RESEARCH PAPER

Dual Diagnostic Dilemmas in Tuberculosis: Mrsa Co-Infection Masking Disease Severity in Extrapulmonary and Spinal Tb

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Introduction

Background

Tuberculosis (TB) remains a major health issue, which is particularly in its extrapulmonary and spinal forms often delayed[1]. The presence of mixed infection compliance clinical appraisal with Methicillin-resistant Staphylococcus aureus (MRSA) is evolving as a specific contributor to diagnostic unpredictability. Specifically, in extrapulmonary and spinal TB symptoms are like abscesses and pain formation, which often resemble bacterial infections, forming it difficult to recognize between primary TB and secondary MRSA involvement. Additionally, standard diagnostic approaches fail to detect dual infection at an early stage, which escalates the risk of complications and lengthy disease progression. This research investigates the role of MRSA co-infection in obscuring TB severity and its meaning for clinical decision-making.

Aim

This study aim is to analyse dual diagnostic dilemmas in tuberculosis of MRSA co-infection masking disease severity in extrapulmonary and spinal TB.

Objectives

- To analyse radiological aspects of tuberculosis, MRSA co-infection that add to diagnostic ambiguity with the overlapping clinical facts.
- To evaluate the impact of MRSA co-infection on diagnostic accuracy and highlighting delays and misdiagnosis in extra-thoracic, spinal tuberculosis cases.
- To identify the effects of dual infection on treatment outcomes, disease progression, and overall patient prognosis.
- To provide complete and integrated diagnostic and supervision strategies to improve premature detection and diagnostic decision-making.

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Question

- How do overlapping clinical and radiographic features of tuberculosis and MRSA co-infection support to diagnostic vagueness?
- What is the shape of MRSA co-infection on diagnostic correctness, including delays and clinical error in extrapulmonary and spinal tuberculosis cases?
- How does dual infection affect management outcomes, disease development, and overall patient prediction?
- What consolidated diagnostic and management frameworks can improve early diagnosis and clinical decision-making?

Problem Statement

MRSA co infection in extrapulmonary and spinal tuberculosis yields clinical and scan-based features to diagnosed vagueness, inappropriate management that makes true disease severity, patient consequences about health situations.

Literature

Tuberculosis (TB) is a bacterial disease condition that is caused by Mycobacterium tuberculosis that commonly modifies the lungs in the human body[2]. These occur in extrapulmonary produces like lymph nodes, bones, and the spine. Spinal TB is a serious form that also leads to deformity and neurological medical consequences. MRSA (Methicillin-resistant Staphylococcus aureus) is a drug-resistant bacterial contamination often associated with clinic and community-acquired disease states. Thus, MRSA co-exists with TB, building a dual identifying quandary. Both infections highlight similar symptoms such as pain, irritation, furuncle development and fever[3]. This overlap makes it difficult to specify the primary disease. MRSA masks the true severity of TB, which is particularly in extrapulmonary and spinal cases. Hospitalized TB patients in high-burden areas and MRSA nasal carriage were found as 21% of patients upon admission, with more potential acquired in the hospital. Also known is that roughly 10.7 million people fell in with TB globally in 2024. The global prevalence of MRSA among all S. aureus infections is so high, and its estimates suggest 20% to 30% of hospital-acquired S. aureus infections are methicillin-resistant.

Method

This research chose primary data collection to examine the collective information through specific case studies. Here, this research also adopts a quantitative design from the prescription of two selected patients presenting extrapulmonary and special clinical information[4]. Thus, this research includes symptoms, diagnostic tests, and imaging findings is systematically extracted from the records. The analysis focuses on overlapping clinical features that contribute to diagnostic ambiguity. Furthermore, this study chooses an abductive comparative approach that evaluates the difference between the initial diagnosis and treatment modifications. Particular attention is given to delays and data interpreted through systematic analysis to identify consistent patterns across both cases and to identify the main research targets[5]. Here, this research chooses primary data capture of real-time clinical evidence directly from patient

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perception. Case study-based research provides strong and authentic evidence compared to secondary data and sources. It allows for the identification of actual diagnostic errors and using real cases reflects practical challenges faced in clinical settings.

