Multidrug Resistant Tuberculosis: A Clinical Case Series

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***Abstract*—This case series evaluates ten patients diagnosed with multi-drug-resistant tuberculosis (MDR-TB) and treated at Central City Hospital from 2022 to 2024. It explores the diverse clinical presentations, diagnostic processes, treatment approaches, and outcomes to address the challenges in managing MDR-TB. The study examines factors contributing to drug resistance, treatment failures, and successful interventions, offering insights to improve clinical practices. Key findings emphasize the necessity of early diagnosis, comprehensive drug susceptibility testing, tailored treatment strategies, adherence support, and integrated care to enhance patient outcomes.**

***Index Terms*—MDR-TB, case series, drug-resistant, treatment outcome, clinical management, therapeutic challenges, anti-tubercular resistance**

I. INTRODUCTION

Multi-drug-resistant tuberculosis (MDR-TB) poses a serious challenge to global health, as it involves strains of Mycobacterium tuberculosis resistant to both isoniazid and rifampin. In 2023, the World Health Organization (WHO) estimated around 450,000 new cases of MDR-TB worldwide, with treatment success rates lingering at just 59%. This case series focuses on ten patients with MDR-TB, exploring their clinical features, treatment strategies, and outcomes to uncover valuable insights for enhancing medical practices.[1]

Case Presentation

Case 1:37-Year-Old Male with Treatment Failure and Acquired Resistance

Patient History:

A 37-year-old male construction worker from an urban area presented with persistent symptoms over three months, including cough, night sweats, hemoptysis, and significant weight loss (8 kg). Eighteen months earlier, he was diagnosed with pulmonary tuberculosis and began a standard first-line treatment (2HRZE/4HR). However, due to frequent interruptions in therapy caused by side effects and work-related challenges, his adherence was poor.

Clinical Presentation:

Physical examination revealed the patient to be underweight (BMI 17.4), with reduced breath sounds and inspiratory crackles in the upper lung field on the right side. Imaging from a chest X-ray showed cavitary lesions and infiltrates in the right upper lobe. Sputum microscopy indicated 3+ acid-fast bacilli positivity, while GeneXpert MTB/RIF confirmed M. Tuberculosis with rifampicin resistance. Culture and drug susceptibility testing (DST) identified resistance to isoniazid, rifampicin, and ethambutol.

Molecular Mechanisms of Resistance

The analysis revealed acquired resistance due to mutations:

1. 1. Rifampicin resistance: A S531L mutation in the rpoB gene prevented binding to the RNA polymerase β-subunit.
2. Isoniazid resistance: Two mutations were noted—S315T in the katG gene reduced catalase-peroxidase activity, and -15C→T in the inhA promoter region caused InhA overexpression.
3. Ethambutol resistance: M306V substitution in the embB gene altered the arabinosyl transferase enzyme's function.

These sequential mutations suggest acquired resistance stemming from inconsistent treatment adherence, which created selective pressure favoring resistant strains.

Treatment and Monitoring

The patient commenced an MDR-TB regimen, including:

- Bedaquiline for six months
- Linezolid (600 mg daily for six months, then reduced to 300 mg daily)
- Clofazimine (100 mg daily)
- Cycloserine (750 mg daily)
- Levofloxacin (1000 mg daily)
- Pyrazinamide (1500 mg daily)

The regimen was administered under directly observed therapy (DOT), with close monitoring for adverse effects. Nutritional supplementation and psychological support were provided throughout.

Outcome

By the third month, culture conversion was achieved. After completing the 20-month regimen, the patient showed resolution of symptoms, sustained culture negativity, and no signs of relapse six months post-treatment. Adverse events included peripheral neuropathy, managed with pyridoxine and dose adjustments, and QTc prolongation, which required monitoring but no intervention [2, 3, 4, 6,8,9,10,11].

