Acta Clin Croat 2023; 62:627-634
doi: 10.20471/acc.2023.62.04.7

Original Scientific Paper

EVALUATION OF THE RELATIONSHIP BETWEEN PARAOXONASE-1 ENZYME ACTIVITY, AFFECTED VOLUME, AND STROKE ONSET IN ACUTE ISCHEMIC STROKE PATIENTS

Serhat Atmaca[1], Suna Eraybar[1], Yasemin Nennicioglu[2], Melih Yuksel[1] and Halil Kaya[1]

[1]Emergency Department, Turkey University of Health Sciences, Bursa, Turkey;
[2]Emergency Service, Nusaybin Government Hospital, Mardin, Turkey

SUMMARY – Acute ischemic stroke has an important place among emergency department admissions. After a rapid diagnosis of the patient admitted with a preliminary diagnosis of stroke, the necessary examination and first intervention should be performed, then diagnostic evaluation should be made. The aim of this study was to investigate whether paraoxonase-1 (PON-1) enzyme activity, measured in patients whose stroke onset is known, is a diagnostic biomarker for stroke by evaluating the relationship between stroke diagnosis, ischemic area volume, and stroke onset. The study included 100 patients over age 18 admitted to the emergency department with acute stroke symptoms, with known stroke onset and diagnosed as acute occlusive cerebrovascular disease within 24 hours, and 100 healthy control subjects. After initial evaluation, computed tomography of the brain and magnetic resonance imaging, PON-1 activity was measured by colorimetric method in patient serum. Comparison of PON-1 levels between the two groups yielded a statistically significant difference. There was a significant decrease in serum PON-1 values in patient group as compared with control group ($p<0.001$). According to these results, no significant relationship was found between the affected ischemic area of the brain, PON-1, and ischemic volume values. The possible relationship between PON-1 values and stroke onset was compared and no statistically significant difference was found ($p=0.311$). The relationship between PON-1 enzyme activity and diagnosis of acute ischemic stroke was found to be significant. PON-1 was found to be lower in stroke patients but no correlation was found with ischemic area volume.

Key words: *Emergency department; Stroke; Paraoxonase-1 enzyme activity; Stroke onset; Ischemic area volume*

Introduction

Stroke is a complex multifactorial disease and one of the most important causes of mortality and morbidity in adults in developed countries. In studies conducted in the general population, 70%-80% of all strokes are found to be ischemic[1]. Oxidative stress has an important role in the pathophysiology of stroke and increases the production of free oxygen radicals and constitutes an independent risk factor for ischemic stroke[2]. Oxidative stress is probably one of the mechanisms involved in the process of neuronal damage induced by ischemia and reperfusion, probably due to lipid peroxidation[3].

Brain tissue loses its function with oxygen radical damage. The lack of energy due to the lack of oxygen and glucose disrupts ion balance. Glutamate is

Correspondence to: *Assoc. Prof. Suna Eraybar, MD*, University of Health Sciences, Bursa Faculty of Medicine, Bursa Şehir Training & Research Hospital
Emergency Department, T.C. Sağlık Bakanlığı Bursa Şehir Hastanesi Doğanköy Mahallesi, 16110 Nilüfer/Bursa, Turkey
E-mail: sunaeraybar@gmail.com

Received May 3, 2021, accepted June 29, 2021

disrupted and calcium input into the cell increases. As a result of all these mechanisms, depending on the onset and severity of ischemia, necrosis or apoptosis or both together can cause death of brain cells[4].

When all laboratory and radiological results are evaluated, stroke diagnosis can be made clinically and radiologically, but there is no definitive laboratory parameter for the diagnosis of stroke. Medical thrombolytic therapy and thrombectomy are the most effective treatment methods for stroke patients, but early treatment is one of the most important factors for success of these methods. Considering the studies performed, there is still no laboratory parameter that can confirm definitive diagnosis of stroke disease. However, many studies are being conducted to provide effective and early treatment. Evaluation of paraoxonase-1 (PON-1) enzyme activity is one of these studies for the diagnosis of stroke.

