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ORIGINAL ARTICLE

Profile and Outcome of Unilateral Tuberculous Lung Destruction in Ilorin, Nigeria

Le profil et les résultats de la destruction unilatérale du poumon tuberculeux à Ilorin, Nigeria

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ABSTRACT

BACKGROUND: **Unilateral tuberculous lung destruction is a serious chronically disabling and often fatal complication of pulmonary tuberculosis. A few previous studies have dealt with some aspects of this entity among Nigerians with pulmonary tuberculosis but these studies may not truly reflect its current trends.**

OBJECTIVE: **To describe the presenting profiles and outcome of unilateral tuberculous lung destruction among patients with pulmonary tuberculosis.**

METHODS: **The study was a chart review of the medical records of adult patients with pulmonary tuberculosis complicated by unilateral lung destruction seen between January 1999–December 2008. Data extracted included demographic, and outcome of treatment. The clinical features, sputum results and illustrative cases are presented.**

RESULTS: **Unilateral lung destruction complicated 74(1.3%) of 5,926 pulmonary tuberculosis cases seen over the review period. Most cases occurred in males [45(60.8%)] and they were predominantly [51(68.9%)] below 40 years of age. Most of them [52(70.2%)] presented after six months of symptoms. Forty-seven (63.5%) of them had had previous exposure to antituberculosis drugs. The left lung was affected in 49 (66.2%) patients. Only 25 (33.8%) of them were cured while the adjusted mortality rate was 25.5%. Male gender, presence of dyspnoea on mild exertion and right ventricular failure were significantly associated with increased mortality.**

CONCLUSION: **Unilateral tuberculous lung destruction has unacceptably high mortality rate which can be prevented by patient presenting early to the hospital, prompt diagnosis and strict adherence to the standard treatment. WAJM 2011; 30(2): 130–135.**

Keywords: **Lung destruction, pulmonary tuberculosis, Nigerians.**

RÉSUMÉ

CONTEXTE: La destruction unilatérale du poumon tuberculeux est un sérieux handicap chronique et souvent une complication mortelle de la tuberculose pulmonaire. Quelques études antérieures ont traité certains aspects de cette entité chez les Nigerians atteints de tuberculose pulmonaire, mais ces études ne reflètent véritablement pas les tendances actuelles.

OBJECTIF: Décrire les profils cliniques révélateurs et le pronostic de la destruction unilatérale du poumon chez les patients atteints de tuberculose pulmonaire.

METHODES: L'étude s'est appuyé sur un examen des dossiers médicaux des patients adultes atteints de tuberculose pulmonaire compliquée par la destruction unilatérale du poumon observée entre Décembre 2008 et Janvier 1999. les données notamment démographiques, et les résultats du traitement ont été recueillies . Les caractéristiques cliniques, les résultats de l'examen des expectorations et des cas illustratifs sont présentés.

RÉSULTATS: La destruction unilatérale compliquée du poumon a été notée chez 74 patients soit (1,3%) des 5.926 cas de tuberculose pulmonaire vu au cours de la période d'examen. La plupart des cas sont survenus chez les hommes [45 (60,8%)] et ils étaient pour la plupart [51 (68,9%)] en dessous de 40 ans. La plupart d'entre eux [52 (70,2%)] ont consulté après six mois de symptômes. Quarante-sept (63,5%) d'entre eux avaient eu un traitement antérieur à des médicaments antituberculeux. Le poumon gauche a été touché chez 49 (66,2%) patients. Seulement 25 (33,8%) d'entre eux ont été guéris alors que le taux ajusté de mortalité était de 25,5%. Le sexe masculin, la présence de dyspnée à l'effort minime et l'insuffisance ventriculaire droite étaient significativement associés à une mortalité accrue.

CONCLUSION: La destruction unilatérale du poumon tuberculeux a un taux de mortalité beaucoup trop élevé qui peut être prévenue par une consultation hospitalière précoce des patients , un diagnostic rapide et l'adhésion stricte au traitement standard. **WAJM 2011; 30(2): 130–135.**

Mots Cles: Destruction du poumon, Tuberculose pulmonaire, Nigérians.

