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Correlation Between Hba1c And IL-6 In Pulmonary Tuberculosis Patients A Cross-Sectional Observation Study

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Abstract

Background: Pulmonary tuberculosis (TB) is associated with persistent systemic inflammation and metabolic disturbances. Interleukin-6 (IL-6), a key pro-inflammatory cytokine, plays an important role in the host immune response against Mycobacterium tuberculosis, while glycated hemoglobin (HbA1c) reflects long-term glycemic status. The interaction between inflammatory activity and glycemic control during anti-tubercular treatment remains incompletely understood.

Objectives: To evaluate changes in serum IL-6 and HbA1c levels between the intensive and continuation phases of anti-tubercular therapy in pulmonary tuberculosis patients and to determine the correlation between these biomarkers.

Materials and Methods: This cross-sectional observational study was conducted in the Department of Biochemistry, RNT Medical College, Udaipur, Rajasthan. A total of 120 adult patients with confirmed pulmonary tuberculosis diagnosed by CBNAAT/GeneXpert were enrolled. Patients with diabetes mellitus, chronic kidney disease, chronic liver disease, pregnancy, immunosuppressive therapy, and other inflammatory conditions were excluded. HbA1c was measured using EDTA blood samples, while serum IL-6 was estimated from separated serum samples. Statistical analysis was performed using SPSS software. Continuous variables were expressed as mean ± standard deviation, and Pearson's correlation coefficient was used to assess the association between IL-6 and HbA1c. A p-value ≤ 0.05 was considered statistically significant.

Results: Serum IL-6 levels were significantly higher during the intensive phase than during the continuation phase (26.13 ± 3.51 pg/mL vs. 12.94 ± 2.15 pg/mL; $p<0.0001$). Similarly, HbA1c levels showed a significant reduction from 7.71 ± 0.74% in the intensive phase to 5.73 ± 0.46% in the continuation phase ($p<0.0001$). Pearson's correlation analysis demonstrated a weak but statistically significant positive correlation between intensive-phase IL-6 and continuation-phase HbA1c levels ($r=0.204$, $p=0.0252$), indicating that greater inflammatory activity was associated with relatively higher glycemic levels.

Conclusion: Effective anti-tubercular therapy is associated with significant reductions in both systemic inflammation and glycemic dysregulation. The positive correlation between IL-6 and HbA1c suggests that inflammatory processes contribute to altered glucose metabolism in pulmonary tuberculosis. Simultaneous assessment of IL-6 and HbA1c may serve as a useful adjunct for monitoring disease activity, treatment response, and metabolic recovery in TB patients. Further multicentric longitudinal studies are warranted to validate these findings.

Keywords: Pulmonary tuberculosis, Interleukin-6, IL-6, HbA1c, Glycemic control, Inflammation, Anti-tubercular therapy, Biomarkers

Introduction

Tuberculosis (TB) remains one of the most significant infectious diseases globally, caused by Mycobacterium tuberculosis (M. tuberculosis). Despite the availability of effective chemotherapy, TB continues to be a leading cause of morbidity and mortality, particularly in low- and middle-income countries. According to global health estimates, millions of new cases occur annually, with a substantial proportion resulting in death, especially among immunocompromised individuals such as those with HIV infection.[1]

India bears the highest burden of TB worldwide, contributing significantly to global incidence. Factors such as overcrowding, malnutrition, poverty, and coexisting infections contribute to the persistence of TB. The disease primarily affects the lungs (pulmonary TB), although extrapulmonary manifestations involving lymph nodes, meninges, bones, and other organs are also common.[2]

TB is transmitted via airborne droplets. Once inhaled, M. tuberculosis bacilli reach the alveoli, where they are phagocytosed by alveolar macrophages. However, the bacilli possess mechanisms to evade intracellular killing, allowing them to survive and replicate within macrophages.[3]

The host immune response plays a crucial role in determining disease outcome. Infected macrophages present antigens to T lymphocytes, initiating a cell-mediated immune response. This leads to the formation of granulomas—organized aggregates of immune cells that attempt to contain the infection.[4]

Granuloma formation is the hallmark of TB and involves a complex interplay of immune cells, including macrophages, T lymphocytes, dendritic cells, and cytokines. The balance between protective immunity and pathological inflammation determines whether infection remains latent or progresses to active disease.[5]

Cytokines are small signaling proteins that regulate immune and inflammatory responses. In TB, cytokines play a central role in host defense as well as disease progression.