Results

Patient 1:

Table 1: Complete Blood Count (CBC) with Trend Analysis

Sample	Test Description	Observed Value	Units	Bio-Reference	Method	Trend / Status
Whole blood	Hemoglobin	9.2	g/dl	12.0 – 15.0	Colorimetric method non cyanide (automated)	⬇️ Low
Whole blood	Haematocrit (PCV)	27.6	%	36.0 – 46.0	Calculated method (Automated)	⬇️ Low
Whole blood	RBC Count	3.19	Millions/cumm	3.8–4.8	Sheath Fluid Impedance	⬇️ Low
Whole blood	MCV	86.6	fl	83–101	Calculated from RBC Histogram	→ Normal
Whole blood	MCH	29.0	pg	27–32	Calculated	→ Normal
Whole blood	MCHC	33.3	%	31.5 – 34.5	Calculated	→ Normal
Whole blood	RDW (CV %)	16.1	%	11–16	Calculated	⬆️ Slightly High
Whole blood	RDW (SD)	51.2	fl	35.0 – 56.0 fl	Calculated	→ Normal
Whole blood	Total WBC Count	10.11	x10⁹/L	4.0–10.0	Laser Flow Cytometry	⬆️ Slightly High

This table highlights hematological parameters, which show mild borderline leukocytosis. Hemoglobin (9.2g/dl), hematocrit (27.6%) and RBC count as 3.19 million/cu mm that is infected with tuberculosis. WBC count is 10.11×10⁹/L, which is slightly elevated, suggesting ongoing inflammation. Others, MCV, MCH and MCHC remain within normal range, which refers to normocytic, normochromic anemia commonly in chronic disease states like TB with possible MRSA co-infection.

Table 2: Differential Leukocyte Count and Platelet Profile with Trend Analysis

Sample	Test Description	Observed Value	Units	Bio-Reference	Method	Trend / Status
Whole blood	Monocytes	3.9	%	2–10	Laser flow cytometry (Auto	→ Normal

					mated)	
Whole blood	Eosinophils	0.1	%	1–6	Automated	⬇ Low
Whole blood	Basophils	0.0	%	<1	Automated	→ Normal
Whole blood	Absolute Neutrophils Count	5.68	×10⁹/L	2–7	Calculated method	→ Normal
Whole blood	Absolute Lymphocyte Count	0.74	×10⁹/L	1–3	Calculated method	⬇ Low
Whole blood	Absolute Monocyte Count	0.26	×10⁹/L	0.09–0.8	Calculated method	→ Normal
Whole blood	Absolute Eosinophil Count	0.01	×10⁹/L	0.02–0.5	Calculated method	⬇ Low
Whole blood	Absolute Basophil Count	0.00	×10⁹/L	0.02–0.1	Calculated method	⬇ Low
Whole blood	Platelet Count	128	×10³/µL	150–410	Sheath fluid impedance	⬇ Low

This table highlights differential leukocyte and platelet profile about mixed inflammatory and immunosuppressed pattern. Monocytes (3.9%) are within the normal range, while eosinophils (0.1%) and basophils (0%) are reduced, which indicates stress-related medullary tissue concerns. Absolute neutrophil count (5.68×10⁹/L) remains high, indicating an active bacterial inflammatory reaction with MRSA contamination. Furthermore, lymphocyte count (0.74×10⁹/L) is low and reflects lymphopenia often seen in tuberculosis and chronic infection. Also, mild thrombocytopenia (128×10³/µL) suggests marrow suppression, infection or drug effects.

