SURGICAL MANAGEMENT OF TUBERCULOSIS INDUCED DESTROYED RIGHT LUNG WITH PATHOLOGIC DEXTROCARDIA AT UNTH, ENUGU (A CASE REPORT)

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ABSTRACT

Chronic lung diseases like relapsed pulmonary tuberculosis can lead to many complications such as unilateral perenchymal destruction of the lung, pulmonary aspergilosis, scar adenocarcinoma of the lung, lung abscess or bronchiectasis among others. This reported case of unilateral right lung destruction with pathologic dextrocardia was a rare presentation in our institution. It was successfully managed by surgery. It should be emphasized that prompt diagnosis of pulmonary tuberculosis and adequate treatment by DOT method will go a long way in reducing if not eliminating the attendant complications.

INTRODUCTION

Unilateral total post-tuberculous lung destruction is a well recognized cause of morbidity and mortality[1,2] worldwide especially in a developing country like Nigeria where the disease is still rampaging. The term tuberculous destroyed lung syndrome is usually used to describe the destructive lung parenchymal changes due to sequelae of pulmonary tuberculosis, which occur over years and cause chronic airway obstruction as well as restrictive disease[3].

As a result of the lung destruction, there is compensatory overexpansion of the contralateral lung which results in mediastinal shift towards the diseased lung, producing bronchial and tracheal compression[4]. In the literature, the report of tuberculous destroyed lung with pathologic dextrocardia managed successfully by surgery is very rare. In this manuscript, we report a case of a tuberculous induced destruction of the right lung, causing pathologic dextrocardia but which corrected after surgical treatment.

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CASE REPORT

Miss OU is a 29-year old female graduate of Linguistic. She developed recurrent cough of 5 years and haemoptysis of 18 months duration. She was in apparent state of good health until about 5 years prior to presentation when she developed cough which was productive of clear sputum but occasionally yellowish and later became progressively blood stained. Cough was not periodic but worse at night.

Initially there was no expectoration of blood and sputum was not copious or foul smelling. There was associated right sided chest pain, sharp in character, non-radiating, worsened by cough and physical activity and relieved transiently by analgesics. Weight loss experienced few years after the onset of cough became progressive with anorexia but no vomiting, jaundice, abdominal discomfort or change in bowel habits. There was associated generalized weakness and joint aches but no history of palpitations, dyspnoea or peripheral leg swelling.

She had a therapeutic trial for pulmonary tuberculosis for about 6 months and was said to have resolution of symptoms. She later relapsed. She was not a known hypertensive and diabetic. There was no history of cigarette smoking or excessive indulgence in alcohol.

Examination revealed a chronically ill-looking young lady, alert, not pale, anicteric, acyanosed, afebrile, not dehydrated, no digital clubbing, no pedal oedema or peripheral lymphadenopathy.

Chest examination revealed respiratory rate to be 20 cycles per minute, asymmetrically depressed(the right hemithorax was slightly concave, which suggests depression rather than flattened) right chest relative to the left. There was ipsilateral reduced excursion, reduced tactile fremitus, dull percussion note and absent breath sound. Prominent resonant note and accentuated vescicular breath sound were noted on the contralateral side.\

Cardiovascular examination showed pulse rate to be 100 beats per minute, moderate volume and regular rhythm. There was no elevation of jugular venous pressure. Apex beat was displaced towards the right hemithorax, just medial to the right midclavicular line.

Abdominal examination revealed normal siting of the intrabdominal viscera (the liver span was found to be 9cm on the right side and the tip of the spleen was felt on the left hypochondrium) and there was no significant finding in the musculoskeletal system including the skin and its skin integument. A working diagnosis of pulmonary tuberculosis with pathologic dextrocardia was made.

A posteriolateral chest x-ray done showed complete opacification of the right hemithorax, extensive mediastinal shift to the right, tracheal deviation and compensatory emphysema of the left lung. The computerized tomography confirmed the findings on the chest x-ray.

