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CASE SERIES

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Destroyed lung syndrome in patients with drug-susceptible pulmonary tuberculosis

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Key Clinical Message

Destroyed lung syndrome is a recognized devastating complication of pulmonary tuberculosis (TB) that affects the quality of life of patients. Clinicians in TB-endemic countries should be aware of this complication aiming at prevention. TB control programs should intensify efforts at early detection and treatment and also provide interventions for post-TB complications.

KEYWORDS

cases, complications, destroyed lung syndrome, Ghana, tuberculosis, x-ray

1 | INTRODUCTION

Tuberculosis (TB) is the second leading infectious cause of death estimated to affect about 10.6 million people worldwide.[1] Although TB is preventable and curable, it continues to remain a public health issue affecting the quality of life of patients and claiming the lives of many people, especially in low-income countries. In Ghana, the prevalence of smear-positive TB is estimated at 111 per 100,000 adult population.[2]

There is a well-established national TB control program in Ghana that offers free treatment for TB.[3] Despite this intervention, patients continue to report late to health facilities for treatment. Delays in detecting TB or missed diagnoses by health professionals also lead to delays in initiating treatment. Another factor that impedes the treatment of TB in Ghana is poor adherence to anti-TB medications.[4] These factors culminate in the development of more complications and greater lung damage from the disease process. Globally, most TB control programs emphasize providing TB treatment services. However, post-treatment complications due to lung damage caused by the disease remain largely unrecognized.[5,6] There is thus a paucity of information related to complications of pulmonary TB most especially on destroyed lung syndrome. A destroyed lung is an irreversible condition of total structural destruction of the lung mostly due to chronic or recurrent lung infection.[7] In this case series of three patients seen at a tertiary hospital in Ghana, we aim to raise awareness about this devastating complication of pulmonary TB-destroyed lung syndrome.

2 | CASE 1

2.1 | Case history and examination

A 35-year-old male was seen with complaints of recurrent cough productive of brownish sputum which had been present for 2 months. This was associated with low-grade fever,

dyspnoea on exertion, significant weight loss of more than 10 kg over the past 6 months, drenching night sweats and pleuritic chest pain. There was no history of any chronic medical or surgical conditions. There was no known history of close contact with a person with TB. The patient lives alone in a single-bedroom house. He is a small-scale miner with one and half pack years of cigarette smoking.

Examination revealed a pale and cachectic man with a weight of 44 kg and a body mass index (BMI) of 17.8 kg/m^2. He had axillary and cervical lymphadenopathy and was in obvious respiratory distress as evidenced by nasal flaring. He had a respiratory rate of 26 bpm with an oxygen saturation of 84% on room air which improved to 94% on intranasal oxygen at 4 L/min. His blood pressure was 130/80 mmHg with a pulse rate of 90 bpm. On auscultation, he had reduced air intensity in both lungs with bronchial breath sounds and coarse crepitation on all lung zones. The rest of the systemic examinations were unremarkable.

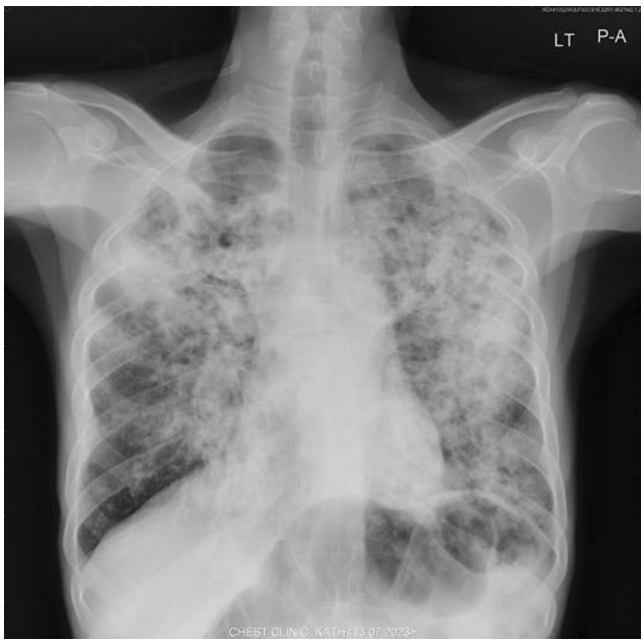

FIGURE 1 Chest x-ray showing markedly reduced lung volumes with heterogeneous opacifications on all lung zones bilaterally and cavitation in the right upper zone.