Paraoxonase, synthesized in the liver, is a serum esterase which is capable of hydrolyzing the active metabolite of paraoxon, parathion, an organic phosphorus insecticide[5,6]. Human serum PON-1 is a protein including 354 amino acids with a molecular weight of 43 kDa and is physically associated with high-density lipoprotein (HDL)[7,8]. PON has two main functions, i.e., to participate in detoxification of organophosphate compounds such as paraoxon, a pesticide, and to protect low-density lipoprotein from oxidation by hydrolyzing lipid peroxides[9]. The role of PON-1 genotypes in preventing atherosclerosis is still controversial and it is widely accepted that individuals with QQ low activity genotypes (homozygous AA) have a higher risk of atherosclerosis. Another feature of paraoxonase is that it has arylesterase activity which does not show polymorphism[10]. The measurement of paraoxonase enzyme activity is based on the spectrophotometric measurement of 4-nitrophenol formed by enzymatic hydrolysis of organophosphates such as paraoxon as substrate[11-13].

Changes in HDL size and shape significantly affect PON-1 binding affinity and stability, resulting in a decrease in antioxidative capacity[14]. PON-1 has an important physiological role in lipid metabolism by trying to prevent the development of atherosclerosis. Many studies have provided important evidence that PON-1 has a protective effect against atherosclerosis[14-16]. Recently, it has been reported that changes in the structure or serum level of PON-1 may also be a risk factor for cerebrovascular diseases[15].

The aim of this study was to assess the utility of PON-1 as a diagnostic biomarker in stroke patients by evaluating the relationship between PON-1 enzyme activity, stroke onset, and ischemic area volume in acute stroke patients.

Patients and Methods

Our study was conducted prospectively in the emergency department of our hospital over a 3-month period. Written approval was obtained from the Faculty Ethics Committee (2011-KAEK_25 2018/07-44). The study included 100 patients over age 18 who were admitted to the emergency department with acute stroke symptoms within 24 hours, diagnosed as acute occlusive cerebrovascular disease after initial evaluation and necessary investigations by emergency physician, and whose onset time was known. Control group consisted of 100 patients aged over 18 who were admitted to the emergency department with a non-stroke symptom, had no history of chronic disease, had a similar age distribution, and voluntarily participated in the study. Demographic characteristics such as age and gender, comorbidities, history of smoking, presence of dysrhythmia on electrocardiogram, and the National Institutes of Health Stroke Scale (NIHSS) scores were recorded.

Patients under age 18 with stroke onset of more than 24 hours, with unknown stroke onset, those with a history of stroke and whose stroke history could not be evaluated were not included in the study.

After initial evaluation, brain computed tomography and magnetic resonance imaging (MRI) examinations, 3 mL of blood was collected into an anticoagulant-free biochemistry tube to measure PON-1 enzyme activity of the patients included in the study. The samples were centrifuged at 7000 rpm for 7 minutes after waiting for 30 minutes at room temperature. Serum was transferred to Eppendorf tubes and kept at -20 °C until the number of patients was completed. PON enzyme activity was measured by colorimetric method.

The affected lobe, infarct area, and ischemic brain tissue volume were measured on MRI images and diffusion-weighted images obtained during the diagnostic process. The volume of ischemic tissue was extracted in four planes as oblique, axial, sagittal and coronal by using a detailed modeling system using point-based and appropriate calibration paint on three dimensional imaging models to form Exponential

ADC imaging obtained on selected patients. The volume area of the infarcted tissue resulting from this extraction was calculated in three dimensions. After the measurements, it was checked by the radiologist.

All data were calculated using SPSS for Windows 21.0. Descriptive values were given as mean ± standard deviation for continuous variables and n and % for categorical variables. Normality test of numerical values was computed by the Kolmogorov-Smirnov test. Mann Whitney U test was used in cases where numerical variables did not show normal distribution in two independent group comparisons. Kruskal Wallis test was used for comparisons of more than two independent groups. Pearson χ^2 was used in 2x2 tables and Fisher Freeman Halton test was used in RxC tables on determining differences between categorical variables. The level of statistical significance was set at $p<0.05$.