Figure 1

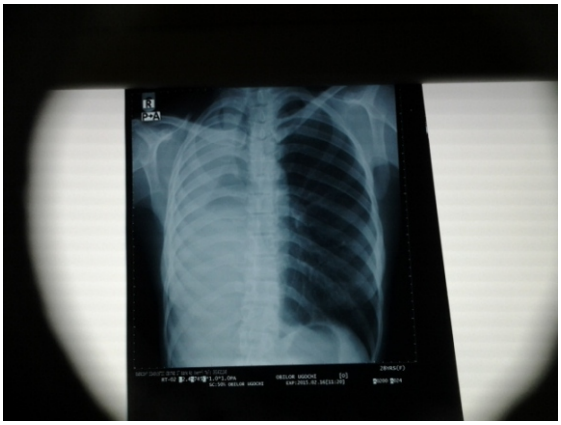


Figure 2

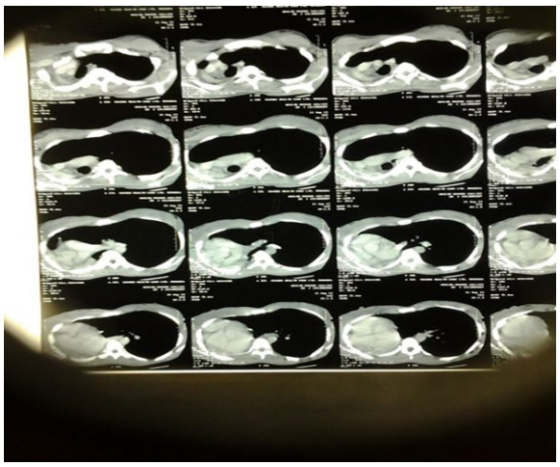
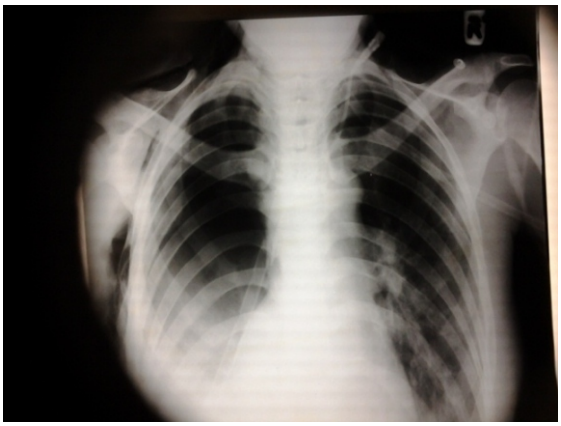


Figure 3



Acid fast bacilli(AFB) x 3 times was negative. Abdominal ultrasound finding was not significant. Haematologic evaluation was normal except for differential lymphoctyosis of 65% and ESR of 25mm(Westergreen). Echocardiography revealed only complete dextroposition of the heart due to extensive right lung lung and mediastinal shift. Bronchoscopy was done and it revealed complete obliteration of the orifice of the right main bronchus.

The patient's KPSS (Karnosfsky Performance Status Scale) was estimated at best to be 60-70%, since she cared for herself but was unable to carry on normal activity or do active work.

The lung function test was actually done. The parameters were as follows: clinical evaluation using dyspnoea grade after 6 minutes walk and pulmonary mechanics (FEV1, FVC, FEV1/FVC) as well as ABG parameters. It was on the basis of the results,

Figure 4

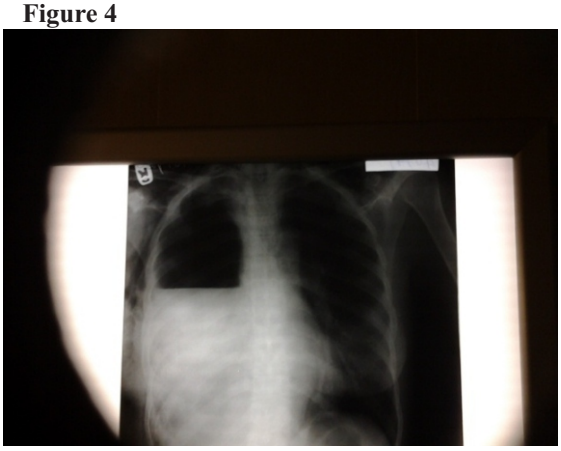
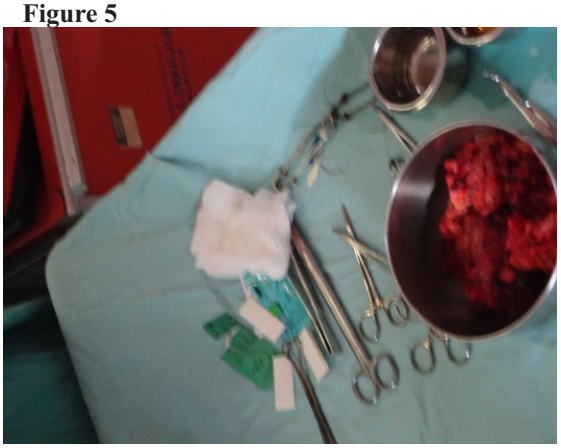