Key cytokines involved in TB include:

1. Interferon-gamma (IFN-γ)
2. Tumor necrosis factor-alpha (TNF-α)
3. Interleukin-1 (IL-1)
4. Interleukin-12 (IL-12)
5. Interleukin-6 (IL-6)

IFN-γ and TNF-α are critical for macrophage activation and granuloma maintenance. However, dysregulation of cytokine production can lead to tissue damage and disease progression.[6]

Among these, IL-6 has emerged as a cytokine of particular interest due to its dual role in immune protection and inflammation.

Interleukin-6 (IL-6) is a pleiotropic cytokine produced by various cell types, including macrophages, monocytes, fibroblasts, endothelial cells, and T lymphocytes. It is encoded by the IL6 gene located on chromosome 7 and functions through binding to the IL-6 receptor (IL-6R) and gp130 signaling complex.[7]

IL-6 is involved in multiple physiological and pathological processes, including:

1. Regulation of immune responses
2. Induction of acute-phase proteins (e.g., CRP, fibrinogen)
3. B-cell differentiation into plasma cells
4. T-cell activation and differentiation
5. Hematopoiesis
6. Inflammation and infection responses

IL-6 signaling occurs via two pathways:

1. Classical signaling (via membrane-bound IL-6 receptor)
2. Trans-signaling (via soluble IL-6 receptor)

These pathways contribute to its diverse biological effects.[8]

Immunological Role of IL-6 in Tuberculosis :

IL-6 in Innate Immunity :

During early TB infection, IL-6 is rapidly produced by macrophages and dendritic cells upon recognition of M. tuberculosis. It acts as an important mediator of the acute inflammatory response and contributes to the recruitment of immune cells to the site of infection.[9]

IL-6 stimulates the production of acute-phase reactants such as CRP and serum amyloid A, which enhance host defense mechanisms. It also influences macrophage activation and promotes antimicrobial activity.[10]

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Experimental studies have demonstrated that IL-6-deficient mice exhibit increased susceptibility to TB, indicating its protective role during early infection.[11]

IL-6 in Adaptive Immunity :

IL-6 plays a critical role in shaping adaptive immune responses. It influences CD4+ T-cell differentiation, particularly:

1. Promoting Th17 cell differentiation
2. Inhibiting regulatory T cells (Treg)
3. Modulating Th1/Th2 balance

Th1 responses, characterized by IFN-γ production, are essential for controlling TB infection. However, excessive IL-6 may suppress Th1 responses and impair bacterial clearance.[12]

Thus, IL-6 has a complex role in TB, acting as both a facilitator of immune defense and a potential contributor to immune dysregulation.

Dual Role of IL-6 in Tuberculosis :

1. IL-6 exhibits both protective and pathogenic effects in TB:
2. Protective Effects
3. Enhances early immune response
4. Promotes acute-phase reaction
5. Supports granuloma formation
6. Contributes to bacterial containment
7. Pathogenic Effects
8. Excessive inflammation leading to tissue damage
9. Suppression of protective Th1 immunity
10. Promotion of chronic infection
11. Association with cytokine storm in severe TB

This dual nature makes IL-6 a key regulator of disease progression.[13]

IL-6 as a Biomarker in Tuberculosis :

Diagnostic Role :

Several studies have shown elevated IL-6 levels in patients with active TB compared to healthy individuals and those with latent TB infection. Increased IL-6 levels have been reported in:

