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Endobronchial Blood Clot as the Presenting Feature of Sputum-Negative Pulmonary Tuberculosis in a Young Adult: The Diagnostic Value of Bronchoscopic Tissue Sampling

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Abstract

Background: Pulmonary tuberculosis (TB) remains the foremost infectious cause of mortality in sub-Saharan Africa, yet endobronchial tuberculosis (EBTB)—defined by mycobacterial invasion of the tracheobronchial mucosa—continues to be diagnostically elusive. Sputum smear microscopy, the most widely available TB diagnostic test in resource-limited settings, is frequently negative in EBTB due to paucibacillary mucosal disease, creating a critical diagnostic gap that non-invasive testing cannot bridge. Haemoptysis from EBTB-related mucosal erosion can culminate in an organised endobronchial blood clot—a rare but potentially life-threatening complication that simultaneously obstructs the airway and harbours the very mycobacterial material needed for diagnosis. This dual clinical and diagnostic significance is underappreciated in practice. To the best of our knowledge, we present the first reported case of this presentation from Kenya, managed through a structured multimodal bronchoscopic protocol, with complete WHO-defined treatment success and objective bilateral radiological resolution at six months. **Case Presentation:** A 21-year-old BCG-naive male (70 kg) presented to a tertiary referral hospital in Nairobi, Kenya, with a seven-day history of moderate haemoptysis (~100 mL per episode), drenching night sweats, fever, and progressive weight loss. Clinical examination revealed haemodynamic stability (HR 87 bpm, BP 120/80 mmHg, SpO_2 99% on room air) with absent right lung breath sounds, directly predictive of the complete right main bronchial obstruction confirmed bronchoscopically. Chest radiograph demonstrated bilateral pulmonary infiltrates with left-predominant consolidation. Contrast-enhanced CT revealed bilateral ground-glass opacities, right lower lobe consolidation, and a centrally cavitating left upper lobe nodule (12.2 mm) with adjacent calcific foci. Laboratory

investigations demonstrated moderate anaemia (Hb 9.0 g/dL), mild thrombocytopenia (platelets 120 × 10^9/L, a recognised manifestation of active TB), elevated CRP (55 mg/L), normal coagulation studies (APTT and INR), normal liver function tests, and normal baseline visual acuity (Snellen 6/6 bilaterally; Ishihara colour vision normal). HIV was confirmed negative by both ELISA and PCR. A BioFire FilmArray Respiratory Panel was comprehensively negative for all viral and atypical bacterial respiratory pathogens, systematically excluding alternative infectious aetiologies. Sputum AFB smear microscopy was negative. Flexible bronchoscopy under general anaesthesia (ETT 7.5 mm, 45-minute procedure) performed by Dr. Yashar Najiaghdam (Consultant Intensivist) identified complete right main bronchial obstruction by an organised blood clot, with diffuse mucosal erythema, petechial haemorrhage, and friability consistent with endobronchial TB throughout the tracheobronchial tree. The clot was extracted by high-flow suction, immediately restoring airway patency. BAL GeneXpert MTB/RIF confirmed Mycobacterium tuberculosis at Very Low level (Ct > 28; RIF-sensitive), directly explaining smear negativity through paucibacillary burden. Ziehl-Neelsen staining of the retrieved clot independently confirmed acid-fast bacilli, providing convergent dual-modality bacteriological and histopathological confirmation. **Management and Outcome:** Standard six-month DS-TB therapy was initiated within 48 hours of bronchoscopic confirmation: intensive phase 2HRZE—Isoniazid 300 mg, Rifampicin 600 mg, Pyrazinamide 1750 mg, Ethambutol 1,050 mg, Pyridoxine 25 mg once daily (weight-based per WHO 2022, 60 - 74 kg band)—followed by continuation phase 4HR. Directly Observed Therapy was arranged. Haemoptysis resolved immediately post-bronchoscopy and did not recur. Sputum conversion was confirmed at Month 2. The patient completed the full regimen without adverse drug reactions or interruption, and was declared a WHO-defined treatment success. Follow-up posteroanterior chest radiograph at six months demonstrated near-complete bilateral radiological resolution, with only mild residual reticular markings consistent with post-inflammatory fibrotic change. **Discussion:** Three clinically critical principles emerge from this case. First, a negative sputum smear must never constitute grounds for diagnostic closure in EBTB: paucibacillary mucosal disease produces bacillary burdens below smear detection thresholds, detectable only by sensitive molecular amplification, as confirmed by the Very Low GeneXpert result (Ct > 28) on BAL. Second, the organised endobronchial clot—a haemorrhagic complication requiring urgent extraction—simultaneously constitutes the most diagnostically informative specimen available, yielding AFB on ZN staining when all non-invasive methods have failed. Third, bronchoscopy under general anaesthesia with systematic BAL collection and submission of all retrieved material for GeneXpert and histopathology enables simultaneous therapeutic resolution and definitive diagnosis in a single procedure, a clinically efficient and lifesaving approach in resource-limited environments. **Conclusion:** This case establishes a clear, reproducible protocol for adolescent sputum-negative haemoptysis in TB-endemic sub-Saharan Africa. Negative sputum smear along-

side a compatible clinical triad, characteristic CT findings, and systematic exclusion of alternative pathogens mandate bronchoscopy without delay. A structured approach—general anaesthesia with endotracheal intubation, clot extraction by high-flow suction, BAL for GeneXpert MTB/RIF, and ZN staining of all retrieved endobronchial material—delivers simultaneous airway clearance and bacteriological confirmation. When MTBC is confirmed RIF-sensitive, immediate weight-based 2HRZE/4HR per WHO guidelines achieves clinical cure, sputum conversion, and near-complete radiological resolution, as documented in this case. This integrated protocol should be adopted as standard practice at bronchoscopy-capable facilities across TB-endemic settings.

Keywords

Pulmonary Tuberculosis, Endobronchial Tuberculosis, Haemoptysis, Endobronchial Blood Clot, Bronchoscopy, Sputum-Negative TB, GeneXpert MTB/RIF, BAL, Ziehl-Neelsen, BioFire, Young Adult, General Anaesthesia, 2HRZE/4HR, Kenya, Sub-Saharan Africa

1. Introduction

Haemoptysis—the expectoration of blood originating from the lower respiratory tract—is an uncommon presenting symptom in the paediatric and adolescent population. Its occurrence, however, carries considerable clinical significance, warranting systematic investigation to exclude serious underlying pulmonary pathology. In contrast to adults, where neoplastic disease and chronic structural lung conditions constitute a substantial proportion of cases, haemoptysis in young adults is predominantly attributable to infectious aetiologies, with pulmonary tuberculosis (TB) representing the most important cause in endemic regions [1] [2].

Sub-Saharan Africa, including Kenya, carries one of the highest global TB burdens. According to the World Health Organization (WHO) Global Tuberculosis Report 2023, Kenya recorded an estimated TB incidence of approximately 292 cases per 100,000 population per year [3]. This epidemiological context fundamentally shapes the clinical reasoning process when an adolescent presents with haemoptysis and constitutional symptoms, as the a priori probability of TB is substantially elevated compared to low-burden settings. Nevertheless, the diagnosis of pulmonary TB in this age group is far from straightforward, and clinicians must contend with a broad differential diagnosis encompassing infectious, structural, vascular, and haematological pathologies [4] [5].

Among the diagnostic tools available, flexible bronchoscopy has emerged as a modality of considerable therapeutic and diagnostic importance. It enables direct airway visualization, mechanical clearance of obstructing material, and procurement of specimens—including bronchoalveolar lavage (BAL), bronchial washings, and endobronchial biopsies—for microbiological, molecular, cytological, and histopathological analysis [6]. In cases of endobronchial TB (EBTB), a dis-

crete variant defined by mycobacterial infection of the tracheobronchial mucosa, sputum smear sensitivity is substantially reduced because the bacillary load in expectorated secretions may fall below the detection threshold for smear microscopy despite histopathologically demonstrable mucosal disease [7] [8].

The advent of the GeneXpert MTB/RIF molecular assay has transformed TB diagnostics, providing rapid detection of MTBC and rifampicin resistance profiling within two hours. When applied to bronchoscopically obtained BAL specimens, GeneXpert has demonstrated substantially higher sensitivity than sputum smear in smear-negative pulmonary TB, with reported sensitivities of 70% - 90% [9] [10]. This combination of therapeutic airway management and molecular tissue diagnosis through a single bronchoscopic procedure represents a clinically efficient approach to diagnostically challenging presentations.

We report a 21-year-old male presenting with haemoptysis and constitutional symptoms in Nairobi, Kenya, in whom the sputum AFB smear was negative, CT imaging was suggestive but non-diagnostic, and flexible bronchoscopy identified an organised endobronchial blood clot from which both BAL GeneXpert MTB/RIF and Ziehl-Neelsen histopathological staining confirmed pulmonary tuberculosis with endobronchial involvement. This case is submitted to the Open Journal of Internal Medicine (OJIM) to highlight the diagnostic limitations of sputum-based testing, the indispensable dual role of bronchoscopy, and the imperative of multimodal tissue-based diagnosis in TB-endemic settings.

2. Case Presentation

2.1. Patient Demographics and Clinical History

A 21-year-old male, resident of Nairobi, Kenya, with no prior documented history of pulmonary tuberculosis, anti-tuberculous therapy, or known immunocompromising conditions, presented to a tertiary-level referral hospital in Nairobi, Kenya, with a seven-day history of haemoptysis. Blood loss per episode was estimated at approximately 100 mL, consistent with moderate haemoptysis by standard clinical classification criteria. The haemoptysis was accompanied by fever, unintentional weight loss, and drenching night sweats—a constitutional triad highly consistent with active pulmonary tuberculosis in an endemic setting. Pleuritic chest pain was absent. No household contacts with known or suspected tuberculosis were identified on initial history. Formal contact tracing was initiated through the Kenya National TB Programme following confirmed diagnosis; outcomes of household contact investigation are not available in the current case records. The patient had not received the Bacille Calmette-Guérin (BCG) vaccination. The patient denied cigarette smoking, recreational drug use, and alcohol consumption. Body weight at presentation was 70 kg.

Clinical note on haemoptysis volume: An estimated blood loss of approximately 100 mL per episode over seven days classifies this presentation as moderate haemoptysis, not massive haemoptysis, consistent with an endobronchial rather than a major vascular source. The absence of pleuritic chest pain argues against pleural

disease or pulmonary infarction as primary aetiologies. The presence of night sweats in conjunction with fever and weight loss fulfills three of the four classical constitutional features of active pulmonary tuberculosis and substantially raises the pre-test probability for TB prior to investigation.

2.2. Physical Examination

On presentation, the patient was alert, oriented, and haemodynamically stable, with no signs of acute respiratory distress at rest. Vital signs on admission: temperature 36.7˚C; heart rate 87 beats per minute; blood pressure 120/80 mmHg; respiratory rate 18 breaths per minute; peripheral oxygen saturation 99% on room air. Despite the preserved oxygen saturation, subsequent bronchoscopic findings of complete right main bronchial obstruction indicate that compensatory ventilation via the unaffected left lung was adequate to maintain normal gas exchange at rest.

Active or recent haemoptysis was evident at assessment, consistent with the reported history. Respiratory examination demonstrated absent breath sounds over the right lung field, a finding consistent with complete endobronchial obstruction of the right main bronchus and confirmed by subsequent bronchoscopy. No wheeze, stridor, or crepitations were identified in the left lung field. Extrathoracic examination was unremarkable: no peripheral lymphadenopathy was detected; digital clubbing was absent; clinical pallor was absent despite the documented moderate anaemia (haemoglobin 9.0 g/dL), suggesting a subacute rather than acute haemorrhagic process. No peripheral oedema, cyanosis, or cutaneous stigmata of systemic disease were identified.

2.3. Radiological Findings

Radiological assessment comprised two modalities: a plain anteroposterior (AP) supine chest radiograph obtained at the initial evaluation, followed by a contrast-enhanced computed tomography (CT) examination of the chest and abdomen performed at Rology Medical Kenya, Ltd (http://www.rology.health/) (Table 1).

Table 1. Chronological clinical timeline from symptom onset to treatment completion.

Time Point	Clinical Event and Detail
Weeks before Presentation	Insidious onset of low-grade fever, progressive unintentional weight loss, and a productive cough.
Days before Presentation	Frank haemoptysis (coughing blood) develops, prompting an emergency presentation.
4 September 2025—Day 0	A 21-year-old male presents to a tertiary hospital in Nairobi (body weight 70 kg). Active haemoptysis, fever, night sweats, and weight loss are confirmed. He is referred for urgent CT and laboratory work-up.

