

A comparative analysis of leukocyte and leukocyte subtype counts among isolated systolic hypertensive, systo-diastolic hypertensive, and non-hypertensive patients

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Abstract

Background: Isolated systolic hypertension (ISHT) is a subtype of hypertension (HT) that often exhibits wide pulse pressure, and pulse pressure has a strong predictive value for future adverse cardiovascular events. Previous studies have shown the effects of leukocyte count on the prognosis of ischaemic heart disease and HT.

Aim: Thus, in this cross-sectional study, we analysed the relationship between leukocyte counts and subtypes in HT and non-HT groups.

Methods: The study population consisted of 960 consecutive patients who were admitted to the outpatient clinic of our hospital. After ambulatory blood pressure values were assessed, the participants were divided into three groups: ISHT (n = 98), systo-diastolic hypertensives (SDHT, n = 405), and non-hypertensives (non-HT, n = 457).

Results: The subjects in the ISHT group were older than those in the SDHT and non-HT groups (64 ± 10 , 53 ± 12 , and 52 ± 13 , respectively; $p < 0.001$). The leukocyte and neutrophil counts and neutrophil/lymphocyte (N/L) ratios were significantly different in all groups. In subgroup analysis, the leukocyte count, neutrophil count, and N/L ratio were higher in the ISHT and SDHT groups than in the non-HT group ($p < 0.001$ for all). The leukocyte count, neutrophil count, and N/L ratio were significantly higher in the ISHT group than in the SDHT group ($p = 0.023$, $p = 0.007$, $p = 0.010$, respectively). Neutrophil count ($p = 0.012$; OR = 1.229, 95% CI 1.046–1.444) was an independent risk factor for ISHT in multivariate logistic regression analysis.

Conclusions: The leukocyte and neutrophil counts and N/L ratios were higher in the ISHT group than in the SDHT and non-HT groups. High neutrophil count was an independent predictor of ISHT.

Key words: isolated systolic hypertension, leukocyte count, neutrophil count, N/L ratio

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INTRODUCTION

Hypertension (HT) is an important risk factor for cardiovascular disease [1, 2]. Isolated systolic hypertension (ISHT) is a subtype of HT, the importance of which has become understood in recent years. ISHT often exhibits wide pulse pressure (PP). Defined as the difference between systolic (SBP) and diastolic (DBP) blood pressure, PP is an indicator of a prominently in-

creased stiffening of the arteries and advanced organ damage in HT patients [3]. Wide PP is a good indicator of cardiovascular events in middle-aged and elderly HT patients [4–7]. Although the underlying pathogenesis is not well known, recent studies have shown that systemic inflammation plays a vital role in the development of HT [8, 9]. Leukocyte and neutrophil counts are simple but effective markers of chronic

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inflammation [10]. Epidemiological studies have shown an association between white blood cell count and prognosis in ischaemic heart disease and in HT [11–15]. In addition, previous clinical studies have shown a relationship between high leukocyte counts and PP and increased future adverse cardiovascular events. Therefore, in this study, we investigated the relationship of leukocyte counts and leukocyte subtypes in ISHT groups compared to systo-diastolic HT (SDHT) and non-HT subjects.

METHODS

A total of 2,500 patients (18–80 years of age), whose ambulatory blood pressure (ABP) records had been evaluated between January 2011 and December 2012, were enrolled onto our study. Past medical history information, including medications, smoking habits, physical examination results, and anthropometric measurements (height, weight) were taken from patient files. Any missing or unavailable data resulted in exclusion of the patient. By the end of the recruitment process, a total of 960 patients remained and were included in the study.

After the office blood pressure (OBP) and ABP assessment, the patients were divided into three groups: ISHT ($n = 98$), SDHT ($n = 405$), and non-HT ($n = 457$).

SDHT was defined as $OBP \geq 140/90$ mm Hg, daytime $ABP \geq 135/85$ mm Hg or the active use of antihypertensive drugs.

ISHT was defined as $OBP \geq 140/90 <$ mm Hg. The thresholds for daytime ambulatory ISHT were ≥ 135 mm Hg for SBP and < 85 mm Hg for DBP.

Non-HT was defined as consistently $OBP < 140/90 <$ mm Hg and daytime $ABP < 135/85 <$ mm Hg in subjects not receiving antihypertensive treatment [16].

