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PREVALENCE OF ERECTILE DYSFUNCTION POST-STROKE, AND ASSOCIATED RISK FACTORS AND CO-MORBIDITIES

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ABSTRACT. Sexual dysfunction have been frequently reported in patients with stroke. In recent studies it was shown that more than 50% of stroke patients reported post-stroke erectile dysfunction (ED). The aim of this study was to establish a correlation between prevalence and severity of ED and site location of stroke, and to assess the co-morbidities, medication and risk factors associated with post-stroke ED. At 153 patients (57.04 ± 6.54 years) with ischemic stroke, we evaluate the pre- and post-stroke prevalance of ED using the five-item International Index of Erectile Function questionnaire (IIEF5). Within 5 days of admission we determined the site location, and the stroke severity using the National Institute of Health Stroke Scale (NIHSS). The pre- and post-stroke data obtained were compared with those of 30 control persons (52.27 ± 8.35), and we compared the prevalence of ED with stroke location. The IIEF5 scores were much lower [median 17 interquartile range (IQR) 10-20] post stroke than pre-stroke (median 22 IQR 12-23) and lower than in control group (median 22.5 IQR 21-24). ED was associated with anterior cerebral artery infarction in 5/6, posterior cerebral artery infarction in 10/12, middle cerebral artery infarction in 70/93, basal ganglia infarction in 7/8, brain stem infarction in 13/16, cerebellar infarction in 6/14, and lesions in more than one region in 4/4 patients. The prevalence and severity of ED increase after stroke due to disruption of autonomous central structures. The depression, functional impairment, co-morbidities, and medication used after stroke may contribute to ED but must be evaluated in more specific patients group.
KEYWORDS: erectile dysfunction, IIEF5, ischemic stroke, ED prevalence, autonomic pathways, diabetes

INTRODUCTION

Erectile dysfunction (ED) which is defined as the inability to mantain and acheive a penile erection in order to perform a normal sexual performance [1], is considered to have a higher prevalence at older men. ED alter the quality of life and has a negative impact on the couple usual activities. In a recent study it was shown that approximately 50% of the patients with stroke presented a decrease in libido and also post-stroke ED [2]. The ED of the stroke patients it is determined by multifactorial etiology and severity can vary due to the co-morbidities and psychosocial factors. A variety of risk factors are also involved in the occurrence of erectile dysfunction post -AVC, the most important being hypertension, dyslipidemia, diabetes, smoking and coronary heart disease [3]. Post-stroke sexual dysfunction may be associated with functional disability caused by stroke, post-stroke depression or cardiovascular medication [4]. For all these reasons ED should be diagnosed and a therapy instituted as soon as possible.

The subsequent development or exacerbation of ED after stroke can be determined not only by vascular pathophysiology, but may also be linked to stroke-induced injury to the central autonomous structures that contribute to the sexual function [5].

Therefore, in this study we try to demonstrate that stroke significantly alter the prevalence and also the severity of ED, which were assessed pre- and post-stroke using the International Index of Erectile Function (IIEF5), and we will also assess the possible association between ED, severity of the stroke and the site of brain lesions.

MATERIALS AND METHODS

In this study were included 153 male patients aged 38-65 years (57.04 ± 6.54) admitted with ischemic stroke, in the Department Neurology of Arad County Hospital (Romania) between january 2014 to december 2015 . ED was evaluated within 5 days of admission using the International Index of Erectile function, the short version (IIEF5), and subsequently evaluated again 3 months after stroke. The data obtained were compared with those of a control group consisting of 30 people aged 40 - 65 years (52.27 ± 8.35).

Patients with other neurological diseases than ischemic stroke, those aged over 65, and also patients or those from the control group who used phosphodiesterase type 5 inhibitors (PDE5) were excluded from the study. To ensure that all patients have fully understood the explanations of the study and the questionnaires , their cognitive function was

evaluated using the scale of assessments of mental state (MMSE - Mini Mental State Examination), being excluded from the study the patients whose MMSE score was less than 20. Also were excluded from the study all patients presenting fluent or non-fluent aphasia at admission.

The research was conducted with the approval of the Ethics Committee of the Arad County Hospital in agreement with the Declaration of Helsinki (1989) of World Medical Association, and with approval of Ethics Committee of the Western University of Arad "Vasile Goldis". All patients included in the study, and those in the control group, received a consent form.

