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Carbohydrate and Lipid Metabolic Profiles in Hospitalized Pulmonary Tuberculosis Patients at Dr. Soetomo General Academic Hospital, Indonesia: A Comparative Study with Healthy Control

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ABSTRACT

Introduction: Tuberculosis (TB) is an infectious disease that can affect various organs, especially the lungs and is caused by Mycobacterium tuberculosis. TB patients experience changes in carbohydrate and lipid metabolism as a result of metabolic modulation by Mtb in order to survive in the host body. ***Objective:*** To analyze the differences in carbohydrate and lipid metabolic profiles between pulmonary TB patients in the inpatient ward compared to healthy controls. ***Methods:*** This study is a retrospective observational analytical study with a case-control design using secondary data in the form of electronic medical records of patients at Dr. Soetomo General Academic Hospital in 2024, with variables including fasting plasma glucose, total cholesterol, LDL cholesterol, HDL cholesterol, and triglyceride. ***Result:*** Most hospitalized pulmonary TB patients were male (60.5%) with the largest age range being 45-59 years (39.5%). Most pulmonary TB patients had a normal BMI (63.2%) and rifampicin-sensitive results in GeneXpert MTB/RIF (78.9%). Comparison test results showed no significant differences in glucose ($p = 0.108$) and triglyceride ($p = 0.496$) levels between the case and control groups. However, significant differences were found in total cholesterol levels ($p < 0.001$), HDL ($p < 0.001$), and LDL ($p = 0.014$). ***Conclusion:*** There were significant differences in lipid metabolism profiles, especially in total cholesterol, HDL, and LDL levels, between pulmonary TB patients hospitalized and the healthy control group. However, no significant differences were found in fasting plasma glucose and triglyceride levels.

Keywords: tuberculosis; metabolism; fasting plasma glucose; lipid profile

INTRODUCTION

Tuberculosis (TB) remains one of the most common infectious diseases that requires global attention due to high morbidity and mortality rate. TB is an infectious disease that can affect various organs, especially the lungs and caused by Acid Fast-Bacilli (AFB) Mycobacterium tuberculosis [1]. Indonesia ranks second in the world for the highest burden of TB cases, after India. The incidence of TB in Indonesia reached 821.200 cases in 2023. This figure represents an increase from 2022, when there were 724.309 cases. East Java Province was the second-highest contributor to TB cases in Indonesia, with 78.334 cases in 2022, and saw an increase to 86.969 cases in 2023. The proportion of pulmonary TB patients in Indonesia is higher among males, at 57,8%, compared to females at 42,2% [2, 3].

In TB patients, one of the classic signs frequently found is massive weight loss accompanied by loss of muscle and fat mass as a result of metabolic wasting syndrome [4]. A body infected by M. tuberculosis experiences an increase in cellular metabolic demand to maintain its physiological functions by increasing energy use even at rest. This process is known as resting energy expenditure [5]. Malnutrition or a decline in nutritional status is one of the factors that can increase the risk of developing TB, particularly in settings with a high TB burden [6].

Pulmonary TB patients are known to experience changes in their body's metabolic processes, such as the metabolism of carbohydrate, lipid, and protein [7]. M. tuberculosis is capable of doing metabolic flexibility, enabling it to survive long-term within the host's body [8]. It is estimated that M. tuberculosis uses glucose as its primary carbon source at the beginning of replication and under aerobic conditions. Over time, M. tuberculosis becomes increasingly dependent on lipids as a carbon source [9].

TB and hyperglycemia are bidirectionally associated. M. tuberculosis is capable of inducing a metabolic shift in Alveolar Macrophages (AM) and Monocyte-derived Macrophages (MDM) toward glycolysis process, thereby facilitating the production of IL-1β to activate the immune response and suppress the production of the anti-inflammatory cytokine IL-10 [10]. The inflammatory process caused by M. tuberculosis infection triggers the release of cytokines and increases the release of stress hormones such as cortisol, catecholamines, epinephrine, norepinephrine, and growth hormone, resulting in elevated blood glucose levels through increased gluconeogenesis process [11].