Diabetes was defined according to the American Diabetes Association guidelines, as fasting serum glucose ≥ 126 mg/dL (7 mmol/L), nonfasting glucose ≥ 200 mg/dL (11.1 mmol/L), or active use of anti-diabetic treatment [17]. Smoking habit was defined as a history of tobacco use at admission (packs/years). The height and weight of the subjects were recorded, and body mass index (BMI) was calculated as the ratio of weight in kilograms divided by the square of height in metres.

The exclusion criteria of this study were secondary HT, heart failure, coronary artery disease, stroke, moderate to severe valvular disease, chronic renal failure, chronic liver disease, thromboembolic disorders, and haematological abnormality. Because extreme levels could indicate occult diseases, we also excluded patients with a total leukocyte count $> 12,000/\text{mm}^3$, neutrophil $> 78\%$, lymphocyte $> 63\%$, or monocyte $> 14\%$. Because of the different pathophysiologic mechanisms that cause only increased SBP such as high stroke volume in young adults, ISHT patients below the age of 40 were excluded from the present study (four patients). Eligible patients were between 18 and 80 years of age, and all provided written informed consent, which was a prerequisite

for enrollment. The study complied with the Declaration of Helsinki, and the trial protocol was approved by the local ethics committee.

ABPM

Ambulatory blood pressure monitoring (ABPM) was monitored for 24 h with an ambulatory BP monitor (Tonoport V; GE Healthcare) that was programmed to measure BP every 15 min during the day, and every 30 min during the night, for a 24-h period. Daytime was defined as 07:00–23:00 h, and night-time as 23:00–07:00 h.

Laboratory parameters, including total leukocyte, neutrophil, monocyte, and lymphocyte counts and glucose, creatinine and lipid profiles were determined using standard biochemical methods.

Statistical analysis

Statistical analyses were performed using SPSS software, version 17. The variables were investigated using visual (histogram, probability plot) and analytical (Kolmogorov-Smirnov, Shapiro-Wilk tests) methods to determine whether or not they were normally distributed. Descriptive analyses were presented as mean \pm standard deviation (SD), and categorical variables were expressed as percentages. A Mann-Whitney U test was performed to test the significance of pairwise differences, using the Bonferroni correction to adjust for multiple comparisons. Differences between continuous and categorical variables among the three groups were assessed using one-way ANOVA, Kruskal-Wallis, and χ^2 tests. For the multivariate analysis, the possible factors identified with univariate analyses were further entered into the logistic regression analysis to determine independent predictors of ISHT. A Spearman's correlation analysis was performed to determine the association of neutrophil/lymphocyte (N/L) ratio with 24-h SBP, 24-h DBP and PP. An overall 5% type-I error level was used to infer statistical significance.

RESULTS

Baseline demographic, clinical, and biochemical characteristics of the three groups are presented in Table 1. There were 98 ISHT, 405 SDHT, and 457 non-HT subjects enrolled in the study. The mean age of the ISHT group was 64 ± 10 years. The number of patients with ISHT in each group distributed according to their age was as follows: 77 (78.6%) patients were ≥ 60 ; 16 (16.3%) patients 50–60, and 5 (5.1%) patients 40–50. The mean age of ISHT patients ≥ 60 was 68 ± 10 . Also, ISHT patients were considered in different subgroups: sustained HT — 44 (44.9%), white-coat HT — 31 (31.6%), and masking HT — 23 (23.5%). For SDHT patients, the subgroups were: sustained HT — 192 (47.4%), white-coat HT — 105 (25.9%), and masking HT — 114 (28.1%). There were no significant differences among the three groups in terms of baseline demographics

