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Serum Neutrophil Gelatinase-Associated Lipocalin (NGAL) as an Early Biomarker of Hypertensive Nephropathy: A Case-Control Study

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Abstract: **Background:** An estimated 1.28 billion people worldwide suffer from hypertension, which often causes subclinical renal and cardiovascular damage prior to its detection using conventional parameters such as serum creatinine. Neutrophil gelatinase-associated lipocalin (NGAL), which is secreted by tubular epithelial and immune cells upon inflammation and tubular stress, has been proposed to be an early indicator of renal injury associated with hypertension. **Objective:** To compare serum NGAL levels between hypertensive patients and healthy controls, and to evaluate its association with blood pressure, lipid profile, and standard renal indices. **Methods:** This case-control study was conducted by the Departments of Biochemistry and Medicine, and comprised 200 participants aged 40 years and older; 100 hypertensive patients and 100 age-matched healthy controls. Blood pressure, fasting glucose, lipid profile, urea, creatinine and serum NGAL were measured in all participants, while Spearman's rank correlation was used to assess the associations between NGAL and the rest of the parameters. **Results:** Serum NGAL was found to be significantly higher among hypertensive patients (98.80 ± 13.29 ng/mL) as compared to the controls (81.55 ± 5.25 ng/mL; $p < 0.001$), with similar patterns seen in both genders. NGAL was positively correlated with systolic blood pressure ($rs = 0.519$), diastolic blood pressure ($rs = 0.480$), and total cholesterol ($rs = 0.392$, all $p < 0.001$), as well as triglycerides, VLDL and LDL ($p < 0.01$), while serum creatinine was found to be similar between the two groups. **Conclusion:** The increased serum NGAL observed among hypertensive patients with normal conventional renal parameters, as well as its association with blood pressure and atherogenic lipids, suggests that it could be used as an early, sex-independent, biomarker of subclinical hypertensive nephropathy.

Keywords: Neutrophil gelatinase-associated lipocalin (NGAL); Hypertension; Chronic kidney disease; Systolic blood pressure; Diastolic blood pressure.

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INTRODUCTION

Hypertension is frequently asymptomatic, and this silent course allows it to persist undetected for years before culminating in cardiovascular disease and irreversible damage to target organs. An increasing amount of evidence suggests that chronic inflammation and dysregulated immune responses play a significant role in the development and progression of the disease [1]. Since early detection is crucial in preventing further complications, many studies have been dedicated to identifying biomarkers that can predict organ damage caused by high blood pressure in advance. Almost one in every four Indian adults has hypertension, with the highest pooled prevalence found in northern India; while the condition has typically been concentrated in urban populations due to less active lifestyles and changes in eating habits, rural prevalence is on the rise with lifestyle transitions and increased screening in the community [2]. Given that hypertension and type 2 diabetes together drive much of India's kidney disease burden, targeted screening of these high-risk groups has been recommended as a practical strategy for curbing the

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expected rise in impaired kidney function [3]. Across the globe, kidney damage caused by high blood pressure is still one of the main reasons people develop chronic kidney disease, affecting over one billion individuals with elevated blood pressure [4].

Serum creatinine, a traditional indicator of kidney function, increases only after a significant decrease in the glomerular filtration rate, limiting its application for the early diagnosis of renal dysfunction [5]. Neutrophil gelatinase-associated lipocalin (NGAL), a 25-kDa glycoprotein produced by neutrophils and expressed in renal, cardiac, and other epithelial cells, may be detected in serum, urine, and other body fluids and increases earlier in response to tubular stress [6,7]. In acute kidney injury, circulating NGAL is correlated with severity and is predictive of mortality [8] and, unlike creatinine, whose levels are affected by muscle mass, sex, and medication use, it seems to be more sensitive to early tubular compromise [9,10].

This study therefore evaluated serum NGAL in hypertensive patients relative to healthy controls and examined its correlation with blood pressure, lipid profile, and conventional renal parameters to clarify its value in detecting subclinical hypertensive nephropathy.

