

Association of Nutritional Status and Glycemic Control with Lipid Profile and Metabolic Syndrome in Adolescents with Type 2 Diabetes Mellitus at Dr. Moewardi Regional Hospital, Indonesia

Hanum Ferdian^{1,2)}, Annang Giri Moelyo^{1,2)}

¹⁾Division of Endocrinology, Department of Pediatrics, Faculty of Medicine, Universitas Sebelas Maret, Indonesia

²⁾Dr. Moewardi Regional Hospital, Surakarta, Indonesia

Received: February 10, 2026; Accepted: March 29, 2026; Available online: May 16, 2026

ABSTRACT

Background: Adolescent type 2 diabetes mellitus (T2DM) is becoming more common worldwide, a trend tied to obesity, metabolic syndrome, and dyslipidemia that raise cardiovascular risk from a young age. In Indonesia, evidence describing adolescents with this condition is still scarce. The present study sought to examine how nutritional status and glycemic control relate to lipid profile and metabolic syndrome among adolescents with T2DM treated at Dr. Moewardi Regional Hospital.

Subjects and Method: A cross-sectional analytic observational design was applied to 51 patients with T2DM, aged 12–18 years, at Dr. Moewardi Regional Hospital. The variables assessed comprised age, sex, body mass index (BMI), presence of metabolic syndrome, HbA1c, and a fasting lipid panel (total cholesterol, LDL, HDL, triglycerides). For bivariate testing, the Chi-Square/Fisher's Exact Test and the Mann-Whitney U test were applied, whereas the link between HbA1c and the lipid panel was examined through Spearman correlation. Independent predictors were then explored using binary logistic regression.

Results: Among the 51 patients, females predominated (92.2%), the 12–14 year bracket was most frequent (49.0%), and the mean HbA1c at diagnosis reached $12.66 \pm 2.88\%$. Obesity was present in 52.9% and metabolic syndrome in 51.0%. Levels of total cholesterol, triglycerides, and LDL were markedly elevated among those with metabolic syndrome (all $p < 0.001$). HbA1c correlated strongly and positively with triglycerides ($r_s = 0.752$; $p < 0.001$) and LDL ($r_s = 0.674$; $p < 0.001$). On multivariate testing, obesity emerged as an independent predictor of metabolic syndrome ($OR = 166.12$; $CI_{95\%} 9.88$ to 2792.23 ; $p < 0.001$).

Conclusion: Obesity, metabolic syndrome, and dyslipidemia were highly prevalent in these adolescents with T2DM. Because both obesity and inadequate glycemic control were significantly associated with the metabolic disease burden, early screening together with comprehensive management of cardiometabolic comorbidities is warranted.

Keywords: Type 2 diabetes mellitus, adolescents, metabolic syndrome

Correspondence:

Hanum Ferdian. Division of Endocrinology, Department of Pediatrics, Dr. Moewardi Regional Hospital, Jl. Kolonel Sutarto 132, Surakarta 57126, Indonesia. Email: lynne06chelin@gmail.com

Cite this as:

Ferdian H, Moelyo AG (2026). Association of Nutritional Status and Glycemic Control with Lipid Profile and Metabolic Syndrome in Adolescents with Type 2 Diabetes Mellitus at Dr. Moewardi Regional Hospital. *J Matern Child Health*. 11(3): 214-225. <https://doi.org/10.26911/thejmch.2026.11.03.06>.



© Hanum Ferdian. Published by Master's Program of Public Health, Universitas Sebelas Maret, Surakarta. This open-access article is distributed under the terms of the [Creative Commons Attribution 4.0 International \(CC BY 4.0\)](https://creativecommons.org/licenses/by/4.0/). Re-use is permitted for any purpose, provided attribution is given to the author and the source is cited.

BACKGROUND

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder defined by insulin resistance alongside progressive failure of pancreatic beta-cell function. Although it was historically regarded as a disease of adulthood, over the last two decades it has increasingly affected children and adolescents, mirroring the worldwide climb in pediatric obesity (Magliano and Boyko, 2021). The International Diabetes Federation (IDF) estimates roughly 41,600 new pediatric and adolescent T2DM cases each year worldwide, with incidence rising by 4.8% annually in the 10–19-year age band (Wu et al., 2022).

