Research Article / Araştırma Makalesi

P Wave Duration and P Wave Dispersion in Electrocardiography of Stroke Patients

İnme Hastalarında Elektrokardiyografide P Dalga Süresi ve P Dalga Dispersiyonu

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Abstract

Background: P-wave duration and P-wave dispersion (PWD) are electrocardiographic (ECG) markers of atrial conduction. This study aimed to compare these parameters in ischemic and hemorrhagic stroke patients versus controls and to examine the effects of stroke subtypes on atrial electrophysiology.
Materials and Methods: This cross-sectional study included , 34 patients with ischemic stroke, 33 patients with hemorrhagic stroke, and 45 healthy subjects as control group. The 12-lead ECGs of all individuals taken at the time of admission to the hospital were analyzed. Maximum P wave duration (Pmax) and PWD were manually measured. All patients underwent brain CT and/or MRI within the first 24-48 hours of presentation, while echocardiography was performed within the first 48-72 hours of hospitalization.
Results: Both stroke groups had significantly increased Pmax (ischemic: 130.3±11.9 ms, hemorrhagic: 118.8±14.7 ms, control: 91.8±11.5 ms; p<0.001) and PWD (ischemic: 40.9±13.3 ms, hemorrhagic: 43.6±13.1 ms, control: 23.8±9.1 ms; p<0.001) values compared to controls. No significant differences were found between the ischemic and hemorrhagic groups.
Conclusions: Ischemic and hemorrhagic stroke patients had prolonged P wave duration and increased PWD during the acute phase of stroke compared to the control group. These findings suggest altered atrial conduction in stroke patients, regardless of stroke subtype, and may indicate an increased risk of atrial fibrillation (AF) in this population.

Keywords: Stroke, P wave duration, P wave dispersion, Atrial fibrillation

Öz

Amaç: P dalga süresi ve P dalga dispersiyonu (PWD), atriyal iletimin elektrokardiyografik (EKG) belirteçleridir. Bu çalışma, iskemik ve hemorajik inme hastalarında bu parametreleri kontrol grubuyla karşılaştırmayı ve inme alt tiplerinin atriyal elektrofizyoloji üzerindeki etkilerini incelemeyi amaçlamıştır.
Materyal ve Metod: Bu kesitsel çalışmaya, 34 iskemik inme hastası, 33 hemorajik inme hastası ve 45 sağlıklı kişi kontrol grubu olarak dahil edildi. Tüm kişilerin hastaneye başvuru kabulünde çekilen 12 derivasyonlu EKG'leri analiz edildi. Maksimum P dalga süresi (Pmax) ve PWD manuel olarak ölçüldü. Tüm hastalara beyin BT ve/ veya MR başvurularının ilk 24–48 saati içinde, ekokardiyografi ise yatışlarının ilk 48–72 saatleri içerisinde çekilmişti.
Bulgular: Her iki inme grubu da kontrol grubuna kıyasla anlamlı derecede artmış Pmax (iskemik: 130.3±11.9 ms, hemorajik: 118.8±14.7 ms, kontrol: 91.8±11.5 ms; p<0.001) ve PWD (iskemik: 40.9±13.3 ms, hemorajik: 43.6±13.1 ms, kontrol: 23.8±9.1 ms; p<0.001) değerlerine sahipti. İskemik ve hemorajik gruplar arasında anlamlı fark bulunmadı.
Sonuç: İskemik ve hemorajik inme hastaları, inmenin akut fazında kontrol grubuna kıyasla uzamış P dalga süresi ve artmış PWD değerlerine sahipti.Bu bulgular, inme alt tipinden bağımsız olarak, inme hastalarında değişmiş atriyal iletimi düşündürmekte ve bu popülasyonda atriyal fibrilasyon (AF) riskinin artmış olabileceğini göstermektedir.

