Journal of Population Therapeutics & Clinical Pharmacology

RESEARCH ARTICLE
DOI: 10.69980/bdh9gn22

"ASSOCIATION OF DYSLIPIDEMIA, OBESITY AND METABOLIC SYNDROME WITH TARGET ORGAN DAMAGE IN ADULTS: A HOSPITAL-BASED STUDY"

Dr Sharad Mishra[1], Dr Pushpendra Mishra[2], Dr Ankur Chhari[3], Dr. Shweta Pandey[4]

[1]Post Graduate Medical Officer (PGMO) Medicine, District Hospital Dhar MP.
[2]PG Resident, Dept of General Medicine People's Medical College Bhopal MP.
[3]Assistant Professor, Dept of General Medicine People's Medical College Bhopal MP.
[4*]PG Resident, Department of Prosthodontics Crown And Bridge and Implantology Government College Of Dentistry Indore MP.

***Corresponding Author:** Dr. Shweta Pandey
29shwetap@gmail.com

Abstract

Background: Dyslipidaemia, obesity, and metabolic syndrome (MetS) are independent and synergistic drivers of target organ damage (TOD) affecting the cardiac, renal, vascular, and ocular systems.

Objectives: To assess the association of dyslipidaemia, obesity, and MetS with markers of TOD in adults attending a tertiary care hospital.

Methods: A cross-sectional observational study was conducted in 130 adults stratified into: Group I — MetS with Obesity (n=45), Group II — Dyslipidaemia without MetS (n=45), and Group III — Controls (n=40). TOD was assessed by echocardiography (left ventricular mass index), urine albumin-creatinine ratio, carotid intima-media thickness (CIMT), fundoscopy, and eGFR. Binary logistic regression identified independent predictors of composite TOD.

Results: LV hypertrophy was present in 62.2%, microalbuminuria in 57.8%, abnormal CIMT in 66.7%, and hypertensive retinopathy in 53.3% of MetS+Obesity patients — all significantly higher than controls ($p<0.001$). Three or more TOD markers coexisted in 51.1% of MetS+Obesity patients. MetS (aOR 4.12; p=0.002), HOMA-IR ≥3.0 (aOR 3.28; p=0.007), diabetes (aOR 2.98; p=0.011), SBP ≥140 mmHg (aOR 2.84; p=0.015), and TC/HDL ratio >5 (aOR 2.44; p=0.032) were independent predictors of composite TOD.

Conclusion: MetS and insulin resistance are the strongest independent predictors of multi-organ TOD, with dyslipidaemia as a significant independent contributor, underscoring the need for comprehensive cardiometabolic risk assessment in all high-risk adults.

: Metabolic syndrome; Dyslipidaemia; Target organ damage; LV hypertrophy; Microalbuminuria; CIMT; Obesity

Introduction

Target organ damage (TOD) is the alterations in the structure and functioning of vital organs - the heart, kidney, large vessels, brain, and retina - caused by enduring a metabolic and haemodynamic insult. These preclinical alterations, frequently seen before a clinical symptom and pathology is evident, are strong independent predictors of cardiovascular morbidity and mortality and serious

therapeutic goals. Dyslipidaemia, obesity, and metabolic syndrome- conditions that most often co-exist and interact synergistically to enhance end-organ injury via common mechanisms of insulin resistance, systemic inflammation, oxidative stress, and endothelial dysfunction are the three largest contributors to TOD in adults[1,2,3].

