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Serum Procalcitonin in Diabetes Mellitus with and Without Pulmonary Tuberculosis

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Abstract. *Diabetes mellitus (DM) is associated with immune dysfunction and increased susceptibility to pulmonary tuberculosis (TB). Procalcitonin (PCT) is widely used as a biomarker of bacterial infection, but its concentrations in patients with DM and pulmonary TB remain insufficiently characterized. To compare serum PCT concentrations between patients with DM with and without active pulmonary TB. This analytical cross-sectional study included 50 patients with DM recruited from two primary healthcare centers in Jambi, Indonesia, from March to June 2025. Participants comprised 34 patients without pulmonary TB and 16 with active pulmonary TB. Serum PCT was measured using fluorescence immunoassay. Non-normally distributed PCT concentrations were summarized using the median and interquartile range (IQR) and compared using the Mann–Whitney U test. PCT concentrations were within the normal reference range (<0.05 ng/mL) in 33/34 (97.1%) patients without pulmonary TB and 13/16 (81.3%) patients with active pulmonary TB. Because measurements at or below 0.05 ng/mL were retained as thresholded values, the median (IQR) was ≤0.05 (≤0.05–≤0.05) ng/mL in both groups. No statistically significant between-group difference was identified (U = 230.000, Z = −1.857, p = 0.063). Serum PCT concentrations did not differ significantly between patients with DM with and without active pulmonary TB. Larger studies with standardized TB confirmation, assessment of treatment status and relevant confounders, and diagnostic-accuracy analyses are warranted.*

Keywords: *Diabetes Mellitus; Pulmonary Tuberculosis; Procalcitonin; Biomarker*

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INTRODUCTION

Diabetes mellitus (DM) is one of the fastest-growing chronic metabolic disorders worldwide and represents a major public health challenge because of its increasing prevalence, long-term complications, and substantial healthcare burden (American Diabetes Association Professional Practice Committee, 2023b, 2023a). According to the International Diabetes Federation, approximately 537 million adults were living with diabetes in 2021, and this number is projected to increase to 643 million by 2030 and 783 million by 2045 (Magliano et al., 2021). Beyond chronic hyperglycemia, DM is characterized by persistent metabolic dysregulation that contributes to vascular complications, impaired immune responses, and increased susceptibility to infectious diseases, thereby substantially increasing morbidity and mortality worldwide (Holt et al., 2024; Yameny, 2024; Kim & Choi, 2025; Khanam et al., 2023).

Among infectious diseases associated with diabetes, pulmonary tuberculosis (TB) remains one of the most important comorbid conditions. The relationship between DM and TB is bidirectional: diabetes increases the risk of developing active tuberculosis, while tuberculosis may worsen glycemic control through systemic inflammation and insulin resistance (Al-Bari et

al., 2024; Franco et al., 2024). Epidemiological studies indicate that individuals with diabetes have approximately two to three times higher risk of developing active TB than those without diabetes (Holt et al., 2024; Peng, 2023; Ye et al., 2024). This association is particularly relevant in countries with a high burden of both diseases, including Indonesia, where diabetes and tuberculosis continue to pose significant public health challenges (Selvan et al., 2024; Mboi et al., 2022; Awad et al., 2022; Kapur & Harries, 2013; Khalil & Hossain, 2025; Susanti et al., 2026).

The increased susceptibility of patients with diabetes to tuberculosis is largely attributed to chronic hyperglycemia-induced immune dysfunction (Al-Bari et al., 2024; Chaubey et al., 2024). Persistent hyperglycemia impairs neutrophil chemotaxis, macrophage phagocytosis, antigen presentation, and T-cell-mediated immune responses, reducing the host's ability to eliminate *Mycobacterium tuberculosis* (Lee et al., 2024; Zhao et al., 2024). In addition, chronic low-grade inflammation and oxidative stress associated with diabetes further compromise innate and adaptive immune function, facilitating infection and disease progression. These pathophysiological mechanisms highlight the need for reliable biomarkers that may assist clinicians in identifying infectious complications among patients with diabetes (Chaubey et al., 2024).

