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Lipid Profile And Target Organ Damage In Hypertension: A Retrospective Hospital-Based Study With Visual Analysis

Manthena Jagadish
MBBS Final Year Part I, SVS Medical College
Mahabubnagar Telangana 509001, India

Dr. Umesh Babu
MG, Professor
Department Of General Medicine
SVSMCH

Abstract
Background: *Hypertension and dyslipidemia frequently coexist and synergistically increase cardiovascular risk through development of target organ damage (TOD).*
Comprehensive evaluation of lipid abnormalities in relation to multi-organ TOD in established hypertension is limited in Indian populations.
Aim: *To retrospectively evaluate the prevalence of dyslipidemia and target organ damage in hypertensive patients and examine associations between lipid parameters and organ damage markers using visual epidemiological analysis.*
Methods: *Hospital medical records from 240 hypertensive adults (May 2025-December 2025) were reviewed. Comprehensive investigations included lipid profile, electrocardiography, echocardiography, urinalysis, retinal examination, and pulse wave velocity. Associations were assessed using t-tests and correlations. Data visualization included pie charts (proportions), bar graphs (prevalence), and scatter plots (correlations).*
Results: *Mean age 54.4 ± 11.3 years (50.4% male); systolic BP 156.7 ± 13.4 mmHg. Dyslipidemia: 238/240 (99.2%). Target organ damage present in 213/240 (88.8%), including microalbuminuria (52.5%), hypertensive retinopathy (46.7%), elevated pulse wave velocity (36.7%), and electrocardiographic left ventricular hypertrophy (34.2%). Multi-organ involvement (≥2 systems) in 126/240 (52.5%).*
Total and non-HDL cholesterol showed weak associations with retinopathy ($p<0.05$). Most lipid parameters demonstrated minimal correlation with individual TOD markers.
Conclusion: *Near-universal dyslipidemia coexists with highly prevalent target organ damage in established hypertension. Blood pressure elevation remains the primary determinant of organ injury; dyslipidemia plays a modulatory role.*
Comprehensive organ evaluation and intensive lipid management should be standard in established hypertension.
Keywords: *hypertension; dyslipidemia; target organ damage; cross-sectional study; cardiovascular risk; retrospective analysis*

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I. Introduction

Hypertension affects over 1.13 billion individuals globally and represents a leading cause of preventable cardiovascular mortality.[1] In India, hypertension prevalence exceeds 30% in urban populations, with many remaining undiagnosed or inadequately treated.[2] Despite pharmacological management, many hypertensive patients develop progressive subclinical target organ damage (TOD) across multiple vascular beds including the heart, kidneys, retina, and arterial wall.

Target organ damage encompasses electrocardiographic left ventricular hypertrophy (LVH), microalbuminuria indicating early glomerular dysfunction, hypertensive retinopathy reflecting microvascular disease, and increased arterial stiffness measured by pulse wave velocity (PWV).[3] The presence of TOD independently predicts future cardiovascular events, progression to chronic kidney disease, and mortality, making it a critical intermediate outcome for risk stratification in hypertensive patients.[4]

Dyslipidemia frequently coexists with hypertension, reported in 60-85% of hypertensive populations.

The concurrent presence of these atherogenic risk factors—termed "lipitension"—creates a synergistic state accelerating atherosclerosis and vascular remodeling.[6] Lipid abnormalities amplify hypertensive organ damage through oxidative stress, inflammation, and endothelial dysfunction.

However, comprehensive data systematically evaluating the relationship between lipid abnormalities and established patterns of multi-organ TOD in Indian hypertensive cohorts remain limited. Most prior studies have examined single organ systems in isolation or focused on incident rather than established hypertension. This study addresses these evidence gaps through detailed retrospective analysis with visual epidemiological presentation.

Our objectives were to: (1) characterize the prevalence of dyslipidemia and individual TOD markers in hypertensive patients; (2) evaluate associations between lipid parameters and organ damage across multiple systems; and (3) provide visual epidemiological analysis through pie charts, bar graphs, and comparative displays suitable for clinical interpretation and teaching.

II. Methods

Study Design and Setting

Retrospective cross-sectional study using hospital medical records from outpatient cardiology, internal medicine, and nephrology departments at SVS Medical College, Mahabubnagar, Telangana, India. Study period: May 2025 to December 2025. The Institutional Ethics Committee approved the protocol with waiver of informed consent (approval number: IEC/DHR-03/(06)/2025, dated [13/12/2025]).