Continued

Day 0	CT Chest and Abdomen (Rology Medical Kenya, Ltd): left upper lobe cavitating nodule ~12.2 mm with adjacent calcific foci; bilateral ground-glass opacities; right lower lobe consolidation with air bronchograms. No mediastinal lymphadenopathy.
4-5 September 2025	Hb 9.0 g/dL (moderate anaemia); platelets 120 × 10^9/L (mild thrombocytopenia); APTT and INR: within normal limits; CRP 55 mg/L (↑); ALT 48 U/L, AST 20 U/L, total bilirubin 0.2 mg/dL (all normal); WBC, eosinophils, ESR, LDH normal; IgE 50 IU/mL; HIV negative (ELISA + PCR). BioFire Respiratory Panel: NEGATIVE.
Day 0 - 1	Sputum AFB smear microscopy: NEGATIVE. TB is not excluded on this basis alone.
4 September 2025	Flexible bronchoscopy performed. Findings: diffuse mucosal erythema, friability, and petechial haemorrhage throughout the tracheobronchial tree (consistent with endobronchial TB). Organised blood clot was identified obstructing the right main bronchus.
4 September 2025	Therapeutic: complete clot extracted by high-flow bronchoscopic suction (45-minute procedure). The right main bronchus patency was immediately restored. BAL was collected at the site of maximal mucosal involvement and submitted for GeneXpert MTB/RIF + cytology + AFB smear.
6-7 September 2025	GeneXpert MTB/RIF on BAL cytology: POSITIVE for Mycobacterium tuberculosis complex. Rifampicin resistance status: RIFAMPICIN-SENSITIVE (RIF-S). The organism is susceptible to standard first-line anti-tuberculous therapy.
6-7 September 2025	Histopathology of the retrieved clot specimen: Ziehl-Neelsen staining POSITIVE for acid-fast bacilli. Definitive diagnosis established: pulmonary tuberculosis with endobronchial involvement.
7-8 September 2025	Standard DS-TB therapy initiated: 2HRZE (Isoniazid 300 mg + Rifampicin 600 mg + Pyrazinamide 1750 mg + Ethambutol 1050 mg + Pyridoxine 25 mg, all administered orally once daily) per WHO 2022 and Kenya NTP 5th Edition guidelines. Directly Observed Therapy (DOT) was arranged.
Month 2 Follow-Up	Sputum conversion confirmed. Haemoptysis is fully resolved. Clinical improvement is documented with progressive weight recovery.
Month 6—Treatment Completion	Full DS-TB regimen (2HRZE/4HR) completed without interruption. Haemoptysis did not recur. Weight was restored. Follow-up CXR (PA erect): near-complete bilateral resolution—consolidation resolved; mild residual reticular markings; no active disease. TREATMENT SUCCESS declared per WHO and Kenya NTP criteria. Notified of national surveillance.

Prepared in accordance with the CARE 2013 guideline Item 5 (Timeline).

Chest Radiograph (AP Supine, 4 September 2025): The plain chest film demonstrated bilateral pulmonary infiltrates. The left lung showed heterogeneous mid-to-lower zone consolidation with air bronchograms visible within the opacified parenchyma. The right lung demonstrated relatively preserved aeration with fine reticular infiltrates and lower zone haziness. No pleural effusion, pneumothorax, or mediastinal shift was identified. The cardiomediastinal silhouette was within normal limits, and the trachea was centrally positioned. Notably, the absence of complete right lung collapse on plain radiograph, despite bronchoscopically confirmed complete right main bronchial obstruction, is consistent with the patient's preserved oxygen saturation of 99% on room air, reflecting compensatory ventilation through residual patent airway segments (Figure 1).

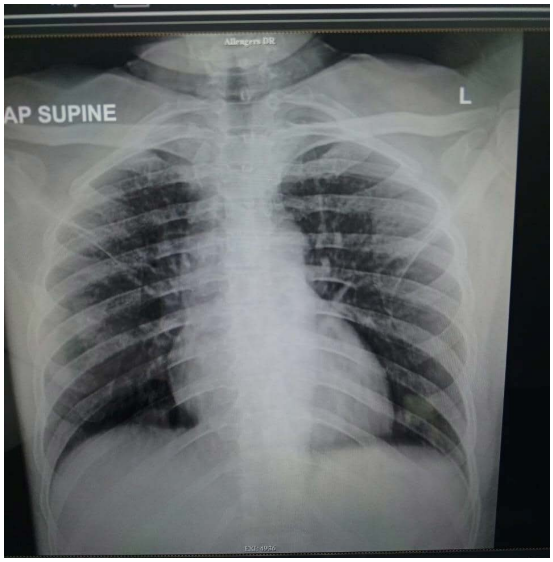

Figure 1. Anteroposterior (AP) supine chest radiograph obtained at presentation (4 September 2025). The radiograph demonstrates bilateral pulmonary infiltrates. The left lung (right side of image, marked "L") shows dense heterogeneous consolidation with patchy opacification in the mid and lower zones, with visible air bronchograms, consistent with active parenchymal infiltration. The right lung (left side of image) demonstrates relative preservation of aeration with fine reticular infiltrates and lower zone haziness. There is no visible pleural effusion or pneumothorax. The cardiomediastinal silhouette is within normal limits with no tracheal deviation, consistent with the absence of complete lobar collapse despite bronchoscopic confirmation of complete right main bronchial obstruction. The relatively preserved right lung aeration on plain radiograph is consistent with the patient's preserved oxygen saturation of 99% on room air at presentation, reflecting adequate compensatory ventilation through the remaining patent airway.

Pulmonary parenchymal assessment demonstrated bilateral ground-glass opacities distributed across both lung lobes, more prominent on the right side. Extensive right lower lobe consolidation with preserved air bronchograms was identified, consistent with active lobar or segmental infective consolidation. Superimposed on this background was a left upper lobe nodule measuring approximately 12.2 mm in maximal diameter, demonstrating central cavitation—a feature that,

in the context of fever, constitutional symptoms, and TB endemicity, raises strong concern for granulomatous infection, though necrotizing bacterial infection cannot be excluded on imaging alone (Figure 2). Adjacent tiny calcific foci, consistent with healed granulomas or prior TB primary complex residua, further support an infectious granulomatous aetiology.

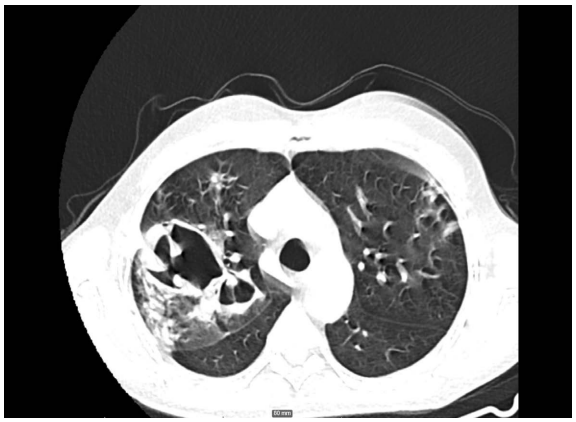

Figure 2. Axial contrast-enhanced CT of the chest (Rology Medical Kenya, Ltd). Extensive right lower lobe consolidation with preserved air bronchograms is demonstrated alongside bilateral ground-glass opacities of varying density. The scale bar at the inferior margin indicates 80 mm. These parenchymal changes are consistent with active post-primary infective consolidation in a TB-endemic setting. The left upper lobe cavitating nodule (~12.2 mm) with adjacent calcific foci is best demonstrated on dedicated axial and coronal reconstructions (available from DICOM archive with patient identifiers removed).

No pleural effusion, pneumothorax, pleural thickening, or pleural-based masses were identified. Mediastinal and hilar structures were within normal limits without significant lymphadenopathy. Of note, the bilateral ground-glass opacities initially characterised as potentially representing viral pneumonitis were subsequently excluded as such by a negative BioFire FilmArray Respiratory Panel, which comprehensively screened for all common viral and atypical bacterial respiratory pathogens; this negative result, combined with positive mycobacterial confirmation, retrospectively supports the interpretation of the ground-glass opacities as representing haematogenous or airway-spread pulmonary TB or a Loeffler-type inflammatory response to mycobacterial antigen—a finding consistent with post-primary TB, in which parenchymal disease predominates over lymph node involvement [10]. Cardiac contour was normal. Abdominal visceral assessment was unremarkable throughout. Full detailed CT findings are available in the contemporaneous radiological report (Rology Medical Kenya, Ltd).

2.4. Laboratory Investigations

Laboratory evaluation demonstrated haemoglobin 9.0 g/dL, consistent with moderate anaemia in a 21-year-old male. In the context of an estimated blood loss of approximately 100 mL per haemoptysis episode over seven days, this degree of anaemia is consistent with cumulative haemorrhagic blood loss compounded by

the anaemia of chronic infection associated with active tuberculosis. Platelet count was 120×10^9/L, representing mild thrombocytopenia. Mild thrombocytopenia is a recognised haematological manifestation of active tuberculosis, attributable to immune-mediated platelet consumption or bone marrow suppression; no clinical bleeding diathesis was documented. C-reactive protein was elevated at 55 mg/L, reflecting an active systemic inflammatory response. White cell count, absolute eosinophil count, ESR, and serum LDH were within normal limits. Pre-procedural coagulation studies, including activated partial thromboplastin time (APTT) and international normalised ratio (INR), were both within normal limits, confirming haemostatic competence and establishing safety for bronchoscopic intervention despite the mild thrombocytopenia documented. Selected laboratory results are summarised in **Table 2**. Serum IgE 50 IU/mL was within the normal reference range (0 - 100 IU/mL), providing no serological support for a helminthic or atopic aetiology. Baseline liver function tests, performed prior to anti-tuberculous therapy initiation, were within normal limits: alanine aminotransferase (ALT) 48 U/L, aspartate aminotransferase (AST) 20 U/L, and total bilirubin 0.2 mg/dL. These values confirmed hepatic competence and established a safe baseline for isoniazid- and rifampicin-containing regimens. HIV status was confirmed negative by two independent methodologies: enzyme-linked immunosorbent assay (ELISA) and polymerase chain reaction (PCR), meeting Kenya national guidelines for dual HIV confirmation in newly diagnosed TB cases [11]. A BioFire FilmArray Respiratory Panel (BioFire Diagnostics, Salt Lake City, USA; FilmArray Respiratory 2 Panel) performed on a respiratory specimen returned a negative result for all targets tested, including influenza A and B, respiratory syncytial virus (RSV), SARS-CoV-2, parainfluenza viruses 1 - 4, adenovirus, human metapneumovirus, rhinovirus/enterovirus, Mycoplasma pneumoniae, Chlamydophila pneumoniae, Legionella pneumophila, and Bordetella pertussis. This comprehensively excluded common viral and atypical bacterial aetiologies for the bilateral ground-glass opacities observed on CT, substantially narrowing the differential and further directing clinical suspicion toward mycobacterial disease. Sputum AFB smear microscopy returned a negative result. GeneXpert MTB/RIF was performed on bronchoalveolar lavage (BAL) fluid obtained at bronchoscopy, submitted to Medlynks By Connect Afya Medical Centre Limited (KENAS Accredited, Nairobi; Pathologist: Dr. Peris W. Thamaini) as a respiratory specimen designated as a sputum sample on the laboratory requisition form—standard practice when BAL is processed via respiratory specimen pathways. The result was POSITIVE for Mycobacterium tuberculosis complex at a semi-quantitative level of Very Low, with rifampicin resistance NOT detected (RIF-sensitive). A Very Low result corresponds to a high cycle threshold (Ct > 28), indicating extremely low bacillary burden—the molecular explanation for the negative sputum smear and the direct evidence of paucibacillary endobronchial disease. A pre-bronchoscopy sputum GeneXpert was not performed as the patient was unable to produce adequate spontaneous sputum, providing additional indication for direct bronchoscopic

sampling.

Table 2. Laboratory investigations at presentation.

Investigation	Result	Reference Range
Sputum AFB Smear	Negative	—
GeneXpert MTB/RIF (BAL/Respiratory Specimen)	**Positive—MTB Detected; Very Low; RIF-Sensitive (Not Resistant)**	—
HIV Serology (ELISA)	Negative (Confirmed)	—
HIV PCR	Negative (Confirmed)	—
Haemoglobin (g/dL)	**9.0 ↓**	13.0 - 17.0
White Cell Count ($\times 10^9$/L)	Within Normal Limits	4.0 - 11.0
Absolute Eosinophil Count ($\times 10^9$/L)	Within Normal Limits	0.0 - 0.5
Platelet Count ($\times 10^9$/L)	**120 ↓**	150 - 400
APTT (Seconds)	Within Normal Limits	25 - 35
INR	Within Normal Limits	0.8 - 1.2
BioFire Respiratory Panel (FilmArray)	NEGATIVE—All Targets	—
CRP (mg/L)	**55 ↑**	<5
ESR (mm/hr)	Within Normal Limits	0 - 15
Serum LDH (U/L)	140	140 - 280
ALT (U/L)	48	7 - 56
AST (U/L)	20	10 - 40
Total Bilirubin (mg/dL)	0.2	0.1 - 1.2
Serum IgE (IU/mL)	50	0 - 100

↑ above reference range; ↓ below reference range. Amber shading = abnormal value. Reference ranges are laboratory-specific and may vary.