Procalcitonin (PCT), the 116-amino acid precursor of calcitonin, has emerged as a promising biomarker for bacterial infection and systemic inflammation (Al-Laith, 2025). Under normal physiological conditions, circulating PCT concentrations are extremely low, whereas bacterial infections stimulate its production in multiple tissues through pro-inflammatory cytokines and bacterial endotoxins. Consequently, PCT has been widely used for diagnosing bacterial infections, assessing disease severity, monitoring treatment response, and guiding antibiotic therapy (Biswas et al., 2025). Nevertheless, PCT expression differs according to the underlying infectious process. Acute pyogenic bacterial infections generally produce marked elevations in circulating PCT, whereas chronic granulomatous infections such as pulmonary tuberculosis often induce relatively modest increases, potentially limiting its diagnostic value in this setting (Leo et al., 2024).

Previous studies investigating procalcitonin in patients with diabetes have primarily focused on acute bacterial infections, particularly diabetic foot infections. Omar et al. reported significantly higher PCT concentrations in patients with infected diabetic foot compared with non-infected diabetic foot, demonstrating excellent discriminatory performance for infection severity (Omar et al., 2023). Similarly, Ahmed El-Banna et al. found that elevated PCT levels were associated with more severe diabetic foot ulcers and complications such as osteomyelitis and peripheral arterial disease, although its diagnostic performance varied according to clinical outcomes (Ahmed El-Banna et al., 2024). Despite these findings, evidence regarding serum procalcitonin concentrations in patients with diabetes complicated by pulmonary tuberculosis remains limited and inconsistent, as tuberculosis elicits a distinct immunopathological response compared with acute bacterial infections.

Although diabetes and tuberculosis frequently coexist in high-burden countries, few studies have specifically compared serum procalcitonin levels between patients with uncomplicated diabetes mellitus and those with diabetes complicated by pulmonary tuberculosis. Moreover, evidence from the Indonesian population remains scarce. Addressing this knowledge gap may improve understanding of the clinical utility of procalcitonin in differentiating infectious complications among patients with diabetes and provide evidence for its potential role in routine clinical assessment. Therefore, this study aimed to compare serum procalcitonin levels between patients with uncomplicated diabetes mellitus and those with diabetes mellitus complicated by pulmonary tuberculosis.

METHODS

This analytical cross-sectional study was conducted from March to June 2025 at Putri Ayu Primary Healthcare Center and Simpang IV Sipin Primary Healthcare Center, Jambi, Indonesia. The study aimed to compare serum procalcitonin (PCT) concentrations between patients with

diabetes mellitus (DM) without pulmonary tuberculosis and those with DM complicated by active pulmonary tuberculosis (TB). The accessible population comprised 94 patients with DM across the two participating healthcare centers. The minimum sample size was determined using the Slovin formula, (n=N/[1+N(e)^2]), where *n* represents the minimum required sample size, *N* the accessible population, and *e* the tolerated margin of error. Using an accessible population of 94 patients and a 10% margin of error, the calculated minimum sample size was 48.45, which was rounded up to 50 participants. A total of 50 participants were ultimately recruited, comprising 25 participants from Putri Ayu Primary Healthcare Center and 25 from Simpang IV Sipin Primary Healthcare Center. Participants were recruited using convenience sampling during the study period. Potential participants attending the two participating healthcare centers were assessed for eligibility and invited to participate until the required sample size was achieved. Participants were eligible if they had DM, attended either participating healthcare center during the recruitment period, and provided written informed consent to participate. Diabetes mellitus was defined as a fasting blood glucose concentration ≥126 mg/dL. Eligible participants were subsequently classified into two study groups according to pulmonary TB status: patients with DM without pulmonary TB (n = 34) and patients with DM complicated by active pulmonary TB (n = 16). Active pulmonary TB status was identified based on a physician-confirmed diagnosis documented in the participants' medical records. The available study records did not systematically document the specific microbiological, radiological, or clinical criteria used by the treating physicians to establish the diagnosis of pulmonary TB.