A previous study has shown that TB patients tend to have lower plasma cholesterol levels than the healthy (general) population. This condition is primarily observed in patients who have not yet undergone treatment [12]. TB is associated with an increase in the number of free radicals in the body due to oxidative stress triggered by increased phagocytosis of M. tuberculosis by monocytes and macrophages, which ultimately affects the catabolism of cell membrane lipids, particularly triglycerides and LDL. Lipids in blood plasma are then used to replace the degraded cell membrane triglycerides and LDL, thereby causing a decrease in plasma lipid levels. HDL cholesterol levels also decrease as a result of increased catabolism during the inflammatory process. The inflammatory response is characterized by increased levels of phospholipase A2 and serum Amyloid A, both of which are responsible for stimulating HDL catabolism [13].

Hypocholesterolemia is also known to be a risk factor for the development of TB. Cholesterol is a lipid molecule that makes up cell membranes. In the host body, lipids (particularly cholesterol) play a role in supporting the immune system. Cholesterol is essential for supporting the cytotoxic function of lymphocytes and helps activate lymphocytes, including CD4+ and CD8+ cells. These cells can trigger the recruitment of macrophages and the release of pro-inflammatory cytokines such as IFN and TNF. Meanwhile, cholesterol is also responsible for phagocytic functions such as cell motility, exocytosis, and endocytosis. All these functions can be disrupted if cholesterol levels are insufficient [12, 14].

Based on these data and phenomena, the authors were interested in conducting a study titled "Carbohydrate and Lipid Metabolic Profiles in Hospitalized Pulmonary Tuberculosis Patients at Dr. Soetomo General Academic Hospital, Indonesia: A Comparative Study with Healthy Controls".

METHODS

This study is an observational analytical study using a case-control design, with the aim of examining or comparing data or variables between the case and control groups. The data were obtained from secondary sources in the form of electronic medical records of patients at Dr. Soetomo General Hospital in Surabaya. The study population was divided into case and control groups. The case population consisted of all adult patients with pulmonary tuberculosis who were hospitalized at Dr. Soetomo General Academic Hospital in Surabaya during 2024, while the control population consisted of healthy individuals who underwent examinations at the Medical Check-Up (MCU) clinic at Dr. Soetomo General Academic Hospital in Surabaya during 2024.

The inclusion criteria for the case group were patients with bacteriologically diagnosed pulmonary TB (GeneXpert MTB/RIF) who were at least 18 years old and hospitalized. Meanwhile, the inclusion criteria for the control group were healthy individuals who were at least 18 years old and had no history of previous TB infection. TB patients whose diagnosis was not confirmed by GeneXpert, those with comorbidities such as HIV/AIDS, diabetes mellitus, cancer, and autoimmune diseases, as well as patients with incomplete medical records, were excluded from the study. Sampling was conducted using consecutive sampling with a 1:1 ratio for each group, resulting in a sample size of 38 patients in the case group and 38 patients in the control group who met the inclusion criteria.

The data collected included patient characteristics, such as gender, age, BMI, and anti-tuberculosis drug sensitivity status in the case group. Carbohydrate metabolism was assessed using Fasting Plasma Glucose (FPG) which was categorized into normoglycemia and hyperglycemias based on PERKENI criteria. While lipid metabolism was assessed using plasma total cholesterol, HDL cholesterol, LDL cholesterol, and triglyceride levels. The data obtained were analyzed using two types of analysis: univariate and bivariate analysis. Univariate analysis was conducted to describe the distributions of patient's characteristics. Bivariate or inferential analysis is also conducted to compare glucose and lipid profile between two groups.

This study was conducted in accordance with the principles of research ethics, including confidentiality, anonymity, and beneficence. This study has received approval and a certificate of ethical compliance from the Ethics Committee of Dr. Soetomo General Academic Hospital, Surabaya. All patient data collected were used exclusively for research and academic purposes.

RESULTS AND DISCUSSION

Sample Characteristics

Table 1 presents the characteristics of the sample from two groups. Among the case group, the majority of patients were male (60.5%), whereas the control group was dominated by female (71.1%). These findings are consistent with previous studies that identified risk factors for TB infection, including a higher prevalence of pulmonary TB among males [15, 16]. This can be explained by differences in behavioral and physiological hypotheses between the males and females. The. The behavioral hypotheses include social role and activities such as traveling, smoking, and alcohol consumption. The physiological hypothesis suggests that male's susceptibility to TB infection is likely due to the modulation of the immune system by sex hormones [16, 17].