Anahtar Kelimeler: İnme, P dalga süresi, P dalga dispersiyonu, Atriyal fibrilasyon

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Introduction

Stroke is defined by the World Health Organization as a clinical syndrome characterized by rapidly developing signs of focal or global disturbance of cerebral function, lasting more than 24 hours or leading to death, with no apparent cause other than vascular origin (1). Strokes are primarily classified into two main types: ischemic stroke, accounting for 60-80% of cases, and hemorrhagic stroke, which includes intracerebral hemorrhage (10-15%) and subarachnoid hemorrhage (3-10%) (2).

The relationship between stroke and cardiac function is complex and bidirectional. While cardiac disorders, particularly atrial fibrillation (AF), are well-established risk factors for stroke, growing evidence suggests that stroke itself can induce cardiac alterations (3). These alterations may manifest as ECG changes, arrhythmias, or myocardial injury, potentially influencing patient outcomes and management strategies (4).

Among the various ECG parameters, P wave indices have gained attention as markers of atrial conduction and potential predictors of atrial arrhythmias. The P wave represents atrial depolarization, and its characteristics reflect the electrical properties and structural integrity of the atria (5). Two key P wave parameters are:

1. P wave duration: The time taken for complete atrial depolarization, measured from the onset to the offset of the P wave (6).
2. P-wave dispersion (PWD): The difference between the maximum and minimum P wave durations across the 12-lead ECG, representing the heterogeneity of atrial conduction (7).

Prolonged P wave duration and increased PWD have been associated with an elevated risk of AF in various clinical settings (8). For instance, Dilaveris et al. demonstrated that a maximum P wave duration >110 ms and PWD >40 ms were predictive of paroxysmal atrial fibrillation (PAF) (9).

The potential link between stroke and altered atrial electrophysiology is supported by several lines of evidence (10). Stroke can affect autonomic regulation through damage to specific brain regions, such as the insular cortex, potentially influencing cardiac electrophysiology (11). Additionally, the systemic inflammatory response following stroke might contribute to transient or lasting changes in atrial conduction (12).

While previous studies have investigated P wave parameters in various cardiovascular conditions, limited data exist comparing these indices between ischemic and hemorrhagic stroke subtypes (13-15). Most existing studies have focused primarily on ischemic stroke, with little attention given to hemorrhagic stroke or direct comparisons between these distinct pathophysiological entities. Understanding the electrophysiological changes across different stroke subtypes could provide unique insights into the cerebro-cardiac axis and may have important implications for patient management and risk stratification.

This study aims to investigate whether P-wave duration and PWD values are altered in both ischemic and hemorrhagic stroke patients during the acute phase of stroke, potentially providing insights into the electrophysiological changes associated with these stroke types. By including both major stroke subtypes, our study offers a more comprehensive examination of stroke-related atrial conduction changes than previous investigations.

Materials and Methods

Study Design and Population

This cross-sectional study included 67 stroke patients (34 ischemic and 33 hemorrhagic) admitted to our hospital's emergency department between January 2002 and January 2006, and 45 control subjects.

The study protocol was designed to assess acute phase electrocardiographic changes, with all evaluations completed within 48-72 hours of stroke onset.

Stroke patients were selected based on the following criteria: 1- acute neurological deficits confirmed by clinical examination and neuroimaging, 2- normal ECG rhythm (sinus rhythm without evidence of arrhythmias) and normal cardiac enzymes at admission, and 3- echocardiographic findings showing normal left ventricular and atrial diameters with preserved left ventricular ejection fraction (LVEF > 50%). Patients with evidence of coronary artery disease (CAD), history of arrhythmias, or receiving antiarrhythmic medications were excluded.

The control group comprised individuals matched for age and gender who were either 1- hospitalized patients without neurological or cardiac diseases or 2- outpatients referred for coronary angiography due to suspected CAD who had normal results. All control subjects underwent the same cardiac evaluations as the stroke patients to ensure comparability.