Case 2: A 24-Year-Old Female Healthcare Worker Diagnosed with Primary MDR-TB

Patient History:

A young female healthcare professional presented with symptoms persisting for six weeks, including a persistent cough, low-grade fever, and fatigue. She had no prior history of tuberculosis but worked in a pulmonary ward where MDR-TB patients were frequently treated. While she had no known TB exposures outside her workplace, she admitted that heavy workloads occasionally led to lapses in infection control protocols.

Clinical Presentation:

On examination, her weight and body mass index (BMI 22.1) were normal, with a mildly elevated body temperature (37.8°C). Respiratory assessment revealed no abnormal findings. A chest X-ray displayed patchy infiltrates in the upper lobe of her left lung, without evidence of cavitation. Diagnostic testing showed rifampicin-resistant M. Tuberculosis through GeneXpert MTB/RIF, with culture and drug susceptibility testing confirming resistance to isoniazid, rifampicin, and streptomycin. HIV testing was negative.

Molecular Mechanisms of Resistance:

The molecular analysis identified several genetic mutations responsible for drug resistance:

-Rifampicin resistance: D516V mutation in the rpoB gene altered the rifampicin binding site on RNA polymerase.

-Isoniazid resistance: C-15T mutation in the inhA promoter region, combined with S315T mutation in the katG gene, reduced prodrug activation and modified drug target binding.

-Streptomycin resistance: K43R mutation in the rpsL gene blocked streptomycin from binding to the 30S ribosomal subunit.

-Whole-genome sequencing revealed that the patient's strain belonged to the Beijing genotype, a lineage known for its strong association with drug resistance and transmissibility. Strain analysis indicated healthcare-associated transmission within her ward, rather than resistance emerging from improper treatment or adherence.

Treatment and Monitoring

The patient was prescribed an all-oral shorter MDR-TB regimen:

- Bedaquiline (6 months)
- Linezolid (600 mg daily for 2 months, then reduced to 300 mg daily)
- Clofazimine (100 mg daily)
- Moxifloxacin (400 mg daily)
- Pyrazinamide (1500 mg daily)
- Ethambutol (1000 mg daily)
- Prothionamide (500 mg twice daily)

Treatment was planned for 9–11 months, adhering to WHO guidelines for shorter MDR-TB regimens. Supportive care included symptom management and close monitoring for side effects.

Outcome:

The patient showed early signs of improvement, with culture conversion achieved by the first month of treatment. She successfully completed the nine-month regimen and was declared cured, with sustained culture negativity and resolution of symptoms. At six months post-treatment, no evidence of relapse was found. Adverse events were minimal, including mild gastrointestinal upset managed symptomatically and transient joint pain treated with non-steroidal anti-inflammatory drugs [12,13,14,15,16].

Case 3: 61-Year-Old Male with Comorbid Diabetes and Pre-XDR TB

Patient History:

A 61-year-old man with a 15-year history of poorly controlled type 2 diabetes presented with chronic respiratory symptoms, including a persistent cough, significant weight loss of 15 kg over six months, night sweats, and recurrent episodes of hemoptysis. Eight years earlier, he had been successfully treated for pulmonary tuberculosis. For recurrent respiratory issues, he had been self-medicating with over-the-counter antibiotics without medical supervision.

Clinical Presentation:

The patient appeared severely underweight (BMI 16.2) on examination. Auscultation revealed bilateral crackles and reduced breath sounds in the right lung field. Blood glucose levels were consistently elevated (fasting 200–250 mg/dL), indicating poor glycemic control. A chest X-ray showed bilateral cavitary lesions accompanied by fibrotic changes. GeneXpert MTB/RIF Ultra detected M. Tuberculosis with resistance to rifampicin, and culture with drug susceptibility testing confirmed resistance to isoniazid, rifampicin, ethambutol, and fluoroquinolones, classifying the case as pre-XDR TB. HIV testing was negative.