1. Serum
2. Plasma
3. Bronchoalveolar lavage fluid
4. Cerebrospinal fluid (in TB meningitis)

These findings suggest that IL-6 may serve as a useful biomarker for diagnosing active TB.[14]

Prognostic Significance :

1. IL-6 levels correlate with:
2. Disease severity
3. Extent of lung involvement
4. Bacterial load
5. Systemic inflammation

Higher IL-6 levels are associated with severe and disseminated TB. During treatment, a decline in IL-6 levels is often observed, indicating therapeutic response.[15]

IL-6 in Extrapulmonary Tuberculosis :

In conditions such as tuberculous meningitis, IL-6 levels in cerebrospinal fluid are significantly elevated and correlate with disease severity and prognosis. This highlights its importance as a biomarker in extrapulmonary TB.[16]

Genetic Polymorphisms of IL-6 and TB Susceptibility :

Genetic variations in the IL-6 gene may influence susceptibility to TB. Polymorphisms in the promoter region of the IL6 gene can affect cytokine production and immune responses.

Studies have shown that certain IL-6 genotypes are associated with:

1. Increased risk of TB
2. Altered disease severity
3. Variations in immune response

These findings suggest a genetic basis for IL-6-mediated immune regulation in TB.[17]

Correlation of IL-6 with Clinical and Laboratory Parameters :

IL-6 levels have been shown to correlate with several clinical and laboratory markers in TB patients:

1. Erythrocyte sedimentation rate (ESR)
2. C-reactive protein (CRP)
3. White blood cell count
4. Radiological severity
5. Sputum smear positivity

Such correlations indicate that IL-6 reflects systemic inflammation and disease activity.[18]

Therapeutic Implications of IL-6 in TB :

Given its central role in TB pathogenesis, IL-6 has potential therapeutic implications. Targeting IL-6

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signaling pathways may help control excessive inflammation and improve outcomes.

However, inhibition of IL-6 must be approached cautiously, as it may impair protective immunity and worsen infection. Further research is needed to explore IL-6-targeted therapies in TB.[19]

In recent years, the dual burden of communicable and non-communicable diseases has become increasingly evident. Among non-communicable diseases, diabetes mellitus (DM) has emerged as a significant risk factor influencing the epidemiology, clinical presentation, and outcomes of TB. The coexistence of TB and diabetes is now recognized as a serious global health concern, particularly in low- and middle-income countries where both conditions are highly prevalent.[20]

Glycated hemoglobin (HbA1c) is a widely used biomarker for assessing long-term glycemic control. It reflects the average blood glucose levels over the previous 8–12 weeks and is considered a reliable indicator for the diagnosis and monitoring of diabetes mellitus.[21] Elevated HbA1c levels indicate poor glycemic control, which has been associated with impaired immune responses and increased susceptibility to infections, including tuberculosis.[22]

The relationship between HbA1c and TB is complex and bidirectional. On one hand, poorly controlled diabetes, as indicated by elevated HbA1c levels, predisposes individuals to active TB infection due to impaired cell-mediated immunity. On the other hand, TB infection itself can worsen glycemic control through stress-induced hyperglycemia and inflammatory responses.[23]

India carries one of the highest burdens of both TB and diabetes globally. The convergence of these two diseases has created a significant public health challenge. It is estimated that diabetes triples the risk of developing active TB, and patients with uncontrolled diabetes are more likely to have severe disease, delayed sputum conversion, and poor treatment outcomes.[24] Therefore, understanding the role of glycemic control, particularly HbA1c levels, in TB patients is crucial for improving disease management and outcomes.

From a pathophysiological perspective, chronic hyperglycemia leads to several immune dysfunctions, including reduced macrophage activity, impaired cytokine production, and decreased T-cell response. These alterations compromise the host's ability to contain Mycobacterium tuberculosis, thereby increasing the risk of infection and disease progression.[25] HbA1c serves as an indirect marker of these metabolic disturbances and can provide valuable insights into the susceptibility and severity of TB in diabetic patients.

