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Inflammatory and nutritional indices in patients with pheochromocytoma

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ABSTRACT

Aims: Pheochromocytoma is characterized by catecholamine excess, which may influence immune regulation and metabolic pathways. However, the relationship between catecholamine activity, systemic inflammation, and immune-nutritional status remains incompletely understood. This study aimed to evaluate inflammatory and nutritional indices in patients with pheochromocytoma and to investigate their association with tumor size and plasma metanephrine levels.

Methods: In this retrospective study, 39 patients with pheochromocytoma were compared with 24 age-, sex-, and body mass index-matched healthy control subjects. Complete blood count parameters and inflammation-based indices, including neutrophil-to-lymphocyte ratio (NLR), Systemic Immune-inflammation Index (SII), Prognostic Nutritional Index (PNI), and Controlling Nutritional Status (CONUT) score, were analyzed. Correlation analyses were performed to assess the relationships between inflammatory indices, plasma metanephrine levels, and tumor size.

Results: Patients with pheochromocytoma had significantly higher WBC, neutrophil, and platelet counts than controls. SII was significantly elevated, whereas NLR, CONUT, and PNI did not differ between groups. Fasting glucose levels were higher in the pheochromocytoma group, while lipid parameters were comparable. Correlation analyses demonstrated PNI was negatively correlated with both tumor size and plasma metanephrine levels. SII showed positive correlations with NLR and PNI, and CONUT was positively correlated with NLR.

Conclusion: Pheochromocytoma is associated with systemic inflammatory activation, as reflected by elevated leukocyte parameters and increased SII. Although overall nutritional indices were not significantly altered, the inverse association between PNI and tumor burden suggests that catecholamine excess may adversely affect immune-nutritional status. These findings highlight the complex immune-metabolic interplay in pheochromocytoma.

Keywords: Pheochromocytoma, catecholamines, Systemic Immune-inflammation Index, Prognostic Nutritional Index, controlling nutritional status

INTRODUCTION

Pheochromocytomas are catecholamine-producing neuroendocrine tumors arising from chromaffin cells of the adrenal medulla.[1] Excess catecholamine secretion leads to increased sympathetic activity and results in characteristic clinical manifestations such as paroxysmal sweating, tachycardia, and flushing. In addition to these symptoms, sustained catecholamine excess contributes to significant metabolic complications, including hypertension, diabetes mellitus, and dyslipidemia.[2,3,4]

Beyond their well-established cardiovascular and metabolic effects, accumulating evidence indicates that catecholamines also exert modulatory effects on modulate the immune system. Catecholamines modulate the production of immune mediators in a receptor-dependent manner.[5] Stimulation of α-adrenergic receptors is generally associated with immunostimulatory effects, whereas activation of β-adrenergic receptors predominantly exerts immunosuppressive actions. When both α- and β-adrenergic receptors are stimulated simultaneously, β-adrenergic effects typically predominate. In addition to their effects on cytokine production, catecholamines influence both the number and function of immune cells. β-adrenergic stimulation is associated with lymphocytosis, while α-adrenergic activation promotes lymphocyte redistribution and tissue homing.[6]

These receptor-mediated alterations in immune cell distribution and function may be reflected in peripheral

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blood inflammatory markers. Changes in neutrophil and lymphocyte counts can be quantified using inflammation-based indices such as the and the neutrophil-to-lymphocyte ratio (NLR) and the Systemic Immune-inflammation Index (SII), which have been widely investigated as indicators of systemic inflammatory status.[7,8]

Furthermore, systemic inflammation is closely linked to nutritional and metabolic status. Indices such as the Prognostic Nutritional Index (PNI) and the Controlling Nutritional Status (CONUT) score integrate immune and nutritional parameters and have been proposed as markers of disease severity in various malignancies. Lower PNI values have been associated with increased inflammatory burden, impaired immune response, and unfavorable clinical outcomes. Higher CONUT scores have been associated with poor nutritional status, increased inflammatory burden, and adverse clinical outcomes. [9,10,11]

Despite growing interest in inflammation-based indices in endocrine tumors, the relationship among catecholamine activity, inflammatory indices, and nutritional status in pheochromocytoma remains unclear.

Therefore, the present study aimed to evaluate systemic inflammatory indices and nutritional parameters in patients with pheochromocytoma and to investigate their associations with tumor size, biochemical catecholamine activity. and histopathological features, including the Pheochromocytoma of the Adrenal Gland Scaled Score (PASS).