Table 3: Follow-up Differential Count and Platelet Profile with Trend Analysis

Sample	**Test Description**	**Observed Value**	**Units**	**Bio. Reference**	**Method**	**Trend / Status**
Whole blood	Monocytes	2.1	%	2–10	laser flow cytometry (Automated)/Manual	→ Normal
Whole blood	Eosinophils	0.0	%	1–6	laser flow cytometry (Automated)/Manual	⬇ Low
Whole blood	Basophils	0.0	%	<1	Laser flow cytometry (Automated)/Manual	→ Normal
Whole blood	Absolute Neutrophils Count	6.95	×10^9/L	2–7	Calculated method	→ High-Normal

Whole blood	Absolute Lymphocyte Count	0.37	×10^9/L	1–3	Calculated method	⬇️ Low
Whole blood	Absolute Monocyte Count	0.16	×10^9/L	0.08–0.8	Calculated method	→ Normal
Whole blood	Absolute Eosinophil Count	0.00	×10^9/L	0.02–0.5	Calculated	⬇️ Low
Whole blood	Absolute Basophil Count	0.00	×10^9/L	0.02–0.1	Calculated	⬇️ Low
Whole blood	PLATELET COUNT	103 (Few large platelet seen)	×10^3/cumm	150 – 410	Manual method	⬇️ Low

The above table shows a low lymphocyte count (0.37×10^9/L) and a reduced platelet count (103×10^3/cu mm), both of which are outside reference ranges, alongside normal neutrophils. Such as hematological abnormalities overlap with tuberculosis-related immune suppression and MRSA induced cytopenia, complicating diagnosis. In extrapulmonary, these overlaps as delay recognition with blur radiological distinctions and foster misdiagnosis. Dual infection worsens prognosis by prolonging treatment, and integrated diagnostic strategies combining microbiology and hematology for perfect management.

Table 4: Renal Function Test and Serum Electrolytes with Trend Analysis

Sample	**Test Description**	**Observed Value**	**Units**	**Bio. Reference**	**Method**	**Trend / Status**
Serum	Urea	58.72	mg/dL	12.84 – 42.8	GLDH-Urease	⬆️ High
Serum	CREATININE	0.60	mg/dL	M: 0.9–1.3 / F: 0.6–1.1	Sarcosine Oxidase	→ Normal (Female) / ⬇️ Low (Male)
Serum	Uric Acid	1.88	mg/dL	M: 4.4–7.6 / F: 2.3–6.6	Uricase – PAP	⬇️ Low
Serum	Electrolytes	—	—	—	—	—
Serum	Sodium	142.5	mEq/L	136 – 145	ISE	→ Normal
Serum	Potassium	3.47	mEq/L	3.5 – 5.1	ISE	⬇️ Slightly Low
Serum	Chloride	107.9	mEq/L	96 – 106	ISE	⬆️ Slightly High

This table covers elevated Urea (58.72 mg/dL) and low Uric Acid (1.88 mg/dL), where its highlights renal stress common in both TB wasting and MRSA sepsis. Hypokalemia (3.47 mEq/L) and hyperchloremia (107.9 mEq/L) suggest metabolic disturbances, which affect prognosis. Thus, TB is a non-specific marker that often leading to

misdiagnosis delays. Its integrated strategies parameters closely as anti-TB medications and MRSA inflammation synergistically worsen kidney function and patient outcomes.

Table 5: Complete Blood Count (CBC) and Differential Count with Trend Analysis

Sample	Test Description	Observed Value	Units	Bio. Reference	Method	Trend / Status
Whole blood	Hemoglobin	8.8	g/dL	12.0–15.0	Colorimetric method non cyanide (automated)	⬇️ Low
Whole blood	Haematocrit (PCV)	25.9	%	36.0–46.0	Calculated method (Automated)	⬇️ Low
Whole blood	RBC Count	3.04	Millions/cmm	3.8–4.8	Sheath Fluid Impedance	⬇️ Low
Whole blood	MCV	85.1	fL	83–101	Calculated from RBC Histogram	→ Normal
Whole blood	MCH	28.9	pg	27–32	Calculated	→ Normal
Whole blood	MCHC	34.0	%	31.5–34.5	Calculated	→ Normal
Whole blood	RDW (CV %)	15.6	%	11–16	Calculated	→ Normal (upper range)
Whole blood	RDW (SD)	48.9	fL	35.0–56.0 fL	Calculated	→ Normal
Whole blood	Total WBC Count	6.69	10^9/L	4.0–10.0	Laser Flowmetry Cytometry	→ Normal
Whole Blood	Differential Count	—	—	—	—	—
Whole Blood	Neutrophils	84.9	%	20–80	Laser Flow Cytometry (Automated)/Manual	⬆️ High
Whole blood	Lymphocytes	11.1	%	22–44	Laser Flow Cytometry (Automated)/Manual	⬇️ Low