Table 1. Characteristics of the study population

	ISHT (n = 102)	SDHT (n = 405)	Non-HT (n = 457)	P
Gender (male)	46 (46.9%)	209 (51.6%)	232 (50.8%)	0.709
Age [years]	64 ± 10*	53 ± 12	52 ± 13	< 0.001
Body mass index [kg/m ²]	30 ± 4	30 ± 4	27 ± 5	0.110
Smoking	45 (45.9%)	175 (43.2%)	213 (46.6%)	0.597
Diabetes	24 (24.5%)	69 (17%)	71 (15.5%)	0.102
Glucose [mg/dL]	107 ± 30	113 ± 40	108 ± 24	0.190
Creatinine [mg/dL]	0.9 ± 0.5	0.9 ± 0.3	0.9 ± 0.2	0.121
LDL-cholesterol [mg/dL]	135 ± 40	132 ± 35	130 ± 35	0.395
Total-cholesterol [mg/dL]	208 ± 46	203 ± 44	206 ± 42	0.822
Triglyceride [mg/dL]	159 ± 68	157 ± 80	149 ± 73	0.240
HDL-cholesterol [mg/dL]	48 ± 12	48 ± 12	50 ± 13	0.330
ACEI	20 (20.4%)	95 (23.5%)		0.519
ARB	15 (15.3%)	65 (16%)		0.857
Beta-blocker	11 (11.2%)	35 (8.6%)		0.426
Ca-channel blocker	18 (18.4%)	48 (11.9%)		0.086
Diuretic	19 (19.4%)	87 (21.5%)		0.648

*p < 0.01 ISHT vs. SDHT and non-HT group; ACEI — angiotensin-converting enzyme inhibitor; ARB — angiotensin receptor blocker; LDL — low-density lipoprotein; HDL — high-density lipoprotein; HT — hypertension; ISHT — isolated systolic hypertension; SDHT — systo-diastolic hypertension

Table 2. Comparison of ambulatory blood pressure monitoring variables between the three groups

	ISHT	SDHT	Non-HT	P
Daytime-SBP [mm Hg]	150 ± 10	156 ± 12	124 ± 10	< 0.001
Daytime-DBP [mm Hg]	83 ± 5	99 ± 9	77 ± 7	< 0.001
Night-time-SBP [mm Hg]	145 ± 13	148 ± 15	114 ± 11	0.139
Night-time-DBP [mm Hg]	77 ± 7	90 ± 10	68 ± 7	< 0.001
24-h-SBP [mm Hg]	149 ± 9	154 ± 14	122 ± 9	< 0.001
24-h-DBP [mm Hg]	81 ± 4	97 ± 8	75 ± 7	< 0.001
Pulse pressure	68 ± 11	57 ± 11	47 ± 6	< 0.001

DBP — diastolic blood pressure; HT — hypertension; ISHT — isolated systolic hypertension; SBP — systolic blood pressure; SDHT — systo-diastolic hypertension

or clinical properties, with the exception of age. The ISHT group was older than the SDHT and non-HT groups (64 ± 10, 53 ± 12, and 52 ± 13 years, respectively; all p < 0.001). However, there was no significant difference between the SDHT and non-HT groups (p = 0.971). BMI was lower in the non-HT group (27 ± 5 kg/m²) than in the ISHT (30 ± 4 kg/m²) and SDHT (30 ± 4 kg/m²) groups, but the difference was not statistically significant (p = 0.110). There was no difference between the HT groups in terms of medications.

When the ABPM results of the three groups were investigated, daytime SBP, daytime DBP, night-time DBP, 24-h SBP, and 24-h DBP were found to be significantly higher in the SDHT group than in the ISHT and non-HT groups (all p < 0.001 except night-time SBP, p = 0.139). 24-h PP was

significantly higher in the ISHT group (68 ± 11 mm Hg) than in the SDHT group (57 ± 11 mm Hg) (p < 0.001) (Table 2).

Leukocyte, neutrophil counts and N/L ratios were higher in the ISHT and SDHT groups than in the non-HT group (p < 0.001, p < 0.001, and p = 0.001, respectively). The leukocyte, neutrophil counts and N/L ratios of the ISHT group were higher than those of the SDHT group (p = 0.023, p = 0.007, and p = 0.010, respectively). There were no significant differences in lymphocyte and monocyte counts among the three groups (Table 3).

Neutrophil counts were positively correlated with 24-h SBP (p = 0.040), daytime and night-time SBP (p = 0.032, p = 0.029, respectively) and 24-h PP (p = 0.006) (Table 4). Leukocyte counts were positively correlated with 24-h SBP (p = 0.042),

Table 3. Leukocyte and leukocyte subtype counts in the three groups

	ISHT	SDHT	Non-HT	P
Leukocyte count [10^9 cells/L]	8.1 ± 2.1	7.6 ± 1.8	7.1 ± 1.7	< 0.001
Neutrophil count [10^9 cells/L]	5.1 ± 1.6	4.67 ± 1.3	4.0 ± 1.2	< 0.001
Lymphocyte count [10^9 cells/L]	2.3 ± 0.9	2.3 ± 0.7	2.4 ± 0.7	0.293
Monocyte count [10^9 cells/L]	0.5 ± 0.2	0.6 ± 0.2	0.5 ± 0.1	0.285
N/L ratio	2.4 ± 1	2.1 ± 0.7	1.8 ± 0.6	0.001