METHODS

Ethics

This study was conducted with the approval of the Scientific and Ethical Review Board for Medical Researches No. 2 at Ankara Bilkent City Hospital (Date: 20.08.2025, Decision No: TABED2-25-1442). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Participants

The records of 39 patients histopathologically diagnosed with pheochromocytoma, who were admitted to Ankara Bilkent City Hospital's Endocrinology clinic between January 2, 2019, and June 30, 2025, were retrospectively examined. Patients aged 18 and above were included in the study. The control group consisted of individuals who attended our hospital for routine clinical evaluation and had no history of chronic disease or regular medication use. Participants with a history of alcohol consumption were excluded to eliminate potential confounding factors. Patients with active infection, chronic inflammatory disease, autoimmune disorders, malignancy other than pheochromocytoma, or current corticosteroid therapy were excluded to minimize potential confounding effects on inflammatory markers. Twenty-four control subjects were matched with the patient group according to age, sex, and body-mass index (BMI).

Procedure

Demographic data, including age and sex, were recorded for all participants. Anthropometric measurements, including height and weight, were obtained at the time of evaluation. BMI was calculated as weight in kilograms divided by the square of height in meters (kg/m^2).

Biochemical parameters were analyzed from fasting blood samples. In addition, catecholamine profiles, including plasma metanephrine, plasma normetanephrine, urinary metanephrine, and urinary normetanephrine levels, were examined.

The lesion size of pheochromocytoma was recorded. Only patients with postoperative histopathological confirmation of pheochromocytoma were included in the study. The PASS was recorded for each patient.

Biochemical Analysis

Fasting venous blood samples were obtained in the morning after following an overnight fast. Fasting plasma glucose (70-99 mg/dl), liver function tests, triglycerides (<150 mg/dl), high-density lipoprotein cholesterol (HDL-C >50 mg/dl), very low-density lipoprotein cholesterol (VLDL-C, 10-40 mg/dl), low-density lipoprotein cholesterol (LDL-C, 100-129 mg/dl), and total cholesterol (<200 mg/dl), levels were measured in all participants.

Plasma glucose and liver function tests were analyzed using a spectrophotometric method (Siemens Atellica, Siemens Healthineers, Germany). Hormone profiles were measured in the morning using the Siemens Atellica IM 1600 system with a chemiluminescent immunoassay method. Twenty-four-hour urinary catecholamines were analyzed by liquid chromatography–tandem mass spectrometry (LC-MS/MS) using the SCIEX 5500 and 5500 PLUS systems.

Complete blood count (CBC) parameters, including neutrophil, lymphocyte, monocyte, and platelet count, were measured using an automated hematology analyzer based on impedance and flow cytometry principles. All measurements were performed according to the manufacturer's instructions and standard laboratory quality control procedures.

The neutrophil-to-lymphocyte ratio (NLR) was calculated as the ratio of absolute neutrophil count to absolute lymphocyte count. The SII was calculated as platelet count×neutrophil count/lymphocyte count.

The Prognostic Nutritional Index (PNI) was calculated using the following formula:

PNI=(10×serum albumin [g/dl])+(0.005×total lymphocyte count [/mm^3]).

The Controlling Nutritional Status (CONUT) score was calculated based on serum albumin, total lymphocyte count, and total cholesterol levels. Serum albumin was scored as 0 (≥3.5 g/dl), 2 (3.0-3.49 g/dl), 4 (2.5-2.99 g/dl), or 6 (<2.5 g/dl). Total lymphocyte count was scored as 0 (≥1600/mm^3), 1 (1200-1599/mm^3), 2 (800-1199/mm^3), or 3 (<800/mm^3). Total cholesterol was scored as 0 (≥180 mg/dl), 1 (140-179 mg/dl), 2 (100-139 mg/dl), or 3 (<100 mg/dl). The total CONUT score

was obtained by summing the individual scores. Nutritional status was classified as normal (0-1 points), mild malnutrition (2-4 points), moderate malnutrition (5-8 points), or severe malnutrition (9-12 points). The CONUT score was calculated according to the original description by Ignacio de Ulíbarri et al.[10]

Radiological Evaluation

All abdominal computed tomography (CT) scans were performed using a 128-slice CT scanner (GE Revolution EVO, GE Medical Systems, Milwaukee, WI, USA), with patients lying supine during full inspiration and without the administration of intravenous contrast. The scanning protocol included a tube voltage of 120 kV and an automated tube current modulation system. The pitch factor was set at 0.98. Axial images were reconstructed with a slice thickness of 1.3 mm using a sharp convolution kernel.

All abdominal CT scans were performed using a 128-slice CT scanner (GE Revolution EVO, GE Medical Systems, Milwaukee, WI, USA). Patients were scanned in the supine position during full inspiration without the administration of intravenous contrast. CT images were used for tumor localization and size assessment.