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Abbreviations: AFB, Acid-Fast Bacilli; **MDR-TB,** Multi-Drug Resistance Tuberculosis; **NTLCP,** National Tubercu-losis and Leprosy Control Programme.

INTRODUCTION

Unilateral lung destruction is a recognized complication of pulmonary tuberculosis.[1,2] Other important causes include various progressively destructive non tuberculous pulmonary infections, primary lung tumors, mediastinal masses, bronchial stricture, and congenital malformations.[3–6] Tuberculous lung destruction causes irreversible changes in lung parenchyma resulting in chronically morbid and sometimes acute life-threatening complications such as massive haemoptysis, empyema, secondary fungal infections, secondary amyloidosis, septicaemia, and pulmonary-systemic shunting.[4,6]

Prompt diagnosis, appropriate chemotherapy and adherence to anti-tuberculosis treatment can easily prevent this chronically disabling and often fatal condition among patients with pulmonary tuberculosis.[7] In Nigeria, late diagnosis and poor adherence to treatment are important reasons for this type of presentation among patients with pulmonary tuberculosis. Although there are no accurate data on tuberculosis in Nigeria, treatment outcomes are poor among those evaluated. Anecdotal reports have also shown that non-adherence and treatment defaults are common.[9,10]

Apart from the morbidity and mortality in the affected patients, unilateral tuberculous lung destruction is also of public health importance because it may be associated with microbial persistence and multi-drug resistance tuberculosis (MDR-TB).[11] Although a few previous reports[1,12] described this problem among Nigerians with pulmonary tuberculosis, reports are old and so may not truly reflect current trends. Apart from the case report in a ten-year old child by Saidu in 2006,[12] we are not aware of any recent work on unilaterally destroyed lung among Nigerians with pulmonary tuberculosis. This study was therefore undertaken to review the presenting profiles and outcome of cases of unilateral tuberculous lung destruction among patients with pulmonary tuberculosis managed at the University of Ilorin teaching hospital in Nigeria. Two illustrative cases are presented in order to further highlight the common mode of presentation and outcome of the patients.

SUBJECTS, MATERIALS, AND METHODS

This was a ten-year (January 1999–December 2008) chart review of patients with pulmonary tuberculosis complicated by unilateral lung destruction. The charts of all adult patients with diagnosis of destroyed lung complicating pulmonary tuberculosis were retrieved from the medical records department. After review, those with clinical and radiological evidence of unilateral lung destruction were selected. Any chart with clinical diagnosis but no radiological report was not included. Data on demography, presenting features, sputum smear microscopy, smoking status, HIV status, presence of right ventricular failure and treatment outcomes (cured, lost to follow up, treatment failure, transferred out and death) were extracted. All the new patients were treated with eight months short course regimen (rifampicin, isoniazid, pyrazinamide and ethambutol for two months, followed by isoniazid and ethambutol for six months) as recommended by the National Tuberculosis and Leprosy Control Programme (NTLCP) of Nigeria. The retreatment cases were treated with standardized retreatment regimen (rifampicin, isoniazid, pyrazinamide, ethambutol and streptomycin for two months, followed by rifampicin, pyrazinamide, isoniazid and ethambutol for one month and rifampicin, isoniazid and ethambutol for five months). The total number of adult patients with pulmonary tuberculosis during the period was 5926.

Definitions

Smear-positive pulmonary tuberculosis: A patient with at least two initial sputum specimens which were positive for AFB by microscopy, or a patient with only one sputum specimen which was positive for AFB by microscopy, and chest radiographic abnormalities consistent with active pulmonary tuberculosis.

Smear-negative pulmonary tuberculosis: A patient with symptoms suggestive of tuberculosis, with at least three sputum specimens negative for AFB by microscopy, and radiologic abnormalities consistent with active pulmonary tuberculosis and lack of clinical response to a course of broad spectrum antibiotic therapy.