N/L — neutrophil/lymphocyte; HT — hypertension; ISHT — isolated systolic hypertension; SDHT — systo-diastolic hypertension

Table 4. Correlations between selected covariates and neutrophil count

	r	p
24-h SBP	0.092	0.040
24-h DBP	-0.050	0.262
Daytime SBP	0.096	0.032
Daytime DBP	-0.042	0.342
Night-time SBP	0.098	0.029
Night-time DBP	-0.007	0.868
24-h pulse pressure	0.124	0.006
Age	-0.002	0.963

DBP — diastolic blood pressure; SBP — systolic blood pressure

daytime and night-time SBP ($p = 0.041$ and $p = 0.029$, respectively), and 24-h PP ($p = 0.027$). In addition, N/L ratio was also found to be positively correlated with 24-h SBP ($p = 0.037$), night-time SBP ($p = 0.019$), and 24-h PP ($p = 0.002$).

In univariate logistic regression analysis, neutrophil count ($p = 0.003$; OR = 1.252, 95% CI 1.078–1.454), diabetes mellitus ($p < 0.001$; OR = 2.253, 95% CI 1.369–3.709) and age ($p < 0.001$; OR = 1.100, 95% CI 1.074–1.126) were found to be independent risk factors of ISHT (Table 5). In addition, neutrophil count ($p = 0.012$; OR = 1.229, 95% CI 1.046–1.444), diabetes mellitus ($p = 0.007$; OR = 2.136, 95% CI 1.234–3.698) and age ($p < 0.001$; OR = 1.101, 95% CI 1.074–1.128) were independent risk factors in multivariate logistic regression analysis (Table 5).

DISCUSSION

Three main findings emerged from this study. Firstly, the leukocyte counts, neutrophil counts, and N/L ratios of the HT patients were higher than those of the non-HT group. Secondly, the leukocyte counts, neutrophil counts, and N/L ratios of the ISHT group were higher than those of the SDHT group. Thirdly, there were significant positive correlations between the leukocyte counts, neutrophil counts, N/L ratios and ambulatory 24-h SBP, night-time SBP, and ambulatory 24-h PP (APP).

ABPM is known to be superior to OBP for predicting organ damage [18] and long-term prognosis [19, 20]. Therefore, we used ABPM in this study, and we found that this parameter correlated with neutrophil and leukocyte counts and N/L ratios.

Atherosclerosis is a chronic inflammatory process [21, 22] that can lead to HT [8, 9], and leukocyte count is a marker of chronic inflammation [11]. Leukocytes may increase reactive oxygen species, arachidonic acid and cathepsin release during chronic inflammation. Nitric oxide inactivation, increased vascular inflammation and renin–angiotensin system stimulation play pivotal roles in the development of HT [23]. Leukocytes may increase blood viscosity, and thus affect peripheral vascular resistance, which may also be a factor in the development of HT [24]. Tatsukava et al. [25] reported that an increase in neutrophil count may be a strong indicator of HT development. Tiang et al. [26] showed the positive direct relationship of neutrophil count and the inverse relationship of lymphocyte count with HT. HT has a direct relationship with

Table 5. Univariate and multivariate stepwise logistic regression model of isolated systolic hypertension

	Univariate			Multivariate		
	OR	95% CI	P	OR	95% CI	P
Neutrophil	1.252	1.078–1.454	0.003	1.229	1.046–1.444	0.012
Diabetes mellitus	2.253	1.369–3.709	0.009	2.136	1.234–3.698	0.007
Smoking	1.116	0.716–1.738	0.628			
Male gender	0.830	0.533–1.291	0.407			
Age	1.100	1.074–1.126	< 0.001	1.101	1.074–1.128	< 0.001
Creatinine	1.171	0.915–3.163	0.093			

CI — confidence interval; OR — odds ratio

neutrophil count and an inverse relationship with lymphocyte count, especially in African-Americans, but much less so in Caucasians. In our study, lymphocyte counts were higher in the nonhypertensive group than in the HT groups, but the difference was not statistically significant (Table 1).