Furthermore, TB infection itself can influence glucose metabolism. The inflammatory cytokines released during TB infection, such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), can induce insulin resistance and hyperglycemia. This creates a vicious cycle where poor glycemic control worsens TB outcomes, and TB infection further deteriorates glucose regulation.[26]

Clinically, patients with TB and elevated HbA1c levels often present with atypical symptoms, more extensive lung involvement, and higher bacterial loads. They are also at increased risk of complications such as treatment failure, relapse, and mortality.[27] Monitoring HbA1c levels in TB patients can therefore aid in risk stratification and guide therapeutic interventions.

In addition, the use of HbA1c as a screening tool for diabetes in TB patients has gained attention. Early identification of undiagnosed diabetes through HbA1c measurement can help in timely management, thereby improving TB treatment outcomes. Conversely, screening TB patients with high HbA1c levels for active TB can facilitate early diagnosis and reduce transmission.[28]

The World Health Organization (WHO) and the International Union Against Tuberculosis and Lung Disease have emphasized the importance of collaborative TB-diabetes care. Integrated management strategies, including routine screening of TB patients for diabetes and monitoring glycemic control using HbA1c, are recommended to address this dual epidemic.[29]

Despite growing evidence, there remains a need for further research to establish the exact relationship between HbA1c levels and TB disease progression, severity, and outcomes. Variations in HbA1c due to factors such as anemia, hemoglobinopathies, and nutritional status, which are common in TB patients, may also affect its reliability as a biomarker in this population.[30]

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Therefore, studying the correlation between HbA1c and TB is of significant clinical and public health importance. It can help in understanding disease mechanisms, improving diagnostic strategies, and optimizing treatment protocols. Such insights are particularly relevant in regions like India, where the burden of both TB and diabetes is high.

In conclusion, HbA1c serves as a crucial link between metabolic and infectious diseases. Its role in predicting susceptibility, severity, and outcomes of TB highlights the need for integrated disease management approaches. Addressing the interplay between glycemic control and TB can significantly contribute to better patient care and improved public health outcomes.

Materials And Methods

A study was conducted in RNT Medical College, Udaipur, Rajasthan, on patients diagnosed with pulmonary Tuberculosis. The source population was all cases of pulmonary tuberculosis at RNT Medical college with a confirmed diagnosis of Tuberculosis using Gene Xpert System (CBNAAT), as reported by department of Microbiology. In Inclusion Criteria Sample Adult patients (18 years and above) with confirmed pulmonary TB (diagnosed by sputum smear microscopy or culture or CB-NAAT).

Patients with known chronic illnesses (e.g. chronic kidney disease, chronic liver disease, Diabetes mellitus), Patients on immunosuppressive therapy, Pregnant women, Pregnant women, Pyrexia & other inflammatory condition which increase IL-6 other than TB was in exclusion criteria.

About 2 ml blood for HBAIC was drawn in EDTA vial (ethylene-diamin-tetraaciti-cacid) using sterile vacutainer. About 2 ml blood was drawn using perfectly dry and sterile vacutainer. The serum was separated with the help of centrifuge machine within 1 hours of collection to prevent artefactual changes in, IL-6. A total number of 120 on treatment patients at RNT Medical college and associated hospital Udaipur with pulmonary tuberculosis, was form the subjects of the present study. In this all patients were on treatment suffering from pulmonary tuberculosis. Efforts will be made to match all anthropometric factors comparable to both the groups of patients.

Clinical Methodology

Symptoms (fever, cough, dyspnea, headache, nasal congestion, rhinorrhea), serum IL-6 and HbA1c parameters levels) were recorded by using Autoanalyzer.

Statistical Analysis

For the quantitative analysis, we used the software SPSS software. In this analysis, all p values reported were two-tailed with the statistical significance set at $\leqslant 0.05$.