Results

The study included 100 patients with acute obstructive cerebrovascular disease and 100 healthy subjects. Six patients in the patient group were excluded from evaluation because the ischemic area volume could not be calculated with MRI results. One subject in the control group was excluded from the study because the paraoxonase enzyme activity could not be measured in serum sample. Table 1 shows demographic data of the study participants.

To determine the change of PON-1 activity in the patient group, PON-1 levels were compared between the patient and control groups, yielding a statistically significant difference. There was a significant decrease in serum PON-1 values of the patient group compared to the control group ($p<0.001$) (Table 2).

The affected ischemic areas of the brain were categorized in the patient group. The minimum, maximum

Table 1. Demographic characteristics of the study and control groups

		Patient				Control			
		M	SD	n	%	M	SD	n	%
Age (yrs)		68.28	13.43			42.02	10.72		
Gender	Male			50	53.2			45	45.5
	Female			44	46.8			54	54.5
Smoking	No			60	63.8			99	100.0
	Yes			34	36.2			0	0.0
Hypertension	No			38	40.4			99	100.0
	1-5 (yrs)			19	20.2			0	0.0
	6-10 (yrs)			18	19.1			0	0.0
	>11 (yrs)			19	20.2			0	0.0
Diabetes	No			64	68.1			99	100.0
	1-5 (yrs)			13	13.8			0	0.0
	6-10 (yrs)			10	10.6			0	0.0
	>11 (yrs)			4	4.3			0	0.0
	Yes (onset unknown)			3	3.2			0	0.0
Coronary artery disease	No			69	73.4			99	100.0
	1-5 (yrs)			13	13.8			0	0.0
	6-10 (yrs)			10	10.6			0	0.0
	>11 (yrs)			2	2.1			0	0.0
Dysrhythmia on electrocardiography	Yes			10	10.6			0	0.0
	No			84	89.4			0	0.0

M = mean; SD = standard deviation

Table 2. Comparison of patient and control groups according to PON-1 levels

		n	Mean	SD	p*
PON-1	Patient	94	216.27	135.31	0.003
	Control	99	297.95	211.41	

*Mann Whitney U test; SD = standard deviation; PON-1 = paraoxonase-1

Table 3. Comparison of PON-1 and ischemic volume values in the affected ischemic area in the brain

	Affected area	n	Mean	SD	SE Lower	95% CI		Min	Max
						Upper			
PON-1	Right	40	236.58	148.70	23.51	189.02	284.13	12	734
	Left	45	209.38	126.04	18.79	171.51	247.25	40	672
	Bilateral	5	168.20	121.49	54.33	17.34	319.06	77	374
	Brainstem	4	150.75	107.81	53.90	-20.80	322.30	68	309
	Total	94	216.27	135.31	13.95	188.55	243.98	12	734
Ischemic volume	Right	40	35.16	65.88	10.41	14.09	56.23	0.14	279.54
	Left	45	40.41	81.19	12.10	16.02	64.81	0.14	357.87
	Bilateral	5	5.95	7.10	3.17	-2.87	14.77	0.95	18.17
	Brainstem	4	0.55	0.40	.20	-0.09	1.19	0.13	1.00
	Total	94	34.65	71.07	7.33	20.09	49.20	0.13	357.87

Kruskal Wallis test; PON-1 = paraoxonase-1; SD = standard deviation; SE = standard error; CI = confidence interval

Table 4. Comparison of PON-1 levels according to the affected lobe

Affected lobe	n	PON-1 Mean	SD	SE	95% CI			
					Lower	Upper	Min	Max
Frontal	3	160.67	110.64	63.87	-114.18	435.51	88	288
Temporal	4	149.25	131.62	65.81	-60.19	358.69	12	322
Parietal	40	223.85	118.59	18.75	185.92	261.78	72	488
Occipital	3	202.67	90.65	52.33	-22.52	427.85	98	256
Brainstem	5	165.20	98.79	44.18	42.53	287.87	68	309
MCA irrigation area	20	243.95	171.76	38.40	163.56	324.34	83	734
Frontal + parietal	9	227.78	188.79	62.93	82.66	372.90	40	672
Cerebellum	8	193.50	126.22	44.62	87.97	299.03	77	412
Parietal + occipital	2	192.50	12.02	8.50	84.50	300.50	184	201
Total	94	216.27	135.31	13.95	188.55	243.98	12	734

