

Pulmonary non-tuberculous mycobacterial disease masquerading as recurrent tuberculosis in Nigeria: diagnostic complexities and therapeutic challenges from a 17-year longitudinal case report

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Case Report

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Abstract

Background

Pulmonary non-tuberculous mycobacterial disease (NTM-LD) is increasingly recognised as an important cause of chronic pulmonary infection worldwide. However, in tuberculosis (TB)-endemic settings, NTM-LD remains underdiagnosed because clinical algorithms and laboratory services are primarily designed to detect *Mycobacterium tuberculosis*. Consequently, patients are frequently misdiagnosed, receive repeated empirical anti-tuberculosis treatment, and experience delays in appropriate management.

Case presentation

We report a 17-year longitudinal case of an immunocompetent Nigerian man whose chronic respiratory illness ultimately proved to be pulmonary NTM disease. The patient had previously completed treatment for pulmonary tuberculosis in 2004 and presented to the University of Port Harcourt Teaching Hospital in 2009 with chronic productive cough, low-grade fever, weight loss, drenching night sweats, and exertional dyspnoea. Chest radiography demonstrated extensive unilateral fibrocavitary lung disease, while three sputum specimens were negative for acid-fast bacilli (AFB). Based on his clinical history and prevailing national tuberculosis treatment guidelines, he was empirically treated for relapsed pulmonary tuberculosis with a Category II anti-tuberculosis regimen. Despite modest clinical improvement, persistent symptoms and progressive radiographic destruction of the left lung prompted further evaluation. Repeat sputum microscopy detected AFB in one of two specimens, raising suspicion of drug-resistant tuberculosis. Mycobacterial culture performed at the Nigerian Institute of Medical Research yielded a non-tuberculous mycobacterium. Subsequent molecular testing confirmed that the isolate was not a member of the *Mycobacterium tuberculosis* complex; however, species identification and drug susceptibility testing were unavailable due to diagnostic limitations. Given the extensive cavitary lung disease, chronic pulmonary aspergillosis was also considered, and empirical itraconazole therapy was initiated while further investigations were pursued. The patient subsequently received rifampicin- and clarithromycin-based therapy with gradual clinical improvement. However, irreversible structural lung damage resulted in destroyed left lung syndrome and chronic bronchiectasis requiring long-term respiratory follow-up.

Conclusions

This case highlights the substantial diagnostic and therapeutic challenges of pulmonary NTM disease in resource-limited, TB-endemic settings. It demonstrates the limitations of relying on AFB microscopy and empirical TB treatment, the importance of recognizing discordant microbiological findings, and the consequences of limited access to mycobacterial species identification and drug susceptibility testing. Strengthening laboratory capacity, integrating NTM into national TB diagnostic algorithms, and

improving clinician awareness are essential for facilitating timely diagnosis and appropriate management.

Introduction

Non-tuberculous mycobacteria (NTM) comprise a diverse group of environmental mycobacteria that are increasingly recognised as important opportunistic pathogens, particularly in chronic pulmonary disease. [1] Unlike members of the *Mycobacterium tuberculosis* complex (MTBC), NTM are acquired from environmental reservoirs, including soil, water, and biofilms, and are not transmitted from person to person. [2] Over the past two decades, the incidence and prevalence of pulmonary NTM disease (NTM-LD) have increased worldwide, particularly in North America, Europe, and parts of Asia. [2] This trend is attributed to aging populations, the increasing prevalence of chronic lung diseases, improved clinician awareness, wider use of advanced imaging, and enhanced laboratory capacity for mycobacterial culture and species identification. [2, 3] In contrast, the epidemiology and clinical burden of NTM-LD in sub-Saharan Africa, including Nigeria, remain poorly defined because of limited surveillance, inadequate laboratory infrastructure, and the continued prioritization of tuberculosis (TB) within national public health programmes. [4]

Pulmonary NTM disease closely resembles pulmonary TB in its clinical and radiological presentation, making differentiation particularly challenging in TB-endemic settings. Patients commonly present with chronic cough, fever, weight loss, night sweats, hemoptysis, and progressive respiratory symptoms, while imaging often shows fibrocavitary disease, bronchiectasis, pulmonary fibrosis, or nodular infiltrates. [5] Furthermore, both NTM and MTBC retain acid-fast staining characteristics, limiting the specificity of sputum acid-fast bacilli (AFB) microscopy, which remains an important diagnostic tool in many low- and middle-income countries (LMICs). [6] Consequently, patients with pulmonary NTM disease are frequently misdiagnosed as having recurrent or drug-resistant TB, particularly where access to mycobacterial culture, molecular diagnostics, and species identification is limited. [7]