Result

The present study between intensive phase treatment and continuation phase treatment showed that level of IL-6 and HbA1c was low in continuation phase treatment patients compare to intensive phase treatment patients (Table.1, Fig.1). And also show insignificant correlation of IL-6 Intensive Phase vs HbA1c Continuation Phase (Table.2, Fig.2).

Table No.1: comparison of IL-6 and HbA1c between intensive phase treatment and continuation phase treatment patient

Parameter	Intensive Phase Mean ± SD	Continuation Phase Mean ± SD	p-value	Significance
IL-6	26.13 ± 3.51	12.94 ± 2.15	<0.0001	Significant
HbA1c	7.71 ± 0.74	5.73 ± 0.46	<0.0001	Significant

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Figure No.1: comparison of IL-6 and HbA1c between intensive phase treatment and continuation phase treatment patient

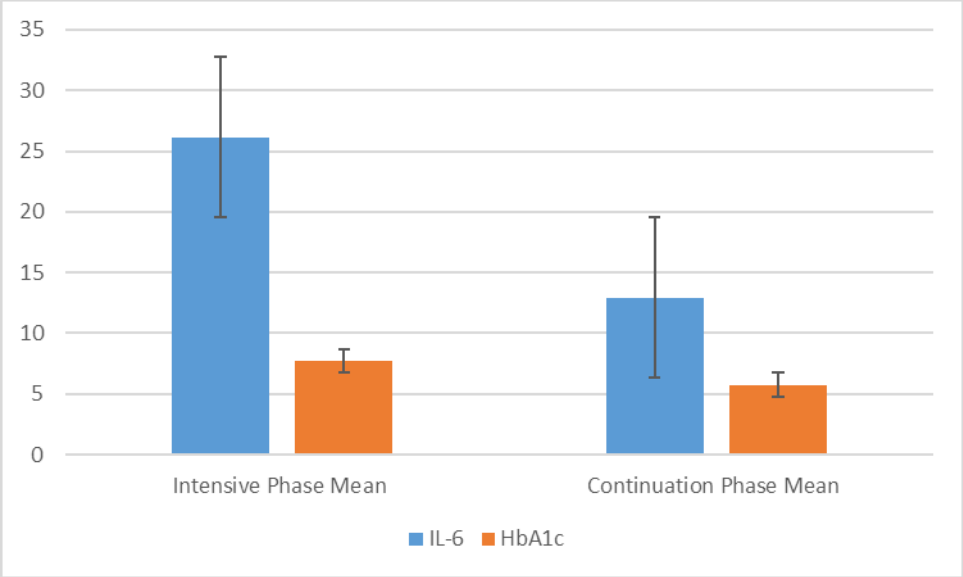
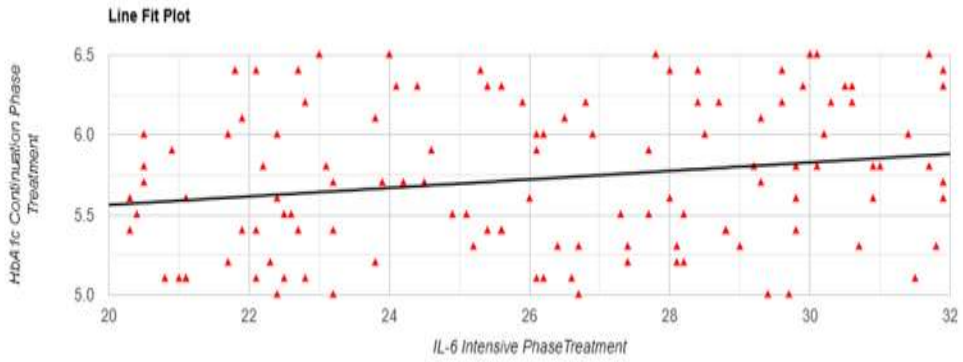