MATERIALS AND METHODS

Study Design and Setting

This case-control study was carried out jointly by the Departments of Biochemistry and Medicine at Sri Guru Ram Das Institute of Medical Sciences and Research, Sri Amritsar, India.

Ethical Considerations

The Institutional Ethics and Research Committee of Sri Guru Ram Das University of Health Sciences, Sri Amritsar approved the protocol (Approval No. SGRD/IEC/2024-288). The participants were informed about the process beforehand, and their voluntary participation without any adverse effects was ensured. In addition, the written consent of all participants was acquired before enrolment.

Sample Size Determination

The sample size was estimated using the data provided by Springer *et al*. [11] that demonstrated a significant increase in serum NGAL levels among hypertensive patients compared to normotensive ones (109.2 ± 48.9 ng/mL vs. 91.9 ± 29.4 ng/mL; p = 0.04). Using the following formula $n = (Z\alpha/2 + Z\beta)^2 \times SD^2 \times 2 / d^2$, with such assumptions as a pooled SD equal to 39.15, a mean difference of 17.3, and an effect size of 0.442, GPower (version 3.1) suggested that 82 participants per group are required at $\alpha = 0.05$ and 80% power. However, enrolling 100 participants per group was proposed to meet the demands of the study.

Study Population

The study involved participants aged above 40 years selected from the outpatient department by a non-probability consecutive sampling technique. There were two groups of patients: the hypertensive group consisting of 100 people randomly selected from those diagnosed with hypertension at the outpatient department of the facility. The diagnosis was made based on the JNC criteria for hypertension; hence, patients with a systolic blood pressure of ≥140 mmHg and/or a diastolic blood pressure of ≥90 mmHg were included. The control group was also made up of 100 people selected from the outpatients' department. It comprised adults of the same age as the hypertensive group but without hypertension. Patients with liver disease, hepatitis B and C, diabetes mellitus, thyroid problems, UTIs, HIV, tuberculosis, chronic or acute kidney disease, and cancer were excluded from the study.

Sample Collection and Processing

To decrease circadian and hydration variability, venous blood samples were collected between 0800 and 1100 h after an overnight fast of at least 8 h followed by 15 min of sitting. Blood was collected in plain tubes (4 ml), left to clot for 30 min at room temperature, then centrifuged at 3500 rpm for 17 min, and the serum was stored at -80 degrees C until measurement of NGAL. Another 2 ml of blood was collected in EDTA tubes and centrifuged at 4000 rpm for 5 min, and the plasma was stored at -20 degrees C for analysis of glucose.

Biochemical And Immunoassay Analysis

Fasting glucose was measured by the glucose oxidase–peroxidase method [12]; urea by the urease method and total cholesterol and triglycerides by enzymatic methods [13]; creatinine by the modified Jaffe method and HDL by an enzymatic method [14]; LDL was calculated using the Friedewald formula (LDL-C = TC − HDL-C − TG/5) and VLDL as TG/5 [15]. Serum NGAL was quantified by ELISA [16]; intra- and inter-assay coefficients of variation ranged from 5.1–5.33% and 5.1–5.26%, respectively, both within accepted precision limits (<10% intra-assay, <15% inter-assay).

Statistical Analysis

The data were entered into the Microsoft Excel spreadsheet and analyzed using SPSS version 23.0. The normality of the data distribution was evaluated using the Shapiro–Wilk test. The independent-samples t-test was applied to assess the intergroup differences for variables that had a normal distribution, whereas for non-normal distributions, the Mann–Whitney U test was used. The chi-square test was used to compare categorical variables between groups. Associations between NGAL and other variables were examined using Spearman's rank correlation coefficient. A two-tailed $p < 0.05$ was considered statistically significant.

RESULTS

Two hundred participants of either sex, aged over 40 years, were enrolled: 100 hypertensive patients (Group 1) and 100 healthy controls (Group 2). Systolic and diastolic blood pressure, fasting glucose, lipid profile,

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serum urea, serum creatinine, and serum NGAL were assessed in all participants.