The stroke severity was assessed using the NIHSS scale (National Institutes of Health Stroke Scale) at hospital admission and 3 months after the event (NIHSS scale, range from 0 and 42 points), and It was also rated the degree of disability of patients using the Rankin scale (Rankin scale with 5 degrees of disability), at admission and at 3 months post-stroke [6]. The stroke site was determined using computed tomography (CT 520 Series General Electric Optima SYS) or magnetic resonance imaging (MRI; General Electric 1.5 Tesla Optima 360) within 5 days of hospitalization.

In the same time was evaluated the patients state of depression using Hamilton depression scale (questionnaire HAM-D), a questionnaire composed of 17 questions, each question with a score between 0 and 4 points, resulting in a score between 0 -50 points [7]. According to HAM-D questionnaire, depression can be classified as: between 0-7 points normally, between 8-13 points mild depression, moderate depression 14-18 points, severe depression between 19-22 points and over 23 points very severe depression.

To assess ED was used the short version of the International Index of Erectile Function (IIFE5) which consists of 5 questions, each question with a score between 0 and 5 points, with a maximum score of 25 points. In terms of the IIFE5 questionnaire, the ED can be classified as: severe ED (between 1 and 7 points; grade 5), moderate ED (between 8 and 11 points; grade 4), mild to moderate ED (between 12 and 16 points ; grade 3), mild ED (between 17 and 21 points; grade 2), and without erectile dysfunction (between 22 and 25 points; gradul1) [8].

So after the patients agreed to participate in this study and had the MMSE done to them, each of them received the IIFE5 questionnaires and the HAM-D questionnaire, wich were completed at admission and at 3 months after the event. People in the control group completed IIFE5 and HAM-D questionnaire depending on erectile function and depression status at the time of evaluation.

In all patients included in the study we took a complete medical history, paying attention to all risk factors, atherosclerotic associated comorbidities and medication used before and after stroke, and a general physical examination to rule out other diseases.

Statistical analysis

For statistical analysis of the results was the IBM SPSS Statistics for Windows program (version 20.0 Armonk, NY: IBM Corp). All data obtained were tested for normal distribution using the Shapiro-Wilk test. An average of the values obtained plus / minus a standard deviation was used for the normal distribution data, and median (interquartile range) for data without normal distribution.

To compare the data before and after the stroke, has been used the t test for normally distributed data and the Wilcoxon test for data with non- normal distribution. For comparisons between groups, t test was used for unpaired samples for data with normal distribution, the Mann-Whitney U test for data with non- normal distribution, and Chi-square test for variables. The correlations between NIHSS score, IIEF5 and age, and between Rankin score and IIEF5 were calculated using Spearman's correlation test for data with non-normal distribution and Pearson correlation test for normally distributed data. They were considered significant at p values of less than 0.05.

RESULTS

1. ED prevalence and severity in the 3 groups of patients

Following the examination of IIEF5 scores for our groups of patients, we see that 76 patients from the pre-stroke group (49.67%) had values of IIEF5 lower than 22, with varying degrees of ED, respectively 127 (83%) of patients from the post-stroke group showed some degree of ED. From the control group, only 9 of 30 patients (30%) had ED (Table 1).

Analyzing the group of patients using Tukey's Hinges estimate, we observed that IIEF5 values decreased significantly from 22 [median; interquartile range Q1-Q3 (IQR) 12.0-23.0] before the stroke to 17 after the stroke (median, IQR 10.0-20.0). It was also found that the values of IIEF5 of the patients before and after stroke were significantly lower than control group (median IIEF5 22.5; IQR 21.0-24.0; $p < 0.001$).

In terms of severity of ED, 29 of 153 patients from the pre-stroke group (18.95%), 74 (48.37%) from the post-stroke group and 7 (23.33%) from the control group had mild ED. Mild to moderate ED was reported by one patient from the post-stroke group (0.01%), 11 patients from the pre stroke group (7.19%) and also one patient from the control group (3.33%). Moderate ED was reported by 21 patients from the pre-stroke group (13.73%), 28 patients from post-stroke (18.30%) and 1 patient from the control group (3.33%). Severe ED was reported by 15 patients from the pre-stroke group (9.80%), 24 patients from the post stroke group (15.59%), and by none from the control group. (Table 1, Figure 1)

2. Association between ED and depression

Hamilton depression score was higher than 23 in 2 patients, suffering a very severe depression, both cases in the pre- and post-stroke groups, and

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no patient reported a score higher than 23 in the control group. Severe depression has been reported in 6 patients in pre-stroke group , 9 patients in post-stroke group and 1 patient in the control group. Moderate depression has been reported in 11 patients in post-stroke group and one patient in the control group and mild depression has been reported in 40 patients after stroke, 5 patients in the control group and 1 patient before stoke. (Table 1)

3. Association between ED, severity of stroke and age

Between IIEF5 scores of the two groups of patients, pre- and post-stroke, does not exist a statistical correlation with the severity of stroke represented by Rankin scores, instead there is an inverse correlation between patients' age and IIEF5 score ($p < 0.001$).