Study Population

Inclusion: (1) Age ≥18 years; (2) Documented hypertension diagnosis (BP ≥140/90 mmHg on ≥2 visits or on antihypertensive therapy); (3) Complete baseline investigations including lipid profile and ≥2 of: 12-lead ECG, echocardiography, spot urine ACR, fundoscopic examination, PWV measurement; (4) Complete data without missing values.

Exclusion: (1) Secondary hypertension; (2) Incomplete medical records; (3) Acute concurrent illness; (4) Severe comorbidities precluding assessment.

Of 280 potentially eligible patients, 240 (85.7%) met criteria. Mean follow-up time from hypertension diagnosis: 4.2 ± 2.8 years.

Data Collection and Measurements

Blood Pressure: Clinic readings extracted from medical records; mean of available measurements used.

Lipid Panel: Fasting total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides measured by enzymatic colorimetric methods. Non-HDL cholesterol calculated as total cholesterol minus HDL. Triglyceride-to-HDL ratio computed for each patient.

Cardiac Assessment: 12-lead ECG evaluated for LVH using voltage criteria (Sokolow-Lyon >3.5 mV or Cornell criteria). Transthoracic echocardiography provided left ventricular mass index (LVMI); LVH defined as LVMI >115 g/m² (males) or >95 g/m² (females).

Renal Evaluation: Spot urine ACR extracted; microalbuminuria defined as ACR 30-300 mg/g. eGFR calculated using CKD-EPI equation.

Retinal Examination: Ophthalmology documentation reviewed; hypertensive retinopathy graded per Wong-Mitchell classification (Grade 0-IV).

Vascular Assessment: Carotid-femoral PWV measured via applanation tonometry; elevated PWV defined as ≥10 m/s.

Data Analysis and Statistics

Continuous variables: mean ± SD (range); categorical: count/percentage.

Normality assessed via Shapiro-Wilk test.

Independent samples t-tests compared lipid values between patients with/without each TOD marker. Pearson and Spearman correlations assessed associations between lipid parameters and continuous TOD measures. Chi-square tests evaluated categorical associations. Two-tailed tests; $\alpha=0.05$.

Data Visualization

Figures generated using Excel 2016 to display: (1) Pie charts—proportion with any TOD and distribution of organ involvement; (2) Bar graphs—prevalence of individual TOD markers and lipid abnormalities; (3) Grouped bars—comparative TOD by dyslipidemia status; (4) Scatter plots—correlations between lipid parameters and continuous TOD measures.

Visualization methodology follows epidemiological principles for clear clinical communication of complex data patterns.

III. Results

Patient Demographics and Baseline Characteristics

Two hundred forty hypertensive patients: mean age 54.4 ± 11.3 years (range 25–85); 121 (50.4%) male, 119 (49.6%) female. Mean systolic BP 156.7 ± 13.4 mmHg (range 140–193); mean diastolic BP 97.2 ± 8.9 mmHg (range 85–122). Mean disease duration 4.2 ± 2.8 years. Baseline characteristics presented in Table 1.

Prevalence of Target Organ Damage

Subclinical organ damage was remarkably prevalent. Any TOD present in 213/240 (88.8%) (Figure 1). Distribution of organ involvement shown in Figure 2: 27 patients (11.3%) no TOD; 87 (36.3%) single-organ; 126 (52.5%) multi-organ involvement.

Individual TOD marker prevalence displayed in Figure 3:
• Microalbuminuria: 126 (52.5%) [mean ACR 48.4 ± 45.7 mg/g]
• Hypertensive retinopathy: 112 (46.7%)
– Grade 0: 128 (53.3%)
– Grade I: 60 (25.0%)
– Grade II: 29 (12.1%)
– Grade III: 15 (6.3%)
– Grade IV: 8 (3.3%)
• Elevated PWV (≥10 m/s): 88 (36.7%) [mean 9.36 ± 1.84 m/s]
• ECG-LVH (voltage criteria): 82 (34.2%)
• Reduced eGFR (<90): 44 (18.3%)
Mean LVMI 100.4 ± 24.7 g/m² (range 60.0–161.8); zero patients met echocardiographic LVH criteria.