Table no 2: correlation of IL-6 Intensive Phase vs HbA1c Continuation Phase

Variables Compared	Pearson Correlation (r)	P value	Interpretation
IL-6 Intensive Phase vs HbA1c Continuation Phase	0.204	0.0252	Positive Correlation

Figure no 2: correlation of IL-6 Intensive Phase vs HbA1c Continuation Phase



Discussion

The present study demonstrated a significant reduction in both serum IL-6 and HbA1c levels between the intensive and continuation phases of anti-tubercular treatment. The mean serum IL-6 concentration decreased from 26.13 ± 3.51 pg/mL during the intensive phase to 12.94 ± 2.15 pg/mL during the continuation phase ($p < 0.0001$). Likewise, HbA1c levels declined significantly from 7.71 ± 0.74% to 5.73 ± 0.46% ($p < 0.0001$). These findings indicate that effective anti-tubercular therapy not only reduces

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systemic inflammation but also improves glycemic status.

Interleukin-6 is an important pro-inflammatory cytokine released by activated macrophages and monocytes during Mycobacterium tuberculosis infection. High IL-6 levels during the intensive phase reflect an increased bacterial load and an exaggerated inflammatory response. As treatment progresses and bacillary burden decreases, inflammatory cytokine production declines, resulting in significantly lower IL-6 concentrations during the continuation phase. Similarly, chronic inflammation is known to induce insulin resistance through cytokine-mediated impairment of insulin signaling pathways, thereby increasing blood glucose levels and HbA1c. Reduction in inflammatory activity with successful treatment therefore contributes to improved glucose metabolism.

The present findings are consistent with Kumar et al. who reported significantly elevated IL-6 levels in patients with active pulmonary tuberculosis, with a marked decline following successful anti-tubercular therapy.[31] Restrepo et al. also demonstrated that tuberculosis-associated inflammation contributes to transient hyperglycemia, which improves after completion of treatment.[32] Likewise, Singh et al. observed significant reductions in both inflammatory markers and HbA1c following effective ATT, suggesting that systemic inflammation plays a crucial role in altered glucose homeostasis in tuberculosis patients.[33]

Thus, the findings of the present study support the hypothesis that monitoring IL-6 along with HbA1c may provide valuable information regarding treatment response and metabolic recovery in patients with pulmonary tuberculosis.

The present study evaluated the correlation between serum IL-6 levels during the intensive phase and HbA1c levels during the continuation phase. Pearson correlation analysis demonstrated a weak but statistically significant positive correlation ($r = 0.204$, $p = 0.0252$).

The positive correlation indicates that patients exhibiting higher inflammatory activity during the intensive phase tended to have relatively higher HbA1c values during the continuation phase. Although the correlation coefficient was weak, the statistically significant p-value suggests that inflammation contributes to disturbances in glucose metabolism among tuberculosis patients.

IL-6 promotes hepatic gluconeogenesis, inhibits insulin receptor signaling, and increases peripheral insulin resistance, thereby contributing to hyperglycemia. However, the relatively weak correlation observed in the present study indicates that HbA1c is influenced by several additional factors including nutritional status, baseline diabetic status, duration of tuberculosis, severity of infection, erythrocyte turnover, and individual metabolic variations.

These observations are in agreement with Magee et al., who reported that inflammatory cytokines including IL-6 are associated with impaired glucose metabolism in tuberculosis patients, although the relationship is influenced by multiple host factors.[34] Dooley and Chaisson also suggested that persistent inflammation contributes to dysglycemia in tuberculosis through cytokine-mediated insulin resistance.[35] Furthermore, Critchley et al. concluded that inflammatory biomarkers and HbA1c show a positive association in tuberculosis patients, supporting the concept that inflammation plays an important role in metabolic dysfunction during active disease.[36]

Overall, the present findings suggest that serum IL-6 may serve as an important inflammatory biomarker reflecting disease activity, while HbA1c may indicate the metabolic consequences of chronic inflammation. Simultaneous assessment of both parameters may therefore improve monitoring of treatment response and identify patients at increased risk of persistent dysglycemia.