2.5. Bronchoscopic Procedure, Findings, and Specimen Retrieval

In view of the active haemoptysis, CT findings, and non-diagnostic sputum smear result, flexible bronchoscopy was performed by Dr. Yashar Najiaghdam, Consultant Intensivist, at a tertiary referral hospital in Nairobi, Kenya. The procedure was conducted under general anaesthesia (GA) with endotracheal intubation (endotracheal tube size 7.5 mm, cuffed) and standard intraoperative monitoring, including continuous pulse oximetry, capnography, non-invasive blood pressure, and electrocardiography. General anaesthesia was selected over conscious sedation in view of: the active haemoptysis with risk of sudden airway compromise; the requirement for definitive airway control and contralateral lung protection; and the anticipated need for high-flow suction and complete mechanical clot ex-

traction without patient movement or laryngospasm. Informed consent was obtained from the patient and his legal guardian prior to the procedure. Pre-procedural coagulation studies (APTT and INR, both within normal limits) confirmed haemostatic competence; the mild thrombocytopenia (platelets 120×10^9/L) was reviewed and deemed clinically acceptable for proceeding without prophylactic platelet transfusion.

Systematic bronchoscopic examination of the trachea and bilateral airways demonstrated diffuse mucosal erythema, prominent submucosal vascular engorgement, superficial mucosal irregularity, and areas of petechial haemorrhage throughout the tracheobronchial tree (Figure 3). The carina showed broadened architecture with blunting of the normal sharp carinal angle and hyperaemic mucosa, consistent with subcarinal inflammatory disease. These endobronchial findings, taken together, are consistent with active endobronchial tuberculosis and provide the plausible anatomical substrate for recurrent airway haemorrhage and secondary clot formation.

Figure 3. Gross macrophotograph of the organised endobronchial blood clot retrieved via flexible bronchoscopy from the right main bronchus. The specimen is displayed on sterile gauze held in sterile gloves. The clot demonstrates a dark red-brown, elongated, and twisted morphology consistent with an organised thrombus. Ziehl-Neelsen staining of the submitted specimen demonstrated the presence of acid-fast bacilli, establishing the histopathological diagnosis of tuberculosis. Patient consent was obtained for image acquisition and publication.

Systematic bronchoscopic examination of the trachea and bilateral airways revealed diffuse mucosal erythema, petechial haemorrhage, and submucosal vascular engorgement consistent with active endobronchial inflammatory disease. Eval-

uation of the right main bronchus demonstrated complete luminal obstruction by an organised blood clot, fully occluding the bronchial lumen and preventing visualisation of distal airways. This finding was consistent with the absent breath sounds identified on pre-procedural clinical examination. The clot was extracted under direct bronchoscopic vision using high-flow bronchoscopic suction applied directly to the proximal surface of the organised clot, with stepwise progressive aspiration and mobilisation. The organised and fibrinised consistency of the clot permitted successful en bloc retrieval by sustained suction without fragmentation. In cases where suction alone is insufficient for organised clot extraction, bronchoscopic biopsy forceps or cryoprobe-assisted extraction represent alternative modalities [6]; however, in this case, suction was effective, and no additional instrumentation was required. Immediate restoration of right main bronchus luminal patency was achieved following extraction, with subsequent visualisation of the distal bronchial tree confirming clearance and no residual endobronchial pathology. Bronchoalveolar lavage (BAL) was performed at the site of maximal mucosal involvement following clot extraction; aspirated material was submitted for GeneXpert MTB/RIF molecular testing and cytological examination. The procedure was completed without complications. Total procedural duration was 45 minutes. The retrieved clot was placed on sterile gauze and photographed immediately prior to histopathological submission (**Figure 3**).

2.6. Histopathological and Microbiological Findings

The retrieved endobronchial clot was submitted in formaldehyde fixative to Medlynks By Connect Afya Medical Centre Limited (KENAS-accredited), Nairobi, for histopathological and special stain analysis under the reporting pathologist, Dr. Peris W. Thamaini.

Haematoxylin and Eosin (H&E) Staining: Histopathological examination of the retrieved clot was performed using haematoxylin and eosin staining. A detailed architectural description of the tissue findings was not available in the documented pathology report at the time of manuscript preparation and is therefore not included here. No fabricated or inferred histopathological characterisation is presented.

Special Stains for Fungal Organisms: Periodic acid-Schiff (PAS) staining was negative for fungal elements. Grocott's methenamine silver (GMS) staining was negative for fungal elements. These results effectively exclude fungal infection as a contributing aetiology within the retrieved specimen.

Acid-Fast Bacillus Staining (Ziehl-Neelsen): Ziehl-Neelsen staining of the submitted clot specimen identified acid-fast bacilli (AFB), providing direct histopathological evidence of mycobacterial infection within the endobronchial material.

Molecular Microbiological Analysis (GeneXpert MTB/RIF): GeneXpert MTB/RIF molecular testing was performed on the bronchoalveolar lavage (BAL) specimen collected at bronchoscopy (**Figure 4**), processed at Medlynks By Connect Afya Medical Centre Limited (KENAS-accredited). The result demonstrated

that Mycobacterium tuberculosis was detected at a very low bacterial load, with rifampicin resistance not detected (RIF-sensitive). The very low quantitative signal corresponds to a high cycle threshold (Ct > 28), consistent with the paucibacillary nature of endobronchial disease and the mechanistic explanation for the negative sputum smear microscopy.

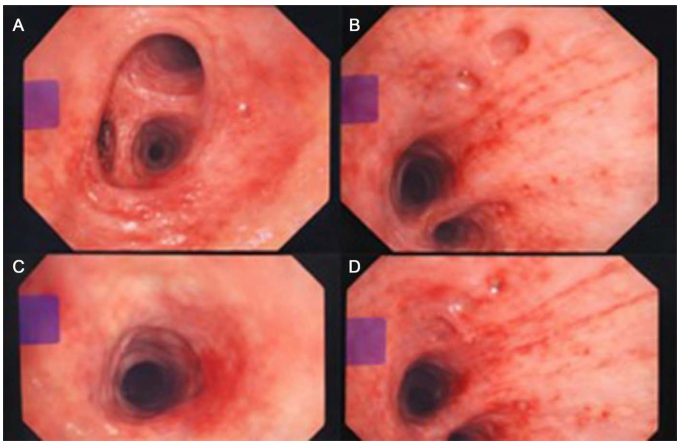

Figure 4. Flexible bronchoscopy: four-panel composite image. (A) Carina demonstrates broadened architecture with erythematous, hyperaemic mucosa and a blunted carina angle, consistent with subcarinal inflammatory disease. (B) Segmental airways showing diffuse mucosal erythema, prominent submucosal vascular engorgement, and superficial mucosal irregularity consistent with active endobronchial involvement. (C) The distal airway lumen is surrounded by diffusely erythematous, friable mucosa with petechial haemorrhagic foci, consistent with active mucosal ulceration and the source of airway bleeding. (D) Further segmental airway view confirming extensive mucosal hyperaemia, submucosal vascular prominence, and haemorrhagic mucosal changes. All four panels demonstrate findings consistent with diffuse endobronchial tuberculosis, providing the anatomical and pathological substrate for recurrent airway haemorrhage and secondary organised clot formation in this case.

Final Diagnosis: The convergence of positive Ziehl-Neelsen staining on the endobronchial clot specimen and positive GeneXpert MTB/RIF (very low bacterial load, RIF-sensitive) on the BAL sample, interpreted in conjunction with the clinical presentation, bronchoscopic findings, and CT imaging, established a confirmed diagnosis of pulmonary tuberculosis with endobronchial involvement. Mycobacterial liquid culture and comprehensive phenotypic drug susceptibility testing for the full first-line panel were not performed at the time of reporting; however, the GeneXpert RIF-sensitive result provided an adequate molecular basis for the initiation of standard drug-sensitive TB (DS-TB) therapy.

2.7. Diagnosis, Management, and Outcome

The integrated clinical, radiological, and dual-modality bronchoscopic evidence—positive GeneXpert MTB/RIF on BAL at Very Low level (Ct > 28, RIF-sensitive) and positive ZN staining on the retrieved endobronchial clot—established an unambiguous diagnosis of pulmonary tuberculosis with endobronchial involvement

in a previously undiagnosed, BCG-naive, 21-year-old young adult. The absence of sputum AFB positivity throughout the diagnostic evaluation was a defining feature of this case; bronchoscopic tissue and molecular analysis served as the sole definitive diagnostic modalities.

2.7.1. Clinical Protocol: Bronchoscopic Management of Endobronchial Clot

The bronchoscopic procedure was conducted under the following protocol at a tertiary referral hospital in Nairobi, Kenya (Table 3):

Table 3. Step-by-step bronchoscopic management protocol as applied in this case.

Protocol Step	Detail and Rationale
Pre-Procedure Assessment	Date: 4 September 2025. Duration: 45 minutes. Patient weight: 70 kg. Operator: Dr. Yashar Najiaghdam, Consultant Intensivist. Haematological profile reviewed: Hb 9.0 g/dL (moderate anaemia); Platelets 120 × 10^9/L (mild thrombocytopenia); APTT and INR within normal limits. Coagulation competence confirmed; platelet transfusion not required. Consent obtained from the patient and legal guardian.
Anaesthesia Choice	General anaesthesia (GA) with endotracheal intubation (ETT 7.5 mm cuffed). Monitoring: pulse oximetry, capnography, NIBP, and ECG. GA selected over conscious sedation: active haemoptysis; definitive airway control required; complete clot extraction needed; contralateral lung protection.
Airway Inspection	Systematic bronchoscopic inspection of the trachea, carina, and bilateral main bronchi. Carina: broadened with a blunted angle and erythematous mucosa (subcarinal inflammation). Bilateral airways: diffuse mucosal erythema, petechial haemorrhage, submucosal vascular engorgement, and friability—consistent with active endobronchial TB throughout.
Clot Identification	COMPLETE obstruction of the right main bronchus confirmed bronchoscopically. Clot: dark red-brown, elongated, firm, twisted morphology, consistent with organised and fibrinised thrombus. Distal airways are not visualisable pre-extraction.
Clot Extraction	Extraction by high-flow bronchoscopic suction with progressive mobilisation under direct vision. Clot was retrieved intact. Right main bronchus patency was immediately restored. Distal airways were visualised and confirmed clear. Procedure was completed without complications.
BAL Collection	Bronchoalveolar lavage (BAL) was performed at the site of greatest mucosal involvement and submitted to Medlynks by Connect Afya Medical Centre Limited (KENAS Accredited, Nairobi) for: GeneXpert MTB/RIF molecular testing; cytological examination; AFB smear; mycobacterial liquid culture.

Continued

Specimen Submission	Retrieved clot submitted in formaldehyde fixative to Medlynks (KENAS-accredited) for: H&E staining (architectural description not available at the time of reporting); ZN staining (POSITIVE for AFB); PAS stain (NEGATIVE for fungal elements); GMS stain (NEGATIVE for fungal elements). Pathologist: Dr. Peris W. Thamaini.
Post-Procedure	The procedure was completed without immediate complications. The patient was monitored in recovery under the standard post-GA observation protocol. Haemoptysis did not recur following clot extraction. The step-by-step procedural protocol is detailed in Table 3.

GA = General Anaesthesia; BAL = Bronchoalveolar Lavage; APTT = Activated Partial Thromboplastin Time; INR = International Normalised Ratio; AFB = Acid-Fast Bacilli; H&E = Haematoxylin and Eosin; ZN = Ziehl-Neelsen; KENAS = Kenya Accreditation Service.

2.7.2. Anti-Tuberculous Treatment Protocol

Anti-tuberculous therapy was initiated within 48 - 72 hours of GeneXpert confirmation of rifampicin-sensitive MTBC, in accordance with the WHO 2022 Treatment of Drug-Susceptible Tuberculosis guidelines [12] and the Kenya National TB Programme 5th Edition guidelines [11]. Drug doses are detailed in Table 4.

Table 4. Anti-tuberculous drug dosing for a 70 kg patient.