Dyslipidemia Prevalence and Lipid Profile

Near-universal dyslipidemia: 238/240 (99.2%), with only 2 (0.8%) having completely normal lipid profiles. Lipid abnormality prevalence displayed in Figure 4.

Total cholesterol 222.1 ± 33.4 mg/dL (range 150–331):
• ≥200 mg/dL: 216 (90.0%)

LDL cholesterol 144.4 ± 39.2 mg/dL (range 60–250):
• ≥130 mg/dL: 175 (72.9%)

HDL cholesterol 39.4 ± 7.6 mg/dL (well below guideline targets):
• <40 mg/dL (males) or <50 mg/dL (females): 189 (78.8%)

Triglycerides 184.4 ± 56.5 mg/dL (range 50–371):
• ≥150 mg/dL: 181 (75.4%)

Non-HDL cholesterol 182.7 ± 34.8 mg/dL:
• ≥130 mg/dL: 232 (96.7%)

Triglyceride-to-HDL ratio 4.88 ± 1.89:
• >3.5 (atherogenic): 166 (69.2%)

Associations between Lipid Parameters and Target Organ Damage

Lipid-TOD associations presented in Table 2. Total cholesterol slightly elevated in retinopathy group (226.8 vs. 218.1 mg/dL, p=0.044). Non-HDL cholesterol similarly elevated with retinopathy (187.5 vs. 178.5 mg/dL, p=0.045). Other lipid parameters showed no significant differences across TOD markers (all p>0.05).

Comparative TOD prevalence by dyslipidemia status shown in Figure 5. Due to near-universal dyslipidemia (99.2%), subgroup comparison severely limited (only 2 normolipidemic patients).

Correlation analyses (Table 3) revealed predominantly very weak correlations between lipid variables and continuous TOD measures ($|r| < 0.20$, $p > 0.05$ for most comparisons). Representative scatter plots displayed in Figure 6 (total cholesterol vs. LVMI, triglycerides vs. PWV).

These weak cross-sectional associations suggest blood pressure elevation remains the primary determinant of accumulated organ damage in established hypertension, with dyslipidemia playing a secondary modulatory role.

Multi-Organ Damage Patterns

More than half the cohort (126/240, 52.5%) demonstrated involvement of ≥2 organ systems (Figure 2). Common patterns: microalbuminuria + retinopathy (n=68, 28.3%), microalbuminuria + elevated PWV (n=54, 22.5%), microalbuminuria + ECG-LVH (n=42, 17.5%). This systemic vascular involvement indicates widespread end-organ damage.

IV. Discussion

Main Findings

In this retrospective cohort of 240 hypertensive patients with comprehensive multi-organ assessment, we found: (1) highly prevalent target organ damage (88.8%), affecting multiple organ systems in over half the cohort; (2) near-universal dyslipidemia (99.2%) with predominantly atherogenic lipid phenotype; (3) weak cross-sectional lipid-TOD associations, indicating blood pressure elevation as the primary driver with dyslipidemia as secondary amplifier.

These findings extend prior literature by demonstrating the magnitude of organ damage in established hypertension and providing visual epidemiological analysis suitable for clinical practice and patient education.

Prevalence of Target Organ Damage in Established Hypertension

The 88.8% TOD prevalence substantially exceeds estimates from cross-sectional surveys of general populations (typically 20-40%), reflecting accumulated injury over years of sustained hypertension. Microalbuminuria prevalence (52.5%) aligns with published hypertensive cohorts (40-60%).[7] Retinopathy prevalence (46.7%) similarly matches prior studies in treated hypertensive populations.

The high prevalence of multi-organ involvement (52.5%) demonstrates systemic vascular disease rather than isolated organ effects. This finding underscores the importance of comprehensive baseline evaluation encompassing cardiac, renal, retinal, and vascular assessment in all hypertensive patients.

Dyslipidemia in Hypertension

The remarkable 99.2% dyslipidemia prevalence illustrates the strong biological coupling between blood pressure elevation and lipid dysregulation (lipitension).[6]

The predominant pattern—elevated triglycerides, low HDL, elevated non-HDL cholesterol—characterizes atherogenic dyslipidemia associated with increased cardiovascular risk and metabolic dysfunction.