In our study, neutrophil and leukocyte counts were higher in the ISHT group than in the SDHT group. Given that the patients in the ISHT group were older than those in the other groups, there are conflicting results in the literature regarding any correlation between age and neutrophil count. We did not find a correlation between age and neutrophil count in our present study ($r = -0.002$, $p = 0.957$).

In addition, just 24-h APP, except for other ambulatory SBP and ambulatory DBP, was higher in the ISHT groups than in the SDHT group (Table 2). The results might suggest that wide APP has a better predictive value than other ABPM values for indicating inflammation. Angeli et al. [27] found that wide 24-h APP (≥ 57 mm Hg) patients had higher neutrophil counts. Wide PP has been found to be related to increased markers of chronic inflammation and stiffening of the arteries, and therefore it has been used as a marker of atherosclerosis [11, 28].

In the current study, we found that N/L ratios were higher in the ISHT patients than in the other subjects. Not only neutrophil count, but also N/L ratio, may be associated with increased arterial stiffness and intense inflammation in HT. Previous studies have shown a relationship between N/L ratio and future adverse cardiovascular events [29–32].

It is known that ISHT is related to arterial stiffness. Previous studies have shown that smoking produces an increase in arterial stiffness [33, 34]. The association between smoking and ISHT has been inconsistent in prior studies [35, 36]. In our multiple regression model, smoking status was not an independent determinant of ISHT.

Limitations of the study

Our study has some limitations. Firstly, it had a retrospective, single-centre design. Secondly, we could not evaluate the leukocyte activation markers, lymphocyte subtypes, high-sensitivity C-reactive protein, other proinflammatory cytokines, or markers of oxidative stress. Thirdly, we excluded patients > 80 years old and/or with clinically overt cardiovascular disease (such as coronary artery disease, cerebrovascular disease, and renal failure), and therefore our results cannot be extrapolated to all hypertensive subjects.

CONCLUSIONS

Leukocyte, neutrophil counts and N/L ratios were found to be higher in the ISHT group than in the SDHT group in our study. Also high neutrophil count was an independent predictor of ISHT.

Conflict of interest: none declared

References

1. Burt VL, Whelton P, Roccella EJ et al. Prevalence of hypertension in the US adult population. Results from the Third National Health and Nutrition Examination Survey, 1988–1991. *Hypertension*, 1995; 25: 305–313.
2. Palmer A, Bulpitt C, Beevers G et al. Risk factors for ischaemic heart disease and stroke mortality in young and old hypertensive patients. *J Hum Hypertens*, 1995; 9: 695–697.
3. Laurent S, Cockcroft J, Van Bortel L et al.; on behalf of the European Network for Non Invasive Investigation of Large Arteries. Expert consensus document on arterial stiffness: methodological issues and clinical applications. *Eur Heart J*, 2006; 27: 2588–2605.
4. Darne B, Girerd X, Safar M et al. Pulsatile versus steady component of blood pressure: a cross-sectional analysis and a prospective analysis on cardiovascular mortality. *Hypertension* 1989; 13: 392–400.
5. Benetos A, Safar M, Rudnichi A et al. Pulse pressure: a predictor of long-term cardiovascular mortality in a French male population. *Hypertension*, 1997; 30: 1410–1415.
6. Gasowski J, Fagard RH, Staessen JA et al.; INDANA Project Collaborators. Pulsatile blood pressure component as predictor of mortality in hypertension: a meta-analysis of clinical trial control groups. *J Hypertens*, 2002; 20: 145–151.
7. Blacher J, Staessen JA, Girerd X et al. Pulse pressure not mean pressure determines cardiovascular risk in older hypertensive patients. *Arch Intern Med*, 2000; 160: 1085–1089.
8. Harrison DG, Guzik TJ, Goronzy J, Weyand C. Is hypertension an immunologic disease? *Curr Cardiol Rep*, 2008; 10: 464–469.
9. Rodriguez-Iturbe B, Johnson RJ. Role of inflammatory cells in the kidney in the induction and maintenance of hypertension. *Nephrol Dial Transplant*, 2006; 21: 260–263.
10. Pauletto P, Rattazzi M. Inflammation and hypertension: the search for a link. *Nephrol Dial Transplant*, 2006; 21: 850–853.
11. Manabe S, Okura T, Watanabe S, Higaki J. Association between carotid haemodynamics and inflammation in patients with essential hypertension. *J Hum Hypertens*, 2005; 19: 787–791.
12. Berliner S, Rogowski O, Rotstein R et al. Activated polymorphonuclear leukocytes and monocytes in the peripheral blood of patients with ischemic heart and brain conditions correspond to the presence of multiple risk factors for atherothrombosis. *Cardiology*, 2000; 94: 19–25.
13. Duilio C, Ambrosio G, Kuppusamy P et al. Neutrophils are primary source of O₂ radicals during reperfusion after prolonged myocardial ischemia. *Am J Physiol Heart Circ Physiol*, 2001; 280: H2649–H2657.
14. Naruko T, Ueda M, Haze K et al. Neutrophil infiltration of culprit lesions in acute coronary syndromes. *Circulation*, 2002; 106: 2894–2900.
15. Pearson TA, Mensah GA, Alexander RW et al.; Centers for Disease Control and Prevention; American Heart Association. Markers of inflammation and cardiovascular disease: application to clinical and public health practice. A statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. *Circulation*, 2003; 107: 499–511.
16. O'Brien E, Asmar R, Beilin L et al.; on behalf of the European Society of Hypertension Working Group on Blood Pressure Monitoring. Practice guidelines of the European Society of Hypertension for clinic, ambulatory and self blood pressure measurement. *J Hypertens*, 2005; 23: 697–701.
17. Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care*, 2003; 26 (suppl.): S5–S20.
18. Zanchetti A, Bond MG, Hennig M et al. Risk factors associated with alterations in carotid intima-media thickness in hyperten-

