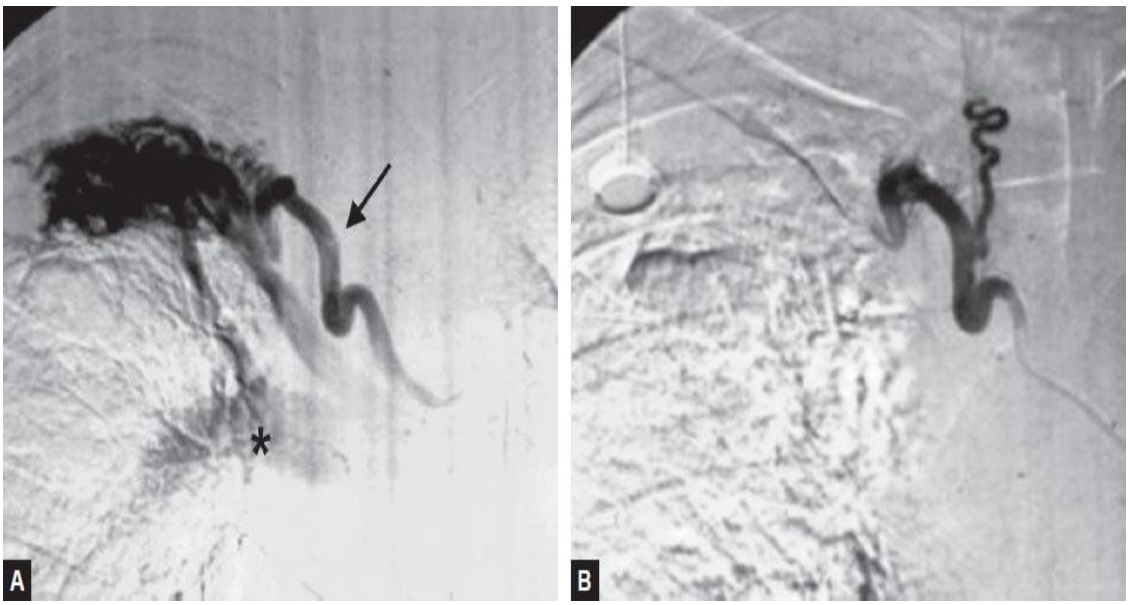
Tranexamic acid is available as oral tablet and injection form. It prevents fibrinolysis by binding to plasminogen. So fibrin unable to bind to plasminogen hence fibrinolysis prevented. Thrombophlebitis of the injected vein is the frequent complication.[9,11,28]

Bronchial arteriography

Patients with severe hemoptysis may require a bronchial arteriography and embolization of the bronchial arteries supplying the bleeding site.[35] This study requires selective catheterization of bronchial arteries.[36] Angiographic technique is individualized to fit particular clinical condition being studied.[26]

Angiography is performed by a technique termed "digital subtraction angiography."[37] With digital subtraction angiography, an early image from the angiogram is recorded by an image intensifier and high-resolution television camera. Image is digitized and stored. A later image is taken in which opacified vessels are handled in a similar manner. First image is subtracted from the second image and the resulting image is displayed. The background body structures are subtracted, leaving an image of contrast-filled blood vessel. The final image is not

obscured by background body images, and angiography can be done with small amount of contrast.[26]



Intra-arterial digital subtraction angiography (IA-DSA) in a patient with right upper lobe pulmonary tuberculosis showing a hypertrophied inter costo-bronchial trunk [arrow] producing contrast extravasation [asterisk] and pulmonary artery filling in the region of fibro cavity lesion [A]. The IA-DSA after embolization with polyvinyl alcohol particles showing obliteration of the angiographic abnormality with patent parent artery [B]

The most common indication for urgent bronchial arteriography is massive hemoptysis.[38] The need for emergency intervention is determined by sudden onset of severe hypoxemia from intrapulmonary bleeding.[39] Mild to moderate hemoptysis rarely causes life-threatening exsanguination. For practical purpose, any amount or rate of bleeding should be considered as emergency when it leads into compromise in airway patency.[40] Massive hemoptysis mostly caused by

fungal infection, bronchiectasis and cystic fibrosis due to chronic inflammation in lung. Neoplasm, such as bronchogenic carcinoma or vascular metastatic disease, can also causes hemoptysis.[29]

Indication for nonemergency bronchial arteriography is mild to moderate hemoptysis that was not responded to medication.[41] Less frequent indications are bronchial artery aneurysms, pseudoaneurysms and arteriovenous fistulas. A failure of a thoracic stent graft caused by bronchial artery collateral flow into aneurysm sac (type II endoleak) is additional rare indication.[42] Bronchial arteriography and intervention had been used for patients with lung cancer and other malignancies that produces significant bleeding by hyper vascular metastases to the chest.[29]

Relative contraindications to bronchial arteriography are severe allergy to iodinated contrast and acute or chronic renal diseases.[43] Severe aortoiliac occlusive disease poses challenges in accessing the bronchial artery origin.[44]

Conservative management for massive hemoptysis is associated with mortality rate of 50% to 100%.[11] Reported mortality rate of surgery, performed for massive hemoptysis is 35%.[22] Bronchial artery embolization is a safe and effective alternative to medical or surgical management.[37] Although BAE have risk of recurrent bleeding, it successfully controls acute life-threatening hemoptysis in 73 to 98% of patients.[44]

The bronchial arteries have variable anatomy with different origin and number, course and branching pattern.[10] The bronchial arteries most commonly originates from the descending thoracic aorta at the level of inter vertebral disc between T5 and T6 but can originate anywhere from T3 to T7 level. Eighty percent individuals will have a right bronchial artery arises as a common intercosto bronchial trunk (ICBT) from posterolateral aspect of the descending thoracic aorta. The other bronchial arteries arise from anterolateral aspect of thoracic aorta. Four branching patterns had been described: two on the left and one on the right arising as an intercosto bronchial trunk (40%); one on the left and one intercosto bronchial trunk on the right (20%); two on the left and two on the right (20%); and one on the left and two on the right (10%). Right and left bronchial arteries could originate from thoracic aorta as a common trunk.[10]

Anomalous bronchial arteries may be found in up to 35% individuals, may arise from the arch of aorta, intercostal arteries, thyrocervical trunk, costocervical trunk, internal mammary arteries, subclavian artery, brachiocephalic artery or inferior phrenic artery.[45] Anomalous bronchial vessels also follow the course of the major bronchi. In pleuro parenchymal diseases, arteries supplying the chest wall and diaphragm recruited as systemic non bronchial collaterals. These collaterals supply the lung after crossing diseased pleura. Unlike bronchial artery collaterals, these systemic collaterals do not follow the course of the bronchi.

Assessing the presence of anomalous or collateral blood supply is important when evaluating hemoptysis.[46] When the bronchial arterial supply to a known parenchymal abnormality is not demonstrated during angiography, the procedure become very difficult and will consume time.[47]

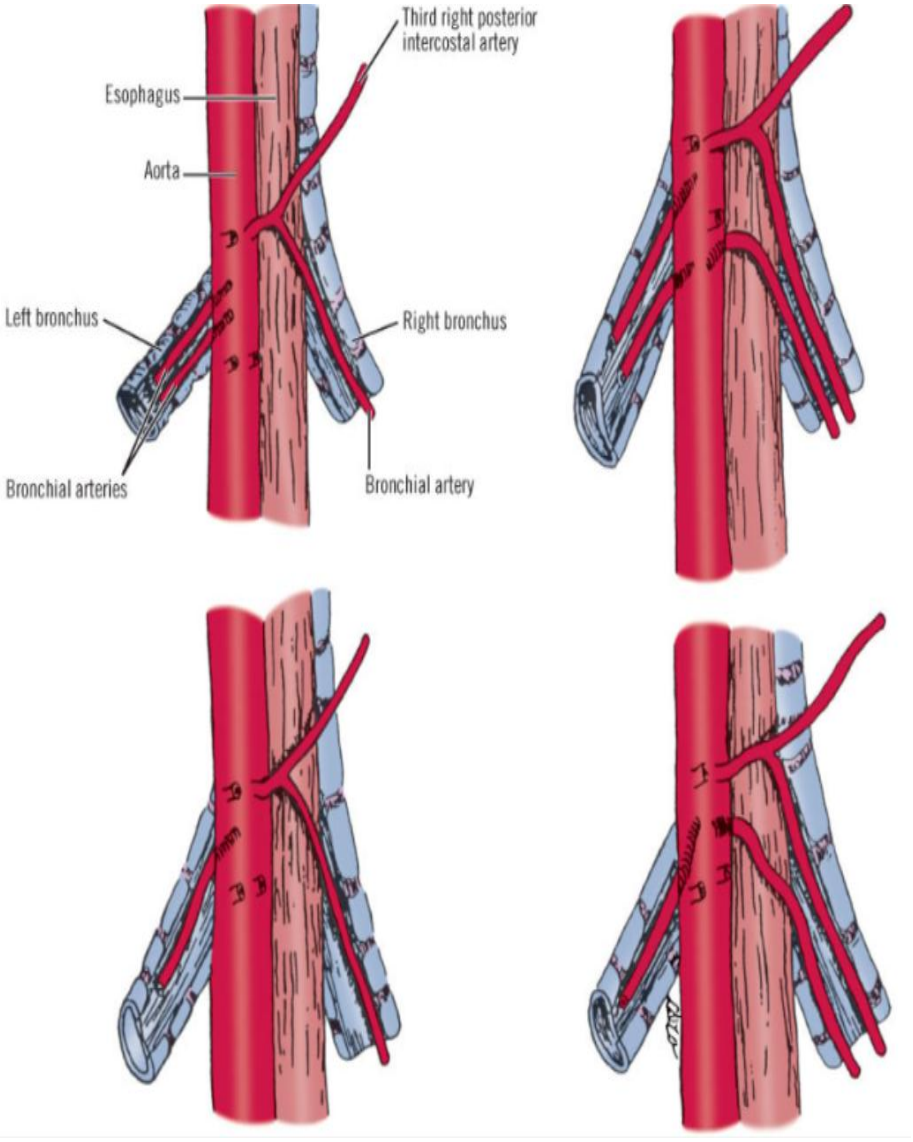


Figure : Four most common sites of origin and numbers of bronchial arteries to the right and left lungs[46]

Number and Origins of Bronchial Arteries in 150 Dissected Autopsy Specimens			
Anatomic Variation	**Number of Right Bronchial Arteries**	**Number of Left Bronchial Arteries**	**Percent Incidence**
I	1	2	40.8
II	1	1	21.3
III	2	2	20.8
IV	2	1	9.7
V	1	3	4.0
VI	2	3	2.0
VII	3	2	0.6
VIII	1	4	0.6
IX	4*	1	0.6

*A branch from the left bronchial artery anterior to the esophagus passing to the right bronchus plus two right bronchial arteries from the aorta and one right bronchial artery from the subclavian artery.

Bronchial artery embolization:

Remy et al first described bronchial artery embolization in the literature in 1970s.[43] It has been published repeatedly in the last few decades as an emergency treatment for hemoptysis.[44]

Bronchial artery embolization is usually indicated to stop massive hemoptysis in patients who were unsuitable for surgical management.[43] The most

common indication are suppurative lung diseases (bronchiectasis) and fibrocavitary disease that causes bronchial artery hypertrophy and consequent bleeding. Less commonly indicated in bronchogenic carcinoma and chronic lung abscess.[11]

Absolute contraindications to bronchial artery embolization are not known. The patient should have hemodynamically stable condition and able to cooperate for doing BAE.[44]

The right intercostal bronchial trunk takes off from aorta at an acute upward angle, whereas the left bronchial arteries leave the aorta more or less at right angles, and special catheters have been designed to facilitate selective catheterization. Selective catheterization of the bronchial artery allows precise delivery of embolic material and prevents spill over into the aorta or inadvertent embolization of spinal artery.

Few criteria exist to determine which bronchial arteries should be embolized when demonstrated angiographically.[32,48] Guidelines are relevant when several bronchial arteries have identified and site of hemorrhage is not obvious from prior thoracic imaging. Embolization is directed towards bleeding vessels which are most likely source of bleeding. Bronchial arteries having diameter >3 mm considered as pathologically enlarged. Embolization done to all significantly enlarged bronchial arteries bilaterally in patients with cystic fibrosis. If abnormal bronchial arteries are not identified, systematic search is made for to identify aberrant bronchial arteries.[48] After embolizing all suspicious systemic

arteries if a patient had hemoptysis, it is necessary to investigate the pulmonary circulation that may be a source of hemorrhage. Embolic materials used for bronchial artery embolization ranging from spheres of polyvinyl alcohol to small pieces of Gelfoam. Coils used in embolization get lodged in the bronchial artery proximally and prevent subsequent catheterization.[32,43,48]

After embolization, many patients had transient fever and chest pain. After 2 days of the procedure some patients had minimal hemoptysis which possibly arises from infarcted bronchial mucosa.[43] Serious complications are rare, the most serious being transverse myelitis, by contrast toxicity rather than inadvertent embolization. Inadvertent spillover of the embolization material into the thoracic aorta may cause ischemia of the leg or abdominal organs. The aim of bronchial artery embolization is immediate control of life-threatening hemoptysis, which is achieved in >75% of patients.[32,43] Failures were usually result from non-identification of bronchial arteries and inability to maintain the catheter position. Recurrent bleed within 6 months after a successful bronchial artery embolization is possible in 20% of individuals. The reasons for recurrent bleed are incomplete embolization, recanalization of previously embolized vessels and hypertrophy of small bronchial arteries which were not initially embolized. However, bronchial artery embolization can be repeated in patients who were re-bleed.[32,43,48]

Surgery

Prevention of hemoptysis and preservation of healthy pulmonary parenchyma are the goals of surgery.[9,28]

Massive hemoptysis associated with active or inactive tuberculosis needs surgical resection. Ideally before surgery the bacterial population should be reduced with drugs as much as possible.[11,28] Patients having aspergilloma should undergo surgery because they were prone for sudden risk of massive hemoptysis. Antifungal agents are ineffective once fungal ball had formed. Due to high risk of complications and mortality, surgical resection was not done frequently. Now surgery is a accepted treatment for aspergilloma after studies showing results with low complication rate. [22]

Life-threatening hemoptysis may give rise to consideration of surgery, although bronchial artery embolization is the initial treatment of choice. A historical adage has been that surgery does not cure bronchiectasis. Innate risk factors are the predisposing factors for recurrence in most cases of bronchiectasis. However, surgery may be considered as appropriate palliative measure in selected cases.[26] Surgery offers three benefits: symptom control, prevention of recurrent hemoptysis and prolongation of better quality of life.[20]

Lobectomy was the commonest surgery done for hemoptysis in the literature.[22] It was done in 82.69% of patients in a study by Khan et al[22]. It was similar to reported by CK Park et al[20] and Jean-Francois Regnard et al[19]. Other procedures done were segmentectomy, bi lobectomy and pneumonectomy. During post-operative period complications found in the above studies were wound infection, air leak, empyema, pneumothorax, hemothorax and wound dehiscence.[22]

AIMS AND OBJECTIVES

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PRIMARY OBJECTIVE:

- To study the causes of moderate to massive hemoptysis in patients admitted at Rajiv Gandhi Government General Hospital, Chennai.

