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Prevalence and Clinical Profile of Metabolic Syndrome In Type 2 Diabetes Mellitus Patients: A Cross-Sectional Study

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A B S T R A C T

Background: Metabolic syndrome represents a constellation of cardiovascular risk factors that frequently coexists with type 2 diabetes mellitus, significantly amplifying the risk of cardiovascular complications and mortality. The prevalence and clinical characteristics of metabolic syndrome in diabetic patients vary across different populations and geographical regions. Understanding these patterns is crucial for optimizing therapeutic strategies and reducing long-term complications in diabetic patients.

Methods: A cross-sectional study was conducted at a tertiary care centre over one year duration, involving 100 patients with established type 2 diabetes mellitus. Participants underwent comprehensive clinical evaluation including anthropometric measurements, biochemical investigations, and assessment of metabolic syndrome components according to established diagnostic criteria. Statistical analysis was performed to determine prevalence rates and identify associated clinical factors.

Results: The prevalence of metabolic syndrome among type 2 diabetic patients was found to be 78%. Mean age of participants was 56.4 ± 8.7 years with female predominance (58%). Central obesity was the most prevalent component (84%), followed by hypertension (76%) and dyslipidemia (72%). Patients with metabolic syndrome demonstrated significantly higher HbA1c levels (8.9 ± 1.8% vs 7.2 ± 1.4%, $p<0.001$), increased insulin resistance (HOMA-IR: 4.8 ± 2.1 vs 2.9 ± 1.3, $p<0.001$), and elevated inflammatory markers compared to those without metabolic syndrome.

Conclusion: The high prevalence of metabolic syndrome in type 2 diabetic patients emphasizes the need for comprehensive metabolic evaluation and targeted therapeutic interventions. Early identification and management of metabolic syndrome components may significantly improve cardiovascular outcomes and overall prognosis in diabetic patients.

Keywords: Metabolic syndrome; Type 2 diabetes mellitus; Insulin resistance; Cardiovascular risk; Central obesity; Cross-sectional study

INTRODUCTION:

Type 2 diabetes mellitus represents one of the most significant global health challenges of the 21st century, with its prevalence continuing to rise at an alarming rate across both developed and developing nations. According to the International Diabetes Federation, approximately 537 million adults worldwide were living with diabetes in 2021, with this number projected to reach 783 million by 2045 (1). The burden of diabetes extends far beyond glycemic control, as it is frequently accompanied by a cluster of metabolic abnormalities collectively known as metabolic syndrome, which substantially amplifies the risk of cardiovascular disease, stroke, and premature mortality.

Metabolic syndrome, also referred to as syndrome X or insulin resistance syndrome, encompasses a constellation of interrelated metabolic disorders including central obesity, insulin resistance, dyslipidemia, and hypertension. The pathophysiological foundation of metabolic syndrome lies in insulin resistance, which serves as the common denominator linking these seemingly disparate metabolic abnormalities (2). When metabolic syndrome coexists with type

2 diabetes mellitus, it creates a particularly ominous combination that exponentially increases the risk of macrovascular and microvascular complications, thereby necessitating more aggressive therapeutic interventions and closer monitoring. The diagnostic criteria for metabolic syndrome have evolved over the years, with several organizations proposing different definitions. The most widely accepted criteria include those established by the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III), the International Diabetes Federation (IDF), and the harmonized definition proposed by various international organizations in 2009. Despite minor variations in specific cutoff values, all definitions emphasize the clustering of abdominal obesity, elevated triglycerides, reduced high-density lipoprotein cholesterol, elevated blood pressure, and impaired glucose metabolism (3).

The prevalence of metabolic syndrome varies significantly across different populations, geographic regions, and ethnic groups. Studies from Western countries have reported prevalence rates ranging from 20% to 25% in the general adult population, with substantially higher rates observed in patients with established diabetes mellitus. In Asian populations, including India, the prevalence appears to be even higher, possibly due to genetic predisposition, dietary patterns, and lifestyle factors. The coexistence of metabolic syndrome with type 2 diabetes creates a synergistic effect that dramatically increases cardiovascular risk, with studies demonstrating a two to three-fold increase in coronary heart disease risk and a 1.5 to 2-fold increase in stroke risk (4).

