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Right Destroyed Lungs Syndrome with Dextrocardia

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Abstract: Chronic lungs disease such relapsed tuberculosis of the lungs could lead to complications that could alter the gross anatomy of a region of the body. Complications have been reported from chronic and relapsed lungs infections such as *Mycobateruim tuberculosis* when they are not properly managed, Although, the indexed case improved within a short time after admission. The reported case was a rare complicated relapsed lungs tuberculosis. He was a 45 years old businessman who presented with history of chronic cough of 25 years duration with associated hemoptysis, treated in the past for lung tuberculosis. He had clinical features and radiological features of destroyed right lung syndrome with dextro cardia following relapsed tuberculosis with possible superimposed infection. He was managed with category two regimen although did not complete his treatment in this facility. Radiographic complications in the index case were mediastinal shift, gross distortion of the right lung with associated fibrosis, contra-lateral lung emphysema which was noticed to compensating for the destroyed right lung. These findings are strongly consistent with some other reports that artificial/pathologic dextro-cardia can be as a result of relapsed tuberculosis with gross distorted chest region. Therefore, physicians should guide against relapsed tuberculosis by revamping Direct Observe Treatment (DOT) through counseling as well as prevent transmission of multi-drug resistance tuberculosis through proper investigation.

Key words: Destroy lungs syndrome, DOT, lungs tuberculosis, lung infection, chronic lung disease

INTRODUCTION

Radiological presentation of destroyed lung syndrome as in the reported case is rare (Adefuye *et al*., 2010). Worldwide, however, they are fairly. Adefuye *et al*. (2010) present in developing nations where tuberculosis is still a rampaging infectious disease among its populace even in this present days of DOTs.

Primary lung tumours, mediastinal masses, vascular abnormalities and various progressively destructive pulmonary infections such as *Mycobacterium tuberculosis* (Dosumu, 2005; Ikeda *et al*., 2012) are considered to be the predominant causes unilateral lung destruction (Rajasekaran *et al*., 1999; Conlan *et al*., 1995; Blyth *et al*., 2002; Le Roux *et al*., 1986; Massard *et al*., 1996; Fischer and del Missier, 1959).

CASE REPORT

Mr. EI was a 45 years old businessman who presented through the accident and emergency department with history of cough of 25 years duration and hemoptysis which started 2 weeks ago with several and recurrent episodes for the past 3 days. Few days before admission, he had sputum smear screened for acid-fast bacilli, performed at a tuberculosis referral centre.

Result was negative for all three smears. He had been diagnosed twice for tuberculosis in the past and treated with anti-tuberculosis drugs twice as well. One of the course lasted for 8 months using category 1 therapy in 2009. A year later, he presented with the above clinical presentation.

On examination he was cachetic, orthopneic, weak, he had features of right pleural effusion with fibrosis (tracheal deviation to the right while auscultation of heart sound was on the right side of the chest. The patient was clinically assessed as a case of destroyed lungs syndrome and dextro-cardia secondary to relapsed tuberculosis with super-imposed infection.

He had X-ray film taken and reported on 2nd day post admission (Fig. 1). Report revealed silhouette sign, indeterminate cardiac size, complete loss of normal cardiac/mediastinal outline, complete mediastinal shift to the right due to collapse of the right lung and thickened chest wall. Inhomogeneous opacities of right lung field (mid and basal lung zones) was noted. There was consistent finding of right upper and middle lobe lung collapse. Ipsilateral apex is devoid of lung marking with air/fluid level thus hydropneumothorax. There was feature of compensating emphysema of the left thoracic lung, focal cavitation and wide spread streaky changes worse in the upper lung zone, thoracic cage was intact.

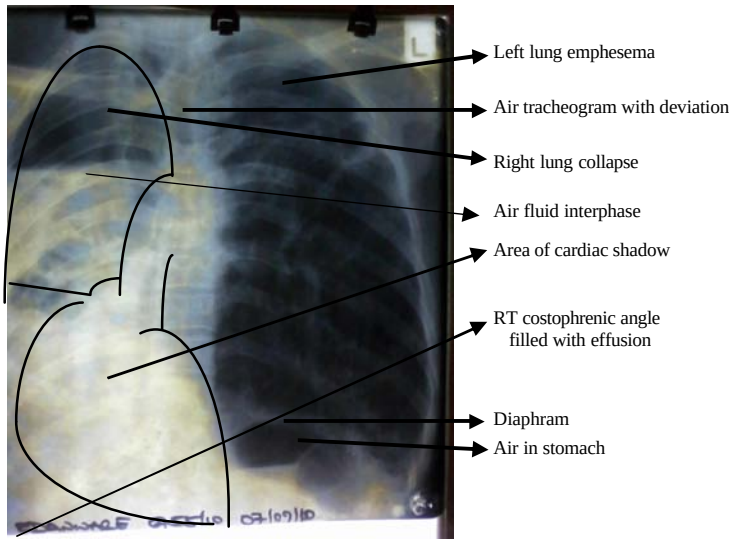

Fig. 1: The 2nd day film of destroyed left lung syndrome

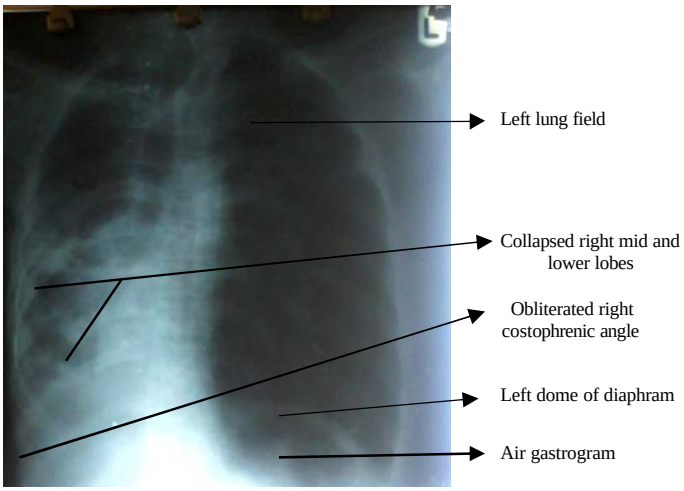

Fig. 2: Resolving pleural effusion

Pleural tap of 4 mL of bloody fluid was collected in 5 mL syringe while in emergency unit, smear showed few lymphocyte with fungal hyphae, probably contaminant no malignant cell seen, therefore malignancy was ruled out.

