

Hollow in the Lung and Hoarseness: An Uncommon Association with Pulmonary Thromboembolism

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Abstract

A case is presented where focus of all investigations centered around a lung cavity which in association hoarseness appeared in all probability a lung cancer after no response to anti tuberculous chemotherapy. Oxygen desaturation on few steps of walk lead to further work up concluding with diagnosis of pulmonary thromboembolism with cavitory infarct and cardiovocal syndrome.

Keywords: *Cavitory infarct; Pulmonary thromboembolism; Cardiovocal syndrome*

Introduction

Pulmonary infarction is an infrequent complication of pulmonary thromboembolism owing to dual blood supply and rich capillary anastomosis. Liquefaction of pulmonary infarct-cavitory infarct is an unusual phenomenon. We report a case of cavitory lung disease in a 53-year-old previously healthy nonsmoker male who was treated for pulmonary tuberculosis without clinical improvement and ultimately proved to be a case of pulmonary thromboembolism with cavitory infarct. Hoarseness, another remarkable symptom in this case is also an exceptionally rare association with secondary pulmonary hypertension.

Case Report

A 53-year-old male presented with non productive cough and breathlessness on exertion. Patient was referred to pulmonology for bronchoscopy to rule out pulmonary malignancy as he was bearing a left upper lobe cavity in the chest x-ray he carried and was not improving with anti-tubercular treatment for last 4 months. Prior to visiting us patient had been through number of physician and specialty consultations for almost 10 months. Patient had been in good health in recent past, happens to be bank manager by occupation, never smoker and tee-totalar with athletic built. His problem begun with shortness of breath which he appreciated on routine morning walks. Few weeks later develops common cold and cough when he shows to a physician. Routine blood test picks up eosinophilia, chest x-ray (Figure 1) is reported unremarkable, spirometry (Figure 2) shows mild obstructive ventilator defect. He is prescribed diethyl carbazine (DEC), inhaled bronchodilators with steroid and anti histaminics. Patient gets no remarkable relief. He continues with dry cough and noticeable shortness of breath to the extent that he leaves morning walks. He shows to a cardiologist. TMT is negative for reversible ischemia, 2 Decho reported mild RA and RV dilatation with normal LV function. Repeat chest skiagram and CT scan (Figure 3, Figure 4) shows Left parahilar consolidation with breakdown. He is prescribed anti TB treatment after consultation with chest physician.

Patient continues ATT but even after four months he is not relieved of symptoms. He also develops hoarseness and ENT examination shows fixed left vocal cord. The repeat CXR and CT scan (Figure 5, Figure 6) now shows a large left upper lobe peripheral cavity with disappearance of the left parahilar lesion. Patient presents to us with this background of information and records.



Figure 1: CXR: May 2008.

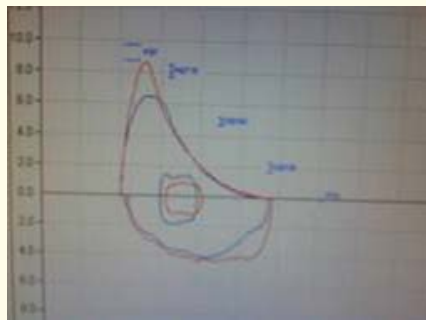


Figure 2: Spirometry: Mild obstruction.



Figure 3: CXR: Aug 2008.

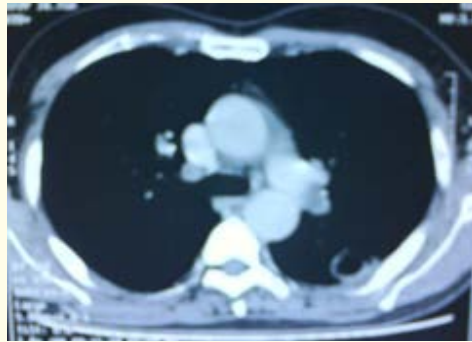


Figure 4: CT thorax: Aug 2008.



Figure 5: CXR: Nov 2008.

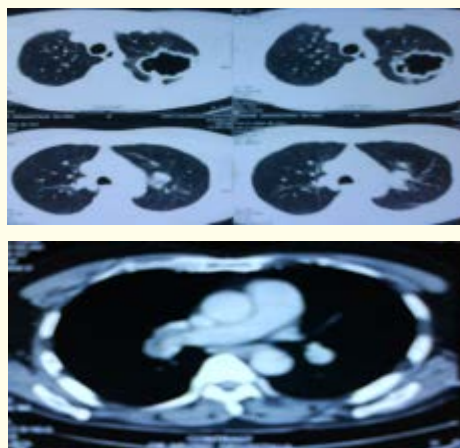


Figure 6: CT thorax: LUL cavity.

Patient is admitted for further workup. Physical examination is remarkable with hoarse voice, resting tachypnea (resp rate: 22/min) and tachycardia (pulse: 132/min, regular good volume). Chest and precordial auscultation reveals normal breath sounds and increased heart rate respectively. spO₂ on ambient air is 94%. Other systemic examination is normal. Patient is made to perform a Six Minute Walk Test. On 6MWT he complains of marked shortness of breath (Borge score 8/10) and marked desaturation is noticed on digital pulse oximetry (spO₂:74%) with extreme tachycardia (pulse rate 162 BPM).