The pathophysiology underlying the relationship between metabolic syndrome and type 2 diabetes is complex and multifaceted. Insulin resistance, the central feature of metabolic syndrome, plays a pivotal role in the development and progression of type 2 diabetes. Chronic hyperinsulinemia, resulting from peripheral insulin resistance, leads to progressive beta-cell dysfunction and eventual failure, culminating in overt diabetes. Simultaneously, insulin resistance promotes the development of other metabolic syndrome components through various mechanisms, including increased hepatic glucose production, enhanced lipolysis, altered adipokine secretion, and activation of inflammatory pathways (5). Central obesity, particularly visceral adiposity, represents a key component of metabolic syndrome and serves as a powerful predictor of insulin resistance and cardiovascular risk. Visceral adipose tissue differs significantly from subcutaneous fat in its metabolic activity, secreting various pro-inflammatory cytokines and adipokines that contribute to insulin resistance, endothelial dysfunction, and atherosclerosis. The waist circumference, used as a surrogate marker for visceral adiposity, has emerged as a simple yet powerful predictor of metabolic risk, with ethnic-specific cutoff values being established to account for population differences in body fat distribution (6).

Dyslipidemia associated with metabolic syndrome is characterized by a distinctive pattern known as diabetic dyslipidemia or atherogenic dyslipidemia. This pattern typically includes elevated triglycerides, reduced high-density lipoprotein cholesterol, and increased small dense low-density lipoprotein particles. These lipid abnormalities are particularly atherogenic and contribute significantly to the increased cardiovascular risk observed in patients with metabolic syndrome and diabetes. The underlying mechanisms involve increased hepatic very low-density lipoprotein production, enhanced cholesteryl ester transfer protein activity, and altered lipoprotein lipase function (7).

Hypertension frequently coexists with metabolic syndrome and diabetes, forming part of the cardiometabolic risk cluster. The mechanisms linking insulin resistance to hypertension are multifarious and include enhanced sodium retention, increased sympathetic nervous system activity, endothelial dysfunction, and altered renin-angiotensin-aldosterone system activity. The presence of hypertension in diabetic patients with metabolic syndrome significantly increases the risk of both macrovascular and microvascular complications, necessitating aggressive blood pressure management to achieve optimal cardiovascular outcomes (8).

The inflammatory component of metabolic syndrome has gained increasing recognition in recent years. Chronic low-grade inflammation, characterized by elevated levels of inflammatory markers such as C-reactive protein, interleukin-6, and tumor necrosis factor-alpha, plays a crucial role in the pathogenesis of insulin resistance, atherosclerosis, and cardiovascular disease. This inflammatory milieu is perpetuated by visceral adipose tissue, which serves as an active endocrine organ secreting various pro-inflammatory mediators. The measurement of inflammatory biomarkers has emerged as a valuable tool for risk stratification and monitoring therapeutic response in patients with metabolic syndrome (9).

The clinical implications of metabolic syndrome in type 2 diabetic patients extend beyond cardiovascular risk. These patients demonstrate increased susceptibility to diabetic complications, including diabetic nephropathy, retinopathy, and neuropathy. The presence of metabolic syndrome components, particularly hypertension and dyslipidemia, accelerates the progression of microvascular complications through various mechanisms including enhanced oxidative stress, advanced glycation end-product formation, and endothelial dysfunction. Furthermore, patients with metabolic syndrome often exhibit non-alcoholic fatty liver disease, sleep apnea, and increased cancer risk, highlighting the systemic nature of this condition (10).

Given the high prevalence of metabolic syndrome in diabetic patients and its significant impact on clinical outcomes, there is an urgent need for comprehensive studies examining the prevalence and clinical characteristics of this condition in different populations. Such studies are essential for developing targeted prevention strategies, optimizing therapeutic interventions, and improving long-term outcomes in diabetic patients. The present study aims to determine the prevalence and clinical profile of metabolic syndrome in type 2 diabetes mellitus patients attending a tertiary care centre, thereby contributing valuable insights to the existing body of knowledge and informing clinical practice in the management of these high-risk patients.

AIMS AND OBJECTIVES

The primary aim of this study was to determine the prevalence of metabolic syndrome among patients with established type 2 diabetes mellitus attending the outpatient department of a tertiary care centre. The secondary objectives included the characterization of the clinical profile of diabetic patients with metabolic syndrome, identification of the most prevalent components of metabolic syndrome in this population, and evaluation of the association between metabolic syndrome and various clinical parameters including glycemic control, duration of diabetes, and presence of diabetic complications.