Drug	Dose (mg/kg)	Actual Dose (70 kg)	Maximum	Rationale/Notes
Isoniazid (H)	5 mg/kg/day	300 mg daily	300 mg	Capped at a maximum of 300 mg. Bactericidal. Pyridoxine 25 mg is co-prescribed to prevent peripheral neuropathy [12].
Rifampicin (R)	10 mg/kg/day	600 mg daily	600 mg	Capped at a maximum of 600 mg. Bactericidal and sterilizing. Take on an empty stomach.
Pyrazinamide (Z)	25 mg/kg/day	1750 mg daily	2000 mg	Within the weight-band dose. Sterilizing activity in an acidic intracellular environment. Intensive phase only.
Ethambutol (E)	15 mg/kg/day	1050 mg daily	1600 mg	Within the weight-band dose. Bacteriostatic. Baseline visual acuity was assessed pre-commencement [12].
Pyridoxine (B6)	—	25 mg daily	—	Co-prescribed with isoniazid. Prevents INH-associated peripheral neuropathy; this is particularly important in patients with nutritional deficits and weight loss [12].

Intensive phase: 2HRZE (2 months). Continuation phase: 4HR (4 months). Total duration: 6 months. Dosing per WHO 2022 weight-band 60 - 74 kg guidance [12] and Kenya NTP 5th Edition [11]. INH = Isoniazid; DS-TB = Drug-Sensitive Tuberculosis.

The patient was notified to the Kenya National TB Programme (TIBU surveillance system) following diagnostic confirmation. Directly Observed Therapy (DOT) was arranged for the continuation phase to ensure adherence. Clinical response was favourable: haemoptysis resolved completely following bronchoscopic clot extraction and did not recur; sputum conversion was confirmed at the Month 2 review; progressive weight recovery was documented. The patient completed the full six-month first-line DS-TB regimen (2HRZE/4HR) without interruption. No adverse drug reactions necessitating regimen modification were recorded. Baseline hepatotoxicity monitoring confirmed normal liver function (ALT 48 U/L, AST 20 U/L, total bilirubin 0.2 mg/dL) (Table 5). Baseline visual acuity assessment prior to initiation of ethambutol demonstrated Snellen acuity of 6/6 bilaterally (equivalent to 20/20), with no colour vision abnormality detected on Ishihara plate testing. No features of optic neuropathy were identified. Ethambutol was commenced at the standard weight-based dose of 1,050 mg daily. Radiological follow-up was performed at treatment completion. A posteroanterior (PA) erect chest radiograph at the six-month review demonstrated marked bilateral improvement compared with the admission radiograph: near-complete resolution of the left-sided consolidation, resolution of bilateral ground-glass opacities and lower zone haziness, with only mild residual reticular markings in the lower zones bilaterally, consistent with post-inflammatory fibrotic change rather than active disease (Figure 5). No new pulmonary infiltrates, pleural effusion, or mediastinal abnormality were identified. At treatment completion, the patient was declared a treatment success in accordance with WHO DS-TB treatment outcome definitions and Kenya NTP 5th Edition outcome criteria.

Table 5. Treatment monitoring protocol and outcomes.

Monitoring Parameter	Timing	Finding/Result	Action Taken
Baseline LFTs (Pre-Treatment)	Pre-treatment	ALT 48 U/L (normal); AST 20 U/L (normal); Total bilirubin 0.2 mg/dL (normal)	All within normal limits. It is safe to initiate isoniazid and rifampicin. No dose modification is required
Visual Acuity (Snellen Chart)	Pre-ethambutol commencement (04 September 2025)	Snellen acuity 6/6 bilaterally (20/20); no color vision abnormality on Ishihara plate testing	Ethambutol was commenced at the standard dose. An ophthalmology review is to be arranged if symptoms develop during therapy
Sputum Smear Conversion	Month 2 (end of the intensive phase)	NEGATIVE—conversion confirmed	Proceeded to the continuation phase (4HR) as planned
Clinical Assessment: Haemoptysis	Following bronchoscopy; Month 2; Month 6	Resolved completely post-bronchoscopy; no recurrence	No further intervention is required

Continued

Weight Monitoring	Monthly	Progressive weight recovery was documented	Confirmed nutritional improvement in therapy
TB Notification	Day of diagnostic confirmation	Notified to Kenya NTP surveillance (TIBU system)	Contact tracing initiated; DOT arranged for the continuation phase
Radiological Follow-Up (CXR PA Erect)	Month 6	Marked bilateral improvement; near-complete resolution of consolidation; mild residual reticular markings; no active infiltrates or effusion	Consistent with treatment success; post-inflammatory fibrotic changes expected
WHO Treatment Outcome	Month 6 (completion of therapy)	Treatment success declared	Full course completed; CXR improvement documented (Figure 5); outcome recorded in the NTP register

LFT = Liver Function Test; ALT = Alanine Aminotransferase; AST = Aspartate Aminotransferase; NTP = National TB Programme; DOT = Directly Observed Therapy; TIBU = TB Information and Notification System (Kenya); WHO = World Health Organization.

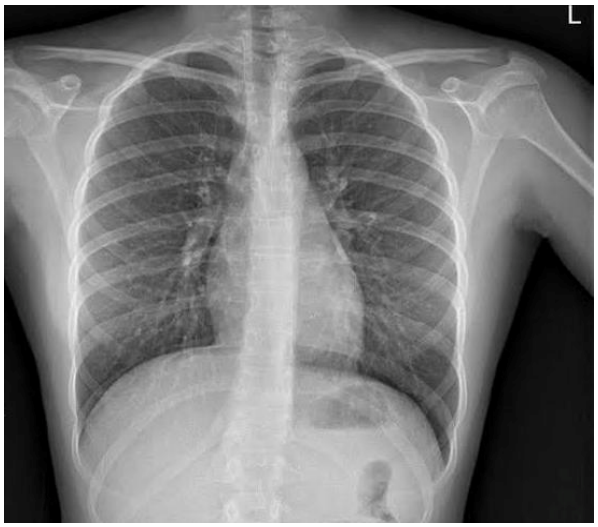

Figure 5. Posteroanterior (PA) erect chest radiograph obtained at six-month post-treatment review, following completion of the standard DS-TB regimen (2HRZE/4HR). In comparison with the admission anteroposterior supine chest radiograph (Figure 1), there is marked bilateral radiological improvement. The left lung demonstrates near-complete resolution of the previously identified mid-to-lower zone consolidation, with only mild residual reticular markings in the lower zone consistent with post-inflammatory fibrotic change. The right lung similarly shows marked improvement, with resolution of the previously identified lower zone haziness and reticular infiltrates. No active consolidation, pleural effusion, or pneumothorax is identified. The cardiomediastinal silhouette remains normal, and the diaphragms are well defined bilaterally. These radiological findings are consistent with a favourable treatment response and support the designation of treatment success in accordance with WHO DS-TB outcome criteria. Patient consent was obtained for image acquisition and publication.

3. Discussion

3.1. Haemoptysis in Adolescents: Differential Diagnosis and the Role of Imaging

Haemoptysis in the adolescent population demands systematic investigation. In sub-Saharan African endemic settings, pulmonary tuberculosis warrants primary consideration given both its prevalence and the severity of consequences if undiagnosed [3]. The clinical tetrad of haemoptysis, fever, weight loss, and night sweats in a 21-year-old Nairobi resident establishes a compelling pre-test probability for TB, appropriately focusing the diagnostic pathway [4]. Night sweats, while non-specific, are a classical constitutional feature of active mycobacterial infection and, in combination with the remaining symptoms, substantially elevate pre-test probability for TB prior to any investigation.

The differential diagnosis is broad and must be approached systematically (Table 6). Infectious causes—TB, bacterial pneumonia with cavitation, pulmonary abscess, paragonimiasis, and fungal infection—predominate in the tropics. Structural causes include bronchiectasis, arteriovenous malformation, congenital pulmonary sequestration, and Rasmussen aneurysm, a vascular complication of cavitatory TB [13]. Haematological disorders, including coagulopathy and thrombocytopenia, must be excluded through laboratory assessment; in the present case, normal platelet count and absence of clinical bleeding diathesis effectively argued against a primary haematological cause. The normal absolute eosinophil count and serum IgE (50 IU/mL) argued strongly against helminthic disease as the primary aetiology, a differential of particular relevance in the East African context [14].

Table 6. Structured differential diagnosis with supporting and excluding features and final diagnostic disposition.

Diagnosis	Supporting Features	Features Against	Disposition
Pulmonary TB (EBTB)	Endemic region; haemoptysis; fever; weight loss; night sweats; cavitating LUL nodule + calcific foci; endobronchial mucosal changes; GeneXpert BAL +ve (Very Low, RIF-S); ZN +ve; BioFire negative (excludes viral/atypical mimics)	Sputum AFB negative; no mediastinal LN	CONFIRMED—dual molecular + histopathological confirmation
Bacterial Lung Abscess	Cavitating lesion; fever; elevated CRP	No leukocytosis; no putrid sputum; no anaerobic risk; GeneXpert MTBC +ve	EXCLUDED by GeneXpert and clinical profile
Pulmonary Fungal Infection	Cavitating nodule; endemic region	Normal IgE; no immunocompromise; GeneXpert MTBC +ve	EXCLUDED—low probability; molecular diagnosis obtained

Continued

Paragonimiasis/Parasitic Cyst	Cavitating lesion; adjacent calcification; East Africa	Normal eosinophils; normal IgE; no dietary exposure; GeneXpert MTBC +ve	EXCLUDED by normal eosinophils, IgE, and molecular diagnosis
Primary Lung Malignancy	Cavitating nodule	Age 16 years; no smoking; calcific foci; GeneXpert MTBC +ve	EXCLUDED—age, epidemiology, and diagnostic confirmation
Coagulopathy/Haematological	Haemoptysis	Mild thrombocytopenia (platelets 120 × 10^9/L) noted; APTT and INR within normal limits, confirming haemostatic competence; no clinical bleeding diathesis; consistent with TB-associated immune-mediated thrombocytopenia; GeneXpert MTBC confirmed.	EXCLUDED as primary cause—mild thrombocytopenia attributed to a TB-associated immune-mediated mechanism

EBTB = Endobronchial Tuberculosis; LUL = Left Upper Lobe; LN = Lymphadenopathy; MTBC = Mycobacterium Tuberculosis Complex; BAL = Bronchoalveolar Lavage; ZN = Ziehl-Neelsen.

CT of the chest is the cornerstone of radiological evaluation in haemoptysis. The combination of a cavitating left upper lobe nodule with adjacent calcific foci and right lower lobe consolidation, as observed in this case, is broadly consistent with an established or evolving post-primary granulomatous process [15]. The absence of mediastinal lymphadenopathy, while atypical for primary TB in younger patients, is consistent with post-primary disease in which parenchymal involvement predominates [10].

Of particular diagnostic value in this case was the BioFire FilmArray Respiratory Panel—a multiplex molecular assay that simultaneously detects over 15 viral and atypical bacterial respiratory pathogens within approximately one hour [16]. The negative result comprehensively excluded influenza A and B, RSV, SARS-CoV-2, parainfluenza viruses 1 - 4, adenovirus, human metapneumovirus, rhinovirus/enterovirus, Mycoplasma pneumoniae, Chlamydophila pneumoniae, Legionella pneumophila, and Bordetella pertussis as aetiologies for the bilateral ground-glass opacities identified on CT. This single investigation effectively resolved a central diagnostic ambiguity created by the non-specific CT appearances, eliminating the most common infectious mimics and directing clinical attention decisively toward mycobacterial disease. The bilateral GGOs, initially characterised as potentially representing viral pneumonitis in the radiological report, are in retrospect more consistently explained as haematogenous or airway-spread pulmonary TB activity, or alternatively as a Loeffler-type inflammatory response to mycobacterial antigen within the pulmonary vasculature. The integration of mul-

tiplex respiratory pathogen panels into the initial workup of adolescent haemoptysis with indeterminate CT ground-glass opacities represents a diagnostically efficient strategy in TB-endemic settings where viral and mycobacterial presentations may be clinically and radiologically indistinguishable.

3.2. Multiplex Respiratory Pathogen Testing as a Diagnostic Adjunct in TB Workup

The BioFire FilmArray Respiratory Panel performed in this case returned a comprehensive negative result for all viral and atypical bacterial respiratory pathogens, including influenza A and B, respiratory syncytial virus, SARS-CoV-2, parainfluenza viruses, adenovirus, human metapneumovirus, rhinovirus/enterovirus, Mycoplasma pneumoniae, Chlamydophila pneumoniae, Legionella pneumophila, and Bordetella pertussis. This single result, delivered within approximately one hour of specimen collection, systematically eliminated the most prevalent alternative infectious causes of bilateral ground-glass opacities and pulmonary consolidation in an adolescent presenting in a Nairobi tertiary facility [16]. Its clinical value in this case was twofold: it excluded co-infection with any of the listed pathogens that might have provided a competing or complicating diagnosis, and it substantially raised the post-test probability of TB as the primary explanation for the CT parenchymal changes.