Adrenal Lesion Evaluation

Tumor size was defined as the largest axial diameter. Attenuation measurements were obtained by positioning regions of interest (ROIs) within the most homogeneous solid portion of the lesion, avoiding areas of necrosis, calcification, or cystic change. Morphological evaluation was based on the presence of necrosis, calcification, and cyst components, as determined by visual inspection and attenuation characteristics.

Statistical Analysis

Quantitative variables were tested for normality. Variables with non-normal distribution were presented as median (minimum–maximum), whereas normally distributed variables were expressed as mean±standard deviation. Comparisons between two groups were performed using the Student's t-test for normally distributed variables and the Mann-Whitney U test for non-normally distributed variables. Categorical variables were compared using the Chi-square test. Correlation analyses between quantitative variables were conducted using Pearson or Spearman correlation coefficients, depending on the data distribution. A p-value<0.05 was considered statistically significant.

RESULTS

A total of 39 patients with histopathologically confirmed pheochromocytoma and 24 matched control subjects were included in the study. There were no significant differences between the groups in terms of age, sex, and BMI. The mean age was comparable between the groups: 49.46±12.30 years in the pheochromocytoma group and 46.96±11.30 years in the control group. There was no significant difference between the pheochromocytoma and control groups in terms of sex distribution (female: 51.3% vs. 41.7%, p=0.458).

The mean WBC count was significantly higher in the pheochromocytoma group compared to controls (p=0.018). In addition, the median neutrophil count was significantly elevated in patients with pheochromocytoma (p=0.042). Lymphocyte count, monocyte count, and hemoglobin levels did not differ significantly between the groups. However, platelet (PLT) levels were significantly higher in the pheochromocytoma group compared to the control group (p=0.003).

There were no significant differences between the groups in terms of CONUT score, PNI, or NLR. However, the SII was significantly elevated in the pheochromocytoma group (p=0.024).

Fasting glucose, lipid profile parameters, and liver function tests were compared between the groups. Fasting glucose levels were significantly elevated in the pheochromocytoma group (p=0.048). No significant differences were observed between the groups in terms of lipid profiles. Comparison of age, BMI, and laboratory characteristics of the groups is presented in **Table 1**.

There were no significant differences between the groups in terms of obesity (30.8% vs 29.2%) and overweight (38.5% vs 37.5%).

Correlation analysis demonstrated a significant negative correlation between PNI and tumor size (r=-0.368, p=0.023). In addition, PNI was negatively correlated with plasma metanephrine levels (r=-0.332, p=0.045). Correlation analysis demonstrated that SII was significantly positively correlated with NLR and PNI (r=0.802, p=0.000; r=0.249, p=0.043, respectively). A significant positive association was observed between CONUT score and NLR (p=0.001). The findings of the correlation analyses are summarized in **Table 2**.

DISCUSSION

In this study, patients with pheochromocytoma exhibited significantly elevated WBC, neutrophil, and platelet counts compared to matched controls. This hematological profile suggests a low-grade systemic inflammatory activation potentially associated with catecholamine excess.

Zelinka et al.[12] compared patients with pheochromocytoma (n=15), primary hyperaldosteronism (n=18), essential hypertension (n=17), and healthy controls. They reported significantly higher leukocyte, neutrophil, and platelet counts in patients with pheochromocytoma. Notably, these hematological parameters normalized after adrenalectomy. The authors suggested that chronic catecholamine exposure increases inflammatory markers and that this inflammatory state is reversible following tumor removal. These findings support the concept that catecholamine excess may have a reversible immunomodulatory effect.

Although leukocyte and neutrophil counts were significantly elevated in patients with pheochromocytoma, NLR did not show a significant difference between the groups. This finding suggests that while absolute neutrophil counts increased, the relative balance between neutrophils and lymphocytes may have been preserved. possibly due to stable lymphocyte