The average age in both groups also had its own characteristics: 51.89 ± 15.39 years in the case group, while the average age in the control group was slightly lower than that of the case group, at 43.16 ± 14.97 years. The age distribution in both groups was dominated by individuals of working age. Previous studies have shown similar results, with the highest percentage of TB patients found in the ≥46-year-old age group, at 42.9% [15]. In previous cohort studies, it has been reported that younger individuals particularly those under 15 years of age tend to have a lower risk of TB infection. This is likely due to the positive protective effect of the BCG vaccination program and the limited diagnostic testing conducted in this age group.

An increased risk of infection may be directly proportional to age, likely due to accumulated exposure over time, including exposure to M. tuberculosis, air pollution, tobacco smoke, weakened immunity, and other determinants [18].

Regarding BMI, it can be observed that the mean BMI in the case group (20.68 ± 3.22 kg/m^2) was indeed slightly lower than that in the control group (24.01 ± 5.35 kg/m^2). And if observed further, the two groups show different characteristics in terms of BMI classification percentages. In the case group, the majority of patients (63.2%) were classified as having a normal BMI. Meanwhile, in the control group, 47.4% fell into the categories of overweight, class I obesity, or class II obesity. While 42.1% had a normal BMI. These findings differ from the previous studies, which reported a bidirectional association between tuberculosis and malnutrition [6]. These differences are likely to be due to variations in population demographics, socioeconomic status, educational levels, and environmental factors that may influence the distribution of BMI in each region. A study explains that an infection can cause changes in a person's nutritional status, and this depends on their prior nutritional status (before the infection occurred), the duration of the infection, and their food intake during treatment [19]. Malnutrition or undernutrition also can impair the immune system's ability to fight pathogens that cause infections, including M. tuberculosis, and therefore may affect the severity of the infection [20].

A study in China found that BMI values exceeding the normal range may be a risk factor for MDR-TB and resistance to certain combinations of anti-TB drugs, such as isoniazid (INH), rifampicin (RFP), and streptomycin (SM). This is likely due to reduced M. tuberculosis virulence and slower replication rates resulting from high-fat exposure, as well as the influence of leptin-mediated immunoregulation [21].

TABLE 1: Sample Characteristics.

Characteristic	Case group (n=38)	Control group (n=38)
Gender/Sex		
Male	23 (60.5)	11 (28.9)
Female	15 (39.5)	27 (71.1)
Age (years), mean±SD	51.89 ± 15.39	43.16 ± 14.97
18-44 years	12 (31.6)	19 (50.0)
45-59 years	15 (39.5)	15 (39.5)
60-74 years	8 (21.1)	3 (7.9)
≥ 75 years	3 (7.9)	1 (2.6)
BMI (kg/m^2), mean±SD	20.68 ± 3.22	24.01 ± 5.35
Underweight	8 (21.1)	4 (10.5)
Normal	24 (63.2)	16 (42.1)
Overweight	1 (2.6)	2 (5.3)
Class I obesity	5 (13.2)	12 (31.6)
Class II obesity	-	4 (10.5)

Values are presented as number (%) or mean; BMI was classified based on Asia-Pacific criteria; SD: Standard Deviation; BMI: Body Mass Index; Underweight: <18.5 kg/m^2; Normal: 18.5-22.9 kg/m^2; Overweight: 23.0-24.9 kg/m^2; Class I obesity: 25.0-29.9 kg/m^2; Class II obesity: ≥30.0 kg/m^2

Drug Sensitivity Status among the Case Group

Table 2 shows the distribution of drug susceptibility to antituberculosis drugs in the case group, as determined by GeneXpert MTB/RIF test. Most of the participants in the case group were found to be susceptible to rifampicin, which was 30 individuals (78.9%), while the remaining 8 individuals (21.1%) were rifampicin resistant. The risk of developing drug-resistant TB is higher among individuals with a previous history of TB treatment. In addition, TB patients with a history of treatment failure, TB recurrence, and treatment dropout are also at higher risk of developing drug-resistant TB [22].

TABLE 2: Distribution of Drug Sensitivity Status.

Status	N	(%)
MTB detected, Rif sensitive	30	78.9
MTB detected, Rif resistant	8	21.1
Total	38	100

Glucose Profile

Glucose levels are classified into two groups: normoglycemia and hyperglycemia, based on the criteria from the Indonesian Endocrinology Association (Perkumpulan Endokrinologi Indonesia [PERKENI]) [23]. In this study, FPG was the variable (data) that was fully available in the medical records of all study subjects. FBG levels were classified as hyperglycemia if the amount was ≥ 126 mg/dL [23].