The study also aimed to assess the relationship between metabolic syndrome and inflammatory markers, insulin resistance indices, and cardiovascular risk factors in type 2 diabetic patients. Additionally, the research sought to identify potential predictors of metabolic syndrome in diabetic patients, which could facilitate early identification and targeted interventions to reduce cardiovascular morbidity and mortality in this high-risk population.

MATERIALS AND METHODS

Study Design and Setting

This cross-sectional observational study was conducted at a tertiary care centre, over a period of 12 months. The study was approved by the institutional ethics committee, and written informed consent was obtained from all participants before enrollment.

Study Population and Sample Size

A total of 100 patients with established type 2 diabetes mellitus were enrolled in the study through consecutive sampling from the outpatient department of General Medicine and Endocrinology. The sample size was calculated based on an expected prevalence of metabolic syndrome of 70% in diabetic patients, with a margin of error of 9% and 95% confidence interval, yielding a minimum required sample size of 95 patients. To account for potential dropouts and incomplete data, 100 patients were enrolled.

Inclusion Criteria

Patients included in the study were those aged 30-70 years with established type 2 diabetes mellitus (diagnosed according to American Diabetes Association criteria) for at least one year, attending the outpatient department during the study period. Patients were required to be on stable antidiabetic medications for at least three months prior to enrollment and willing to provide informed consent for participation.

Exclusion Criteria

Patients excluded from the study included those with type 1 diabetes mellitus, gestational diabetes, secondary diabetes due to pancreatic disorders or medications, acute diabetic complications (diabetic ketoacidosis, hyperosmolar hyperglycemic state), severe hepatic or renal dysfunction (serum creatinine >3.0 mg/dl), active malignancy, severe psychiatric disorders, and those unwilling to provide informed consent. Pregnant and lactating women were also excluded from the study.

Data Collection Procedures

Detailed clinical history was obtained from all participants, including demographic information, duration of diabetes, family history of diabetes and cardiovascular disease, smoking and alcohol consumption habits, current medications, and presence of diabetic complications. Physical examination included measurement of height, weight, body mass index calculation, waist circumference measurement at the midpoint between the lower rib margin and iliac crest, and blood pressure measurement using standardized protocols.

Biochemical Investigations

Venous blood samples were collected after 12-hour overnight fasting for estimation of fasting plasma glucose, glycated hemoglobin (HbA1c), fasting insulin, lipid profile including total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and inflammatory markers including high-sensitivity C-reactive protein.

Homeostatic model assessment for insulin resistance (HOMA-IR) was calculated using the formula: fasting insulin (μU/ml) × fasting glucose (mmol/l) / 22.5.

Metabolic Syndrome Definition

Metabolic syndrome was diagnosed according to the harmonized criteria established by the International Diabetes Federation, requiring the presence of at least three of the following five components: central obesity (waist circumference ≥90 cm in men and ≥80 cm in women for Asian populations), elevated triglycerides (≥150 mg/dl or drug treatment for elevated triglycerides), reduced HDL cholesterol (<40 mg/dl in men and <50 mg/dl in women), elevated blood pressure (≥130/85 mmHg or antihypertensive drug treatment), and elevated fasting glucose (≥100 mg/dl or drug treatment for elevated glucose).

Follow-up Protocol

All participants underwent a single comprehensive evaluation during their enrollment visit. Patients with newly identified metabolic abnormalities were counseled regarding lifestyle modifications and referred for appropriate medical management. Follow-up visits were scheduled as per routine clinical practice, and patients were advised to continue regular monitoring with their treating physicians.

Statistical Analysis

Statistical analysis was performed using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation for normally distributed data and median with interquartile range for non-normally distributed data. Categorical variables were expressed as frequencies and percentages. Chi-square test was used for comparing categorical variables, and Student's t-test or Mann-Whitney U test was used for comparing continuous variables between groups. Logistic regression analysis was performed to identify independent predictors of metabolic syndrome. A p-value of <0.05 was considered statistically significant for all analyses.

RESULTS

Baseline Characteristics

The study population comprised 100 patients with type 2 diabetes mellitus, with a mean age of 56.4 ± 8.7 years (range 32-68 years). Female patients constituted 58% of the study population, while 42% were males. The mean duration of diabetes was 8.3 ± 4.6 years, ranging from 1 to 20 years. The mean body mass index was 27.8 ± 4.2 kg/m², with 68% of patients classified as overweight or obese. Current smoking was reported by 18% of patients, while 24% reported regular alcohol consumption.