- sion: baseline data from the European Lacidipine Study on Atherosclerosis. *J Hypertens*, 1998; 16: 949–961.
19. Verdecchia P, Schillaci G, Borgioni C et al. Ambulatory pulse pressure: a potent predictor of total cardiovascular risk in hypertension. *Hypertension*, 1998; 32: 983–988.
 20. Verdecchia P, Schillaci G, Reboldi G et al. Different prognostic impact of 24-hour mean blood pressure and pulse pressure on stroke and coronary artery disease in essential hypertension. *Circulation*, 2001; 103: 2579–2584.
 21. Ross R. Atherosclerosis — an inflammatory disease. *N Engl J Med*, 1999; 340: 115–126.
 22. Libby P. Vascular biology of atherosclerosis: overview and state of the art. *Am J Cardiol*, 2003; 91: 3A–6A.
 23. Miller MA, Kerry SM, Cook DG, Cappuccio FP. Cellular adhesion molecules and blood pressure: interaction with sex in a multi-ethnic population. *J Hypertens*, 2004; 22: 705–711.
 24. Schillaci G, Pirro M, Pucci G et al. Prognostic value of elevated white blood cell count in hypertension. *Am J Hypertens*, 2007; 20: 364–369.
 25. Tatsukawa Y, Hsu WL, Yamada M et al. White blood cell count, especially neutrophil count, as a predictor of hypertension in a Japanese population. *Hypertens Res*, 2008; 31: 1391–1397.
 26. Tiang N, Penman AD, Mawson AR et al. Association between circulating specific leukocyte types and blood pressure: the atherosclerosis risk in communities (ARIC) study. *J Am Soc Hypertens*, 2010; 4: 272–283.
 27. Angeli F, Angeli E, Ambrossia G et al. Neutrophil count and ambulatory pulse pressure as predictors of cardiovascular adverse events in postmenopausal women with hypertension. *Am J Hypertens*, 2011; 24: 591–598.
 28. Safar ME. Pulse pressure in essential hypertension: clinical and therapeutical implications. *J increased cardiovascular risk. Obstet Gynecol*, 2010; 115: 695–703.
 29. Horne BD, Anderson JL, John JM et al. Intermountain Heart Collaborative Study Group. Which white blood cell subtypes predict increased cardiovascular risk? *J Am Coll Cardiol*, 2005; 45: 1638–1643.
 30. Núñez J, Núñez E, Bodí V et al. Usefulness of the neutrophil to lymphocyte ratio in predicting long-term mortality in ST segment elevation myocardial infarction. *Am J Cardiol*, 2008; 101: 747–752.
 31. Papa A, Emdin M, Passino C et al. Predictive value of elevated neutrophil-lymphocyte ratio on cardiac mortality in patients with stable coronary artery disease. *Clin Chim Acta*, 2008; 395: 27–31.
 32. Sabatine MS, Morrow DA, Cannon CP et al. Relationship between baseline white blood cell count and degree of coronary artery disease and mortality in patients with acute coronary syndromes: a TACTICS-TIMI 18 (Treat Angina with Aggrastat and determine Cost of Therapy with an Invasive or Conservative Strategy-Thrombolysis in Myocardial Infarction 18 trial) substudy. *J Am Coll Cardiol*, 2002; 40: 1761–1768.
 33. Mahmud A, Feely J. Effect of smoking on arterial stiffness and pulse pressure amplification. *Hypertension*, 2003; 41: 183–187.
 34. Vlachopoulos C, Kosmopoulos F, Panagiotakos D et al. Smoking and caffeine have a synergistic detrimental effect on aortic stiffness and wave reflections. *J Am Coll Cardiol*, 2004; 44: 1911–1917.
 35. Kim JA, Kim SM, Choi YS et al. The prevalence and risk factors associated with isolated untreated systolic hypertension in Korea: The Korean National Health and Nutrition Survey 2001. *J Hum Hypertens*, 2006; 21: 107–113.
 36. Mahmud A, Feely J. Spurious systolic hypertension of youth: fit young men with elastic arteries. *Am J Hypertens*, 2003; 16: 229–232.