The use of multiplex respiratory pathogen panels as an adjunct in the diagnostic workup of suspected TB in endemic settings has not been systematically evaluated in the published literature, and their inclusion in TB diagnostic algorithms has not yet been incorporated into WHO or Kenya NTP guidelines. This case provides an illustrative precedent for their potential utility. In resource-rich settings, the BioFire panel has been widely adopted for rapid exclusion of viral respiratory infections; in TB-endemic settings, its value may extend further—providing a rapid, high-specificity exclusion of competing diagnoses that would otherwise require time-consuming serial investigations. This is particularly important when CT findings such as bilateral ground-glass opacities carry a broad differential diagnosis that includes both TB and viral pneumonitis.

It is important to note that the BioFire panel does not test for Mycobacterium tuberculosis and is not a TB diagnostic tool per se; its role in this case was purely exclusionary. Its negative result did not confirm TB but removed plausible alternatives, enabling clinicians to proceed with confidence toward bronchoscopy as the next diagnostic step. The cost and infrastructure requirements of multiplex PCR panels remain a barrier to routine use at lower-tier facilities in sub-Saharan Africa; however, at bronchoscopy-capable tertiary centres—where the clinical decision-making stakes are highest—the addition of a multiplex pathogen panel to the haemoptysis workup algorithm merits prospective evaluation. Future multicentre studies in TB-endemic settings should examine the diagnostic efficiency gains and cost-effectiveness of incorporating BioFire or equivalent multiplex panels into structured haemoptysis diagnostic protocols.

3.3. Plain Chest Radiograph versus CT in the Evaluation of Haemoptysis: Complementary Roles

The availability of both a plain anteroposterior chest radiograph and a contrast-enhanced CT of the chest in this case provides an instructive opportunity to examine the complementary diagnostic contributions of these two modalities in the evaluation of adolescent haemoptysis. The admission chest radiograph (**Figure 1**) demonstrated bilateral pulmonary infiltrates with left-predominant consolidation, consistent with an active bilateral infective process. However, it did not demonstrate the left upper lobe cavitating nodule with adjacent calcific foci that was subsequently identified on CT—a finding that provided the key imaging evidence of granulomatous infection and substantially focused the clinical diagnosis toward TB. This diagnostic gap between plain radiography and CT reflects a well-documented limitation of plain chest films in detecting nodular pulmonary lesions less than 15 mm in diameter, particularly in the upper lobes, where overlying bony and vascular structures reduce conspicuity [15].

The CT chest additionally contributed two findings that could not be inferred from the plain radiograph alone: the characterisation of the right lower lobe consolidation as having preserved air bronchograms (a feature suggesting lobar infiltration rather than complete collapse, consistent with the normal SpO_2 of 99%), and the absence of mediastinal or hilar lymphadenopathy (a finding arguing against primary TB and supporting post-primary disease). The plain radiograph, by contrast, provided a rapid, widely accessible overview of the global pulmonary burden of disease, demonstrated the bilateral nature of the infiltrates at initial assessment, and forms the baseline against which radiological progression or improvement can be measured. The six-month follow-up PA erect chest radiograph (**Figure 5**) directly documented treatment response in a format that is readily interpretable, widely available, and sufficient for WHO treatment outcome documentation purposes—without the radiation exposure, contrast requirements, or resource burden of repeat CT imaging.

The clinical lesson is clear: in resource-limited settings, CT of the chest should be reserved for cases where plain radiography has identified pulmonary abnormalities but failed to characterise them adequately for diagnostic decision-making, or where clinical findings—such as absent unilateral breath sounds suggestive of bronchial obstruction—raise specific structural questions that plain film cannot resolve. In the present case, the combination of plain radiograph for initial assessment and CT for structural characterisation represented an optimal and cost-effective imaging strategy. Follow-up plain radiography at treatment completion provided sufficient radiological documentation of treatment response. This stepwise imaging approach should be incorporated into institutional haemoptysis protocols in TB-endemic settings as a standard of care.

3.4. Cavitary Lung Lesions: Radiological and Clinical Considerations

Cavitatory pulmonary nodules represent one of the most diagnostically challeng-

ing categories of pulmonary pathology. Infectious causes predominate in endemic regions, principally pulmonary tuberculosis, bacterial abscess, and endemic mycoses [17]. Non-infectious causes—granulomatosis with polyangiitis, pulmonary infarction, and primary malignancy—were considered less likely given the patient's age, epidemiology, and molecular diagnostic confirmation.

The morphological features of the nodule in this case—12 mm, centrally cavitating, with adjacent calcific satellite foci—are more consistent with infectious or granulomatous pathology than malignancy. The Fleischner Society and subsequent literature have identified wall thickness, cavity margin characteristics, and satellite lesions as key discriminating features [18]. The calcific foci adjacent to the nodule are a recognised hallmark of healed TB granulomas and prior mycobacterial disease activity, providing supportive indirect evidence for an infectious aetiology [15].

3.5. Endobronchial Tuberculosis and Clot Formation: Pathogenesis

Endobronchial tuberculosis (EBTB) is defined as mycobacterial infection of the tracheobronchial tree and accounts for 10% - 40% of pulmonary TB cases in some series, with higher prevalence among younger patients [8] [19]. Pathogenesis involves mucosal extension from contiguous parenchymal lesions via lymphatic or haematogenous routes, producing the spectrum of bronchoscopic appearances observed in this case: erythema, friability, petechial haemorrhage, and mucosal ulceration.

Haemorrhage in EBTB arises from erosion of the mucosal and submucosal bronchial vessels. Recurrent or sustained bleeding produces intraluminal accumulation, which undergoes progressive organisation through fibrin polymerisation and inflammatory cell infiltration—resulting in the firm, twisted, dark red-brown clot observed in **Figure 3**. This organised clot paradoxically constitutes a valuable diagnostic substrate: it may harbour mycobacteria shed from adjacent bronchial mucosa, rendering it amenable to ZN staining even when expectorated sputum is negative [20].

The temporal relationship between the onset of haemoptysis and the formation of an organised endobronchial clot is clinically important and directly relevant to understanding the presentation in this case. The patient reported a seven-day history of haemoptysis prior to bronchoscopy, during which time an estimated 100 mL of blood was lost per episode. The macroscopic appearance of the retrieved clot—dark red-brown, firm, elongated, and twisted, with histopathological evidence of fibrinisation on Ziehl-Neelsen staining—is consistent with a thrombus that has undergone progressive organisation over several days. Fresh intraluminal haemorrhage is initially fluid, but begins to organise within 24 - 48 hours through platelet aggregation and fibrin polymerisation; by 3 - 7 days, the thrombus acquires a firm, cohesive structure through infiltration by fibroblasts and inflammatory cells [6] [20]. The seven-day interval between symptom onset and bronchos-

copy in this case is therefore consistent with the degree of clot organisation observed, providing a mechanistically coherent temporal narrative.

This timeline has practical clinical implications. Organised clots are substantially more resistant to fragmentation and aspiration than fresh haemorrhagic material, and their mechanical removal by suction requires sustained high-flow aspiration or mechanical engagement rather than simple lavage. Conversely, their firm consistency makes them less likely to fragment and cause distal bronchial occlusion during extraction—a recognised complication of manipulating less-organised intraluminal blood. The seven-day symptom duration also implies that the patient had sustained repeated episodes of mucosal haemorrhage for approximately one week before presentation; had bronchoscopy been performed earlier—for instance, within 24 hours of haemoptysis onset—the clot might have been less organised and potentially more amenable to simple suction alone, while also yielding less fibrinous material for ZN staining. Clinicians should be aware that a delay of several days between haemoptysis onset and bronchoscopy, while not ideal from an airway safety standpoint, may paradoxically enhance the diagnostic yield of the retrieved specimen by allowing clot organisation and mycobacterial embedding to progress.

3.6. The Dual Role of Bronchoscopy: Therapeutic and Diagnostic

The clinical-radiological-bronchoscopic correlation in this case is particularly instructive. The plain chest radiograph demonstrated bilateral infiltrates with left-predominant consolidation rather than complete right lung opacification, despite bronchoscopically confirmed complete right main bronchial obstruction. This apparent paradox is explained by the adequacy of compensatory left lung ventilation—maintaining SpO_2 at 99% on room air—and the presence of bilateral TB-related parenchymal disease producing left-sided consolidation on the plain film. The absent breath sounds over the right lung field on clinical examination directly predicted the bronchoscopic finding of complete right main bronchial obstruction. This physical examination sign, while non-specific in isolation, acquires considerable diagnostic weight when considered alongside the history of haemoptysis, moderate haemoptysis volume, and seven-day duration—providing a coherent anatomical explanation for the CT right lower lobe consolidation and reinforcing the appropriateness of proceeding to bronchoscopy without further delay. The preserved oxygen saturation (99% on room air) at presentation, despite complete right main bronchial obstruction, reflects adequate compensatory ventilation from the unaffected left lung—a physiological reserve that may mask the severity of obstruction and delay presentation in some patients [6]. The central therapeutic contribution of bronchoscopy in this case was mechanical clearance of the organised endobronchial clot from the right main bronchus, restoring luminal patency and averting distal atelectasis, post-obstructive pneumonia, and potential respiratory compromise [6] [20]. In a 21-year-old with active infection, timely bronchoscopic intervention may have been decisive in preventing respiratory de-

compensation.

The diagnostic contribution was equally significant. Bronchoscopically obtained BAL cytology subjected to GeneXpert MTB/RIF returned a positive result for MTBC within 48 - 72 hours, providing rapid molecular confirmation without awaiting mycobacterial culture. Simultaneously, ZN staining of the retrieved clot provided independent histopathological confirmation. The delivery of both therapeutic airway management and dual-modality molecular and histopathological diagnosis through a single bronchoscopic procedure represents an efficient and clinically decisive approach. Reported sensitivities for GeneXpert on BAL specimens in smear-negative pulmonary TB range from 70% to 90% across published series [9] [10], substantially exceeding the performance of sputum smear microscopy in this diagnostic context.

The decision to perform bronchoscopy under general anaesthesia (GA) rather than conscious sedation merits specific comment. In active haemoptysis, GA with endotracheal intubation offers decisive advantages: definitive airway control, protection of the contralateral lung from aspiration of blood, unrestricted high-flow suction capability, and complete mechanical clot extraction without patient agitation or laryngospasm risk [6]. Conscious sedation carries substantially higher procedural risk when sudden airway compromise may require emergency conversion to definitive airway management. The pre-procedural coagulation profile in this case—APTT and INR both within normal limits despite mild thrombocytopenia—confirmed haemostatic competence and supported proceeding under GA without prophylactic platelet transfusion. Systematic coagulation assessment prior to bronchoscopy in any patient with haemoptysis and thrombocytopenia is a mandatory pre-procedural step, both for clinical safety and medicolegal transparency [6] [20].

The mild thrombocytopenia (platelets 120 × 10^9/L) documented in this case warrants independent clinical comment. Thrombocytopenia is a recognised haematological manifestation of active pulmonary tuberculosis, observed in approximately 5% - 17% of cases in published series, generally attributed to immune-mediated platelet destruction, bone marrow suppression by mycobacterial disease, or hypersplenism in advanced infection [21]. Critically, the preservation of normal APTT and INR argues definitively against a primary coagulopathy as the cause of haemoptysis—a distinction of both clinical and diagnostic importance. The thrombocytopenia in this case is interpreted as a secondary haematological manifestation of active TB rather than an independent bleeding risk factor, and is expected to resolve with effective anti-tuberculous therapy, providing an additional indirect marker of treatment response (**Figure 6**).

3.7. Sputum-Histopathology Discordance: Mechanistic Explanation and Clinical Implications

The present case provides a particularly instructive example of multilevel diagnostic discordance: sputum AFB smear microscopy was persistently negative, yet

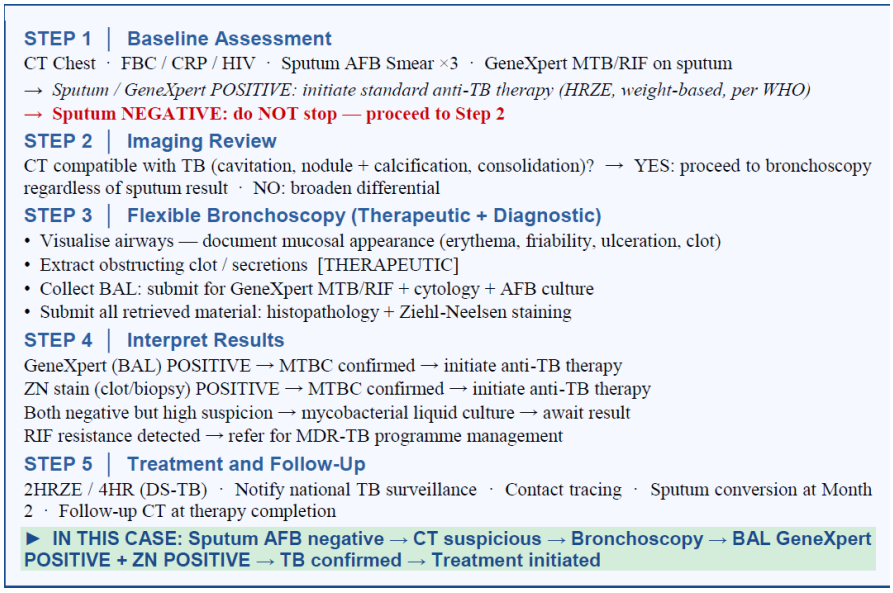

Figure 6. Proposed stepwise diagnostic algorithm for adolescent haemoptysis in a TB-endemic region. DS-TB = Drug-Sensitive Tuberculosis; MDR-TB = Multidrug-Resistant Tuberculosis; HRZE = Isoniazid, Rifampicin, Pyrazinamide, Ethambutol; BAL = Bronchoalveolar Lavage; ZN = Ziehl-Neelsen; MTBC = Mycobacterium Tuberculosis Complex.

two independent bronchoscopic diagnostic modalities — GeneXpert MTB/RIF on BAL cytology and ZN histopathological staining of the endobronchial clot — were both positive for Mycobacterium tuberculosis complex. This triangulation of molecular and morphological evidence, obtained from a single bronchoscopic procedure, delivered unambiguous diagnostic confirmation that non-invasive methods had entirely failed to provide.