Cured patient: A patient who completed treatment with negative bacteriology result at the end of treatment and on at least one previous occasion.

Lost to follow up: These are patients who were not transferred out but failed to complete the prescribed treatment regimen.

Treatment failure: a patient who was sputum smear-positive at five months or later during treatment.

Patients transferred out: patients whose treatment results were unknown due to transfer to another health facility.

Dead patients: patients who died from any cause during the course of treatment. Statistical analysis.

Data were analyzed using SPSS version 13.0. The mean ± standard deviation was calculated for the ages while percentages were calculated for the categorical variables. Data comparison for the categorical variables was done using the chi-squared or Fisher's exact test as was appropriate. P value < 0.05 was considered significant.

RESULTS

Seventy-four out of the 5926 patients with pulmonary tuberculosis had unilateral lung destruction giving a prevalence rate of 1.3% over the review period. They were predominantly [45(60.8%)] males with a mean age of 36.0 ± 14.2 and a range of 17–72 years. Majority of them were in the younger age group with 51 (68.9%) of them being below the age of 40 years. Most of them [52 (70.2%)] presented late; after six or more months of onset of symptoms. They were mostly [47(63.5%)] retreatment cases. The left lung was affected in 49 (66.2%) patients. Most of the other presenting features were similar to those in pulmonary tuberculosis patients without associated lung destruction (Table 1).

Table1: Presenting Characteristics of Patients with Unilateral Tuberculous Lung Destruction.

Characteristic	Number(%)
Age group(years)	
<40	51(68.9)
≥40	23(31.1)
Gender	
Male	45(60.8)
Cough Present	**74(100)**
Fever	
Low	34(45.9)
High	5(6.8)
None	35(47.3)
Weight loss	
Present	60(81.1)
Chest pain	
Present	17(23.0)
Haemoptysis	
Mild	15(20.3)
Moderate	7(9.5)
None	52(70.2)
Dyspnoea on exertion	
Present	26(35.1)
Duration of symptoms*	
<6months	22(29.8)
≥6months	52(70.2)
Sputum smear	
Positive	41(55.4)
Cigarette smoking	
Present	11(14.9)
HIV status	
Positive	9(12.2)
Site of destruction	
Right lung	25(33.8)
Left lung	49(66.2)
Right ventricular failure	
Present	14(18.9)
Treatment status	
New	27(36.5)
Retreatment	47(63.5)

**Duration of symptoms before presenting to hospital*

Table 2: Treatment Outcome in Patients with Unilateral Tuberculous Lung Destruction

Outcome	Frequency N(%)
Cured	25(33.8)
Lost to follow-up	32(43.2)
Treatment failure	3(4.1)
Transferred out	1(1.4)
Dead*	13(17.5)

**Adjusted mortality rate=25.5%*

Only 25(33.8%) patients were cured while 32(43.2%) were lost to follow up. Thirteen patients died giving a crude mortality rate of 17.5% (Table 2). However, after adjusting for the patient who was transferred to other centre and the patients who were lost to follow up, the adjusted mortality rate was 25.5%. Table 3 shows the presenting features of prognostic significance in the patients who were cured and those who died. Male gender, presence of dyspnoea on mild exertion and right ventricular failure were significantly associated with increased mortality.

Illustrative Cases

Case 1 was a 46-year old retired soldier who presented in December 2005 on account of recurrent dyspnoea on mild exertion and cough productive of whitish sputum of about three years duration without haemoptysis. There was occasional low grade fever associated with weight loss despite good appetite. He had prolonged contact with a friend with chronic cough. He was diagnosed of pulmonary tuberculosis about three years earlier but defaulted after two months of treatment because he got better. He also received anti-tuberculous treatment for three months a year before presenting to our clinic when the symptoms recurred. He had smoked 5 pack-year of cigarette but stopped about 10 years prior to presentation based on personal conviction to stop. He drank lager beer usually during social events but stopped about 10 years ago. There was no history of extramarital affair.