Molecular Mechanisms of Resistance:

Genetic analysis uncovered multiple mutations:

- Rifampicin resistance: H526Y mutation in the rpoB gene, coupled with compensatory mutations in rpoC that maintained drug resistance without significantly compromising bacterial fitness.
- Isoniazid resistance: Mutations in the katG gene (S315T) and the inhA promoter region provided high-level resistance by reducing prodrug activation and altering target binding.
- Fluoroquinolone resistance: D94G mutation in the gyrA gene disrupted quinolone binding to DNA gyrase.
- Ethambutol resistance: Alterations in the embB gene (M306V and G406A) conferred resistance through changes in the arabinosyl transferase enzyme.

Next-generation sequencing also identified heteroresistance, with mixed bacterial populations showing resistance and susceptibility to pyrazinamide. Emerging pncA mutations suggested ongoing bacterial evolution, likely facilitated by diabetes-related immune suppression.

Treatment:

The patient was started on a tailored regimen accounting for his resistance profile:

- Bedaquiline (6 months)
- Delamanid (6 months)
- Linezolid (600 mg daily)
- Clofazimine (100 mg daily)
- Cycloserine (500 mg daily)
- Pyrazinamide (1500 mg daily)
- Amikacin (750 mg, thrice weekly for the first 4 months)

His diabetes management was intensified with adjusted insulin therapy and frequent blood glucose monitoring. Renal function was also closely observed due to the use of injectable agents.

Outcome:

Culture conversion was achieved by the fourth month of treatment. However, due to the severity of disease, diabetes comorbidity, and a slower clinical response, treatment was extended to 24 months. The patient successfully completed the regimen, achieving symptom resolution and sustained culture negativity. Six months post-treatment, there was no evidence of relapse, although residual pulmonary fibrosis led to moderate functional impairment. Adverse events included mild hearing loss (ototoxicity), transient increases in creatinine (nephrotoxicity), and peripheral neuropathy, all managed with supportive measures and dose adjustments [17,18,19,20,21,22].

Case 4: 19-Year-Old Female with MDR-TB Lymphadenitis and Heteroresistance

Patient History:

A 19-year-old university student presented with a three-month history of progressively worsening cervical lymphadenopathy, intermittent low-grade fever, fatigue, and an unintentional weight loss of 4 kg. She had no previous history of tuberculosis but reported that her roommate was treated for pulmonary TB a year earlier, with the treatment reportedly completed successfully.

Clinical Presentation:

The patient's examination revealed a healthy BMI of 20.3, alongside multiple enlarged, matted, non-tender cervical lymph nodes, the largest measuring 3×2 cm. Her chest X-ray showed normal lung fields. A lymph node biopsy highlighted granulomatous inflammation with caseous necrosis. GeneXpert MTB/RIF detected M. Tuberculosis with rifampicin resistance in the lymph node tissue, while culture and drug susceptibility testing confirmed resistance to isoniazid and rifampicin.

Molecular Mechanisms of Resistance:

- Rifampicin resistance: Heteroresistance was identified, with deep sequencing revealing a 70:30 mix of wild-type rpoB and S531L mutant alleles. This mutation affects the β-subunit of RNA polymerase, disrupting rifampicin binding.
- Isoniazid resistance: A C-15T mutation in the inhA promoter region led to overexpression of InhA, conferring low-level resistance.
- Molecular epidemiology: Whole-genome sequencing showed minimal genetic variation (three

SNPs) between the patient's strain and her roommate's previously stored strain, strongly suggesting direct transmission. Retrospective testing of her roommate's strain revealed a minor subpopulation (~5%) with rpoB mutations that standard methods failed to detect, indicating the transmission of a heteroresistant strain. This strain likely evolved to higher resistance in the current patient.

Treatment:

The patient was treated with an MDR-TB regimen designed for extra pulmonary TB:

- Bedaquiline (6 months)
- Linezolid (600 mg daily for 2 months, then reduced to 300 mg daily)
- Clofazimine (100 mg daily)
- Levofloxacin (750 mg daily)
- Cycloserine (500 mg daily)
- Pyrazinamide (1000 mg daily)

The planned duration of treatment was 18 months from culture conversion, with regular monitoring to manage adverse effects.