The high TG/HDL ratio prevalence (69.2%) is noteworthy, as this simple marker has been proposed as surrogate for atherogenic dyslipidemia and insulin resistance in population studies.[8]

Visual Epidemiological Analysis

This study employed comprehensive data visualization (Figures 1-6) to present complex epidemiological relationships in clinically meaningful formats:

- Pie charts (Figures 1-2) rapidly convey proportion with disease
- Bar graphs (Figures 3-4) enable quick visual comparison of prevalence across markers and abnormalities
- Grouped bars (Figure 5) facilitate comparison between patient subgroups

This visualization approach follows epidemiological best practices for communicating data to diverse audiences including clinicians, researchers, patients, and public health officials.

Weak Lipid-TOD Associations: Interpretation

Despite atherogenic lipid abnormalities in nearly all patients, lipid parameters showed minimal association with individual TOD markers. Several explanations:

1. Near-universal dyslipidemia provides insufficient variation for meaningful comparison; with 99.2% prevalence, statistical power severely limited.
2. In established hypertension, sustained blood pressure elevation remains dominant driver of cumulative organ damage, overwhelming lipid effects in cross-sectional relationships.

3. Cross-sectional design cannot establish temporal relationships; lipid values at review may not reflect values when organ damage initially occurred.
4. Medication use (lipid-lowering, antihypertensive agents) modified lipid values in many patients, attenuating lipid-TOD associations.

This pattern—weak lipid-TOD associations despite high dyslipidemia prevalence—differs from some prior reports, but aligns with recent studies demonstrating that in established hypertension, blood pressure control remains the critical priority with lipid management as secondary strategy.

Clinical Implications
These findings support several clinical recommendations:
1. Comprehensive organ evaluation should be standard at baseline hypertension assessment, including ECG, renal function, urinalysis, fundoscopy, and arterial stiffness measurement (if available).
2. The high microalbuminuria prevalence (52.5%) indicates ACR should be measured in all hypertensive patients, not limited to high-risk subgroups.
3. Retinopathy presence should trigger treatment intensification and more aggressive BP targets.
4. Multi-organ involvement in over half the cohort justifies intensive cardiovascular risk reduction: (a) aggressive BP control (<130/80 mmHg targets for those with TOD); (b) high-intensity statin therapy; (c) structured lifestyle interventions; (d) consideration of organ-protective agents.
5. Visual epidemiological displays (Figures 1-6) provide tools for clinician education and patient communication regarding cardiovascular risk.

Strengths and Limitations
Strengths: (1) large cohort (n=240) with comprehensive multi-organ assessment; (2) use of validated measurement techniques; (3) complete data; (4) real-world clinical population; (5) comprehensive visual epidemiological analysis.
Limitations: (1) Retrospective design limits temporal relationships; (2) Selection bias (patients with documented investigations preferentially included); (3) 99.2% dyslipidemia severely limits statistical power for lipid-TOD comparisons; (4) Lack of information on medication adherence, prior lipid values, or disease duration; (5) Single institution limits generalizability; (6) No inflammatory markers assessed.

V. Conclusion

In this retrospective analysis of 240 hypertensive patients with comprehensive organ evaluation and visual epidemiological analysis, we demonstrate that subclinical target organ damage is highly prevalent (88.8%), affecting multiple organ systems in over half the cohort, while dyslipidemia is near-universal (99.2%). Blood pressure elevation remains the primary determinant of accumulated organ damage in established hypertension, with dyslipidemia playing a secondary modulatory role.

These findings argue for: (1) comprehensive baseline and periodic organ evaluation in all hypertensive patients; (2) universal ACR screening; (3) routine ophthalmologic examination; (4) intensive cardiovascular risk reduction in patients with documented TOD; and (5) use of visual epidemiological displays to communicate complex cardiovascular risk patterns to clinical audiences and patients.

Future prospective studies should examine whether baseline lipid abnormalities predict organ damage progression and whether intensive lipid management reduces adverse outcomes in hypertensive cohorts with established TOD.