On examination, he was a middle-aged man, chronically ill looking, afebrile, without significant peripheral lymph-adenopathy, or pedal oedema. Respiratory system examination revealed respiratory rate of 20 cycles per minute, no obvious respiratory distress at rest, trachea was deviated to the right side with diminished chest movement on the same side. The percussion note was dull over the right lung field with low-pitch bronchial breath sound and coarse crepitations. There was clinical evidence of hyperinflation of the left lung. The only abnormal finding in the cardiovascular system was the presence of the apex beat in the 5th right intercoastal space just lateral to the sternum. An assessment of destroyed right lung secondary to poorly treated pulmonary tuberculosis was made.

Two of his three sputum specimens were positive on microscopy for acid-fast bacilli (AFB), but mycobacterial culture and sensitivity was not done because there was no facility for such in our centre. Sputum cytology did not show any malignant cells. Chest X-ray showed fibrocystic shadows in the right lung with complete shifting of the mediastinum to the ipsilateral side and compensatory emphysema in the left lung field (Figure 1). Electrocardiography was reportedly within normal limit. HIV screening was non reactive, erythrocyte sedimentation rate was raised (45mm/hour) and other blood tests were normal. He was managed on retreatment regimen with rifampicin, isoniazid, pyrazinamide, ethambutol and streptomycin for two months, followed by rifampicin, pyrazinamide, isoniazid and ethambutol for one month. He however failed to return to the clinic for the continuation phase of his treatment despite adequate counseling on the need for strict adherence to the treatment regimen.

Case 2 was a 30-year-old driver, who was referred to our clinic in March 2008 on account of cough productive of greenish sputum and progressively worsening dyspnoea of four months duration. There was low grade fever with weight loss of three months duration and progressive bilateral leg swelling of two weeks duration. There was orthopnoea, but no paroxysmal nocturnal dyspnoea or palpitation. He denied a history of contact with anybody with chronic cough. He had been treated for pulmonary tuberculosis about five years earlier but defaulted after four months of treatment. There was no history of cigarette smoking, ingestion of alcoholic drinks, or extramarital affair.

Examination revealed a young man, chronically ill looking, afebrile with tinge of central cyanosis, and bilateral pitting pedal oedema up to the lower third of the leg. Respiratory examination revealed respiratory rate of 32 cycles per minute, trachea was deviated to the left side with diminished chest movement on the same side. The percussion note was dull over

Table3: Features of Prognostic Significance in Patients who were Cured or Died.

Variable	Cured (n)	Dead (n)	χ^2	P value
Age(years)				
<40	13	9		
≥40	12	4	1.04	0.31
Gender				
Male	14	12		
Female	11	1	5.22	0.02†
Chest pain				
Present	5	3		
Absent	20	10	0.49	0.57
Haemoptysis				
Mild	4	1		
Moderate	3	1		
None	18	11	1.15	0.77
Dyspnoea on mild exertion				
Present	5	8		
Absent	20	5	6.56	0.01†
Duration of symptoms*				
<6months	11	2		
≥ 6months	14	11	3.11	0.08
Sputum smear				
Positive	12	8		
Negative	13	5	0.63	0.43
Cigarette smoking				
Present	2	5		
Absent	15	4	5.77	0.06
HIV status				
Positive	1	2		
Negative	24	11	0.66	0.33
Site of destruction				
Right lung	9	5		
Left lung	16	8	0.02	0.89
Right ventricular failure				
Present	1	9		
Absent	24	4	18.77	0.01†
Treatment status				
New	9	3		
Retreatment	16	10	1.52	0.27

**Duration of symptoms before presenting to hospital †Statistically significant difference.*

the left lung field with low-pitch bronchial breath sound and coarse crepitations, and with clinical evidences of hyper-inflation of the right lung. On cardio-vascular examination arterial pulse was 108 beats per minute, regular and of full volume, blood pressure was 110/70 mmHg, jugular venous pressure was raised above the angle of the jaw, apex beat was in the 5th left intercoastal space at the anterior *axillary* line. There was S_3 gallop rhythm with grade 4 pansystolic murmur loudest at the left lower sternal edge. Abdominal examination revealed a tender liver six cm below the right coastal margin with a span of 18cm and ascites demonstrable by fluid thrill. An assessment of poorly treated pulmonary tuberculosis complicated by destroyed left lung and cor pulmonale was made.