Data from the SEARCH for Diabetes in Youth study showed that adolescent T2DM incidence grew by 4.8% per year between 2002 and 2018, with steeper increases among females and ethnic minority populations (Lawrence et al., 2021). This escalating burden is particularly worrying because disease that begins in youth follows a harsher course than its adult counterpart, marked by more rapid loss of beta-cell function, greater difficulty achieving glycaemic targets, and earlier appearance of both microvascular and macrovascular complications (Nadeau et al., 2016).

In the Treatment Options for Type 2 Diabetes in Adolescents and Youth (TODAY) trial, fewer than 15 years after diagnosis over 60% of adolescent participants had acquired at least one microvascular complication and about 28% had developed several (TODAY Study Group, 2021). Such observations underscore how essential timely detection and accurate clinical profiling are in this age group. The pathogenesis of adolescent T2DM is closely intertwined with central adiposity, insulin resistance, and additional metabolic syndrome features including dyslipidaemia

and hypertension (Kelsey and Zeitler, 2016). Bjornstad et al. (2021) further noted that affected adolescents carry an atherogenic lipid pattern—elevated triglycerides, lower dense LDL particles, and lowered HDL cholesterol—that heightens the likelihood of premature cardiovascular disease. The 2022 consensus of the International Society for Pediatric and Adolescent Diabetes (ISPAD) therefore recommends that every adolescent receiving a T2DM diagnosis undergo comprehensive evaluation for cardiometabolic comorbidities (Shah et al., 2022).

While the interplay of obesity, glycemic control, and metabolic disturbance has been examined extensively in adults and in Western adolescent cohorts, comparable evidence from Asian adolescents—Indonesians in particular—remains sparse. Detailed cardiometabolic profiles of adolescents with T2DM have rarely been published locally, even though the 2018 National Basic Health Research (Riskesdas) documented a rising prevalence of diabetes across every age group (Ministry of Health of the Republic of Indonesia, 2019). Differences in body phenotype, fat distribution, and genetic determinants that drive insulin resistance at comparatively lower BMI levels mean that Asian adolescents with T2DM may present clinically in ways distinct from Caucasian populations (Yoon et al., 2006).

Serving as a tertiary referral centre for Central Java, Dr. Moewardi Regional Hospital draws adolescent T2DM patients from across the region, creating a useful setting in which to study how clinical and metabolic variables interrelate in this group. Accordingly, this investigation set out to characterise the relationship of nutritional status and glycaemic control with lipid profile and metabolic syndrome in adolescents with T2DM, aiming to support

evidence-based screening, management protocols, and prevention of long-term complications.

SUBJECTS AND METHOD

1. Study Design

An analytic observational study of cross-sectional design was carried out using medical-record data from adolescents with T2DM managed at the Pediatric Endocrinology Division of Dr. Moewardi Regional Hospital, Surakarta.

2. Population and Sample

Eligible participants were all patients aged 12–18 years whose T2DM had been established according to American Diabetes Association (ADA) and ISPAD criteria—that is, (1) Fasting plasma glucose ≥ 126 mg/dL (7.0 mmol/L), confirmed on repeat testing; or (2) Two-hour plasma glucose ≥ 200 mg/dL (11.1 mmol/L) during a 75-g oral glucose tolerance test (OGTT); or (3) HbA1c $\geq 6.5\%$ (≥ 48 mmol/mol), confirmed on repeat testing; or (4) Random plasma glucose ≥ 200 mg/dL (11.1 mmol/L) with classic symptoms of hyperglycemia accompanied by features of insulin resistance (obesity, acanthosis nigricans) together with preserved C-peptide levels (≥ 0.6 ng/mL) confirming preserved endogenous insulin secretion (Shah et al., 2022; American Diabetes Association, 2022). Fifty-one patients met these criteria from January 2021 to December 2025.

3. Study Variables

The dependent variables were lipid profile abnormalities and the presence of metabolic syndrome. Lipid profile consisted of total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, and triglyceride levels. The independent variables were nutritional status and glycemic control. Nutritional status was assessed using body mass index (BMI)-for-age according to the World

Health Organization (WHO) growth reference and categorized as obese and non-obese. Glycemic control was assessed using glycated hemoglobin (HbA1c) levels. Demographic characteristics including age and sex were also collected and analyzed as descriptive variables.

4. Operational Definition of Variables

Nutritional status was determined using BMI-for-age based on the WHO growth reference for children and adolescents aged 5–19 years. BMI was calculated as body weight (kg) divided by height squared (m^2). Subjects were classified as obese when BMI-for-age was $> +2$ standard deviations (SD), while those with BMI-for-age $\leq +2$ SD were classified as non-obese. The variable was measured on a categorical scale.