2.2 | Differential diagnosis, investigations and treatment

His labs were significant for severe microcytic hypochromic anemia (hemoglobin of 5.8 g/dL, mean corpuscular volume of 59.0 fL, mean corpuscular hemoglobin of 17.6 pg) with normal platelets (159×10^3/μL) and white cell counts (5.62×10^3/μL). Erythrocyte sedimentation rate (ESR) was increased at 77 mm/h however renal function test and electrolytes were normal. Hepatitis B and C virus infection and retroviral infection screen were all negative. Sputum microscopy and culture did not reveal any fungal hyphae or bacterial growth. However, nucleic acid amplification test (Gene Xpert) detected *Mycobacterium tuberculosis* (high) rifampicin resistance was not detected. Chest x-ray imaging showed markedly reduced lung volumes with heterogeneous opacifications on all lung zones bilaterally with cavitation in the right upper zone (Figure 1).

The patient was diagnosed with pulmonary TB with destroyed lung syndrome and severe anemia. He was hemotransfused with three units of whole blood and was started on anti-TB medication; intensive phase of oral isoniazid 200 mg daily, oral rifampicin 450 mg daily, oral pyrazinamide 1000 mg daily, oral ethambutol 800 mg daily for 2 months and continuation phase of oral isoniazid 200 mg daily and oral rifampicin 450 mg daily for 4 months. He was also put on oral pyridoxine 50 mg daily for 6 months and hematinic.

2.3 | Outcome and follow-up

The patient could not be weaned off oxygen and was assessed for long-term oxygen therapy. His partial pressure of oxygen was 7.0 kPa and thus was advised to get mobile oxygen at home before discharge. He was followed up monthly at the TB clinic and sputum for acid-fast bacilli and Gene Xpert for *M. tuberculosis* done at months two, five and six of treatment were all negative. The patient still requires home oxygen therapy 3 months post-treatment and is currently unable to carry out his daily activities without support.

3 | CASE 2

3.1 | Case history and examination

A 47-year-old male, with 10 pack years of cigarette smoking who lives in a mining community and is known to have smear-positive pulmonary TB in his 6th month of first-line anti-TB treatment presented with a persistent cough of 7 months duration productive of whitish sputum. Although he was adherent to his anti-TB medications the cough persisted and was relieved temporarily after taking over-the-counter antitussive syrup dextromethorphan. He also had grade 2 dyspnoea per the modified Medical Research Council dyspnoea scale.

On examination, he was cachectic with a weight of 45 kg and a BMI of 17.1 kg/m^2. He was anicteric, not pale, not cyanosed, with no peripheral lymphadenopathy, or pedal oedema. He had stage three finger clubbing. He was afebrile with a temperature of 36.8°C, pulse rate of 75 bpm, blood pressure of 126/86 mmHg, respiratory rate of

22 bpm, and oxygen saturation of 94% on room air. There was a drop in oxygen saturation to 89% on room air after doing a 10 m walk. There was apical flattening on the right chest wall with deviation of the trachea to the right. Chest expansion was reduced on the right with a dull percussion note. On auscultation, breath sound intensity was markedly reduced with bronchial breath sounds and coarse crackles on the right lung zones. The left chest wall however had a hyperresonant percussion note, with mildly reduced air entry on auscultation and vesicular breath sounds on the left lung zones. All other systemic examinations were unremarkable.

3.2 | Differential diagnosis, investigations, and treatment

His laboratory investigations including renal function test and electrolytes, liver function test and complete blood count were essentially normal albeit a mild normocytic normochromic anemia was present (hemoglobin of 10.6 g/dL, mean corpuscular volume of 85.4 fL, mean corpuscular hemoglobin of 27.1 pg). Sputum microscopy and culture did not reveal any fungal hyphae, acid-fast bacilli, or bacterial growth. Chest x-ray showed trachea deviation to the right, reduced rib spaces on the right, and homogenous opacification on the right hemithorax with compensatory hyperinflation on the left. Chest wall ultrasound revealed no pleural effusion. The patient however could not afford a computed tomography scan of the chest. (Figure 2).