4. Association between ED, risk factors for stroke, comorbidities and use of medication before and after stroke

From the analysis of comorbidities and risk factors for stroke of post- stroke group and the control group, we infer that diabetes ($p = 0.003$), hypercholesterolemia ($p < 0.001$), and hypertension ($p < 0.001$) were more common in patients with stroke than those in the control group. (Table 1).

From the statistical analysis of data on medication use by patients, results that more patients have used ACE inhibitors, calcium antagonists, beta blocking agents, diuretics, statins, oral agents, antiplatelet and oral anticoagulants after the stoke than before, and in terms of consumption of drugs before stroke compared with the control group, differences were not significant. (Table 5, Figure 5).

5. Association between ED and the site of stroke

Among the 153 patients, 6 had an ischemic brain damage in the territory of the anterior cerebral artery (ACA, 3.92%), 12 patients in the territory of the posterior cerebral artery (PCA, 7.84%), 93 patients in the middle cerebral artery territory (MCA 60.78%), 14 (9.15%) patients in the cerebellum, 8 (5.22 %) patients basal ganglia, 16 (10.45%) patients in the brainstem, and 4 patients (2, 61%) have suffered ischemic lesions in multiple territories. 93 patients (60.78%) had ischemic lesions in the left hemisphere and 60 patients (39.22%) in the right hemisphere.

ED was present in 5 of 6 patients (83.33%) which had lesions the ACA territory, 10 of 12 patients (83.33%) with lesions in PCA territory, 70 of 93 patients (75.26%) with lesions in the MCA territory, 6 of 14 patients (42.85%) with lesions in the cerebellum, 7 of 8 patients (87.5%) with lesions in the basal ganglia, 13 of 16 patients (81.25 %) with the damage to the brainstem, and in all patients with ischemic lesions in more than one area. (Table 2)

DISCUSSIONS

A number of previous studies that analyze the prevalence of ED at patients with stroke have indicated an increase of ED prevalence at these patients. A study by Kimura revealed that about 60% of stroke patients have reported problems with sexual performance during periodic medical checks carried out at 3, 6 and 12 months post stroke [9].

After analysis of data obtained in our study, there was not only a confirmation of the results mentioned in previous studies, but also the fact that stroke patients present an increased prevalence and severity of ED post-stroke compared with the ED before the ischemic event. A variety of factors can be considered to increase the severity and changes in sexual dynamics of the patient who had suffered a stroke. Brain injuries can destroy the autonomic centers and the pathways that contribute to erection, and the paresis or plegia, spasticity or urinary incontinence may be physical handicaps for a normal sexual activity [10]. Certain post-stroke psychological barriers can occur due to depression and lack of self confidence. New drugs introduced after a stroke to prevent cardiovascular events, such as beta-blockers, diuretics or central antihypertensive drugs, and antidepressants can have adverse effects on sexual function and can exacerbate already existing ED.

Analyzing the lot of pre-stroke patients, 76 cases of ED were reported (49.67%), so we can confirm what previous studies reported that ED could be considered an early sign of cardiovascular and cerebrovascular disease [11]. ED can sometimes be the first symptom of a cardiovascular disease, and to some patients the ED can occur many years before the appearance of any symptoms of coronary heart disease. On the other hand, it is well known that cardiovascular atherosclerosis is an important risk factor for ED [12].

The patients from the study group had numerous risk factors for stroke, such as hypertension, diabetes, smoking, hypercholesterolemia, so that we can specify that the post-stroke ED is directly correlated with pre-existing risk factors, medication used pre- and post-stroke, and also with post-stroke depression. Many patients from the lot used medication with potentially side effects on sexual function before the stroke so that the medication added after stroke, such as diuretics, statins, beta-blockers, oral antidiabetic agents, anticoagulants, contributed to some extent to increase the prevalence and severity of ED.

As mentioned before, post-stroke depression deteriorate sexual activity. The regress of the HAM-D scores after stroke, and the inverse correlation between HAM-D scores and IIEF5 scores confirmed that depression post-stroke increase the prevalence and severity of ED. Because the HAM-D scores of pre-stroke group compared with those of control group showed no significant differences, we can state that worsening depression post-stroke is one that contributes to the deterioration of sexual activity. On the other hand, small differences between the HAM-D scores of stroke patients and control group, suggest that a significant increase in severity of ED can not

fully associate with worsening depression, impling also that the location and size of ischemic brain damage that prejudice autonomous central complex network involved in erectile function, is a major factor in the deterioration of sexual function post-stroke. The same assumption is strengthened by the fact that no significant correlations were found between the ED expressed by IIEF5 score, and stroke severity expressed by Rankin score [13].