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Tables

Table 1. Baseline Characteristics of Study Population

Variable	Mean ± SD	Range	n (%)
Age (years)	54.4 ± 11.3	25–85	
Sex (Male)			121 (50.4)
Systolic BP (mmHg)	156.7 ± 13.4	140–193	
Diastolic BP (mmHg)	97.2 ± 8.9	85–122	
Disease duration (years)	4.2 ± 2.8	1–15	
Total cholesterol (mg/dL)	222.1 ± 33.4	150–331	
LDL cholesterol (mg/dL)	144.4 ± 39.2	60–250	
HDL cholesterol (mg/dL)	39.4 ± 7.6	25–61	
Triglycerides (mg/dL)	184.4 ± 56.5	50–371	
Non-HDL cholesterol (mg/dL)	182.7 ± 34.8	97–293	
TG/HDL ratio	4.88 ± 1.89	1.32–10.80	
Serum creatinine (mg/dL)	0.95 ± 0.24	0.50–1.93	
eGFR (mL/min/1.73m²)	105.8 ± 18.0	61–150	
Urine ACR (mg/g)	48.4 ± 45.7	5.0–231.9	
LVMI (g/m²)	100.4 ± 24.7	60.0–161.8	
PWV (m/s)	9.36 ± 1.84	6.50–15.80	

Table 2. Lipid Parameters Compared Between Patients With and Without Target Organ Damage Markers

Lipid variable	TOD marker	With TOD (mean ± SD)	Without TOD (mean ± SD)	p-value
Total cholesterol (mg/dL)	Microalbuminuria	222.1 ± 30.1	222.2 ± 36.9	0.979
	Retinopathy	226.8 ± 34.3	218.1 ± 32.1	0.044*
	Elevated PWV	223.2 ± 33.1	221.5 ± 33.6	0.698
Non-HDL cholesterol (mg/dL)	Microalbuminuria	182.7 ± 31.9	182.7 ± 37.9	0.989
	Retinopathy	187.5 ± 35.6	178.5 ± 33.7	0.045*
	Elevated PWV	183.4 ± 35.3	182.3 ± 34.7	0.821
Triglycerides (mg/dL)	Microalbuminuria	189.4 ± 57.3	179.0 ± 55.4	0.156
	Retinopathy	188.1 ± 54.3	181.2 ± 58.4	0.348
	Elevated PWV	180.5 ± 51.8	186.7 ± 59.1	0.416
TG/HDL ratio	Microalbuminuria	5.0 ± 1.9	4.7 ± 1.9	0.227

Lipid variable	TOD marker	With TOD (mean ± SD)	Without TOD (mean ± SD)	p-value
	Retinopathy	5.0 ± 1.8	4.8 ± 2.0	0.515
	Elevated PWV	4.8 ± 1.9	4.9 ± 1.9	0.467

Table 3. Correlation Analysis: Lipid Parameters and Continuous Target Organ Damage Measures

Lipid parameter	Echo LVMI r	Echo LVMI p	Urine ACR r	Urine ACR p	PWV r	PWV p
Total cholesterol	0.104	0.109	0.060	0.356	-0.007	0.912
LDL cholesterol	0.154	0.017	0.045	0.485	-0.035	0.589
HDL cholesterol	-0.089	0.167	0.031	0.631	0.042	0.522
Triglycerides	-0.067	0.302	0.089	0.172	0.054	0.411
Non-HDL cholesterol	0.121	0.071	0.073	0.270	-0.015	0.834
TG/HDL ratio	-0.055	0.415	0.102	0.129	0.038	0.563

Note: All correlations weak (|r| <0.2), most p>0.05, indicating minimal linear relationships between lipid variables and continuous TOD measures.

Figures And Visual Analysis

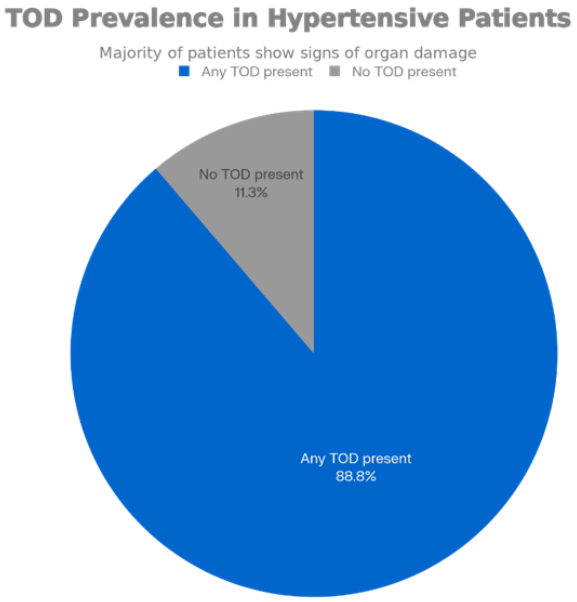

Figure 1. Target Organ Damage Prevalence in Hypertensive Cohort

Data for Figure 1:
- Any TOD present: 213/240 (88.8%) [Blue pie slice, large]
- No TOD present: 27/240 (11.3%) [Gray pie slice, small]

Legend: The majority of hypertensive patients demonstrated evidence of at least one target organ damage marker. Nearly 9 of every 10 patients had subclinical evidence of organ involvement.