At this time he had completed his anti-TB treatment regimen and was managed as post-pulmonary TB with a destroyed right lung. The patient was counseled on the condition and the need to prepare for long-term oxygen therapy. Dietary counseling including the need for a high-protein diet, increased fruit and vegetable intake and adequate daily hydration was given to the patient. He was also advised to take influenza vaccination annually and the pneumococcal vaccine. The patient was counseled to avoid overcrowded places and to minimize any lifestyle or behaviors that predispose him to infection.

3.3 | Outcome and follow-up

As part of his TB care, there was a follow-up sputum for acid-fast bacilli at months 2, 5, and 6 of the treatment period which were all negative. He is currently being followed at the TB clinic with no significant change in his condition. He is unable to return to work. Post-treatment chest x-ray showed no significant improvement compared to the pre-treatment chest x-ray. Repeat chest x-ray revealed mediastinal herniation to the right with crowding of the right ribs and hyperinflation on the left as shown in Figure 2B.

4 | CASE 3

4.1 | Case history and examination

The third patient is a 64-year-old female who presented with 6 months of cough that was productive of brownish sputum associated with unintentional weight loss, night sweats, and low-grade fever. She became alarmed after symptoms became associated with bothersome dyspnea on exertion though there was no orthopnea, paroxysmal nocturnal dyspnoea, or pedal swelling. She had a history

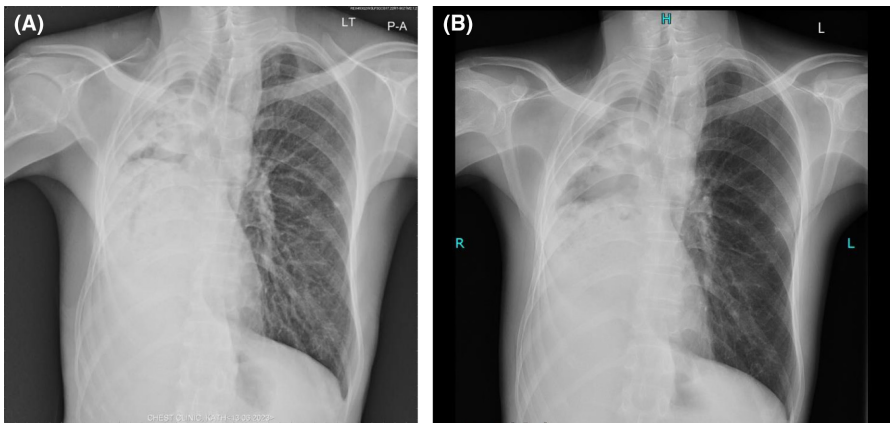

FIGURE 2 (A) Chest x-ray showing trachea deviation to the right, reduced rib spaces on the right, homogeneous opacification on the right hemithorax with compensatory hyperinflation on the left. Pretreatment x-ray for case 2. (B) Post-treatment chest x-ray for case 2 showing no significant improvement compared to the pre-treatment chest x-ray. There is mediastinal herniation to the right with crowding of the right ribs and hyperinflation on the left.

of type 2 diabetes which was well controlled on metformin 500 mg twice daily. She is a lifetime non-smoker and a retired school teacher.

On examination, She was mildly pale, afebrile, anicteric, well hydrated, and was not in any respiratory distress. She had stage three finger clubbing and axillary lymphadenopathy which were discrete and non-tender, the largest measuring 3 cm by 2 cm. Her weight was 55 kg with a BMI of 22 kg/m^2. Her respiratory rate was 18 bpm, her blood pressure was 138/80 mmHg and her pulse rate of 80 bpm. The random blood glucose checked was 8.0 mmol/L. Chest examination showed a tracheal shift to the left, with reduced chest expansion and dull percussion note bilaterally. On auscultation breath sound intensity was reduced globally with bronchial breath sounds in the middle and lower zones bilaterally. Fine end-inspiratory crepitation was heard bilaterally but worse on the left lung zones. Abdominal, cardiovascular, and nervous system examinations were all normal.