SECONDARY OBJECTIVE:

- To assess the outcome of moderate to massive hemoptysis management in patients during admission to 3 months after discharged from Rajiv Gandhi Government General Hospital, Chennai.

MATERIALS AND METHODS

MATERIALS AND METHODS

STUDY CENTRE:

Rajiv Gandhi Government General Hospital, Chennai

DURATION OF THE STUDY:

7 months from December 2018 to June 2019

STUDY DESIGN:

Descriptive Observational Study

SAMPLING FRAME:

Prospective - Patients with moderate to Massive Hemoptysis admitted in Rajiv Gandhi Govt General Hospital, Chennai.

SAMPLING METHOD:

Consecutive Sampling

SAMPLE SIZE : 93

ETHICAL CLEARANCE : APPLIED

CONSENT:

Informed written consent obtained from all eligible patients.

SUBJECT SELECTION:

INCLUSION CRITERIA:

1. Willingness for informed written consent
2. Patients with moderate to massive hemoptysis admitted in thoracic medicine ward of Rajiv Gandhi Govt General Hospital, Chennai

EXCLUSION CRITERIA:

1. Not willing for informed written consent for the study
2. Previous hospitalisation within 3 months for the same complaints
3. Bleeding from upper respiratory tract

STUDY PROTOCOL:

All prospective patients came to Thoracic Medicine out-patient department with history of hemoptysis were screened. Patients who had true hemoptysis only were included in this study after excluding hematemesis by clinical history and relevant investigations. Amount of hemoptysis enquired from the patient and his/her relatives for quantification and classification. Patients with moderate to massive hemoptysis were admitted in Thoracic Medicine ward of Rajiv Gandhi Government General Hospital, Chennai for further evaluation. Ethical committee approval from the above hospital was obtained before the study was started.

Totally 110 cases with history of moderate to massive hemoptysis came to thoracic medicine out patient department from December 2018 to June 2019 for further management. As per the inclusion/exclusion criteria 15 cases were

excluded. Out of 15 patients 10 had history of previous hospitalisation within 3 months for the same complaints and 5 patients had causes other than hemoptysis like hematemesis and upper respiratory tract bleeding.

Written informed consent was obtained from each patient after giving information about the aims and methods of this study. 2 patients were not given consent for this study. So totally 93 patients were included in this study.

Following data were collected from all the 93 patients

Demographic data

- Name
- Age
- Sex
- Address and contact mobile number

Detailed clinical history with following particulars were collected

History of hemoptysis: duration, frequency, amount of hemoptysis per day, number of episodes in the past.

Constitutional symptoms: fever, loss of appetite, loss of weight

Hemoptysis was classified according to the amount of blood expectorated as[4]

- Mild : <30 ML/Day
- Moderate : 31 – 100 ML/Day
- Severe : 101 – 600 ML/Day

- Massive : >600 ML/Day or any amount of hemoptysis associated with hemodynamic and respiratory compromise

H/o previous ATT: under DOTS or private treatment. pulmonary or extrapulmonary, number of spells of ATT, outcome after completion of treatment
Co morbid conditions like diabetes mellitus, systemic hypertension, coronary artery diseases, chronic obstructive pulmonary disease, chronic kidney disease, chronic liver disease etc., were obtained

No one in our study had chronic kidney disease.

H/O smoking with duration of smoking

Smoking status of the patient was recorded using CDC guidelines

Never smoker: who have never smoked a cigarette or smoked fewer than 100 cigarettes in their life time

Current smoker: who have smoked 100 cigarettes in their lifetime and currently smoking cigarettes everyday (daily) or some days (nondaily)

Former smoker: who had smoked more than 100 cigarettes in their lifetime and does not currently smoke.

H/O drug intake of antiplatelet agents, anticoagulants or antihypertensives obtained. No one in our study were taking antiplatelet agents or anticoagulants.

- Through physical examination of following were done. General examination from head to foot done to note pallor, clubbing, cyanosis, icterus, lymphadenopathy and pedal edema.

- Vital signs: pulse rate, blood pressure, respiratory rate and temperature were recorded.

- Spo2 was recorded by hand held pulse oximetry

- Respiratory and other system examination were done thoroughly

- Coughed out blood examined for colour and food particles if any present.

- ENT examination to rule out upper respiratory tract bleeding in doubtful cases. 2 patients with history of hoarseness of voice were referred for ENT examination and found no evidence of upper respiratory tract bleeding.

Following investigations were done in all the 93 patients

- Blood taken for routine investigations - complete hemogram, liver function test, renal function test, serum for electrolytes and random blood sugar.
- Blood grouping and Rh typing, Prothrombin time, INR were done in all 93 patients. Arterial blood gas analysis was done in patients having severe breathlessness or Spo2 of < 92%.
- Viral markers for HBsAg, HCV and ICTC were done in all study patients.

After controlling hemoptysis, patient was advised to collect sputum free of blood in a screw caped container. Sputum was examined for bacterial and fungal culture. 2 samples of Sputum for Acid fast bacilli smear was done in RNTCP lab by using fluorescent microscope. Sputum was reported with grading of smear. Sputum was positive for acid fast bacilli in 5 patients. Sputum CBNAAT test was done by using Cepheid Gene X pert machine, if both sputum samples were negative for AFB. Sputum cytology for malignant cells was done in patients with constitutional symptoms, smokers and who had negative smear for acid fast bacilli.

Chest radiography and computed tomography (CT) of the chest were taken in all 93 patients to find out location and extent of the lesion.

ECG and echocardiography were done in all 93 patients to find out the cardiac status of the patients.

Cardiologist opinion was obtained for undergoing bronchoscopy or surgery only in indicated patients.

Bronchoscopy was done in 88 out of 93 patients after stabilising their hemodynamic status and control of hemoptysis. 5 patients were excluded because their sputum showed presence of acid fast bacilli. Prothrombin time, INR, blood platelet count, viral markers and cardiologist opinion were checked before procedure. Lignocaine test dose was given a day before the procedure. No one had allergy to lignocaine among the 88 patients. Patient was advised nil per oral in the

early morning for 4 to 5 hours before the procedure. On the day of procedure informed written consent was obtained in local language (tamil) from patient and their relatives. Injection glycopyrrolate was given intravenously 30 – 40 minutes before the procedure to reduce secretions and to prevent vasovagal syncope. Bronchoscopy was done in lying down position under Local anaesthesia. First nasal orifices were plugged with 2% lignocaine gel and patient was asked to take deep inhalation until it reaches the throat. To anaesthetise trachea and proximal airways, 2 ml of 2% lignocaine injection was given in trans cricoid route or 2% lignocaine topical solution sprayed over posterior pharyngeal wall. During the procedure continuous monitoring of electrocardiograph and Spo2 were done. Oxygen was administered in needed cases when the saturation falls below 90%. Fibre optic video bronchoscope was used to examine the airways. Separate bronchoscope was used for those who had positive viral markers. 4 patients had HBsAg positive and 1 had HCV positive. Bronchoscopy findings and site of bleeding were noted. Bronchial wash was taken from the diseased side for AFB smear, CBNAAT, cytology for malignant cells, culture (bacterial and fungal) and drug sensitivity.

Endobronchial biopsy from intra luminal growth and trans bronchial biopsy from diseased side in suspected malignancy or vasculitis were taken. Biopsy material was sent for histopathological examination and immunohistochemistry. During procedure if active bleeding was found iced cold saline or adrenaline given topically at the site via bronchoscope and injection tranexamic acid or adrenochrome monosemicarbazone given intravenously. Post

procedure patient was advised not to take per orally both solids and liquids for 2 hours.

Duration of stay in hospital, interventions done, complications and final outcome were noted.

Confidentiality of data collected from individuals or contributory source was maintained.

Diagnosis was based on following criteria

Active pulmonary tuberculosis was diagnosed by presence of acid-fast bacilli in sputum or bronchial wash and radiological features suggestive of active pulmonary tuberculosis (thick walled cavity and or air space opacity) in whom having constitutional symptoms such as low grade fever, night sweats, loss of appetite and significant weight loss.

Pulmonary tuberculosis sequelae was diagnosed on the basis of absence of acid fast bacilli in sputum & bronchial wash, radiological features (thin walled fibro cavity or calcification) with absence of systemic symptoms other than hemoptysis in whom had previously cured or treatment completed for pulmonary tuberculosis.

Bronchiectasis was diagnosed by history of recurrent respiratory tract infection since childhood, physical examination findings of pan digital clubbing, persistent coarse leathery crackles on auscultation of the chest and confirmed with

radiological signs of bronchiectasis in High resolution computed tomography of the chest (HRCT).

Aspergilloma was diagnosed on the basis of previous history of lung disease in whom showing positive sputum or bronchial wash fungal culture or positive serum aspergillus specific IgG antibody titre and typical radiographic appearance of cavity with air crescent sign in computed tomogram of chest and or bronchoscopic evidence of fungal ball.

Lung cancer was diagnosed by systemic symptoms (significant weight loss, loss of appetite), pressure symptoms (dysphagia, hoarseness of voice, edematous face and limbs), radiological features (mass, nodule or cavity), positive sputum or bronchial wash cytology, bronchoscopy findings and histopathological examination of lung tissue with immunohistochemistry showing type of lung carcinoma.

Pneumonia was diagnosed on the basis of clinical symptoms and sputum or bronchial wash culture showing organism and radiographic findings (consolidation, nodules, cavity or bulging fissure).

Lung abscess was diagnosed by risk factors of aspiration, evidence of organism in sputum or bronchial wash culture and chest radiographic (cavity with air fluid level) findings.

Pulmonary Vasculitis was diagnosed on the basis of physical examination, HRCT of the chest, bronchoscopy, biopsy and serum markers.

All possible treatment options were given and none was withheld

All patients were initially treated with conservative management to control hemoptysis.

Conservative management:

Absolute bed rest, lateral decubitus position of diseased side, protection and maintenance of patent airway by suctioning, oxygen supplementation, intravenous fluids, plasma expanders, whole blood or packed red blood cell transfusion, anti-tussive and anti-fibrinolytic drugs. Anti-tuberculous treatment given only for active pulmonary tuberculosis. Antibiotics given wherever indicated.

Bronchial artery embolization:

Digital subtraction angiography was done prior to bronchial artery embolization. It was indicated in ongoing bleeding not controlled with conservative management or re-bleeding and was done by interventional radiologist of our hospital.

Surgical excision of diseased portion wherever indicated.

Pre-operative evaluation with pulmonary function test was done who were eligible for surgery. Counselling for nutrition and smoking cessation was given during admission and before surgery. Surgery was done under general anaesthesia. Surgery was performed through posterolateral thoracotomy incision

by cardiothoracic surgeon of our hospital. To prevent post-operative atelectasis adequate analgesics and chest physiotherapy were given.

All the Patients were monitored from admission to till discharge in our ward. At the time of discharge patients were advised to come for follow up on weekly basis or immediately if an episode of hemoptysis happens. Follow up was done in thoracic medicine out-patient department of our hospital. All the 89 patients were followed up till 3 months from discharge date, for outcome, complications and recurrent bleeding episodes. During follow up detailed clinical history and physical examination was done in out-patient department. Chest x ray of postero- anterior or lateral view was taken in needed cases.

Descriptive statistics applied and results were given as mean with standard deviation and percentage.

RESULTS

RESULTS

PATIENT CHARACTERISTICS:

A total number of 93 patients with moderate to massive hemoptysis were included in our study after satisfying the inclusion and exclusion criteria.

AGE DISTRIBUTION:

The number of patients in the age group of <30, 31-40, 41-50, 51-60, >60 years were 7 (7.5%), 23 (24.7%), 36 (38.7%), 20 (21.5%) and 7 (7.5%) respectively.

Maximum incidence was seen in 41-50 years group followed by 31-40 years. Minimum age was 22 years and maximum age was 85 years. Mean age was 45.74 years with standard deviation of 10.663.

TABLE 1: AGE DISTRIBUTION

Age	Frequency	Percentage
<= 30 years	7	7.5
31 - 40 years	23	24.7
41 - 50 years	36	38.7
51 - 60 years	20	21.5
> 60 years	7	7.5
Total	93	100

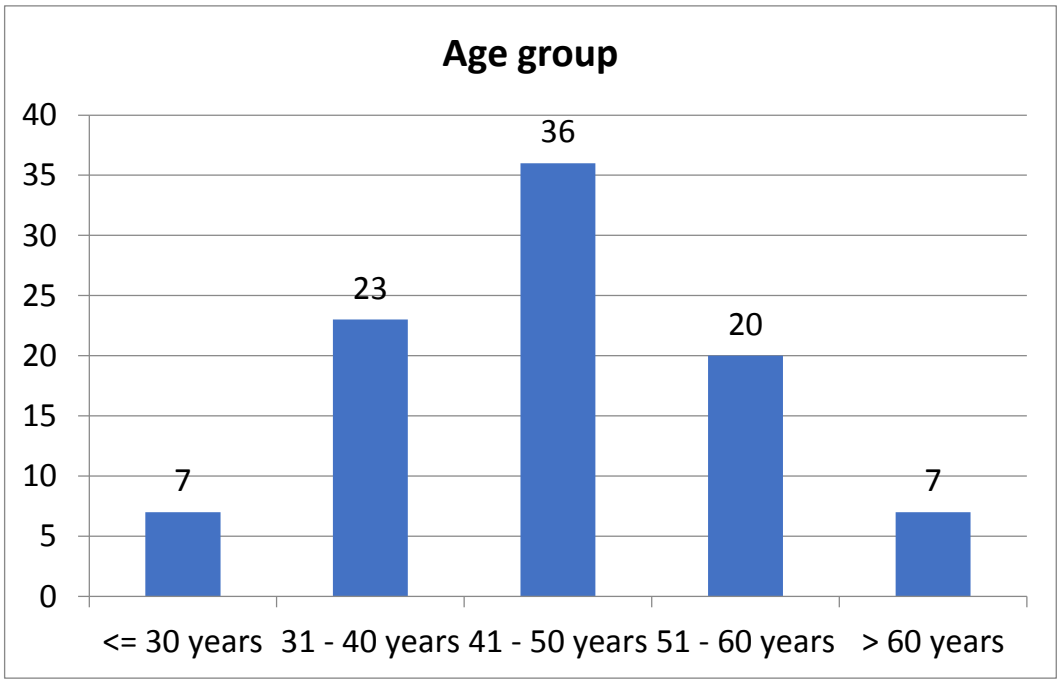

FIGURE 1: AGE DISTRIBUTION

GENDER DISTRIBUTION

Among the 93 patients enrolled in the study 71 were male and 22 were females. Majority of patients were male (76.3%) in this study. Rest of them constitutes females (23.7%).