Clinical significance: This high prevalence demonstrates the importance of comprehensive baseline organ evaluation in all hypertensive patients.

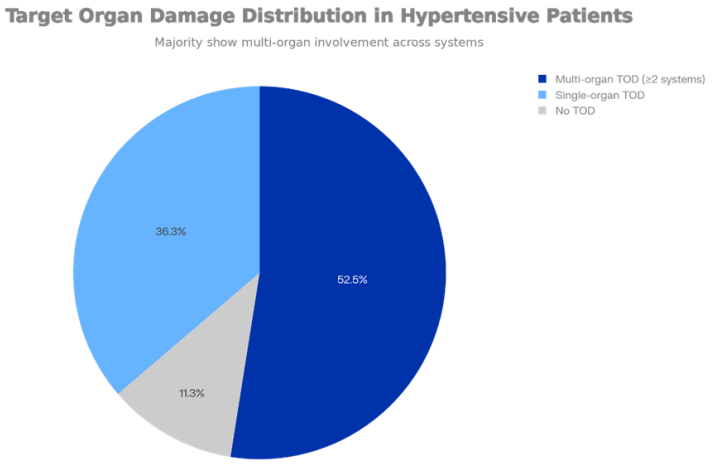

Figure 2. Distribution of Target Organ Involvement: Single versus Multi-Organ Patterns

Data for Figure 2:
- No TOD: 27/240 (11.3%) [Light gray slice]
- Single-organ TOD: 87/240 (36.3%) [Light blue slice]
- Multi-organ TOD (≥2 systems): 126/240 (52.5%) [Dark blue slice]

Legend: More than half of hypertensive patients exhibited involvement of two or more organ systems, indicating systemic vascular disease. Multi-organ involvement was identified in 52.5% of the cohort.

Clinical significance: The predominance of multi-organ damage justifies comprehensive evaluation and intensive risk reduction strategies.

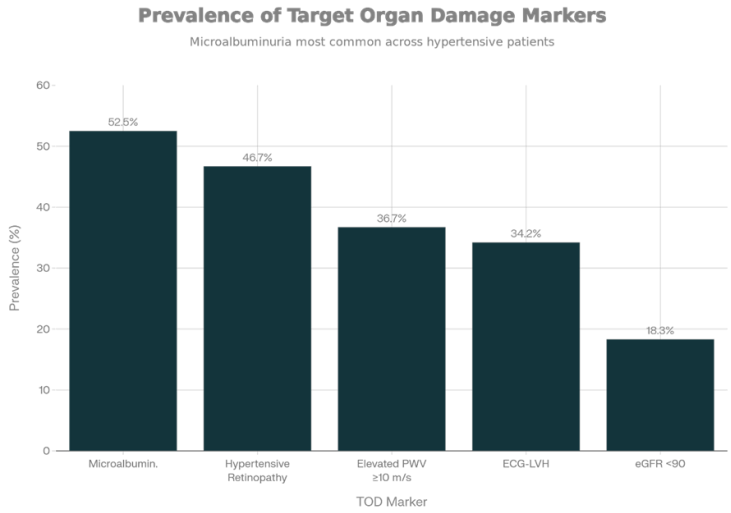

Figure 3. Prevalence of Individual Target Organ Damage Markers

Data for Figure 3 (bars in descending order by prevalence):
- Microalbuminuria: 126/240 (52.5%)
- Hypertensive Retinopathy: 112/240 (46.7%)
- Elevated PWV (≥10 m/s): 88/240 (36.7%)
- ECG-LVH (voltage criteria): 82/240 (34.2%)
- Reduced eGFR (<90): 44/240 (18.3%)

Y-axis scale: 0-60%
Bar color: Dark blue
Data labels: Percentage above each bar

Legend: Among individual organ systems, renal involvement (microalbuminuria in 52.5%) and retinal changes (46.7%) were most prevalent, followed by vascular stiffness markers and cardiac involvement. Approximately one-third of patients showed electrocardiographic evidence of left ventricular hypertrophy.