4.2 | Differential diagnosis, investigations, and treatment

Her laboratory results including complete blood count, renal function test and electrolytes, and liver function tests were normal, however, her serum globulins were elevated (39.1 g/L). ESR was high at 78 mm/h with a negative screen for Hepatitis B and C virus infection and retroviral infection. Sputum microscopy for acid-fast bacilli was positive (2+) and the culture did not reveal bacterial growth. Gene Xpert detected *M. tuberculosis* (high), rifampicin resistance was not detected.

Chest x-ray showed trachea deviation to the left, reduced rib spaces on the left hemithorax and heterogeneous opacification throughout the lungs but more prominent on the left lung zones and left lung fibrosis as shown in Figure 3.

The patient was diagnosed as a case of drug-susceptible pulmonary TB with destroyed lung syndrome. She was counseled on the diagnosis and the need for medication adherence. She was started on oral isoniazid 300 mg daily, oral rifampicin 450 mg daily, oral pyrazinamide 1200 mg daily, and oral ethambutol 1000 mg daily for 2 months. The continuation phase of oral isoniazid 300 mg daily, and oral rifampicin 450 mg daily has been initiated and will be continued for 4 months. She is also on oral pyridoxine 50 mg daily to counteract the peripheral neuropathy caused by isoniazid. The patient was given dietary and infection prevention counseling and advised to take influenza vaccination annually and the pneumococcal vaccine.

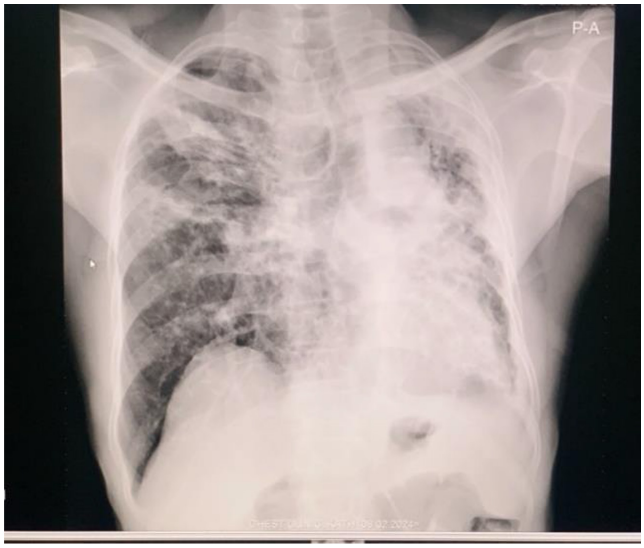

FIGURE 3 Chest x-ray showing trachea deviation to the left, reduced rib spaces on the left hemithorax and heterogeneous opacification throughout the lungs but more prominent on the left lung zones which indicate left lung fibrosis.

4.3 | Outcome and follow-up

She is currently being followed up at the TB clinic and has completed two 2-month intensive phases of treatment and is continuing with the consolidation phase for 4 months. There is improvement in the cough, night sweats, and weight loss. Sputum for acid-fast bacilli after 2 months of treatment was negative.

5 | DISCUSSION

Destroyed lung syndrome is an outcome of irreversible damage to one or both lungs with compromised lung function most commonly caused by pulmonary TB. Other notable causes include cystic bronchiectasis, aspergillosis, emphysema, multiple or extensive lung abscesses, necrotizing pneumonia, empyema thoracis, and mycobacteria other than TB.[8,9] It is a common complication affecting the quality of life of patients and places a huge financial burden on patients and their families.