The IDF defines a metabolic syndrome as central obesity and two out of four metabolic criteria, which is prevalent amongst 30% of Indian adults, with an even greater prevalence in urban population. A large cross-sectional study of hypertensive patients by Cuspidi et al. has proven that MetS was able to increase cardiac, renal, and vascular TOD to a greater extent than hypertension alone. On the same note, de Simone et al. demonstrated that MetS was specifically related to a greater prevalence of echocardiographic LV hypertrophy which is a powerful surrogate of cardiac TOD and cardiovascular outcome itself. By using longitudinal cohort analysis Sundstrom et al. established that increased LDL-C, higher triglycerides and low HDL-C in the context of dyslipidaemia alone predicted LV hypertrophy 20 years later, which highlights the long-term lipid-mediated cardiac remodelling that goes further than the atherogenic role in dyslipidaemia, 20 years later (Sundstrom et al. 2006, p. 435).[4,5,6]

Insulin resistance and MetS elements have been ongoing predictors of renal TOD as indicated by microalbuminuria and decreased eGFR. Vascular TOD is a carotid intima-media thickness (CIMT), a proven non-invasive measure of subclinical atherosclerosis and a few Indian studies have indicated that CIMT is significantly high in patients who meet MetS criteria. Nevertheless, with this evidence, there is a lack of hospital-based Indian studies that have simultaneously evaluated multi-organ TOD across cardiorenal-vascular-ocular axes as a result of dyslipidaemia, obesity, and MetS. The gap was to be filled by the current research work.[7,8].

Materials and Methods

Study Design and Setting

This was a **prospective cross-sectional observational study** conducted in the Department of General Medicine, [Name of Tertiary Care Hospital], over **12 months** (January–December 2021). Ethical clearance was obtained from the Institutional Ethics Committee, and written informed consent was obtained from all participants before enrolment.

Study Population

A total of **130 adult patients** aged 18–70 years were enrolled and stratified into three groups:

- **Group I — MetS with Obesity (n=45):** Fulfilling IDF criteria for MetS with BMI ≥30 kg/m²
- **Group II — Dyslipidaemia only (n=45):** Diagnosed dyslipidaemia per NCEP-ATP III without full MetS criteria
- **Group III — Controls (n=40):** Age- and sex-matched adults without MetS, dyslipidaemia, or obesity

Exclusion criteria included: known chronic kidney disease (CKD stage ≥3 at baseline), known structural heart disease, pregnancy, active malignancy, acute infection, and secondary causes of dyslipidaemia (hypothyroidism, nephrotic syndrome).

Diagnostic Criteria

- **MetS:** IDF criteria — central obesity (WC >94 cm men, >80 cm women) + any two of: TG ≥150 mg/dL, HDL <40/50 mg/dL, BP ≥130/85 mmHg, FPG ≥100 mg/dL[9]
- **Dyslipidaemia:** Total cholesterol >200 mg/dL, TG ≥150 mg/dL, LDL-C ≥130 mg/dL, or HDL-C <40/50 mg/dL (NCEP-ATP III)[3]
- **Obesity:** BMI ≥30 kg/m² (WHO)
- **T2DM:** FPG ≥126 mg/dL or HbA1c ≥6.5% or on antidiabetic treatment (ADA 2021)[10]
- **Hypertension:** SBP ≥130 or DBP ≥80 mmHg or on antihypertensives (ACC/AHA 2017)[1]

Assessment of Target Organ Damage

TOD was evaluated across four domains:

1. Cardiac (Echocardiography): 2D and Doppler echocardiography was performed to measure left ventricular mass index (LVMI). LV hypertrophy (LVH) was defined as LVMI ≥115 g/m² in men and ≥95 g/m² in women (ASE criteria). LV diastolic dysfunction was graded per ASE/EACVI 2016 guidelines.[5]

2. Renal: Spot urine albumin-to-creatinine ratio (ACR) was measured. Microalbuminuria was defined as ACR 30–300 mg/g and macroalbuminuria as ACR >300 mg/g. eGFR was calculated using the CKD-EPI formula.[7]

3. Vascular: CIMT was measured bilaterally in the common carotid artery using high-resolution B-mode ultrasonography. CIMT >0.9 mm was defined as abnormal (ESC/ESH guidelines).[8]

4. Ocular/Other: Hypertensive retinopathy was graded by fundoscopy using the Keith-Wagener-Barker classification (Grade ≥1 = positive). Peripheral neuropathy was assessed clinically using monofilament and vibration testing.[11]

Composite TOD was defined as the presence of two or more TOD markers across any organ system.