Investigations showed that all his 3 sputum specimens were scantily positive on microscopy for AFB. Chest X-ray revealed reticulonodular changes on the left lung field with complete shift of the mediastinum to the left side and compensatory emphysema of the right lung field (see Figure 2). Electro-cardiography showed resting sinus tachycardia, right axis deviation, right atria enlargement and incomplete right bundle branch block. Echocardiography showed mild pericardial effusion and dilated right sided chambers especially the right ventricle. There were severe right ventricular diastolic dysfunction and moderate tricuspid regurgitation with estimated pulmonary arterial pressure of 51mmHg. Packed cell volume was 35%, white blood cell count was 11.7 x 10^9 per liter with neutrophil of 73% and lymphocyte of 16%, erythrocyte sedimentation rate was 67mm/hour and HIV screening was non reactive. Liver function tests and electrolyte, urea and creatinine did not show remarkable derangements.

He was placed on intranasal oxygen from oxygen concentrator, intravenous frusemide and oral spironolactone. He was also commenced on retreatment regimen for tuberculosis. He showed minimal improvement in his clinical condition and was bed ridden for about four months before he finally died.

DISCUSSION

Despite the availability of very potent short-course drug regimens for treating and therefore preventing the occurrence of serious complications of tuberculosis, 1.3% of our patients still developed unilateral lung destruction. This is most likely due to poor adherence and high treatment default which were reported in an earlier study from our centre,[9] and may explain why 63.5% of patients with this complication were retreatment cases. This is similar to an earlier report[11] in which 76.4% of patients with tuberculous lung destruction had previously received antituberculous drugs. Another major contributor to the development of unilateral lung destruction in our patients was late presentation to hospital. Ashour et al[2] also reported late presentation and poor compliance with treatment for tuberculosis as the major reasons for lung destruction in patients with pulmonary tuberculosis in Saudi Arabia. The insidious nature of the symptoms of tuberculosis which makes it wax and wane

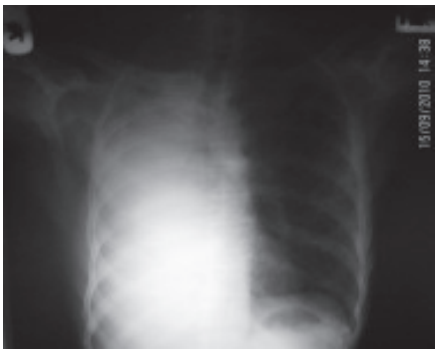

Fig. 1: Chest X-ray showing destruction of the right lung with ipsilateral shift of the mediastinum and compensatory emphysema in the left lung of a patient with unilateral tuberculous destruction of the right lung.

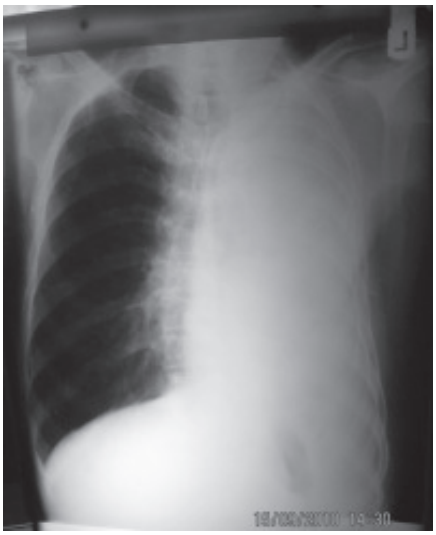

Fig. 2: Chest X-ray showing destruction of the left lung with ipsilateral shift of the mediastinum and compensatory emphysema in the right lung of a patient with unilateral tuberculous destruction of the left lung.

may not always encourage patients to seek early relief.