Late detection of pulmonary TB or delayed treatment coupled with drug non-adherence and drug resistance is a major contributor to developing complications such as destroyed lung syndrome post-TB treatment as it leads to extensive destruction of the lungs.[10] The patients described in this case series had significant symptoms for several months before treatment was initiated. Also, some risk factors that could be identified included small-scale mining and smoking as was in the case of the first and second patients, respectively. Also, our third patient was elderly and had diabetes. Small-scale miners are known to

have a high burden of both silicosis and TB which tends to be complicated.[11]

Destroyed lung syndrome is characterized by pulmonary cavitation, cystic bronchiectasis, loss of lung volume, pleuroparenchymal fibrosis, unilateral near-complete lung parenchymal anomalies, or combinations of the above findings. It is predominantly left-sided in location with contralateral lung parenchymal compensatory hyperinflation manifested as emphysema, and pull of contralateral lung and mediastinal structures to the diseased side (radiologically termed as mediastinal herniation). There is also a reduction of intercostal spaces resulting in the crowding of ipsilateral ribs as seen in Figure 2B. There is a decrease in the diameter of the ipsilateral pulmonary vessels, hypertrophy of the ribs and/or thickening of extrapleural fat.[7,12,13] Patients commonly present with prolonged episodes of easy fatigability on minimal exertion, wasting, recurrent breathlessness, pleuritic chest pains, and a history of chronic infection or pulmonary TB as described in this case series. However, the patient may be asymptomatic or have mild symptoms especially when it is affecting only one lung with compensatory hyperinflation or increased lung volume on the unaffected side. The diagnosis is mostly dependent on the radiological findings.

There is no definitive treatment guideline available for the management of destroyed lung syndrome, however, there is some consensus on clinical standards for the assessment, management and rehabilitation of post-TB lung disease.[14] Treatment is aimed at alleviating the symptoms and improving the quality of life of the patients. Treatment may be either conservative or surgical. Conservative treatment involves the use of bronchodilators such as long-acting beta-2 agonists or long-acting muscarinic antagonists and inhaled corticosteroids may provide some symptomatic relief for respiratory distress.[15] Antifibrotic agents may be considered to reduce the rate of fibrosis. However, no studies have been conducted on the use of antifibrotic agents on such patients. We recommend larger studies such as randomized controlled trials could be considered in endemic countries to ascertain the beneficial effect of antifibrotic agents in these people. Surgical management involving pulmonary resection of the non-functioning lungs may be indicated in the management of destroyed lungs to either resolve or avert complications.[16] Ruan et al., have shown that surgical treatment of tuberculous destroyed lung is associated with reduced rates of dyspnea (as compared to the preoperative rate) prior to discharge and at the end of long-term follow-up.[16] Destroyed lung syndrome may lead to massive hemoptysis, superinfections, empyema thoracis, respiratory failure, and death.[17,18] Although there is overwhelming evidence of lung impairment post-TB,[19] there is a seeming lack of urgency on reporting and management of post-TB complications including destroyed lung syndrome.

Destroyed lung syndrome is a recognized devastating complication of pulmonary TB that adversely affects the quality of life of the patients afflicted. Clinicians in TB-endemic countries should be aware of this complication and strive towards preventing it. TB control programs should intensify efforts at early detection and treatment of TB as well as monitoring and providing interventions for post-TB complications.

AUTHOR CONTRIBUTIONS

Solomon Gyabaah: Conceptualization; data curation; investigation; methodology; project administration; resources; supervision; validation; writing – original draft; writing – review and editing. **Kwadwo Faka Gyan:** Data investigation; methodology; validation; writing – original draft; writing – review and editing. **Harris Osei Mensah:** Conceptualization; data curation; investigation; validation; writing – original draft; writing – review and editing. **Eric Amoako Darkwa:** Conceptualization; investigation; writing – original draft; writing – review and editing. **Michael Asiedu Owiredu:** Data curation; investigation; writing – original draft; writing – review and editing. **Vera Selorm Nyamuame:** Data curation; investigation; project administration; writing – original draft; writing – review and editing. **Gordon Manu Amponsah:** Investigation; validation; writing – original draft; writing – review and editing. **Divine Aseye Amenuke:** Conceptualization; investigation; supervision; writing – original draft; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

Data is available upon reasonable request to the corresponding Author.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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REFERENCES