Glycemic control was assessed using glycated hemoglobin (HbA1c) measured at diagnosis. HbA1c values were expressed as percentages (%). Lower HbA1c values indicated better glycemic control, whereas higher values reflected poorer glycemic control. The variable was analyzed as a continuous scale.

Lipid profile consisted of fasting serum total cholesterol, LDL cholesterol, HDL cholesterol, and triglyceride concentrations measured in mg/dL. These variables were analyzed as continuous variables.

Metabolic syndrome was defined according to the International Diabetes Federation (IDF) criteria for children and adolescents. Patients were classified as having metabolic syndrome when they fulfilled the diagnostic criteria and as not having metabolic syndrome when the criteria were not met. This variable was measured on a dichotomous scale (yes/no).

5. Study Instruments

Data was obtained from patients' medical records at the Pediatric Endocrinology Division of Dr. Moewardi Regional Hospital. The parameters studied were: (1)

age and sex; (2) anthropometry—height, weight, and body mass index (BMI)—interpreted against CDC 2000 growth charts (BMI-for-age ≥ 85 th to < 95 th percentile: overweight; ≥ 95 th percentile: obese), consistent with ADA/ISPAD guideline recommendations and standard clinical practice at our institution; (3) metabolic syndrome defined by the International Diabetes Federation (IDF) paediatric and adolescent criteria (Zimmet et al., 2007): for subjects aged 10–15 years, central obesity (waist circumference ≥ 90 th percentile) as the obligatory criterion plus ≥ 2 of the following: triglycerides ≥ 150 mg/dL, HDL < 40 mg/dL, systolic BP ≥ 130 or diastolic ≥ 85 mmHg, or fasting glucose ≥ 100 mg/dL. For subjects ≥ 16 years, adult IDF criteria were applied using Asian-specific waist circumference thresholds (≥ 90 cm males, ≥ 80 cm females). Since all enrolled patients had confirmed T2DM, the glucose criterion was automatically fulfilled in all subjects; (4) blood pressure classified per the 2017 American Academy of Paediatric Lipid Guidelines; (5) HbA1c recorded at first diagnosis; and (6) a fasting lipid panel comprising total cholesterol, LDL, HDL, and triglycerides, interpreted following the National Heart, Lung, and Blood Institute (NHLBI) Expert Panel for Paediatric Lipid Guidelines.

6. Data Analysis

Participants were summarized descriptively: continuous variables appeared as mean

$\pm$ standard deviation (SD) or as median (interquartile range/IQR) depending on the distribution determined by the Shapiro-Wilk test, and categorical variables as counts and percentages. At the bivariate level, the HbA1c–lipid relationships were tested with Spearman correlation, links between BMI category and metabolic syndrome or dyslipidemia with the Chi-Square or Fisher's Exact Test (the latter when expected cell counts were < 5), and between-group lipid comparisons with the Mann-Whitney U test or independent t-test. Binary logistic regression was then used to identify independent predictors of metabolic syndrome, entering any variable with $p < 0.25$ in bivariate testing into the model. Results were reported as odds ratios (OR) with 95% confidence intervals (CI95%), and a p-value below 0.05 denoted statistical significance.

7. Research Ethic

The study received ethical clearance from the Health Research Ethics Committee of Dr. Moewardi Regional Hospital.

RESULTS

1. Sample Characteristics

Insert your text here. Fifty-one adolescents with T2DM took part. Most were female (92.2%), and the 12–14-year group was the largest (49.0%). At diagnosis the mean HbA1c stood at $12.66 \pm 2.88\%$, signalling severe hyperglycaemia at presentation (Table 1).

Table 1. Demographic and Clinical Characteristics (n=51)

Characteristic	Frequency (n)	Percentage (%)
Sex		
Female	47	92.2
Male	4	7.8
Age Group		
12–14 years	25	49.0
15–16 years	10	19.6
17–18 years	16	31.4
Nutritional Status (BMI)		
Normal	12	23.5

Characteristic	Frequency (n)	Percentage (%)
Overweight	12	23.5
Obese	27	52.9
Metabolic Syndrome		
Present	26	51.0
Absent	25	49.0
Hypertension		
Present	1	2.0
Absent	50	98.0

Regarding the lipid profile of study subjects, considerable dyslipidemia was evident across the cohort (Table 2). High total cholesterol (≥ 200 mg/dL) was found in 35.3% of subjects and hypertriglyceri-

demia in 49.0%. Even so, most participants kept LDL cholesterol within normal limits (60.8%) and HDL cholesterol in the acceptable-to-desirable range (90.2%).