The laboratory data demonstrate the diagnostic challenges of TB/MRSA co-infection. The seven anemia (Hb 8.8 g/dL) and high Neutrophil to Lymphocyte Ratio (-7.6) produce ambiguity as healthcare providers might emphasize acute bacterial sepsis while disregarding indolent spinal TB. This led to misdiagnosis and delayed treatment. Such of dual infection worsen prognosis by accelerating immune exhaustion and complicating drug induced metabolic strain. Definitive diagnosis was achieved through CSF analysis following lumbar puncture. The findings were crucial in identifying central nervous system infection. This

investigation played a decisive role where initial laboratory tests and imaging were inconclusive.

Table 6: CSF Fluid Analysis

Sample	Test Description	Observed Value	Units	Bio. Reference	Method	Trend / Status
CSF	CSF Fluid for Total Count	59	Cells/cumm	—	—	Slightly Elevated
CSF	Differential WBC Count					—
	Neutrophils	01	%	—	Manual	Low
	Lymphocytes	99	%	—	Manual	High

CSF analysis shows elevated cell count (59 cells/cumm) with marked lymphocytic predominance (99%) and minimal neutrophils (1%), suggesting non-bacterial meningitis, likely viral or tubercular, requiring further clinical correlation. CSF fluid analysis checks the fluid around your brain and spine for infection or disease. Results show increased cells with lymphocytes, suggesting possible viral or tuberculosis infection; further tests are needed.

The patient presented with decreased responsiveness, fever, and a chest wall abscess, suggesting a serious systemic infection. Wound and blood culture revealed MRSA, and antibiotics were initiated accordingly. However, imaging showed no significant findings, creating diagnostic uncertainty. Despite appropriate treatment, the patient's condition did not improve, raising suspicion of an underlying or coexisting condition. This highlighted the limitation of relying only on initial tests and imaging in complex infections.

Definitive diagnosis was achieved through CSF analysis, where GeneXpert was positive, confirming tuberculous meningoencephalitis. This explained the neurological deterioration and emphasized the importance of advanced investigations in identifying central nervous system tuberculosis.

Patient 2:

Table 7: Differential Leukocyte Count and Platelet Profile with Trend Analysis

Sample	Test Description	Observed Value	Units	Bio. Reference	Method	Trend / Status
Whole blood	Monocytes	1.0	%	2–10	laser flow cytometry (Automated)/Manual	⬇ Low
Whole blood	Eosinophils	0.0	%	1–6	laser flow cytometry (Automated)/Manual	⬇ Low
Whole blood	Basophils	0.0	%	<1	Laser flow cytometry (Automated)/Manual	→ Normal
Whole blood	Absolute Neutrophils Count	18.43	$\times 10^9$/L	2–7	Calculated method	⬆ High
Whole blood	Absolute Lymphocyte Count	0.98	$\times 10^9$/L	1–3	Calculated method	⬇ Slightly Low

Whole blood	Absolute Monocyte count	0.20	×10⁹/L	0.08–0.8	Calculated method	→ Normal
Whole blood	Absolute Eosinophil Count	0.00	×10⁹/L	0.02–0.5	Calculated method	⬇ Low
Whole blood	Absolute Basophil Count	0.00	×10⁹/L	0.02–0.1	Calculated method	⬇ Low
Whole blood	PLATELET COUNT	361	×10³/cumm	150–410	Sheath fluid impedance	→ Normal

The absolute Neutrophil Count (18.43) is clearly elevated, whereas the Absolute Lymphocyte Count (0.98) is low. This strictly high Neutrophil-to-Lymphocyte Ratio (NLR) as a significant market for diagnostic ambiguity that strongly indicates acute pyogenic stress (MRSA). This stress also clinically overshadows the lymphocyte-driven response on typical of TB.