Descriptively, both groups have almost the same characteristics, with the majority of subjects classified as normoglycemic, although the percentage of normoglycemic subjects in the case group (84.2%) was lower than in the control group (97.4%). The proportion of hyperglycemia in the case group was higher (15.8%) than in the control group (2.6%). However, this difference was not statistically significant, as indicated by a p = 0.108.

Previous studies have shown that glucose metabolism in TB patients can change significantly as a result of systemic inflammation that disrupts the body's ability to regulate blood glucose, leading to hyperglycemia or permanent insulin resistance in some cases [24].

However, the non-significant difference in proportions observed in this study is likely due to the hyperglycemia mechanism caused by stress. In TB patients without diabetes mellitus, the hyperglycemia that occurs is stress-induced hyperglycemia and is reversible in most cases, so glucose levels will return to normal once the trigger or source of the infection is eliminated. In active TB infection, pro-inflammatory cytokines such as IL-1, IL-6, IL-10, and TNF-α are produced, which stimulate the hypothalamic-pituitary axis to increase the production of counter-insulin hormones such as cortisol, ACTH, prolactin, and growth hormone. This leads to increased gluconeogenesis and glycolysis, increased insulin resistance, and decreased glucose uptake by tissues. Meanwhile, macrophages release nitric oxide (NO) and reactive oxygen species (ROS), which also contribute to increased insulin resistance in adipose tissue, muscle, and the liver [25].

The non-significant results of this study may also be due to differences in the duration of treatment among patients. One study reported that hyperglycemia was more common among TB patients in the early stages of diagnosis. However, after undergoing TB treatment, 68% of patients experienced an improvement in their glucose levels, which returned to normal [26]. However, there haven't been any studies confirming the duration of hyperglycemia in TB patients who do not have diabetes or how far this can be a predisposing factor for diabetes or persistent hyperglycemia.

TABLE 3: FBG Distribution and Comparison between Two Groups.

	Case group (n=38)	Control group (n=38)	p-value
FPG			
Normoglycemia	32 (84.2)	37 (97.4)	0.108
Hyperglycemia	6 (15.8)	1 (2.6)	
Total	38 (100)	38 (100)	

Values are presented as number (%); p-value was calculated using the Fisher's Exact test; FPG: Fasting Plasma Glucose; Normoglycemia: FPG <126 mg/dL; Hyperglycemia: FPG ≥ 126 mg/dL

Lipid Profile

In general, plasma lipid levels in the case group tended to be lower than those in the control group, based on both average and median (Q1–Q3) values. However, statistically, only 3 of the 4 plasma lipid parameters showed a significant difference between the two groups. These 3 variables were total cholesterol, HDL cholesterol, and LDL cholesterol.

The case group had a median total cholesterol level of 148.50 (125.75–179.25) mg/dL, while the control group had a median level of 185.00 (170.00–214.25) mg/dL; this difference was statistically significant (p < 0.001). Furthermore, the median HDL cholesterol level in the case group (35.50 (26.00-44.75) mg/dL) was also found to be lower than in the control group (53.00 (49.00-65.50) mg/dL). This difference was statistically significant (p < 0.001). A significant difference was also found in LDL cholesterol levels (p = 0.014).

A previous study has reported low plasma lipid concentrations in TB patients at the time of initial diagnosis [12]. Activated macrophages after *M. tuberculosis* invasion trigger the release of free radicals, which can reduce the body's antioxidant capabilities. Malondialdehyde and nitric oxide as markers of oxidative stress, are elevated in TB patients, accompanied by decreased lipid levels [27, 28].

In contrast to total cholesterol, HDL, and LDL levels, this study found no statistically significant difference in triglyceride levels between the case group and the healthy control group (p = 0.496). This finding differs from a previous study, which reported significant differences in triglyceride levels between the two groups [14]. One factor that may explain the different results of our study is fasting status prior to lipid testing. Triglyceride levels are affected by a patient's nutritional intake or prandial status. In hospitalized pulmonary TB patients, the patients' prandial status at the time of blood sample collection for lipid profile testing cannot be determined.