Several mechanistic explanations account for this discordance. First, sputum smear microscopy requires a bacillary burden of approximately 5000 - 10,000 organisms per millilitre for reliable detection; in EBTB, the mycobacterial load in expectorated secretions may fall below this threshold despite active mucosal disease [8]. Second, intermittent shedding of mycobacteria—well-documented throughout the natural history of TB—means that any given set of sputum samples may fail to capture a positive result [22]. Third, adolescent patients may not generate adequate sputum volumes or quality for reliable smear examination [23]. In the present case, the patient was unable to produce adequate spontaneous sputum for pre-bronchoscopy molecular testing, which served as an additional indication for bronchoscopy and precluded a formal sputum GeneXpert prior to the procedure. The GeneXpert Very Low result on BAL (corresponding to Ct > 28) confirms that even if adequate sputum had been obtained, the low bacillary burden may have produced an indeterminate or negative result on standard sputum GeneXpert analysis, underscoring the superiority of BAL as a diagnostic specimen in endobronchial disease. The elevated CRP of 55 mg/L without haematological derangement is consistent with a subacute infective process, providing supportive biochemical evidence but insufficient specificity to replace direct microbiological

or histopathological confirmation. Critically, the GeneXpert RIF-sensitive result directly informed the treatment decision, confirming that the standard six-month first-line regimen (2HRZE/4HR) was appropriate without the need for specialist MDR-TB programme referral—a particularly important determination in a resource-limited setting where empirical MDR-TB therapy carries significant toxicity and cost.

These findings carry direct clinical implications. A negative sputum AFB result in a clinically compatible presentation should not terminate the diagnostic pathway. WHO guidelines and the Kenya National TB Programme explicitly recommend escalation to GeneXpert and bronchoscopy in smear-negative suspects [3] [11]. Had bronchoscopy not been performed in this case, or had BAL and clot specimens not been submitted for GeneXpert and ZN analysis, the diagnosis of TB would have been significantly delayed, with consequent risks of ongoing transmission, progressive pulmonary destruction, and delayed curative therapy.

3.8. Tuberculosis in Kenya: Epidemiological Context and Multimodal Diagnosis

Kenya's position among the WHO's 30 high TB-burden countries [3] establishes the epidemiological imperative for a low diagnostic threshold in adolescents presenting with haemoptysis. Adolescents occupy an intermediate clinical and bacteriological position between paediatric TB (paucibacillary, smear-negative) and adult TB (cavitatory, smear-positive), producing heterogeneous presentations that may elude standard diagnostic algorithms [23]. A multimodal diagnostic approach—integrating clinical assessment, CT characterisation, sputum-based testing, HIV serology, molecular diagnostics, and bronchoscopy with tissue sampling when indicated—is essential to reduce diagnostic delays and improve case detection in this vulnerable population [11]. The absence of BCG vaccination in this patient is noteworthy: BCG vaccination in infancy does not reliably prevent post-primary pulmonary TB in adolescents and young adults [23], but its absence removes a potential source of false-positive tuberculin skin test results and reflects the incomplete vaccine coverage documented in certain Kenyan communities. In the present case, this approach resulted in the timely initiation of appropriate DS-TB therapy, haemoptysis resolution, sputum conversion at two months, and treatment success at six months—a clinically favourable outcome that underscores the importance of pursuing a complete diagnostic workup rather than accepting sputum-negative results as exclusionary.

3.9. Comparative Context: Published Cases of Endobronchial TB with Haemoptysis

Table 7 summarizes selected published cases and series in which endobronchial tuberculosis presented with haemoptysis in younger patients, contextualizing the current report within the existing literature. This is not a systematic review; cases were identified through a narrative literature search for educational comparison.

Table 7. Comparative summary of selected published cases of endobronchial tuberculosis presenting with haemoptysis.

Author (Year)	Age	Presentation	Sputum AFB	Bronchoscopic Finding	Key Learning Point
Ozkaya *et al.* (2012) [8]	Adults (series)	Cough, haemoptysis, dyspnoea	Negative in 40% of EBTB	Mucosal ulceration, granulation, fibrostenosis	Sputum negativity is common in EBTB; bronchoscopy is essential for diagnosis
Kashyap *et al.* (2003) [19]	Adults (series)	Haemoptysis; constitutional	Variable—frequently negative	Active ulcerative endobronchial lesions	EBTB is a distinct clinicopathological entity requiring bronchoscopic confirmation
Han *et al.* (1995) [20]	Adults	Haemoptysis; recurrent pneumonia	Not reported	Fibrostenosis; granulomatous change	Endobronchial TB can produce irreversible stricture if untreated—early diagnosis is critical
Xue *et al.* (2011) [24]	Adults (review)	Haemoptysis; dyspnoea; wheeze	Negative in the majority of EBTB	Multiple subtypes, including ulcerative and tumorous	GeneXpert and bronchoscopic biopsy increase diagnostic yield beyond sputum smear alone
Present Case (2026)	21-year-old young adult male	Haemoptysis; fever; weight loss	Negative	Diffuse erythema; petechial hemorrhage; organized right main bronchus clot	Dual confirmation (GeneXpert BAL + ZN histopathology) despite sputum negativity; treatment success at 6 months (haemoptysis resolved, sputum converted at Month 2, weight recovery); to the best of our knowledge, the first published report of adolescent EBTB presenting as organized endobronchial clot from Kenya

EBTB = Endobronchial Tuberculosis; BAL = Bronchoalveolar Lavage; ZN = Ziehl-Neelsen; OJIM = Open Journal of Internal Medicine. Green Row = Present Case.

3.10. Endobronchial Tuberculosis in Young Adults versus Older Adults: A Clinicopathological Comparison

Endobronchial tuberculosis exhibits distinct clinicopathological characteristics depending on patient age. The present patient, a 21-year-old young adult, occupies a clinical position at the boundary of the adolescent and adult TB patterns, where paucibacillary disease with sputum negativity—classically associated with younger patients—may coexist with the parenchymal and endobronchial features of post-primary disease more typical of adult TB. While EBTB is documented across all age groups, it carries particular diagnostic implications in younger pa-

tients (aged 15 - 25 years), in whom the clinical, microbiological, and bronchoscopic profiles may differ meaningfully from those of older adults [8] [19] [25]. Understanding these differences is essential for clinicians practicing in TB-endemic settings where adolescent TB is disproportionately common and disproportionately underdiagnosed [23] [26].

In adults, post-primary pulmonary TB typically produces high-bacillary-burden cavitatory disease with smear-positive sputum, enabling conventional diagnostic pathways. In adolescents, disease is more likely to resemble the paediatric paucibacillary pattern—characterised by lower bacillary burden, smear negativity, predominant lymphatic involvement, and, when endobronchial, a propensity for ulcerative and inflammatory mucosal disease rather than fibrotic stricturing [23] [25]. The present case exemplifies this adolescent pattern: diffuse mucosal erythema, petechial haemorrhage, and organised clot formation rather than frank cavitatory disease or fibrostenosis. Table 8 summarises the key clinicopathological differences between EBTB in younger and older patients.

Table 8. Clinicopathological comparison of endobronchial tuberculosis (EBTB) in adolescents versus adults.

Feature	Young Adult EBTB (15 - 25 years)	Adult EBTB
Age Range	15 - 25 years (present case: 21 years)	20 - 60+ years
Sex Predilection	No consistent predilection has been reported	Higher prevalence in women in most series [8] [19]
Sputum AFB Smear	Frequently negative; paucibacillary pattern [23]	Positive in 40% - 60% of cases; varies by subtype [8]
Predominant TB Pattern	Post-primary or primary progressive; intermediate between paediatric and adult [23]	Post-primary; cavitary disease is common [8] [10]
Bronchoscopic Appearance	Diffuse erythema, friability, petechial hemorrhage, active ulceration (present case)	Multiple subtypes: ulcerative, granular, fibrostenotic, tumorous [8] [24]
Clot/Haemoptysis Mechanism	Mucosal ulceration, erosion of submucosal vessels; organized clot (present case)	Mucosal erosion; Rasmussen aneurysm in cavitary disease [13]
Sputum Conversion during Treatment	Expected by Month 2; may be rapid given the paucibacillary burden [23]	Typically confirmed at Month 2; culture conversion is used [3]
Risk of Bronchial Stricture	Lower in the acute ulcerative phase; fibrostenosis may develop if untreated [19] [25]	Higher in chronic disease; cicatricial stenosis is well documented [20]
Recommended Diagnostic Approach	Bronchoscopy with BAL GeneXpert + ZN staining; tissue biopsy if a lesion is visible	Sputum-based first; bronchoscopy if smear is negative; biopsy for ulcerative/tumorous subtypes
Treatment Regimen	Standard DS-TB: 2HRZE/4HR (weight-based per WHO 2022) [11]	Standard DS-TB: 2HRZE/4HR (adult dosing per WHO 2022) [3]

HRZE = Isoniazid, Rifampicin, Pyrazinamide, Ethambutol; DS-TB = Drug-Sensitive Tuberculosis; BAL = Bronchoalveolar Lavage; ZN = Ziehl-Neelsen.

3.11. Anaemia in Active Tuberculosis: Pathophysiology of the Dual Mechanism

The moderate anaemia documented in this case (haemoglobin 9.0 g/dL) is best understood as the product of two simultaneous pathophysiological processes: 1) acute haemorrhagic blood loss from recurrent endobronchial haemorrhage, and 2) the anaemia of chronic disease (ACD), also termed anaemia of inflammation, associated with active mycobacterial infection. Distinguishing these mechanisms has both diagnostic and therapeutic implications [27] [28].

Acute haemorrhagic anaemia in this case was driven by an estimated blood loss of approximately 100 mL per episode over seven days. While individual episodes of this volume are classified as moderate haemoptysis rather than massive haemorrhage, cumulative blood loss over a seven-day period without compensatory marrow response in the context of pre-existing nutritional deficiency and weight loss is consistent with the degree of anaemia observed. The anaemia of chronic disease, independently operating in active TB, results from inflammatory cytokine-mediated suppression of erythropoiesis, reduced erythropoietin responsiveness, shortened erythrocyte lifespan, and iron sequestration by macrophages under the influence of hepcidin, an acute-phase reactant elevated in active mycobacterial infection [27]-[29].

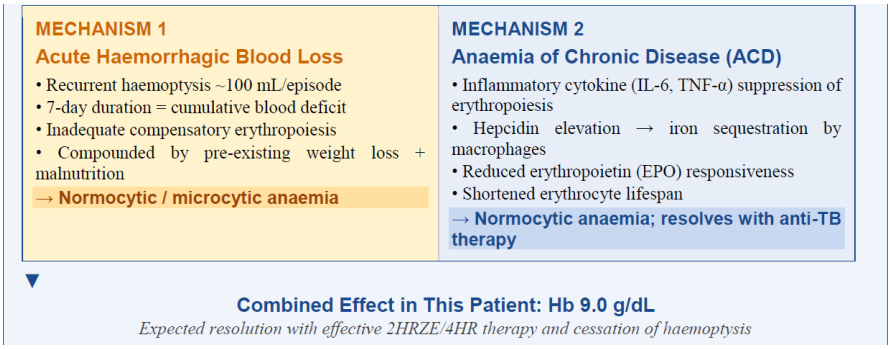

Figure 7. Dual mechanism of anaemia in this adolescent patient with active pulmonary tuberculosis. ACD = Anaemia of Chronic Disease; IL-6 = Interleukin-6; TNF-α = Tumour Necrosis Factor-Alpha; EPO = Erythropoietin; Hb = Haemoglobin; HRZE = Isoniazid, Rifampicin, Pyrazinamide, Ethambutol.