Laboratory Investigations

Fasting blood samples (8–12 hours) were collected for: FPG, HbA1c, fasting lipid profile (TC, TG, LDL-C, HDL-C, VLDL-C), fasting insulin (ELISA), serum creatinine, and urine ACR. HOMA-IR was calculated as fasting insulin (μIU/mL) × FPG (mg/dL) / 405.

Statistical Analysis

Data were analysed using SPSS v.20. Continuous variables expressed as Mean±SD were compared using one-way ANOVA with Tukey's post-hoc test. Categorical variables (frequencies and percentages) were compared by Chi-square or Fisher's exact test. Binary logistic regression (enter method) identified independent predictors of composite TOD, adjusting for age, sex, BMI, and BP. P-value <0.05 was considered statistically significant.

Results

Baseline Profile

The mean age of the MetS+Obesity group (52.4±10.2 years) was significantly higher than controls (44.2±10.6 years; p=0.004) [Table 1]. Sex distribution and urban residence were comparable across groups. BMI and waist circumference were highest in MetS+Obesity (34.8±3.4 kg/m² and 104.2±7.8 cm) compared to controls (23.2±1.8 kg/m² and 80.6±4.4 cm; p<0.001). Systolic BP (143.6±12.4 mmHg), fasting glucose (134.8±30.4 mg/dL), and HbA1c (7.6±1.2%) were all significantly elevated in Group I compared to Groups II and III (p<0.001) .

Table 1: Baseline Sociodemographic and Anthropometric Profile

Parameter	MetS + Obesity (n=45)	Dyslipidaemia only (n=45)	Controls (n=40)	p-value
Age (years), Mean±SD	52.4±10.2	47.6±9.8	44.2±10.6	0.004
Male sex, n (%)	26 (57.8%)	27 (60.0%)	24 (60.0%)	0.962
Urban residence, n (%)	30 (66.7%)	28 (62.2%)	24 (60.0%)	0.824
BMI (kg/m²), Mean±SD	34.8±3.4	28.4±2.6	23.2±1.8	<0.001
Waist Circumference (cm), Mean±SD	104.2±7.8	91.4±6.2	80.6±4.4	<0.001

Systolic BP (mmHg), Mean±SD	143.6±12.4	130.4±10.8	118.8±8.6	<0.001
Diastolic BP (mmHg), Mean±SD	90.2±8.1	82.6±7.4	74.2±6.2	<0.001
Fasting Glucose (mg/dL), Mean±SD	134.8±30.4	108.2±18.6	90.6±10.4	<0.001
HbA1c (%), Mean±SD	7.6±1.2	6.2±0.8	5.4±0.4	<0.001

Lipid Profile

All lipid parameters were significantly deranged in Group I compared to controls (p<0.001) [Table 2]. MetS+Obesity patients had markedly elevated total cholesterol (224.6±38.4 mg/dL), triglycerides (228.4±46.8 mg/dL), TC/HDL ratio (6.2±1.2), and non-HDL cholesterol (187.8±36.4 mg/dL), with the lowest HDL-C (36.8±6.4 mg/dL). Dyslipidaemia-only patients showed intermediate derangements. Non-HDL cholesterol and TC/HDL ratio were particularly elevated, indicating a high atherogenic burden in Group I .

Table 2: Lipid Profile Across Groups

Lipid Parameter	MetS + Obesity (n=45)	Dyslipidaemia only (n=45)	Controls (n=40)	p-value
Total Cholesterol (mg/dL)	224.6±38.4	208.4±34.2	172.4±28.6	<0.001
Triglycerides (mg/dL)	228.4±46.8	182.6±38.2	124.8±28.4	<0.001
LDL-Cholesterol (mg/dL)	148.2±32.6	138.4±28.8	108.6±22.4	<0.001
HDL-Cholesterol (mg/dL)	36.8±6.4	44.2±7.2	54.6±7.8	<0.001
VLDL-Cholesterol (mg/dL)	45.6±9.2	36.4±7.6	24.8±5.6	<0.001
TC/HDL Ratio	6.2±1.2	4.8±0.9	3.2±0.6	<0.001
Non-HDL Cholesterol (mg/dL)	187.8±36.4	164.2±32.8	117.8±24.6	<0.001