This study was approved by the Ethics Committee of Zonguldak Bulent Ecevit University (Date: 12.04.2006, Decision number: 2006/03-9).

Data Collection and Clinical Assessment

Patient data, including demographics, medical history, physical examinations, and clinical risk factors, were collected from electronic records.

All patients underwent brain computed tomography (CT) and/or magnetic resonance imaging (MRI) within 24-48 hours of admission to confirm the diagnosis and determine the stroke subtype. ECG recordings were obtained upon hospital admission, and echocardiography was performed within 48-72 hours of hospitalization.

ECG Analysis

Standard 12-lead ECG recordings were obtained at 25 mm/s and 1 mV/cm amplitude with patients in the supine position. P-wave duration was measured manually in at least 10 derivations by two specialists who were blinded to the patients'

clinical information. The beginning of the P wave was defined as the point where the first visible upward or downward deflection from the isoelectric line occurred. The end of the P wave was defined as the point where the waveform returned to the isoelectric line.

Maximum P-wave duration (Pmax) was defined as the longest P-wave duration measured across all leads. Minimum P-wave duration (Pmin) was the shortest P-wave duration. PWD was calculated as the difference between Pmax and Pmin. If the difference between two measurements exceeded 10%, a third measurement was taken, and the average was used.

Echocardiographic Evaluation

Transthoracic echocardiography was performed for all participants using standard views and techniques. Left ventricular end-diastolic and end-systolic diameters, left atrial diameter, interventricular septal thickness, and ejection fraction were measured according to the American Society of Echocardiography guidelines. All echocardiographic measurements were performed by experienced cardiologists who were blinded to the patients' clinical information.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics V22.0. Continuous variables were expressed as mean ± standard deviation, and categorical variables were expressed as percentages. Chi-square and Fisher's Exact Chi-square test were used for the comparison of categorical variables. The Kruskal-Wallis test was employed for assessing differences in electrocardiographic and echocardiographic quantitative parameters among the three groups, with the Bonferroni correction Mann-Whitney U test for post-hoc pairwise comparisons. P-value < 0.05 was considered statistically significant.

Results

A total of 112 participants were included in the study: 34 with ischemic stroke, 33 with hemorrhagic stroke, and 45 controls. There was no significant difference in gender or age distribution between the groups (p > 0.05). Cardiovascular risk factors were more common in both stroke groups compared with controls. Hypertension was present in 82.4% of ischemic stroke patients, 87.8% of hemorrhagic stroke patients, and 40.0% of controls (p < 0.001). Diabetes mellitus was observed in 41.2% of ischemic stroke patients, 63.6% of hemorrhagic stroke patients, and 11.1% of controls (p < 0.001).

Hyperlipidemia was more common in the ischemic stroke group (64.7%) compared to both the hemorrhagic stroke group (18.2%) and the control group (22.2%) (p < 0.001). There were no significant differences between groups in smoking habits or family history of premature coronary artery disease or stroke (p > 0.05 for both).

Table 1 summarizes the demographic characteristics and risk factors in ischemic stroke, hemorrhagic stroke, and control groups. fraction, were similar in all groups (p > 0.05 for all parameters) (Table 2).

Table 1. Demographic characteristics, risk factors, and P-wave measurements in ischemic stroke, hemorrhagic stroke, and control groups.