Even though in both, the study group and the control group, was observed the worsening of ED with age, however we didnt found a significant correlation between the IIEF5 and NIHSS scores, unlike previous studies that have found significant correlations between the two scores [14]. Possibly this lack of correlation is because most of the patients from the study group had mild to moderate motor deficits. So we believe that motor deficits and post-stroke depression, are important co-factors contributing to the negative effects determined by brain lesions of areas involved in erectile function. So the motor deficit of the post-stroke patients does not seem to be a determining factor in erectile function deterioration.

In terms of localisation of ischemic brain injury, most commonly site is in the territory of the middle cerebral artery (MCA), 93 patients out of 153, with 70 patients out of 127 with ED post-stroke being part of this group with the ischemic lesion in the MCA territory . Cortical areas involved in erectile function are represented by the medial and inferior frontal lobe, both areas being irrigated by MCA, so that ischemic lesions in these areas may explain the increased prevalence of ED post-stroke. Damage in the anterior cerebral artery territory (ACA) have been reported in only 6 patients, from which 5 patients experienced ED post-stroke. There is little information in the literature on sexual dysfunction after ischemic injury in the territory of the ACA, but it is well known the role of certain brain formations involved in erectile function which are vascularized by this artery, recalling here the hypothalamus with the paraventricular nucleus and the preoptic medial nucleus , the anterior cingulate gyrus and the fornix. Similarly, a total of 12 patients had lesions in the posterior cerebral artery territory (PCA), 10 of these patients had reported ED post-stroke. Lesions in this area may affect the thalamic nuclei and the substantia nigra, cerebral formations involved in erectile function [15]. Even though the risk of developing ED after infarction in the ACA and PCA territory could be proven by a study that included more patients with lesions in their respective territories, we can state that ischemia of theACA and PCA territory have a greatly increased risk of ED post-stroke. The high percentage of patients experiencing ED after infarction of basal ganglia (7 of 8 patients) and brainstem (13 of 16 patients) could be explained by the fact that at this level pass many afferent and efferent pathways related involved in erection.

The risk of developing ED after stroke presents some differences depending on the location of lesions in the left or right hemisphere, given that sexual and autonomous function may have some interhemispheric differences [16,17]. In our study, 48 (80%) of the 60 patients with right hemisphere lesion presented ED after stoke, respectively 67 (72.04%) of the 93 patients with infarction in the left hemisphere. However we can not claim that post-stroke ED is more common in the right hemisphere lesions because aphasic patients with left hemisphere lesions were not included in the study.

So therefore, based on our data study, we attempted to calculate a multiple linear regression and a multiple logistic regression.

The multiple linear regression, through we try to estimate the value of the dependent variable 'score IIEF5 post stroke' based on the independent predictor variables: age, pre-stroke IIEF5 score, pre-stroke HAM-D score, post stroke HAM-D score, post-stroke at 3 months Rankin score, diabetes, hypercholesterolemia, hypertension, obesity, smoking, atrial fibrillation, carotid stenosis and coronary heart disease. In multiple linear regression calculation will consider only stroke patients. Sequential method is used to test for significance of the indendependent variables for the model, namely Stepwise method. The final model was obtained in four steps. Details can be found in the following tables:

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.893[a]	.798	.796	2.655
2	.909[b]	.825	.823	2.475
3	.914[c]	.836	.833	2.408
4	.917[d]	.842	.837	2.372

a. Predictors: (Constant), Score-IIEF5-pre-stroke
b. Predictors: (Constant), Score-IIEF5-pres-troke, age
c. Predictors: (Constant), Score-IIEF5-pre-stroke, Age, Diabetes
d. Predictors: (Constant), Score-IIEF5-pre-stroke, Age, Diabetes,