TABLE 2: GENDER DISTRIBUTION

Sex	Frequency	Percentage
Male	71	76.3
Female	22	23.7
Total	93	100.0

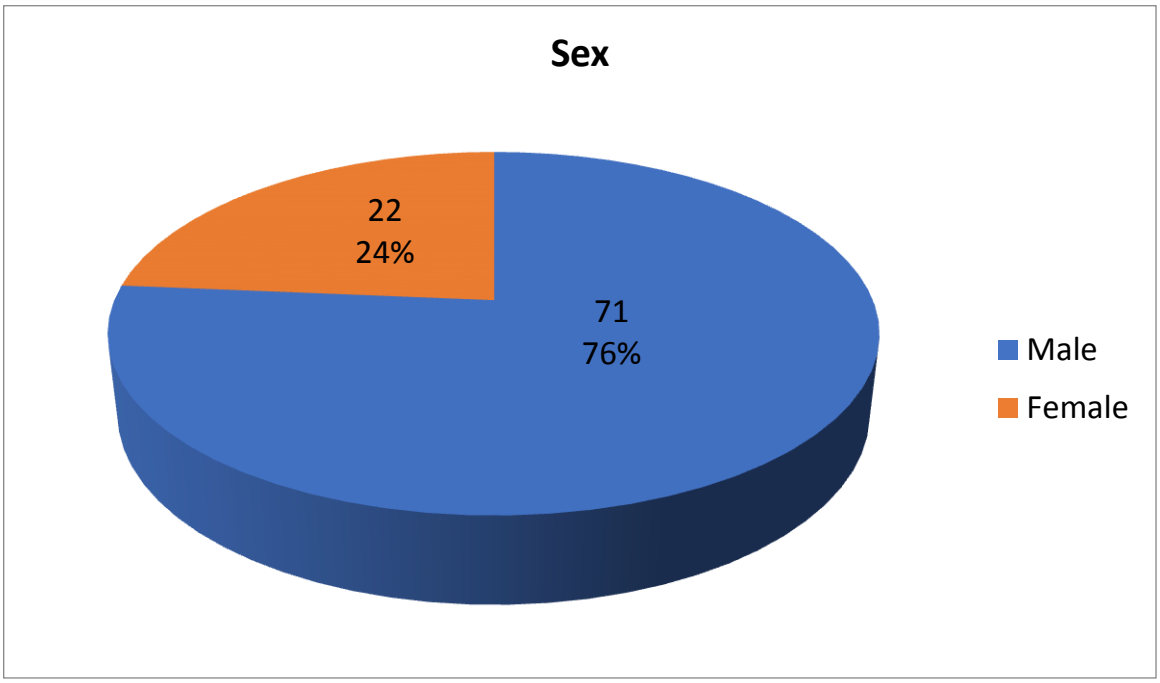

FIGURE 2: GENDER DISTRIBUTION

In the present study 41 – 50 years was the most common age group affected for both males and females. 28 males and 8 females were in the same age group which constitutes 38.7% of the total patients.

TABLE 3: AGE VS SEX

AGE * SEX Crosstabulation				
		SEX		Total
		Male	Female	
AGE	<= 30 years	4	3	7
	31 - 40 years	17	6	23
	41 - 50 years	28	8	36
	51 - 60 years	16	4	20
	> 60 years	6	1	7
Total		71	22	93

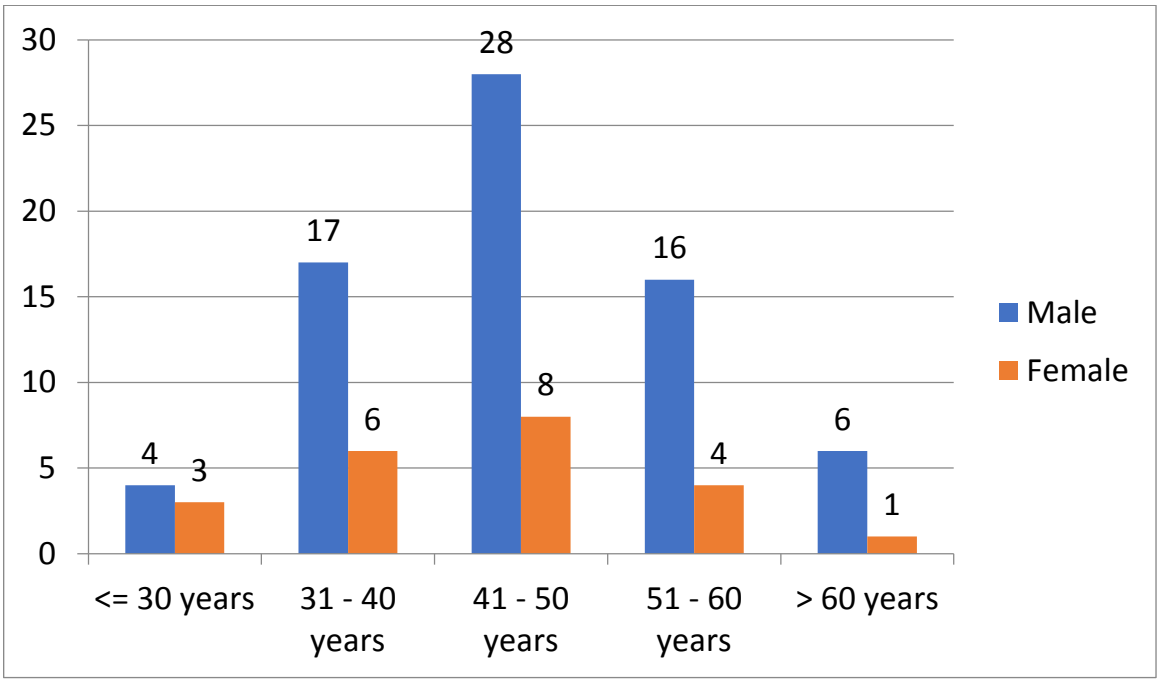

FIGURE 3: AGE VS SEX

DURATION OF HEMOPTYSIS:

Minimum duration of hemoptysis in our study was 1 day and maximum duration was 8 days with standard deviation of 0.907. More number of patients were seek admission prior to hospital admission had 2 days duration of hemoptysis which constitutes 45.1% of the study population. Severe to Massive hemoptysis seeks hospital admission within 1-3 days of onset whereas in moderate hemoptysis they wait from 1 day to 8 days before going to hospital for admission.

TABLE 4: DURATION OF HEMOPTYSIS WITH SEVERITY

Duration of hemoptysis prior to admission	Moderate	Severe	Massive	Total Number of patients	Percentage
1 day	17	7	2	26	28.0
2 days	34	3	5	42	45.1
3 days	14	4	1	19	20.4
4 days	3	0	0	3	3.2
5 days	2	0	0	2	2.2
>= 6 days	1	0	0	1	1.1
Total	71	14	8	93	100.0

CLASS OF HEMOPTYSIS:

Hemoptysis was moderate in 71 (76.3%) patients. Severe in 14 (15.1%) and massive in 8 (8.6%) patients. Moderate hemoptysis was the most common class in study patients.

TABLE 5. CLASS OF HEMOPTYSIS

Class of hemoptysis	Number of patients	Percentage
Moderate	71	76.3
Severe	14	15.1
Massive	8	8.6

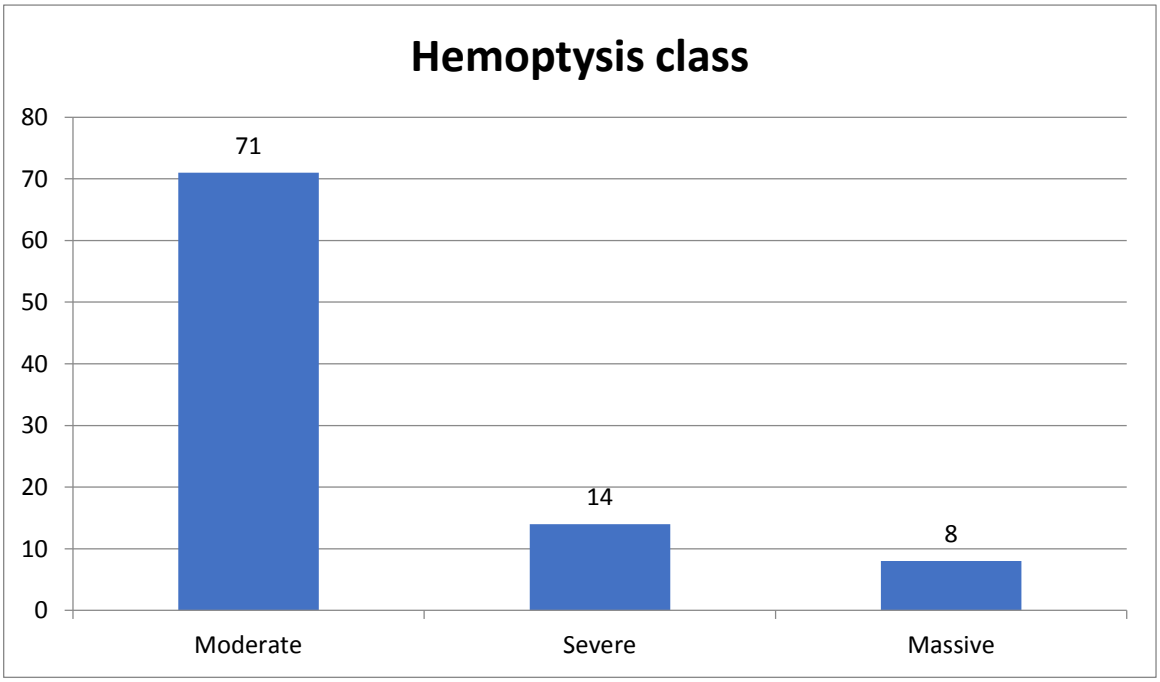

FIGURE 4. DISTRIBUTION OF HEMOPTYSIS SEVERITY IN STUDY PATIENTS

PAST HISTORY OF TB

75 (80.6%) patients had taken treatment for tuberculosis previously. Rest (19.4%) had not taken drugs for tuberculosis in the past. So majority of them were treated for tuberculosis in the past.

TABLE 6. PAST HISTORY OF TUBERCULOSIS

Past history of TB	Total number of patients	Percentage
YES	75	80.6
NO	18	19.4
Total	93	100

Massive hemoptysis was more common in previously treated tuberculosis patients. 9.33% of hemoptysis in previously treated tuberculosis patients was massive hemoptysis in our study. Among the massive hemoptysis 87.5% of were occurred in previously treated tuberculosis patients. Here the p value was not significant.

TABLE 7. COMPARISON OF PAST HISTORY OF TUBERCULOSIS WITH CLASS OF HEMOPTYSIS

Hemoptysis class	Past H/o TB		Total	Fisher exact p value
	Yes	No		
Moderate	55 (73.33%)	16 (88.88%)	71 (76.34%)	0.095
Severe	13 (17.33%)	1 (5.55%)	14 (15.05%)	
Massive	7 (9.33%)	1 (5.55%)	8 (8.6%)	
Total	75 (100%)	18 (100%)	93 (100%)	

CO MORBID CONDITIONS:

The commonest co morbidity was chronic obstructive pulmonary disease found in 32 patients (34.4%). None of them had acute exacerbation of COPD at the time of admission. Diabetes mellitus was found in 18 (19.35%) patients and constitutes second position. Others were coronary artery disease 1 (1.07%) and chronic liver disease 1 (1.07%). No one had chronic kidney disease.

TABLE 8: CO-MORBIDITY STATUS

Comorbidities	Count	%
CAD	1	1.1%
CLD, COPD	1	1.1%
COPD	29	31.2%
DM	15	16.1%
DM, CAD	1	1.1%
DM, COPD	2	2.2%
NO	44	47.3%

SMOKING STATUS:

In the present study majority (40) were never smoker (43%). 34 patients (36.6%) were current smoker. 19 patients (20.4%) were former smoker not taking tobacco since last 6 months.

TABLE 9. SMOKING STATUS OF STUDY POPULATION

Smoking status	Total number of patients	Percentage
Current	34	36.6
Former	19	20.4
Never	40	43.0
Total	93	100

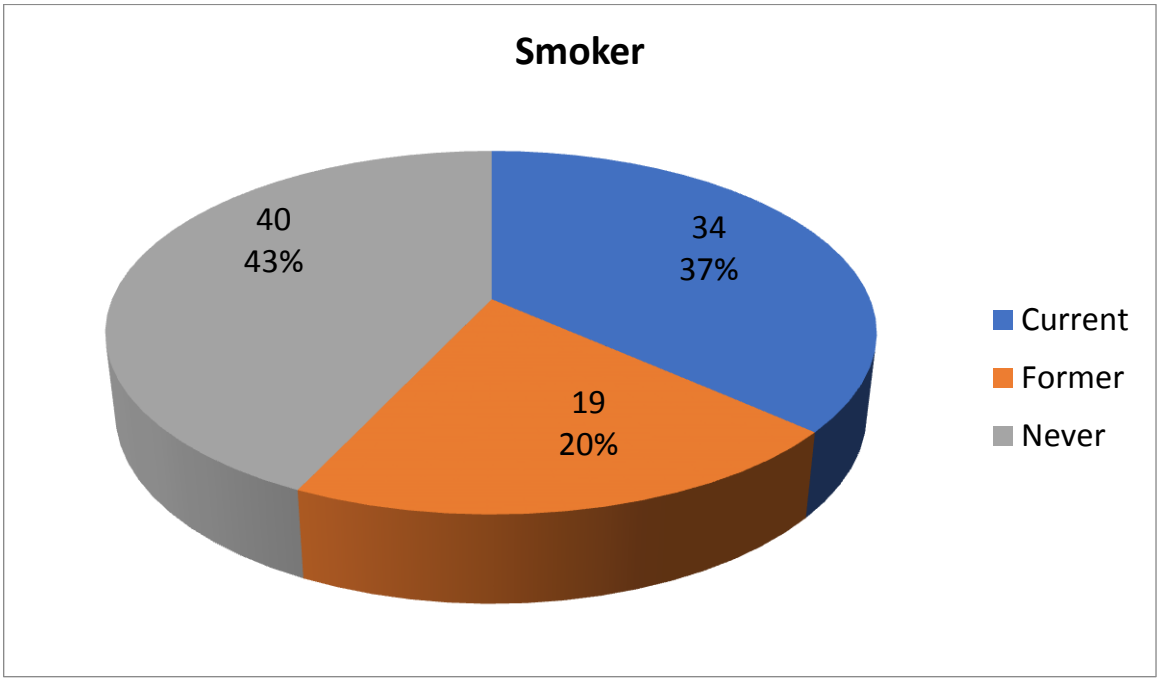

FIGURE 5. DISTRIBUTION OF PATIENTS ACCORDING TO SMOKING STATUS

BASELINE PARAMETERS

Minimum duration of hospital stay was 1 day and maximum day was 23 days with standard deviation of 4. Mean duration of hospital stay was 11 days. Mean pulse rate, systolic blood pressure, hemoglobin, blood cell count, total protein and albumin were within normal limits.