In countries with a high burden of TB, including Nigeria, national TB control programmes appropriately prioritize the rapid identification and treatment of *Mycobacterium tuberculosis* infection to reduce transmission and mortality. [8] However, this approach may inadvertently delay recognition of pulmonary NTM disease, especially in patients with persistent respiratory symptoms despite appropriate anti-tuberculosis therapy or discordant microbiological findings. Such delays may result in repeated empirical TB treatment, prolonged morbidity, progressive structural lung damage, unnecessary drug exposure, and missed opportunities for appropriate antimicrobial therapy. [9]

Although Nigeria remains one of the countries with the highest TB burdens globally, data describing the prevalence, species distribution, and clinical outcomes of pulmonary NTM disease are scarce. [10] Available studies from sub-Saharan Africa suggest that NTM may account for a proportion of patients investigated for pulmonary TB, particularly among those with persistent symptoms or microbiological findings that are inconsistent with *M. tuberculosis* infection. [11, 12] Nevertheless, routine evaluation for

NTM is rarely incorporated into standard diagnostic algorithms, and awareness of the disease among clinicians remains limited.

Pulmonary NTM disease has traditionally been associated with immunosuppression and pre-existing structural lung disease. However, growing evidence suggests it may also occur in immunocompetent individuals, particularly those with prior pulmonary TB, bronchiectasis, or other chronic pulmonary abnormalities that predispose to persistent mycobacterial colonization and infection. [13] Diagnosing NTM-LD requires integration of compatible clinical features, radiological abnormalities, and microbiological evidence, as recommended by the American Thoracic Society (ATS) and the Infectious Diseases Society of America (IDSA). [14] Implementation of these criteria remains difficult in many resource-limited settings because repeat cultures, species identification, and drug susceptibility testing are often unavailable.

We report the long-term clinical course of an immunocompetent young Nigerian man whose chronic respiratory illness was initially managed as recurrent pulmonary tuberculosis before subsequent mycobacterial culture and molecular testing established pulmonary NTM disease. This case illustrates the diagnostic ambiguity, therapeutic challenges, and long-term pulmonary sequelae associated with NTM-LD in a resource-limited, high-TB-burden setting. It further highlights the consequences of limited access to definitive mycobacterial diagnostics, the potential pitfalls of empirical anti-tuberculosis treatment, and the need to integrate NTM into routine diagnostic algorithms and clinician training in low- and middle-income countries.

Case Presentation

Clinical presentation

A 28-year-old Nigerian male engineering student at Rivers State University of Science and Technology presented to the Medical Outpatient Clinic of the University of Port Harcourt Teaching Hospital (UPTH), Port Harcourt, Nigeria, on 22 June 2009, with a one-month history of persistent cough and low-grade fever. The cough was non-paroxysmal, distressing, worse at night, and productive of moderate amounts of whitish sputum, occasionally purulent, brownish, and foul-smelling. There was no history of haemoptysis. Associated symptoms included unintentional weight loss, drenching night sweats, anorexia, and exertional dyspnea. He denied chest pain, orthopnea, or paroxysmal nocturnal dyspnea. The fever was intermittent and low-grade, with temporary relief after antipyretics.

His medical history was significant for microbiologically unconfirmed pulmonary tuberculosis, for which he had received and completed an 8-month first-line anti-tuberculosis regimen (rifampicin, isoniazid, pyrazinamide, and ethambutol [RIPE]) at a private hospital in 2004, five years before the current presentation. He had no history of cigarette smoking or other significant comorbidities.

On physical examination, he appeared chronically ill and cachectic with pallor and grade III digital clubbing. His body weight was 55 kg. Vital signs revealed a temperature of 37.2°C, respiratory rate of 24

breaths/min, pulse rate of 92 beats/min, and blood pressure of 90/60 mmHg. Chest examination demonstrated clinical evidence of left upper lobe fibrosis with consolidation involving the left mid and lower lung zones. Cardiovascular, abdominal, and neurological examinations were otherwise unremarkable.