Table 1. Sex-wise comparison of serum NGAL between hypertensive and control groups

Sex	N (HTN)	Hypertensive Mean ± SD (ng/mL)	N (Control)	Control Mean ± SD (ng/mL)	p-value
Female	48	98.81 ± 13.52	57	81.44 ± 5.40	< 0.001**
Male	52	98.79 ± 13.21	43	81.69 ± 5.09	< 0.001**

***p < 0.001, highly significant; Mann–Whitney U test applied within each sex subgroup.*

Serum NGAL was significantly higher in hypertensive patients than controls within both sexes (Table 1). Among females (control n = 57; hypertensive n = 48), mean NGAL was 98.81 ± 13.52 ng/mL in the hypertensive subgroup versus 81.44 ± 5.40 ng/mL in controls ($p < 0.001$); among males (control n = 43; hypertensive n = 52), corresponding values were 98.79 ± 13.21 ng/mL and 81.69 ± 5.09 ng/mL ($p < 0.001$). Figure 1 B illustrates the comparable magnitude of elevation in both sexes.

Table 2. Demographic and anthropometric comparison between hypertensive and control groups

Parameter	Hypertensive (n=100) Mean ± SD	Control (n=100) Mean ± SD	p-value
Age (years)	57.70 ± 9.08	49.00 ± 6.45	< 0.001*
Height (cm)	162.45 ± 6.87	160.75 ± 6.56	0.054
Weight (kg)	65.33 ± 11.63	66.42 ± 11.43	0.506
SBP (mmHg)	140.31 ± 13.24	112.68 ± 6.30	< 0.001*
DBP (mmHg)	90.61 ± 7.59	73.58 ± 4.72	< 0.001*

**p < 0.05 considered statistically significant. Mann–Whitney U test applied for non-normally distributed variables. SBP = systolic blood pressure; DBP = diastolic blood pressure.*

Hypertensive patients were significantly older than controls (57.70 ± 9.08 vs 49.00 ± 6.45 years, $p < 0.001$), while height and weight did not show significant differences between groups (Table 2). As expected, systolic and diastolic blood pressure were substantially higher in the hypertensive group (both $p < 0.001$).

Table 3. Biochemical parameters in hypertensive and control groups

Parameter	Hypertensive (n=100) Mean ± SD	Control (n=100) Mean ± SD	p-value
Fasting glucose (mg/dL)	96.27 ± 7.06	94.40 ± 9.04	0.107
Serum urea (mg/dL)	28.00 ± 6.35	25.96 ± 6.64	0.029*
Serum creatinine (mg/dL)	0.92 ± 0.19	0.91 ± 0.18	0.650
Total cholesterol (mg/dL)	215.63 ± 27.02	186.01 ± 16.49	< 0.001*
HDL (mg/dL) **	52.37 ± 10.27	53.75 ± 7.59	0.283
Triglycerides (mg/dL)	195.51 ± 55.65	155.15 ± 33.81	< 0.001*
VLDL (mg/dL)	38.81 ± 11.21	31.03 ± 6.76	< 0.001*
LDL (mg/dL)	114.54 ± 17.70	101.23 ± 17.76	< 0.001*

**p < 0.05 considered statistically significant. **HDL was normally distributed (Shapiro–Wilk p > 0.05); independent-samples t-test applied. Mann–Whitney U test applied for other, non-normally distributed variables. VLDL = very low-density lipoprotein; LDL = low-density lipoprotein; HDL = high-density lipoprotein.*

Fasting glucose, serum creatinine, and HDL were comparable between groups, whereas serum urea was modestly but significantly higher in hypertensive patients (28.00 ± 6.35 vs 25.96 ± 6.64 mg/dL, $p = 0.029$) (Table 3). Serum creatinine levels did not differ significantly (0.92 ± 0.19 vs 0.91 ± 0.18 mg/dL; Mann–Whitney $U = 4814.5$, $Z = -0.453$, $p = 0.650$). Total cholesterol, triglycerides, VLDL, and LDL were all significantly higher in hypertensive patients ($p < 0.001$ for each).