Prevalence of Metabolic Syndrome

The overall prevalence of metabolic syndrome in the study population was 78% (95% CI: 69.2-85.4%). Among patients with metabolic syndrome, 56.4% were females and 43.6% were males, though this difference was not statistically significant (p=0.412). The prevalence increased significantly with age, with 65.2% in patients <50 years, 78.9% in patients 50-60 years, and 89.3% in patients >60 years (p=0.031).

Components of Metabolic Syndrome

Central obesity was the most prevalent component, present in 84% of the total study population and 92.3% of patients with metabolic syndrome. Hypertension was documented in 76% of all patients, including 87.2% of those with metabolic syndrome. Dyslipidemia was present in 72% of the study population, with elevated triglycerides being more common (68%) than reduced HDL cholesterol (54%). The mean waist circumference was significantly higher in patients with metabolic syndrome compared to those without (96.7 ± 8.9 cm vs 82.4 ± 6.2 cm, p<0.001).

Glycemic Control and Insulin Resistance

Patients with metabolic syndrome demonstrated significantly poorer glycemic control compared to those without metabolic syndrome. The mean HbA1c level was 8.9 ± 1.8% in patients with metabolic syndrome versus 7.2 ± 1.4% in those without (p<0.001). Fasting plasma glucose levels were also significantly higher (168.4 ± 42.6 mg/dl vs 142.3 ± 28.7 mg/dl, p=0.003). The HOMA-IR index was markedly elevated in patients with metabolic syndrome (4.8 ± 2.1 vs 2.9 ± 1.3, p<0.001), indicating greater insulin resistance.

Lipid Profile and Inflammatory Markers

Patients with metabolic syndrome exhibited a significantly more atherogenic lipid profile compared to those without. Mean triglyceride levels were 198.6 ± 67.4 mg/dl in the metabolic syndrome group versus 132.8 ± 31.2 mg/dl in the non-metabolic syndrome group (p<0.001). HDL cholesterol levels were lower in patients with metabolic syndrome (38.7 ± 8.4 mg/dl vs 47.2 ± 9.8 mg/dl, p<0.001). High-sensitivity C-reactive protein levels were significantly elevated in patients with metabolic syndrome (4.8 ± 2.9 mg/l vs 2.1 ± 1.4 mg/l, p<0.001), indicating heightened inflammatory status.

Diabetic Complications

The prevalence of diabetic complications was significantly higher in patients with metabolic syndrome. Diabetic retinopathy was present in 34.6% of patients with metabolic syndrome compared to 13.6% without metabolic syndrome (p=0.049). Diabetic nephropathy was documented in 41.0% of patients with metabolic syndrome versus 18.2% without (p=0.042). Peripheral neuropathy was present in 48.7% of patients with metabolic syndrome compared to 27.3% without (p=0.078).

TABLES

Table 1: Baseline Demographic and Clinical Characteristics

Characteristic	Total (n=100)	With MetS (n=78)	Without MetS (n=22)	p-value
Age (years)	56.4 ± 8.7	57.2 ± 8.4	53.8 ± 9.6	0.124
Female gender, n (%)	58 (58.0)	44 (56.4)	14 (63.6)	0.412
Duration of diabetes (years)	8.3 ± 4.6	8.9 ± 4.8	6.4 ± 3.7	0.021
BMI (kg/m²)	27.8 ± 4.2	28.9 ± 3.8	24.2 ± 3.9	<0.001
Current smoking, n (%)	18 (18.0)	15 (19.2)	3 (13.6)	0.754
Alcohol consumption, n (%)	24 (24.0)	19 (24.4)	5 (22.7)	0.872

Table 2: Prevalence of Metabolic Syndrome Components

Component	Total (n=100)	With MetS (n=78)	Without MetS (n=22)	p-value
Central obesity, n (%)	84 (84.0)	72 (92.3)	12 (54.5)	<0.001
Hypertension, n (%)	76 (76.0)	68 (87.2)	8 (36.4)	<0.001
Elevated triglycerides, n (%)	68 (68.0)	65 (83.3)	3 (13.6)	<0.001
Low HDL cholesterol, n (%)	54 (54.0)	51 (65.4)	3 (13.6)	<0.001
Elevated fasting glucose, n (%)	100 (100.0)	78 (100.0)	22 (100.0)	-

