EVALUATION OF THE PRESENCE OF LEUKOARAIOSIS IN CORRELATION WITH SELECTED RISK FACTORS FOR VASCULAR DISEASES IN PATIENTS WITH NEWLY DIAGNOSED STROKE

Dawid Mamak[1], Maciej Horyniecki[2], Mateusz Rajchel[1], Aleksandra Lupa[1], Justyna Szalajko[1], Kaja Skowronek[1], Monika Adamczyk-Sowa[2]

1. Students Scientific Group affiliated to Department of Neurology in Zabrze, Medical University of Katowice, Katowice, Poland
2. Department of Neurology in Zabrze, Medical University of Silesia, Katowice, Poland

#Corresponding author: Dawid Mamak, MD, e-mail: davidmamak@wp.pl, Students Scientific Group affiliated to Department of Neurology in Zabrze, Medical University of Katowice, Sleczka St. 6C/18, p.o. box 41-800 Zabrze, Poland, phone number: +48 508 485 627

RUNNING TITLE	Presence of leukoaraiosis in patients diagnosed with stroke
KEYWORDS	stroke; leukoaraiosis; clock drawing test; doppler ultrasound
WORD COUNT	2552
CONFLICT OF INTERESTS	no conflicts of interest

ABSTRACT

Stroke is one of the most common causes of mortality and disability in the elderly and promotes the development of cognitive functions. Risk factors for stroke include hypertension, dyslipidemia, ischemic heart disease, arrhythmia, atherosclerosis and leukoaraiosis. Prospective assessment of the dependency between the occurrence of leukoaraiosis, selected blood laboratory parameters, ultrasound results of carotid duplex ultrasonography and cognitive impairment in patients with newly diagnosed stroke was performed. The study group consisted of 50 patients at the age of 71.3 ± 11.8 years hospitalized in the Department of Neurology in Zabrze due to a stroke. Patients were subjected to physical examination, neurological examination with the NIHSS (National Institutes of Health Stroke Scale) and the MMSE (Mini-Mental State Exmination) test. Laboratory blood tests, carotid duplex ultrasonography and CT (computer tomography) scan of the head were performed. Statistical analysis was performed by using the Statistica program. Leukoaraiosis was found more frequently in patients over 74 years old (mean age 80 vs 69, p=0.008) and its presence correlated with higher LDL (low density lipoprotein) cholesterol (p=0.04) and higher CIMT (carotid intima-media thickness) in carotid arteries (p=0.04). Patients with leukoaraiosis achieved worse results in the clock test (p=0.04), median score 1 vs 5. Significantly lower incidence of leukoaraiosis was observed among patients taking beta-blockers (p=0.003) and calcium channel blockers (p=0.04). The occurrence of leukoaraiosis among patients with newly diagnosed stroke is associated with an intensity of other vascular risk factors and cognitive impairment.

BACKGROUND

The stroke constitute is a significant problem worldwide. Because of this disease, 6 million people die every year, making it the third most common cause of mortality [1]. What's more, it is the most common cause of disability of people over 45 years old. After stroke, not only physical impairments occur but also cognitive impairment [2, 3]. It has been shown that even minor strokes affect daily functioning, limiting executive and cognitive functions, making it difficult for patients to return to work, and thus lowering the quality of life [4, 5].

In recent decades, there has been a continuing upward trend in the incidence of strokes, which is due to the continuous aging of the population. Newer and better treatment methods significantly reduces patients mortality. These is associated with increase in treatment costs, more frequent hospitalizations and the number of disabled people who need long-term care [3, 6]. All these aspects have caused that recent years have been abounding in numerous studies on risk factors for stroke, their modification, early treatment, and prevention of stroke. Among the risk factors of stroke there are hypertension, dyslipidemia, lack of physical activity, ischemic heart disease, arrhythmia, atherosclerosis and leukoaraiosis [7, 8].

Leukoaraiosis (LA) was firstly described by Hachinski in 1987. For many years it was only a radiological notion describing spilled or multifocal, poorly demarcated areas of reduced density of the white matter of the brain, located bilaterally mainly in the periventricular regions (found in neuroimaging studies) [9, 10]. In CT examination, it is in the form of hypodense changes, with varying degrees of density reduction compared to the normal white matter. In magnetic resonance imaging (MR) in T2-dependent images and FLAIR (fluid-attenuated inversion recovery) sequences, these are changes of higher intensity (hyperintensive) in relation to the normal white matter [11, 12]. It is now known that leukoaraiosis is not only a radiological term, but a complex clinical syndrome whose etiopathogenesis and symptoms have not yet been fully understood[13, 14]. Leukoaraiosis is an independent risk factor for stroke. It occurs significantly more often among patients with stroke than in the healthy population. Many studies, including a Kissel's and collaborators study showed that leukoaraiosis not only increases the risk of stroke but also affects the severity of the effects and worsens the prognosis, rated using Modified Rankin Scale (mRS) [10, 14, 15]. The first of them shows a 0.47 higher evaluation of patients with leukoaraiosis in 90 days observation [15] and the second a 0.8 higher mRS in semi-annual observation [16]. It was also shown that the presence of leukoaraiosis is associated with an increase in patients' mortality from cardiovascular causes [17, 18].