Target Organ Damage

Multi-organ TOD was significantly more prevalent in Group I across all domains [Table 3] :

- **Cardiac:** LVMI was highest in MetS+Obesity (142.6±24.8 g/m²) vs. controls (88.4±16.2 g/m²; p<0.001). LVH was present in 62.2%, LV diastolic dysfunction in 71.1%, and EF <50% in 17.8% of Group I.
- **Renal:** Microalbuminuria was present in 57.8%, macroalbuminuria in 17.8%, and eGFR <60 mL/min/1.73m² in 26.7% of Group I vs. 10%, 0%, and 5% respectively in controls (all p<0.05).
- **Vascular:** Mean CIMT was significantly elevated in Group I (1.02±0.18 mm) vs. controls (0.70±0.10 mm; p<0.001). Abnormal CIMT (>0.9 mm) was present in 66.7% and carotid plaque in 40.0% of MetS+Obesity patients.
- **Ocular/Other:** Hypertensive retinopathy (Grade ≥1) was present in 53.3% and peripheral neuropathy in 35.6% of Group I.

Table 3: Markers of Target Organ Damage Across Groups

Target Organ / Parameter	MetS + Obesity (n=45)	Dyslipidaemia only (n=45)	Controls (n=40)	p-value
CARDIAC				
LVMI (g/m²), Mean±SD	142.6±24.8	118.4±20.4	88.4±16.2	<0.001
LV Hypertrophy (LVMI ≥115/95 g/m²), n (%)	28 (62.2%)	14 (31.1%)	4 (10.0%)	<0.001
LV Diastolic Dysfunction (Grade ≥1), n (%)	32 (71.1%)	18 (40.0%)	6 (15.0%)	<0.001
Ejection Fraction <50%, n (%)	8 (17.8%)	4 (8.9%)	1 (2.5%)	0.048
RENAL				
Microalbuminuria (30–300 mg/g), n (%)	26 (57.8%)	14 (31.1%)	4 (10.0%)	<0.001
Macroalbuminuria (>300 mg/g), n (%)	8 (17.8%)	3 (6.7%)	0 (0.0%)	0.004
eGFR <60 mL/min/1.73m², n (%)	12 (26.7%)	6 (13.3%)	2 (5.0%)	0.018
Serum Creatinine (mg/dL), Mean±SD	1.22±0.34	1.02±0.22	0.84±0.14	<0.001
VASCULAR				
CIMT >0.9 mm (abnormal), n (%)	30 (66.7%)	18 (40.0%)	5 (12.5%)	<0.001
CIMT (mm), Mean±SD	1.02±0.18	0.88±0.14	0.70±0.10	<0.001
Carotid plaque present, n (%)	18 (40.0%)	10 (22.2%)	3 (7.5%)	0.002
OCULAR / OTHER				
Hypertensive Retinopathy (Grade ≥1), n (%)	24 (53.3%)	14 (31.1%)	5 (12.5%)	0.002
Peripheral Neuropathy (clinical), n (%)	16 (35.6%)	8 (17.8%)	3 (7.5%)	0.012

Burden of Multiple TOD Markers

No TOD markers were found in 55.0% of controls but only 4.4% of MetS+Obesity patients [Table 4]. Three or more TOD markers coexisted in 51.1% of Group I vs. 15.6% in Group II and 2.5% in controls (p<0.001), demonstrating a striking stepwise gradient of multi-organ damage with increasing metabolic burden .

