

CASE REPORT

ENDOBRONCHIAL TUBERCULOSIS MIMICKING PULMONARY EMBOLISM

*Dr Nazim Hussain Bokhari
MCPS (Med), MCPS (Chest), MD (Chest), FCCP

**Dr Talha Mehmood
DTCD, MCPS (Chest), FCPS

*Associate Professor Pulmonology
**Senior Registrar Pulmonology

Department of Pulmonology
Shaikh Zayed Hospital Lahore

SUMMARY:

A 35 years old lady had multiple hospital admissions on account of persistent fever, shortness of breath, dry cough and weight loss of two months duration. Initially she was admitted in a general hospital because of fever with chills and progressive shortness of breath. On account of non responsive dyspnoea and persistent wheeze she was referred to the Cardiac Institute on suspicion of pulmonary embolism. In the cardiology unit pulmonary embolism was ruled out. Because of persistent hypoxia she was suspected to have developed respiratory distress syndrome and was referred to our unit. In the Pulmonology department pulmonary embolism and diffuse parenchymal lung disease were excluded. Persistent fever and wheeze lead to the search for isolation of AFB. As sputum smear came to be positive for acid fast bacilli she was diagnosed to have endobronchial tuberculosis. She responded well to antituberculous chemotherapy along with inhaled bronchodilators and steroids

CASE HISTORY:

A 35 year old housewife resident of Lahore presented to various family physicians with complaints of off and on fever with chills, hacking cough and shortness of breath of two weeks duration. There has been no relief in her symptoms inspite of repeated courses of antibiotics and expectorants. Because of rapidly worsening shortness of breath and considerable weight loss she was referred to a local teaching hospital.

In the hospital she was found to have tachypnoea, cyanosis and exhaustion with an ECG showing sinus tachycardia; CXR showing bilateral mid zone haziness with sparing of apical and basal areas. ABG's showed hypoxemia but normal PCO₂. On account of her clinical presentation diagnosis of Pulmonary Embolism was suspected and she was referred to the Cardiac Institute.

She was reviewed and evaluated in the A/E department of cardiac institute. Her investigation included, echocardiography which showed normal heart chambers but presence of pulmonary hypertension. ABG analysis was again consistent with type-I respiratory failure (pH 7.412 PO₂ 55 mmHg, PCO₂ 30 mmHg, HCO₃ 20 meq/L, SaO₂ 78%). Diagnosis of pulmonary embolism was ruled out. She was assessed to have respiratory tract infection complicated by diffuse lung injury (ARDS) and was referred to the Pulmonology department of Shaikh Zayed Hospital .

In the A&E department of the hospital she was found to have dry hacking cough, wheeze & respiratory distress with cyanosis. Her vital signs included a pulse rate of 110/min temp: 101 F, B.P: 100/60 mmHg and respiratory rate of 46/min

Respiratory system examination revealed use of accessory muscles, nasal flaring & intercostal in drawing. Breath sounds were vesicular but expiration was prolonged. High pitched rhonchi and inspiratory crackles were audible all over the chest.

No other significant abnormality was detected on the examination of other systems. Repeat investigations included, ABG's consistent with type- 1 respiratory failure, ECG: sinus tachycardia, chest x-ray, bilateral mid & lower zone mottling (Fig:1). The resident on call made a differential diagnosis of, bronchopneumonia, recurrent pulmonary embolism, acute severe asthma, alveolar cell carcinoma and military tuberculosis. On the provisional diagnosis of bronchopneumonia leading to respiratory failure, she was empirically put on antibiotics and transferred to the Pulmonology unit. She was prescribed high flow oxygen (8-10 L/min) through rebreathing mask, nebulized salbutamol & ipratropium bromide 6 hourly, and i/v levofloxacin and ceftriaxone. After three days of hospitalization she showed minimal clinical improvement, was still febrile upto 100F and dyspnoeic inspite of maintaining acceptable SO₂. She complained of pain & tenderness in both legs though the peripheral pulses were normal. Plasma D-Dimer levels were checked and found to be high (>2000) and remained on Heparin infusion for two days. In the meantime Doppler USG of legs was found normal. She also underwent 99mTc-MAA lung perfusion & 99mTc-DTPA aerosol lung ventilation

scintigraphy that showed hypo perfusion of right mid & lower zones and multiple small sub segmental defects in left lung, enlarged cardiac silhouette and ventilation defects in both lungs. This was reported as low probability for pulmonary emboli. Anticoagulation was stopped.

