THE NEW ARMENIAN MEDICAL JOURNAL
Vol.15 (2021), No 4, p.42-49

NEUTROPHIL-TO-LYMPHOCYTE RATIO AND RENAL FUNCTION IN HYPERTENSIVE CRISIS PATIENTS

Hartono R.T.D.[1*], Purwanto A.[1], Pangarso D.C.[1], Ludfi A.S.[2]

[1]Department of Emergency, Citra Husada Hospital, Jember, Indonesia
[2]Department of Internal Medicine, Citra Husada Hospital, Jember, Indonesia

Received 29.09.2020; accepted for printing 07.08.2021

Abstract

Background: *The incidence of hypertensive crisis patients worldwide has been estimated at 1-2% of the population. The objective of this study was to evaluate the correlation between neutrophil-to-lymphocyte ratio and renal function in the acute state of hypertensive crisis.*

Methods: *The study was conducted in Citra Husada hospital in Jember, East Java, Indonesia, including 167 consecutive patients admitted to the hospital between January and December 2019 to diagnose hypertensive crisis. The neutrophil-to-lymphocyte ratio was counted as neutrophil count (/μL) divided by lymphocyte count (/μL). The estimated glomerular filtration rate (ml/min/1.73 m^2) was calculated with the Modification of Diet in Renal Disease equation.*

Results: *Hypertensive crisis proportion was lower in male (40.7%, 68 patients) than females (59.3%, 99 patients). The hypertensive emergency incidence was higher (92.2%, 154 patients) with cerebral events as the most frequent target organ damage (64.1%, 107 patients). neutrophil-to-lymphocyte ratio was significantly lower in hypertensive emergency patients (p=0.033), while estimated glomerular filtration rate was significantly higher in hypertensive emergency patients (p=0.046). neutrophil-to-lymphocyte ratio and estimated glomerular filtration rate correlation was significant (p=0.036) with a very weak score (r=-0.163) and coefficient of determination 0.266. A decrease of estimated glomerular filtration rate of 26.6 % will follow each increase in neutrophil-to-lymphocyte ratio.*

Conclusion: *This study suggested a relationship between the increasing neutrophil-to-lymphocyte ratio with decreasing estimated glomerular filtration rate in hypertensive crisis patients. Further studies are needed to evaluate whether the increase in neutrophil-to-lymphocyte ratio was an acute or chronic disease process.*

Keywords: *. eGFR, hypertensive crisis, hypertensive emergencies, hypertension urgencies, NLR*

Introduction

Hypertension is the most prevalent chronic medical condition afflicting people globally [*Varounis C et al., 2017*]. High blood pressure (BP) is a highly prevalent ailment, and its repercussions (including heart disease, stroke, and kidney failure) are significant public health problems [*Salim H et al., 2020*]. Hypertension contributes as a risk factor for the leading causes of death in Indonesia: stroke and ischemic heart disease [*Centers for Disease Control and Prevention, 2018*]. Hypertension comorbid was one of the causes of coronary heart disease (CHD). High blood pressure would cause direct trauma to the walls of the coronary arteries. Therefore, it facilitated coronary atherosclerosis (coronary factors) [*Saputri F et al., 2020*]. The incidence of hypertensive crisis patients around the world has been estimated at 1-2% of the population [*Chakraborty S, 2017*]. In 2004, hypertension was the third most prevalent condition seen in outpatient clinics in Indonesia. By 2006, it had risen to the second position (4.67 percent). According to risk factor surveys for cardiovascular disease (CVD), the prevalence of hypertension has increased to 13.6 percent in men and 16 percent in women in Indonesia [*Sulistiawati S et al., 2020*].