Table 8: Renal Function, Electrolytes, and Liver Function Test with Trend Analysis

Sample	**Test Description**	**Observed Value**	**Units**	**Bio.Reference**	**Method**	**Trend / Status**
Serum	Urea	83	mg/dL	12.84–42.8	GLDH-Urease	⬆ High
Serum	CREATININE	2.36	mg/dL	Male: 0.9–1.3 / Female: 0.6–1.1	Sarcosine Oxidase	⬆ High
Serum	Uric Acid	3.52	mg/dL	Male: 4.4–7.6 / Female: 2.3–6.6	Uricase–PAP	⬇ Low (Male) / → Normal (Female)
Serum	Electrolytes	—	—	—	—	—
Serum	Sodium	132.9	mEq/L	136–145	ISE	⬇ Low
Serum	Potassium	4.79	mEq/L	3.5–5.1	ISE	→ Normal
Serum	Chloride	95.8	mEq/L	96–106	ISE	⬇ Slightly Low
Serum	Liver Function Test	—	—	—	—	—
Serum	Bilirubin (Total)	0.88	mg/dL	Adult: 0.3–1.1	Vanadate oxidizing method	→ Normal
Serum	Bilirubin (Direct)	0.66	mg/dL	0.0–0.2	Vanadate oxidizing method	⬆ High

Serum	Bilirubin (Indirect)	0.22	mg/dL	0.2–0.9	Calculated	→ Normal
Serum	Aspartate aminotransferase (SGOT)	21.1	IU/L	Male: <35 / Female: <31	UV Kinetic – IFCC	→ Normal
Serum	Alanine aminotransferase (SGPT)	7.3	IU/L	Male: <45 / Female: <34	UV Kinetic – IFCC	→ Normal

The severely elevated Urea (83) and creatinine (2.36), which is indicate significant renal impairment, are variables in TB/MRSA research. This is also likely acute kidney injury (AKI) from MRSA providing sepsis or anti-TB drug toxicity, creating diagnostic ambiguity. Additionally, hyponatremia (132.9) and elevated direct bilirubin (0.66) suggest systemic involvement in the dissemination of TB. Also, TB treatment clinicians assume findings are significant markers of delta-specific TB treatment and integrated management targets on renewal protection and metabolic stabilization to develop a prognosis in dual infection cases.

Table 9: Liver Function and Protein Profile with Trend Analysis

Sample	**Test Description**	**Observed Value**	**Units**	**Bio.Reference**	**Method**	**Trend / Status**
Serum	Alkaline phosphatase	292.0	IU/L	M(17–60y): 53–128 / F(17–60y): 42–98	PNP AMP Kinetic	⬆️ High
Serum	Gamma-glutamyl transferase	77.4	IU/L	Male: <55 / Female: <38	Szasz method / IFCC standard	⬆️ High
Serum	Total Protein	6.72	g/dL	6.0–7.8 gm/dL	Biuret	→ Normal
Serum	Albumin	2.5	g/dL	3.35–5.2	Bromocresol Green	⬇️ Low
Serum	Globulin	4.22	g/dL	2.5–3.5	Calculated	⬆️ High
Serum	A/G Ratio	0.59	—	1.2:1 – 2:1	Calculated	⬇️ Low

The elevated Alkaline Phosphatase (292.0) and low A/G Ratio (0.59) are the most significant findings. High ALP is a marker for bone involvement in spinal TB, yet it rises in MRSA-related hepatic stress, a diagnostic overlap. The low Albumin (2.5) and high Globulin (4.22) provide a chronic inflammatory state and malnutrition (TB-related wasting) vs the acute phase response of MRSA. Thus, clinicians misinterpret these markers as general infection rather its localized extrapulmonary TB.