3.12. Nutritional Rehabilitation as a Component of Anti-Tuberculous Therapy

The nutritional dimension of this case deserves specific attention. The patient presented with unintentional weight loss as part of the constitutional triad of active TB, compounded by moderate anaemia (haemoglobin 9.0 g/dL) arising from the dual mechanism of haemorrhagic blood loss and anaemia of chronic disease. Malnutrition is both a risk factor for TB and a consequence of it: protein-energy deficiency impairs cell-mediated immunity, increasing susceptibility to mycobacterial infection, while active TB infection drives a catabolic state that perpetuates and deepens nutritional deficits [27] [28]. This bidirectional relationship between nu-

tritional status and TB disease severity has been recognised by the WHO, which recommends nutritional assessment and support as an integral component of TB management, particularly in adolescent and paediatric patients [3].

In this case, pyridoxine (Vitamin B6) 25 mg daily was co-prescribed with isoniazid to prevent peripheral neuropathy—a risk magnified by nutritional deficiency and reduced dietary intake of pyridoxine-containing foods [12]. Progressive weight recovery was documented throughout the treatment course and confirmed at six-month follow-up, consistent with effective anti-mycobacterial therapy reversing the catabolic state and resolving the anaemia of chronic disease. The normalisation of haemoglobin over the treatment period, while not serially documented in this case, is anticipated based on the dual-mechanism model (**Figure 7**): cessation of haemoptysis eliminates the haemorrhagic component, while effective TB treatment reduces the inflammatory cytokine burden responsible for the chronic disease component, restoring normal erythropoiesis. Clinicians managing adolescent TB should ensure that nutritional assessment, dietary counselling, and micronutrient supplementation are incorporated into the treatment package alongside anti-TB pharmacotherapy, particularly in patients presenting with weight loss and moderate anaemia.

3.13. Post-Treatment Follow-Up: WHO Outcome Definitions and Monitoring Framework

The availability of a follow-up chest radiograph at six months provides objective radiological documentation of treatment response, a dimension that many published case reports of endobronchial TB lack. The PA erect chest radiograph obtained at treatment completion (**Figure 5**) demonstrated near-complete resolution of the bilateral pulmonary consolidation and ground-glass opacities identified at presentation, with only mild residual reticular markings in the lower zones bilaterally—a pattern consistent with post-inflammatory fibrotic change rather than residual active TB. This radiological evolution, from bilateral active infiltrates to near-complete resolution over six months of DS-TB therapy, provides compelling visual evidence of treatment efficacy and directly corroborates the WHO treatment success designation based on sputum conversion and clinical response.

The attainment of treatment success in this case, at six months—defined by WHO as sputum smear or culture conversion without subsequent reversion, completion of the full prescribed treatment course, and absence of treatment failure criteria—represents the primary objective of DS-TB management [3] [12]. Documentation of WHO-standardized outcome classifications is not merely a clinical formality; it contributes to national and international TB surveillance data, informs programme evaluation, and provides the epidemiological evidence base required to justify current first-line regimen recommendations [3] [11].

For adolescent patients with endobronchial TB completing therapy, several additional follow-up considerations merit attention beyond sputum conversion.

Bronchial stricture, a well-recognised late complication of EBTB, may develop silently following apparent clinical resolution, particularly in cases with extensive mucosal involvement or fibrostenotic subtype bronchoscopic findings [20] [24] [25]. Although the bronchoscopic appearances in this case were predominantly ulcerative and haemorrhagic rather than fibrostenotic, the risk of post-inflammatory cicatricial narrowing cannot be entirely excluded without follow-up bronchoscopy or high-resolution CT at therapy completion [30]. **Table 9** summarises the recommended post-treatment monitoring framework for endobronchial TB, integrating WHO outcome criteria, Kenya NTP guidance, and radiological surveillance recommendations.

Table 9. Recommended post-treatment monitoring framework for adolescent endobronchial tuberculosis.

Time Point	Assessment	Expected Finding (Treatment Success)	Action If Abnormal
Month 2 (End of Intensive Phase)	Sputum smear + culture; clinical assessment; weight; CXR	Sputum conversion; symptom improvement; weight gain; CXR improvement	Consider an extended intensive phase; reassess DST; check adherence
Month 5	Sputum smear + culture	Smear negative; culture negative	Investigate treatment failure; urgent DST; specialist referral
Month 6 (Treatment Completion)	Sputum smear + culture; CXR or CT chest; weight; LFTs; visual acuity (post-ethambutol)	Culture confirmed negative; radiological improvement; weight restored; LFTs normalized	If culture is positive at Month 5 or 6: classify as treatment failure; DST; MDR evaluation
Month 12 (1-Year Post-Completion)	Clinical review; CXR; spirometry if symptomatic; consider follow-up bronchoscopy	No relapse; stable or improved CXR; no breathlessness or wheezing	Investigate relapse; repeat sputum culture; perform bronchoscopy to exclude bronchial stricture
If Symptoms Recur	Sputum smear + culture; bronchoscopy; CT chest	N/A—symptom recurrence requires investigation	Exclude relapse vs. reinfection; exclude bronchial stricture; repeat DST

DS-TB = Drug-Sensitive Tuberculosis; DST = Drug Susceptibility Testing; CXR = Chest Radiograph; LFT = Liver Function Test; MDR = Multidrug-Resistant; WHO = World Health Organization.

3.14. Public Health Implications: Sputum-Negative TB and Transmission Risk

The public health dimensions of this case extend beyond the individual patient and carry implications for TB control strategy in Kenya and comparable endemic settings. Sputum-negative pulmonary tuberculosis has historically been considered a lower transmission risk than smear-positive disease; however, this assump-

tion has been progressively challenged by molecular epidemiological evidence demonstrating that smear-negative patients contribute meaningfully to community TB transmission [22] [31].

Behr *et al.* demonstrated that patients with smear-negative culture-positive TB are responsible for an estimated 17% of secondary cases in high-burden settings, a figure that substantially exceeds what would be predicted if transmission were exclusively from smear-positive individuals [22]. In adolescents, the epidemiological stakes are particularly high: school attendance, household crowding, and limited access to healthcare create conditions for rapid intergenerational spread. A 21-year-old with active endobronchial TB attending school in Nairobi represents a potentially significant source of community transmission, particularly given the seven-day symptom-to-presentation interval and the negative sputum smear result that might, in the absence of bronchoscopy, have led to diagnostic delay and continuation of community exposure [3] [23].

The Kenya National TB Programme mandates contact tracing and household investigation for all confirmed TB cases [11]. In smear-negative cases, the yield of contact tracing may be lower than in smear-positive disease, but the process remains obligatory and clinically important. The adoption of GeneXpert MTB/RIF across Kenyan facilities has substantially improved detection of smear-negative TB, but coverage remains incomplete, particularly at lower-tier health facilities [11] [32]. Cases such as this underscore the importance of: 1) not allowing sputum negativity to falsely reassure clinicians and delay definitive investigation; 2) ensuring that molecularly confirmed cases are promptly notified and contact-traced regardless of smear status; and 3) recognizing adolescents with constitutional symptoms and haemoptysis as a high-priority demographic for accelerated TB diagnosis in endemic settings (**Table 10**).

Table 10. Public health implications and recommended actions for sputum-negative adolescent tuberculosis in an endemic setting.

Public Health Domain	Considerations in Sputum-Negative Young Adult TB	Recommended Action
Transmission Risk	Smear-negative TB contributes ~17% of secondary cases [22]; adolescent school attendance amplifies exposure risk	Prompt notification upon molecular/histopathological confirmation, regardless of smear status
Contact Tracing	Mandatory per Kenya NTP [11]; lower yield than smear-positive, but obligatory	Household + school contact screening; tuberculin skin testing or IGRA in contacts
Notification	All bacteriologically confirmed cases (including GeneXpert-positive) are notifiable [11]	Register the case; report to the county TB coordinator; enter into the national TIBU system
Diagnostic Delay and Spread	7-day symptom-to-presentation interval in this case; sputum negativity could have caused further delay	Establish a fast-track bronchoscopy pathway for haemoptysis and sputum-negative presentations

Continued

GeneXpert Access	Incomplete facility-level coverage in Kenya [32]; BAL GeneXpert is required where sputum GeneXpert fails	Advocate for GeneXpert availability at bronchoscopy-capable facilities
Treatment as Prevention	Prompt initiation of effective anti-TB therapy reduces transmission rapidly	Initiate 2HRZE/4HR within 24 - 48 hours of GeneXpert/histopathological confirmation

NTP = National Tuberculosis Programme; IGRA = Interferon-Gamma Release Assay; GeneXpert = GeneXpert MTB/RIF; TIBU = TB Information and Notification System (Kenya).

3.15. Future Research Directions and Implications for Clinical Guidelines

This case raises several questions that merit systematic investigation and have direct implications for the development of evidence-based clinical guidelines for the management of adolescent haemoptysis in TB-endemic settings. We propose the following research priorities and policy recommendations, grounded in the clinical experience documented here.

First, the prevalence and clinical characteristics of endobronchial TB presenting as organised haemoptysis and airway clot formation in adolescents in sub-Saharan Africa remain undocumented. A prospective multicentre registry of adolescent haemoptysis at bronchoscopy-capable tertiary facilities across Kenya and comparable high-burden settings would provide epidemiological data to define the true burden of this presentation, identify risk factors for clot formation, and establish evidence-based thresholds for proceeding to bronchoscopy. The Kenya National TB Programme and the Respiratory Society of Kenya are well-positioned to coordinate such a registry.

Second, the diagnostic pathway employed in this case—combining multiplex respiratory pathogen exclusion (BioFire FilmArray), BAL GeneXpert MTB/RIF molecular testing, and histopathological ZN staining of retrieved bronchoscopic material—represents a novel multimodal protocol not previously described in EBTB case reports from Africa. A prospective clinical protocol study evaluating this pathway against the standard sputum-based approach would provide comparative sensitivity, specificity, time-to-diagnosis, and cost-effectiveness data that could support its incorporation into national TB diagnostic guidelines for sputum-negative haemoptysis presentations.

Third, the use of general anaesthesia versus conscious sedation for flexible bronchoscopy in active haemoptysis has not been evaluated in a randomised or prospective observational study in resource-limited settings. While GA was clinically appropriate in this case based on the degree of haemoptysis and complete airway obstruction, the additional resource requirements of GA—anaesthetist availability, theatre time, post-anaesthesia monitoring—may not be feasible at all facilities. A structured prospective study comparing the safety and diagnostic outcomes of GA versus conscious sedation bronchoscopy in moderate haemoptysis

would inform facility-level protocol development across different tiers of the Kenyan healthcare system.

Finally, the BCG-naive status of this patient warrants comment in the context of TB prevention policy. While BCG vaccination does not reliably prevent post-primary pulmonary TB in adolescents, it significantly reduces the risk of severe disseminated TB in infancy and childhood [23]. The absence of BCG vaccination in this 21-year-old patient reflects a gap in vaccine coverage that contributes to unprotected exposure during the critical adolescent period. Strengthening BCG vaccination coverage and catch-up programmes for unvaccinated older children and adolescents in Kenya represents an actionable public health recommendation with direct relevance to preventing cases such as this.

3.16. Limitations

This case report is subject to several limitations. Certain microbiological data—notably mycobacterial liquid culture and full phenotypic drug susceptibility testing beyond rifampicin resistance profiling—were unavailable at the time of writing. Rifampicin sensitivity was confirmed by GeneXpert, providing the molecular basis for initiation of standard DS-TB therapy (2HRZE/4HR); however, formal phenotypic DST for isoniazid, pyrazinamide, and ethambutol remains pending, and resistance to non-rifampicin first-line agents cannot be excluded on molecular data alone. Detailed physical examination data, including vital signs, are not documented in available records. Treatment outcome data are available and documented in Section 2.8: the patient achieved treatment success at six months, with haemoptysis resolution, sputum conversion, and weight recovery. Radiological follow-up at treatment completion is now available: a PA erect chest radiograph at six months demonstrated near-complete resolution of bilateral consolidation, providing objective documentation of structural pulmonary response to therapy (**Figure 5**). High-resolution CT at treatment completion was not performed; post-treatment HRCT would have provided more detailed characterisation of residual fibrotic changes and exclusion of bronchial stricture, representing a limitation of the post-treatment radiological assessment. The haemoptysis duration (7 days), estimated volume per episode (~100 mL, moderate classification), and constitutional symptoms (night sweats, fever, weight loss) are now documented; the precise temporal relationship between haemoptysis onset and clot organisation within the bronchus cannot be definitively established from available clinical records. A detailed H&E architectural description was not available in the documented pathology report at the time of manuscript preparation; this limitation is explicitly acknowledged in Section 2.7 without inference or fabrication. PAS and GMS fungal stains were both negative and are documented. Mycobacterial liquid culture was submitted, but the final culture result and comprehensive phenotypic DST for the full first-line panel (isoniazid, pyrazinamide, ethambutol resistance) were not available at the time of manuscript preparation. This represents a limitation in the completeness of drug susceptibility documentation; however, the GeneXpert RIF-sensitive result provided an adequate molecular basis for standard

DS-TB therapy, and the patient achieved treatment success on 2HRZE/4HR without evidence of drug resistance or treatment failure. Tuberculin skin testing (TST) and interferon-gamma release assay (IGRA) were not performed; while the bacteriological diagnosis in this case is confirmed by GeneXpert and ZN staining and does not require immunological corroboration, the absence of IGRA or TST documentation represents a minor limitation in the completeness of the immunological diagnostic workup. Additionally, the exact numerical Ct value from GeneXpert analysis was not recorded in the available laboratory documentation; the semi-quantitative designation of Very Low is used as reported by the laboratory, corresponding to an estimated Ct > 28. These limitations are acknowledged in accordance with CARE 2013 reporting standards.