Table 2. Lipid Profile of Adolescents with Type 2 Diabetes Mellitus (n=51)

Lipid Parameter	Frequency (n)	Percentage (%)
Total Cholesterol		
Normal (<170 mg/dL)	31	60.8
Borderline (170–199 mg/dL)	2	3.9
High (≥ 200 mg/dL)	18	35.3
Triglycerides		
Normal (<90 mg/dL)	23	45.1
Borderline (90–129 mg/dL)	3	5.9
High (≥ 130 mg/dL)	25	49.0
LDL Cholesterol		
Normal (<110 mg/dL)	31	60.8
Borderline (110–129 mg/dL)	2	3.9
High (≥ 130 mg/dL)	18	35.3
HDL Cholesterol		
Desirable (>45 mg/dL)	18	35.3
Acceptable (40–45 mg/dL)	28	54.9
Low (<40 mg/dL)	5	9.8

2. Comparison of Lipid Profile by Metabolic Syndrome Status

A comparison of lipid measures in subjects with versus without metabolic syndrome is shown in Table 3. Those with metabolic

syndrome had significantly greater total cholesterol, triglycerides, LDL, and HbA1c, whereas HDL was statistically comparable between the two groups ($p=0.453$).

Table 3. Comparison of Lipid Profile by Metabolic Syndrome Status

Parameter	Metabolic Syndrome (+) (n=26)	Metabolic Syndrome (-) (n=25)	p
	Median (IQR)	Median (IQR)	
Total Cholesterol (mg/dL)	564.00 (167.00–564.00)	150.00 (150.00–156.00)	<0.001 ^a
Triglycerides (mg/dL)	831.00 (255.00–831.00)	50.00 (43.00–70.00)	<0.001 ^a
LDL Cholesterol (mg/dL)	211.00 (118.00–211.00)	100.00 (100.00–100.00)	<0.001 ^a
HDL Cholesterol (mg/dL)	44.00 (44.00–44.00)	44.00 (41.00–52.00)	0.453 ^a
HbA1c (%)	15.30 (14.00–15.30)	11.20 (10.00–13.10)	<0.001 ^a

^aMann-Whitney U test

3. Association of Nutritional Status (BMI) with Metabolic Syndrome and Dyslipidemia

Table 4 displays how BMI category related to metabolic syndrome and to lipid abnormalities. BMI category was significantly linked with metabolic syndrome ($p < 0.001$), high total cholesterol ($p < 0.001$), hyper-

triglyceridemia ($p < 0.001$), and high LDL cholesterol ($p < 0.001$). Metabolic syndrome affected 92.6% of obese subjects—far exceeding the 8.3% seen in the overweight group and 0% in those with normal BMI. By contrast, low HDL cholesterol showed no significant relationship with BMI category ($p = 0.097$).

Table 4. Association of Nutritional Status with Metabolic Syndrome and Dyslipidemia

Variables	Normal (n=12) n (%)	Overweight (n=12) n (%)	Obese (n=27) n (%)	P
Metabolic Syndrome				$<0.001^b$
Present	0 (0.0)	1 (8.3)	25 (92.6)	
Absent	12 (100.0)	11 (91.7)	2 (7.4)	
High Total Cholesterol				$<0.001^b$
Yes	0 (0.0)	0 (0.0)	18 (66.7)	
No	12 (100.0)	12 (100.0)	9 (33.3)	
Hypertriglyceridemia				$<0.001^b$
Yes	1 (8.3)	2 (16.7)	22 (81.5)	
No	11 (91.7)	10 (83.3)	5 (18.5)	
High LDL Cholesterol				$<0.001^b$
Yes	0 (0.0)	0 (0.0)	18 (66.7)	
No	12 (100.0)	12 (100.0)	9 (33.3)	
Low HDL Cholesterol				0.097^b
Yes	3 (25.0)	0 (0.0)	2 (7.4)	
No	9 (75.0)	12 (100.0)	25 (92.6)	

^bChi-Square / Fisher's Exact Test

4. Correlation between HbA1c and Lipid Profile Parameters

Spearman analysis revealed significant correlations between HbA1c and each lipid parameter (Table 5). HbA1c was moderately and positively related to total cholesterol

($r = 0.483$; $p < 0.001$) and weakly inversely related to HDL cholesterol ($r = -0.290$; $p = 0.039$), while showing strong positive correlations with triglycerides ($r = 0.752$; $p < 0.001$) and LDL cholesterol ($r = 0.674$; $p < 0.001$).