Hypertensive crisis consists of hypertensive

Address for Correspondence:
Rahma Tansy Dea Hartono, MD
Department of Emergency, Citra Husada Hospital, Jember, Indonesia
Jl. Teratai No.22, Patrang, Jember 68117, Indonesia
Tel.: +6281327619500
E-mail: rahmatansydea@gmail.com

emergency (HE) and hypertensive urgency (HU) [*Chakraborty S, 2017*] are acute life-threatening condition [*Derhaschnig U et al., 2014*]. Over the last year, hypertensive patients have experienced a decrease in production due to inability to work (4%) and 28.2 percent sick leave [*Sulistiawati S et al., 2020*]. HE and HU are a condition of severe hypertension in the presence of target organ damage [*Lukito A et al., 2019*]. The target organs include the brain, retina, large arteries, and kidneys [*Unger T et al., 2020*]. This condition can lead to hypertensive encephalopathy, hemorrhagic and non-hemorrhagic stroke, acute coronary syndromes, pulmonary edema, aortic dissection, renal failure, sympathetic crises, and eclampsia. HU is the isolated elevation of BP without the involvement of target organs with the evidence of clinical, laboratory, or instrumental evidence [*Paini A et al., 2018*]. Systolic blood pressure (SBP) >179 *mmHg* or diastolic blood pressure (DBP) >109 *mmHg* is considered hypertensive crisis [*Varounis C et al., 2017*].

The pathophysiology of hypertensive crises is unknown. This event is thought to be initiated by a sudden increase in vascular resistance caused by humoral vasoconstrictors [*Derhaschnig U et al., 2014*]. BP mechanism of self-regulation hypertensive crisis occurs when the brain, heart, and kidneys are unable to maintain stable blood flow. Mechanical stress is caused by autoregulatory dysfunction in the vascular bed and blood flow, which results in an abrupt increase in blood pressure and systemic vascular resistance [*Varounis C et al., 2017*]. Ischemia and vasoactive mediators release lead injury to endothelial. The renin-angiotensin system is activated, resulting in increased vasoconstriction and the generation of proinflammatory cytokines, such as IL-6 [*Haas A, Marik P, 2006*].

Kidneys are one of organs affected by hypertension, and malignant hypertension can cause decrease in renal function [*Derhaschnig U et al., 2014*]. On the other hand, hypertension places a mechanical strain on the glomerulus, which is closely correlated with chronic kidney disease (CKD) progression. Therefore, CKD and high blood pressure often occur simultaneously, characterized by increased blood pressure with decreased kidney function resulting in poor clinical outcomes [*Thaha M et al., 2019*]. Globally, the prevalence of CKD is growing. The prevalence of CKD is estimated to be between 40–60 cases per million people in poor nations. In Indonesia, nephrology centers estimated a prevalence of CKD of between 200 and 250 cases per million population [*Suprapti B et al., 2019*].

Renal function can be evaluated by the laboratory testing of creatinine and estimated glomerular filtration (eGFR) ratio [*Derhaschnig U et al., 2014*]. An increase will follow the decrease in eGFR in CKD in inflammation characterized by increased cytokines, such as IFN, TNFa, IL-1, IL-6, and IL-10. Surge in cytokines is caused by a disruption of the elimination of these cytokines in the kidneys, repeated infections, increased oxidative stress, uremic conditions, metabolic acidosis also affects the increase in inflammatory conditions [*Hertanto D et al., 2019*]. Additionally, hypertensive patients, particularly those with pre-existing severe renal disease, may see an increase in creatinine [*Derhaschnig U et al., 2014*].

Microinflammation is a significant component of CKD and has a significant role in the pathophysiology of CVD complications, as well as protein-energy deficiency (PED) and mortality [*Thaha M, Widiana I, 2019*]. The inflammatory process can be assessed from increased leukocytes and their subtypes. One of the hallmarks investigated regarding cardiovascular risk is the neutrophil-to-lymphocyte ratio (NLR) [*Sevencan N, Ozkan A, 2018*]. The neutrophil is a polymorphonuclear mature granular leukocyte that has an affinity to the immune complex and phagocytosis. The lymphocyte is one specific immune system and has specific antigens, which are its ligands [*Azikin A et al., 2018*]. NLR is a marker of cardiac or non-cardiac disorders and a predictor of mortality in patients with CVD. NLR has been linked to inflammatory indicators such as IL-6 or high sensitivity-CRP (hs-CRP), as well as endothelial dysfunction and cardiovascular risk in individuals with CKD [*Yoshitomi R et al., 2019*]. Other studies also