TABLE 10: BASELINE PARAMETERS (n = 93)

	Mean	**Standard Deviation**	**Minimum**	**Maximum**
Duration of Hospital Stay	11	4	1	23
PR	95	14	68	130
BP	115	15	90	140
HB	11.8	2.0	7.0	15.6
RBC	3.94	0.73	2.53	5.72
PLATELET	2.73	0.69	1.64	5.50
TC	10.63	3.38	5.75	21.20
TP	7.2	0.6	5.8	8.2
ALB	3.6	0.5	2.6	4.6

DISEASED LOBE FOUND BY CT CHEST:

CT Chest was done in all 93 patients. Many of them (44.1%) had multiple lobe involvement. Right upper lobe only was involved in 25.8% and Left upper lobe was in 20.4% of patients. 4.3% had right lower lobe and 3.2% had left lower lobe involvement by CT Chest. Right middle lobe was involved in only 2.2%.

TABLE 11: DISEASED LOBE FOUND BY CT CHEST

	Lobe involvement	Count	Percentage
AREA	Left Lower Lobe	3	3.2%
	Left Upper Lobe	19	20.4%
	Multi-lobar	41	44.1%
	Right Lower Lobe	4	4.3%
	Right Middle Lobe	2	2.2%
	Right Upper Lobe	24	25.8%

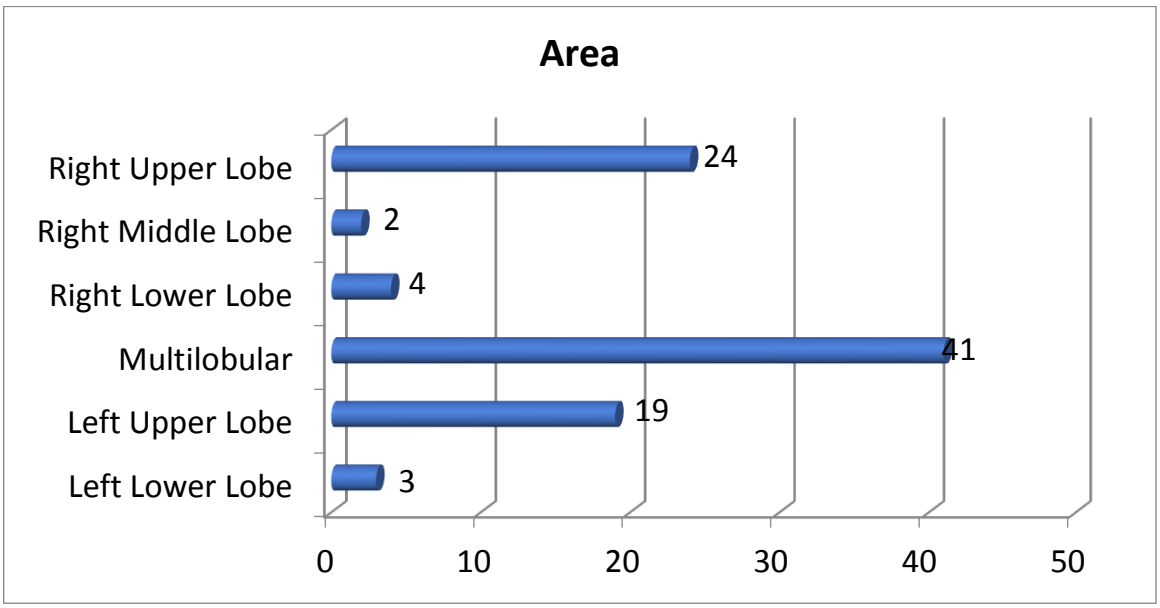

FIGURE 6. LOBE INVOLVEMENT IN HEMOPTYSIS

RADIOLOGICAL LESIONS IN CT CHEST:

Consolidation was the most common radiological lesion found in 35 (37.6%) and fibrosis or cavity or both was the second commonest found in 25 (26.9%) patients. 22 (23.7%) patients has bronchiectatic changes. Air crescent sign in cavity was seen in 6 (6.5%) patients. 5 (5.4%) patients had mass lesion in CT Chest.

TABLE 12: LESIONS IN CT CHEST

	Count	Percentage
Aspergilloma	6	6.5%
Bronchiectasis	22	23.7%
Consolidation	35	37.6%
Fibrosis/Cavity	25	26.9%
Mass	5	5.4%

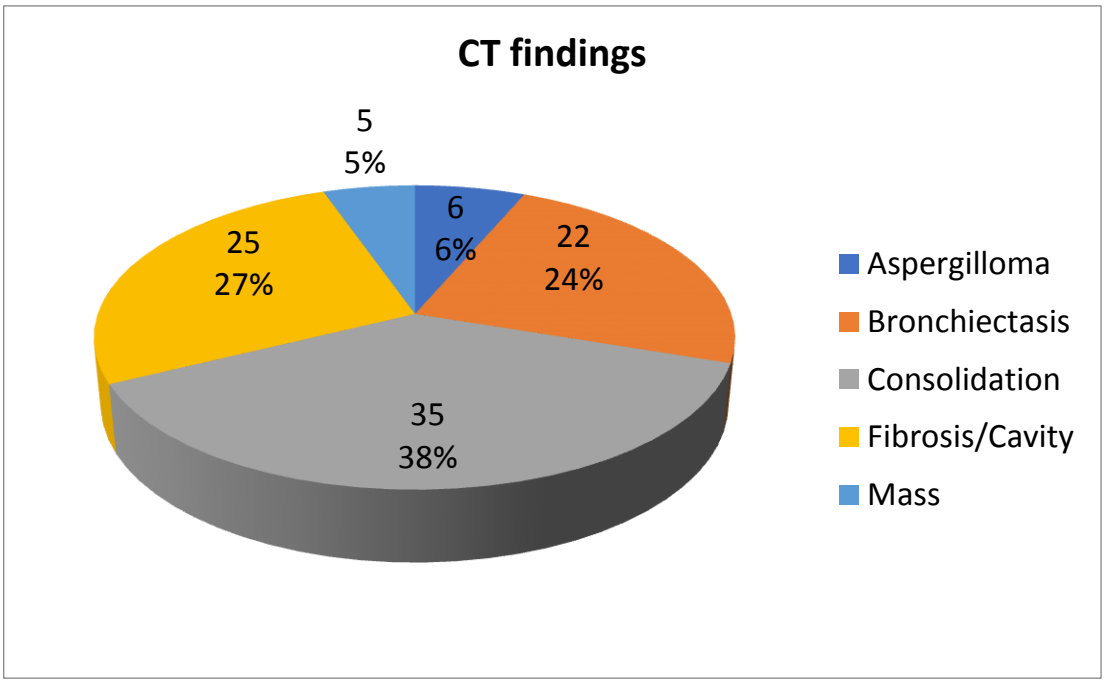

FIGURE 7. CT CHEST FINDINGS IN HEMOPTYSIS

CAUSES OF HEMOPTYSIS:

Tuberculosis was the most common cause of moderate to massive hemoptysis found in 73.1% (68) of patients. Among them active pulmonary tuberculosis was noted in 44.12% (30) and sequelae of pulmonary tuberculosis was found in 55.88% (38) of patients.

TABLE 13: CAUSE OF MODERATE TO MASSIVE HEMOPTYSIS

		Count	%
Cause of Hemoptysis	Aspergilloma	6	6.5%
	Bronchiectasis	8	8.6%
	Carcinoma lung	5	5.4%
	Lung Abscess	2	2.2%
	Pneumonia	3	3.2%
	PT sequelae	38	40.9%
	Active PTB	30	32.3%
	Pulmonary Vasculitis	1	1.1%

Bronchiectasis was in 8.6% (8) and aspergilloma was in 6.5% (6) of patients found as etiology. Primary carcinoma of lung was diagnosed in 5.4% (5) of patients. The other important causes were community acquired pneumonia 3.2% (3), lung abscess 2.2% (2) and vasculitis 1 (1.1%).

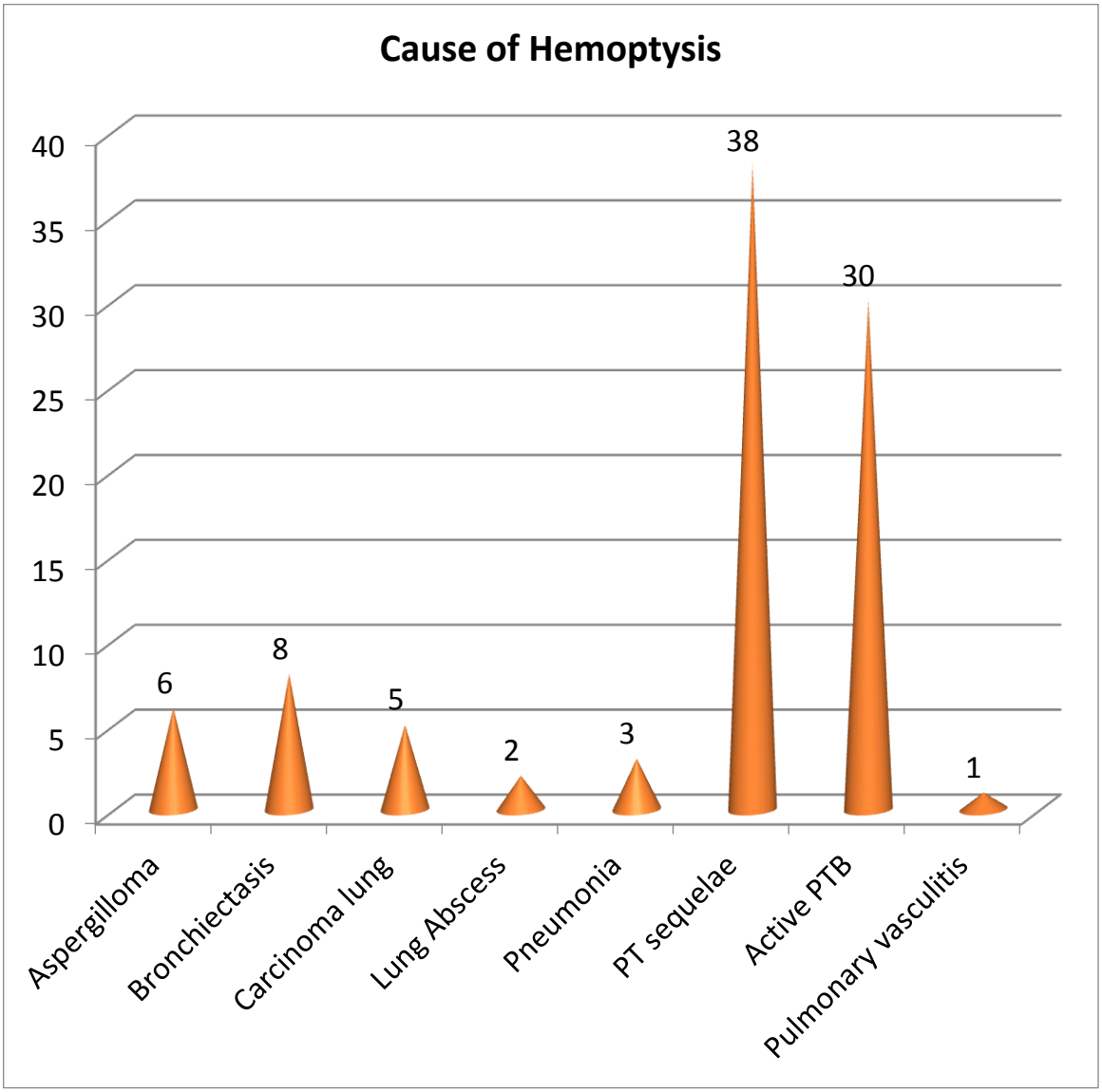

FIGURE 8. CAUSE OF MODERATE TO MASSIVE HEMOPTYSIS

Severe to massive hemoptysis in our study was mainly due to pulmonary tuberculosis (active and sequelae), aspergilloma and bronchiectasis. Carcinoma lung, pneumonia, lung abscess and pulmonary vasculitis in our study only caused moderate hemoptysis. Here the p value is 0.022 and is significant in our study.

TABLE 14. COMPARING CAUSES WITH SEVERITY OF HEMOPTYSIS

Causes of Hemoptysis	**Hemoptysis class**						**Fisher exact p value**
	Moderate		**Severe**		**Massive**		
	Count	**%**	**Count**	**%**	**Count**	**%**	
Aspergilloma	3	50.0%	2	33.3%	1	16.7%	**0.022**
Bronchiectasis	5	62.5%	2	25.0%	1	12.5%	
Carcinoma lung	5	100.0%	0	0.0%	0	0.0%	
Lung Abscess	2	100.0%	0	0.0%	0	0.0%	
Pneumonia	3	100.0%	0	0.0%	0	0.0%	
PT sequelae	28	73.7%	7	18.4%	3	7.9%	
Active PTB	24	80.0%	3	10.0%	3	10.0%	
Pulmonary Vasculitis	1	100.0%	0	0.0%	0	0.0%	

Multiple lobe involvement (44%) were seen in pulmonary tuberculosis (active and sequelae), bronchiectasis, pneumonia and pulmonary vasculitis in our study. Right upper lobe is more commonly involved than other lobes followed by left upper lobe, right lower lobe, left lower lobe and right middle lobe. Here the P value is significant 0.001.

TABLE 15. COMPARISON OF CAUSES WITH LOBE INVOLVEMENT IN LUNG

Causes of Hemoptysis	AREA						Fisher exact p value
	Right Upper Lobe	Right Middle Lobe	Right Lower Lobe	Left Upper Lobe	Left Lower Lobe	Multiple lobe	
Aspergilloma	2	0	0	4	0	0	**0.001**
Bronchiectasis	2	0	0	0	2	4	
Carcinoma lung	3	0	0	1	1	0	
Lung Abscess	0	1	1	0	0	0	
Pneumonia	0	0	0	2	0	1	
PT sequelae	7	0	1	6	0	24	
Active PTB	10	1	2	6	0	11	
Pulmonary Vasculitis	0	0	0	0	0	1	

Radiologically consolidation was the most common lesion found in 90% of active pulmonary tuberculosis. Other lesions were bronchiectasis and fibrocavity found in already treated patient. Now they presents with recurrence. Fibrocavity was the most common lesion of PT sequelae and was found in 61% of them. Pulmonary vasculitis presented with radiologically as consolidation. Here the p value was 0.001 and is significant.

TABLE 16. COMPARISON OF CAUSES AND FINDINGS OF CT CHEST

Cause of Hemoptysis	**CT Findings**					**Total**	**Fisher exact p value**
	Aspergilloma	**Bronchiectasis**	**Consolidation**	**Fibrosis / Cavity**	**Mass**		
Aspergilloma	6	0	0	0	0	6	**0.001**
Bronchiectasis	0	8	0	0	0	8	
Carcinoma lung	0	0	0	0	5	5	
Lung Abscess	0	0	2	0	0	2	
Pneumonia	0	0	3	0	0	3	
PT sequelae	0	13	2	23	0	38	
Active PTB	0	1	27	2	0	30	
Pulmonary Vasculitis	0	0	1	0	0	1	
Total	6	22	35	25	5	93	

Secondary bacterial infection as a cause of active hemoptysis was diagnosed in 35 patients. The most common organisms were Pseudomonos (20), Klebsiella (14) and Acinetobacter (1) isolated in sputum or bronchial wash culture. Secondary bacterial infection were common in bronchiectasis and PTB sequelae.