Kruskal Wallis test; PON-1 = paraoxonase-1; SD = standard deviation; SE = standard error; CI = confidence interval; MCA = middle cerebral artery

and mean values of PON-1 and ischemic volume variables were calculated according to the condition of the affected ischemic area of the brain. According to these data, no statistically significant difference was found between the affected ischemic area of the brain and PON-1 and ischemic volume values (Table 3).

The patient group was categorized according to the affected ischemic brain lobes. The minimum, maximum and mean values of PON-1, NIHSS and ischemic volume variables were calculated according to the affected ischemic brain lobe. No statistically significant difference was found according to these data (Kruskal

Table 5. Comparison of PON-1 levels according to stroke onset

Stroke onset	n	PON-1 Mean	SD	SE	95% CI			
					Lower	Upper	Min	Max
<1 hour	7	269.14	170.30	64.36	111.64	426.65	83	488
1-2 hours	13	184.23	90.03	24.97	129.83	238.64	68	342
2-3 hours	14	285.86	217.55	58.14	160.24	411.47	83	734
3-4 hours	10	148.10	101.74	32.17	75.31	220.89	12	316
4-5 hours	8	182.63	96.29	34.04	102.12	263.13	40	337
>5 hours	42	216.81	110.63	17.07	182.33	251.29	88	473
Total	94	216.27	135.31	13.95	188.55	243.98	12	734

Kruskal Wallis test; PON-1 = paraoxonase-1; SD = standard deviation; SE = standard error; CI = confidence interval

Table 6. Comparison of PON-1 levels according to ischemic volume

Ischemic volume	n	PON-1 mean	SD	SE	95% CI		Min	Max
					Lower	Upper		
<1 cm^3	28	200.50	109.85	20.76	157.90	243.10	68	457
1-10 cm^3	33	214.79	124.11	21.60	170.78	258.80	12	488
>10 cm^3	33	231.12	164.85	28.69	172.66	289.58	83	734
Total	94	216.27	135.31	13.95	188.55	243.98	12	734

Kruskal Wallis test; PON-1 = paraoxonase-1; SD = standard deviation; SE = standard error; CI = confidence interval

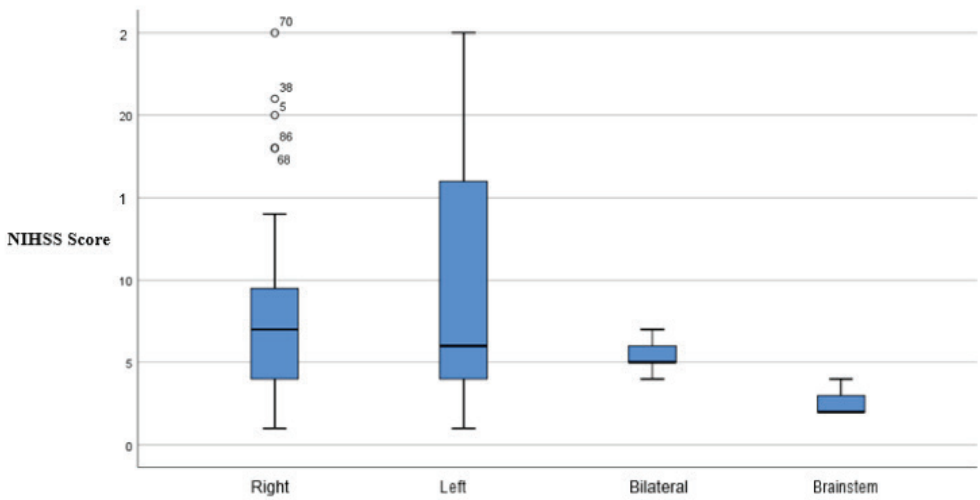

Fig. 1. Comparison of NIHSS scores according to the affected ischemic area of the brain.