Conditions that may independently influence circulating PCT concentrations were considered potential confounding factors. However, the original study records did not systematically document comprehensive exclusion of concomitant acute bacterial infections other than pulmonary TB, sepsis, renal impairment, recent major surgery or trauma, or systemic inflammatory diseases. Detailed information regarding anti-tuberculosis treatment status, treatment duration, TB disease severity, and the timing of blood collection relative to the initiation of anti-tuberculosis therapy was also not systematically recorded. These factors were therefore considered potential sources of residual confounding and were taken into account when interpreting the findings. Serum samples were obtained from all participants and analyzed for PCT using a fluorescence immunoassay (FIA) with the STANDARD F200 Analyzer (SD Biosensor, Republic of Korea) according to the standard operating procedure. Before analysis, the reagents, buffer, and serum samples were brought to room temperature. Participant identification was entered into the analyzer, and a PCT-specific test cassette was inserted into the instrument, with assay information automatically recognized through the 2D barcode. For each measurement, a 100-µL aliquot of serum was transferred into the extraction buffer and mixed thoroughly. The resulting reaction mixture was transferred to the PCT test cassette and immediately analyzed. The analyzer automatically processed the assay and provided a quantitative PCT result in ng/mL after 15 minutes. Instrument calibration was performed periodically and whenever a new test-kit lot was introduced using the CAL-1 calibrator in accordance with the standard operating procedure. Information regarding the initial venous blood collection volume, exact collection time, serum separation and storage conditions, analytical detection limit, and whether measurements were performed in duplicate was not available in the original study records. For descriptive interpretation, serum PCT concentrations <0.05 ng/mL were classified as within the normal range, whereas concentrations of 0.05–0.50 ng/mL were considered mildly to moderately elevated according to the reference ranges used in the study.

Categorical variables were summarized as frequencies and percentages. The distribution of continuous variables was assessed using the Shapiro–Wilk test. Normally distributed continuous variables were summarized as mean and standard deviation, whereas non-normally distributed variables were summarized as median and interquartile range (IQR). Because serum PCT concentrations were not normally distributed, differences in PCT concentrations between patients with DM without pulmonary TB and those with DM complicated by active pulmonary TB were evaluated using the Mann–Whitney U test. All statistical analyses were performed using IBM

SPSS Statistics version 24.0. All statistical tests were two-sided, and a *p*-value <0.05 was considered statistically significant. Ethical approval for the study was obtained from the Health Research Ethics Committee of Progres Ilmiah Kesehatan (Komisi Etik Penelitian Kesehatan Progres Ilmiah Kesehatan), approval No. 036/PROMISE/KEPK/II/2025. Written informed consent was obtained from all participants prior to study enrollment. Participant confidentiality and privacy were maintained throughout the study.

RESULT AND DISCUSSION

Characteristics of the study participants

A total of 50 patients with diabetes mellitus (DM) were included in the study, comprising 34 patients with DM without pulmonary tuberculosis (TB) and 16 patients with DM complicated by active pulmonary TB. Overall, 29 participants (58%) were male and 21 (42%) were female. The largest age group was 46–65 years (46%), followed by 26–45 years (44%) and >65 years (10%). Thirty-one participants (62%) had lived with DM for five years or longer, whereas 19 (38%) had a duration of DM of less than five years. All participants had fasting blood glucose concentrations ≥126 mg/dL. Detailed participant characteristics are presented in **Table 1.**

Table 1. Characteristics of The Respondents

Characteristics		Frequency	Percentage
Gender	Male	29	58%
	Female	21	42%
Age	26-45 y.o	22	44%
	46-65 y.o	23	46%
	>65 y.o	5	10%
Duration of DM	Newly Diagnosed (< 5 thn)	19	38%
	Long-Standing (≥ 5 thn)	31	62%
DM Complication Status	Uncomplicated	34	68%
	Pulmonary Tb	16	32%
Fasting Blood Glucose	Normal (<100 mg/dL)	-	-
	Prediabetes (100-125 mg/dL)	-	-
	DM (≥ 126 mg/dL)	50	100%

The predominance of male participants is consistent with previous epidemiological evidence indicating a substantial burden of both diabetes and tuberculosis among men (Buasroung et al., 2022). Behavioral and metabolic factors, including smoking, reduced physical activity, and visceral adiposity, have been associated with insulin resistance and may contribute to the burden of diabetes among males (Cho et al., 2022; Jiang et al., 2022; Marzoa Jansana et al., 2025; van der Velde et al., 2023). Men may also experience greater exposure to behavioral and environmental risk factors associated with pulmonary TB (Vega et al., 2024).

Most participants were aged 46–65 years and had lived with diabetes for at least five years. Increasing age and prolonged hyperglycemia are associated with chronic inflammation, endothelial dysfunction, and alterations in host immune responses (Lodi et al., 2026; Pacinella et al., 2022). Long-standing diabetes can impair neutrophil function, macrophage activity, and cell-mediated immunity, thereby increasing susceptibility to infectious diseases, including tuberculosis (Alexander et al., 2024; Thimmappa et al., 2023). These mechanisms provide a biologically plausible context for the coexistence of DM and pulmonary TB in the present study.