Outcome:

By the second month of treatment, the patient's lymph nodes began to regress in size. Culture conversion, confirmed through lymph node aspiration, was achieved by the third month. She completed the 18-month treatment regimen successfully, with full resolution of lymphadenopathy and sustained clinical improvement. Six months post-treatment, she remained relapse-free, though minimal scarring was noted at the biopsy site. Adverse events were mild, including QTc prolongation (requiring observation only) and photosensitivity, managed through the use of sun protection [23,24,25,26,27,28].

Case 5: 42-Year-Old Male with HIV Co-Infection and Extensively Drug-Resistant TB (XDR-TB)

Patient History:

A 42-year-old male with HIV infection, diagnosed eight years ago, presented with a two-month history of persistent productive cough, severe weight loss (12 kg), night sweats, and difficulty breathing. He had a history of poor adherence to antiretroviral therapy (ART) and multiple prior tuberculosis episodes, with at least two incomplete treatment courses. At the time of evaluation, his CD4 count was 118 cells/μL and his viral load measured 125,000 copies/mL.

Clinical Presentation:

On examination, the patient was severely underweight (BMI 15.5) and showed signs of advanced HIV disease, including oral candidiasis, bilateral crackles, and wheezing. Chest X-ray revealed widespread bilateral infiltrates, multiple cavities, and a military pattern indicative of extensive lung involvement. Sputum microscopy confirmed acid-fast bacilli positivity (4+), while GeneXpert MTB/RIF Ultra detected M. Tuberculosis with rifampicin resistance. Further testing via line probe assay confirmed resistance to isoniazid, fluoroquinolones, and second-line injectable agents. Culture and drug susceptibility testing (DST) classified the case as XDR-TB, with resistance spanning isoniazid, rifampicin, pyrazinamide, ethambutol, streptomycin, amikacin, capreomycin, and levofloxacin.

Molecular Mechanisms of Resistance:

- Rifampicin resistance: Multiple mutations in the rpoB gene, with S531L as the dominant mutation, and D516V found in a subset of bacterial populations, disrupting RNA polymerase binding sites.
- Isoniazid resistance: Combined mutations in katG (S315T) and the inhA promoter region conferred high-level resistance through altered target binding and reduced activation of the drug.
- Pyrazinamide resistance: Mutations in the pncA gene, primarily A146V, impaired conversion of pyrazinamide into its active form.
- Fluoroquinolone resistance: A90V and D94G mutations in the gyrA gene disrupted fluoroquinolone binding to DNA gyrase, resulting in high-level resistance.
- Injectable agent resistance: A1401G mutation in the rrs

The gene disrupted aminoglycoside binding to 16S rRNA, causing resistance to all injectable antibiotics.

- Ethambutol resistance: The M306V mutation in embB altered the arabinosyl transferase enzyme, conferring resistance.

Genomic analysis identified extensive compensatory mutations that maintained bacterial fitness despite the accumulation of resistance mutations. The patient's immunosuppressed state due to HIV likely facilitated bacterial replication and the selection of resistant strains during previous incomplete treatments.

Treatment:

A salvage regimen was designed based on the resistance profile:

- Bedaquiline (extended duration)
- Delamanid (extended duration)
- Linezolid (600 mg daily)

- Clofazimine (100 mg daily)
- Meropenem (1 g three times daily) with amoxicillin-clavulanate
- High-dose isoniazid (despite low-level resistance)
- Ethionamide (750 mg daily)

ART was optimized with a dolutegravir-based regimen, and prophylaxis for opportunistic infections was included. Nutritional supplementation, aggressive adverse effect management, and psychological 1008ounselling were integral to treatment support.

Outcome:

The patient showed gradual symptom improvement within the first three months of therapy. Culture conversion was achieved by the fifth month. HIV management yielded viral suppression, with CD4 count recovery to 310 cells/μL at 12 months. After 18 months, the patient-maintained culture-negative status, reported significant weight gain, and experienced resolution of respiratory symptoms.