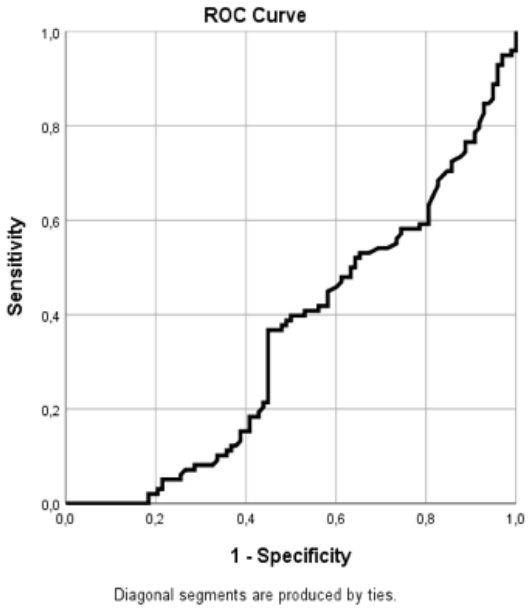

Fig. 2. ROC curve for paraoxonase-1 variable.

Wallis H: 4.207, p=0.649) (Table 4). Comparison of NIHSS scores according to the affected ischemic area of the brain is illustrated in Figure 1.

The patient group was categorized in terms of stroke onset. The minimum, maximum and mean values of PON-1 were calculated. No statistically significant difference was found between stroke onset and PON-1 values (Kruskal Wallis H: 4.775, p=0.311) (Table 5). PON-1 values were compared with affected ischemic volume measurements in patients with acute stroke. There was no statistically significant difference between these two variables (Kruskal Wallis H: 0.422, p=0.810 (Table 6).

In the ROC analysis to determine the efficacy of PON-1 in predicting ischemic stroke, the area under the curve was 0.352; p<0.001; and 95% confidence interval 0.276-0.429 (Fig. 2).

Discussion

The diagnosis of ischemic stroke is clinically predictable but it cannot be diagnosed without radiological imaging. In this disease, appropriate treatment should be started as soon as possible. However, the desired diagnostic speed could not be reached and no diagnostic laboratory marker could be obtained. In our study, diagnostic value of PON-1 in ischemic stroke and its relationship with ischemic area volume and stroke onset was evaluated.

In the study group, 53% of patients were male and 47% were female. Their mean age was 68.2±13.43 years. When other studies were examined, similar results were observed. In many studies, male gender ratios in ischemic stroke patients vary between 48.3% and 73.4%, and male gender is considered as a risk factor in the stroke group[17,18].

The study conducted in Framingham showed that smoking is an important factor for the increase in the incidence of stroke and appears to be an independent risk factor. In the study, the stroke risk increased 1.62 times for females and 1.42 times for males[19]. In another study investigating distribution of stroke risk factors by Rostohar Bijelic *et al.*, it is emphasized that male patients had a significantly higher prevalence of smoking[20]. In an animal model investigated by Reed *et al.*, tobacco use was shown to directly reduce PON-1 and indirectly lower HDL cholesterol levels[21]. The rate of smoking was 42.5% in the patients in our study, but no significant decrease was found on comparison of PON-1, NIHSS and ischemic volume values. In a study conducted by Chawhan *et al.*, PON-1 was evaluated as a risk factor for cerebrovascular disease in patients with ischemic stroke and in healthy subjects[22].

In another study, 48 ischemic stroke patients and 46 healthy control subjects were compared and PON-1 was found to be significantly lower in ischemic stroke patients[23]. In a study including 185 ischemic stroke patients and 185 healthy control subjects, PON-1 was found to be significantly lower in the patient group[24]. In our study, PON-1 was found to be significantly lower in the patient population, which is consistent with the literature.