Other investigations done include pulmonary function tests which was reported as restrictive dysfunction due to poor respiratory effort. Echocardiography showed normal valves, enlarged RV, with loss of "a-dip" on pulmonary valve due to pulmonary hypertension. Ultrasonography of abdomen was normal. Serological markers for viral hepatitis were negative, RA Factor & ANA were also negative. CBC revealed Hb: 9.3g/dl, TLC: 6.8 3/dL, platelets: 350,000/cmm, MCV 77.4 fl MCHC 31.6 pg/cell. Peripheral Smear showed microcytosis, hypochromia, target cells & a few macrophages. Serum biochemistry included, BUN 20mg/dl, serum creatinine 0.8mg/dl, Alb 3.3Gm/dL, ALP 226u/l, ALT 28u/l, AST 13u/l, Ca 9.2mg/dl, T.Bil 0.4mg/dl, T.Prot 6.6g/dl, Urine examination did not show any abnormality while stool for occult blood was negative. In the second week of hospitalization she was continued with Levofloxacin 500mg I.Vx B.D & Ceftriaxone 1G I.Vx B.D, aerosolized bronchodilators, with O₂ inhalation at 3-4 L/min, saturation being maintained at 90%.

There was moderate degree of clinical improvement but low-grade temperature spikes up to 99 F and bronchospasm persisted. There was also change of dry cough to productive type with 20-30mL of yellowish sputum per day. She also noticed nocturnal worsening of cough along with wheez. Her Bronchoscopy was planned for BAL & bronchial biopsy but was delayed due to persistent dyspnoea, and patient's unwillingness. Her Sputum was sent for C/S, ZN & Gram Staining which revealed acid fast bacilli in two of the samples.

She was diagnosed as having endobronchial tuberculosis, leading to bronchial hyper reactivity and persistent airway obstruction. Anti-tuberculous chemotherapy was initiated with FDC tab of (RHEZ) and pyridoxine. She was maintained on inhaled salbutamol, and steroids via MDI with spacer device. After 10 days of in hospital stay she had SO₂ of >90% maintained on room air. She underwent bronchoscopy which revealed generalized mucosal inflammation, hyperemia and fine ulceration (Fig:3, 4) but no hypertrophic changes. These were considered to be of good prognosis with little subsequent complications.

She was discharged on request after 2 weeks of hospitalization and was counseled regarding infectiousness of tuberculosis, importance of chemotherapy and regular follow up.

She has successfully completed anti TB course. She didn't agree for follow up bronchoscopies, however clinically there were no sign of airway obstruction and she did not require bronchodilators after four months of chemotherapy.

DISCUSSION:

Infection of the tracheobronchial tree with microbial & histopathological evidence is defined as endobronchial tuberculosis EBTB [1]. It is found in 10-40% of active pulmonary tuberculosis. When it occurs in isolation it is a diagnostic challenge for physicians as it is a highly infectious illness on one hand and may have no obvious abnormality on chest radiographs. As a sequelae some degree of bronchial stenosis is seen in more than 90% of the cases [2].

Endobronchial involvement can occur through five possible mechanisms which are; erosion of lymph node into the bronchus retrograde lymphatic spread from parenchyma to the peribronchial region direct extension from adjacent parenchymal focus implantation of organisms from infected sputum. and hematogenous dissemination.

Because of generalized airway inflammation and oedema the disease may have an acute presentation like bronchial asthma, foreign body aspiration or pneumonia and can also have an insidious onset like bronchial carcinoma. There may be signs of collapse with monophonic wheeze on chest auscultation. All that wheezes is not asthma [3]. Patient may present with barking cough, bronchorrhea [4], chest pain dyspnoea, and haemoptysis. Constitutional symptoms are uncommon. Young adults are mostly affected with female preponderance [5]. The diagnostic modalities include sputum smear and culture which provide relatively fewer yields in EBTB [16 to 53.3%] compared with lung parenchymal disease. Endobronchial disease with ulceration & mucosal involvement is likely to have higher sputum positivity (>70%). For suspected cases bronchoscopy is highly desirable, both for diagnostic purpose and followup.