Despite many years of intensive research on leukoaraiosis, many important questions about its etiopathogenesis, its impact on the patient's condition and its prevention remain unambiguous.

Therefore, the aim of the following study was a prospective assessment of the relationship between the occurrence of leukoaraiosis and lipidogram parameters, results of Doppler ultrasonography of the carotid and vertebral arteries and presence of the cognitive disorders in the patient with a newly diagnosed stroke.

MATERIAL AND METHODS

Study Population

The presented data was part of a single-centre, prospective study and included patients hospitalized in the Stroke Ward of the Silesian Neurology Clinic in Zabrze of the Medical University of Silesia in Katowice with a newly diagnosed stroke. The study group consisted of 50 patients, including 31 women and 19 men. The average age in the study population was 71.3±11.8 years. Patients with impaired consciousness or aphasia were excluded from the study due to the lack of cognitive functions in these patients. All examined patients were subjected to physical examination, neurological examination with the NIHSS assessment during admission to the ward. In order to assess cognitive dysfunctions, the MMSE test was performed, the results of which were adjusted for the age and length of the learning period, as well as the clock drawing test (CDT). All patients underwent computed tomography (CT) examination of the head, total cholesterol, LDL, HDL (high density lipoprotein), triglycerides, as well as carotid duplex ultrasonography. On the basis of CT results, patients were divided into two groups: patients with leukoaraiosis (GSL test group with leukoaraiosis, n=12) and patients with no leukoaraiosis (GC control group, n=35). Due to the ambiguous description of the CT scan of the head, three patients were excluded from the study.

Ethics

All of the patients obtained appropriate information about their participation in the studies. The written consent for treatment, hospitalization was collected. Patients consent for the use of patients data for medical researches. All of the obtained clinical data were collected as a result of the diagnostic and treatment procedures which were in pursuance with appropriate for stroke guidelines. The study protocol which was in line with ethical standards.

Statistical analysis

Results were expressed as means with standard deviations unless otherwise specified (continuous parameters) and with percentages and frequencies (categorical variables). Statistical significance was considered as a p value less than 0.05. Comparative group analysis was performed using Chi^2 Pearsona or Chi^2 Yatesa test for dichotomous parameters and Student's t-test for continuous variables. A logistic regression model with multiple-chance odds ratios was performed. All analyses were performed using the software package Statistica (version 12.5, StatSoft Inc., Tulsa, OK, USA).

RESULTS

The analysis of quantity and the division of studied population

In a group of 50 patients with newly diagnosed stroke a neuroimaging examination was performed. The results showed that 24% of them have leukoaraiosis (GSL). 6% of patients had ambiguous image, however in 70% of

remaining group the characteristic lesions for this illness (GC; control group) were not found. The patients without any lesions in CT image were qualified as a control group, which was compared with patients with leukoaraiosis [Figure 1].

Comparative analysis of demographic and pharmacotherapy among study group

Both GSL and GC were composed of more women than men. The sex doesn't have an impact on occurrence of leukoaraiosis (LA). The patients with LA were older than CG (p=0.008) [Figure 2]. In both groups they found high level of comorbidities that are classified as risk factors for stroke. The most common comorbidities were: hypertension, dyslipidemia and diabetes. There was no difference in frequency of these diseases in both GSL and GC.

It is proved that taking dihidropirydine type drug causes 7-fold reduced occurrence of LA (p=0.04). Beta blockers therapy is associated with 9-fold reduced risk of LA (p=0.003). No other drug-related correlation was found.

Among the patients with LA smokers represented smaller fraction than non-smokers. However smoking is associated with significantly earlier age at which the stroke occurs (the average age: 66±10 vs 75±12; p=0.007) [Figure 3].

In Table 1 the details of characteristics of both group are presented.

Comparative analysis of laboratory and imaging research, neurological condition and cognitive functions in both groups

There were no significant differences in blood pressure values in both groups of patients. Whereas the patients with LA presented substantially higher level of LDL (p=0.04). There was no other correlation between blood laboratory parameter and the occurrence of LA.

In GSC, higher average values of IMC (intima-media complex) in carotid arteries were showed. The thickness of IMC in left common carotid artery was accordingly 1.12±0.28 in GSL group and 0.92±0.17 in GC group (p=0.04) [Figure 4].

The connection between leukoaraiosis presence and NIHSS scale score were not demonstrated.

Whereas in a group with LA substantially more frequent occurrences of motor congruency disorders (p=0.037) and oculomotor nerves damage: III, IV and VI cranial nerves (p=0.014) were showed.