His full blood count showed packed cell volume of 24% white blood cell count of 79×10^9 cells L^{-1}. Among the white blood cells, neutrophil and lymphocyte consist 75 and 24%, respectively which was reported 7th day later after admission. HE was transfused with 2 units of sedimented blood cells and 1 unit of whole blood. He was placed on category 11 anti tuberculosis therapy. A combination therapy consisting Rifampicin, Isoniazid, Pyrazinamide and Ethambutol. Three tablets of these combination daily was administered as well as intramuscular streptomycin, (0.75 g) 1.5 mL in accordance with his weight estimation. Streptomycin was taken three times in a week, a least alternate days. He had his medications for 8 days before referral to another centre to continue his medications for close proximity. Before he left, the severity of orthopnoea, dyspnoea and cough had drastically reduced.

A repeat X-ray film (Fig. 2) of the chest performed 14 days later showed silhouette sign with associated inhomogeneous opacities and cavitations within the right lungs field. Features were consisted with right lung collapse, especially the mid and lower lobes. There was obliteration of the right costophrenic and

diaphragmatic angle with effusions but reduced as compared to previous X-ray film (Fig. 1). The left lung field was spared and bony thorax was intact.

DISCUSSION

Destroyed lungs syndrome is one of the morbidity of relapsed lungs tuberculosis and commonly affect the left (Adefuye *et al*., 2010) because of its anatomical characteristics. Adefuye *et al*. (2010) demonstrated in a poster presentation that destroyed lung syndrome could involve the right lung rather than the left lung which was the same with the reported case. A clinical study (Dosumu, 2005) that was carried out between January 1996 and December 1997, showed that of 500 newly diagnosed cases of Pulmonary Tuberculosis that were treated with Directly Observed Short-course Treatment (DOT). Hundred of them had chest radiographic examination done. Cases of destroyed lung syndrome stands at 5 (5%) while 80% where in HIV sero-positive patients. This fact proves that the reported complication is not a common phenomenon and is more associated with HIV sero-positive patients rather than sero-negative patient. The index case was HIV sero-negative. Moreover, there was fibrosis in the right lungs field as confirmed in the X-ray film. This complication was noticed in my client as well. The reviewed study (Dosumu, 2005) stated that 70% of studied films had fibrosis in about 57.1% patients which were sero-negative. Other complications seen in the reported patients were pleural effusion, contra-lateral emphysema with cavitation. The listed complications in the reported patient showed some of the radiographic patterns as reported by Dosumu (2005). Obviously, the patient did not have congenital situs-inversus because even on the both X-ray film, the stomach lies in the left side of the film as proven by the lucency of gastric air bubble therefore congenital situs-inversus was ruled out. Detection of destroyed lung are quite easy beginning from clinical examination of the patient which may reveal heart sounds on contra-lateral side, increased tympanicity of the compensating lung zone. Other investigations that can be carried out are chest X-ray (Fig. 1 and 2) and computed topography (Ikeda *et al*., 2012).

The presence of fibrosis, mediastinal shift to the right, contra-lateral left compensatory emphysema and right sided tracheal deviation in the patient are strong clues for the possible cause of the pathologic dextrocardia demonstrated by the absence of cardiac shadow on the left side of both radiographic films (Fig. 1 and 2).

Therefore, the hypothetical pathologic process that followed as aftermath of chronic lung parenchyma disease could be described as thus Lung collapse resulted from air leak into intra-thoracic space which has negative pressure followed by fibrosis formed from scar tissue of destroyed air sacs, lung tissue and capillaries then next to follow is tracheal deviation and mediastinal shift due to contracted fibrous tissue. Dosumu (2005) went further stating that upper lung lobes are affected by tuberculosis other rather than the lower lobes as seen in the patient. However, the left lung is reported (Adefuye *et al*., 2010) to be more affected in destroyed lung syndrome due to the anatomical structure of the left main bronchus but for the index patient radiographic film shows otherwise. Pleura tap of the reported showed no malignancy but reported that there was fungal hyphae as contaminant. It could be possible that there was fungal infection which was part of the pathology the lung. Other reports (Ikeda *et al*., 2012; Kosar *et al*., 2010) have notice fungal infection following pulmonary tuberculosis and that surgery is a viable treatment option. Example of surgical options for destroyed lungs (Conlan *et al*., 1995) from chronic infections such as pulmonary tuberculosis and Aspergilosis are thoracostomy with chest tube insertion and pneumonectomy (Kosar *et al*., 2010). This may be considered as options where conservative management and DOT fails putting into perspective, the clinical state of the respiratory system.

CONCLUSION

The report clearly indicates that there should be a strong advocacy for multi-drug resistance testing (Adefuye *et al*., 2010) to strengthened DOTs and prevent recurrence, thereby reducing morbidity and mortality that may arise.

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