Table 5. Correlation between HbA1c and Lipid Profile Parameters

Parameter	r	P
Total Cholesterol	0.483	<0.001
Triglycerides	0.752	<0.001
LDL Cholesterol	0.674	<0.001
HDL Cholesterol	-0.290	0.039

5. Comparison of Metabolic Disorder Prevalence across Age Groups

Prevalence of metabolic disturbances by age band appears in Table 6. The 12–14

year group carried the greatest burden of obesity (84.0%), metabolic syndrome (84.0%), high total cholesterol (72.0%), and hypertriglyceridemia (80.0%), each differ-

ing significantly across the age bands ($p < 0.001$). Low HDL cholesterol, however, was similar between groups ($p = 0.242$).

Mean HbA1c likewise varied significantly by age (Kruskal-Wallis, $p < 0.001$), peaking in the 12–14 year group ($14.39 \pm 2.15\%$).

Table 6. Prevalence of Metabolic Disorders by Age Group

Variable	12–14 years (n=25) n (%)	15–16 years (n=10) n (%)	17–18 years (n=16) n (%)	p
Obesity	21 (84.0)	4 (40.0)	2 (12.5)	$< 0.001^b$
Metabolic Syndrome	21 (84.0)	4 (40.0)	1 (6.2)	$< 0.001^b$
High Total Cholesterol	18 (72.0)	0 (0.0)	0 (0.0)	$< 0.001^b$
Hypertriglyceridemia	20 (80.0)	5 (50.0)	0 (0.0)	$< 0.001^b$
Low HDL Cholesterol	4 (16.0)	1 (10.0)	0 (0.0)	0.242 ^b
HbA1c (Mean; SD)	14.39 (2.15)	12.56 (1.34)	10.79 (1.14)	$< 0.001^c$

^bChi-Square / Fisher's Exact Test; ^cKruskal-Wallis test

6. Multivariate Analysis — Predictors of Metabolic Syndrome

Variables reaching $p < 0.25$ in bivariate testing were carried forward into binary logistic regression. Obesity proved to be a significant independent predictor of metabolic syndrome (OR=166.12; CI95%

9.88 to 2792.23; $p < 0.001$), whereas neither above-median HbA1c nor age group retained significance in the final model. Overall, the model classified cases with 98.0% accuracy (sensitivity 96.2%, specificity 100%) and a Nagelkerke $R^2 = 0.868$ (Table 7).

Table 7. Binary Logistic Regression Analysis — Predictors of Metabolic Syndrome

Variables	OR	95% CI	p
Nutritional Status			
Non-obese	Ref.		
Obese	166.12	9.88 to 2792.23	< 0.001
HbA1c			
≤median	Ref.		
>median	2.35	0.09 to 61.89	0.609
Age Group			
12–14 years	Ref.		
15–16 years	0.43	0.02 to 11.22	0.609
17–18 years	0.05	0.001 to 3.25	0.160
Chi square= 5.23			
p=0.733			
Nagelkerke $R^2 = 86.8\%$			

DISCUSSION

This work offers a detailed portrait of the clinical features and inter-variable associations of adolescents with T2DM seen at a tertiary referral centre in Central Java. Its key observations are fourfold: (1) a female preponderance with disease emerging in early adolescence; (2) strikingly high HbA1c at diagnosis; (3) substantial rates of obesity, metabolic syndrome, and dyslipidemia; and (4) meaningful associations linking nutri-

tional status and glycaemic control to the broader metabolic disease burden.

1. Demographic and Clinical Characteristics

The marked female predominance (92.2%) observed in this study, while higher than typically reported, is consistent with established biological mechanisms underpinning sex differences in youth-onset T2DM. During puberty, females experience proportionally greater physiological insulin

resistance compared to males, driven by estrogen-mediated redistribution of adipose tissue (particularly increased visceral fat accumulation), growth hormone-induced transient insulin insensitivity, and the additive metabolic burden imposed by androgen excess in those with underlying polycystic ovary syndrome (PCOS)—a condition that substantially elevates T2DM risk and is common among adolescent females in Asian populations (Nadeau et al., 2016; Tricò et al., 2022). These biological factors create a more pronounced insulin-resistant milieu in genetically susceptible females during the pubertal transition, increasing vulnerability to pancreatic beta-cell decompensation.