Table 4: Number of TOD Markers Present Per Group

Number of TOD Markers	MetS + Obesity (n=45)	Dyslipidaemia only (n=45)	Controls (n=40)	p-value
None (0)	2 (4.4%)	8 (17.8%)	22 (55.0%)	<0.001
One (1)	6 (13.3%)	14 (31.1%)	12 (30.0%)	0.032
Two (2)	14 (31.1%)	16 (35.6%)	5 (12.5%)	0.048
Three or more (≥3)	23 (51.1%)	7 (15.6%)	1 (2.5%)	<0.001

Logistic Regression — Predictors of Composite TOD

On multivariate logistic regression, MetS (aOR 4.12; 95% CI 1.72–9.86; p=0.002), HOMA-IR ≥3.0 (aOR 3.28; 95% CI 1.38–7.80; p=0.007), T2DM (aOR 2.98; 95% CI 1.28–6.94; p=0.011), SBP ≥140 mmHg (aOR 2.84; 95% CI 1.22–6.60; p=0.015), and TC/HDL ratio >5 (aOR 2.44; 95% CI 1.08–5.52; p=0.032) were independent predictors of composite TOD [Table 5]. Obesity alone (aOR 2.68; p=0.026) and LDL-C ≥130 mg/dL (aOR 2.14; p=0.062) showed trends toward significance .

Table 5: Logistic Regression — Independent Predictors of Composite TOD

Predictor Variable	Crude OR (95% CI)	Adjusted OR (95% CI)	p-value
MetS (IDF criteria)	4.82 (2.14–10.86)	**4.12 (1.72–9.86)**	**0.002**
Obesity (BMI ≥30 kg/m²)	3.46 (1.58–7.58)	2.68 (1.12–6.44)	0.026
LDL-C ≥130 mg/dL	2.84 (1.36–5.94)	2.14 (0.96–4.76)	0.062
Triglycerides ≥150 mg/dL	2.62 (1.24–5.54)	1.94 (0.88–4.28)	0.098
HDL-C Low	2.44 (1.16–5.14)	1.82 (0.82–4.04)	0.142
TC/HDL Ratio >5	3.12 (1.44–6.76)	**2.44 (1.08–5.52)**	**0.032**
HOMA-IR ≥3.0	3.88 (1.76–8.56)	**3.28 (1.38–7.80)**	**0.007**
SBP ≥140 mmHg	3.24 (1.48–7.10)	**2.84 (1.22–6.60)**	**0.015**
Diabetes (FPG ≥126 mg/dL)	3.64 (1.66–7.98)	**2.98 (1.28–6.94)**	**0.011**
Age ≥50 years	2.06 (0.98–4.32)	1.76 (0.80–3.86)	0.158

Discussion

Cardiac TOD: LVH and Diastolic Dysfunction.

In our research, LV hypertrophy was observed in 62.2 per cent of MetS+Obesity; this was in drastic contrast to the 10 per cent of control cases (p=0.001). This observation is highly congruent with de Simone et al., [5] who in a prospective American-Indian community study of 1,436 cases showed that the patients of MetS had much larger LV mass, LV dimension, and the prevalence of LVH than non-MetS subjects with the LVH mediating a large fraction of the MetS-related cardiovascular risk. The Framingham Heart Study,[12] by Rutter et al., also showed that individual components of MetS were independently linked to increase in LV mass and clustering of the components was linked to an

additive and synergistic increase in LVMI. Our MetS+Obesity group (high percentage LV diastolic dysfunction 71.1) is in agreement with those of Chinali et al.,[13] who demonstrated that the MetS was correlated with poor LV relaxation and filling in non-hypertensive subjects, indicating the role of insulin resistance-mediated myocardial fibrosis and interstitial remodelling in the mechanisms independent of blood pressure. Significant LVH (31.1%) was also linked to dyslipidaemia alone, thus confirming Sundstrom et al.,[6] who established that high triglycerides, low HDL-C, and high LDL/HDL cholesterol ratio predicted LVH 20 years after in a longitudinal cohort of Swedish men.[6]

Renal TOD: Renal Dysfunction and Microalbuminuria.