4. Conclusions

This case documents a 21-year-old male resident of Nairobi, Kenya, in whom pulmonary tuberculosis with endobronchial involvement presented as haemoptysis, constitutional symptoms, and an organised blood clot obstructing the right main bronchus. Sputum AFB smear microscopy was negative throughout. Flexible bronchoscopy provided both therapeutic restoration of airway patency through clot extraction and definitive diagnosis through two independent bronchoscopic modalities: GeneXpert MTB/RIF on BAL cytology and ZN histopathological staining of the retrieved clot, both positive for Mycobacterium tuberculosis complex.

The patient completed the full six-month first-line regimen (2HRZE/4HR). Haemoptysis resolved following bronchoscopic clot extraction and did not recur. Sputum conversion was confirmed at two months, weight recovery was documented progressively, and a follow-up chest radiograph at six months demonstrated near-complete bilateral radiological resolution—collectively meeting WHO treatment success criteria for DS-TB. The central lessons of this case are clear. First, in TB-endemic settings, a negative sputum AFB result must not terminate the diagnostic pathway in a young adult with compatible clinical and radiological features. Second, flexible bronchoscopy serves an indispensable dual therapeutic and diagnostic role—one that cannot be replicated by any non-invasive modality. Third, all bronchoscopically retrieved material should be submitted for both GeneXpert molecular testing and histopathological ZN staining as a matter of standard practice: in this case, GeneXpert on BAL additionally confirmed rifampicin sensitivity, enabling immediate initiation of standard six-month DS-TB therapy (2HRZE/4HR) without MDR-TB referral. Fourth, the multimodal diagnostic approach demonstrated in this case—integrating clinical, radiological, molecular, and morphological evidence—represents the optimal standard of care for adolescent sputum-negative haemoptysis in TB-endemic sub-Saharan Africa.

Patient Perspective

The patient, a 21-year-old male, expressed relief following the bronchoscopic procedure, reporting immediate improvement in his ability to breathe following res-

toration of airway patency. He completed the full six-month anti-tuberculous treatment course and was reported by his family to have returned to his normal daily activities, including school attendance, by the time of treatment completion. The patient and his legal guardian provided written informed consent for this publication and expressed willingness for their experience to contribute to the medical literature for the benefit of future patients with similar presentations [Patient statement or further perspective to be added with consent].

Patient Consent Statement

Written informed consent was obtained from the patient and his legal guardian for the publication of this case report and all accompanying images, in accordance with the Declaration of Helsinki (2013 revision) and the guidelines of the Open Journal of Internal Medicine. All identifying information has been removed or redacted to preserve patient anonymity. Ethical clearance for the publication of de-identified clinical data was obtained from the relevant institutional authority in accordance with applicable national research governance requirements.

Care Guidelines Compliance

This case report was prepared in accordance with the CARE (Case Report) 2013 guidelines for the complete and transparent reporting of clinical cases (Gagnier *et al.*, 2013, BMJ). The completed CARE checklist is available upon request from the corresponding author.

Author's Contributions

Yashar Tolentino Najiaghdam: conceptualization, clinical management, data acquisition, manuscript preparation, and final approval of the submitted version.

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Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

References

[1] Coss-Bu, J.A., Sachdeva, R.C., Bricker, J.T., Harrison, G.M. and Jefferson, L.S. (1997) Hemoptysis: A 10-Year Retrospective Study. *Pediatrics*, **100**, e7. https://doi.org/10.1542/peds.100.3.e7

[2] Bidwell, J.L. and Pachner, R.W. (2005) Hemoptysis: Diagnosis and Management.

American Family Physician, **72**, 1253-1260.

[3] World Health Organization (2023) Global Tuberculosis Report. World Health Organization. https://www.who.int/publications/i/item/9789240083851

[4] Gaude, G.S. (2010) Hemoptysis in Children. *Indian Pediatrics*, **47**, 245-254. https://doi.org/10.1007/s13312-010-0044-z

[5] Susarla, S.C. and Fan, L.L. (2007) Diffuse Alveolar Hemorrhage Syndromes in Children. *Current Opinion in Pediatrics*, **19**, 314-320. https://doi.org/10.1097/mop.0b013e3280dd8c4a

[6] Sakr, L. and Dutau, H. (2010) Massive Hemoptysis: An Update on the Role of Bronchoscopy in Diagnosis and Management. *Respiration*, **80**, 38-58. https://doi.org/10.1159/000274492

[7] Charoenratanakul, S., Dejsomritrutai, W. and Chaiprasert, A. (1995) Diagnostic Role of Fiberoptic Bronchoscopy in Suspected Smear Negative Pulmonary Tuberculosis. *Respiratory Medicine*, **89**, 621-623. https://doi.org/10.1016/0954-6111(95)90231-7

[8] Ozkaya, S., Bilgin, S., Findik, S., Kök, H.Ç., Yuksel, C. and Atıcı, A.G. (2012) Endobronchial Tuberculosis: Histopathological Subsets and Microbiological Results. *Multidisciplinary Respiratory Medicine*, **7**, Article No. 34. https://doi.org/10.1186/2049-6958-7-34

[9] Lawn, S.D. and Zumla, A.I. (2011) Tuberculosis. *The Lancet*, **378**, 57-72. https://doi.org/10.1016/s0140-6736(10)62173-3

[10] Leung, A.N. (1999) Pulmonary Tuberculosis: The Essentials. *Radiology*, **210**, 307-322. https://doi.org/10.1148/radiology.210.2.r99ja34307

[11] Kenya Ministry of Health (2020) National Tuberculosis, Leprosy and Lung Disease Pro-gramme: Guidelines for Management of Tuberculosis and Leprosy in Kenya. 5th Edition, Ministry of Health, Kenya.

[12] World Health Organization (2013) Definitions and Reporting Framework for Tuberculosis—2013 Revision: Updated December 2014 and January 2020. https://www.who.int/publications/i/item/9789241505345

[13] Chatterjee, K., Colaco, B., Colaco, C., Hellman, M. and Meena, N. (2015) Rasmussen's Aneurysm: A Forgotten Scourge. *Respiratory Medicine Case Reports*, **16**, 74-76. https://doi.org/10.1016/j.rmcr.2015.08.003

[14] Vijayan, V.K. (2008) Tropical Parasitic Lung Diseases. *Indian Journal of Chest Diseases and Allied Sciences*, **50**, 49-66.

[15] Im, J.G., Itoh, H., Shim, Y.S., Lee, J.H., Ahn, J., Han, M.C., *et al.* (1993) Pulmonary Tuberculosis: CT Findings—Early Active Disease and Sequential Change with Antituberculous Therapy. *Radiology*, **186**, 653-660. https://doi.org/10.1148/radiology.186.3.8430169

[16] Poritz, M.A., Blaschke, A.J., Byington, C.L., Allen, L., Nilsson, K., Jones, D.E., *et al.* (2011) Filmarray, an Automated Nested Multiplex PCR System for Multi-Pathogen Detection: Development and Application to Respiratory Tract Infection. *PLOS ONE*, **6**, e26047. https://doi.org/10.1371/journal.pone.0026047

[17] Gadkowski, L.B. and Stout, J.E. (2008) Cavitary Pulmonary Disease. *Clinical Microbiology Reviews*, **21**, 305-333. https://doi.org/10.1128/cmr.00060-07

[18] MacMahon, H., Naidich, D.P., Goo, J.M., Lee, K.S., Leung, A.N.C., Mayo, J.R., *et al.* (2017) Guidelines for Management of Incidental Pulmonary Nodules Detected on CT Images: From the Fleischner Society 2017. *Radiology*, **284**, 228-243. https://doi.org/10.1148/radiol.2017161659

[19] Kashyap, S., Mohapatra, P.R. and Saini, V. (2003) Endobronchial Tuberculosis. The

Indian Journal of Chest Diseases & Allied Sciences, **45**, 247-256.

[20] Han, J.K., Im, J.G., Park, J.H., Han, M.C. and Kim, C.W. (1995) Tuberculous Fibrostenosis of the Central Airways: CT Findings. *Journal of Computer Assisted Tomography*, **19**, 579-583.

[21] Steingart, K.R., Henry, M., Ng, V., Hopewell, P.C., Ramsay, A., Cunningham, J., *et al.* (2006) Fluorescence versus Conventional Sputum Smear Microscopy for Tuberculosis: A Systematic Review. *The Lancet Infectious Diseases*, **6**, 570-581. https://doi.org/10.1016/s1473-3099(06)70578-3

[22] Behr, M., Warren, S., Salamon, H., Hopewell, P., de Leon, A.P., Daley, C., *et al.* (1999) Transmission of Mycobacterium Tuberculosis from Patients Smear-Negative for Acid-Fast Bacilli. *The Lancet*, **353**, 444-449. https://doi.org/10.1016/s0140-6736(98)03406-0

[23] Perez-Velez, C.M. and Marais, B.J. (2012) Tuberculosis in Children. *New England Journal of Medicine*, **367**, 348-361. https://doi.org/10.1056/nejmra1008049

[24] Xue, Q., Wang, N., Xue, X. and Wang, J. (2011) Endobronchial Tuberculosis: An Overview. *European Journal of Clinical Microbiology & Infectious Diseases*, **30**, 1039-1044. https://doi.org/10.1007/s10096-011-1205-2

[25] Aggarwal, A.N., Gupta, D., Joshi, K., Behera, D. and Jindal, S.K. (1999) Endobronchial Involvement in Tuberculosis: A Report of 24 Cases Diagnosed by Fiberoptic Bronchoscopy. *Journal of Bronchology*, **6**, 247-250. https://doi.org/10.1097/00128594-199910000-00004

[26] Snow, K.J., Cruz, A.T., Seddon, J.A., Ferrand, R.A., Chiang, S.S., Hughes, J.A., *et al.* (2020) Adolescent Tuberculosis. *The Lancet Child & Adolescent Health*, **4**, 68-79. https://doi.org/10.1016/S2352-4642(19)30337-2

[27] Weiss, G. and Goodnough, L.T. (2005) Anemia of Chronic Disease. *New England Journal of Medicine*, **352**, 1011-1023. https://doi.org/10.1056/nejmra041809

[28] Isanaka, S., Mugusi, F., Urassa, W., Willett, W.C., Bosch, R.J., Villamor, E., *et al.* (2012) Iron Deficiency and Anemia Predict Mortality in Patients with Tuberculosis. *The Journal of Nutrition*, **142**, 350-357. https://doi.org/10.3945/jn.111.144287

[29] Ganz, T. (2011) Hepcidin and Iron Regulation, 10 Years Later. *Blood*, **117**, 4425-4433. https://doi.org/10.1182/blood-2011-01-258467

[30] Lee, J.Y. (2015) Diagnosis and Treatment of Extrapulmonary Tuberculosis. *Tuberculosis and Respiratory Diseases*, **78**, 47-55. https://doi.org/10.4046/trd.2015.78.2.47

[31] Tostmann, A., Kik, S.V., Kalisvaart, N.A., Sebek, M.M., Verver, S., Boeree, M.J., *et al.* (2008) Tuberculosis Transmission by Patients with Smear-negative Pulmonary Tuberculosis in a Large Cohort in the Netherlands. *Clinical Infectious Diseases*, **47**, 1135-1142. https://doi.org/10.1086/591974

[32] Oliwa, J.N., Maina, J., Ayieko, P., Gathara, D., Kathure, I.A., Masini, E., *et al.* (2018) Variability in Distribution and Use of Tuberculosis Diagnostic Tests in Kenya: A Cross-Sectional Survey. *BMC Infectious Diseases*, **18**, Article No. 328. https://doi.org/10.1186/s12879-018-3237-z