The correlation between occurrence of leukoaraiosis and cognitive functions disorders was proved. Patients with GSC reached a lower score in a Clock-Drawing test than patients from control group. Median score of Clock-Drawing test in a group of patients with LA was 1, meanwhile others patients obtained 5 points (p=0.04) [Figure 5]. The observation shown no link between the occurrence of leukoaraiosis and the results of MMSE test.

The details concerning comparison of both groups and above-cited results were presented in a Table 2 and Table 3.

Logistic regression – prediction of leukoaraiosis based on an interview

The influence of selected factors on risk of leukoaraiosis using logistic regression was analyzed. The monofactoral analysis showed a significant predictive value of age, smoking and taking of beta-blockers. The use of dihydropyridine calcium channel blockers and the high LDL level values were also close to the predictive relevance [Table 4].

Multivariate analysis showed the usefulness of a model composed of such predictive parameters as: age (p=0.03), use of beta-blockers (p=0.01), and dihydropyridine calcium channel blockers (p=0.02). The LDL level was close to the predictive suitability in this model (p=0.053). Knowledge about smoking (p=0.79) turned out to be useless.

It has been shown that based on the knowledge of patient's age, about the lack of use of beta-blockers and dihydropyridine calcium channel blockers by patients, presence of leukoaraiosis can be predicted with a sensitivity of 92%, and specificity of 89% [Figure 6].

DISCUSSION

Leukoaraiosis is often noticed in patients with diagnosed stroke [14, 19]. This fact is also confirmed by our research in which 24% of all patients had identify LA.

Previous studies reveal connection between presence of LA and age of studied population [10, 20, 21]. They show, such as results in our study, that older age is a strong predictor of the presence of LA. Unfortunately, the definition of threshold for the age beyond which patients have higher risk of occurrence of LA is not clear. So far, studies have most often indicated a significant increase in LA risk in the seventh decade of life [20, 21]. Our results indicate that this takes place mainly in the eighth decade.

Although we didn't noticed a statistically significant influence of the gender on presence of LA in patients after stroke, it is worthy to highlight that higher percentage of patients with LA is represented by women. This information seems to be important, because previous studies proved, that female are more burdened with risk of LA [20, 21]. One of the possibilities of this state is loss of protective effect of estrogens after menopause [20, 22].

Results of our research unequivocally indicate, that presence of LA is correlated with severity of cardiovascular risk factors. One of them is significantly higher rate of intima-media complex (IMC) in left common carotid artery (CCA) in patients with LA. Despite it's an index for macroangiopathy and in etiology of LA should be considered ischemic changes related to microvascular disorders. Not only our study, but also previous researches shown a distinct connection between higher rate of CCA-IMT in patients with LA [23, 24, 25]. Probably it's connected with more complex etiology of LA than previously expected in which is involved chronic cerebral hypoperfusion. Information which may be important is fact that increase of the IMC value, was noticed only on the left side. Well-known observation is increased IMC value only on the left side as a result of different haemodynamic tensions on both sides, which is associated with anatomical differences in left and right

carotid artery (place where this arteries leaves). Differences in tensions may exacerbate hypertension, which often coexists with LA (in our study up to 83%), which is probably also associated with increased white matter changes on the left hemisphere [25, 26, 27].

From the view of cardiovascular diseases other important correlation is presence of higher values of LDL level in serum in patients with LA compared to control group. Atherogenic potential connected with LDL level is well-known, therefore this information seems to be relevant in clinical term, because early normalization of values of LDL cholesterol can be an effective method in prevention of the occurrence of LA. Association between presence of LA and lipid disturbances were already confirmed by other research. Nevertheless, they demonstrate correlation between presence of LA, hypertriglyceridemia and metabolic syndrome. In contrast to this studies, our results doesn't show any relationship between level of triglycerides and presence on LA [23, 28].

In patients with LA there are often noticed various degrease of cognitive impairment [10]. Diffuse changes on kind of LA are considered as an important factor in pathogenesis of vascular dementia [29]. However impact of LA in cognitive functions is more complex and it is not only related with vascular dysfunctions [30, 31]. There is often observed poorly increased frontal lobe disorder and signs of subcortical structures damages in patients with LA, which leads to slowdown in thinking and blunted affect [10]. In our research we were trying to evaluate influence of LA on cognitive functions. For this purpose it was used clock drawing test and MMSE. Outcomes were ambiguous, because presence of LA have a significant impact on lower results in CDT. On the other hand presence of LA in obtained results have not influence on MMSE score. Also a new research lead by Genlong Zhong et al. presents inconclusive influence of the presence of LA on results in MMSE. Although the authors showed that LA is related to the result of the MMSE, the severity of lesions LA that were also evaluated in the study did not affected on the worse results of this test [32]. It seems that on the basis of current knowledge, it can be assumed that LA has an impact on cognitive function disorders but a thorough assessment of its impact requires further research.