Clinical significance: Renal and retinal involvement are most frequent, highlighting importance of routine urinalysis and ophthalmologic examination.

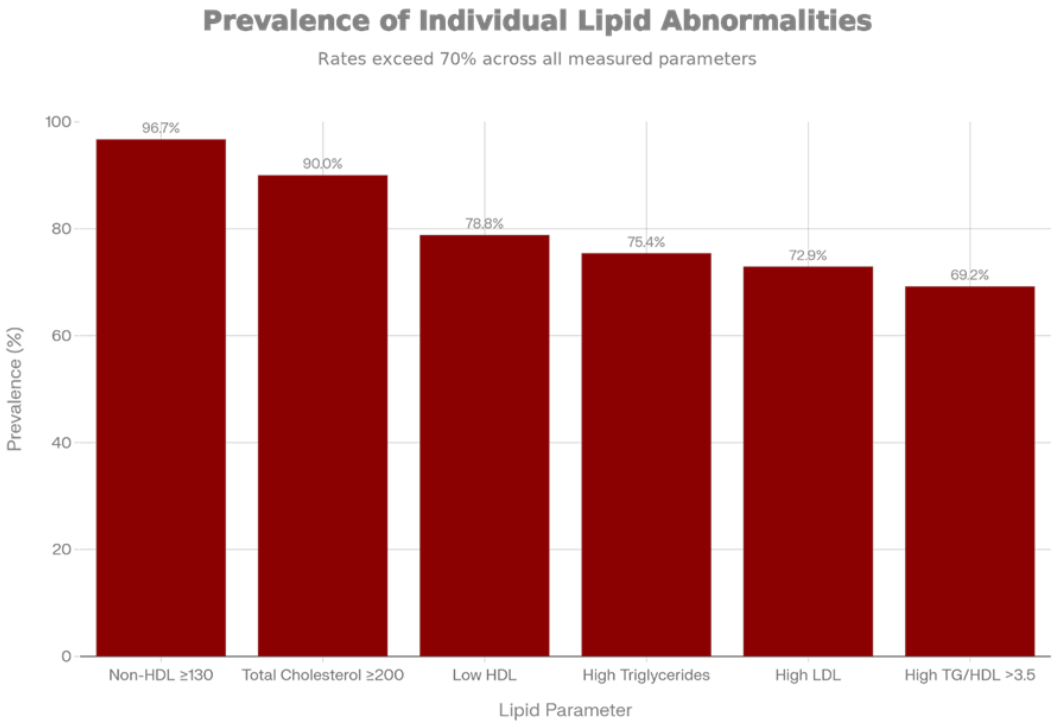

Figure 4. Prevalence of Individual Lipid Abnormalities

Data for Figure 4 (bars in descending order by prevalence):
- Non-HDL Cholesterol ≥130 mg/dL: 232/240 (96.7%)
- Total Cholesterol ≥200 mg/dL: 216/240 (90.0%)
- Low HDL Cholesterol: 189/240 (78.8%)
- High Triglycerides ≥150 mg/dL: 181/240 (75.4%)
- High LDL Cholesterol ≥130 mg/dL: 175/240 (72.9%)
- High TG/HDL Ratio >3.5: 166/240 (69.2%)

Y-axis scale: 0-100%
Bar color: Dark red/maroon
Data labels: Percentage above each bar

Legend: Nearly all hypertensive patients had at least one lipid abnormality, with near-universal dyslipidemia (99.2% had ≥1 abnormality). Non-HDL cholesterol elevation (96.7%) and total cholesterol elevation (90.0%) were most prevalent, followed by low HDL (78.8%) and elevated triglycerides (75.4%). An atherogenic triglyceride-to-HDL ratio (>3.5) was present in approximately 69% of the cohort.

Clinical significance: The atherogenic lipid phenotype (high triglycerides, low HDL, high non-HDL) predominates, indicating metabolic dysfunction amenable to lifestyle intervention and statin therapy.