Consistent with those pathophysiological mechanisms, epidemiological data demonstrate a robust female predominance in youth-onset T2DM globally; the SEARCH for Diabetes in Youth study reported an incidence approximately twice as high in adolescent girls compared to boys (Lawrence et al., 2021). In the SEARCH for Diabetes in Youth study, T2DM incidence among adolescent girls was roughly double that of boys (Lawrence et al., 2021). Contributing factors include the physiological insulin resistance of female puberty, sex-related differences in fat distribution, and ovarian androgen activity associated with polycystic ovary syndrome (PCOS) (Nadeau et al., 2016; Tricò et al., 2022). Consistent with this, about two-thirds of TODAY participants were female, reflecting a pattern repeated across populations (TODAY Study Group, 2021).

That the 12–14-year group predominated coincides with the height of pubertal development. Rising growth hormone and sex-steroid levels during puberty lower insulin sensitivity by some 30–50%, making this stage especially

conducive to T2DM expression in adolescents who are genetically predisposed and obese (Kelsey and Zeitler, 2016; Goran and Gower, 2001). Such findings lend support to the ADA and ISPAD advice to begin T2DM screening in overweight or obese children from age 10 or once puberty sets in (Shah et al., 2022; American Diabetes Association, 2022).

At 12.66 (SD= 2.88%), the mean diagnostic HbA1c far exceeds figures from Western series (7.9% in TODAY and 8.0% in RISE) (TODAY Study Group, 2021; RISE Consortium, 2018). Such pronounced elevation points to diagnosis being delayed at the primary-care level. Pulungan et al. similarly found that the majority of Indonesian children and adolescents with diabetes present already in severe hyperglycaemia (Pulungan et al., 2020). The clinical consequences of this delay are considerable, since it accelerates beta-cell deterioration and raises the risk of early microvascular complications (Nadeau et al., 2016).

2. Association of Metabolic Syndrome with Lipid Profile

The observation that lipid profiles were significantly poorer in the metabolic syndrome group (total cholesterol, triglycerides, LDL; all $p < 0.001$) fits the recognised pathophysiology of dyslipidemia in insulin-resistant states. Insulin resistance accelerates lipolysis in adipose tissue, drives hepatic very-low-density lipoprotein (VLDL) output, and curtails lipoprotein lipase activity, together yielding higher triglycerides, lower HDL, and the generation of atherogenic small dense LDL particles (Bardini et al., 2019). In a meta-analysis, Reisinger et al. confirmed that adolescent metabolic syndrome is reliably accompanied by atherogenic dyslipidemia and a greater risk of cardiovascular events in early adulthood (Reisinger et al., 2021).

The lack of a meaningful HDL difference ($p=0.453$) may stem from the narrow HDL variability in this cohort and from unmeasured genetic and dietary influences.

3. Association of Nutritional Status with Metabolic Burden

The strong link between obesity and both metabolic syndrome and the individual dyslipidemia components reaffirms that adiposity lies at the core of cardiometabolic derangement in adolescent T2DM. Metabolic syndrome was present in 92.6% of obese subjects, versus merely 8.3% of overweight subjects and none of those with normal BMI. That 23.5% of participants had a normal BMI accord with Asian data describing a "metabolically obese normal weight" phenotype, whereby insulin resistance and T2DM can arise at lower BMI because visceral fat is proportionally greater (Yoon et al., 2006; Misra et al., 2018).

4. Correlation between HbA1c and Lipid Profile

The positive associations between HbA1c and atherogenic lipids hold notable clinical relevance. Sustained hyperglycaemia fosters non-enzymatic glycation of lipoproteins, hampers LDL clearance through its receptor, and promotes hepatic triglyceride synthesis (Bardini et al., 2019). These results echo Bjornstad et al., who showed that inadequate glycaemic control in adolescents with T2DM tracks closely with early arterial stiffening and endothelial dysfunction (Bjornstad et al., 2021). Clinically, these findings suggest that improved glycemic control may be associated with a more favorable lipid profile in adolescents with T2DM, although the cross-sectional design of this study does not permit establishment of a causal or directional relationship.

Prospective and interventional studies are needed to determine whether pharma-

cological or lifestyle-mediated reductions in HbA1c translate into corresponding improvements in atherogenic lipid parameters in this population. The tightest correlations linked HbA1c with triglycerides ($r=0.75$) and LDL cholesterol ($r=0.67$), while its relationship with HDL was only weakly inverse ($r=-0.29$; $p=0.039$).