TABLE 17: CAUSES OF SECONDARY BACTERIAL INFECTION

Organisms	Count	Percentage
Nil	58	62.4
Acinetobacter	1	1.1
Klebsiella	14	15.1
Pseudomonos	20	21.5

MANAGEMENT OF HEMOPTYSIS:

71% (66) of patients were responded to conservative treatment and hemoptysis was controlled. Twenty-seven patients (29%) underwent intervention for controlling ongoing and recurrent bleeding. Out of 27 patients, bronchial artery embolization was done in 23 (85%) and surgery done in 4 (15%) patients. Out of 93 patients with above management 19 (20.4%) patients had recurrent hemoptysis.

TABLE 18. MANAGEMENT DONE FOR HEMOPTYSIS

		Count	Percentage
Management	Conservative	66	71.0
	BAE	23	24.7
	Surgery	4	4.3

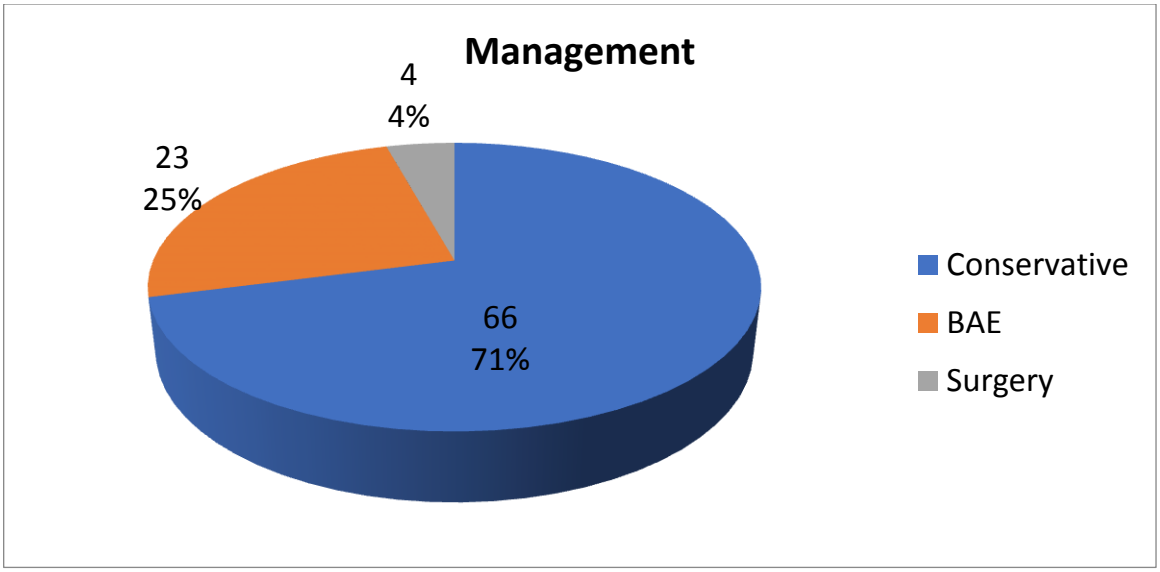

FIGURE 9. TYPES OF MANAGEMENT DONE IN HEMOPTYSIS

4 Patients were died due to massive hemoptysis and asphyxiation of blood in spite of the all resuscitative measures. The overall mortality rate was 4.3%.

TABLE 19. OUTCOME OF MANAGEMENT

		Count	Percentage
Outcome	Alive	89	95.7
	Dead	4	4.3

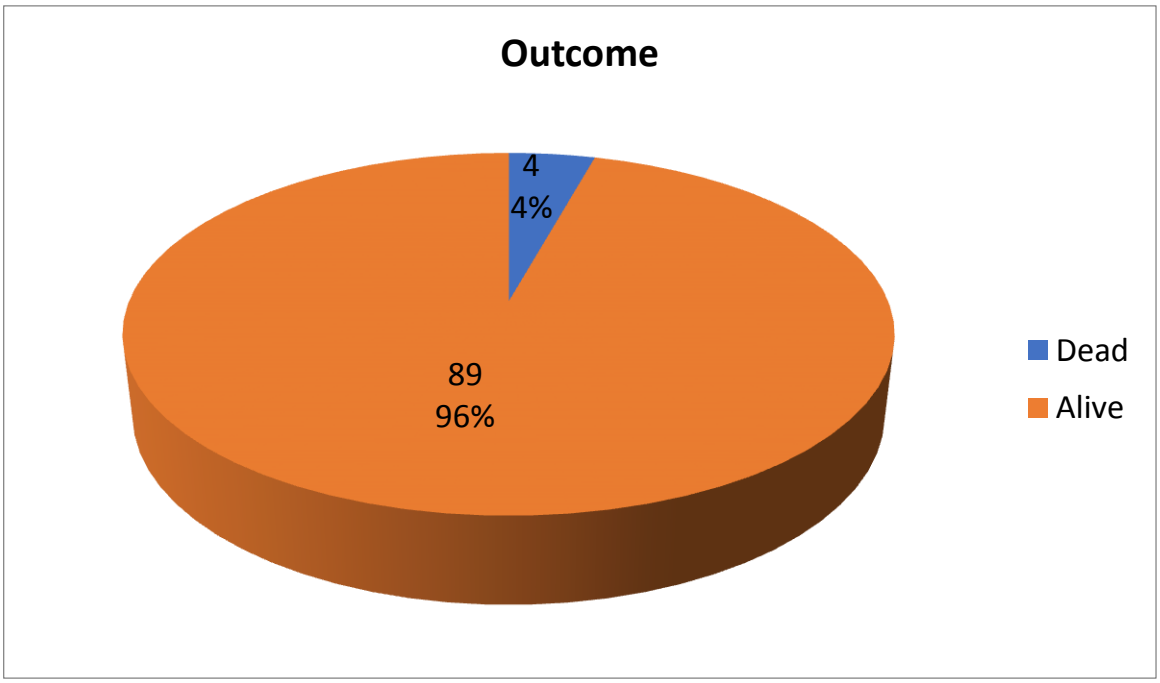

FIGURE 10. FINAL OUTCOME OF MODERATE TO MASSIVE HEMOPTYSIS

Hemoptysis of 2 to 3 days duration prior to admission was associated with mortality in 4 out of 61 patients in our study. All the 4 patients had massive hemoptysis. Here the p value was 0.188 and was not significant in our study.

TABLE 20: COMPARISON OF DURATION OF HEMOPTYSIS WITH DEATH

Duration of Hemoptysis	Outcome		Total	Fisher exact p value
	Dead	Alive		
1	0 (0%)	26 (100%)	26 (100%)	0.188
2	3 (7.31%)	38 (92.68%)	41 (100%)	
3	1 (5%)	19 (95%)	20 (100%)	
4	0 (0%)	3 (100%)	3 (100%)	
5	0 (0%)	2 (100%)	2 (100%)	
>=6	0 (0%)	1 (100%)	1 (100%)	
Total	4 (4.3%)	89 (95.69%)	93 (100%)	

In the present study moderate and severe hemoptysis associated with good prognosis. Massive hemoptysis had poor outcome. 4 out of 8 (50%) patients were died in massive hemoptysis group.

TABLE 21: COMPARISON OF SEVERITY OF HEMOPTYSIS WITH OUTCOME

Hemoptysis class	Outcome		Total	Fisher exact p value
	Dead	Alive		
Moderate	0 (0%)	71 (100%)	71 (100%)	**< 0.001**
Severe	0 (0%)	14 (100%)	14 (100%)	
Massive	4 (50%)	4 (50%)	8 (100%)	
Total	4 (4.3%)	89 (95.69%)	93 (100%)	

All the 4 died patients had taken anti-tuberculous treatment previously. Among the patients who had tuberculosis in the past had a mortality rate of 5.33%. p value of this was 0.416 not significant.

TABLE 22: COMPARING OUTCOME WITH PAST HISTORY OF TUBERCULOSIS

Past H/o TB	Outcome		Total	Fisher exact p value
	Dead	Alive		
Yes	4 (5.33%)	71 (94.66%)	75 (100%)	0.416
No	0 (0%)	18 (100%)	18 (100%)	
Total	4 (4.3%)	89 (95.69%)	93 (100%)	

Out of 4 death 2 were former smoker and remaining 2 were never smoker. Out of 19 former smoker 2 patients (10.52%) were died and 2 patients (5%) out of 40 never smoker were died in our study.

TABLE 23: COMPARING SMOKER WITH OUTCOME OF HEMOPTYSIS

Smoker	Outcome		Total	Fisher exact p value
	Dead	Alive		
Current	0 (0%)	34 (100%)	34 (100%)	0.160
Former	2 (10.52%)	17 (89.47%)	19 (100%)	
Never	2 (5%)	38 (95%)	40 (100%)	
Total	4 (4.3%)	89 (95.69%)	93 (100%)	

In this study duration of hospital stay, blood pressure and total leukocyte count had significant P value of 0.001 by ‘t’ test.

TABLE 24. VITAL SIGNS AND BLOOD PARAMETERS

	GROUP	N	MEAN	STD. DEVIATION	p VALUE BY ‘t’ TEST
Duration of Hemoptysis	Dead	4	2.25	0.50	0.678
	Alive	89	2.06	0.92	
Duration of hospital Stay	Dead	4	5.75	4.03	**0.009**
	Alive	89	11.01	3.84	
PR	Dead	4	93.00	15.87	0.749
	Alive	89	95.26	13.69	
BP	Dead	4	100.00	8.16	**0.034**
	Alive	89	115.84	14.56	
HB	Dead	4	10.35	0.87	0.136
	Alive	89	11.86	1.99	

	GROUP	N	MEAN	STD. DEVIATION	p VALUE BY 't' TEST
RBC	Dead	4	3.81	0.69	0.699
	Alive	89	3.95	0.73	
PLATELET	Dead	4	2.90	0.66	0.622
	Alive	89	2.73	0.70	
TC	Dead	4	15.98	3.87	**0.001**
	Alive	89	10.39	3.17	
TP	Dead	4	6.90	0.93	0.586
	Alive	89	7.18	0.56	
ALB	Dead	4	3.63	0.48	0.954
	Alive	89	3.64	0.53	

Among the 4 died 50% had multiple lobe involvement and another 50% had left upper lobe involvement. Mortality rate among the multiple lobe involvement is 4.87% (2) and left upper lobe involvement is 10.52% (2). P value was 0.168 and not significant.

TABLE 25. COMPARISON OF LOBE INVOLVEMENT WITH OUTCOME

Lobular Involvement	Outcome		Total	Fisher exact p value
	Dead	Alive		
Multi-lobar	2 (4.87%)	39 (95.12%)	41 (100%)	0.168
Left Upper Lobe	2 (10.52%)	17 (89.47%)	19 (100%)	
Other Lobes	0 (0%)	33 (100%)	33 (100%)	
Total	4 (4.3%)	89 (95.69%)	93 (100%)	

Out of the 4 patients died 2 had consolidation, 1 had bronchiectasis and remaining one had fibro cavity in CT scan of the Chest. P value was 0.2 and not significant.

TABLE 26. COMPARING OUTCOME WITH CT CHEST FINDINGS

CT Findings	Outcome		Total	Fisher exact p value
	Dead	Alive		
Aspergilloma	0 (0%)	6 (100%)	6 (100%)	0.200
Bronchiectasis	1 (4.54%)	21 (95.45%)	22 (100%)	
Consolidation	2 (5.71%)	33 (94.28%)	35 (100%)	
Fibrosis/Cavity	1 (4%)	24 (96%)	25 (100%)	
Mass	0 (0%)	5 (100%)	5 (100%)	
Total	4 (4.3%)	89 (95.69%)	93 (100%)	

Four patients were died from this study by asphyxiation and the etiology were active pulmonary tuberculosis in 2, pulmonary tuberculosis sequelae in 1 and bronchiectasis in 1. P value was 0.001 and is significant.

TABLE 27. COMPARISON OF CAUSES AND OUTCOME OF HEMOPTYSIS

Cause of Hemoptysis	**Outcome**		**Total**	**Fisher exact p value**
	Dead	**Alive**		
Aspergilloma	0	6	6	
Bronchiectasis	1	7	8	
Carcinoma lung	0	5	5	
Lung Abscess	0	2	2	
Pneumonia	0	3	3	**0.001**
PT sequelae	1	37	38	
Active PTB	2	28	30	
Pulmonary Vasculitis	0	1	1	
Total	4	89	93	

4 patients (6.06%) out of 66 patients managed by conservative treatment were died. No one from BAE or surgery were died. But not significant and the p value was 0.246.

TABLE 28. COMPARING OUTCOME WITH MANAGEMENT

Management	Outcome		Total	Fisher exact p value
	Dead	Alive		
Conservative	4 (6.06%)	62 (93.93%)	66 (100%)	0.246
BAE	0 (0%)	23 (100%)	23 (100%)	
Lobectomy	0 (0%)	4 (100%)	4 (100%)	
Total	4 (4.3%)	89 (95.69%)	93 (100%)	

Pulmonary tuberculosis (active/sequelae), bronchiectasis and aspergilloma were associated with recurrent hemoptysis in our study. This was significant with p value of 0.021.

TABLE 29. COMPARING CAUSES WITH RECURRENT BLEEDING

Cause of Hemoptysis	**Recurrence**		**Total**	**Fisher exact p value**
	Yes	**No**		
Aspergilloma	1	5	6	**0.021**
Bronchiectasis	1	7	8	
Carcinoma lung	0	5	5	
Lung Abscess	0	2	2	
Pneumonia	0	3	3	
PT sequelae	8	30	38	
Active PTB	9	21	30	
Pulmonary Vasculitis	0	1	1	
Total	19	74	93	

21.05% of recurrent bleeding is associated with death among moderate to massive hemoptysis in our study population.

TABLE 30. COMPARING RECURRENT HEMOPTYSIS WITH OUTCOME

Recurrence	Outcome		Total	Fisher exact p value
	Dead	Alive		
Yes	4 (21.05%)	15 (78.95%)	19 (100%)	0.394
No	0 (0%)	74 (100%)	74 (100%)	
Total	4 (4.3%)	89 (95.69%)	93 (100%)	

TABLE 31. COMPARISON OF EACH MANAGEMENT WITH RECURRENT HEMOPTYSIS AND OTHER COMPLICATIONS

		Management						Fisher exact p value
		Conservative		BAE		Lobectomy		
		Count	%	Count	%	Count	%	
Recurrent bleeding	**Yes**	17	89.4%	2	10.6%	0	0.0%	**0.023**
	No	49	66.2%	21	28.3%	4	5.4%	
Complications	**Nil**	66	75.9%	17	19.5%	4	4.6%	**0.01**
	Chest pain	0	0.0%	5	100.0%	0	0.0%	
	Mono paresis	0	0.0%	1	100.0%	0	0.0%	

Recurrent hemoptysis was more common after conservative management. 89.4% of recurrent hemoptysis were occur after conservative treatment, remaining 10.6% was after embolization therapy. One major complication in our study was right lower limb monoparesis after bronchial artery embolization due to inadvertent spinal artery embolization. Minor complication was chest pain after embolization found only in 5 patients.