To overcome it is possible, due to the uniting the knowledge and will of all doctors in the world

state that hs-CRP and NLR have correlated with kidney functions such as eGFR, cystatin c, and blood urea nitrogen (BUN) [*Rochmanti M et al., 2020*]. IL-6 is also one of the most prominent pro-inflammatory cytokines that can be found in CKD patients. Various studies have shown that IL-6 levels increased in CKD, especially in later stage and in a dialysis patient [*Bramantya R et al., 2020*].

Only a few research have examined the link between NLR and renal function in patients with hypertensive crises. The purpose of this study was to determine whether NLR corresponds with renal function in individuals experiencing an acute hypertensive crisis.

METHODS

Patients and Study Design

The study took place at Citra Husada Hospital in Jember, East Java, Indonesia. The data for this cross-sectional study were gathered from medical records between January and February 2020. We included 167 consecutive patients who were admitted to the hospital between January and December 2019 with the diagnosis of hypertensive crisis. Incomplete information from medical records was a criterion for exclusion. The study was approved by the Ethics Committee of Citra Husada Hospital, Jember, Indonesia (approval no.158/RSCH/I/2020).

Clinical and Laboratory Assessment

Demographic information (age and sex) and history of hypertension were recorded for each patient on admission. The BP of patients was measured on the presentation at the emergency room with an automated device (Omron) or a manual device (Riester). HE was defined as a severe elevation of SBP >179 *mmHg* or DBP >109 *mmHg* with target organ damage. HU was defined as a marked rise in blood pressure in the absence of evidence of target organ damage.

Blood samples were obtained from each patient at admission in the emergency room for complete blood count and serum creatinine (SCrz). The NLR was counted as neutrophil count (*/μL*) divided by lymphocyte count (*/μL*). The eGFR rate (eGFR, *ml/min*/1.73 m^2) was calculated with the Modification of Diet in Renal Disease equation [*Levey A et al., 1999*] eGFR = 186 x [SCr x 0.0011312] $^{-1.154}$x [age (years)] $^{-0.203}$x [0.742 if female] x [1.212 if black] where GFR (*ml/min*/1.73 m^2), Scr (μmol/L) is serum creatinine, age (years).

Statistical Analysis

IBM SPSS, version 24.0, was used to analyze the data. If the distribution was not normal, continuous data were presented as mean standard deviation (e.g., age) and median (min.-max). Categorical data are expressed as number (%). The independent t-test was used to compare two continuous variables with a normal distribution and the Mann-Whitney U test was used to compare two

TABLE 1.

Baseline Characteristics

Variable	n = 167
Age (year), mean ± SD	58.34 ± 11.56
Sex, n (%)	
Male	68 (40.7)
Female	99 (59.3)
History of Hypertension, n (%)	85 (50.9)
Systolic blood pressure *(mmHg)*, median (Min.-Max.)	190 (132-260)
Diastolic blood pressure *(mmHg)*, median (Min.-Max.)	110 (80-190)
Hypertensive Crisis, n (%)	
Hypertensive urgencie	13 (7.8)
Hypertensive emergencies	154 (92.2)
Target Organ Damage, n (%)	
Cardiac	38 (22.8)
Pulmonary Edema	6 (3.6)
Myocardial Infarction	7 (4.2)
Congestive Heart Failure	25 (15.0)
Cerebral Event	107 (64.1)
Stroke Hemorrhagic	22 (13.2)
Stroke Non-Hemorrhagic	84 (50.3)
Transient Ischemic Attack	1 (0.6)
Renal Failure	11 (6.6)
Neutrophil (*/μL*), median (Min.-Max.)	74 (47-94)
Lymphocyte (*/μL*), median (Min.-Max.)	18 (3-40)
neutrophil-to-lymphocyte ratio, median (Min.-Max.)	4.33 (1.18-31.33)
serum creatininer (*mg/dL*), median (Min.-Max.)	1.30 (1.00-21.00)
estimated glomerular function rate (*ml/min/1.73m²*), median (Min.-Max.)	51.70 (2.5-112.9)