Table 10: Complete Blood Count and Differential Leukocyte Count with Trend Analysis

Sample	**Test Description**	**Observed Value**	**Units**	**Bio. Reference**	**Method**	**Trend / Status**
Whole blood	Hb	8.9	g/dL	12–15	Photometric	⬇️ Low
Whole bl	Total WBC Count	25.17	×10⁹/L	4.0–10.0	Laser Flowmetry Cytometry	⬆️ High

ood						
Whole Blood	Differential Count	—				
Whole Blood	Neutrophils	88.7	%	20–80	Laser Flow Cytometry (Automated)/Manual	⬆️ High
Whole blood	Lymphocytes	6.8	%	22–44	Laser Flow Cytometry (Automated)/Manual	⬇️ Low
Whole blood	Monocytes	3.3	%	2–10	Laser flow cytometry (Automated)/Manual	→ Normal
Whole blood	Eosinophils	1.1	%	1–6	Laser flow cytometry (Automated)/Manual	→ Normal
Whole blood	Basophils	0.1	%	<1	Laser flow cytometry (Automated)/Manual	→ Normal
Whole blood	Absolute Neutrophils Count	22.32	×10⁹/L	2–7	Calculated method	⬆️ High
Whole blood	Absolute Lymphocyte Count	1.71	×10⁹/L	1–3	Calculated method	→ Normal
Whole blood	Absolute Monocyte count	0.83	×10⁹/L	0.08–0.8	Calculated method	⬆️ Slightly High
Whole blood	Absolute Eosinophil Count	0.28	×10⁹/L	0.02–0.5	Calculated method	→ Normal
Wh	Absolute Basophil Count	0.03	×10⁹/L	0.02–0.1	Calculated method	→ Normal

The extreme Leukocytosis (WBC 25.17) and neutrophilia (88.7%) provide a severe acute phase response, which is highly characteristic of a pyogenic MRSA infection. The low Hb (8.9 g/dl) suggests underlying chronic diseases which is a dominant high Absolute Neutrophil Count (22.32) may lead clinicians to treat for acute sepsis alone. Thus, the results overlap in misdiagnosis of TB intervention, worsening patient prognosis and disease progression.

Table 11: Complete Blood Count (CBC) and Differential Count with Trend Analysis

Sample	**Test Description**	**Observed Value**	**Units**	**Bio. Reference**	**Method**	**Trend / Status**
Whole	Hemoglobin	8.5	g/dL	12.0 – 15.0	Colorimetric meth	⬇️ Low

blood					od (automated)	
Whole blood	RBC Count	3.47	Millions/cumm	3.8–4.8	Sheath Fluid Impedance	⬇️ Low
Whole blood	MCV	72.8	fL	83–101	Calculated from RBC Histogram	⬇️ Low
Whole blood	MCH	24.5	pg	27–32	Calculated	⬇️ Low
Whole blood	MCHC	33.6	%	31.5–34.5	Calculated	→ Normal
Whole blood	RDW (CV%)	16.8	%	11–16	Calculated	⬆️ High
Whole blood	RDW (SD)	45.7	fL	35.0–56.0 fL	Calculated	→ Normal
Whole blood	Total WBC Count	22.35	×10⁹/L	4.0–10.0	Laser Flowmetry Cytometry	⬆️ High
Whole Bl	Differential	—	—	—	—	—
ood	Count					
Whole Blood	Neutrophils	87.6	%	20–80	Laser Flow Cytometry (Automated)	⬆️ High
Whole blood	Lymphocytes	7.3	%	22–44	Laser flow cytometry (Automated)	⬇️ Low

MCV values (MCV 72.8, Hb 8.5) suggest nutritional depletion, common in advanced TB. However, the extreme Leukocytosis (22.35) and Neutrophilia (87.6%) shift the clinical targets on an acute MRSA infection. The extreme inflammatory response of MRSA clinically drowns out the slower, lymphocyte-mediated markers of TB. coordinated diagnostic strategies simultaneously screen for both to prevent therapy deferments, which strengthens the overall patient prognosis.