Similar to earlier reports,[2,4,6,11] unilateral tuberculous lung destruction was commoner in males. The exact reason for this is not clear but may be partly related to higher treatment default among men as reported by earlier review from our centre[9] and elsewhere in Nigeria.[10] Other workers outside Nigeria have also reported that males are at an increased risk of a poor treatment outcome compared to females.[13,14] Most of our patients with unilateral tuberculous lung destruction were below the age of 40 years because majority of the tuberculosis cases occurred in this age group.

Left lung destruction was predominant in our patients, occurring in 66.2% of them. Ashour *et al*[2] and Rajasekaran et al[11] had earlier reported predominantly left destruction in their patients. In both studies, the left main bronchus remained patent in all their tuberculous patients with left lung destruction. This suggests that a different mechanism, other than the proneness of the left main bronchus to extraluminal compression, was responsible for the higher frequency of left lung destruction in tuberculosis. Other mechanisms adduced for the higher frequency of tuberculous lung destruction on the left side include the reduced drainage of pulmonary secretions due the more horizontal and longer course of the left main bronchus as well as its narrower diameter which is approximately 15% lower than that of the right main bronchus.[15] Reduced or absent pulmonary arterial flow to the diseased lung may also favour progression of the disease and eventual lung destruction.[16]

Lung destruction was commoner (87.8%) among those who were seronegative for HIV. The presentation of tuberculosis in HIV patients depends on the degree of immunosuppression.[7] In the early stage of HIV infection, tuberculosis presents typically with upper lobe cavitations, pulmonary fibrosis and shrinkage. The atypical form, with diffuse interstitial or military infiltrates without cavitations, is observed in the advanced stage. One of the most dreaded complications of tuberculous lung destruction, massive haemoptysis, was not common in this study. The exact reason for this is not clear. Most of the other presenting features in our patients were similar to those in patients with non- complicated pulmonary tuberculosis.

The treatment outcome in our patients was similar to findings reported in earlier studies.[9,10,17,18] Overall, 43.2% of the patients did not complete their treatment. This may further aggravate the problems of microbial persistence and MDR-TB associated with tuberculous lung destruction. Unless steps are taken to improve treatment outcomes in our patients, the efficacy of the available standard tuberculosis chemotherapy may be further jeopardized by the development of drug resistance thereby further worsening the outlook for better tuberculosis control in Nigeria.

The mortality rate in this study is unacceptably high despite the availability of effective drugs that can prevent this fatal complication. This is especially so because tuberculosis is curable and its serious complications such as complete unilateral lung destruction can be prevented by early diagnosis, prompt commencement of treatment and strict adherence to treatment regimen. Presence of dyspnoea on mild exertion, right ventricular failure and male gender were significantly associated with increased mortality. Presence of dyspnoea and right ventricular failure suggested the development of severe pulmonary hypertension in the affected patients as demonstrated in illustrative Case 2. However, we did not include data on pulmonary pressures in the analysis because evidences of elevated pulmonary pressures were documented in the chart of very few patients. The higher mortality among the males may be related to the higher rate of occurrence of lung destruction as well as higher treatment default observed among them.

The major limitations to the interpretation of this report are the small number of patients involved and the use of retrospective records with the associated problems of missing data.

In spite of these limitations, this study has shown that unilateral tuberculous lung destruction still accounts for unacceptably high mortality among patients with pulmonary tuberculosis in Nigeria. Late presentation, poor adherence and high treatment default were the identified main factors responsible for this highly fatal but preventable complication in our patients with pulmonary tuberculosis. Early presentation to health facility, prompt diagnosis and strict adherence to standardized treatment will help to prevent the development of this complication and the associated

problems of microbial persistence and MDR-TB in Nigeria.