continuous variables with an abnormal distribution. When comparing more than two continuous variables with normal distribution, one-way analysis of variance (ANOVA) was used; when the data were not normally distributed, the Kruskal-Wallis test was used. Chi-square test was applied for the comparison of the categorical variable. Spearman's rho test was performed to analyze the associations between NLR and eGFR. A *p*-value below 0.05 indicated a significant difference.

Results

A total of 167 patients were analyzed, 3 patients were excluded due to incomplete data in medical records. Baseline characteristics are shown in Table 1. Hypertensive crisis proportion was lower in male (40.7%, 68 patients) than females (59.3%, 99 patients). The HE incidence was higher (92.2%, 154 patients) with cerebral events as the most frequent target organ damage (64.1%, 107 patients).

Clinical characteristics of hypertensive emergencies and hypertensive urgencies of patients are shown in Table 2. Lymphocyte count and NLR were significantly lower in HE patients (p=0.029 and p=0.033, respectively), while eGFR was significantly higher in HE patients (p=0.046). There were no significant differences in age, sex, history of hypertension, BP, neutrophil count, and SCr between HU and HE patients.

Table 3 shows clinical characteristics of target organ damage in HE. SBP was significantly different in cerebral event, cardiac, and renal groups (p=0.023), with the highest median of SBP in the cerebral event 193 (154-260), followed by cardiac 190 (132-230) and renal 190 (150-213). In contrast, DBP was not significantly different. SCr and eGFR were also significantly different (p=0.000 and p=0.000, respectively). The highest median of SCr was 6.90 (1.00-21.00) followed by the lowest median of eGFR 7.40 (2.50-53.70) in the renal group.

Correlations were calculated between NLR and eGFR in HU and HE patients with analysis in each group of target organ damage (Table 4). Significance correlation was found in the HE and cardiac event groups (p=0.028 and p=0.030, respectively). The correlation between NLR and eGFR in HE patients was negative, with a very weak score (r=-

Table 2.

Clinical Characteristics of Hypertensive Emergencies and Hypertensive Urgencies

Variable	Hypertensive urgencies	Hypertensive emergencies	p
Age (year), mean ± SD	55.54 ± 12.91	58.57 ± 11.45	0.488[a]
Sex			
Male	3 (23.1%)	65 (42.2%)	0.244[b]
Female	10 (76.9%)	89 (57.8%)	
History of Hypertension			
Yes	4 (30.8%)	81 (52.6%)	0.156[b]
No	9 (69.2%)	73 (47.4%)	
Systolic blood pressure (*mmHg*), median (min.-max.)	190 (156-215)	191 (132-260)	0.577[c]
diastolic blood pressure (*mmHg*), median (min.-max.)	114 (90-180)	110 (80-190)	0.511[c]
Neutrophil (*/μL*), median (min.-max.)	70 (49-84)	75 (47-94)	0.072[c]
Lymphocyte (*/μL*), median (min.-max.)	24 (11-40)	17 (3-40)	0.029[c]*
Neutrophil-to-lymphocyte ratio, median (min.-max.)	2.92 (1.23-7.64)	4.40 (1.18-31.33)	0.033[c]*
Serum creatinin (*mg/dL*), median (min.-max.)	1.60 (1-3)	1.20 (1.00-21.00)	0.083[c]
Estimated glomerular function rate (*ml/min/1.73 m²*), median (min.-max.)	43.00 (20.90-78.00)	53.55 (2.50-112.90)	0.046[c]*

Notes: *[a]-Independent-t-test: p<0.05; significant, [b]-Chi-square test: p<0.05; significant. [c]-Mann-Whitney U test: p<0.05; significant*

TABLE 3.