A study has shown that there are changes in the average peak lipid levels 1–6 hours after a meal, specifically a 26 mg/dL increase in triglycerides, an 8 mg/dL decrease in total cholesterol and LDL, while HDL is not affected by prandial status [29]. Dietary cholesterol and triglycerides are absorbed in the intestinal lumen by the enterocytes, then assembled into chylomicrons and transported via the lymphatic system into the bloodstream, where they are hydrolyzed by lipoprotein lipase [30]. Triglycerides are lipid components that tend to be more easily affected by various short-term factors. Therefore, another possible factor contributing to the non-significant triglyceride test results is the distribution of nutritional status or BMI, which could not be controlled in this study. Each subject group had a different BMI distribution, and the majority of patients in the case group had a normal BMI. This allowed triglyceride levels in the case subjects to be relatively similar to those in the control group.

Hypocolesterolemia may be a risk factor for tuberculosis infection [14, 31]. Low cholesterol levels in macrophage cell membranes can cause macrophages to be unable to internalize *M. tuberculosis*, thereby weakening the body's defence mechanisms against *M. tuberculosis* and allowing latent TB to develop into active disease [32]. Cholesterol levels and the risk of TB infection are inversely related. However, this does not mean that a high-cholesterol diet can help reduce an individual's risk of TB infection [33].

TABLE 4: Lipid Profile Comparison between Two Groups.

Variable	Groups	Mean ± SD (mg/dL)	Median (Q1-Q3) (mg/dL)	p-value
Total cholesterol	Case group	148.37 ± 54.11	148.50 (125.75-179.25)	< 0.001
	Control group	187.03 ± 35.58	185.00 (170.00-214.25)	
HDL cholesterol	Case group	38.32 ± 20.77	35.50 (26.00-44.75)	< 0.001
	Control group	56.24 ± 13.45	53,00 (49.00-65.50)	
LDL cholesterol	Case group	96.61 ± 34.19	95.50 (76.25-116.50)	0.014
	Control group	116.61 ± 34.724	115.00 (93.00-139.75)	
Triglyceride	Case group	106.29 ± 58.13	97.50 (72.00-131.50)	0.496
	Control group	118.82 ± 81.98	104.50 (87.25-130.25)	

Values are presented as mean ± SD and median (Q1-Q3); p-values were calculated using the Independent T-Test for LDL (normally distributed variable) and also the Mann-Whitney U Test for total cholesterol, HDL cholesterol, and triglyceride (non-normally distributed variables); SD: Standard Deviation; HDL: High-Density Lipoprotein; LDL: Low-Density Lipoprotein.

CONCLUSION

From an epidemiological aspect, hospitalized pulmonary TB patients (case group) are predominantly male and of working age, with the majority having a normal BMI. There were significant differences in lipid metabolism profiles, particularly in total cholesterol, HDL, and LDL levels, between hospitalized pulmonary TB patients (case group) and the healthy control group. However, no significant differences were found in fasting plasma glucose and triglyceride levels, which were likely due to differences in treatment duration, nutritional intake, and prandial status that could not be controlled in this study.

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REFERENCES

[1] A. Rasyid and L. Heryawan, "Klasifikasi Penyakit Tuberculosis (TB) Organ Paru Manusia Berdasarkan Citra Rontgen Thorax Menggunakan Metode Convolutional Neural Network (CNN)," J. Manaj. Inf. Kesehat. Indones., vol. 11, no. 1, Mar. 2023, doi: 10.33560/JMIKI.V11I1.484.

[2] Kemenkes RI, "Laporan Program Penanggulangan Tuberkulosis Tahun 2023," 2024, [Online]. Available: https://www.tbindonesia.or.id/wp-content/uploads/2024/12/Laporan-Program-Penanggulangan-TBC-2023_Final.pdf

[3] Depkes RI, "Laporan Program Penanggulangan Tuberkulosis Tahun 2022," Kemenkes RI, pp. 1–147, 2023, [Online]. Available: https://tbindonesia.or.id/pustaka_tbc/laporan-tahunan-program-tbc-2021/

[4] Y. Ding et al., "Tuberculosis causes highly conserved metabolic changes in human patients, mycobacteria-infected mice and zebrafish larvae," Sci. Rep., vol. 10, no. 1, p. 11635, Dec. 2020, doi: 10.1038/S41598-020-68443-Y.

[5] Nurjannah and I. M. Sudana, "Analisis Pengaruh Fase Pengobatan, Tingkat Depresi dan Konsumsi Makanan Terhadap Status Gizi Penderita Tuberkulosis (TB) Paru Di Wilayah Kerja Puskesmas se-Kecamatan Genuk Kota Semarang," Public Heal. Perspect. J., vol. 2, no. 3, pp. 215–233, 2017.