Figure 5



especially, FEV1 which was more than 60%, that pneumonectomy was carried out. The issue of postoperative oxygen dependency will not arise.

On the basis of the above findings, a working diagnosis of tuberculous destroyed right lung was entertained and patient was scheduled for right pneumonectomy.

Intraoperative findings included dense pleurofibrosis, thickened fibrotic lung that was adherent to the pericardium. Right main bronchus was narrowed with obliterated lumen. Surgically, upon entering the thoracic cavity, blunt and sharp dissection was done anteriorly and posteriorly to mobilize the lung from the chest wall. This was followed by transection of the inferior pulmonary ligament to free the base from the diaphragm. Consequently, the inferior pulmonary vein was identified and securely ligated and transected.

Hilar dissection anterioly, exposed the right superior pulmonary vein. It was securely ligated and transected. This further exposed the right pulmonary artery. It was doubly clamped and transected in between clamps. The proximal stump was doubly ligated with vicryl 0 suture while the specimen side was singly ligated. Further blunt dissection and control of haemostasis by cautery, exposed the right main bronchus, which was clamped with bronchial clamp, transected and specimen removed. The proximal stump was closed securely with ethibond suture, size 2-0. Haemostasis was further carried out, chest irrigated with warm normal saline, intercostals nerve block appropriately instituted and chest closed in layers with chest tube drain in-situ. The chest tube drain was removed the following day after satisfactorily establishing that haemostasis was adequate and mediastinal stablisation has occurred, thereby allowing the postpneumonectomy space to fill and organize.

DISCUSSION

Destroyed lung is an end stage phenomenon prone to serious complications and irreversible changes in the lung p'arenchyma[5]. It is commoner in adults and rare in children. Our patient is a young adult, aged 29 years. This recognized phenomenon occur as a primary disease or re-infection. Such patients affected can report for the first time as for diagnosis or after completion of treatment as was the case in the index patient. They can also be referred as noresponders to anti-tuberculoses treatment on account of harbouring drug resistant organisms[6].

Presently, there are potent short course drug regimens affecting early bactrericidal and sterilizing actions to prevent lung destruction. However, some patients still present to hospitals with significant lung destruction. In a study done by S. Rajasekara et al, in India in 1991[7], 51% of 871 newly diagnosed patients had far advanced tuberculosis because of delay in seeking for treatment and this is due to the

fact that occasionally tuberculosis symptoms being insidious are likely to wax and wane and such patients decline to present for early treatment. In another study by S. Rajasekara et al, in India in 1999[8], it was found that 81.8% of 60 patients studied had left lung destruction. In this study, our patient had right lung destruction with pathologic dextrocardia.

Cessation of pulmonary blood flow to the destroyed lung[9] is likely to result in reduced levels of chemotherapeutic agents reaching affected sites and, thus, ineffective killing of tubercle bacilli and consequently, results in microbial persistence leading to relapsed tuberculosis and treatment failure with multresistant anti-tuberculosis(MDR-TB,)[10]

Surgically, a classic posteriolateral thoracotomy through the 5th interspace was employed with careful adhesiolysis with electrocautery to release the pericardium that was adherent to the destroyed lung. A double lumen endotracheal intubation was used. Such approach was used in a study done by Altug kosar et al, where pneumonectomy was done for 18 children with destroyed lung[11].

Several authors have concluded that pneumonectomy should be avoided whenever possible[12] but there are situations when pneumonectomy or pleuropneumonectomy remain the only curative treatment modality for benign inflammatory lung diseases such as destroyed lung, significant hemoptysis, main bronchial stenosis, MDR-TB or significant symptoms such as a productive cough[13].

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