Investigations and management

The initial working diagnosis was relapsed pulmonary tuberculosis (PTB) based on his previous treatment history, constitutional symptoms, and respiratory findings. In accordance with the Nigerian National Tuberculosis and Leprosy Control Programme (NTBLCP) guidelines in use at the time, empirical retreatment with the Category II anti-tuberculosis regimen (rifampicin, isoniazid, pyrazinamide, ethambutol, and injectable streptomycin) was commenced.

Baseline investigations showed three consecutive sputum specimens negative for acid-fast bacilli (AFB) on Ziehl–Neelsen microscopy. The erythrocyte sedimentation rate (ESR) was markedly elevated at 121 mm/hour, and HIV 1 and 2 screening tests were negative. Chest radiography demonstrated extensive left lung fibrosis with multiple cystic, nodular, and cavitary lesions accompanied by compensatory hyperinflation of the right lung. Other laboratory parameters are presented in Table 1.

Table 1
Laboratory parameters and radiologic findings of the patient

S/N	Investigations	Results	Normal Range
1	Full Blood Count		
	Haemoglobin (g/dl)	10.2	13–18
	Packed Cell Volume (%)	30.6	40–45
	White Blood Cell Count ($10^9/L$)	9	4-10.8
	Lymphocytes (%)	25	20–45
	Monocytes (%)	08	2–10
	Eosinophils (%)	04	1–6
	Basophils (%)	01	0–1
	Neutrophils (%)	62	40–75
	Platelets ($10^9/L$)	150	90–300
2	ESR (mm/hr)	121	5–7
3	Liver Function Test		
	Total Bilirubin ($\mu\text{mol/L}$)	5.6	< 21
	Conjugated Bilirubin ($\mu\text{mol/L}$)	3.5	< 5
	ALP (IU/L)	65	30–120
	ALT (IU/L)	8	< 15
	AST (IU/L)	15	< 35

At review on 6 March 2010, after completing the retreatment regimen, the patient continued to experience persistent respiratory symptoms despite modest clinical improvement. His body weight had increased from 55 kg to 60 kg, and he appeared less cachectic. Repeat sputum microscopy performed on two sputum specimens detected AFB in one specimen. Follow-up chest radiography demonstrated extensive unilateral destruction of the left lung, consistent with destroyed lung syndrome.

Given the persistence of symptoms despite two completed courses of anti-tuberculosis therapy and progressive structural lung destruction, drug-resistant pulmonary tuberculosis (DR-TB) was initially suspected. The patient was referred to the National Tuberculosis Reference Laboratory at the Nigerian Institute of Medical Research (NIMR), Yaba, Lagos, for mycobacterial culture and drug susceptibility testing (DST). Mycobacterial culture yielded growth of a mycobacterial species; however, DST could not be performed because of laboratory limitations. Subsequent molecular genotypic testing demonstrated

that the isolate did not belong to the *Mycobacterium tuberculosis* complex, raising suspicion for pulmonary non-tuberculous mycobacterial (NTM) disease.

In view of the extensive cavitary lung destruction, chronic constitutional symptoms, and previous pulmonary tuberculosis, chronic pulmonary aspergillosis (CPA) was also considered an important differential diagnosis because of its well-recognized association with post-tuberculous structural lung disease. Owing to limited access to advanced fungal diagnostic investigations, including *Aspergillus*-specific serology and fungal culture, a definitive diagnosis of CPA could not be established or excluded. Consequently, the patient was commenced on empirical itraconazole 200 mg orally once daily while further microbiological investigations were pursued.

Because species-level identification of the causative mycobacterium was not routinely available locally at the time, additional mycobacterial culture and molecular characterization were undertaken to establish the diagnosis. These investigations confirmed infection with a non-tuberculous mycobacterium (NTM), thereby excluding recurrent pulmonary tuberculosis.

Following confirmation of pulmonary NTM disease in 2011, treatment was initiated with rifampicin 600 mg orally once daily, clarithromycin 500 mg orally twice daily, itraconazole 200 mg orally once daily, and zinc 50 mg orally once daily. The patient showed gradual clinical improvement after starting therapy. To further exclude recurrent tuberculosis, an Xpert MTB/RIF assay performed in April 2012 was negative for the *Mycobacterium tuberculosis* complex.