Presence of LA was associated with higher appearance of depressive disorders [33, 34]. In our study there was not noticed this relationship, but higher percentage of patients with depression in GCS may indicate on accuracy of this conclusion.

Very often, however, LA does not show any symptoms or they are unnoticeable. Although LA does not manifest symptoms it has many negative consequences, such as increased risk of stroke, and higher mortality from cardiovascular causes [14, 17, 18]. Therefore, it is important to be alert and select people from LA risk groups, in which the prediction model of leukoaraiosis presented in our study can be helpful.

We have demonstrated that the use of beta blockers and dihydropyridine calcium blockers is associated with a lower incidence of LA [Table 4]. This is most likely associated with the providing a better blood supply to the central nervous system, which reduces the development of leukoaraiosis in the mechanism of ischemia. Therefore, it can be assumed that patients in the 8th decade of life who do not use the above drugs have a high risk of developing LA. This is also confirmed by the results of our logistic regression analysis [Figure 6].

Limitations of the study

The study was single-center, non-randomized and carried out on a small group of patients, so our results may not reflect on the real state of the world population. The study did not take into account the severity of LA assessed on the Fazekas scale, so presence of the symptoms were not related to LA severity but only to its presence [34]. To confirm our conclusions, a large randomized clinical trial is needed. In order to confirm the usefulness of a multiple-sensitivity of LA prediction model, it would be necessary to carry out a study assessing the occurrence of LA, in patients fulfill the criteria for the risk of presence of LA, before they experienced a stroke.

CONCLUSIONS

Presence of LA in patients with newly diagnosed stroke is correlated with severity of risk factors of cardiovascular diseases, and also reveal connection with occurrence of cognitive impairment.

CITE THIS AS

MEDtube Science Sep, 2018, Vol. VI (3), 17 – 26

ABBREVIATIONS

ACEI – angiotensin converting enzyme inhibitors
ARB – angiotensin receptor blockers
ASA – acidum acetylsalicylicum
BMI – Body Mass Index
BPM – beats per minute
CCA – common carotid artery
CDT – clock drawing test
CIMT – carotid intima-media thickness
CN – Cranial nerve
CT – computer tomography
FLAIR – fluid-attenuated inversion recovery
GC – control group
GSL – test group with leukoaraiosis
HCT – Hematocrit
HDL – high-density lipoprotein
HGB – Hemoglobin
HR – Heart Rate
IHD – ischemic heart disease
IMC – intima-media complex
IMT – intima-media thickness
IPP – proton pump inhibitors
LA – leukoaraiosis
LDL – low-density lipoprotein
MMSE – Mini-Mental State Examination
mRS – Modified Rankin Scale
MRI – magnetic resonance imaging
NIHSS – National Institutes of Health Stroke Scale
NSAIDs – Non-Steroidal Anti-Inflammatory Drugs other than ASA
OR – odds ratio
PLT – Blood Platelets

PU – confidence interval
RBC – Red Blood Cell
SD – standard deviation
WBC – White Blood Cell