Table 1. Comparison of age, BMI, and laboratory characteristics of the groups

	Pheochromocytoma (n=39)	Control (n=24)	t or U	p
Age (years)	50±11	47±12	0.683	0.498
BMI (kg/m²)	27.6±5	27.3±3.9	0.398	0.692
Glucose (mg/dl) (M (IQR))	94 (15)	89 (11)	-2.033	0.048*
Albumin (g/L)	45±3.6	45±3.2	-0.015	0.879
WBC	7.7±1.8	6.7±1.3	2.437	0.018*
Neutrophil (×10⁹/L), (M (IQR))	4.9 (2.1)	4 (1.7)	-2.033	0.042*
Lymphocyte (×10⁹/L), (M (IQR))	2.00 (0.7)	1.8 (0.3)	1.086	0.282
Monocyte (×10⁹/L), (M (IQR))	0.4 (0.1)	0.3 (0.1)	1.544	0.128
hemoglobin	13.7±1.6	14.4±1.1	-1.895	0.063
Platelet (M (IQR))	290 (59)	243 (53)	3.084	0.003*
ALT (U/L) (M (IQR))	27(15)	20(14)	-1.599	0.110
LDL-C (mg/dl)	110±35	126±31	-1.928	0.054
TG (mg/dl) (M (IQR))	119 (151)	123 (66)	-0.598	0.550
VLDL-C (mg/dl) (M (IQR))	24(30)	24 (13)	-0.569	0.570
HDL-C (mg/dl) (M (IQR))	43(19)	43 (11)	-0.222	0.825
Total Chol (mg/dl)	187±40	198±34	-1.155	0.253
CONUT (M (IQR))	1.00 (1)	0.00 (1)	-1.312	0.190
PNI	450±35	452±32	-0.234	0.816
SII (M (IQR))	709 (397)	531 (257)	-2.254	0.024*
NLO (M (IQR))	2.54 (1.04)	2.23 (1.24)	-0.867	0.386

BMI: Body-mass index, WBC: White blood cell count, ALT: Alanine aminotransferase, LDL-C: Low-density lipoprotein cholesterol, TG: Triglyceride, VLDL-C: Very low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol, CONUT: Controlling nutritional status score, NLR: Neutrophil-to-lymphocyte ratio, PNI: Prognostic Nutritional Index, SII: Systemic Immune-inflammation Index, Total Chol: Total cholesterol, M: Median, IQR: Interquartile range,

Table 2. The relationship between catecholamines and inflammatory/nutritional markers in patients with pheochromocytoma

	PMET (r; p)	PNMET	UMET	UNMET	Lesion size	PASS score	PNI	NLR	CONUT
PNI	-0.332 0.045	-0.271 0.116	-0.108 0.519	-0.177 0.287	-0.368 0.023	-0.43 0.801	1	0.238 0.055	0.028 0.823
CONUT	-0.094 0.574	-0.188 0.272	-0.071 0.666	0.077 0.643	-0.157 0.339	0.071 0.670	0.028 0.823	0.404 0.001	1
NLR	-0.322 0.052	-0.015 0.933	-0.079 0.636	0.169 0.310	0.013 0.938	-0.025 0.885	0.238 0.055	1	0.404 0.001
SII	-0.317 0.056	-0.022 0.900	0.027 0.870	0.222 0.181	0.015 0.931	0.163 0.334	0.249 0.043	0.802 0.000	0.224 0.071
Lesion size	0.094 0.575	0.427 0.009	-0.026 0.876	0.229 0.161	1	0.362 0.026	-0,368 0.023	0.013 0.938	-0.157 0.339

PMET: Plasma metanephrine, PNMET: Plasma normetanephrine, UMET: Urinary metanephrine, UNMET: Urinary normetanephrine, PNI: Prognostic Nutritional Index, NLR: Neutrophil-to-lymphocyte ratio, CONUT: Controlling nutritional status score, HU: Hounsfield unit, SII: Systemic Immune-inflammation Index

counts, limited sample size, or disease-specific inflammatory characteristics.

Mochizuki et al.[13] compared NLR values in patients with adrenocortical carcinoma (ACC) (n=13) and benign adrenal tumors (n=46). They reported significantly higher NLR levels in patients with ACC compared to those with adrenal adenomas. Furthermore, elevated NLR was associated with poorer prognosis in ACC patients.

In contrast to these findings, NLR did not differ significantly between the pheochromocytoma and control groups in our study. This discrepancy may be explained by the biological differences between ACC, a highly aggressive malignancy, and pheochromocytoma, which is often benign and may not induce the same degree of systemic inflammatory imbalance.

Although inflammatory markers were elevated in patients with pheochromocytoma, no significant differences were observed in the CONUT score or PNI between the groups. This suggests that catecholamine-related inflammatory activation may not necessarily translate into overt nutritional impairment, particularly in patients without advanced or malignant disease.

In correlation analyses, PNI demonstrated a significant inverse association with both plasma metanephrine levels and tumor size. Although overall PNI values did not differ between groups, this finding suggests that increasing tumor burden and catecholamine activity may be associated with deterioration in immune-nutritional status. Elevated catecholamine levels may contribute to metabolic stress, inflammatory activation,

and alterations in protein metabolism, which could partly explain the observed decline in PNI with larger tumors.