1. Tuberculosis. https://www.who.int/news-room/fact-sheets/detail/tuberculosis
2. Bonsu F, Addo KK, Alebachew Z, et al. National population-based tuberculosis prevalence survey in Ghana, 2013. *Int J Tuberc Lung Dis*. 2020;24(3):321-328. doi:10.5588/IJTLD.19.0163
3. National TB Programe|Unite To End TB. https://www.tbghana.gov.gh/
4. Danso E, Addo IY, Ampomah IG. Patients' compliance with tuberculosis medication in Ghana: evidence from a Periurban community. *Adv Public Health*. 2015;2015:1-6. doi:10.1155/2015/948487
5. Desta KT, Masango TE, Nkosi ZZ. Performance of the National Tuberculosis Control Program in the post conflict Liberia. *PLoS One*. 2018;13:e0199474. doi:10.1371/journal.pone.0199474
6. Malwe S, Bawiskar D, Wagh V. Tuberculosis and the effectiveness of the revised National Tuberculosis Control Program (RNTCP) to control tuberculosis: a narrative review. *Cureus*. 2023;15(12):e51418. doi:10.7759/CUREUS.51418
7. Yadav S. Destroyed lung syndrome in a young Indian male: a case report. *Cureus*. 2023;15(4):e38174. doi:10.7759/CUREUS.38174
8. Misirlioğlu AK, Bayram S, Kiral H, et al. Factors affecting complication rates of pneumonectomy in destroyed lung. *Turk Gogus Kalp Damar Cerrahisi Derg*. 2018;26(2):272-278. doi:10.5606/TGKDC.DERGISI.2018.14635
9. Kosar A, Orki A, Kiral H, Demirhan R, Arman B. x. *Ann Thorac Surg*. 2010;89(1):226-231. doi:10.1016/j.athoracsur.2009.10.007
10. Bai L, Hong Z, Gong C, Yan D, Liang Z. Surgical treatment efficacy in 172 cases of tuberculosis-destroyed lungs. *Eur J Cardiothorac Surg*. 2012;41(2):335-340. doi:10.1016/J.EJCTS.2011.05.028/2/S1010794011005616O2.GIF
11. Moyo D, Zishiri C, Ncube R, et al. Tuberculosis and silicosis burden in artisanal and small-scale gold miners in a Large Occupational Health Outreach Programme in Zimbabwe. *Int J Environ Res Public Health*. 2021;18(21):11031. doi:10.3390/IJERPH182111031
12. Varona Porres D, Persiva O, Pallisa E, Andreu J. Radiological findings of unilateral tuberculous lung destruction. *Insights Imaging*. 2017;8(2):271-277. doi:10.1007/S13244-017-0547-4
13. Patil S, Narkar S, Raka V, Dahiphale J, Choudhari S, Gondhali G. Destroyed lung as post tuberculosis sequel: A preventable stigma of disease of concern of millennium. *Saudi J Med*. 2023;8:112. doi:10.36348/sjm.2023.v08i03.007
14. Migliori GB, Marx FM, Ambrosino N, et al. Clinical standards for the assessment, management and rehabilitation of post-TB lung disease. *Int J Tuberc Lung Dis*. 2021;25(10):797-813. doi:10.5588/IJTLD.21.0425
15. Rhee CK, Yoo KH, Lee JH, et al. Clinical characteristics of patients with tuberculosis-destroyed lung. *Int J Tuberc Lung Dis*. 2013;17(1):67-75. doi:10.5588/IJTLD.12.0351
16. Ruan H, Liu F, Li Y, et al. Long-term follow-up of tuberculosis-destroyed lung patients after surgical treatment. *BMC Pulm Med*. 2022;22(1):1-8. doi:10.1186/S12890-022-02139-Z/TABLES/6
17. Genovés Crespo M, Agustín Martínez F, Callejas González FJ. Destroyed lung complicated with empyema. *Imaging Med*. 2016;8(4):113-115.
18. Katoto PDMC, Musole P, Maheshe G, et al. A miner with no left lung: extensive pulmonary destruction in delayed effective multi-drug-resistant tuberculosis treatment. *Respir Med Case Rep*. 2020;31:101234. doi:10.1016/J.RMCR.2020.101234
19. Ivanova O, Hoffmann VS, Lange C, Hoelscher M, Rachow A. Post-tuberculosis lung impairment: systematic review and meta-analysis of spirometry data from 14 621 people. *Eur Respir Rev*. 2023;32(168):220221. doi:10.1183/16000617.0221-2022

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