Clinical Characteristics of Target Organ Damage in hypertensive emergencies

Variable	Cerebral Event n=107	Cardiac n=38	Renal n=11	p
Age (year), mean ± SD	59.35 ± 10.93	58.21 ± 12.22	55.09 ± 14.36	0.499[a]
Sex				
Male	49 (45.8%)	13 (34.2%)	3 (27.3%)	0.278[b]
Female	58 (54.2%)	25 (65.8%)	8 (72.7%)	
History of Hypertension				
Yes	60 (56.1%)	19 (50.0%)	4 (36.4%)	0.414[b]
No	47 (43.9%)	19 (50.0%)	7 (63.6%)	
Systolic blood pressure (*mmHg*), median (min.-max.)	193 (154-260)	190 (132-230)	190 (150-213)	0.023[c]*
Diastolic blood pressure (*mmHg*), median (min.-max.)	111 (80-190)	110 (90-144)	110 (87-126)	0.161[c]
Neutrophil (*/μL*), median (min.-max.)	75 (53-94)	73 (47-90)	82 (60-93)	0.065[c]
Lymphocyte (*/μL*), median (min.-max.)	17 (3-39)	19 (3-40)	14 (4-31)	0.076[c]
Neutrophil-to-lymphocyte ratio, median (min.-max.)	4.56 (1.36-31.33)	3.84 (1.18-27.00)	5.86 (1.94-23.25)	0.080[c]
Serum creatinine (*mg/dL*), median (min.-max.)	1.20 (1.00-17.00)	1.30 (1.00-5.00)	6.90 (1.00-21.00)	0.000[c]*
Estimated glomerular function rate (*ml/min/1.73 m²*), median (min.-max.)	54.1 (3.0-107.1)	54.0 (14.2-112.9)	7.40 (2.50-53.70)	0.000[c]*

NOTES: *[a]-One-Way ANOVA: p<0.05; significant; [b]-Chi-square test: p<0.05; significant; [c]-Kruskal-Wallis test: p<0.05; significant*

TABLE 4.

Correlation between NLR and eGFR in HU and HE patients

Variable	Neutrophil-to-lymphocyte ratio Med (min.-max.)	Estimated glomerular function rate Med (min.-max.)	p	r	r^2
Hypertensive urgencies	2.92 (1.23-7.64)	43.00 (20.90-78.00)	0.109	-0.465	0.216
Hypertensive emergencies	4.40 (1.18-31.33)	53.55 (2.50-112.90)	0.028*	-0.177	0.208
Cerebral Event	4.56 (1.36-31.33)	54.10 (3.00-107.10)	0.422	-0.078	0.006
Cardiac	3.84 (1.18-27.00)	54.00 (14.2-112.9)	0.030*	-0.357	0.127
Renal	5.86 (1.94-23.25)	7.40 (2.50-53.70)	0.629	0.164	0.190

NOTE: **Spearman's rho test: p<0.05; significant*

0.177). The coefficient of determination 0.208 means that each increase in NLR will be followed by a decrease of eGFR 20.8% in HE patients. The correlation between NLR and eGFR in HE patients with cardiac as the target organ damage was also negative with a weak score (r=-0.357). The coefficient of determination 0.127 means that each increase in NLR will be followed by a decrease of eGFR 12.7% in HE patients with cardiac as target organ damage.

We also performed NLR and eGFR correlation analysis in all patients. The result was significant (p=0.036), with a very weak score (r=-0.163) and coefficient of determination 0.266. Each increase in NLR was followed by a decrease of eGFR 26.6% (Figure).

DISCUSSION

This study demonstrated that increased NLR had a significant risk of decreased renal function, which was evaluated by eGFR. The result of the analysis was significant, with a very weak score.