Summary:

The present study evaluated the serum levels of Interleukin-6 (IL-6) and HbA1c in pulmonary tuberculosis patients during the intensive and continuation phases of anti-tubercular treatment. The study also assessed the correlation between IL-6 and HbA1c to determine the relationship between systemic inflammation and glycemic status.

The results demonstrated a statistically significant reduction in both IL-6 and HbA1c levels following progression from the intensive phase to the continuation phase of treatment. The mean serum IL-6 level decreased from 26.13 ± 3.51 pg/mL during the intensive phase to 12.94 ± 2.15 pg/mL during the

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continuation phase ($p < 0.0001$). Similarly, the mean HbA1c level declined significantly from $7.71 \pm 0.74\%$ to $5.73 \pm 0.46\%$ ($p < 0.0001$). These findings suggest that successful anti-tubercular therapy is associated with a reduction in systemic inflammation and improvement in glycemic control.

Correlation analysis revealed a weak but statistically significant positive correlation between IL-6 during the intensive phase and HbA1c during the continuation phase ($r = 0.204$, $p = 0.0252$). This indicates that increased inflammatory activity is associated with higher HbA1c levels, although other factors also contribute to glucose metabolism in tuberculosis patients.

Overall, the study highlights the important role of inflammatory cytokines in the metabolic alterations associated with tuberculosis and suggests that simultaneous assessment of IL-6 and HbA1c may be useful in monitoring treatment response and metabolic recovery.

Conclusion

The present study concludes that pulmonary tuberculosis patients exhibit significantly elevated serum IL-6 and HbA1c levels during the intensive phase of anti-tubercular therapy, reflecting active systemic inflammation and impaired glycemic status. Both parameters decreased significantly during the continuation phase, indicating that effective anti-tubercular treatment reduces inflammatory burden and improves glucose metabolism.

Furthermore, a weak but statistically significant positive correlation between IL-6 and HbA1c suggests that inflammation contributes to dysglycemia in tuberculosis patients. Although IL-6 alone does not fully explain changes in HbA1c, it appears to be an important inflammatory biomarker associated with metabolic alterations during active disease.

Therefore, measurement of serum IL-6 along with HbA1c may serve as a useful adjunct in assessing disease activity, monitoring therapeutic response, and identifying tuberculosis patients at risk of persistent dysglycemia. Larger multicentric longitudinal studies are recommended to further establish the clinical utility of these biomarkers in routine tuberculosis management.

Eithical Issues:

Research project approved by the ethics committee of RNT Medical College Udaipur- 313005, Rajasthan, INDIA.

References:

1. 1.World Health Organization. Global tuberculosis report 2023. Geneva: WHO; 2023.
2. Central TB Division. India TB report 2023. New Delhi: MoHFW; 2023.
3. Philips JA, Ernst JD. Tuberculosis pathogenesis and immunity. Annu Rev Pathol. 2012; 7:353–84.
4. Flynn JL, Chan J. Immunology of tuberculosis. Annu Rev Immunol. 2001; 19:93–129.
5. O'Garra A, et al. The immune response in tuberculosis. Annu Rev Immunol. 2013; 31:475–527.
6. Dorhoi A, Kaufmann SH. Cytokines in tuberculosis. Nat Rev Immunol. 2014; 14:451–65.
7. Kishimoto T. IL-6: from laboratory to clinic. Clin Rev Allergy Immunol. 2005; 28:177–86.
8. Tanaka T, Narazaki M, Kishimoto T. IL-6 signaling pathways. Cold Spring Harb Perspect Biol. 2014;6: a016295.
9. Singh PP, Goyal A. IL-6 in TB infection. Springerplus. 2013; 2:686.
10. Hunter CA, Jones SA. IL-6 in immunity and inflammation. Nat Immunol. 2015; 16:448–57.
11. Ladel CH, et al. IL-6 deficient mice and TB susceptibility. Infect Immun. 1997; 65:4843–9.
12. Linge I, et al. IL-6 in adaptive immunity in TB. Front Immunol. 2022; 13:750068.
13. Boni FG, et al. IL-6 and cytokine storm in TB. Infect Genet Evol. 2022; 97:105166.
14. Kumar NP, et al. IL-6 as biomarker in TB. PLoS One. 2013;8: e63879.
15. Djoba Siawaya JF, et al. Cytokine levels in TB patients. J Infect. 2007; 54:36–43.
16. Thuong NT, et al. IL-6 in TB meningitis. Clin Infect Dis. 2007; 45:875–81.
17. Wu S, et al. Cytokine gene polymorphisms and TB. Sci Rep. 2019; 9:2507.
18. Verbon A, et al. Serum cytokines in TB. Clin Exp Immunol. 1999; 115:110–3.
19. Wallis RS, et al. Host-directed therapies in TB. Clin Chest Med. 2019; 40:807–19.
20. Jeon CY, Murray MB. Diabetes mellitus increases the risk of active tuberculosis: a systematic review. PLoS Med. 2008;5(7): e152.