Table 4. Serum NGAL levels in hypertensive and control groups

Parameter	Hypertensive (n=100) Mean ± SD	Control (n=100) Mean ± SD	p-value
Serum NGAL (ng/mL)	98.80 ± 13.29	81.55 ± 5.25	< 0.001*

**Mann–Whitney U test, U = 1162.0, Z = −9.378, p < 0.001.*

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Mean serum NGAL was significantly higher in hypertensive patients (98.80 ± 13.29 ng/mL) than in controls (81.55 ± 5.25 ng/mL; U = 1162.0, Z = −9.378, p < 0.001) (Table 4, Figure 1A), confirming markedly elevated NGAL in hypertension.

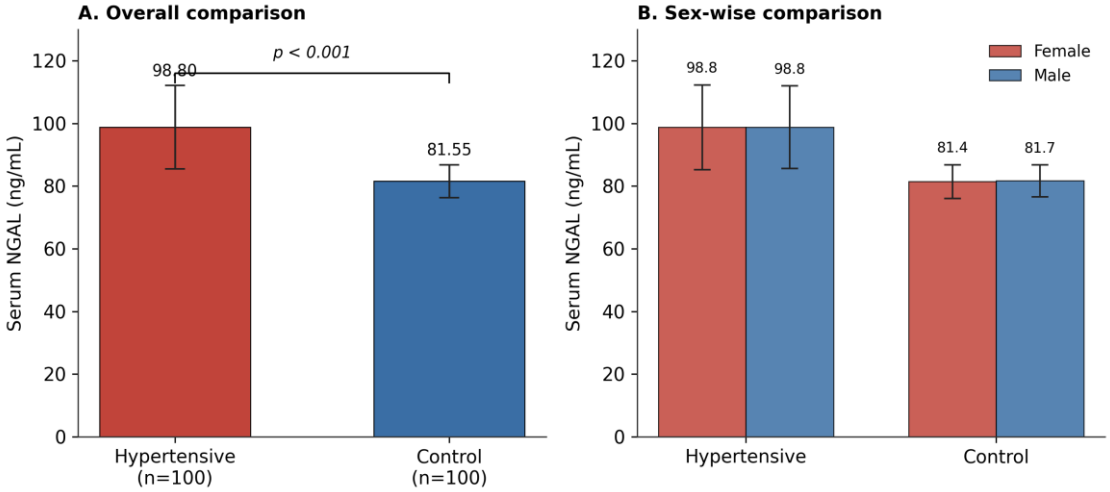

Figure 1. Serum NGAL levels (mean ± SD) in hypertensive and control groups (A) Overall comparison between groups. (B) Comparison stratified by sex, revealing comparable elevation in male and female hypertensive patients relative to their respective controls.

Table 5. Spearman's correlation of serum NGAL with clinical and biochemical parameters (n = 200)

Parameter	rs (NGAL)	p-value
Age (years)	0.196**	0.005
Height (cm)	0.093	0.189
Weight (kg)	−0.040	0.577
SBP (mmHg)	0.519*	< 0.001
DBP (mmHg)	0.480*	< 0.001
Fasting glucose (mg/dL)	0.013	0.855
Serum urea (mg/dL)	0.119	0.094
Serum creatinine (mg/dL)	0.032	0.657
Total cholesterol (mg/dL)	0.392*	< 0.001
HDL (mg/dL)	0.003	0.968
Triglycerides (mg/dL)	0.233**	0.001
VLDL (mg/dL)	0.221**	0.002
LDL (mg/dL)	0.205**	0.004

**Correlation significant at the 0.05 level (2-tailed). **Correlation significant at the 0.01 level (2-tailed). rs = Spearman's correlation coefficient.*

On Spearman correlation analysis across the full cohort, NGAL correlated positively with age (rs = 0.196, p = 0.005), systolic blood pressure (rs = 0.519, p < 0.001), diastolic blood pressure (rs = 0.480, p < 0.001), total cholesterol (rs = 0.392, p < 0.001), triglycerides (rs = 0.233, p = 0.001), VLDL (rs = 0.221, p = 0.002), and LDL (rs = 0.205, p = 0.004) (Table 5). No significant correlation emerged with height, weight, fasting glucose, urea, creatinine, or HDL.