DISCUSSION

DISCUSSION

Hemoptysis is a common life-threatening situation in India and all other parts of the world. Causes of hemoptysis varies from infection to malignancy. It depends upon geographical area, the institution where research is conducted and time of study within the same institution.

In this descriptive study various causes of hemoptysis were evaluated from patients admitted in a tertiary care centre. A total of 93 patients with moderate to massive hemoptysis were evaluated in our study.

Analysis of age distribution showed that hemoptysis was commonly found in the age group 41-50 years (38.7%) followed by 31 – 40 years (24.7%) in this study. This Age group was common for both males and females. It is corresponded with a study done by Das et al[1] which reported as most common age group was 40-49 years (23%) followed by 50-59 years (22%). Age group of 30 to 50 years is common in a study conducted by Rachakonda et al[6] at Guntur, south India which was similar to our study results.

Male to female ratio in the present study was 3.22:1 concluding male were thrice more susceptible than females for hemoptysis. Nawal et al[2] found that hemoptysis was 2.23 times more common in male than female. Our findings were similar to those found by Bhalla et al[4] (3.57:1).

Moderate hemoptysis contributes to majority and was found in 76.3% in the present study. It was similar to various studies conducted in India. Singh et al[5] found it in 73.4% of his total 346 patients.

Smoking is considered as important risk factor for development of hemoptysis. In the present study former and current smokers contributes to 57% of hemoptysis. Similar results were seen in Rachakonda et al[6] and Nawal et al[2]. Among the 4 patients died 2 were former smoker who quit smoking within 6 months of time. Nawal et al[2] in their study showed 64.55% of smokers (current and former) in hemoptysis patients. Never smoker in my study was 43%, that was similar to study conducted by Das et al[1] which was 46%.

Diabetes mellitus was found in 19% of our study population. It was 16% in a study done by Ronald win b et al[3]. 62.5% of massive hemoptysis had diabetes. Out of the total 18 diabetics 27.7% had massive hemoptysis.

Pseudomonos and Klebsiella were the commonest organism causing secondary infection in our study which was similar to study conducted by Bhalla et al[4].

Cause of hemoptysis was different in developing countries like India when compared to developed countries. Infections are still a major causative factor in developing countries. Due to changing epidemiology of each disease, cause may vary over a period of time in the same geographical area. Hemoptysis was considered as one of the symptoms suggestive of tuberculosis is now being

replaced by other diseases too like bronchiectasis, lung malignancy and pneumonia.

Studies done during the 1940s and 1950s in developed countries showed tuberculosis was the most common cause of hemoptysis.[49] Abbot OA[49] done a study in USA (Atlanta) reported as Tuberculosis was a most common cause for hemoptysis in 22% of patients closely followed by bronchiectasis (21%) and malignancy(21%) which were ranked second position. Subsequent studies in developed countries during 1977-1985, 1974-1981 and 1980-1995 demonstrated decreasing trend of tuberculosis from 22 to 1%. Pulmonary tuberculosis is now becoming less important cause of hemoptysis in developed countries.

Study from India by Rao in 1960 reported as tuberculosis was the most common cause of hemoptysis. This scenario remains unchanged as evidence from this present study (73.2%) and other published studies from India. [2,5,15,17] In our present study pulmonary tuberculosis (active and sequelae) was the most common cause of moderate to massive hemoptysis and it contributes to 73.2% of patients. Out of this 73.2% active pulmonary TB contributes 44% (30). Remaining 56% (38) were PT sequelae patients. 79.2% of patients with hemoptysis were diagnosed was tuberculosis in a study by Prasad et al[15] on 2009 and by Singh et al[5] on 2016. Reports from other developing countries also projects pulmonary tuberculosis remains the most common cause of hemoptysis.[50,51] All the active pulmonary tuberculosis patients in our study were confirmed microbiologically from sputum or bronchial wash.

Presence of hemoptysis does not merely indicate presence of active pulmonary tuberculosis. It may occur as an initial clinical manifestation of active pulmonary tuberculosis, during treatment or even after cure of the disease. In the present study pulmonary tuberculosis sequelae was responsible for 40.9% out of 93 patients and the bacilli was absent in biological specimen. Both active pulmonary tuberculosis and sequelae are most common cause of hemoptysis as evidence from this current study. Higher incidence of tuberculosis in present study was due to prevalence of tuberculosis in our country is still high.

In pulmonary tuberculosis sequelae structural damage formed already by organism result in stagnation of secretion leads in to secondary infections and that may cause hemoptysis.[15] Secondary bacterial infections were present in patients with bronchiectasis and pulmonary tuberculosis sequelae of this present study. In these patients anti-tuberculous treatment was not needed to control active bleeding. Secondary bacterial infection is an additional factor for causing hemoptysis in the study patients, justify the role of antibiotics in conservative management.

If a patient had episode of hemoptysis during anti tuberculous treatment, drugs should not be withheld and continued along with other conservative management. He should be investigated further for drug resistant strains after confirming good adherence with drugs. In the present study no patient was diagnosed as drug resistant tuberculosis.

Bronchiectasis was the second commonest cause of life threatening hemoptysis in this present study showing 8.6%. Bhalla et al[4] reported 9.3% of hemoptysis in their study was caused by bronchiectasis in 2017. It was closely similar to our study report. Patel et al[7] reported bronchiectasis in 4% of their 50 patients on 2015. Other studies from India had a incidence of bronchiectasis in up to 6.6% of patients[2,15,17,33,52,53]. Highest incidence of bronchiectasis (14%) than other etiology found in a study by Das et al[1] in north eastern India during 2016-2017 .

Aspergilloma was the third leading cause in our present study. It constitutes 6.5% of 93 patients. Aspergilloma was a cause in 1.39% (3) of hemoptysis patients in a study by Rachakonda et al[6] at Guntur, south India. Our study result was almost similar to Ronald win b et al[3] Study at Thiruvananthapuram, south India which showed aspergilloma was the cause in 4% of their patients.

In the present post tuberculosis era, nontuberculous causes like bronchiectasis, malignancy and pneumonia are the other important causes of hemoptysis in developed countries.

Incidence of malignancy in various developed countries had ranging from 5 – 44%.[54–57] Indian studies had no high incidence of lung malignancies as a cause for moderate to massive hemoptysis as on now. Patel et al[7] in their study on 2015 reported as bronchogenic carcinoma was the second most common cause accounting for 14% of their patients. Singh et al[5] in their study on 2016 showed

lung carcinoma as second commonest cause found in 7.2%. Both of the above studies they included patients with mild hemoptysis of <100 ml/ day. Around 84% of lung carcinoma were caused by mild hemoptysis in Singh et al[5] study. Most of the time patients with malignancy had mild hemoptysis which was true by the above studies. Lung carcinoma was a cause in 5.2% of patients in a study conducted by Prasad et al[15] on 2009. Recently Bhalla et al in their study on 2017 reported as lung carcinoma contributes 6.25% of hemoptysis.[4] In the present study result of 5.4% is closely similar to Bhalla et al[4] and Prasad et al.[15] But in developed countries 19% were caused by lung carcinoma reported by Hirshberg et al.[58] Here the inference is lung malignancy also an important cause of hemoptysis in India but not high like in developed countries.

Community acquired pneumonia was present in 3.2% of patients in the present study. Indian studies shows it ranging from 1.7% to 25.5%.[2,15,17] Pneumonia was reported in 10.7% of 64 patients in their study by Bhalla et al[4]. Patel et al reported 10% of hemoptysis was due to pneumonia on 2015.[7] It was one of the four main causes of hemoptysis in developed countries as evidence by Hirshberg et al[58] study which showed 16%. In the present study incidence was lower because mild hemoptysis was not included in our study who were the major population in Patel et al[7].

Most of the patients in the present study were managed with conservative treatment and less than 30% requires interventions like bronchial artery embolization and surgery to control ongoing bleeding and to prevent rebleeding.

Hemoptysis was successfully controlled by conservative management in 76.6% of patients in a study by Mishra et al[44]. In our study 66 (70.96%) patients were managed with conservative treatment but the success rate was 74.24% (49).

Definitive treatment of hemoptysis needs medical treatment or surgical excision of diseased portion of the lung.

Bronchial artery embolization is a interventional procedure that stops bleeding transiently but in many cases it prevents occurrence of re bleeding in long term also. 78% Successful outcome after BAE was seen in a study conducted by Ramakantan et al[59] in 140 patients with tuberculosis. Recently 94% immediate successful outcome by BAE was seen in 163 of 169 patients in a study by Shin et al[43]. On further follow up by Shin et al[43] study reported 29% recurrence. Mishra A et al[44] in their study on 2017 reported immediate success rate of BAE in 92% of patients. In our study we had done only short time follow up which showed 91.3% successful outcome without significant recurrent bleeding. Our study results were similar to study done by Shin et al[43] and Mishra et al[44].

Recurrent hemoptysis within 6 months was usually due to undetected arteries during embolization or partial embolization of feeding artery or neovascularization from adjacent vessels. Late recurrence after 6 months is usually due to collateral vessel formation or disease progression. If underlying disease was adequately cured many of them may not have recurrence.

The complication rate after BAE is in decreasing trend for the past few years. Highest complication rate of 29% by Shin et al[43] and 27% by Ramakantan et al was due to all minor complications were included by them. Most of the other studies showed less than 10% complications. Major complication such as monoparesis was seen in 1 (1.9%) patient due to inadvertent spinal artery embolization in a study done by Mishra A et al[44]. In our study also one patient out of 23 had right lower limb monoparesis (4.3%) and improved by medical management and physiotherapy. No one was died after the BAE till 3 months of follow up in our study.

4 patients in my study were died due to massive hemoptysis and asphyxiation. All the 4 had history of irregular anti-tuberculous chemotherapy in the past and finally cured before came to our hospital. Out of the 4 patients 3 had uncontrolled diabetes mellitus at the time of admission. Mortality rate of present study was 4.3% which was lower than reports from other studies conducted in India. Mortality rate was ranging from 8.2% to 18.8% in previous studies of hemoptysis at India.[15,17] Difference in the study group may had impact on differences in the mortality rate. Study by Prasad et al[15] had a mortality rate of 8.2% and more than 25% of patients in their study had moderate to massive hemoptysis. Lower mortality in this present study was may be due to interventional managements like bronchial artery embolization and surgery were decided by us sooner. This points out that aggressive management of life threatening hemoptysis could save many lives.

CONCLUSION

CONCLUSION

Hemoptysis is a non-specific but dangerous symptom of underlying disease that should be investigated for better treatment and outcome. Pulmonary tuberculosis is still remain a major cause of hemoptysis in India. Both active pulmonary tuberculosis and post tuberculous sequelae can cause hemoptysis. In our study pulmonary tuberculous sequelae is the most common cause of hemoptysis, that gives importance to the control of tuberculosis.

Hemoptysis may occur during anti-tuberculous treatment in patients with pulmonary tuberculosis. Conservative management along with anti-tuberculous treatment is sufficient in these patients. Change of anti-tuberculous treatment is needed only if the biological specimen shows drug resistant pattern.

Hemoptysis also develop in cured and treatment completed patients of pulmonary tuberculosis. Anti-tuberculous treatment should not be started without clinico-radiological or microbiological evidence.

Most common parenchymal lesion predispose to aspergilloma is cavity produced by tuberculosis in our country. Malignancy is one of the four important causes in our study. So mild to moderate hemoptysis of even one episode needs thorough investigation workup.

Blood sugar control in diabetics is more important for the control of active bleeding, to prevent rebleeding and to reduce mortality.

Early interventional management can reduce recurrence and mortality. Surgery is indicated for massive and recurrent hemoptysis. Surgery is effective in prevention of hemoptysis as our study shows no recurrent hemoptysis post operatively and during follow up period.

Bronchial artery embolization is an effective and safe intervention for the control of acute and massive hemoptysis. This procedure may be repeated in recurrent hemoptysis if they have contraindication for surgery.

Bronchial artery embolization or surgery can be used for definitive management of hemoptysis.

LIMITATIONS OF STUDY

LIMITATIONS OF THE STUDY

Study was conducted in a single centre with small number of patients that may not represent the community.

Study centre being the tertiary referral unit, majority of cases were referreal cases from nearby centre after failed control of hemoptysis that may not represent the whole affected population of hemoptysis.

Short term follow-up of 3 months was only done which may not be adequate to assess the treatment response. Prolonged follow up will establish the long term prognosis of each successful management.

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ANNEXURES

ABBREVIATIONS

TB	–	Tuberculosis
AFB	–	Acid Fast Bacilli
RNTCP	–	Revised National Tuberculosis Control Programme
CBNAAT	–	Catridge Based Nucleic Acid Amplification Test
HBsAg	–	Hepatitis B Surface Antigen
HCV	–	Hepatitis C Virus
ICTC	–	Integrated Counselling and Testing Centre
BAE	–	Bronchial Artery Embolization
COPD	–	Chronic Obstructive Pulmonary Disease
Nd-YAG	–	Neodymium-doped Yttrium Aluminum Garnet
INR	–	International Normalized Ratio
HRCT	–	High Resolution Computed Tomography
ECG	–	Electrocardiograph
ANCA	–	Anti-Neutrophil Cytoplasmic Antibodies
DAH	–	Diffuse Alveolar Hemorrhage
SLE	–	Systemic Lupus Erythematosus

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CERTIFICATE - II

This is to certify that this dissertation work titled "**CAUSES AND OUTCOME OF MODERATE TO MASSIVE HEMOPTYSIS AMONG ADMITTED PATIENTS FROM RAJIV GANDHI GOVERNMENT GENERAL HOSPITAL IN CHENNAI"** of the candidate **DR. UMAPATHI S** with registration Number **201827003** for the award of **M.D., DEGREE** in the branch of **RESPIRATORY MEDICINE.** I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **1 percentage** of plagiarism in the dissertation.