Table 12: Differential Leukocyte Count and Platelet Profile with Trend Analysis

Sample	**Test Description**	**Observed Value**	**Units**	**Bio. Reference**	**Method**	**Trend / Status**
Whole blood	Monocytes	3.6	%	2–10	laser flow cytometry (Automated)/Manual	→ Normal
Whole bl	Eosinophils	1.4	%	1–6	laser flow cytometry	→ Nor

ood					(Automated)/Manual	mal
Whole blood	Basophils	0.1	%	<1	Laser flow cytometry (Automated)/Manual	→ Normal
Whole blood	Absolute Neutrophils Count	19.59	×10⁹/L	2–7	Calculated method	⬆ High
Whole blood	Absolute Lymphocyte Count	1.63	×10⁹/L	1–3	Calculated method	→ Normal
Whole blood	Absolute Monocyte count	0.80	×10⁹/L	0.08–0.8	Calculated method	→ High - Normal
Whole blood	Absolute Eosinophil Count	0.31	×10⁹/L	0.02–0.5	Calculated method	→ Normal
Whole blood	Absolute Basophil Count	0.02	×10⁹/L	0.02–0.1	Calculated method	→ Normal
Whole bl	PLATELET CO	356	×10³/cumm	150–410	Sheath fluid impedance	→ Normal
ood	UNT					

The Absolute Neutrophil Count (19.59) is adjacent to 3x the upper limit of normal. This table delivers that the subject is meeting intense acute reactive feedback, which is commonly seen in pyogenic bacteriological infections like MRSA. Furthermore, the ratio between Neutrophils and Lymphocytes (NLR) is high (~12:1). The Platelet Count (356) is normal, and it suggests that irritative neutropenia is the body's bone marrow, which yields platelets. Monocytes are often elevated in chronic TB. Low normal monocytes also end the percentage range as 3.6%, which shows how the acute neutrophil response is drowning out cellular markers.

Table 13: Prothrombin Time (PT-INR) Analysis with Trend Status

Sample	**Test Description**	**Observed Value**	**Units**	**Bio.Reference**	**Method**	**Trend / Status**
Citrated Plasma	PROTHROMBIN TIME (PT-INR)	—	—	—	—	—
Citrated Plasma	TEST	14.5	Seconds	—	Automated	⬆ Slightly Prolonged
Citrated Plasma	CONTROL	11.2	Secs	—	Automated	→ Reference
Citrated Plasma	INR	1.28	—	Normal: 0.9–1.1	Automated	⬆ High

This table highlights that the elevated INR (1.28) indicates a slight clotting delay, which is a common

sign of liver stress in TB-related wasting. This biochemical shift diagnostic ambiguity, and clinicians attribute irregularities to accurate MRSA induced sepsis or DIC, which potentially overlooks the chronic hepatic impact of TB. Moreover, the 1.28 INR exceeds the 0.9 to 1.1 range, which underscores slower clot development. The patient presented with paraplegia (power - 0/5) and back pain, indicating possible spinal cord involvement. Pleural fluid analysis showed elevated ADA levels suggestive of tuberculosis, although GeneXpert was negative. MRSA detection pointed towards a bacterial infection. These concurrent findings created diagnostic confusion, as most investigations supported a bacterial cause. This overlap delayed definitive identification and required careful clinical correlation.

Table 14: Glycated Hemoglobin (HbA1c) Analysis with Trend Status

Sample	Test Description	Observed Value	Units	Bio.Reference	Method	Trend / Status
Whole blood	HbA1c	7.27	%	Normal: <5.7% / Prediabetes: 5.7%–6.4% / Diabetes: ≥6.5%	HPLC	⬆ High (Diabetic Range)

The trend/status column indicates deviation from reference ranges in the absence of serial measurements. The HbA1c (7.27%) provides chronic hyperglycemia, and it is a crucial factor for diagnostic ambiguity. MRSA is also masks the systemic wasting of TB. High glucose levels act as a substrate for bacterial growth, fueling both infection types and tissue repair. The 7.27% value is well above the diabetic threshold of a minimum of 6.5% that reflecting poor glycemic control over the last 90 days.