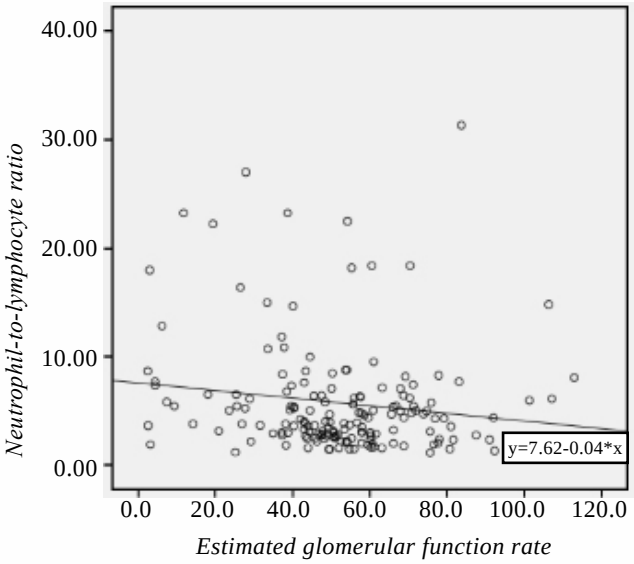

Figure 1. *Correlation between Neutrophil-to-lymphocyte ratio and Estimated glomerular function rate*

In this study, each increase in NLR will be followed by a decrease in eGFR. SCr and eGFR were also significantly different. This study also shows a significant correlation between the HE groups and the cardiac event group. The correlation between NLR and eGFR in HE patients was negative, with a very weak score. These results are also similar in HE patients with cardiac as the target organ damage.

This study shows that increasing in NLR will be followed by a decrease of eGFR in HE patients with cardiac as target organ damage. Increased NLR levels were found to be a significant predictor of adverse renal outcomes in one investigation. It was caused by increasing oxidative stress or inflammatory markers [*Yoshitomi R et al., 2019*]. NLR could be a valuable marker to predict renal dysfunction, cardiovascular complication, and stress response of the patients. This ratio is composed of two different pathways of immunity. The neutrophil count shows the inflammation process [*Yuan Q et al., 2019*]. Lymphocyte count shows general health and physiological stress [*Sevencan N, Ozkan A, 2018*]. In a four-year follow-up research, NLR was identified as an independent predictor of the development of significant adverse cardiac events in the diabetic group [*Azab B et al., 2012*]. This parameter is inexpensive to collect, generally available, and somewhat stable when compared to other parameters (ex: leukocyte, neutrophil, lymphocyte, monocyte, and platelet counts) [*Akase T et al., 2020*].

In this study, hypertensive crisis proportion is lower in males than females. The HE incidence is higher in patients with cerebral events as the most frequent target organ damage. SBP is significantly different in the cerebral event, cardiac, and renal groups, with the highest median of SBP in the cerebral event 193 (154-260), followed by cardiac 190 (132-230) and renal 190 (150-213). In contrast, DBP was not significantly different. In HE patients, lymphocyte count and NLR were much lower, although eGFR was significantly greater. Between HU and HE patients, there were no significant differences in age, sex, history of hypertension, blood pressure, neutrophil count, or SCr.

This study had some limitations. The selection of subjects was limited only to one hospital. The data were collected from medical records, so the information was based on the previous examiners, and the baseline NLR and eGFR were not known due to the cross-sectional study design. Cohort studies need to be done to decrease the bias of information and standardize data collection procedures.

Conclusion

This study suggested a relationship between the increasing NLR with decreasing eGFR in hypertensive crisis patients. Further studies are needed to evaluate whether the increase in NLR was an acute or chronic disease process.