In a study conducted by Kim *et al.*, PON-1 and ischemic stroke were compared and a statistically significant correlation was found[25]. We investigated the relationship between this significant decrease, the affected area and stroke onset. However, no statistically significant difference was found between PON-1 and the affected brain lobe. PON-1 activity alone in assessing the affected lobe or stroke onset was not sufficient for clinical prediction. As a result of the ROC analysis between PON-1 and ischemic stroke, the area under the curve in the effectiveness of PON-1 in predicting ischemic stroke was 0.352; p<0.001; and 95% confidence interval 0.276-0.429. These results showed the efficacy of PON-1 in predicting ischemic stroke, but no diagnostic cut-off value could be determined.

When the relationship between PON-1 and ischemic area volume and stroke onset, which were the leading parameters of our study, were evaluated, no statistically significant difference was found. However, we believe that more studies are needed to investigate the issue.

Conclusion

This study aimed to investigate the relationship between serum PON-1 and stroke onset and ischemic area volume in acute stroke patients and to evaluate its use as a diagnostic biomarker. Thus, we think that acute stroke patients can be treated faster and more effectively.

In this study, a significant correlation was found between PON-1 enzyme activity and the diagnosis of acute ischemic stroke. In the patient population, PON-1 values were found to be statistically significantly lower, but no significant difference was found between stroke onset and ischemic area volume. No significant cut-off value could be determined for diagnostic PON-1 enzyme activity. We believe that PON-1 enzyme activity may be a valid parameter in the diagnosis of acute ischemic stroke, however, warranting additional studies on this issue.

Limitations

Our study was carried out in 24-hour stroke cases in the emergency department. The number of patients included in our study was limited due to this time limitation considering thrombolytic and thrombectomy indications.

We could not evaluate the efficacy of PON-1 levels in the treatment or survival of stroke patients in the diagnostic process in the emergency department. Further studies are needed on this issue.