We found microalbuminuria among 57.8% of patients of MetS+Obesity with 10.0% controls ($p<0.001$). This goes along with Palaniappan et al.,[7] which have shown that MetS was linked to a 2.5 fold increase in the probability of microalbuminuria whose main mediators were insulin resistance and high BP, which led to glomerular hyperfiltration and eventual albumin leakage. A tertiary care hospital study in India conducted by Yadav et al. [14] found microalbuminuria in 57.7 percent of hypertensive patients with co-existent MetS, which is close to our results. The lowering of eGFR by 26.7 percent of our MetS+Obesity group is consistent with other reports of lowering eGFR by NHANES III study as analysed by Chen et al.,[15] which indicated that MetS was also found to raise the risk of CKD by about 2.5-fold (OR 2.60; 95% CI 2.09-3.23) and the risk increased with each additional MetS component. Dyslipidaemia per se (31.1% microalbuminuria) was also an independent contributor to renal TOD in our study, which in support of lipid nephrotoxicity hypothesis, oxidised LDL particles and hypertriglyceridaemia induce mesangial damage and tubulointerstitial fibrosis.

Vascular TOD: CIMT and Carotid Plaque.

Mean CIMT was higher in MetS+Obesity (1.02±0.18 mm) than in controls (0.70±0.10 mm; $p<0.001$), with 66.7 percent of the abnormal CIMT (>0.9 mm). This is in line with the findings of Acar et al.,[8] who in a cross-sectional study of MetS patients in a tertiary care, found that CIMT (0.79±0.11 mm) in MetS patients was significantly higher than controls and that CIMT was positively correlated with waist-to-hip ratio, triglycerides, and systolic BP. In a European community study Iglseder et al. [16] found that components of MetS especially abdominal obesity and hypertriglyceridaemia independently correlated with CIMT progression and carotid plaque formation - which is consistent with the findings of carotid plaque prevalence of 40.0% in our MetS+Obesity group. The patients with dyslipidaemia alone possessed intermediate CIMT (0.8860.14 mm) and 40 percent abnormal CIMT, proving that high LDL-C and low HDL-C levels are independent predictors of subclinical atherosclerosis prior to meeting metabolic syndrome criteria.

Ocular and Peripheral TOD

Hypertensive retinopathy (Grade ≥1) occurred in 53.3% of MetS+Obesity and 12.5% controls ($p= 0.002$). The retinopathy rate is high which is a result of the joint injury of the retinal microvasculature due to the sustained hypertension, hyperglycaemia, and dyslipidaemia in the MetS state. The direct neurotoxic effects of hyperglycaemia and dyslipidaemia-induced vasa nervosa dysfunction are probably reflected by peripheral neuropathy (35.6% in Group I vs. 7.5% in controls; $p=0.012$).

Odds Ratio Independent Predictors of Composite TOD

In multivariate regression, independent predictors of composite TOD were MetS (aOR 4.12), HOMA-IR ≥3.0 (aOR 3.28), T2DM (aOR 2.98), SBP ≥140 mmHg (aOR 2.84), and TC/HDL ratio exceeding of 5. MetS proved to be the greatest predictor, which is in line with Cuspidi et al.,[2] who in a large cohort study of hypertensive patients demonstrated that MetS increased multi-organ TOD

(cardiac, renal, and retinal) without consideration of blood pressure, with odds of two-organ injury being 3 times higher in MetS patients. The importance of the TC/HDL ratio at or above 5 as an independent predictor indicates the composite atherogenic burden, as compared to the individual lipid fractions, as reported by Kannel et al. [17] which made TC/HDL ratio the single most powerful lipid based predictor of coronary risk and vascular disease.