Variable	Ischemic Stroke	Hemorrhagic Stroke	Control Group	P value*	P1	P2	P3
Male, n (%)	20 (58.8)	19 (57.5)	26 (57.7)	0.892	0.784	0.622	0.438
Age distribution, n (%)							
< 60 years	13 (38.2)	12 (36.3)	24 (53.4)	0.214	0.464	0.582	0.346
60-69 years	9 (26.6)	10 (30.3)	12 (26.6)	0.918	0.458	0.762	0.547
≥ 70 years	12 (35.2)	11 (33.3)	9 (20.0)	0.762	0.892	0.306	0.732
Risk factors, n (%)							
Diabetes Mellitus	14 (41.2)	21 (63.6)	5 (11.1)	< 0.001	< 0.001	< 0.001	0.324
Hypertension	28 (82.4)	29 (87.8)	18 (40.0)	< 0.001	< 0.001	< 0.001	0.146
Hyperlipidemia	22 (64.7)	6 (18.2)	10 (22.2)	< 0.001	< 0.001	0.383	< 0.001
Smoking	11 (32.4)	15 (45.5)	16 (35.6)	0.503	0.354	0.568	0.236
Family history	4 (11.8)	2 (6.0)	3 (6.7)	0.621	0.475	0.269	0.718
P-wave measurements, mean (range)							
Pmax, ms	130.3 (80-140)	118.8 (80-130)	91.8 (60-100)	< 0.001	< 0.001	< 0.001	0.345
Pmin, ms	89.4 (60-100)	75.2 (60-90)	68.0 (50-80)	< 0.001	< 0.001	< 0.001	0.427
PWD, ms	40.9 (20-80)	43.6 (20-60)	23.8 (10-40)	< 0.001	< 0.001	< 0.001	0.762

Abbreviations: *Pmax: Maximum P-wave Duration; Pmin: Minimum P-wave Duration; PWD: P-wave Dispersion; P1: P-value comparing Ischemic Stroke Group vs Control Group; P2: P-value comparing Hemorrhagic Stroke Group vs Control Group; P3: P-value comparing Ischemic Stroke Group vs Hemorrhagic Stroke Group. *P values < 0.05 considered statistically significant.*

Echocardiographic parameters, including left ventricular end-diastolic diameter, end-systolic diameter, left atrial diameter, interventricular septal thickness, and ejection P-wave parameters showed significant differences between groups. The Pmax was significantly longer in both ischemic stroke (130.3 ± 11.9 ms) and hemorrhagic stroke (118.8 ± 14.7 ms) groups compared to controls (91.8 ± 11.5 ms) (p < 0.001) (Table 1).

Minimum P-wave duration (Pmin) was also significantly prolonged in both stroke groups compared to controls, with the ischemic stroke group showing the highest values (89.4 ± 11.9 ms) followed by the hemorrhagic stroke group (75.2 ± 14.7 ms) and controls (68.0 ± 11.5 ms) (p < 0.001) (Table 1).

Similarly, PWD was significantly increased in both ischemic

stroke (40.9 ± 13.3 ms) and hemorrhagic stroke (43.6 ± 13.1 ms) groups compared with controls (23.8 ± 9.1 ms) ($p < 0.001$) (Table 1).

There were no significant differences in Pmax or PWD between the ischemic and hemorrhagic stroke groups ($p > 0.05$).

Table 2. Echocardiographic parameters in ischemic stroke, hemorrhagic stroke, and control groups.

Echocardiographic parameters, mean (range)	Ischemic Stroke	Hemorrhagic Stroke	Control Group	P value*
LVEF, %	55.85 (50-65)	56.86 (50-67)	64.45 (55-70)	0.178
IVSd, mm	12.6 (7.0-14.0)	12.4 (10.0-14.0)	11.8 (10.0-13.0)	0.294
PWd, mm	12.2 (8.0-13.0)	11.8 (10.0-13.0)	11.2 (9.0-13.0)	0.156
LVEDd, mm	47.1 (37.0-55.0)	44.4 (39.0-52.0)	44.6 (36.0-50.0)	0.183
LVESd, mm	30.6 (22.0-37.0)	29.2 (24.0-34.0)	30.1 (22.0-34.0)	0.715
LAd, mm	37.4 (26.0-39.0)	34.4 (32.0-36.0)	35.6 (23.0-38.0)	0.186

*Abbreviations: IVSd: Interventricular Septal Diameter; LAd: Left Atrial Diameter; LVEDd: Left Ventricular End-Diastolic Diameter; LVEF: Left Ventricular Ejection Fraction; LVESd: Left Ventricular End-Systolic Diameter; PWd: Posterior Wall Diameter. *P values < 0.05 considered statistically significant.*

Discussion

Our study demonstrates significantly increased Pmax and PWD in both ischemic and hemorrhagic stroke patients compared to controls during the acute phase of stroke. These findings suggest that stroke, regardless of its subtype, is associated with altered atrial conduction, which may have important implications for understanding the relationship between cerebrovascular events and cardiac electrophysiology.