Outcome and follow-up

Outcome and follow-up

Following initiation of NTM-directed therapy, the patient's constitutional symptoms improved, and he experienced partial clinical recovery. However, extensive structural destruction of the left lung resulted in chronic bronchiectatic changes. He remains under follow-up at the Respiratory Clinic of UPTH for the management of post-infectious bronchiectasis, although clinic attendance has been inconsistent. No further microbiological evidence of pulmonary tuberculosis has been documented during follow-up. Table 2 summarises the chronological sequence of major clinical events.

Table 2
Timeline of clinical presentation, investigations, diagnosis, treatment, and follow-up

Date/Period	Clinical events	Investigations	Management/Outcome
2004	Diagnosed with pulmonary tuberculosis at a private hospital.	Details of microbiological confirmation unavailable.	Completed an 8-month first-line anti-tuberculosis regimen (rifampicin, isoniazid, pyrazinamide, and ethambutol [RIPE]) with symptomatic improvement.
May–June 2009	Developed a one-month history of productive cough, intermittent low-grade fever, and weight loss,		Presented to the Medical Outpatient Clinic, University of Port Harcourt Teaching Hospital (UPTH).
22 June 2009	Chronically ill-looking, pale, with grade III digital clubbing. Respiratory examination showed left upper lobe fibrosis and left mid/lower zone consolidation.	Chest radiograph demonstrated extensive left lung fibrosis with cystic, nodular, and cavitary lesions, with compensatory hyperinflation of the right lung. ESR was 121 mm/hour. Three sputum specimens were negative for acid-fast bacilli (AFB).	Diagnosed clinically as relapsed pulmonary tuberculosis and commenced empirical Category II anti-tuberculosis regimen (rifampicin, isoniazid, pyrazinamide, ethambutol, and streptomycin) according to the national guideline in use at the time.
June 2009–February 2010	Persistent respiratory symptoms during treatment.	Serial clinical assessment.	Completed Category II anti-tuberculosis therapy.
6 March 2010	Persistent cough despite modest clinical improvement; weight increased from 55 kg to 60 kg.	Repeat sputum microscopy (two specimens) detected AFB in one specimen. Chest radiograph demonstrated destroyed left lung syndrome.	Drug-resistant pulmonary tuberculosis was suspected, and the patient was referred to the National Tuberculosis Reference Laboratory, Nigerian Institute of Medical Research (NIMR), Lagos.
2010	Evaluation at the National TB Reference Laboratory in Lagos.	Mycobacterial culture yielded growth of a mycobacterial species. Drug susceptibility testing was not performed because of laboratory limitations. Molecular genotypic testing identified a non-tuberculous mycobacterium (NTM), excluding the <i>Mycobacterium tuberculosis</i> complex.	Diagnosis revised from recurrent pulmonary tuberculosis to pulmonary NTM disease.

Date/Period	Clinical events	Investigations	Management/Outcome
2011	Persistent chronic respiratory disease.	Clinical review.	Commenced oral rifampicin 600 mg once daily and clarithromycin 500 mg twice daily, with gradual clinical improvement.
April 2012	Follow-up evaluation.	Xpert MTB/RIF assay negative for <i>Mycobacterium tuberculosis</i> complex.	Continued management as pulmonary NTM disease.
Long-term follow-up	Residual chronic respiratory symptoms secondary to structural lung damage.	Clinical and radiological follow-up.	Follow-up at the Respiratory outpatient clinic for post-infectious bronchiectasis has been irregular.

Patient perspective

I had been experiencing a persistent cough for many years, sometimes bringing up sputum, along with occasional fever and gradual weight loss. At different times, I was told it might be tuberculosis, and I received treatment, but my symptoms never fully went away. This was frustrating and confusing, especially as the illness kept affecting my daily life and ability to work.

Over time, I became worried about my health as the symptoms persisted despite treatment. I also felt discouraged having to repeatedly seek care without clear answers. When I was eventually told that my condition was due to a different infection (non-tuberculous mycobacteria), it helped me understand why previous treatments had not worked.