REFERENCES

1. Adebonajo SA, Adebo OA, Osinowa O, Grillo IA. Management of tuberculous destroyed lung in Nigeria. *J Natl Med Assoc* 1981; **73:** 39–42.
2. Ashour M, Pandya L, Mezraqji A, Qutashaf W, Desouki M, al-Sharif N, *et al*. Unilateral post-tuberculous lung destruction: the left bronchus syndrome. *Thorax* 1990; **45:** 210–212.
3. Conlan AA, Scott EK. Pneumonectomy for benign disease. Deslauries J, Faber LP. Chest surgery clinics of North America. Philadelphia: Saunders; 1999. p. 311–325.
4. Conlan AA, Lucanich JM, Shutz J, Hurwitz SS. Elective pneumonectomy for benign lung disease: modern-day mortality and morbidity. *J Thorac Cardiovasc Surg*. 1995;**110:** 1118–1124.
5. Blyth DF. Pneumonectomy for inflammatory lung disease. *Eur J Cardiothorac Surg*. 2000; **18:** 429–434.
6. Halezeroglu S, Keles M, Uysal A, Celik M, Senol C, Haciibrahimoglu G, *et al.* Factors affecting postoperative morbidity and mortality in destroyed lung. *Ann Thorac Surg*. 1997; **64:** 1635–1638.
7. World Health Organization. Treatment of tuberculosis: guidelines for national programes 3rd edition. Geneva, World Health Organization 2003 (document WHO/CDS/TB2003.313).
8. World Health Organization. Global tuberculosis control: surveillance, planning, financing. WHO report 2007. Geneva, World Health Organization 2007. (document WHO/HTM/TB/ 2007.376).
9. Salami AK, Oluboyo PO. Management outcome of pulmonary tuberculosis: A nine year review in Ilorin. *West Afr J Med* 2003; **22:** 114–119.
10. Daniel OJ, Oladapo OT, Alausa OK. Default from tuberculosis treatment programme in Sagamu, Nigeria. *Nig J Med* 2006; **15:** 63–67.
11. Rajasekaran S, Vallinayagi V, Jeyaganesh D. Unilateral lung destruction: a computed tomographic evaluation. *Ind J Tub* 1999; **46:** 183–187.
12. Saidu SA. Unusual occurrence of destroyed lung syndrome in childhood: a case report. *Nigerian Journal of Surgical Research* 2006; **7:** 46–47.
13. Lienhardt C, Manneh K, Bonchier V, Lahai G, Milligan PJ, McAdam KP. Factors determining the outcome of treatment of adult smear positive tuberculosis cases in the Gambia. *Int J Tuberculosis Lung Dis.* 1998; **2:** 712–8.
14. Diel R, Niemann S. Outcome of tuberculosis treatment in Hamburg: A survey, 1997–2001. *Int J Tuberculosis Lung Dis.* 2003; **7:** 124–31.
15. Groth S, Mortensen J, Lange P, Munch EP, Sorensen PG, Rossing N. Imaging of airways of bronchoscintigraphy for the study of mucociliary clearance. *Thoarx* 1988; **48:** 360–365.
16. Charles BT, Rama R. Lung imaging-unilateral absence or near absence of pulmonary perfusion on lung scanning. *Semin Nucl Med* 1983; **13:** 388–90.
17. Kharsany ABM, Olowolagba A, Abdool Karim SS, Abdool Karim Q. TB treatment outcomes following directly-observed treatment outcomes at an urban out patient specialist TB facility in South Africa. *Tropical Doctor* 2006; **36:** 23–25.
18. Tessema B, Muche A, Bekele A, Reissig D, Emmrich F, Sack U. Treatment outcome of tuberculosis patients at Gondar University teaching hospital, North West Ethiopia. A five-year retrospective study. BMC public Health 2009; 9: 371 epub. Available from http://www.biomedcentral.com/ 1471-2458/9/371 .