Chest radiograph: is normal in 10-20% of all the cases so a clear chest radiograph does not exclude the diagnosis of EBTB. There may be segmental or lobar collapse, broncholithiasis due to erosion of calcified nodes into the bronchi. Upper lobe bronchiectatic changes may also complicate EBTB.

Multiplaner & 3D images on CT chest are helpful for typical appearance of branching linear shadows of similar caliber arising from single stalk [the 'tree in bud appearance'] [6] and to pick up focal airway stenosis. CT scan chest accurately depicts the bronchial abnormality in 93 to 100% of cases. The findings include isolated [41 to 43%] lung segmental bronchial narrowing with concentric wall thickening, complete endobronchial obstruction [32%] and extrinsic compression by adjacent adenopathy [23 to 50%].

Fibreoptic Bronchoscopy is essential for diagnosis and treatment planning.

Bronchoscopy commonly shows diffuse mucosal congestion, edema & mass lesions & has 90% yield on smear as well as culture [7]. Endobronchial appearance include; actively caseating, edematous hyperemic, fibrostenotic, tumorous, granular, ulcerative and nonspecific bronchitis type lesions.

Complications of endobronchial tuberculosis include: collapse of dependent portions of lungs, bronchospasm & respiratory failure. During healing tracheal and bronchial stenosis and strictures can lead to permanent airway obstruction. Upper lobe bronchiectasis, haemoptysis, and fibrosis and scarring of pulmonary

parenchyma are other complications. Because of nonspecific symptoms, loss of weight and low grade fever lung cancer is the most important differential diagnosis. Other disease can mimick EBTB, such as, simple infective bronchitis, sarcoidosis, bronchial asthma, foreign body aspiration, endobronchial actinomycosis and endobronchial Kaposi's sarcoma particularly in patients with AIDS [8,9,10,11]. EBTB simulating pulmonary embolism is rarely reported. Early diagnosis and treatment may alter the natural course of the disease. Early introduction of standard ATT including Rifampicin, Isoniazid, Pyrazinamide, and Ethambutol/ Streptomycin should control the progression of EBTB. Aerosolized antituberculous drugs have also been tried . Corticosteroids have a debatable role [12]. However they are used with encouraging results. Potential benefits may be because of two reasons. Firstly, it reduces the bronchial narrowing to promote drainage & reduce the extent of post-stenotic lung damage, and secondly, it may help to reduce the long-term evolution of high-grade bronchial stenosis. Use of oral steroids in endobronchial TB, in addition to standard chemotherapy seems to prevent bronchostenosis to some extent. Demonstration of rapid healing & complete resolution of EBTB by local endoscopic injection of corticosteroids has been observed in different settings [13]. Other treatment options for complication include aerosolized isoniazid, streptomycin steroids, balloon dilatation and self-expanding metallic stents [14,15, 16]. Endobronchial laser and curettage have been used with encouraging results [17]. Bronchoplasty, with resection and anastamosis are other available options. Prognosis in EBTB is good for granular and non-specific bronchitis type but poor for tumorous type which can lead to bronchial stenosis despite adequate treatment, and is grave for edematous hyperemic type that results in fibrostenosis in 2/3rd of patients.

The case in discussion presented to a general hospital with severe dyspnoea, bronchospasm, which was non responsive to conventional treatment. On suspicion of pulmonary embolism she was rightly referred to the cardiology department. Though pulmonary embolism was ruled out yet the persistent hypoxemia was unexplained and development of respiratory distress syndrome was suspected. Endobronchial tuberculosis is predominantly airway disease with marked inflammatory changes resulting in bronchospasm, hypoxemia, variable degree of fever and constitutional symptoms. Permanent airway obstruction can occur after healing of inflamed areas requiring intervention. Fibreoptic bronchoscopy is the best way of monitoring airway remodeling. As in this case many patients respond ultimately to appropriate chemotherapy and inhaled steroids.

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Fig: 1
Chest radiograph showing bilateral mid and lower zone mottling along with ground glass haze.



Fig:2 Chest radiograph on discharge showing much clearance



Fig.3:
Bronchoscopic appearance depicting generalized mucosal inflammation and ulceration.



Fig.4
Close up of endobronchial appearance revealing granular appearance