The treatment process has not been easy, but I feel more hopeful now that there is a clearer diagnosis and plan. I have learnt the importance of follow-up and proper evaluation. I would encourage others with long-standing cough or similar symptoms to seek further medical care if they are not improving and to ask questions about their diagnosis.

Discussion

This case illustrates the diagnostic complexity and therapeutic challenges of pulmonary non-tuberculous mycobacterial disease (NTM-LD) in a tuberculosis (TB)-endemic, resource-limited setting. It highlights how the substantial clinical and radiological overlap between pulmonary TB and NTM-LD, coupled with limited diagnostic capacity and entrenched TB-focused clinical algorithms, can delay recognition of NTM infection and contribute to irreversible pulmonary damage despite repeated healthcare encounters.

One of the principal lessons from this case is the difficulty of distinguishing pulmonary NTM disease from recurrent pulmonary TB in high-burden settings. The patient presented with chronic productive

cough, constitutional symptoms, and extensive unilateral fibrocavitary lung disease several years after completing treatment for pulmonary TB. These features appropriately raised clinical suspicion for relapsed TB and justified empirical retreatment according to the Nigerian National Tuberculosis and Leprosy Control Programme guidelines that were applicable at the time. The initial diagnosis, therefore, reflected the prevailing standard of care rather than inappropriate clinical practice. However, persistent symptoms despite completion of therapy underscored the need to reconsider alternative diagnoses, including NTM-LD and other chronic pulmonary infections.

The diagnostic pathway in this case also demonstrates the limitations of conventional microbiological investigations. Initial sputum AFB microscopy was repeatedly negative despite extensive radiographic disease, illustrating the imperfect sensitivity of smear microscopy. Subsequently, AFB was detected in one of two sputum specimens, raising concern for drug-resistant TB. Although acid-fast staining confirms the presence of mycobacteria, it cannot differentiate *Mycobacterium tuberculosis* from non-tuberculous mycobacteria. [6] Consequently, reliance on smear microscopy alone may lead to diagnostic uncertainty and inappropriate therapeutic decisions, particularly where molecular diagnostics and mycobacterial culture are not routinely available. Studies from sub-Saharan Africa suggest that a proportion of patients investigated for pulmonary TB, particularly those with discordant microbiological findings or poor response to treatment, are ultimately found to have NTM disease. [11, 12, 15]

The chronology of this case is particularly instructive. At the time of the patient's initial presentation in 2009, rapid molecular assays such as Xpert MTB/RIF were not widely available within the Nigerian TB programme. Clinical decisions therefore depended largely on symptoms, radiographic findings, treatment history, and sputum microscopy. After empirical retreatment failed and the left lung progressively deteriorated, referral to the Nigerian Institute of Medical Research enabled mycobacterial culture and molecular testing, which showed the isolate was not MTBC and established the diagnosis of pulmonary NTM disease. This sequence highlights the critical role of reference laboratories in confirming diagnoses that cannot be resolved using routine investigations available in many low-resource settings.

An additional diagnostic consideration in this patient was chronic pulmonary aspergillosis (CPA). Previous pulmonary TB, extensive fibrocavitary lung destruction, persistent respiratory symptoms, and weight loss are recognized risk factors for CPA, and the condition frequently coexists with or mimics chronic mycobacterial lung disease. Consequently, empirical itraconazole therapy was initiated because definitive fungal diagnostic tests, including *Aspergillus*-specific serology and fungal culture, were unavailable. This aspect of the case further illustrates the complexity of evaluating chronic cavitary lung disease in resource-limited settings, where multiple infectious aetiologies often coexist but comprehensive diagnostic investigations remain inaccessible.

The absence of species identification and drug susceptibility testing represents another important challenge highlighted by this case. NTM comprises more than 200 recognized species with considerable differences in pathogenicity, antimicrobial susceptibility, and recommended treatment regimens. [1–3]

Current ATS/IDSA guidelines recommend integrating compatible clinical symptoms, characteristic radiological abnormalities, and repeated microbiological confirmation, including species identification, before establishing the diagnosis of pulmonary NTM disease. [14] Species identification is particularly important because treatment differs substantially between slowly growing organisms, such as the *Mycobacterium avium* complex, and rapidly growing species, including the *Mycobacterium abscessus* complex. Without species identification and susceptibility testing, individualized therapy is difficult, and clinicians must often rely on empirical treatment based on available expertise and limited evidence.