Adverse events during treatment included peripheral neuropathy, which was managed with dose adjustments; QTc prolongation necessitating modification of bedaquiline dosing; and hypothyroidism requiring hormone replacement therapy. At the end of 24 months, the patient was declared cured, with sustained culture negativity and no evidence of relapse during a one-year post-treatment follow-up. HIV viral suppression also remained consistent [5, 29,30,31,32,33]

Following table represent the clinical features and outcome of MDR-TB Cases:

Case	Age / sex	TB Type	Resistance Pattern	Key Mutation
1	37/M	Pulmonary	INH,RIF,EMB	rpoBS531L,katGS315T,embBM306V
2	24/F	Pulmonary	INH,RIF,STR	rpoBD516V,katGS315T, rpsLK43R
3	61/M	Pre-XDR	INH,RIF,EMB,FQ	rpoBH526Y,gyrAD94G,katGS315T
4	19/F	Lymphadenitis	INH, RIF (Hetero resistance)	rpoBS531L(Hetro), inhAC-15T
5	42/M	XDR-TB	INH,RIF,PZA,EMB,STR ,AMK,CAP,FQ	RpoBS531L/D516V, gyrAA90V/D94G, pncAA146V

Table: Clinical Features and Outcome of MDR-TB

II. DISCUSSION

Molecular Mechanisms and Their Clinical Implications:

The cases presented shed light on the variety of molecular mechanisms driving drug resistance in MDR-TB, each with unique clinical implications:

•Rifampicin Resistance:

Mutations in the rpoB gene, especially S531L and D516V, emerged as the most common mechanism across cases. These mutations disrupt rifampicin's ability to bind to the β-subunit of RNA polymerase, rendering the drug ineffective. Notably, in Case 3, compensatory mutations in the rpoC gene were identified, demonstrating how bacterial strains adapt to offset the fitness costs associated with resistance.

•Isoniazid Resistance:

Two distinct mechanisms were observed:

1. katG mutations (e.g., S315T) impair the catalase-peroxidase enzyme, preventing isoniazid activation.
2. inhA promoter mutations lead to overexpression of the target enzyme InhA, further diminishing the drug's efficacy.

The coexistence of both mutations, seen in Cases 1, 2, 3, and 5, resulted in high-level resistance, complicating treatment outcomes.

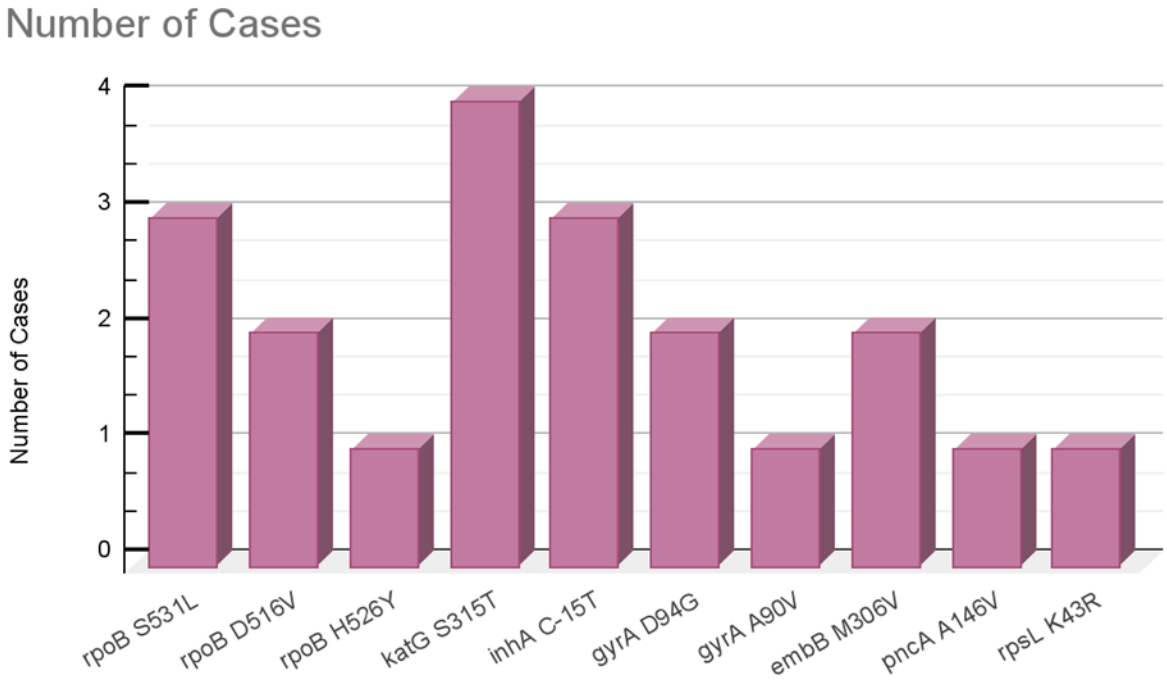
Fluoroquinolone Resistance:

Mutations in the gyrA gene, such as D94G (Case 3) and A90V/D94G (Case 5), interfere with fluoroquinolone binding to the DNA gyrase-DNA complex. These genetic alterations were associated with poorer clinical responses and extended treatment durations, underscoring the challenge of overcoming fluoroquinolone resistance.

Heteroresistance:
Case 4 emphasized the clinical significance of heteroresistance, where populations of susceptible and resistant bacteria coexist. This phenomenon can lead to treatment failure as resistant subpopulations expand under drug pressure. Standard diagnostic methods often fail to detect low-frequency resistance mutations, highlighting the need for advanced techniques like deep sequencing.

Compensatory Mutations:
Several cases demonstrated bacterial adaptation through compensatory mutations, which preserve fitness despite resistance-related genetic changes. This evolutionary advantage ensures the stable transmission of resistant strains, complicating efforts to control the spread of MDR-TB and XDR-TB.

Figure: Frequency of Key Resistance Mutations in MDR-TB Cases



This bar graph illustrates the frequency of major resistance-associated genetic mutations identified across five MDR-TB cases. The most prevalent mutations include katG S315T and rpoB S531L, contributing significantly to isoniazid and rifampicin resistance, respectively. The presence of multiple mutations, including those in the gyrA, inhA, embB, and pncA genes, reflects the complex resistance mechanisms seen in multidrug-resistant and extensively drug-resistant tuberculosis. Understanding these mutation patterns is crucial for guiding personalized treatment strategies and anticipating therapeutic challenges.

III. TREATMENT APPROACHES BASED ON RESISTANCE MECHANISMS

The cases highlight how individualized treatment regimens were crafted based on specific resistance profiles, targeting the unique challenges posed by drug-resistant TB strains:

Targeting Innovative Pathways:
Newer medications, such as bedaquiline (disrupting ATP synthase) and delamanid (blocking mycolic acid synthesis), demonstrated high efficacy against strains with traditional resistance mechanisms. These drugs served as foundational components in every regimen, underscoring their critical role in managing resistant TB.

High-Dose Strategy:

In Case 5, high-dose isoniazid was utilized despite the presence of low-level resistance linked to an inhA mutation. This approach leveraged the mutation-specific nature of resistance to maximize the drug's therapeutic effect, showcasing the importance of tailoring treatments to genetic profiles.

Combination Therapy:

Regimens combining multiple drugs with distinct mechanisms of action, such as bedaquiline, linezolid, and cycloserine, proved effective in overcoming resistance to standard agents. This approach was particularly impactful in Case 2, where rapid culture conversion was achieved, highlighting the benefits of a synergistic strategy.