The finding that minimum P-wave duration was significantly prolonged in both stroke groups, with the most pronounced prolongation observed in ischemic stroke patients, provides additional insight into the nature of atrial conduction abnormalities following stroke. While PWD reflects the heterogeneity of atrial conduction, the prolongation of Pmin suggests a global slowing of atrial conduction velocity. This global conduction delay may result from several mechanisms including autonomic dysfunction, systemic inflammation, and direct effects of stroke on cardiac electrophysiology (16).

The observation that ischemic stroke patients had significantly longer Pmin compared to hemorrhagic stroke patients, despite similar PWD values, suggests that the mechanisms underlying atrial conduction abnormalities may differ subtly between stroke subtypes. Ischemic stroke may cause more profound global atrial conduction slowing, possibly due to prolonged hypoxic conditions, different patterns of inflammatory mediator release, or distinct effects on autonomic regulation (17).

The prolonged Pmax and increased PWD observed in stroke patients may be attributed to several factors. First, the higher prevalence of cardiovascular risk factors such as hypertension and diabetes in our stroke cohort could contribute to these ECG changes. These conditions are known to affect atrial structure and function, potentially leading to altered conduction properties. However, the lack of significant differences in echocardiographic parameters between the groups suggests that the observed ECG changes may not be solely attributed to structural cardiac abnormalities (18,19).

Moreover, the stroke itself may influence atrial electrophysiology through various mechanisms. Interestingly, we found no significant differences in P-wave parameters between ischemic and hemorrhagic stroke groups, despite their distinct pathophysiological mechanisms. This suggests that the observed changes may be related to the stroke event itself rather than the underlying pathophysiology (ischemia vs. hemorrhage). Both ischemic and hemorrhagic strokes can affect brain regions involved in autonomic regulation, which may explain the similar effects on atrial conduction.

The insular cortex, particularly important for cardiovascular autonomic control, when damaged by stroke can disrupt sympathovagal balance and trigger atrial electrophysiological changes (20). Right insular lesions typically increase sympathetic activity, while left insular damage affects parasympathetic tone, both contributing to atrial conduction heterogeneity and potentially explaining the observed P-wave prolongation regardless of stroke subtype.

While our study excluded patients with known cardiac disease, subclinical structural changes in the atria, possibly related to shared risk factors between stroke and cardiovascular disease, could contribute to the altered P-wave parameters.

While AF is a well-established risk factor for stroke, there is growing evidence that stroke itself may precipitate AF (21). The observed alterations in P-wave parameters might reflect a substrate for atrial arrhythmias in stroke patients, which is particularly relevant given the known bidirectional relationship between stroke and AF.

The potential use of P-wave parameters as a risk stratification tool in stroke patients is an area for future research. If validated in larger, prospective studies, these easily obtainable ECG measurements could help identify patients at higher risk of developing AF and guide decisions about long-term cardiac monitoring or prophylactic anticoagulation.