The patient's gradual clinical improvement following rifampicin- and clarithromycin-based therapy supports the clinical relevance of the cultured NTM isolate. Nevertheless, delayed diagnosis resulted in irreversible structural lung damage manifested by destroyed left lung syndrome and chronic bronchiectasis requiring long-term respiratory follow-up. Previous pulmonary TB likely predisposed the patient to subsequent NTM infection through structural distortion of the lung, a relationship that has been increasingly recognized in immunocompetent individuals. [13] This case therefore reinforces the importance of considering NTM disease in patients with chronic respiratory symptoms and persistent radiographic abnormalities following apparently adequate treatment for pulmonary TB.

Beyond the individual patient, this case has important implications for TB control programmes in high-burden countries. Because NTM disease is not routinely considered within national diagnostic algorithms, patients with persistent symptoms may receive repeated empirical anti-tuberculosis therapy before alternative diagnoses are investigated. Such practice may expose patients to unnecessary drug toxicity, increase healthcare costs, delay appropriate treatment, and underestimate the true burden of NTM disease within national surveillance systems. [8, 9] Incorporating mycobacterial culture, molecular identification, and clear referral pathways for patients with persistent symptoms or discordant microbiological results would improve diagnostic accuracy and patient outcomes.

The principal strength of this report lies in its long-term longitudinal follow-up, spanning approximately 17 years, which provides a unique perspective on the evolution of chronic pulmonary disease within a resource-constrained healthcare system. The case also documents real-world clinical decision-making during a period when advanced molecular diagnostics were not widely available in Nigeria. However, several limitations should be acknowledged. First, species identification and drug susceptibility testing were unavailable, preventing definitive characterization of the infecting organism and limiting assessment of treatment appropriateness. In addition, the diagnosis of CPA could neither be confirmed nor excluded because fungal diagnostic investigations were unavailable. As with all single-case reports, the findings are not intended to be generalisable but rather to highlight important diagnostic and health-system challenges that warrant further investigation.

In conclusion, this case demonstrates the diagnostic complexity of pulmonary NTM disease in TB-endemic settings and emphasizes that persistent respiratory symptoms after appropriate anti-tuberculosis treatment should prompt consideration of alternative diagnoses, including NTM-LD. Strengthening laboratory capacity for mycobacterial culture, species identification, molecular

diagnostics, and drug susceptibility testing, alongside improved clinician awareness and incorporation of NTM into national TB diagnostic algorithms, will be essential to reducing diagnostic delays and improving outcomes for patients with chronic mycobacterial lung disease.

Abbreviations

AFB	Acid-fast bacilli
ATS	American Thoracic Society
CPA	Chronic pulmonary aspergillosis
DR-TB	Drug-resistant tuberculosis
DST	Drug susceptibility testing
ESR	Erythrocyte sedimentation rate
IDSA	Infectious Diseases Society of America
LMICs	Low- and middle-income countries
MTBC	<i>Mycobacterium tuberculosis</i> complex
NIMR	Nigerian Institute of Medical Research
NTBLCP	National Tuberculosis and Leprosy Control Programme
NTM	Non-tuberculous mycobacteria
NTM-LD	Non-tuberculous mycobacterial lung disease
PTB	Pulmonary tuberculosis
RIPE	Rifampicin, isoniazid, pyrazinamide, and ethambutol
TB	Tuberculosis
UPTH	University of Port Harcourt Teaching Hospital
WHO	World Health Organization
Xpert MTB/RIF	Xpert <i>Mycobacterium tuberculosis</i> /Rifampicin assay

Declarations

Clinical trial number

Not applicable

Ethics approval and consent to participate

Ethics approval was obtained from the Research and Ethics Committee of the University of Port Harcourt Teaching Hospital. Research was conducted in accordance with the Declaration of Helsinki.

Consent to publish

Written informed consent (Consent to Participate and Consent to Publish) was obtained from the patient.

Data Availability Statement

All data generated or analysed during this study are included in this published article

Competing interests

The authors declare no competing interests.

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Author contributions

CO conceptualized the study and wrote the initial manuscript; APO-I collected the data and revised the final manuscript. S.O developed the Table and collected data. All authors critically revised and approved the final manuscript for submission.

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