REFERENCES

1. Ryglewicz W. Epidemiologia udarow mozgu. Medycyna 2000 1992; 27/28: 38-40.
2. Strong K, Mathers C, Bonita R. Preventing stroke: saving lives around the world. Lancet Neurology 2007; 6: 182–187.
3. Ryglewicz D. Choroby naczyń mózgowych w Polsce. [In:] Strzelecki Z, Szymborski J. Diagnoza i rekomendacje. Rządowa rada ludnościowa. Zachorowalność i umieralność na choroby układu krążenia a sytuacja demograficzna Polski 2015; 140-147.
4. Lees R, Fearon P, Harrison JK, Broomfield NM, Quinn TJ. Cognitive and mood assessment in stroke research: focused review of contemporary studies. Stroke 2012; 43(1): 678–1680.
5. Fride Y, Adamit T, Maeir A et al. What are the correlates of cognition and participation to return to work after first ever mild stroke? Top Stroke Rehabil 2015; 22(5): 317–325.
6. Kwolek A. Rehabilitacja w udarze mozgu. Wydawnictwo Uniwersytetu Rzeszowskiego 2009; 1: 33-36.
7. Renna R, Pilato F, Profice P et al. Risk factor and etiology analysis of ischemic stroke in young adult patients. Journal of stroke and cerebrovascular diseases: the official journal of National Stroke Association 2014 Mar; 23(3): 221-227.
8. Qing L, Wen-Qing H, Qi-Lin Ma et al. Incidence and risk factors of leukoaraiosis from 4683 hospitalized patients. Medicine 2017 Sep; 96(39): 7682.
9. Hachinski VC, Potter P, Merskey H. Leukoaraiosis. Archives of Neurology 1987; 44: 21–23.
10. Mieszko M. Zagrajek, Anna Pokryszko-Dragan. The characteristic and clinical signs of leukoaraiosis. Via Medica Udar Mozgu 2005; 7(2): 56–60.
11. Lopez OL, Becker JT, Jungreis CA et al. Computed tomography — but not magnetic resonance imaging — identified periventricular white-matter lesions predict symptomatic cerebrovascular disease in probable Alzheimer's disease. Archives of Neurology 1995; 52: 659–664.
12. Gupta SR, Naheedy MH, Young JC, Ghobrial M, Rubino FA, Hindo W. Periventricular white matter changes and dementia: clinical, neuropsychological and pathological correlation. Archives of Neurology 1988; 45: 637–641.
13. Huo D, Feng HL. New Progress in the research of leukoaraiosis. Med Recapitulate 2015: 269-271.
14. Smith E. Leukoaraiosis and Stroke. Stroke 2010; 41(10): 139–143.
15. Kissela B, Lindsell CJ, Kleindorfer D et al. Clinical prediction of functional outcome after ischemic stroke: the surprising importance of periventricular white matter disease and race. Stroke 2009; 40: 530–536.
16. Arsava EM, Rahman R, Rosand J et al. Severity of leukoaraiosis correlates with clinical outcome after ischemic stroke. Neurology 2009; 72: 1403–1410.
17. Debette S, Beiser A, DeCarli C et al. Association of MRI markers of vascular brain injury with incident stroke, mild cognitive impairment, dementia, and mortality: The Framingham Offspring Study. Stroke 2010; 41: 600–606.
18. Inzitari D. Leukoaraiosis: An independent risk factor for stroke? Stroke 2003; 34: 2067–2071.
19. Junying Sh, Kai Hao, Peihong Qi et al. Confirmation of the abnormal lipid metabolism as a risk factor for the disease of leukoaraiosis. Saudi Journal of Biological Sciences 2017; 24(3): 508-513.
20. de Leeuw FE, de Groot JC, Achten E et al. Prevalence of cerebral white matter lesions in elderly people: a population based magnetic resonance imaging study: the Rotterdam scan study. Journal of Neurology, Neurosurgery, and Psychiatry 2000; 70: 9–14.
21. Lin Q, Huang WQ, Ma QL et al. Incidence and risk factors of leukoaraiosis from 4683 hospitalized patients: A cross-sectional study. Medicine 2017; 96(39): 7682.
22. Seo SK, Jung I, Lee SM et al. Relationship between leukoaraiosis and menopause in healthy middle-aged women. Fertility and sterility 2013; 100(2); 500-504.
23. Manolio T, Burke G, O'Leary D et al. Relationships of cerebral MRI findings to ultrasonographic carotid atherosclerosis in older adults: the Cardiovascular Health Study. Arterioscler Thromb Vasc Biol 1999; 19: 356-365.
24. Lucatelli P, Raz E, Saba L et al. Relationship between leukoaraiosis, carotid intima-media thickness and intima-media thickness variability: Preliminary results. European radiology 2016; 26(12): 4423-4431.
25. Tanaka T, Shimizu T, Fukuhara T. The relationship between leukoaraiosis volume and parameters of carotid artery duplex ultrasonographic scanning in asymptomatic diabetic patients. Computerized Medical Imaging and Graphics 2009; 33(6): 489-493.
26. Rosfors S, Hallerstam S, Jensen-Urstad K, Zetterling M, Carlström C. Relationship between intima-media thickness in the common carotid artery and atherosclerosis in the carotid bifurcation Stroke 1998; 29: 1378-1382.
27. Rodriguez Hernandez SA, Kroon AA, van Boxtel MP et al. Is there a side predilection for cerebrovascular disease? Hypertension 2003; 42: 56-60.
28. Park K, Yasuda N, Toyonaga S et al. Significant association between leukoaraiosis and metabolic syndrome in healthy subjects. Neurology 2007; 69(10): 974-978.
29. Shimada K, Kawamoto A, Matsubayashi K, Ozawa T. Silent cerebrovascular disease in the elderly. Hypertension 1990, 16, 692–699.
30. Vernooij MW, Ikram MA, Vrooman HA et al. White matter microstructural integrity and cognitive function in a general elderly population. Archives of General Psychiatry 2009; 66: 545–553.
31. Nitkunan A, Barrick TR, Charlton RA, Clark CA, Markus HS. Multimodal MRI in cerebral small vessel

disease: its relationship with cognition and sensitivity to change over time. Stroke 2008; 39: 1999–2005.
32. Zhong G, Ruiting Z, Yerfan J et al. Better Correlation of Cognitive Function to White Matter Integrity than to Blood Supply in Subjects with Leukoaraiosis. Frontiers in Aging Neuroscience 2017; 9: 185.
33. Kuo HK, Lipsitz LA. Cerebral white matter changes and geriatric syndromes: is there a link?. The journals of gerontology. Series A, Biological sciences and medical sciences 2004; 59: 818-826.
34. Brown MM. Leukoaraiosis. [In]: Donnan GA et al. Lacunar and other subcortical infarctions. Oxford University Press 1995: 181–198.