5. Predictors of Metabolic Syndrome

Logistic regression identified obesity as the most powerful independent predictor of metabolic syndrome, implying that weight control deserves the highest therapeutic priority. The significant positive ties between HbA1c and atherogenic lipid markers further argue for vigorous glycaemic management, this finding should be interpreted with caution. The IDF pediatric metabolic syndrome criteria require waist circumference ≥ 90 th percentile as an obligatory defining criterion, which shares conceptual overlap with BMI-based general obesity. This definitional proximity may partially inflate the observed odds ratio, and the true independent effect of general adiposity on the composite metabolic syndrome phenotype may be lower than suggested.

Furthermore, the wide confidence interval reflects limited statistical precision due to the small sample size. Future studies with larger samples and concurrent measurement of waist circumference are needed to more precisely estimate this association. The practical value of these findings is immediate: recognising adolescents who carry the principal risk factors—obesity and/or strikingly high HbA1c—at diagnosis enables early risk stratification and more intensive intervention, encompassing intensive lifestyle change, optimisation of glucose-lowering pharmacotherapy, and, in keeping with the ISPAD 2022 guidelines, consideration of lipid-lowering drugs (Shah et al., 2022).

Several limitations warrant mention. The cross-sectional structure precludes any inference of causality, and the modest sample (n=51) limits the statistical power of the multivariate model. Because all data originated from a single centre, generalisation should be approached cautiously. Variables such as Tanner pubertal stage, family history of type 2 diabetes, and direct measures of insulin resistance (HOMA-IR, C-peptide) were not systematically assessed. Larger, prospective, multicentre studies are needed to corroborate these observations and to clarify the temporal relationship between early clinical features and long-term outcomes in the Indonesian setting.

In summary, obesity (52.9%), metabolic syndrome (51.0%), and dyslipidaemia were highly common among the adolescents with type 2 diabetes attending Dr. Moewardi Regional Hospital. Subjects with metabolic syndrome showed substantially higher total cholesterol, triglycerides, LDL cholesterol, and HbA1c ($p<0.001$). Obesity was the strongest independent predictor of metabolic syndrome, and HbA1c correlated strongly and positively with both LDL cholesterol and triglycerides. The heaviest metabolic burden fell on the 12–14 year age group. These findings underscore associations between early-onset obesity and dysglycemia with cardiometabolic disturbances in Indonesian adolescents with T2DM, and support the importance of early lipid screening and integrated weight management from the time of diagnosis.

AUTHOR CONTRIBUTION

Hanum Ferdian raised the initial research question, managed data collection, performed statistical analysis, and prepared tables and figures. Annang Giri Moelyo refined the research questions, planned the study design, interpreted the results, and

wrote up the manuscript. Both authors read and approved the final version of the manuscript for publication.

CONFLICT OF INTEREST

The authors declare that the study was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

FUNDING AND SPONSORSHIP

This work was supported by: Nil.

ACKNOWLEDGEMENT

The authors would like to express their gratitude to the medical team at the Pediatric Endocrinology Division of Dr. Moewardi Regional Hospital, Surakarta, for their support throughout the data collection process.

REFERENCE

- American Diabetes Association Professional Practice Committee (2022). Children and adolescents: Standards of Medical Care in Diabetes—2022. *Diabetes Care*. 45(1): S208–S231. <https://doi.org/10.2337/dc22-s014>.
- Bardini G, Rotella CM, Giannini S (2019). Dyslipidemia and diabetes: reciprocal impact of impaired lipid metabolism and Beta-cell dysfunction on micro- and macrovascular complications. *Rev Diabet Stud*. 9(2–3): 82–93. <https://doi.org/10.1900/RDS.2012.9.82>.
- Bjornstad P, Drews KL, Caprio S, Gubitosi-Klug R, Nathan DM, Tesfaldet B, et al. (2021). Long-term complications in youth-onset type 2 diabetes. *N Engl J Med*. 385(5): 416–426. <https://doi.org/10.1056/nejmoa2100165>.
- Expert Panel on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adoles-