[6] J. Ockenga et al., "Tuberculosis and malnutrition: The European perspective," Clin. Nutr., vol. 42, no. 4, pp. 486–492, Apr. 2023, doi: 10.1016/J.CLNU.2023.01.016/ATTACHMENT/80BBF1B3-E3A5-408A-AA10-72039A32DEBA/MMC1.DOCX.

[7] E. A. Daniel et al., "Predictive metabolite signatures for risk of progression to active TB from QuantiFERON supernatants of household contacts of TB patients," Emerg. Microbes Infect., vol. 14, no. 1, Dec. 2025, doi: 10.1080/22221751.2024.2437242.

[8] D. P. Shan Chang and X. L. Guan, "Metabolic versatility of mycobacterium tuberculosis during infection and dormancy," Metabolites, vol. 11, no. 2, pp. 1–21, 2021, doi: 10.3390/metabo11020088.

[9] D. F. Warner, "Mycobacterium tuberculosis Metabolism," Cold Spring Harb. Perspect. Med., vol. 5, no. 4, p. a021121, 2015, doi: 10.1101/CSHPERSPECT.A021121.

[10] L. E. Gleeson et al., "Cutting Edge: Mycobacterium tuberculosis Induces Aerobic Glycolysis in Human Alveolar Macrophages That Is Required for Control of Intracellular Bacillary Replication," J. Immunol., vol. 196, no. 6, pp. 2444–2449, 2016, doi: 10.4049/jimmunol.1501612.

[11] M. Byers and E. Guy, "The Complex Relationship Between Tuberculosis and Hyperglycemia," Diagnostics, vol. 14, no. 22, p. 2539, Nov. 2024, doi: 10.3390/DIAGNOSTICS14222539.

[12] G. Gebremicael et al., "Lipid Profile in Tuberculosis Patients with and without Human Immunodeficiency Virus Infection," Int. J. Chronic Dis., vol. 2017, p. 3843291, 2017, doi: 10.1155/2017/3843291.

[13] Taparia P, Yadav D, Koolwal S, and Mishra S, "Study of lipid profile in pulmonary tuberculosis patients and relapse cases in relation with disease severity - A pilot study," Int. J. Sci. Appl. Res., vol. 2, no. 1, pp. 41–50, 2015, doi: 10.65122/IJSAR.

[14] A. P. Mani, S. K, D. K. K, and S. Yadav, "Assessment of Lipid Profile in Patients with Pulmonary Tuberculosis: An Observational Study," Cureus, vol. 15, no. 5, p. e39244, May 2023, doi: 10.7759/CUREUS.39244.

[15] F. Fahdhienie, M. Mudatsir, T. F. Abidin, and N. Nurjannah, "Risk factors of pulmonary tuberculosis in Indonesia: A case-control study in a high disease prevalence region," Narra J, vol. 4, no. 2, p. e943, Aug. 2024, doi: 10.52225/NARRA.V4I2.943.

[16] M. Humayun, J. Chirenda, W. Ye, I. Mukeredzi, H. A. Mujuru, and Z. Yang, "Effect of Gender on Clinical Presentation of Tuberculosis (TB) and Age-Specific Risk of TB, and TB-Human Immunodeficiency Virus Coinfection," Open Forum Infect. Dis., vol. 9, no. 10, p. ofac512, Oct. 2022, doi: 10.1093/OFID/OFAC512.

[17] S. Nhamoyebonde and A. Leslie, "Biological differences between the sexes and susceptibility to tuberculosis," J. Infect. Dis., vol. 209, no. SUPPL. 3, 2014, doi: 10.1093/infdis/jiu147.

[18] Z. Dong et al., "Age–period–cohort analysis of pulmonary tuberculosis reported incidence, China, 2006–2020," Infect. Dis. Poverty 2022 111, vol. 11, no. 1, pp. 85-, Jul. 2022, doi: 10.1186/S40249-022-01009-4.

[19] N. S. Scrimshaw and J. P. SanGiovanni, "Synergism of nutrition, infection, and immunity: An overview," Am. J. Clin. Nutr., vol. 66, no. 2, 1997, doi: 10.1093/ajcn/66.2.464S.

[20] P. Sinha et al., "Undernutrition and Tuberculosis: Public Health Implications," J. Infect. Dis., vol. 219, no. 9, p. 1356, Apr. 2018, doi: 10.1093/INFDIS/JIY675.