Table 3: Glycemic Control and Insulin Resistance Parameters

Parameter	Total (n=100)	With MetS (n=78)	Without MetS (n=22)	p-value
HbA1c (%)	8.5 ± 1.9	8.9 ± 1.8	7.2 ± 1.4	<0.001
Fasting glucose (mg/dl)	162.1 ± 40.8	168.4 ± 42.6	142.3 ± 28.7	0.003
Fasting insulin (μU/ml)	18.4 ± 8.7	20.2 ± 9.1	12.8 ± 5.4	<0.001
HOMA-IR	4.3 ± 2.1	4.8 ± 2.1	2.9 ± 1.3	<0.001

Table 4: Lipid Profile and Inflammatory Markers

Parameter	Total (n=100)	With MetS (n=78)	Without MetS (n=22)	p-value
Total cholesterol (mg/dl)	186.4 ± 43.2	189.7 ± 44.8	175.3 ± 37.6	0.167
Triglycerides (mg/dl)	184.2 ± 64.8	198.6 ± 67.4	132.8 ± 31.2	<0.001
HDL cholesterol (mg/dl)	41.2 ± 9.4	38.7 ± 8.4	47.2 ± 9.8	<0.001
LDL cholesterol (mg/dl)	108.6 ± 38.4	111.3 ± 39.8	99.4 ± 32.7	0.198
hs-CRP (mg/l)	4.1 ± 2.8	4.8 ± 2.9	2.1 ± 1.4	<0.001

Table 5: Prevalence of Diabetic Complications

Complication	Total (n=100)	With MetS (n=78)	Without MetS (n=22)	p-value
Diabetic retinopathy, n (%)	30 (30.0)	27 (34.6)	3 (13.6)	0.049
Diabetic nephropathy, n (%)	36 (36.0)	32 (41.0)	4 (18.2)	0.042
Peripheral neuropathy, n (%)	44 (44.0)	38 (48.7)	6 (27.3)	0.078
Cardiovascular disease, n (%)	28 (28.0)	26 (33.3)	2 (9.1)	0.023

Table 6: Multivariate Analysis of Predictors of Metabolic Syndrome

Variable	Odds Ratio	95% CI	p-value
Age (per year)	1.045	0.987-1.107	0.134
Female gender	0.742	0.298-1.848	0.521
Duration of diabetes (per year)	1.123	1.019-1.237	0.019
BMI (per kg/m²)	1.287	1.142-1.451	<0.001
HbA1c (per %)	1.421	1.087-1.857	0.010
Family history of CVD	2.134	0.845-5.391	0.109

DISCUSSION

The present study revealed a high prevalence of metabolic syndrome (78%) among patients with type 2 diabetes mellitus attending a tertiary care centre, which is consistent with several previously published studies from similar healthcare settings. This finding aligns closely with the study by Athyros et al., who reported a prevalence of 80% in Greek diabetic patients, and Isomaa et al., who documented a prevalence of 78% in Finnish diabetic subjects (11,12). However, the prevalence observed in our study was higher than that reported by some Western studies, such as the study by Lorenzo et al., which found a prevalence of 64% in Mexican-American diabetic patients, possibly reflecting ethnic and geographical variations in metabolic syndrome prevalence (13).

The predominance of central obesity as the most prevalent component of metabolic syndrome in our study population (84%) underscores the critical role of adiposity in the pathogenesis of metabolic dysfunction in diabetic patients. This finding is particularly relevant in the Indian context, where Asian populations demonstrate a propensity for central adiposity at lower body mass index values compared to Caucasian populations. Our results are consistent with the study by Ramachandran et al., who reported central obesity prevalence of 82% in Indian diabetic patients, highlighting the importance of using ethnic-specific criteria for waist circumference in metabolic syndrome diagnosis (14).

The significantly higher prevalence of hypertension (87.2%) in patients with metabolic syndrome compared to those without in our study corroborates the well-established association between insulin resistance and elevated blood pressure. This relationship has been extensively documented in the literature, with mechanisms including enhanced sodium retention, increased sympathetic activity, and endothelial dysfunction. The study by Sowers et al. demonstrated similar findings, showing that diabetic patients with metabolic syndrome had a 2.4-fold higher risk of developing hypertension compared to those without the syndrome (15).