LIST OF TABLES

LIST OF FIGURES

TAB. 1. COMPARATIVE ANALYSIS OF DEMOGRAPHIC AND PHARMACOTHERAPY AMONG STUDY GROUP.

Variable	GC (n=35)		GSL (n=12)	
	No.	[%]/SD	No.	[%]/SD
Age (years)*	68.7	12	80.3	5.5
Male	15	43	3	25
BMI>30	12	38	2	20
Loss of consciousness in stroke	3	9	3	25
Stroke in the past	9	26	3	25
Myocardial infarction in past	11	31	1	8
Head trauma in the past	20	57	1	8
Hypertension	33	94	10	83
IHD	11	31	3	25
Dyslipidemia	24	69	9	75
Depression	3	9	2	17
Diabetes Mellitus	12	34	5	42
Smokers*	19	54	2	16
Alcohol drinking	8	23	1	8
Used of statins	31	89	12	100
Used of dihydropyridine calcium channel blockers*	14	40	1	8
Used of beta-blockers*	23	66	2	17
Used of nitrate	4	11	2	17

Used of diuretics	19	54	6	50
Used of ACEI/ARB	23	66	8	67
Used of IPP	17	49	5	42
Used of NSAIDs	2	6	0	0
Used of ASA	21	60	8	67
Used of anticoagulants	12	34	3	25

Values presented as means±SD or percentage of subjects. ACEI=angiotensin converting enzyme inhibitors; ARB=angiotensin receptor blockers; ASA=acidum acetylsalicylicum; BMI=Body Mass Index; GC=Control group; GSL=Study group with leukoaraiosis; IHD=ischemic heart disease; IPP=proton pump inhibitors; NSAIDs=Non-Steroidal Anti-Inflammatory Drugs other than ASA; SD=standard deviation.

* – p value <0.05 compared study group (GSL) with control group (GC)

TAB. 2. COMPERATIVE ANALYSIS FOR CLINICAL AND LABORATORY RESULTS AMONG STUDY GROUPS.

Variable	GC	GSL
HR [BPM]	78±11	67±9
Systolic blood pressure [mmHg]	134±19	137±23
Diastolic blood pressure [mmHg]	75±10	75±11
MMSE adjusted for age and education (points)	24±5	22±7
HCT [%]	40±4	40±6
RBC [$x10^{12}$/l]	4.4±0.5	4.4±0.4
HGB [$x10^{12}$/l]	13.8±1.4	13.4±2.2
WBC [$x10^{12}$/l]	8.1±3	7.8±1.8
PLT [$x10^{12}$/l]	250±65	269±103
HDL level in serum [mmol/L]	1.2±0.5	1.3±0.3
LDL level I serum* [mmol/L]	2.8±1.3	3.7±1.2
Total cholesterol level [mmol/L]	4.9±1.6	5.4±1
Triglycerides [mmol/L]	1.75±1.04	1.81±1.1
Serum albumin [g/l]	41±4	41±3
Serum protein [g/l]	67±5	68±6
Creatynine [umol/l]	90±44	90±27

Values presented as means±SD or percentage of subjects. BPM=Beats per minute; GC=Control group; GSL=Study group with leukoaraiosis; HCT=Hematocrit; HGB=Hemoglobin; HDL=High-density lipoprotein; HR=Heart Rate; LDL=Low density lipoprotein; MMSE=Mini Mental State Examination Scale; PLT=Blood Platelets; RBC=Red Blood Cell; WBC=White Blood Cell.

* – p value <0.05 compared study group (GSL) with control group (GC)

TAB. 3. COMPARATIVE ANALYSIS IN NEUROLOGICAL EXAMINATION AMONG STUDY GROUPS.

Variable	GC (n=35)		GSL (n=12)	
	No.	[%]/SD	No.	[%]/SD
Muscle strength on Lovett's scale	4	1	3.8	0.7
Abnormal tendon reflexes	4	11	0	0
Sensory disturbance	3	9	1	8

Ataxia*	3	9	1	8
Presence of pathological symptoms in neurological examination	10	29	1	8
NIHSS score on admission	5.9	3.8	5.6	3
Failure of III CN*	0	0	2	17
Failure of IV CN*	0	0	2	17
Failure of V CN	10	29	1	8
Failure of VI CN*	0	0	2	17
Failure of VII CN	21	60	8	67
Failure of VIII CN	10	29	0	0
Failure of IX CN	8	23	3	25
Failure of X CN	8	23	3	25
Failure of XI CN	0	0	0	0
Failure of XII CN	11	31	3	25

Values presented as means±SD or percentage of subjects. CN=Cranial nerve; GC=Control group; GSL=Study group with leukoaraiosis; NIHSS=National Institutes of Health Stroke Scale; SD=standard deviation.