References

1. Xian Y, Holloway RG, Chan PS, Noyes K, Shah MN, Ting HH, *et al*. Association between stroke center hospitalization for acute ischemic stroke and mortality. JAMA. 2011; 305(4):373-80. doi: 10.1001/jama.2011.22.
2. Ferretti G, Bacchetti T, Masciangelo S, Nanetti L, Mazzanti L, Silvestrini M, *et al*. Lipid peroxidation in stroke patient. Clin Chem Lab Med. 2008;46(1):113-7. doi: 10.1515/CCLM.2008.011. PMID: 18034641.
3. Chen H, Yoshioka H, Kim GS, Jung JE, Okami N, Sakata H, *et al*. Oxidative stress in ischemic brain damage: mechanisms of cell death and potential molecular targets for neuroprotection. Antioxid Redox Signal. 2011;14(8):1505-17. doi: 10.1089/ars.2010.3576.
4. Walsh KB, Hart K, Roll S, Sperling U, *et al*. Apolipoprotein A-I and paraoxonase-1 are potential blood biomarkers for ischemic stroke diagnosis. J Stroke Cerebrovasc Dis. 2016;25(6):1360-5. doi: 10.1016/j.jstrokecerebrovasdis.2016.02.027.
5. Juretic D, Tadijanovic M, Rekic B, Simean-Rudolf V, Reiner E, Baricic M. Serum paraoxonase activities in hemodialyzed uremic patients: cohort study. Clin Sci. 2001;42:146-50. PMID: 11259735.
6. Marsillach J, Costa LG, Furlong CE. Paraoxonase-1 and early-life environmental exposures. Ann Glob Health. 2016;82(1):100-10. doi: 10.1016/j.aogh.2016.01.009.
7. Précourt LP, Amre D, Denis MC, Lavoie JC, Delvin E, Seidman E, *et al*. The three-gene paraoxonase family: physiologic roles, actions and regulation. Atherosclerosis. 2011 Jan;214(1):20-36. doi: 10.1016/j.atherosclerosis.2010.08.076.
8. Boemi M, Leviev I, Sirolla C, Pieri C, Marra M, James RW. Serum paraoxonase is reduced in type 1 diabetic patients compared to non-diabetic first degree relatives; influence on the ability of HDL to protect LDL from oxidation. Atherosclerosis. 2001 Mar;155(1):229-35. doi: 10.1016/s0021-9150(00)00556-6.
9. Mackness B, Mackness MI, Arrol S, Turkie W, Durrington PN. Effect of the human serum paraoxonase 55 and 192 genetic polymorphisms on the protection by high density lipoprotein against low density lipoprotein oxidative modification. FEBS Lett. 1998 Feb 13;423(1):57-60. doi: 10.1016/s0014-5793(98)00064-7.
10. Rahman MF, Hashad IM, Abou-Aisha K, Abdel-Maksoud SM, Gad MZ. Addressing the link between paraoxonase-1 gene variants and the incidence of early onset myocardial infarction. Arch Med Sci. 2015 Jun 19;11(3):513-20. doi: 10.5114/aoms.2015.52353.
11. Qujeq D, Mahrooz A, Alizadeh A, Masoumi P, Annemohammadzadeh S, Boorank R. Genotype and phenotype of salt-stimulated paraoxonase 1 (PON1) is associated with atherogenic indices in type 2 diabetes. J Diabetes Metab Disord. 2018 Mar 26;17(1):1-10. doi: 10.1007/s40200-018-0332-z.
12. Menini T, Gugliucci A. Paraoxonase 1 in neurological disorders. Redox Rep. 2014 Mar;19(2):49-58. doi: 10.1179/1351000213Y.0000000071.
13. Ceron JJ, Tecles F, Tvarijonaviciute A. Serum paraoxonase 1 (PON1) measurement: an update. BMC Vet Res. 2014 Mar 25;10:74. doi: 10.1186/1746-6148-10-74.
14. Liu JL, Li JP, Wang XL, Yang Y. Relationship between Q192R polymorphisms in paraoxonase 1 gene and young ischemic. Stroke. 2010;90(13):912-6. PMID: 20646512.
15. Lazaros L, Markoula S, Kyritsis A, Georgiou I. Paraoxonase gene polymorphisms and stroke severity. Eur J Neurol. 2010;17(5):757-9. doi: 10.1111/j.1468-1331.2009.02860.x.
16. Shin BS, Oh SY, Kim YS, Kim KW. The paraoxonase gene polymorphism in stroke patients and lipid profile. Acta Neurol Scand. 2008;117(4):237-43. doi: 10.1111/j.1600-0404.2007.00929.x.
17. Bustamante A, Lopez-Cancio E, Pich S, Penalba A, Giralt D, Garcia-Berrocoso T, *et al*. Blood biomarkers for the early diagnosis of stroke: the Stroke-Chip study.

Stroke. 2017;48(9):2419-25. doi: 10.1161/STROKEAHA.117.017076.