[21] W. mei Song et al., "Association between body mass index and newly diagnosed drug-resistant pulmonary tuberculosis in Shandong, China from 2004 to 2019," BMC Pulm. Med., vol. 21, no. 1, p. 399, Dec. 2021, doi: 10.1186/S12890-021-01774-2.

[22] B. Kedokteran Masyarakat, E. Nurjanah Widiastuti, Y. Wijayanti Subronto, and D. Promono, "Determinan kejadian multi-drug resistant tuberculosis di rumah sakit Dr. Sardjito Yogyakarta," Ber. Kedokt. Masy., vol. 33, no. 7, pp. 325–330, Jul. 2017, doi: 10.22146/bkm.18290.

[23] PERKENI, "Pedoman Pengelolaan dan Pencegahan Diabetes Melitus Tipe 2 Dewasa di Indonesia 2021," p. 46, 2021, [Online]. Available: www.ginasthma.org.

[24] M. K. Bisht, P. Dahiya, S. Ghosh, and S. Mukhopadhyay, "The cause–effect relation of tuberculosis on incidence of diabetes mellitus," Front. Cell. Infect. Microbiol., vol. 13, p. 1134036, Jun. 2023, doi: 10.3389/FCIMB.2023.1134036/XML/NLM.

[25] M. J. Magee, A. D. Salindri, N. T. T. Kyaw, S. C. Auld, J. S. Haw, and G. E. Umpierrez, "Stress Hyperglycemia in Patients with Tuberculosis Disease: Epidemiology and Clinical Implications," Curr. Diab. Rep., vol. 18, no. 9, p. 71, Sep. 2018, doi: 10.1007/S11892-018-1036-Y.

[26] N. Boillat-Blanco et al., "Association between tuberculosis, diabetes and 25 hydroxyvitamin D in Tanzania: a longitudinal case control study," BMC Infect. Dis. 2016 161, vol. 16, no. 1, pp. 626-, Nov. 2016, doi: 10.1186/S12879-016-1960-X.

[27] O. O. Samuel, A. A. Adeyemi, T. O. Olusegun, R. O. Nihinlade, and E. Ayokunle, "Oxidative Stress and Lipid Profile Status in Pulmonary Tuberculosis Patients in South Western Nigeria," Greener Journal of Medical Sciences. Accessed: Dec. 06, 2025. [Online]. Available: https://gjournals.org/GJMS/archive/vol-3-6-august-2013-2/oyedeji-et-al.html

[28] V. Wagh, S. Rajopadhye, S. Mukherjee, A. Urhekar, and D. Modi, "Assessment of oxidative stress in serum of pulmonary tuberculosis patients," Int. J. Res. Med. Sci., vol. 4, no. 8, pp. 3328–3332, 2016, doi: 10.18203/2320-6012.IJRMS20162288.

[29] B. G. Nordestgaard et al., "Fasting is not routinely required for determination of a lipid profile: clinical and laboratory implications including flagging at desirable concentration cut-points—a joint consensus statement from the European Atherosclerosis Society and European Feder…," Eur. Heart J., vol. 37, no. 25, pp. 1944–1958, Jul. 2016, doi: 10.1093/EURHEARTJ/EHW152.

[30] B. G. Nordestgaard, "A Test in Context: Lipid Profile, Fasting Versus Nonfasting," J. Am. Coll. Cardiol., vol. 70, no. 13, pp. 1637–1646, Sep. 2017, doi: 10.1016/J.JACC.2017.08.006.

[31] C. Pérez-Guzmán and M. H. Vargas, "Hypocholesterolemia: A major risk factor for developing pulmonary tuberculosis?," Med. Hypotheses, vol. 66, no. 6, pp. 1227–1230, Jan. 2006, doi: 10.1016/J.MEHY.2005.12.041.

[32] K. Divya, K. G. R. Mallan, and R. Thamarai, "An Observational Study of Lipid Profile in Pulmonary Tuberculosis Patients," J. Assoc. Pulmonologist Tamil Nadu, vol. 8, no. 3, pp. 109–113, Sep. 2025, doi: 10.4103/JAPT.JAPT_24_25.

[33] Y. S. Jo et al., "Relationship between total cholesterol level and tuberculosis risk in a nationwide longitudinal cohort," Sci. Reports 2021 111, vol. 11, no. 1, pp. 16254-, Aug. 2021, doi: 10.1038/s41598-021-95704-1.