Acknowledgments: *This paper is made in the term of the internship program. We want to thank Medical Record employees in Citra Husada Hospital for all help in data collecting processes and Citra Husada Hospital for allowing us to make this paper done.*

REFERENCES

1. *Akase T, Kawamoto R, Ninomiya D, Kikuchi A, Kumagi T* (2020). Neutrophil-to-lymphocyte ratio is a predictor of renal dysfunction in Japanese patients with type 2 diabetes. Diabetes Metab. Syndr. Clin. Res. Rev. 14(4):481–487. doi:10.1016/j.dsx.2020.04.029
2. *Azab B, Chainani V, Shah N, Mcginn JT* (2012). Neutrophil–lymphocyte ratio as a predictor of major adverse cardiac events among diabetic population: a 4-year follow-up study. Angiology. 64(6):456–465. doi: 10.1177/0003319712455216
3. *Azikin A, Mangarengi F, Bahrun U* (2018). Relationship between neutrophil-lymphocyte ratio and decreased glomerular fiiltration rate in diabetic. Indones. J. Clin. Pathol. Med. Lab. 24(2):131–135. file:///C:/Users/User/Downloads/RELATIONSHIP_BETWEEN_NEUTROPHIL_-_LYMPHOCYTE_RATIO%20(1).pdf
4. *Bramantya RR, Mohani CI, Soelistijo SA* (2020). Interleukin-6 associated with insulin resistance in non-diabetic predialysis chronic kidney disease patients. Syst. Rev. Pharm. 11(4):354–358. https://www.sysrevpharm.org/articles/interleukin6-associated-with-insulin-resistance-in-nondiabetic-predialysis-chronic-kidney-disease-patients.pdf
5. *Centers for Disease Control and Prevention* (2018). Indonesia top 10 causes of death. https://www.cdc.gov/globalhealth/countries/indonesia/pdf/indonesia-fs.pdf
6. *Chakraborty S* (2017). Hypertension urgencies & emergencies. In: Medicine Update The Association of Physicians of India. Retrieved from https://apiindia.org/wp-content/uploads/pdf/medicine_update_2017/mu_139.pdf
7. *Derhaschnig U, Testori C, Riedmueller E, Hobl EL, Mayr FB, Jilma B* (2014). Decreased renal function in hypertensive emergencies. J. Hum. Hypertens. 28(7):427–431. doi: 10.1038/jhh.2013.132
8. *Haas AR, Marik PE* (2006). Current diagnosis and management of hypertensive emergency. Semin. Dial. 19(6):502–512. doi: 10.1111/j.1525-139X.2006.00213.x
9. *Hertanto DM, Ashariati A, Mardiana N, Santoso D* (2019). The relationship between estimated glomerular filtration rate (EGFR) with hepcidin in CKD patients that have not undergone dialysis. Indian J. Public Heal. Res. Dev. 10(4):1163–1166.
10. *Lukito AA, Harmeiwaty E, and Hustrini NM* (2019). Indonesian Society of Hypertension. Hypertension Management Consensus 2019. Published in Indonesian, Jakarta. https://fliphtml5.com/acptd/ebpd/basic/
11. *Levey AS, Bosch JP.,Lewis JB.,Greene T., Rogers N., Roth D* (1999). A More Accurate Method To Estimate Glomerular Filtration Rate from Serum Creatinine: A New Prediction Equation. Annals of Internal Medicine, 130(6), 461-470. doi:10.7326/0003-4819-130-6-199903160-00002
12. *Paini A, Aggiusti C, Bertacchini F, Rosei CA, Maruelli G, Arnoldi C, et al* (2018). Definitions and epidemiological aspects of hypertensive urgencies and emergencies. High Blood Press. Cardiovasc. Prev. 25(3):241-244. doi: 10.1007/s40292-018-0263-2. Epub 2018 Jun 18
13. *Rochmanti M, Tsania NM, Salsabila SA, Thaha M* (2020). Anti-inflammatory and antioxidative therapeutic approach in chronic kidney disease with statin consumption. Sys Rev Pharm. 11(11):1070–1079. *Salim HM, Alam IP, Kharisma WD* (2020). Organ damage due to elevation of blood pressure on NaCl-induced. Biomol. Heal. Sci. J. 3(2):108.
14. *Saputri FB, Fauziah D, Hindariati E* (2020). Prevalence proportion of patient with coronary heart disease in inpatient room of RSUD Dr. Soetomo Surabaya in 2017. Biomol. Heal. Sci. J. 3(2):95-100. http://dx.doi.org/10.20473/bhsj.v3i2.22094
15. *Sevencan NO, Ozkan AE* (2018). Associations between neutrophil/lymphocyte ratio, platelet/lymphocyte ratio, albuminuria and uric acid and the estimated glomerular filtration rate in hypertensive patients with chronic kidney disease stages 1-3. Arch Med Sci. 15(5):1232–1239. doi: 10.5114/aoms.2018.76262
16. *Sgueglia GA, Varounis C, Katsi V, Nihoyannopoulos P, Lekakis J, Tousoulis D* (2017). Cardiovascular hypertensive crisis : recent evidence and review of the literature. Front. Cardiovasc. Med.3:51. doi: 10.3389/fcvm.2016.00051