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CERTIFICATE OF APPROVAL

To

DR. UMAPATHI S
POST GRADUATE IN TUBERCULOSIS AND CHEST DISEASES
MADRAS MEDICAL COLLEGE & RAJIV GANDHI GOVT. GENERAL HOSPITAL
CHENNAI - 600003

Dear DR. UMAPATHI S,

The Institutional Ethics Committee has considered your request and approved your study titled **"CAUSES AND OUTCOME OF MODERATE TO MASSIVE HEMOPTYSIS AMONG ADMITTED PATIENTS FROM RAJIV GANDHI GOVERNMENT GENERAL HOSPITAL IN CHENNAI "- NO.31112018**

The following members of Ethics Committee were present in the meeting held on **13.11.2018** conducted at Madras Medical College, Chennai 3

1. Prof.P.V.Jayashankar :Chairperson
2. Prof.R.Jayanthi,MD.,FRCP(Glasg)., Dean,MMC,Ch-3 : Deputy Chairperson
3. Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3 : Member Secretary
4. Prof.N.Gopalakrishnan,MD,Director,Inst.of Nephrology,MMC,Ch : Member
5. Prof.S.Tito,MD,Prof. Inst. of Int.Med,MMC, Ch-3 : Member
6. Prof.Afee Asma,Director, Inst. of Gen.Surgery,MMC : Member
7. Prof.Shobha, Prof, Inst.of O&G, Chennai : Member
8. Prof.Rema Chandramohan,Prof.of Paediatrics,ICH,Chennai : Member
9. Prof. Sudha, Prof. Inst. of Pharmacology,MMC,Ch-3 : Member
10. Prof.K.Ramadevi,MD., Director, Inst. of Bio-Chemistry,MMC,Ch-3 : Member
11. Prof.Bharathi Vidya Jayanthi,Director, Inst. of Pathology,MMC,Ch-3: Member
12. Thiru S.Govindasamy, BA.,BL,High Court,Chennai : Lawyer
13. Tmt.Arnold Saulina, MA.,MSW., :Social Scientist
14. Thiru K.Ranjith, Ch- 91 : Lay Person

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary - Ethics Committee

INFORMATION SHEET

We are conducting a study on **"CAUSES AND OUTCOME OF MODERATE TO MASSIVE HEMOPTYSIS AMONG ADMITTED PATIENTS FROM RAJIV GANDHI GOVERNMENT GENERAL HOSPITAL IN CHENNAI"** among patients attending Rajiv Gandhi Government General Hospital, Chennai. The purpose of this study is to find the cause and outcome of patients with active hemoptysis admitted in Rajiv Gandhi Government General Hospital. Those patients who admitted in the ward and come under the inclusion criteria will be included in the study and will undergo certain investigations required to diagnose the disease.We are selecting certain cases and if you are found eligible, we would like to perform extra tests and special studies which in any way do not affect your final report or management.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator

Signature/left thumb impression of Participant

Date :

Place :

PATIENT CONSENT FORM

Study Detail : **CAUSES AND OUTCOME OF MODERATE TO MASSIVE HEMOPTYSIS AMONG ADMITTED PATIENTS FROM RAJIV GANDHI GOVERNMENT GENERAL HOSPITAL IN CHENNAI**

Study Centre : Department of Thoracic Medicine, Rajiv Gandhi Government General Hospital, Chennai.

Patient's Name :

Patient's Age :

Identification Number :

Patient may check (☑) these boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ❑

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ❑

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ❑

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms. ❑

I hereby consent to participate in this study. ❑

I hereby give permission to undergo complete clinical examination ,and necessary investigations. ❑

Signature of Investigator Signature/thumb impression

Study Investigator's Name: Patient's Name and Address:

Dr. UMAPATHI S

ஆராய்ச்சி தகவல் தாள்

ஆராய்ச்சி தலைப்பு

இருமும்போது சளியில் இரத்தம் வரும் பிரச்சனை உள்ளவர்களுக்கு , காரணம் மற்றும் விளைவுகள் கண்டறியும் ஆராய்ச்சி

ஆய்வாளர் பெயர் : மரு. சி. உமாபதி

ஆய்வு நிலையம் : நெஞ்சக நோய் மருத்துவ துறை,
சென்னை மருத்துவக் கல்லூரி, சென்னை.

இந்த ஆய்வில் தங்களை பங்கேற்க அழைக்கிறோம். இந்த தகவல் அறிக்கையில் கூறப்பட்டிருக்கும் தகவல்கள் தாங்கள் இந்த ஆராய்ச்சியில் பங்கேற்கலாமா வேண்டாமா என்பதை முடிவு செய்ய உதவியாக இருக்கும். இந்த படிவத்தில் உள்ள தகவல்கள் பற்றி உள்ள சந்தேகங்களை நீங்கள் தயங்காமல் கேட்கலாம்.

இந்த ஆராய்ச்சியில் இருமும்பொது சளியில் இரத்தம் வரும் பிரச்சனை உள்ளவர்களுக்கு, காரணம் கண்டறிய இரத்தம், சளி பரிசோதனை, ஸ்கேன் (CXR, CT CHEST) மற்றும் மூச்சுக் குழல் ஆய்வு (Bronchoscopy) செய்யப்பட்டு தேவைப்படின் திசு பரிசோதனை செய்யப்படும் . அதற்கு தங்கள் ஒத்துழைப்பு தேவை.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். அதனால் தங்களது நோயின் ஆய்வறிக்கைக்கோ அல்லது சிகிச்சைக்கோ பாதிப்புக்கு ஏற்படாது என்பதையும் தெரிவித்து கொள்கிறோம்.

முடிவுகளை அல்லது கருத்துக்களை வெளியிடும் போதோ அல்லது ஆய்வின் போதோ தங்களது பெயரையோ அல்லது அடையாளத்தையோ வெளியிடமாட்டோம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

இந்த சிறப்பு பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின் போது அல்லது ஆராய்ச்சியின் முடிவின் போது தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்து கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம்: பங்கேற்பாளர் கையொப்பம்:

ஆராய்ச்சி ஒப்புதல் படிவம்

ஆராய்ச்சியின் தலைப்பு

இருமும்போது சளியில் இரத்தம் வரும் பிரச்சனை உள்ளவர்களுக்கு , காரணம் மற்றும் விளைவுகள் கண்டறியும் ஆராய்ச்சி.

ஆய்வு நிலையம் : நெஞ்சக நோய் மருத்துவ துறை,
சென்னை மருத்துவக் கல்லூரி, சென்னை.

பங்கு பெறுவரின் பெயர் :
பங்கு பெறுவரின் எண் :

பங்குபெறுபவர் இதனை (√) குறிக்கவும்

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது.	
நான் இவ்வாய்வில் தன்னிச்சையாகத்தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கு உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.	
இந்த ஆய்வு சம்மந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.	
இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக்கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கின்றேன்.	
இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிகிறேன்.	

பங்கேற்பவரின் கையொப்பம்.....
கட்டைவிரல் ரேகை

இடம்: சென்னை.
நாள்.....

PROFORMA

S.No:

NAME OF THE PATIENT : D.O.Admn.:

AGE / SEX : D.O.Intven.:

OP/IP NUMBER : D.O.Discg.:

ADDRESS : Dura.O.Stay:

CONTACT NUMBER :

COMPLAINTS : Amount and duration of hemoptysis

PAST HISTORY : H/O ATT, COMORBIDITY

SMOKING HISTORY :

TREATMENT HISTORY : ANTIPLATELETS, ANTICOAGULANTS

GENERAL EXAMINATION: Lymphadenopathy

VITALS : Pulse Rate: BP: Temperature:

BASIC BLOOD INVESTIGATIONS

RBC, TC, HB, PLATELETS, PCV:

RBS: Urea: Creatinine:

Total Protein: Albumin:

PROTHROMBIN TIME: INR: BLOOD GROUPING AND Rh:

CXR:

CT-CHEST: (PLAIN / CONTRAST / HRCT)

SPUTUM / BRONCHIAL WASH EXAMINATION:

LED FM :

CBNAAT: MTB detected / Not detected RIF: Sensitive / Resistant

LPA: H : Sensitive / Resistant R : Sensitive / Resistant

MGIT FOR AFB

NT C/S

FUNGAL KOH

FUNGAL C/S

BRONCHOSCOPY FINDINGS AND REPORTS:

HPE of LUNG TISSUE:

OTHER INVESTIGATIONS:

TREATMENT:

INTERVENTIONS DONE: BAE / SURGERY

COMPLICATIONS:

FOLLOW UP CHECK UP: complaints, recurrent bleed, complications, management

FINAL OUTCOME:

MASTER CHART

S.NO	AGE	SEX	DU. HEM	CLASS	PAST TB	CO-MOR	SMOKER	DU. STAY	PR	BP	HB	RBC	PLATELET	TC	TP	ALB	AREA	CT FINDIG	CAUSE	2 INFEC	MANAGE	OUTCOME	RECURR	COMPLI
1	48	M	2	MODERA	YES	NO	FORMER	8	78	110	10	4.1	3.78	12.9	8	3.1	MULTI	CONSOL	PTB	NO	CONSER	ALIVE	NO	NO
2	85	M	4	MODERA	YES	DM,COPD	FORMER	16	108	140	7	2.9	2.13	7.6	6	3.5	MULTI	BRONCHI	PT SEQ	KLEBS	CONSER	ALIVE	NO	NO
3	47	M	3	MODERA	YES	COPD	CURRENT	6	70	130	9.2	3.1	1.64	8.9	7	3.5	MULTI	CONSOL	PTB	NO	CONSER	ALIVE	NO	NO
4	48	M	2	MASSIVE	YES	DM	FORMER	1	98	110	9.6	4.1	3.74	13.7	6	3	MULTI	BRONCHI	BRONCHI	NO	CONSER	DIED	RECURR	NO
5	50	M	2	MASSIVE	YES	COPD	FORMER	8	110	100	11	4.4	2.6	16.5	8	4	LEFT UL	CONSOL	PTB	NO	CONSER	DIED	RECURR	NO
6	41	M	1	SEVERE	YES	CAD	NEVER	16	130	90	15	5.1	3.07	9.3	8	4.1	MULTI	FIBCAVIT	PT SEQ	KLEBS	BAE	ALIVE	NO	MONOPA
7	50	M	3	MODERA	NO	DM	NEVER	11	110	110	12	3.9	2.56	6.4	7	3.3	RIGHT UL	CONSOL	PTB	NO	CONSER	ALIVE	RECURR	NO
8	51	M	2	MODERA	YES	DM	NEVER	23	125	90	12	4.2	2.85	17.2	7	4.1	RIGHT UL	FIBCAVIT	PT SEQ	PSEUD	BAE	ALIVE	NO	NO
9	55	M	2	MASSIVE	YES	DM	NEVER	4	72	90	11	4	3.06	21.2	8	3.5	MULTI	FIBCAVIT	PT SEQ	NO	CONSER	DIED	RECURR	NO
10	39	M	2	MODERA	YES	NO	NEVER	14	68	120	12	5.1	2.56	12.1	8	3.7	RIGHT UL	FIBCAVIT	PT SEQ	KLEBS	CONSER	ALIVE	NO	NO
11	50	M	3	SEVERE	YES	NO	NEVER	10	82	130	14	5.6	2.39	13.6	8	4.4	MULTI	CONSOL	PTB	NO	BAE	ALIVE	NO	NO
12	40	M	1	SEVERE	YES	DM	FORMER	12	78	110	15	5.2	2.26	8	7	4.5	RIGHT UL	BRONCHI	BRONCHI	PSEUD	BAE	ALIVE	NO	NO
13	68	M	2	MODERA	YES	DM,CAD	FORMER	7	78	140	13	4.5	2.61	7.53	7	3.5	LEFT UL	MASS	CA	NO	CONSER	ALIVE	NO	NO
14	40	M	3	SEVERE	YES	NO	CURRENT	7	98	90	12	4.2	2.32	6.4	7	2.7	MULTI	BRONCHI	PT SEQ	KLEBS	CONSER	ALIVE	NO	NO
15	27	M	1	MODERA	YES	NO	NEVER	15	102	110	11	3.9	2.19	8.5	7	2.9	RIGHT UL	BRONCHI	BRONCHI	NO	BAE	ALIVE	NO	NO
16	52	M	2	MODERA	NO	COPD	CURRENT	10	108	140	12	4.6	2.7	15.6	7	4.3	LEFT UL	CONSOL	PNEUMO	NO	CONSER	ALIVE	NO	NO
17	45	M	2	MODERA	YES	COPD	CURRENT	15	92	120	12	3.3	2.08	6.7	6	3.1	RIGHT UL	FIBCAVIT	PT SEQ	PSEUD	BAE	ALIVE	NO	NO
18	61	M	3	MODERA	YES	COPD	FORMER	7	86	130	9.2	3	2.96	9.1	7	3.2	RIGHT UL	MASS	CA	NO	CONSER	ALIVE	NO	NO
19	33	M	3	MODERA	YES	NO	NEVER	12	72	110	12	4.6	2.48	7.8	6	3.4	MULTI	BRONCHI	PT SEQ	PSEUD	BAE	ALIVE	NO	NO
20	55	M	2	MODERA	YES	COPD	CURRENT	8	78	130	8.8	2.9	1.75	13.2	7	4.3	MULTI	FIBCAVIT	PT SEQ	NO	BAE	ALIVE	NO	NO
21	44	M	1	SEVERE	YES	NO	FORMER	17	120	100	7.2	2.6	2.15	10.4	8	4	LEFT UL	ASPERGIL	ASPERGIL	NO	LOBECTO	ALIVE	NO	NO
22	38	M	2	MODERA	YES	NO	NEVER	10	82	120	11	3.1	2.27	16.3	8	3.4	MULTI	BRONCHI	BRONCHI	PSEUD	CONSER	ALIVE	NO	NO
23	62	M	3	MODERA	YES	COPD	CURRENT	7	110	130	9.8	3.1	2.56	15.4	8	3.6	MULTI	BRONCHI	PT SEQ	KLEBS	CONSER	ALIVE	RECURR	NO
24	55	M	3	MODERA	YES	COPD	CURRENT	15	85	120	12	4.1	2.43	5.75	8	3.5	LEFT UL	FIBCAVIT	PT SEQ	PSEUD	BAE	ALIVE	NO	CHST PAIN
25	48	M	2	MODERA	YES	COPD	FORMER	11	92	130	12	4.5	2.73	8.8	8	3.4	MULTI	BRONCHI	BRONCHI	KLEBS	CONSER	ALIVE	NO	NO
26	42	M	2	MODERA	YES	DM	CURRENT	9	98	110	12	3.9	2.19	11.8	7	4	MULTI	BRONCHI	PT SEQ	NO	CONSER	ALIVE	RECURR	NO