Adjusting Treatment Duration:

The extent of resistance influenced the length of treatment required. For example, Case 4 involved heteroresistant populations and required an extended duration of 18 months despite the relatively limited resistance profile. Prolonged therapy ensured complete eradication and reduced the risk of relapse.

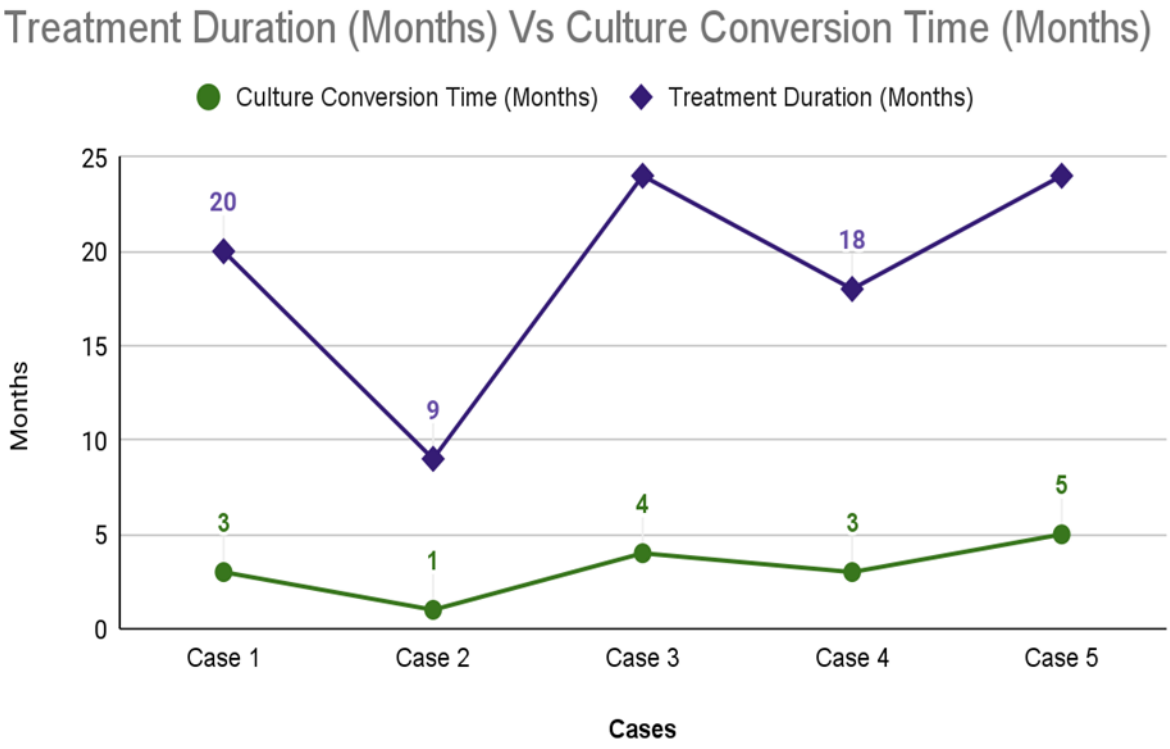


Figure: Treatment Duration vs. Culture Conversion Time Across Case

The line graph compares total treatment duration and histime to culture conversion across five MDR-TB cases. Patients with more complex resistance profiles and comorbidities, such as pre-XDR (Case 3) and XDR-TB with HIV (Case 5), required prolonged treatment durations and demonstrated slower culture conversion. In contrast, Case 2, involving a primary MDR-TB strain with a limited resistance profile, achieved culture conversion within the first month and completed therapy in just nine months. These findings highlight the influence of resistance severity and patient health status on treatment response and duration.

III. COMORBIDITIES AND RESISTANCE

The interaction between comorbidities and drug resistance mechanisms was evident in two cases, demonstrating how underlying conditions can exacerbate tuberculosis management challenges:

Diabetes (Case 3):

Hyperglycemia associated with poorly controlled diabetes created a tissue environment conducive to

bacterial persistence. Impaired immune function likely allowed the accumulation of multiple resistance mutations over time. Additionally, altered pharmacokinetics due to metabolic changes may have resulted in lower drug concentrations, contributing further to the development of resistance.