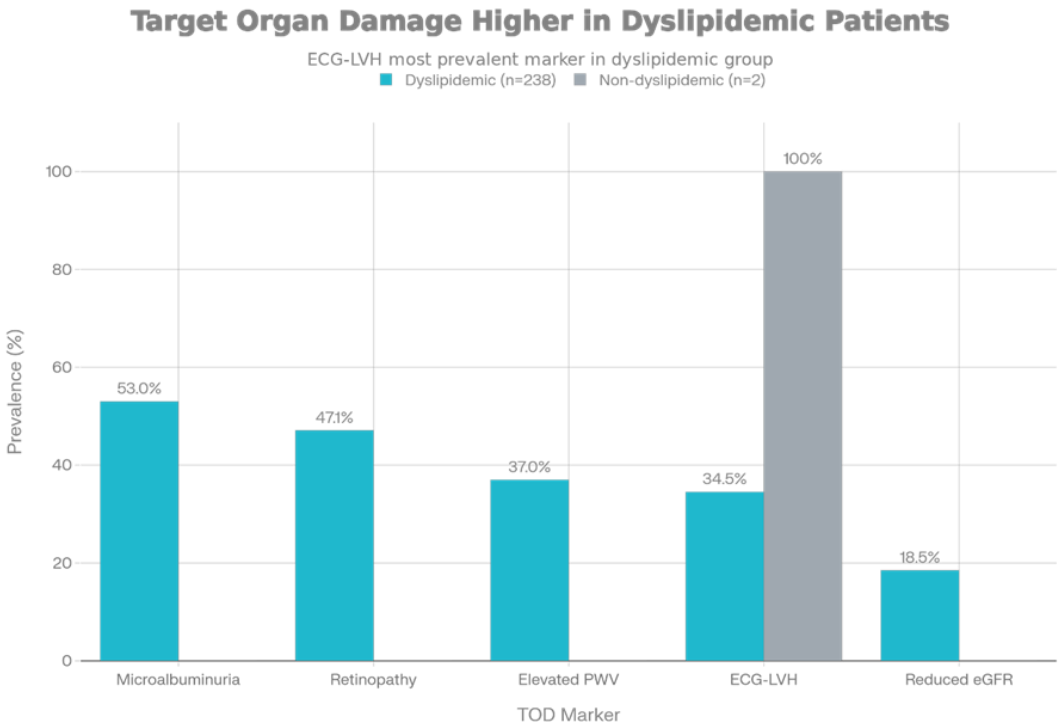

Figure 5. Target Organ Damage Prevalence Stratified by Dyslipidemia Status

Data for Figure 5:
Dyslipidemic patients (n=238):
- Microalbuminuria: 126/238 (53.0%)
- Retinopathy: 112/238 (47.1%)
- Elevated PWV: 88/238 (37.0%)
- ECG-LVH: 82/238 (34.5%)
- Reduced eGFR: 44/238 (18.5%)

Non-dyslipidemic patients (n=2):
- Microalbuminuria: 0/2 (0%)
- Retinopathy: 0/2 (0%)
- Elevated PWV: 0/2 (0%)
- ECG-LVH: 2/2 (100%)
- Reduced eGFR: 0/2 (0%)

Y-axis scale: 0-100%
Grouped bars: Blue (dyslipidemic) and light gray (non-dyslipidemic)
Data labels: Percentage above bars

Legend: Comparison of target organ damage prevalence between dyslipidemic and non-dyslipidemic patients. While dyslipidemic patients showed similar or slightly higher prevalence across most markers, the extremely limited number of non-dyslipidemic patients (n=2) restricted meaningful subgroup analysis.

Nevertheless, the very high prevalence of TOD in both groups indicates that blood pressure elevation remains the dominant determinant of organ damage.

Clinical significance: The rarity of normolipidemia in hypertension highlights the strong coupling between these conditions ("lipitension").

Summary Of Visual Epidemiological Analysis

Figure Type	Purpose	Clinical Application
Pie Charts (Figures 1-2)	Rapid communication of proportions; disease burden visual at a glance	Patient education Clinician training Public health messaging
Bar Graphs (Figures 3-4)	Comparison of prevalence across multiple markers and abnormalities	Identify most common complications Prioritize screening
Grouped Bars (Figure 5)	Subgroup comparisons demonstrating disease pattern differences	Risk stratification Identify high-risk populations

This comprehensive visual epidemiological analysis provides multiple formats for communicating complex hypertension data to diverse professional and patient audiences.