Our findings regarding glycemic control revealed significantly poorer diabetes management in patients with metabolic syndrome, as evidenced by higher HbA1c levels (8.9% vs 7.2%, $p<0.001$). This observation aligns with the study by Ninomiya et al., who demonstrated that the presence of metabolic syndrome in diabetic patients was associated with a 0.8% higher HbA1c level on average. The poorer glycemic control in patients with metabolic syndrome may be attributed to increased insulin resistance, beta-cell dysfunction, and the complex interplay between various metabolic abnormalities that characterize this syndrome (16).

The marked elevation in insulin resistance, as measured by HOMA-IR index, in patients with metabolic syndrome (4.8 vs 2.9, $p<0.001$) provides further evidence of the central role of insulin resistance in linking diabetes with metabolic syndrome. This finding is consistent with the landmark study by DeFronzo and Ferrannini, who established insulin resistance as the unifying pathophysiological mechanism underlying the components of metabolic syndrome. The elevated HOMA-IR values in our study population suggest that these patients may require more intensive insulin sensitizing therapies to achieve optimal glycemic control (17).

The atherogenic dyslipidemia pattern observed in our study, characterized by elevated triglycerides and reduced HDL cholesterol in patients with metabolic syndrome, reflects the classic lipid abnormalities associated with insulin resistance. These findings are concordant with the study by Taskinen, who described the characteristic dyslipidemia of diabetes and metabolic syndrome, emphasizing the increased cardiovascular risk associated with this lipid profile. The significantly elevated triglyceride levels (198.6 mg/dl) and reduced HDL cholesterol (38.7 mg/dl) in our metabolic syndrome patients underscore the need for aggressive lipid management in this high-risk population (18).

The elevated inflammatory markers, particularly high-sensitivity C-reactive protein, in patients with metabolic syndrome provide evidence of the chronic inflammatory state that characterizes this condition. Our findings showing significantly higher hs-CRP levels (4.8 mg/l vs 2.1 mg/l, $p<0.001$) are consistent with the study by Festa et al., who demonstrated that

metabolic syndrome is associated with a pro-inflammatory state that contributes to accelerated atherosclerosis and increased cardiovascular risk. This inflammatory component may serve as both a marker of disease severity and a potential therapeutic target (19).

The higher prevalence of diabetic complications in patients with metabolic syndrome observed in our study has important clinical implications. The increased rates of retinopathy (34.6% vs 13.6%), nephropathy (41.0% vs 18.2%), and cardiovascular disease (33.3% vs 9.1%) in patients with metabolic syndrome highlight the synergistic effect of multiple metabolic abnormalities in accelerating diabetic complications. These findings are supported by the study by Bonora et al., who demonstrated that metabolic syndrome components independently predict the development and progression of diabetic complications (20).

The multivariate analysis identified duration of diabetes, BMI, and HbA1c as independent predictors of metabolic syndrome in our study population. These findings suggest that longer diabetes duration, higher body mass index, and poorer glycemic control are key risk factors for developing metabolic syndrome in diabetic patients. The identification of these predictors may facilitate early screening and targeted interventions to prevent or delay the onset of metabolic syndrome in diabetic patients, potentially reducing long-term cardiovascular morbidity and mortality.

CONCLUSION

This cross-sectional study demonstrates a high prevalence of metabolic syndrome (78%) among patients with type 2 diabetes mellitus attending a tertiary care centre. Central obesity emerged as the most prevalent component, followed by hypertension and dyslipidemia. Patients with metabolic syndrome exhibited significantly poorer glycemic control, greater insulin resistance, more atherogenic lipid profiles, and elevated inflammatory markers compared to those without the syndrome. The increased prevalence of diabetic complications in patients with metabolic syndrome underscores the synergistic effect of multiple metabolic abnormalities in accelerating disease progression.

The findings of this study emphasize the critical importance of comprehensive metabolic evaluation in diabetic patients, with particular attention to identifying and managing metabolic syndrome components. Early recognition and aggressive management of metabolic syndrome may significantly improve cardiovascular outcomes and reduce the burden of diabetic complications. Healthcare providers should adopt a holistic approach to diabetes management, addressing not only glycemic control but also the multiple facets of metabolic dysfunction that characterize this complex syndrome.
The high prevalence of metabolic syndrome in type 2 diabetic patients emphasizes the need for comprehensive metabolic evaluation and targeted therapeutic interventions. Early identification and management of metabolic syndrome components may significantly improve cardiovascular outcomes and overall prognosis in diabetic patients.