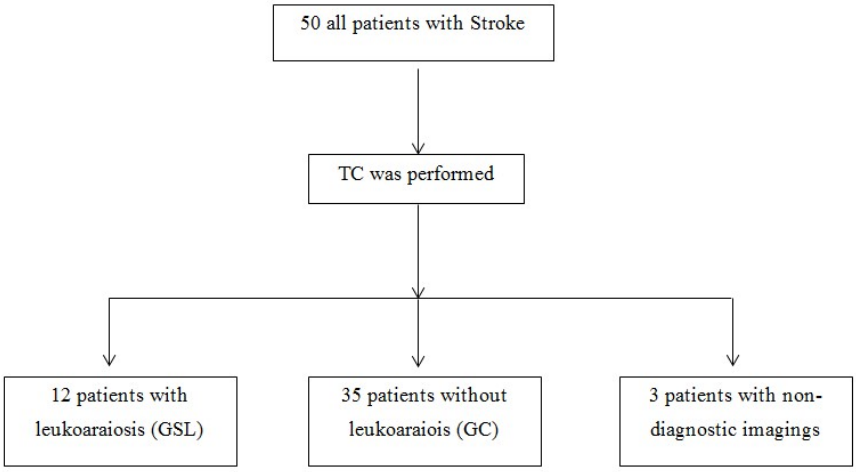
* – p value <0.05 compared study group (GSL) with control group (GC)

TAB. 4. ONE-FACTOR ANALYSIS OF LOGISTIC REGRESSION EVALUATED INFLUENCE OF SELECTED FACTORS ON PRESENCE OF LEUKOARAIOSIS.

Variable	OR (95% PU)	P
Age	1.153 (1.038 – 1.281)	**0.0078**
Used of dihydropyridine calcium channel blockers	0.136 (0.016 – 1.178)	0.07
Used of beta-blockers	0.104 (0.02 – 0.555)	**0.008**
Tobacco smoking	0.168 (0.985 – 2.863)	**0.035**
LDL level in serum	1.679 (0.985 – 2.863)	0.06

PU = confidence interval; OR = odds ratio

FIG. 1. ANALYSIS OF NUMBER AND DISTRIBUTION OF THE EXAMINED POPULATION.



GC=Control group; GSL=Study group with leukoaraiosis; TC=Computer Tomography

FIG. 2. COMPARISON OF THE PATIENTS AGE IN BOTH STUDIES GROUPS.

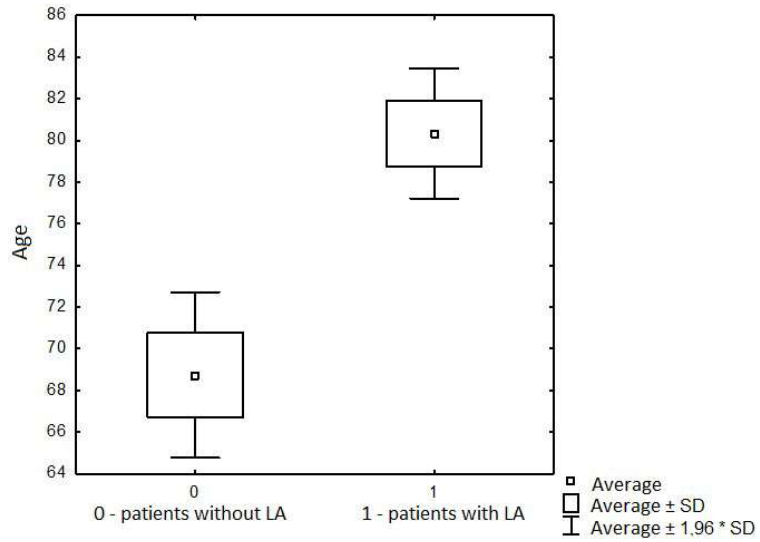


FIG. 3. INFLUENCE OF SMOKING ON PATIENTS AGE IN BOTH EXAMINED GROUPS.