Serum procalcitonin concentrations

Most participants in both groups had serum PCT concentrations within the normal reference range. Among patients with DM without pulmonary TB, 33 of 34 participants (97.1%) had PCT concentrations <0.05 ng/mL, whereas one participant (2.9%) had a mildly to moderately elevated concentration. Among patients with DM complicated by active pulmonary TB, 13 of 16 participants (81.3%) had PCT concentrations within the normal range, whereas three

participants (18.8%) had mildly to moderately elevated concentrations. Overall, 46 of 50 participants (92.0%) had PCT concentrations within the normal reference range (Table 2).

Table 2. Distribution of Serum Procalcitonin Categories According to Pulmonary Tuberculosis Status

Serum PCT category	Complication Status		Total
	DM without pulmonary TB, n (%)	DM with active pulmonary TB, n (%)	
Normal (<0.05 ng/mL)	33 (97.1)	13 (81.3)	46 (92.0)
Mildly to moderately elevated (0.05–0.50 ng/mL)	1 (2.9)	3 (18.8)	4 (8.0)

Procalcitonin is synthesized in response to bacterial endotoxins and pro-inflammatory cytokines, particularly interleukin-6, interleukin-1β, and tumor necrosis factor-α. Consequently, elevated circulating PCT concentrations are commonly observed in acute bacterial infections and sepsis (Ali et al., 2025). In contrast, pulmonary tuberculosis is characterized by chronic granulomatous inflammation mediated predominantly by macrophages and T lymphocytes (Sossen et al., 2023). This immunological profile may explain why most participants with pulmonary tuberculosis in the present study still exhibited relatively low serum procalcitonin concentrations despite having an active infectious disease.

These findings are biologically plausible and consistent with previous reports suggesting that procalcitonin responds more strongly to acute pyogenic bacterial infections than to chronic mycobacterial infections (Xu et al., 2022). Therefore, pulmonary tuberculosis alone may not induce a sufficient systemic inflammatory response to produce marked elevations in circulating procalcitonin (Kong et al., 2026).

Comparison of serum procalcitonin levels

Serum PCT concentrations were non-normally distributed in both study groups (Shapiro–Wilk, $p < 0.001$) and were therefore summarized using the median and interquartile range (IQR). Because PCT measurements at or below 0.05 ng/mL were retained in the study dataset as ≤0.05 ng/mL rather than as exact numerical values, the median (IQR) was reported as ≤0.05 (≤0.05–≤0.05) ng/mL in both groups. The Mann–Whitney U test showed no statistically significant difference in the distribution of serum PCT concentrations between patients with DM without pulmonary TB and those with DM complicated by active pulmonary TB (U = 230.000, Z = −1.857, $p = 0.063$) (Table 3).

Table 3. Distribution of Procalcitonin Levels by Diabetes Status

Study Group	n	Median IQR, ng/mL	U	Z	*P*-Value
DM without pulmonary TB	34	0.05 (0.05–0.05)	230.000	−1.857	0.063
DM with active pulmonary TB	16	0.05 (0.05–0.05)			

Procalcitonin production is strongly stimulated by bacterial endotoxins and pro-inflammatory cytokines, particularly interleukin-6, interleukin-1β, and tumor necrosis factor-α, and marked elevations are commonly associated with acute systemic bacterial infections and sepsis (Ali et al., 2025; Xu et al., 2022). In contrast, pulmonary tuberculosis is characterized by a predominantly chronic, cell-mediated immune response and granuloma formation rather than the intense systemic inflammatory response typically observed in acute pyogenic bacterial infections (Weeratunga et al., 2024). This difference in immunopathology may partly explain why most participants with active pulmonary TB in the present study had PCT concentrations within the normal reference range.

Diabetes may further modify this inflammatory response. Chronic hyperglycemia is associated with impaired innate and adaptive immune function, including altered macrophage activity and neutrophil dysfunction, which may influence host responses to *Mycobacterium tuberculosis* (Chaubey et al., 2024; Lee et al., 2024; Ye et al., 2024). Nevertheless, the present study did not identify a statistically significant difference in serum PCT concentrations between the two study groups. This finding should be interpreted strictly as a between-group comparison and not as evidence regarding the diagnostic or discriminatory performance of PCT for pulmonary TB.