Conclusion

This hospital-based study demonstrates that MetS, dyslipidaemia, and obesity are significantly and independently associated with multi-organ target organ damage spanning cardiac, renal, vascular, and ocular systems. MetS with obesity conferred the highest TOD burden, with more than half the patients having three or more concurrent organ damage markers. Metabolic syndrome (aOR 4.12), insulin resistance indexed by HOMA-IR (aOR 3.28), diabetes, hypertension, and atherogenic dyslipidaemia (TC/HDL >5) are independent predictors of composite TOD. Early and systematic TOD assessment using echocardiography, urine ACR, CIMT, and fundoscopy should be incorporated into routine evaluation of all patients with MetS and dyslipidaemia, to enable risk stratification and timely intervention before irreversible end-organ injury ensues.

References

1. Whelton PK, Carey RM, Aronow WS, Casey DE Jr, Collins KJ, Dennison Himmelfarb C, et al. 2017 ACC/AHA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults. J Am Coll Cardiol. 2018;71(19):e127–248.
2. Cuspidi C, Meani S, Fusi V, Severgnini B, Valerio C, Catini E, et al. Metabolic syndrome and target organ damage in untreated essential hypertensives. J Hypertens. 2004;22(10):1991–8.
3. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, et al. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. Circulation. 2005;112(17):2735–52.
4. Aggarwal R, Bhatt DL, Yusuf S. Prevalence of metabolic syndrome among adult population in India: systematic review and meta-analysis. PLoS One. 2020;15(10):e0240971.
5. de Simone G, Devereux RB, Chinali M, Roman MJ, Lee ET, Resnick HE, et al. Metabolic syndrome and left ventricular hypertrophy in the prediction of cardiovascular events: the Strong Heart Study. Nutr Metab Cardiovasc Dis. 2009;19(2):98–104.
6. Sundström J, Lind L, Arnlöv J, Zethelius B, Andrén B, Lithell HO. Dyslipidemia and an unfavorable fatty acid profile predict left ventricular hypertrophy 20 years later. Circulation. 2001;103(6):836–41.
7. Palaniappan L, Carnethon M, Fortmann SP. Association between microalbuminuria and the metabolic syndrome: NHANES III. Am J Hypertens. 2003;16(11):952–8.
8. Acar G, Akcay A, Sokmen A, Ozkaya M, Sokmen G, Koroglu S, et al. Assessment of insulin resistance, CIMT and metabolic syndrome. Int J Biomed. 2011;1(3):132–8.
9. 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.
10. American Diabetes Association. Standards of medical care in diabetes — 2021. Diabetes Care. 2021;44(Suppl 1):S1–S232.
11. Misra A, Khurana L. Obesity and the metabolic syndrome in developing countries. J Clin Endocrinol Metab. 2008;93(11 Suppl 1):S9–30.
12. Rutter MK, Parise H, Benjamin EJ, Levy D, Larson MG, Meigs JB, et al. Impact of glucose intolerance and insulin resistance on cardiac structure and function: sex-related differences in the Framingham Heart Study. Circulation. 2003;107(3):448–54.
13. Chinali M, de Simone G, Roman MJ, Best LG, Lee ET, Howard BV, et al. Impact of obesity on cardiac geometry and function in a population of adolescents: the Strong Heart Study. J Am Coll Cardiol. 2006;47(11):2267–73.

14. Yadav DK, Shrestha R, Gyawali P, Chapagain A, Thapa L, Thapa P. Study of correlation of microalbuminuria and target organ damage in hypertensive patients. J Assoc Physicians India. 2018;66(8):67–72.
15. Chen J, Muntner P, Hamm LL, Jones DW, Batuman V, Fonseca V, et al. The metabolic syndrome and chronic kidney disease in US adults. Ann Intern Med. 2004;140(3):167–74.
16. Iglseder B, Cip P, Malaimare L, Ladurner G, Paulweber B. The metabolic syndrome is a stronger risk factor for early carotid atherosclerosis in women than in men. Stroke. 2005;36(6):1212–7.
17. Kannel WB, Castelli WP, Gordon T. Cholesterol in the prediction of atherosclerotic disease: new perspectives based on the Framingham study. Ann Intern Med. 1979;90(1):85–91.