Allover, the patient exhibited with severe back pain and entire lower limb immobility, raising suspicion of spinal cord pathology. Initial pleural fluid analysis showed elevated ADA levels suggestive of Tuberculosis, although GeneXpert was negative. Concurrent identification of Methicillin-resistant Staphylococcus aureus and other observations indicated a coexisting microbial infection, generating evaluative complexity. However, contrast-enhanced MRI Spine confirmed Tuberculous spondylodiscitis, explaining the neurological deficit and guiding definitive treatment. The development of paraplegia (0/5 power) indicated critical spinal cord involvement necessitating urgent evaluation.

Table 15: Pleural Fluid Culture & Sensitivity

Category	Parameter / Antibiotic	Result / Finding	Status / Trend
Specimen	Pleural Fluid	Sample collected	—
Gram Stain	Microscopy	Gram-positive cocci seen	Abnormal
Organism Isolated	MRSA	Methicillin-resistant Staphylococcus aureus	Pathogenic
Antibiotic	Clindamycin (CD)	Sensitive	Effective
Antibiotic	Co-Trimoxazole (STX)	Sensitive	Effective
Antibiotic	Linezolid (LZ)	Sensitive	Highly Effective
Antibiotic	Azithromycin (AZ)	Resistant	Not Effective
Antibiotic	Cefoxitin (CX)	Resistant	Not Effective
Antibiotic	Ciprofloxacin (CI)	Resistant	Not Effective
Antibiotic	Erythromycin (EM)	Resistant	Not Effective
Antibiotic	Gentamicin (GM)	Resistant	Not Effective

Antibiotic	Levofloxacin (LE)	Resistant	Not Effective
Antibiotic	Penicillin (P)	Resistant	Not Effective

The pleural fluid culture shows MRSA infection with sensitivity to linezolid, clindamycin, and co-trimoxazole, while resistance to multiple antibiotics indicates limited treatment options and need for targeted therapy.

Paraplegia indicated spinal cord involvement, while MRSA suggested bacterial infection. Elevated ADA raised suspicion for tuberculosis, and MRI confirmed spinal TB, explaining neurological deficits and back pain despite initially conflicting investigations.

Discussion

The commercial clinical data reveal two patients with hyperinflammatory states, such as TB/MRSA co-infection. The most striking finding is the extreme leukocytosis (WBC 25.17) and neutrophilia (88.7%), which point to an aggressive, acute bacterial attack from MRSA. Simultaneously, the HbA1c of 7.27% confirms diabetes. High blood sugar acts as fuel for bacteria, especially MRSA, while paralyzing the white blood cells that fight against it. This metabolic breakdown is characterized by low albumin (2.5) and anemia (Hb 8.5), which refers to a body in deep wasting mode and is a classic hallmark of long-term TB. Furthermore, the elevated urea (83) and creatinine (2.36) signal acute kidney stress. This organ damage is a direct hit from infection of heavy medications. Existing facts which refer to patients with poorly controlled diabetes opened the door for a dual threat infection, where an acute MRSA crisis makes chronic TB, severely complicating the path to recovery[6]. Therefore, MRSA co-infection causes significant diagnostic ambiguity by triggering an acute inflammatory surge that clinically masks the chronic, mediated markers of TB.

Conclusion

This research concludes that significant diagnostic ambiguity is created when MRSA co infection present alongside extrapulmonary and spinal TB. Across patient cases highlight leukocytosis (WBC 22.35 - 25.17) and neutrophilia (87-88%) act as a smokescreen, clinically drawing out the chronic, lymphocyte-mediated markers typically for identifying tuberculosis. Overall, this research overlaps with hematological and metabolic profiles leads to frequent misdiagnosis and treatment delays.

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