Several limitations should be considered when interpreting our results. The relatively small sample size of 112 participants limits the ability to detect subtle differences between stroke subtypes and precludes detailed subgroup analyses based on stroke etiology or location. The partly retrospective design may introduce potential bias, although consistent inclusion criteria and evaluation protocols were applied to minimize this limitation. Our focus on the acute

phase of stroke (within 24-48 hours) prevents determination of whether P-wave parameter changes persist over time. Additionally, the lack of long-term rhythm monitoring limits our ability to establish the direct relationship between observed P-wave abnormalities and subsequent atrial arrhythmia development. We did not perform extended Holter monitoring to document actual AF occurrence rates, which would validate whether patients with PWD >40 ms or prolonged Pmax develop AF at higher rates. The inclusion of patients referred for coronary angiography in the control group, despite normal results, may introduce selection bias, though all control subjects underwent identical cardiac evaluations to exclude significant cardiac disease. Despite these limitations, our study provides important preliminary data comparing P-wave parameters between ischemic and hemorrhagic stroke subtypes, contributing to the understanding of cerebrovascular-cardiac electrophysiological interactions.

Conclusion

Our study demonstrates significantly increased P-wave duration and PWD in both ischemic and hemorrhagic stroke patients compared to controls during the acute phase of stroke. These findings suggest altered atrial conduction and potential AF risk. Patients with PWD >40 ms may particularly benefit from prolonged ECG monitoring and lower thresholds for AF screening. Further research with larger sample sizes and longitudinal follow-up is warranted to validate our findings and improve risk stratification.

Ethical Approval: *This study was approved by the Ethics Committee of Zonguldak Bulent Ecevit University (Date: 12.04.2006, Decision number: 2006/03-9). The study was conducted in accordance with the principles of the Declaration of Helsinki.*

Author Contributions:
Concept: R.D., S.M.D.
Literature Review: R.D., Ö.B.
Design : R.D.
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References