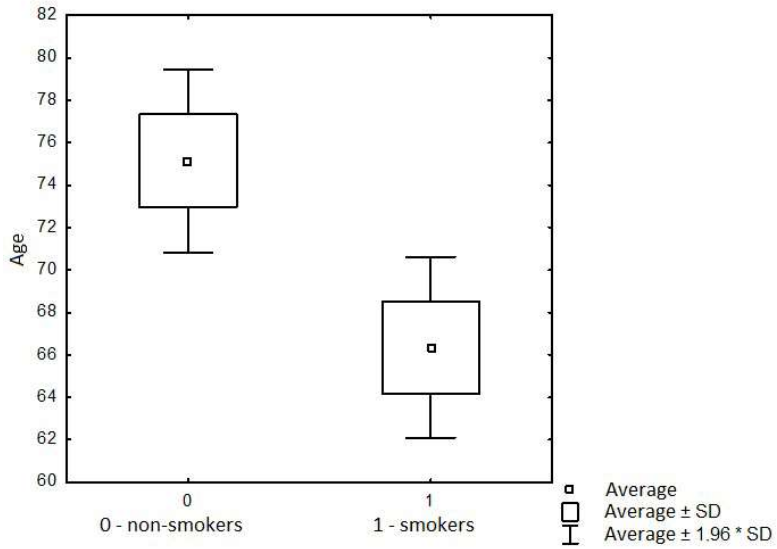
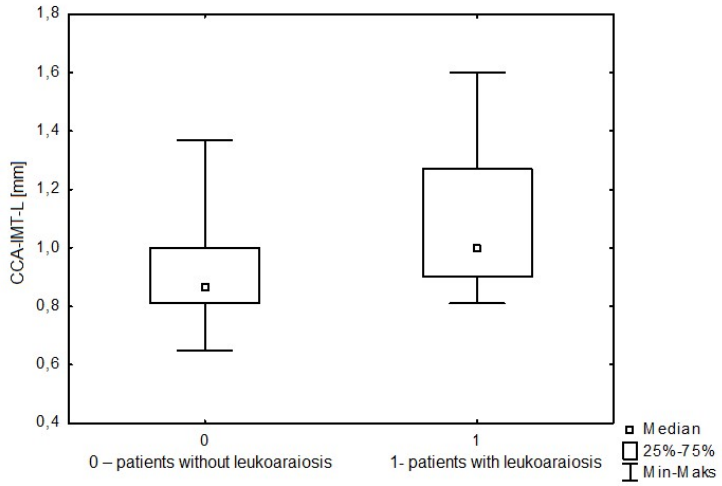


FIG. 4. COMPARISON OF THE VALUE OF IMT COMMON CAROTID ARTERY IN BOTH GROUPS.



IMT = intima-media thickness

FIG. 5. COMPARISON OF CLOCK DRAWING TEST SCORE IN BOTH GROUPS.

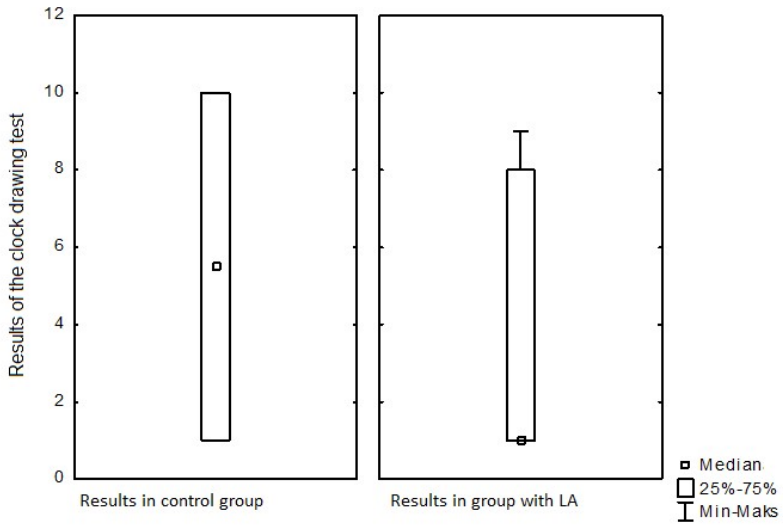
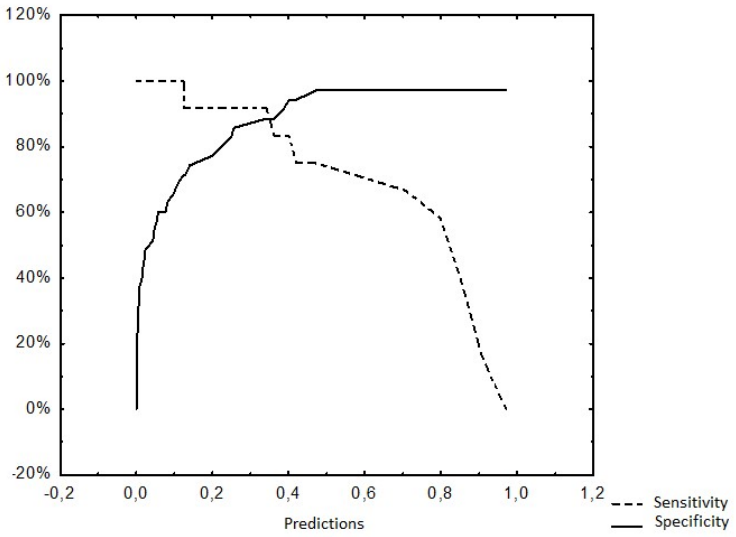


FIG. 6. EVALUATION OF THE SENSITIVITY AND SPECIFICITY OF A THREE-FACTOR LEUKOARAIOSIS PREDICTION MODEL.




MEDtube
sharing
medical
knowledge™