- cents; National Heart, Lung, and Blood Institute (2011). Expert panel on integrated guidelines for cardiovascular health and risk reduction in children and adolescents: summary report. *Pediatrics*. 128(5): S213–S256. <https://doi.org/10.1542/peds.2009-2107C>.
- Flynn JT, Kaelber DC, Baker-Smith CM, Blowey D, Carroll AE, Daniels SR, et al. (2017). Clinical practice guideline for screening and management of high blood pressure in children and adolescents. *Pediatrics*. 140(3): e201-71904. <https://doi.org/10.1542/peds.-2017-1904>.
- Goran MI, Gower BA (2001). Longitudinal study on pubertal insulin resistance. *Diabetes*. 50(11): 2444–2450. <https://doi.org/10.2337/diabetes.50.11.2444>.
- Kelsey MM, Zeitler PS (2016). Insulin resistance of puberty. *Curr Diab Rep*. 16(7): 64. <https://doi.org/10.1007/s11892-016-0751-5>.
- Lawrence JM, Divers J, Isom S, Saydah S, Imperatore G, Pihoker C, et al. (2021). Trends in prevalence of type 1 and type 2 diabetes in children and adolescents in the US, 2001–2017. *JAMA*. 326(8): 717–727. <https://doi.org/10.1001/jama.2021.11165>.
- Magliano DJ, Boyko EJ (2021). *IDF Diabetes Atlas*. 10th ed. Brussels: International Diabetes Federation. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK581934/>.
- Misra A, Soares MJ, Mohan V, Anoop S, Abhishek V, Vaidya R, et al. (2018). Body fat, metabolic syndrome and hyperglycemia in South Asians. *J Diabetes Complications*. 32(11): 1068–1075. <https://doi.org/10.1016/j.jdiacomp.2018.08.001>.
- Nadeau KJ, Anderson BJ, Berg EG, Chiang JL, Chou H, Copeland KC, et al. (2016). Youth-onset type 2 diabetes consensus report: current status, challenges, and priorities. *Diabetes Care*. 39(9): 1635–1642. <https://doi.org/10.2337/dc16-1066>.
- Ministry of Health of the Republic of Indonesia. (2019). National Report of the 2018 Basic Health Research (Riskesdas 2018). Jakarta: National Institute of Health Research and Development.
- Pulungan AB, Annisa D, Imada S (2020). Diabetes mellitus type 1 and type 2 in children and adolescents in Indonesia: a systematic review. *Paediatr Indones*. 60(3): 117–127. <https://doi.org/10.1297/cpe.30.11>.
- Reisinger C, Nkeh-Chungag BN, Fredriksen PM, Goswami N (2021). The prevalence of pediatric metabolic syndrome—a critical look on the discrepancies between definitions and its clinical importance. *Int J Obes (Lond)*. 45(1): 12–24. <https://doi.org/10.1038/s41366-020-00713-1>.
- RISE Consortium (2018). Metabolic contrasts between youth and adults with impaired glucose tolerance or recently diagnosed type 2 diabetes. *Diabetes Care*. 41(8): 1696–1706. <https://doi.org/10.2337/dc18-0244>.
- Shah AS, Zeitler PS, Wong J, Pena AS, Wicklow B, Arslanian S, et al. (2022). ISPAD Clinical Practice Consensus Guidelines 2022: type 2 diabetes in children and adolescents. *Pediatr Diabetes*. 23(7): 872–902. <https://doi.org/10.1111/pedi.13409>.
- TODAY Study Group (2021). Long-term complications in youth-onset type 2 diabetes. *N Engl J Med*. 385(5): 416–426. <https://doi.org/10.1056/NEJMoa2100165>.

- Tricò D, Chiriaco M, Nouws J, Vash-Margita A, Kursawe R, Tarabra E, et al. (2022). Alterations in adipose tissue distribution, cell morphology, and function mark primary insulin hypersecretion in youth with obesity. *Diabetes*. 71(7): 1392–1404. <https://doi.org/10.2337/db23-0450>.
- Wu H, Patterson CC, Zhang X, Ghani RBA, Magliano DJ, Boyko EJ, et al. (2022). Worldwide estimates of incidence of type 2 diabetes in children and adolescents in 2021. *Diabetes Res Clin Pract*. 185: 109785. <https://doi.org/10.1016/j.diabres.2022.109785>.
- Yoon KH, Lee JH, Kim JW, Cho JH, Choi YH, Ko SH, et al. (2006). Epidemic obesity and type 2 diabetes in Asia. *Lancet*. 368(9548): 1681–1688. [https://doi.org/10.1016/s0140-6736-\(06\)69703-1](https://doi.org/10.1016/s0140-6736-(06)69703-1).
- Zimmet P, Alberti KGMM, Kaufman F, Tajima N, Silink M, Arslanian S, et al. (2007). The metabolic syndrome in children and adolescents — an IDF consensus report. *Pediatr Diabetes*. 8(5): 299–306. <https://doi.org/10.1111/j.1399-5448.2007.00271.x>.