S.NO	AGE	SEX	DU. HEM	CLASS	PAST TB	CO-MOR	SMOKER	DU. STAY	PR	BP	HB	RBC	PLATELET	TC	TP	ALB	AREA	CT FINDIG	CAUSE	2 INFEC	MANAGE	OUTCOME	RECURR	COMPLI
27	44	M	4	MODERA	YES	COPD	CURRENT	10	98	120	10	3.6	2.48	13.5	8	4.2	RIGHT UL	FIBCAVIT	PT SEQ	NO	BAE	ALIVE	NO	NO
28	39	M	1	SEVERE	YES	DM	NEVER	14	116	90	7.8	3.2	2.06	13.9	6	3.7	MULTI	BRONCHI	BRONCHI	PSEUD	CONSER	ALIVE	NO	NO
29	56	M	1	MODERA	NO	COPD	CURRENT	8	96	130	12	4.3	2.82	18.4	8	4.6	LEFT LL	MASS	CA	NO	CONSER	ALIVE	NO	NO
30	50	M	1	MODERA	YES	DM	NEVER	9	78	126	11	4.9	2.81	9.43	7	4.3	MULTI	FIBCAVIT	PT SEQ	NO	CONSER	ALIVE	RECURR	NO
31	62	M	2	MODERA	YES	COPD	CURRENT	10	90	138	15	5.1	3.2	6.5	7	4	LEFT UL	FIBCAVIT	PT SEQ	KLEBS	CONSER	ALIVE	NO	NO
32	45	M	1	MODERA	YES	NO	FORMER	9	70	126	14	4.2	2.51	16.3	8	4.2	RIGHT LL	CONSOL	PTB	NO	CONSER	ALIVE	NO	NO
33	42	M	1	MODERA	NO	NO	CURRENT	14	98	130	14	4.3	3.18	13.9	8	4.4	LEFT UL	CONSOL	PTB	NO	CONSER	ALIVE	NO	NO
34	36	M	1	MODERA	YES	NO	CURRENT	12	84	120	12	4	2.5	9.31	8	3.6	MULTI	FIBCAVIT	PT SEQ	NO	CONSER	ALIVE	NO	NO
35	60	M	2	MODERA	YES	COPD	CURRENT	10	76	130	12	3.8	2.64	10.7	7	2.8	RIGHT UL	MASS	CA	NO	CONSER	ALIVE	NO	NO
36	55	M	2	MODERA	YES	COPD	CURRENT	14	80	140	15	4.9	3.07	7.18	7	3	LEFT UL	FIBCAVIT	PT SEQ	KLEBS	BAE	ALIVE	NO	NO
37	42	M	3	MODERA	NO	NO	FORMER	12	102	120	12	4.1	3.03	14.9	8	4.1	MULTI	CONSOL	PNEUMO	NO	CONSER	ALIVE	NO	NO
38	45	M	2	MODERA	YES	COPD	CURRENT	14	106	110	14	4.3	2.83	7.1	8	3.7	LEFT UL	FIBCAVIT	PT SEQ	KLEBS	BAE	ALIVE	NO	CHST PAIN
39	44	M	2	MODERA	YES	NO	NEVER	11	92	130	12	4.2	3.11	7.5	7	3.3	MULTI	FIBCAVIT	PT SEQ	ACINETE	CONSER	ALIVE	NO	NO
40	38	M	1	MODERA	YES	NO	CURRENT	12	96	120	16	4.9	3.8	15.4	6	2.9	RIGHT UL	CONSOL	PTB	NO	CONSER	ALIVE	NO	NO
41	40	M	1	MODERA	YES	NO	CURRENT	10	100	120	14	4.1	2.63	14	6	3.1	MULTI	CONSOL	PTB	NO	CONSER	ALIVE	NO	NO
42	30	M	2	MODERA	NO	NO	CURRENT	12	112	120	13	4	2.96	15.6	8	3.9	LEFT UL	CONSOL	PNEUMO	NO	CONSER	ALIVE	NO	NO
43	45	M	2	MODERA	YES	NO	FORMER	11	106	130	14	4.5	3.22	13.8	6	3.5	MULTI	CONSOL	PTB	NO	CONSER	ALIVE	NO	NO
44	48	M	1	SEVERE	YES	COPD	NEVER	16	115	120	12	4	2.9	8.6	7	3.4	RIGHT UL	ASPERGIL	ASPERGIL	NO	CONSER	ALIVE	NO	NO
45	55	M	2	MODERA	YES	COPD	CURRENT	10	92	140	15	4.1	3.09	9.3	7	2.8	MULTI	BRONCHI	PT SEQ	PSEUD	CONSER	ALIVE	NO	NO
46	42	M	2	MODERA	YES	NO	NEVER	16	108	120	12	3.7	2.54	16.5	8	3.2	MULTI	FIBCAVIT	PT SEQ	NO	BAE	ALIVE	NO	NO
47	47	M	3	MODERA	YES	NO	CURRENT	17	94	110	14	4.2	2.77	8.9	7	3.3	RIGHT LL	FIBCAVIT	PT SEQ	PSEUD	BAE	ALIVE	RECURR	NO
48	34	M	1	MODERA	YES	NO	CURRENT	10	92	120	14	4.4	2.81	13.6	7	2.9	MULTI	CONSOL	PTB	NO	CONSER	ALIVE	NO	NO
49	54	M	2	MODERA	YES	COPD	CURRENT	9	102	130	15	4.1	3.63	6.5	8	4	MULTI	FIBCAVIT	PT SEQ	PSEUD	CONSER	ALIVE	NO	NO
50	36	M	1	MODERA	YES	NO	CURRENT	9	98	110	13	4.1	2.88	12.2	8	3.5	RIGHT UL	CONSOL	PTB	NO	CONSER	ALIVE	NO	NO
51	45	M	2	MODERA	YES	NO	FORMER	16	116	110	13	4.6	2.75	7.7	8	3.5	LEFT LL	BRONCHI	BRONCHI	PSEUD	CONSER	ALIVE	NO	NO
52	43	M	2	MODERA	YES	DM,COPD	CURRENT	12	102	120	13	3.9	2.54	14.5	8	3.1	MULTI	CONSOL	PTB	NO	CONSER	ALIVE	NO	NO
53	47	M	8	MODERA	YES	COPD	CURRENT	5	108	110	13	4	5.5	11.8	8	3.2	RIGHT UL	BRONCHI	PTB	NO	CONSER	ALIVE	NO	NO

S.NO	AGE	SEX	DU. HEM	CLASS	PAST TB	CO-MOR	SMOKER	DU. STAY	PR	BP	HB	RBC	PLATELET	TC	TP	ALB	AREA	CT FINDIG	CAUSE	2 INFEC	MANAGE	OUTCOME	RECURR	COMPLI
54	22	M	1	MODERA	NO	NO	NEVER	6	88	100	9.8	3.3	1.88	12.4	7	3.6	MULTI	CONSOL	PTB	NO	CONSER	ALIVE	NO	NO
55	53	M	3	MODERA	YES	COPD	FORMER	7	90	110	12	4.2	3.62	9	6	2.8	RIGHT UL	BRONCHI	PT SEQ	NO	CONSER	ALIVE	NO	NO
56	39	M	2	MASSIVE	YES	DM	CURRENT	9	86	90	12	4.6	3.52	6.3	7	3.6	MULTI	BRONCHI	PT SEQ	PSEUD	BAE	ALIVE	NO	NO
57	58	M	2	SEVERE	YES	COPD	CURRENT	9	110	100	7.4	3	2.15	7.8	7	3.6	MULTI	FIBCAVIT	PT SEQ	KLEBS	BAE	ALIVE	NO	NO
58	31	M	3	MODERA	NO	NO	NEVER	5	102	100	14	4.1	3.16	13.2	8	4	LEFT UL	CONSOL	PTB	NO	CONSER	ALIVE	NO	NO
59	46	M	2	MODERA	NO	DM	FORMER	9	94	130	13	3.8	2.44	7.3	7	3.4	RIGHT ML	CONSOL	PTB	NO	CONSER	ALIVE	RECURR	NO
60	42	M	3	MODERA	NO	DM	CURRENT	15	98	120	12	4.3	3.9	12.3	7	4.3	RIGHT LL	CONSOL	LU ABS	PSEUD	CONSER	ALIVE	NO	NO
61	33	M	3	MODERA	YES	NO	CURRENT	8	102	90	11	4.1	4.65	7.9	7	3	LEFT UL	CONSOL	PT SEQ	KLEBS	CONSER	ALIVE	NO	NO
62	38	M	2	SEVERE	YES	NO	CURRENT	7	112	90	11	5	2.35	11.4	7	3.3	RIGHT UL	FIBCAVIT	PTB	NO	CONSER	ALIVE	RECURR	NO
63	67	M	3	SEVERE	YES	COPD	NEVER	10	118	90	12	4.4	2.82	9.2	7	3.6	RIGHT UL	BRONCHI	PT SEQ	PSEUD	BAE	ALIVE	NO	NO
64	52	M	1	MASSIVE	YES	NO	FORMER	15	110	100	11	3.9	5.22	5.9	8	3	LEFT UL	ASPERGIL	ASPERGIL	NO	CONSER	ALIVE	RECURR	NO
65	55	M	3	MODERA	YES	COPD	FORMER	8	90	110	14	5.4	4.12	9.6	7	3.8	MULTI	BRONCHI	PT SEQ	PSEUD	CONSER	ALIVE	NO	NO
66	29	M	5	MODERA	NO	NO	CURRENT	6	80	100	13	4.3	2.46	8.3	7	2.6	RIGHT UL	CONSOL	PTB	NO	CONSER	ALIVE	NO	NO
67	55	M	2	SEVERE	YES	NO	NEVER	8	104	110	11	3.3	1.85	14.2	7	3.3	RIGHT UL	FIBCAVIT	PT SEQ	KLEBS	BAE	ALIVE	NO	NO
68	37	M	2	MASSIVE	NO	DM	CURRENT	10	100	90	9.9	4.1	3.84	11.7	7	3.4	MULTI	CONSOL	PTB	NO	CONSER	ALIVE	RECURR	NO
69	37	M	4	MODERA	YES	NO	NEVER	10	93	100	15	5.7	2.01	8.2	8	4.5	MULTI	FIBCAVIT	PT SEQ	PSEUD	BAE	ALIVE	NO	CHST PAIN
70	45	M	5	MODERA	YES	CLD,COPD	CURRENT	15	88	110	15	5	2.94	8.9	7	3.1	MULTI	FIBCAVIT	PTB	NO	CONSER	ALIVE	RECURR	NO
71	55	M	3	SEVERE	YES	COPD	FORMER	7	100	100	13	4.6	1.67	8.01	7	3.6	MULTI	BRONCHI	PT SEQ	PSEUD	CONSER	ALIVE	RECURR	NO
72	28	F	2	MODERA	YES	NO	NEVER	20	82	110	8.5	2.9	3.19	8	6	3.4	LEFT UL	ASPERGIL	ASPERGIL	NO	LOBECTO	ALIVE	NO	NO
73	35	F	1	MODERA	NO	NO	NEVER	12	96	120	12	4.4	2.07	7.1	7	4.2	LEFT UL	CONSOL	PTB	NO	CONSER	ALIVE	NO	NO
74	59	F	2	MODERA	YES	COPD	NEVER	18	86	130	12	3.3	2.1	7.9	8	4.1	MULTI	FIBCAVIT	PT SEQ	NO	BAE	ALIVE	NO	NO
75	42	F	1	MODERA	YES	NO	NEVER	14	100	120	12	3	2.32	8.3	8	4.5	MULTI	FIBCAVIT	PT SEQ	PSEUD	BAE	ALIVE	NO	NO
76	50	F	2	MODERA	YES	COPD	NEVER	10	78	140	13	3.4	2.54	12	7	3.6	RIGHT UL	CONSOL	PTB	NO	CONSER	ALIVE	NO	NO
77	37	F	1	MODERA	YES	NO	NEVER	15	70	110	13	2.9	3.08	9	7	4	LEFT LL	BRONCHI	BRONCHI	KLEBS	CONSER	ALIVE	NO	NO
78	29	F	1	MODERA	NO	NO	NEVER	14	112	120	12	2.5	2.16	13.3	7	4.3	RIGHT UL	CONSOL	PTB	NO	CONSER	ALIVE	RECURR	NO
79	50	F	2	MODERA	YES	COPD	FORMER	12	102	140	13	3.3	3.01	9.8	7	4.4	MULTI	FIBCAVIT	PT SEQ	PSEUD	CONSER	ALIVE	NO	NO
80	35	F	2	MODERA	YES	NO	NEVER	8	98	120	13	3.6	2.4	6.3	8	4	RIGHT ML	CONSOL	LU ABS	NO	CONSER	ALIVE	NO	NO

S.NO	AGE	SEX	DU. HEM	CLASS	PAST TB	CO-MOR	SMOKER	DU. STAY	PR	BP	HB	RBC	PLATELET	TC	TP	ALB	AREA	CT FINDIG	CAUSE	2 INFEC	MANAGE	OUTCOME	RECURR	COMPLI
81	42	F	1	MODERA	YES	NO	NEVER	8	86	110	12	3.2	2.63	11.9	7	3	MULTI	CONSOL	PTB	NO	CONSER	ALIVE	NO	NO
82	48	F	3	MASSIVE	YES	DM	NEVER	10	92	100	9.6	2.8	2.21	12.5	6	4	LEFT UL	CONSOL	PTB	NO	CONSER	DIED	RECURR	NO
83	55	F	2	MODERA	YES	COPD	NEVER	7	84	130	11	3.1	2.39	10.2	7	3.2	RIGHT UL	ASPERGIL	ASPERGIL	NO	LOBECTO	ALIVE	NO	NO
84	44	F	2	MODERA	YES	DM	NEVER	12	110	130	8.2	2.6	2.01	8.9	7	4.5	RIGHT LL	CONSOL	PTB	NO	CONSER	ALIVE	RECURR	NO
85	36	F	2	MODERA	YES	NO	NEVER	9	70	100	12	3.5	2.18	7.9	8	4.6	MULTI	CONSOL	VASCULIT	NO	CONSER	ALIVE	NO	NO
86	54	F	1	SEVERE	YES	DM	NEVER	15	78	100	7.4	2.6	2.44	15.1	7	3.1	MULTI	BRONCHI	PT SEQ	NO	BAE	ALIVE	RECURR	CHST PAIN
87	38	F	1	MODERA	YES	NO	NEVER	22	98	120	8.5	3.1	2.11	10.4	8	4.5	LEFT UL	ASPERGIL	ASPERGIL	NO	LOBECTO	ALIVE	NO	NO
88	72	F	3	MODERA	YES	COPD	NEVER	7	102	130	11	4.3	2.06	7	7	4.3	MULTI	CONSOL	PT SEQ	KLEBS	CONSER	ALIVE	RECURR	NO
89	45	F	2	MODERA	NO	NO	NEVER	5	98	120	11	3.9	2.14	8.2	7	4.2	RIGHT UL	CONSOL	PTB	NO	CONSER	ALIVE	NO	NO
90	36	F	1	SEVERE	NO	NO	NEVER	7	104	100	9	3.1	3.23	9.1	6	3.2	LEFT UL	CONSOL	PTB	NO	CONSER	ALIVE	NO	NO
91	54	F	2	MODERA	NO	NO	NEVER	9	82	120	12	4.3	2.12	6.5	7	4	RIGHT UL	MASS	CA	NO	CONSER	ALIVE	NO	NO
92	25	F	2	MODERA	NO	NO	NEVER	6	94	110	10	3.6	1.98	8.8	6	3.1	RIGHT UL	CONSOL	PTB	NO	CONSER	ALIVE	NO	NO
93	48	F	1	MASSIVE	YES	NO	NEVER	8	106	90	9.9	3.3	2.97	11	7	3.6	LEFT UL	BRONCHI	PT SEQ	PSEUD	BAE	ALIVE	NO	CHST PAIN