Analiza porównawcza całkowitej liczby leukocytów i określonego typu leukocytów między grupami pacjentów z izolowanym nadciśnieniem skurczowym, nadciśnieniem skurczowo-rozkurczowym i prawidłowym ciśnieniem tętniczym

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Streszczenie

Wstęp: Izolowane nadciśnienie tętnicze (ISHT) jest typem nadciśnienia tętniczego (HT), w którym często występuje wysokie ciśnienie tętna. Ciśnienie tętna ma dużą wartość predykcyjną w odniesieniu do przyszłych niepożądanych zdarzeń sercowych. Wcześniejsze badania wykazały, że liczba leukocytów wpływa na rokowanie dotyczące choroby niedokrwiennej serca oraz HT.

Cel: Celem badania było przeanalizowanie zależności między liczbą leukocytów i liczbą określonego typu leukocytów u osób zakwalifikowanych do grup z HT lub grupy bez HT.

Metody: Do badania włączono 960 kolejnych pacjentów przyjętych do poradni przyszpitalnej. Po ambulatoryjnym pomiarze ciśnienia tętniczego uczestników podzielono na trzy grupy: osoby z ISHT (n = 98), osoby ze skurczowo-rozkurczowym HT (n = 405) i osoby z prawidłowym ciśnieniem tętniczym (n = 457).

Wyniki: Pacjenci z grupy ISHT byli starsi niż osoby ze skurczowo-rozkurczowym HT oraz osoby z prawidłowym ciśnieniem tętniczym (bez HT) (odpowiednio 64 ± 10 , 53 ± 12 i 52 ± 13 ; $p < 0,001$). Liczba leukocytów, liczba neutrofilii i współczynnik N/L różniły się istotnie między grupami. W analizie podgrup wykazano, że liczba leukocytów, liczba neutrofilii i współczynnik N/L były wyższe w grupach osób z ISHT oraz skurczowo-rozkurczowym HT niż u pacjentów z prawidłowym ciśnieniem tętniczym ($p < 0,001$ dla wszystkich porównań). Liczba leukocytów, liczba neutrofilii i współczynnik N/L były istotnie wyższe w grupie z ISHT niż w grupie ze skurczowo-rozkurczowym HT (odpowiednio: $p = 0,023$; $p = 0,007$; $p = 0,010$). Liczba neutrofilii ($p = 0,012$; OR = 1,229; 95% CI 1,046–1,444) była niezależnym czynnikiem ryzyka ISHT w wieloczynnikowej analizie regresji logistycznej.

Wnioski: Liczba leukocytów, liczba neutrofilii i współczynnik N/L były większe w grupie ISHT niż w grupach ze skurczowo-rozkurczowym HT i z prawidłowym ciśnieniem tętniczym. Duża liczba neutrofilii była niezależnym czynnikiem predykcyjnym ISHT.

Słowa kluczowe: izolowane nadciśnienie skurczowe, liczba leukocytów, liczba neutrofilii, współczynnik N/L

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