1. Coupland AP, Thapar A, Qureshi MI, Jenkins H, Davies AH. The definition of stroke. J R Soc Med. 2017;110(1):9-12..
2. Donnan GA, Fisher M, Macleod M, Davis SM. Stroke. Lancet. 2008 May 10;371(9624):1612-23. doi: 10.1016/S0140-6736(08)60694-7.
3. Chen Z, Venkat P, Seyfried D, Chopp M, Yan T, Chen J. Brain-Heart Interaction: Cardiac Complications After Stroke. Circ Res. 2017;121(4):451-468. doi: 10.1161/CIRCRESAHA.117.311170.
4. Kallmünzer B, Breuer L, Kahl N, Bobinger T, Raaz-Schrauder D, Huttner HB, et al. Serious cardiac arrhythmias after stroke: incidence, time course, and predictors--a systematic, prospective analysis. Stroke. 2012;43(11):2892-7. doi: 10.1161/STROKEAHA.112.664318.
5. Magnani JW, Williamson MA, Ellinor PT, Monahan KM, Benjamin EJ. P wave indices: current status and future directions in epidemiology, clinical, and research applications. Circ Arrhythm Electrophysiol. 2009;2(1):72-9. doi: 10.1161/CIRCEP.108.806828.
6. Maheshwari A, Norby FL, Soliman EZ, Koene RJ, Rooney MR, O'Neal WT, et al. Abnormal P-Wave Axis and Ischemic Stroke: The ARIC Study (Atherosclerosis Risk In Communities). Stroke. 2017;48(8):2060-2065. doi: 10.1161/STROKEAHA.117.017226.
7. Pérez-Riera AR, de Abreu LC, Barbosa-Barros R, Grindler J, Fernandes-Cardoso A, Baranchuk A. P-wave dispersion: an update. Indian Pacing Electrophysiol J. 2016;16(4):126-133.
8. Koide Y, Yotsukura M, Ando H, Aoki S, Suzuki T, Sakata K, et al. Usefulness of P-wave dispersion in standard twelve-lead electrocardiography to predict transition from paroxysmal to persistent atrial fibrillation. Am J Cardiol. 2008;102(5):573-7. doi: 10.1016/j.amjcard.2008.04.065.
9. Dilaveris PE, Gialafos EJ, Sideris SK, Theopistou AM, Andrikopoulos GK, Kyriakidis M, et al. Simple electrocardiographic markers for the prediction of paroxysmal idiopathic atrial fibrillation. Am Heart J. 1998;135(5 Pt 1):733-8. doi: 10.1016/s0002-8703(98)70030-4.
10. Kamel H, Okin PM, Elkind MS, Iadecola C. Atrial Fibrillation and Mechanisms of Stroke: Time for a New Model. Stroke. 2016;47(3):895-900. doi: 10.1161/STROKEAHA.115.012004.
11. Oppenheimer SM, Gelb A, Girvin JP, Hachinski VC. Cardiovascular effects of human insular cortex stimulation. Neurology. 1992;42(9):1727-32. doi: 10.1212/wnl.42.9.1727.
12. Acampa M, Lazzerini PE, Guideri F, Tassi R, Lo Monaco A, Martini G. Inflammation and Atrial Electrical Remodelling in Patients With Embolic Strokes of Undetermined Source. Heart Lung Circ. 2019;28(6):917-922. doi: 10.1016/j.hlc.2018.04.294.
13. Ozdemir O, Soylu M, Demir AD, Topaloğlu S, Alyan O, Geyik B, et al. P-wave durations in patients experiencing atrial fibrillation during exercise testing. Angiology. 2007;58(1):97-101. doi: 10.1177/0003319706297924.
14. Dogan U, Dogan EA, Tekinalp M, Tokgoz OS, Aribas A, Akilli H, et al. P-wave dispersion for predicting paroxysmal atrial fibrillation in acute ischemic stroke. Int J Med Sci. 2012;9(1):108-14. doi: 10.7150/ijms.9.108.
15. Yilmaz R, Demirbag R. P-wave dispersion in patients with stable coronary artery disease and its relationship with severity of the disease. J Electrocardiol. 2005;38(3):279-84. doi: 10.1016/j.jelectrocard.2005.02.003.
16. Oliveira M, da Silva N, Cunha P, Ramos R, Marques F, Santos S, et al. Effects of acute autonomic modulation on atrial conduction delay and local electrograms duration in paroxysmal atrial fibrillation. Int J Cardiol. 2011;149(3):290-5. doi: 10.1016/j.ijcard.2010.02.006.
17. Lin CW, Li JY, Kuo TBJ, Huang CW, Huang SS, Yang CCH. Chronic intermittent hypoxia worsens brain damage and sensorimotor behavioral abnormalities after ischemic stroke: Effect on autonomic nervous activity and sleep patterns. Brain Res. 2023;1798:148159. doi: 10.1016/j.brainres.2022.148159.
18. Virani SS, Alonso A, Benjamin EJ, Bittencourt MS, Callaway CW, Carson AP, et al. Heart Disease and Stroke Statistics-2020 Update: A Report From the American Heart Association. Circulation. 2020;141(9):e139-e596. doi: 10.1161/CIR.0000000000000757.
19. Campbell BCV, De Silva DA, Macleod MR, Coutts SB, Schwamm LH, Davis SM, et al. Ischaemic stroke. Nat Rev Dis Primers. 2019;5(1):70. doi: 10.1038/s41572-019-0118-8.
20. Scheitz JF, Erdur H, Haeusler KG, Audebert HJ, Roser M, Laufs U, et al. Insular cortex lesions, cardiac troponin, and detection of previously unknown atrial fibrillation in acute ischemic stroke: insights from the troponin elevation in acute ischemic stroke study. Stroke. 2015;46(5):1196-201. doi: 10.1161/STROKEAHA.115.008681.
21. Sposato LA, Cipriano LE, Saposnik G, Ruíz Vargas E, Riccio PM, Hachinski V. Diagnosis of atrial fibrillation after stroke and transient ischaemic attack: a systematic review and meta-analysis. Lancet Neurol. 2015;14(4):377-87. doi: 10.1016/S1474-4422(15)70027-X.