The present findings differ from studies evaluating PCT in acute diabetic infections. Todorova et al. (Todorova et al., 2023) reported higher serum PCT concentrations in patients with infected diabetic foot and demonstrated diagnostic utility for mild-to-moderate diabetic foot infection. Similarly, evidence from diabetic foot populations suggests that PCT may increase in association with acute infectious complications (Ardian, 2024; Omar et al., 2023). These findings are not directly comparable with the present study because diabetic foot infections are predominantly acute pyogenic processes, whereas pulmonary TB represents a chronic granulomatous infection. The differing inflammatory pathways may therefore result in different circulating PCT responses.

Although mildly to moderately elevated PCT concentrations occurred proportionally more often among participants with active pulmonary TB (18.8%) than among those without pulmonary TB (2.9%), this descriptive difference should not be interpreted as clinically meaningful. The present study did not establish a minimum clinically important difference for PCT, nor was it designed as a diagnostic-accuracy study. No receiver operating characteristic analysis, sensitivity, specificity, predictive values, or diagnostic cutoff optimization was performed. Consequently, the findings do not permit conclusions regarding the ability of serum PCT to discriminate pulmonary TB among patients with DM.

Several limitations should be considered when interpreting these findings. First, the study included a relatively small and unequal number of participants in the two comparison groups (34 patients with DM without pulmonary TB and 16 with active pulmonary TB), which may have limited statistical power to detect between-group differences. Second, convenience sampling and recruitment from only two primary healthcare centers may have introduced selection bias and limit the generalizability of the findings. Third, active pulmonary TB status was based on physician-confirmed diagnoses documented in medical records, while the specific microbiological, radiological, and clinical criteria underlying these diagnoses were not systematically available in the retained study records. Fourth, detailed information regarding TB disease severity, anti-tuberculosis treatment status, treatment duration, and the timing of blood collection relative to treatment initiation was unavailable. These factors may influence systemic inflammatory activity and circulating PCT concentrations.

In addition, glycemic control was assessed using fasting blood glucose, while longer-term glycemic status, particularly HbA1c, was not evaluated. Potential conditions capable of independently increasing PCT, including concomitant acute bacterial infection, sepsis, renal impairment, recent surgery or major trauma, and systemic inflammatory disease, were not comprehensively documented in the retained dataset. Residual confounding from these conditions therefore cannot be excluded. Finally, most PCT measurements at or below 0.05 ng/mL were retained as thresholded values rather than exact numerical concentrations, limiting the precision of descriptive distributional statistics.

Despite these limitations, the study provides preliminary data on serum PCT concentrations among patients with DM with and without active pulmonary TB in a primary healthcare setting. Future studies should employ larger and more balanced samples, multicenter recruitment, standardized microbiological and radiological confirmation of pulmonary TB, detailed assessment of TB severity and treatment status, HbA1c measurement, renal-function assessment, and systematic evaluation of concomitant infections and inflammatory conditions. Prospective diagnostic-accuracy studies incorporating receiver operating characteristic analysis,

sensitivity, specificity, and predictive values would be required to determine whether PCT has diagnostic utility for identifying pulmonary TB among patients with diabetes mellitus.

CONCLUSION

This study did not identify a statistically significant difference in serum procalcitonin concentrations between patients with diabetes mellitus without pulmonary tuberculosis and those with diabetes mellitus complicated by active pulmonary tuberculosis. Most participants in both groups had PCT concentrations within the normal reference range, and the between-group comparison was not statistically significant (U = 230.000, Z = −1.857, p = 0.063). These findings should be interpreted as evidence from a between-group comparison rather than as an assessment of the diagnostic or discriminatory performance of procalcitonin.

SUGGESTION

Future studies should include larger and more balanced samples, multicenter recruitment, standardized microbiological and radiological confirmation of pulmonary tuberculosis, detailed assessment of tuberculosis severity and treatment status, measures of long-term glycemic control such as HbA1c, and systematic evaluation of conditions that may independently influence PCT concentrations. Diagnostic-accuracy studies incorporating receiver operating characteristic analysis, sensitivity, specificity, and predictive values are also needed to determine the potential clinical utility of PCT in patients with diabetes mellitus and pulmonary tuberculosis.

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