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21. American Diabetes Association. Standards of medical care in diabetes—2024. Diabetes Care. 2024;47(Suppl 1): S1–S350.
22. Restrepo BI. Diabetes and tuberculosis. Microbiol Spectr. 2016;4(6):10.1128.
23. Baker MA, Harries AD, Jeon CY, et al. The impact of diabetes on tuberculosis treatment outcomes. BMC Med. 2011; 9:81.
24. Stevenson CR, Critchley JA, Forouhi NG, et al. Diabetes and risk of tuberculosis: a systematic review. Int J Tuberc Lung Dis. 2007;11(7):693–700.
25. Martinez N, Kornfeld H. Diabetes and immunity to tuberculosis. Eur J Immunol. 2014;44(3):617–626.
26. Dooley KE, Chaisson RE. Tuberculosis and diabetes mellitus: convergence of two epidemics. Lancet Infect Dis. 2009;9(12):737–746.
27. Alisjahbana B, Sahiratmadja E, Nelwan EJ, et al. The effect of type 2 diabetes mellitus on TB treatment outcomes. Clin Infect Dis. 2007;45(4):428–435.
28. Harries AD, Kumar AMV, Satyanarayana S, et al. Diabetes mellitus and tuberculosis: programmatic management. Int J Tuberc Lung Dis. 2015;19(8):879–886.
29. WHO & The Union. Collaborative framework for care and control of TB and diabetes. Geneva: WHO; 2011.
30. .Sacks DB. Hemoglobin variants and HbA1c interpretation. Clin Chem. 2012;58(2):351–353.
31. Kumar NP, Banurekha VV, Nair D, Babu S. Circulating cytokines are associated with pulmonary tuberculosis disease severity and treatment response. Cytokine. 2016;86:1-8.
32. Restrepo BI. Diabetes and tuberculosis. Microbiol Spectr. 2016;4(6):TNMI7-0023-2016.
33. Singh SK, Kashyap GC, Puri A, et al. Changes in inflammatory biomarkers and glycemic status during anti-tuberculosis treatment: a prospective study. Indian J Tuberc. 2021;68(3):328-334.
34. Magee MJ, Salindri AD, Kornfeld H, et al. Diabetes mellitus and tuberculosis: current status of the dual epidemic. Lancet Diabetes Endocrinol. 2018;6(9):710-719.
35. Dooley KE, Chaisson RE. Tuberculosis and diabetes mellitus: convergence of two epidemics. Lancet Infect Dis. 2009;9(12):737-746.
36. Critchley JA, Restrepo BI, Ronacher K, et al. Defining a research agenda to address the converging epidemics of tuberculosis and diabetes. Part Fibre Toxicol. 2017;14(Suppl 1):50

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