Findings of 6MWT lead us to suspect pulmonary thromboembolism and investigations directed to PTE are pursued. 2D echo reveals dilated RA and RV, diminished RV contractility with PASP of > 60 mmHg. CT pulmonary angiogram (Figure 7, 8, 9) shows large cavity in left upper lobe with thick and irregular wall and thrombi in bilateral descending pulmonary arteries. Venous Doppler lower limbs picks up deep vein thrombosis in distal half of right superficial femoral vein. The laboratory investigation of patient was remarkable for eosinophilia which was related to background history of allergic rhinitis. Thrombophilia profile was remarkable for protein C deficiency. Bronchoscopy is done as requested by the admitting physician and it shows left vocal cord palsy (Figure 11) with normal appearance of tracheobronchial tree. Bronchial washings are negative for malignancy in cytology and negative for AFB on ZN staining.



Figures 7,8,9: CT pulmonary angiogram: Filling defects in descending pulmonary arteries and LUL cavity.

Patient is started on low molecular weight heparin and to which he responds with remarkable relief in symptoms and review 2D echo showing decreased PASP. Patient is continued on low molecular weight heparin and is prepared for thoracotomy to rule out any possibility of malignancy as cause of hoarseness still remains unexplained. Left upper lobe wedge resection is done. Histopathology reveals extensive inflammation and fibrosis with organized thrombi in supplying vessels. IVC filter is placed in the femoral vein in the same sitting. Patient is discharged in improved general condition on oral anticoagulant and follows regularly in OPD. International Normalized Ratio (INR) is maintained between 2-3. His hoarseness completely recovered in 2 months and he is able to continue his professional career and active personal life as before.

Discussion

Cavitary lung disease can be caused by a wide variety of pathologic conditions. Possible etiologies include infection, metastatic malignancies, septic pulmonary emboli, granulomatous vasculitides and rarely pneumoconiosis and pulmonary sequestration. Cavitation resulting from bland pulmonary infarction is often not considered in differential diagnosis [1-4]. Pulmonary infarction occurs in only 10% of patients of pulmonary thromboembolism. Cavitation after pulmonary infarction is even a rare event. Large autopsy series reveal cavitation in 4-5% of all pulmonary infarcts [3]. It is general agreement that cavitation occurs when the infarct is more than 4 cms. It chiefly involves the upper and middle zones of the lungs with only 20% involvement of the lower lobes. Morphologically, the infarct is typically hemorrhagic with coagulative necroses of parenchymal frame work, which heals with minimal fibrosis. On the other hand, liquefactive necrosis is unusual with incidence of 2-4%. Liquefaction usually follows septic thromboembolism, though it may occur in bland thrombi

[3,5,6]. The natural history of cavitory infarctions is not well documented or understood. There have been number of case reports describing various complications and high mortality rates associated with this condition. Mortality rate as high as 41% in non infected and 73% in infected pulmonary infarcts have been reported. An aggressive surgical approach to management of cavitory infarcts with use of measures to prevent further emboli has been advocated by some authors. It is important to remember that cavitory pulmonary infarction though rare but nevertheless forms one of the differential diagnosis of cavitory lung lesions [10,11].



Figure 10: CXR: Post LUL wedge resection.

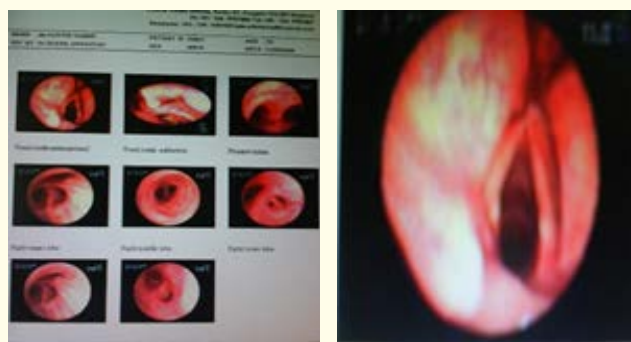


Figure 11: Video laryngobronchoscopy: LVC palsy.

Hoarseness was a prominent complaint in our patient and video laryngoscopy confirmed the left vocal cord palsy. Ortnier's Syndrome (described 118 years ago in 1897) is a clinical entity with hoarseness due to a left recurrent laryngeal nerve (LRLN) palsy caused by cardiac disease [7]. Left recurrent laryngeal nerve palsy has been reported with extreme rarity in association with moderate to severe pulmonary hypertension [8]. The mechanical cause has been advocated due to compression of RLN between enlarged pulmonary artery and aorta at ligamentum arteriosum [9].

Conclusion

This case is unique because of following reasons:

- a. The difficulty in diagnosis imposed by conundrum of breathlessness and hoarseness with angry looking large cavity in left upper lobe lung which ultimately turned out to be venous thromboembolism with cavitory infarct. This naturally incurred hot debates among the treating team viz. physician, pulmonologist, radiologists and cardiovascular surgeon.

- b. The hoarseness which confused and misdirected the attention towards malignancy and even lead to lung surgery later could get explanation of cardiovocal syndrome (Ortner's syndrome).
- c. Last but not the least, the simple observation which gave the clue towards embolic phenomenon was not anything special but the digital pulse oxymetry during walk (Six-minute walk test).

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