17. *Sulistiawati S, Dewanti L, Pratama AP, Atika A, Fatmaningrum W, Nuswantoro D, et al* (2020). Profile and lifestyle of hypertensive patients, cardiovascular comorbidity, and complications in a primary health center in Surabaya, Indonesia. Open Access Maced. J. Med. Sci. 8(E)219–223.

18. *Suprapti B, Nilamsari WP, Rachmania, Widodo, Alderman C* (2019). Medical problems in patients with chronic kidney disease undergoing hemodialysis and their therapy. J. Basic Clin. Physiol. Pharmacol. 30(6):jbcpp-2019-0250.

19. *Thaha M, Kadariswantiningsih IN, Empitu MA* (2019). Association of high blood pressure with elevated oxidative stress, inflammatory marker and albuminuria in chronic kidney disease patients. J. Med. 20(1):12–18. doi:10.3329/jom.v20i1.38815

20. *Thaha M, Widiana IGR* (2019). The role of inflammation in chronic kidney disease. Indones. J. Kidney Hypertens. 22(3):4–13.

21. *Yuan Q, Wang J, Peng Z, Zhou Q, Xiao X, et al* (2019). Neutrophil-to-lymphocyte ratio and incident end-stage renal disease in chinese patients with chronic kidney disease: results from the chinese cohort study of chronic kidney disease (C-STRIDE). J. Transl. Med. 17(1):86. doi: 10.1186/s12967-019-1808-4

22. *Unger T, Borghi C, Charchar F, Khan NA, Poulter NR, Prabhakaran D, et al* (2020). 2020 International society of hypertension global hypertension practice guidelines. Hypertension. 2020;75(6):1334-1357. doi: 10.1161/HYPERTENSIONAHA.120.15026

23. *Yoshitomi R, Nakayama M, Sakoh T, Fukui A, Katafuchi E, Seki M* (2019). High neutrophil/lymphocyte ratio is associated with poor renal outcomes in Japanese patients with chronic kidney disease. Ren. Fail. 41(1):238–243. doi: 10.1080/0886022X.2019.1595645

THE NEW ARMENIAN MEDICAL JOURNAL
Vol.15 (2021). No 4

CONTENTS

The Journal is founded by
Yerevan State Medical
University after M. Heratsi.

Address for correspondence:

Yerevan State Medical University
2 Koryun Street, Yerevan 0025,
Republic of Armenia

Phones:

(+37410) 582532 YSMU
(+37410) 580840 Editor-in-Chief
Fax: (+37410) 582532
E-mail: namj.ysmu@gmail.com, ysmiu@mail.ru
URL: http//www.ysmu.am

Our journal is registered in the databases of Scopus, EBSCO and Thomson Reuters (in the registration process)

SCOPUS EBSCO THOMSON REUTERS

Copy editor: Tatevik R. Movsisyan

Printed in "collage" LTD
Director: A. Muradyan
Armenia, 0002, Yerevan,
Saryan St., 4 Building, Area 2
Phone: (+374 10) 52 02 17,
E-mail: collageltd@gmail.com

OFFICIAL PUBLICATION OF THE YEREVAN STATE MEDICAL UNIVERSITY