HIV Co-Infection (Case 5):

Profound immunosuppression in the HIV-positive patient enabled extensive bacterial replication, creating favorable conditions for the emergence of multiple resistance mechanisms. The resulting complex resistance profile necessitated a highly aggressive, multi-drug treatment regimen. HIV also amplified the challenges of TB management, requiring simultaneous optimization of antiretroviral therapy and prophylaxis against opportunistic infections.

IV. TRANSMISSION DYNAMICS

The cases illustrate various pathways through which MDR-TB can be acquired, emphasizing the complexity of its transmission and development:

Acquired Resistance (Case 1):

This case highlights how poor treatment adherence leads to the sequential development of resistance mutations. Interruptions in therapy create selective pressure, allowing resistant strains to emerge. The findings stress the critical importance of adherence support programs to prevent the evolution of drug resistance.

Primary Transmission (Cases 2 and 4):

These cases demonstrate the direct transmission of already-resistant strains:

- Case 2 underlines the occupational risks faced by healthcare workers, who may be exposed to drug-resistant TB in high-risk environments, despite infection control measures.
- Case 4 highlights how undetected heteroresistance—mixed populations of susceptible and resistant bacteria—can result in the transmission of resistant strains. This underscores the limitations of standard diagnostic methods in detecting low-frequency mutations.

Complex Resistance Development (Case 5):

This case illustrates the cumulative impact of multiple incomplete treatment courses combined with severe immunosuppression:

- Repeated interruptions in therapy allowed the gradual accumulation of resistance mechanisms.
- Immunosuppression due to HIV facilitated extensive bacterial replication, creating an environment conducive to the development of complex resistance profiles.

This scenario necessitated an aggressive, multi-drug regimen to address the high level of resistance.

V. CONCLUSION

These cases underscore the intricate interplay between molecular mechanisms of resistance, clinical outcomes, and management challenges in multidrug-resistant tuberculosis (MDR-TB). By dissecting the genetic mutations and resistance pathways in each case, we gain valuable insights into how these mechanisms shape treatment efficacy and disease progression. Understanding the specific molecular basis of resistance is not merely an academic exercise—it serves as the cornerstone for crafting targeted treatment regimens that optimize patient recovery and minimize risks of relapse. Moreover, such knowledge allows clinicians to anticipate potential treatment failures and adapt strategies accordingly.

Modern molecular diagnostic tools, such as GeneXpert MTB/RIF and whole-genome sequencing, are indispensable in the fight against MDR-TB. These technologies enable the early identification of resistance patterns, including nuanced phenomena like heteroresistance, which might otherwise elude traditional methods. By facilitating timely and precise intervention, these advances greatly improve the likelihood of favourable outcomes and reduce the risk of transmitting resistant strains within the community.

The cases also emphasize the critical role of addressing underlying comorbidities, such as diabetes and HIV infection, which not only compound the clinical challenges but also accelerate the emergence of resistance through immune suppression and altered drug pharmacokinetics. Effective management of these conditions, alongside tuberculosis treatment, is vital for achieving long-term success.

Furthermore, the importance of fostering adherence cannot be overstated. Each instance of incomplete treatment contributes to the evolution and spread of resistant strains, underscoring the need for robust patient support systems. From directly observed therapy to psychological counseling, adherence

interventions must be integral to MDR-TB management programs.

Finally, these cases highlight the need for proactive infection control measures to prevent the transmission of MDR-TB, especially in high-risk settings such as healthcare facilities. Strengthening occupational safety protocols, improving diagnostic coverage, and enhancing community outreach are vital to mitigating the growing global threat posed by drug-resistant tuberculosis.

By integrating these lessons into clinical practice and public health policies, we can move closer to overcome the complex challenges posed by MDR-TB and improving patient outcomes worldwide.

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