18. Chen CY, Weng WC, Wu CL, Huang WY. Association between gender and stroke recurrence in ischemic stroke patients with high-grade carotid artery stenosis. J Clin Neurosci. 2019 Sep;67.62-7. doi: 10.1016/j.jocn.2019.06.021.
19. Oshunbade AA, Yimer WK, Valle KA, Clark D, Kamimura D, White WB, *et al*. Cigarette smoking and incident stroke in blacks of the Jackson Heart study. J Am Heart Assoc. 2020 Jun 16;9(12):e014990. doi: 10.1161/JAHA.119.014990.
20. Rostohar Bijelić B, Petek M, Kadojić M, Bijelić N, Kadojić D. Distribution of stroke risk factors in eastern Croatia. Acta Clin Croat. 2018 Mar;57(1):103-9. doi: 10.20471/acc.2018.57.01.12.
21. Reed RM, Borgan SM, Eberlein M, Goldklang M, Lewis J, Miller M, Navab M, Kim BS. Tobacco smoke exposure reduces paraoxonase activity in a murine model. Int J Biomed Sci. 2017 Mar;13(1):20-5. PMID: 28533733; PMCID: PMC5422641.
22. Chawhan SS, Mogarekar MR, Wagh RV, Das RR, Pramanik SS, Sonune SM, *et al*. Relation of paraoxonase 1, arylesterase and lipid profile in ischemic stroke patients. J Clin Diagn Res. 2015 Nov;9(11):BC01-3. doi: 10.7860/JCDR/2015/15345.6707.
23. Gokdemir MT, Karakilcik AZ, Gokdemir GS. Prognostic importance of paraoxonase, arylesterase and mean platelet volume efficiency in acute ischaemic stroke. J Pak Med Assoc. 2017 Nov;67(11):1679-83. PMID: 29171559.
24. Kotur-Stevuljevic J, Bogavac-Stanojevic N, Jelic-Ivanovic Z, Stefanovic A, Gojkovic T, Joksic J, *et al*. Oxidative stress and paraoxonase 1 status in acute ischemic stroke patients. Atherosclerosis. 2015 Jul;241(1):192-8. doi: 10.1016/j.atherosclerosis.2015.05.016.
25. Kim NS, Kang K, Cha MH, Kang BJ, Moon J, Kang BK, *et al*. Decreased paraoxonase-1 activity is a risk factor for ischemic stroke in Koreans. Biochem Biophys Res Commun. 2007 Dec 7;364(1):157-62. doi: 10.1016/j.bbrc.2007.09.119.

Sažetak

PROCJENA ODNOSA IZMEĐU AKTIVNOSTI ENZIMA PARAOKSONAZE-1, ZAHVAĆENOG VOLUMENA I NASTUPA MOŽDANOG UDARA U BOLESNIKA S AKUTNIM ISHEMIJSKIM MOŽDANIM UDAROM

S. Atmaca, S. Eraybar, Y. Nennicioglu, M. Yuksel i *H. Kaya*

Akutni ishemijski moždani udar zauzima važno mjesto kod prijma na hitni odjel. Poslije brzo postavljene dijagnoze kod bolesnika primljenog s preliminarnom dijagnozom moždanog udara treba provesti potrebne pretrage i prvu intervenciju, nakon čega se provodi dijagnostička procjena. Cilj ovog istraživanja bio je ispitati je li aktivnost enzima paraoksonaze-1 (PON-1) dijagnostički biološki biljeg za moždani udar, mjeren u bolesnika s poznatim vremenom nastupa moždanog udara, i to kroz procjenu odnosa između dijagnoze moždanog udara, volumena područja zahvaćenog ishemijom i nastupa moždanog udara. Studija je uključila 100 bolesnika starijih od 18 godina primljenih na hitni odjel sa simptomima moždanog udara, poznatim vremenom nastupa moždanog udara i dijagnosticiranih kao akutna okluzivna cerebrovaskularna bolest unutar 24 sata te 100 zdravih kontrolnih osoba. Nakon početne procjene, kompjutorizirane tomografije mozga i snimanja magnetskom rezonancijom izmjerena je aktivnost PON-1 u serumu bolesnika kolorimetrijskom metodom. Usporedba razina PON-1 između dvije skupine ispitanika pokazala je značajnu razliku. U skupini bolesnika zabilježen je značajan pad vrijednosti PON-1 u serumu u usporedbi s kontrolnom skupinom ($p<0,001$). Prema ovim rezultatima nije zabilježena značajna povezanost dijela mozga zahvaćenog ishemijom, PON-1 i vrijednosti volumena zahvaćenog ishemijom. Usporedba moguće povezanosti vrijednosti PON-1 i nastupa moždanog udara nije pokazala značajnu razliku ($p=0,311$). Utvrđena je značajna povezanost aktivnosti enzima PON-1 i dijagnoze akutnog ishemijskog moždanog udara. Vrijednosti PON-1 bile su niže kod bolesnika s moždanim udarom, ali nije utvrđena korelacija s volumenom područja zahvaćenog ishemijom.

Ključne riječi: *Odjel hitne medicine; Moždani udar; Aktivnost enzima paraoksonaza-1; Nastup moždanog udara; Volumen ishemijskog područja*