DISCUSSION

Hypertension is still a leading contributor to cardiovascular and renal morbidity globally, and a growing body of literature links it with subclinical inflammation, oxidative stress and endothelial injury occurring prior to overt organ damage [17]. NGAL, which reflects the aforementioned processes, was significantly elevated in hypertensive patients irrespective of sex within the present cohort, and this finding is in keeping with Bonnard *et al*. [18], who associated increased NGAL expression with hypertension-related renal dysfunction regardless of gender, and which implies NGAL acts as a sex-independent marker of early renal injury. Gupta *et al*. [19] have also reported a balanced sex distribution among comparable case-control groups.

Height and weight, fasting glucose and creatinine were found to be similar between groups, indicating that the biochemical alterations detected were due to hypertension rather than obesity, overt diabetes, or an advanced phase of renal impairment, an observation

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which can be paralleled with that of Tripathy *et al*. [20] in newly diagnosed hypertensive patients. Serum urea was modestly but significantly elevated among hypertensive patients despite an absence of change in creatinine, indicating renal involvement at a stage preceding conventional detection. Kaul *et al*. [21] have also documented progressive elevations in renal injury biomarkers alongside worsening blood pressure [22].

The rise in serum NGAL levels among hypertensive patients despite normal creatinine values further supports its role as an early biomarker of tubular stress, as proposed by Danquah *et al*., [6], who also observed increased levels of plasma NGAL among the hypertensive patients. The previous studies by Buonafine *et al*., [17] and Kumela Goro *et al*., [23] also reported increased levels of NGAL despite no change in creatinine. The latter authors attributed this to subclinical tubular injury and inflammatory activity occurring ahead of any measurable loss of filtration capacity. Within the present series, NGAL correlated positively with age, systolic and diastolic pressure, and atherogenic lipid parameters, reflecting findings by Danquah *et al*. [6] which associate elevated NGAL and creatinine with worse blood pressure control and glycaemic status.

Mechanistically, persistent pressure overload and the activation of the renin-angiotensin-aldosterone system are postulated to promote tubulo-interstitial ischaemia; Angiotensin II-induced NADPH-oxidase activity increases levels of reactive oxygen species, as well as activates NF-κB and TGF-β signalling pathways within renal tissue, and this in turn stimulates tubular NGAL synthesis [7]. Unlike the urinary albumin-to-creatinine ratio, which reflects glomerular capillary leak, NGAL indicates tubular injury which may occur ahead of any albuminuria altogether [24]; more than half of patients with raised NGAL show no concurrent rise in albuminuria [25], making NGAL a complementary rather than replacement marker for early onset of hypertensive nephropathy [26].

Because we excluded patients with preexisting chronic kidney disease, both urea and creatinine were within the normal range, despite the slight increase in urea that may have limited our ability to identify associations with conventional renal indexes. However, the distinct rise in NGAL against the normal background of creatinine can be used as a sensitive indicator of subclinical tubular damage beyond the limits of conventional renal function tests.

Limitations

This study was conducted at a single tertiary-care centre, which may limit generalisability. NGAL was measured at a single time point without longitudinal follow-up, so we could not assess its predictive value for future chronic kidney disease. Because patients with pre-existing kidney disease or deranged renal function were excluded, results are applicable only to hypertensive patients with preserved renal function. Other emerging renal biomarkers, including kidney injury molecule-1, cystatin C, and fatty acid-binding protein, were not evaluated for comparison.

CONCLUSION

Serum NGAL was significantly higher in hypertensive patients than in controls, despite normal conventional renal indices including urea and creatinine, and positively correlated with age, blood pressure, and atherogenic lipid parameters. These results suggest that serum NGAL is a promising sex-independent early biomarker for subclinical hypertensive nephropathy that may facilitate earlier detection and surveillance of at-risk patients when used alongside routine clinical and biochemical evaluation.

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