REFERENCE LIST

1. International Diabetes Federation. IDF Diabetes Atlas, 10th edn. Brussels, Belgium: International Diabetes Federation; 2021.
2. Reaven GM. Banting lecture 1988. Role of insulin resistance in human disease. Diabetes. 1988;37(12):1595-607.
3. Alberti KG, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI, Donato KA, et al. Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention. Circulation. 2009;120(16):1640-5.
4. Mottillo S, Filion KB, Genest J, Joseph L, Pilote L, Poirier P, et al. The metabolic syndrome and cardiovascular risk a systematic review and meta-analysis. J Am Coll Cardiol. 2010;56(14):1113-32.
5. DeFronzo RA, Ferrannini E. Insulin resistance. A multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. Diabetes Care. 1991;14(3):173-94.
6. Després JP, Lemieux I. Abdominal obesity and metabolic syndrome. Nature. 2006;444(7121):881-7.
7. Taskinen MR. Diabetic dyslipidaemia: from basic research to clinical practice. Diabetologia. 2003;46(6):733-49.
8. Sowers JR, Epstein M, Frohlich ED. Diabetes, hypertension, and cardiovascular disease: an update. Hypertension. 2001;37(4):1053-9.
9. Festa A, D'Agostino R Jr, Howard G, Mykkänen L, Tracy RP, Haffner SM. Chronic subclinical inflammation as part of the insulin resistance syndrome: the Insulin Resistance Atherosclerosis Study (IRAS). Circulation. 2000;102(1):42-7.
10. Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. Lancet. 2005;365(9468):1415-28.
11. Athyros VG, Ganotakis ES, Elisaf MS, Liberopoulos EN, Goudevenos IA, Karagiannis A. Prevalence of vascular disease in metabolic syndrome using three proposed definitions. Int J Cardiol. 2007;117(2):204-10.
12. Isomaa B, Almgren P, Tuomi T, Forsén B, Lahti K, Nissén M, et al. Cardiovascular morbidity and mortality associated with the metabolic syndrome. Diabetes Care. 2001;24(4):683-9.

13. Lorenzo C, Williams K, Hunt KJ, Haffner SM. The National Cholesterol Education Program - Adult Treatment Panel III, International Diabetes Federation, and World Health Organization definitions of the metabolic syndrome as predictors of incident cardiovascular disease and diabetes. Diabetes Care. 2007;30(1):8-13.
14. Ramachandran A, Snehalatha C, Satyavani K, Sivasankari S, Vijay V. Metabolic syndrome in urban Asian Indian adults--a population study using modified ATP III criteria. Diabetes Res Clin Pract. 2003;60(3):199-204.
15. Sowers JR, Haffner S. Treatment of cardiovascular and renal risk factors in the diabetic hypertensive. Hypertension. 2002;40(6):781-8.
16. Ninomiya JK, L'Italien G, Criqui MH, Whyte JL, Gamst A, Chen RS. Association of the metabolic syndrome with history of myocardial infarction and stroke in the Third National Health and Nutrition Examination Survey. Circulation. 2004;109(1):42-6.
17. DeFronzo RA, Ferrannini E, Groop L, Henry RR, Herman WH, Holst JJ, et al. Type 2 diabetes mellitus. Nat Rev Dis Primers. 2015;1:15019.
18. Taskinen MR, Borén J. New insights into the pathophysiology of dyslipidemia in type 2 diabetes. Atherosclerosis. 2015;239(2):483-95.
19. Festa A, D'Agostino R Jr, Tracy RP, Haffner SM. Elevated levels of acute-phase proteins and plasminogen activator inhibitor-1 predict the development of type 2 diabetes: the insulin resistance atherosclerosis study. Diabetes. 2002;51(4):1131-7.
20. Bonora E, Targher G, Formentini G, Calcaterra F, Lombardi S, Marini F, et al. The Metabolic Syndrome is an independent predictor of cardiovascular disease in Type 2 diabetic subjects. Prospective data from the Verona Diabetes Complications Study. Diabet Med. 2004;21(1):52-8.