SII was significantly elevated in the pheochromocytoma group, further supporting systemic inflammatory activation in these patients. As SII incorporates neutrophil, lymphocyte, and platelet counts, it may more comprehensively reflect inflammatory and thrombo-inflammatory alterations associated with catecholamine excess. The increase in SII, despite stable NLR values, indicates that platelet involvement may contribute to the inflammatory profile seen in pheochromocytoma.

Parazzoli et al.[14] retrospectively evaluated 68 patients with pheochromocytoma and compared them with individuals with nonfunctioning adrenal adenomas and essential hypertension, demonstrating significantly elevated leukocyte, neutrophil, and platelet counts in the pheochromocytoma group. In addition, inflammation-based indices, including NLR and SII, were found to be higher in these patients. Consistent with these findings, our cohort exhibited significantly elevated WBC, neutrophil, and platelet counts, along with elevated SII levels, supporting systemic inflammatory activation associated with catecholamine excess. In addition, Parazzoli et al.[14] reported that plasma metanephrine levels were positively correlated with NLR and negatively correlated with PNI. In our study, we similarly observed a significant negative correlation between PNI and plasma metanephrine levels.

Parazzoli et al.[14] additionally performed BMI-stratified analyses and reported that inflammatory markers were higher in patients with BMI<30 kg/m², whereas NLR and SII tended to be lower in obese individuals. In our cohort, approximately one-third of patients were obese, which may have attenuated between-group differences in NLR and could partly explain the lack of statistical significance.

While NLR did not differ significantly between groups, SII was markedly elevated in patients with pheochromocytoma. This discrepancy may indicate that platelet involvement plays a relevant role in the inflammatory profile associated with catecholamine excess. Therefore, SII may serve as a more sensitive and clinically useful marker of inflammatory status in patients with pheochromocytoma.

Correlation analysis demonstrated significant positive associations between SII and both NLR and PNI. The positive correlation between SII and NLR is not unexpected, as these indices share common hematologic components, particularly neutrophil and lymphocyte counts. However, the association between SII and PNI may reflect a broader interaction between systemic inflammatory activation and immune-nutritional status in pheochromocytoma.

The observed correlation between CONUT score and NLR may partly be explained by shared hematological components, particularly lymphocyte count. Nevertheless, this association supports the concept that inflammatory activation and nutritional parameters may be interrelated rather than independent processes.

In our study, fasting glucose levels were significantly elevated in patients with pheochromocytoma compared to controls, consistent with the well-established metabolic effects of catecholamine excess. Catecholamines promote hepatic gluconeogenesis, glycogenolysis, and insulin resistance, which may contribute to hyperglycemia in these patients.[15]

Limitations

This study has several limitations. First, its retrospective design may have introduced selection bias and limited the ability to establish causal relationships. Second, the sample size was relatively small, which may have reduced the statistical power to detect subtle differences, particularly in nutritional indices such as CONUT and PNI. However, this limitation is largely attributable to the rarity of pheochromocytoma. Third, postoperative inflammatory and nutritional parameters were not available, preventing evaluation of the reversibility of inflammatory changes after tumor removal and limiting the ability to assess causal relationships. In addition, detailed cytokine profiling, including key inflammatory markers such as CRP, IL-6, and TNF-α, was not performed; therefore, the mechanistic pathways underlying the observed inflammatory alterations could not be directly assessed.

CONCLUSION

As a result, pheochromocytoma is associated with a measurable systemic inflammatory response, as evidenced by elevated WBC, neutrophil, and platelet counts, and increased SII. While overall nutritional indices did not differ significantly between groups, the inverse association between PNI and tumor size, as well as plasma metanephrine levels, suggests that increasing tumor burden and catecholamine activity may negatively impact immune-nutritional status. These findings highlight the complex interplay between catecholamine excess, inflammation, and metabolic alterations in pheochromocytoma. Further prospective studies with larger cohorts are warranted to clarify the clinical implications of these immune-metabolic changes.

ETHICAL DECLARATIONS

Ethics Committee Approval

This study was conducted with the approval of the Scientific and Ethical Review Board for Medical Researches No. 2 at Ankara Bilkent City Hospital (Date: 20.08.2025, Decision No: TABED2-25-1442).

Informed Consent

As this was a retrospective study, formal written informed consent was not required and was therefore not obtained.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Financial Disclosure

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Author Contributions

Concept: BÖTB, MSD, NNI; Design: BÖTB, NÇS, BÇ; Control: OT, RE, BÇ; Data Collection and/or Interpretation: BÖTB, MSD; Literature Review: BÖTB, NNI; Article Writing: BÖTB, NÇS; Critical Review: BÖTB, MSD, NÇS, OT, RE, BÇ.

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