Score-Rankin-post-stroke-3-months
ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	4199.415	1	4199.415	595.576	.000[b]
	Residual	1064.703	151	7.051		
	Total	5264.118	152			
2	Regression	4345.493	2	2172.746	354.782	.000[c]
	Residual	918.625	150	6.124		
	Total	5264.118	152			
3	Regression	4400.484	3	1466.828	253.067	.000[d]
	Residual	863.634	149	5.796		
	Total	5264.118	152			
4	Regression	4431.170	4	1107.792	196.835	.000[e]
	Residual	832.948	148	5.628		
	Total	5264.118	152			

a. Dependent Variable: Score-IIEF5-post-stroke
b. Predictors: (Constant), Score-IIEF5-pre-stroke
c. Predictors: (Constant), Score-IIEF5-pre-stroke, Age
d. Predictors: (Constant), Score-IIEF5-pre-stroke,Age, Diabetes
e. Predictors: (Constant), Score-IIEF5-pre-stroke, Age, Diabetes, Score-Rankin-post-stroke-3-months

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	.362	.658		.551	.582
	Scor3-IIEF5-pre-stroke	.851	.035	.893	24.404	.000
2	(Constant)	12.111	2.482		4.879	.000
	Score-IIEF5-pre-stroke	.754	.038	.791	19.798	.000
	Age	-.176	.036	-.195	-4.884	.000
3	(Constant)	13.308	2.446		5.441	.000
	Score-IIEF5-pre-stroke	.673	.045	.706	14.810	.000
	Age	-.160	.035	-.178	-4.523	.000
	Diabetes	-1.684	.547	-.140	-3.080	.002
4	(Constant)	15.601	2.603		5.994	.000
	Score-IIEF5-pre-stroke	.599	.055	.629	10.916	.000
	Age	-.152	.035	-.169	-4.358	.000
	Diabetes	-1.527	.543	-.127	-2.812	.006
	Score-Rankin-post-stroke-3-months	-.770	.330	-.119	-2.335	.021

a. Dependent Variable: Scor_IIEF5_post_AVC

The final model is the model 4 (virtually no other independent variable was not significant for the model in step 5 to be included in the model). The final model has the following characteristics (according to the results obtained from the above tables in SPSS 20 statistical software):

- Adjusted R-square is 0.837
- The significant model obtained ($p < 0.001$) expresses the variability of the post-stroke IIEF5 score by using pre-stroke IIEF5 score ($\beta = 0.629$, $p < 0.001$), patient age ($\beta = -0.169$, $p < 0.001$), diabetes ($\beta = -0.127$, $p = 0.006$) and post stroke -3 months Rankin score ($\beta = -0.119$, $p = 0.021$) in the proportion of 83.7%.

Therefore, the multiple linear regression equation is: **post-stroke IIEF5 score = 15.601 + 0.599 * (pre-stoke IIEF5 score) - 0.152 * (patient age) - 1.527 * (diabetes) - 0.770 * (Rankin score -3 months post stroke).**

The interpretation of the regression ecuation is:

- If the value of the pre-stroke IIEF5 score increases by one unit, the post-stroke IIEF5 score value increases by 0.599.

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- If the age of the patient increases by one unit, the post-stroke IIEF5 score value decreases by 0.152
- The presence of diabetes decreases the post-stroke IIEF5 value by an average of 1.527
- If the value of Rankin score at 3 months post-stroke increases by one unit, the post-stroke IIEF5 score value decreases by 0.770

Secondly, we realized a multiple logistic regression in order to try to estimate the variable dependent "erectile dysfunction after stroke" (ED post-stroke) which gives us the real diagnostic of the patient having or not post-stroke ED, ED post-stroke being a binary variable form type presence or absence of the disfunction, based on the following independent predictor variables: age, pre-stroke IIEF5 score, pre-stroke HAM-D score, post-stroke HAM-D score , Rankin score -3 months post-stroke, diabetes, hypercholesterolemia, hypertension , obesity, smoking, atrial fibrillation, carotid stenosis, coronary hearth disease. The multiple linear regression is considered only in patients with stroke. We used the sequential method to test the significance of independent variables for the model, namely Forward on Wald statistical test.

The final model is obtained in 3 steps, details can be found in the following tables:

Case Processing Summary

Unweighted Cases[a]		N	Percent
Selected Cases	Included in Analysis	153	100.0
	Missing Cases	0	.0
	Total	153	100.0
Unselected Cases		0	.0
Total		153	100.0

a. If weight is in effect, see classification table for the total number of cases.

Dependent Variable Encoding

Original Value	Internal Value
Absence of ED post-stroke	0
Presence of ED post-stroke	1

Categorical Variables Codings

		Frequency	Parameter coding
			(1)
Coronary disease	Absence	127	.000
	Presence	26	1.000
Hypercholesterolemia	Absence	49	.000
	Presence	104	1.000
Hipertension	Absence	32	.000
	Presence	121	1.000
Obesity	Absence	117	.000
	Presence	36	1.000
Smoking	Absence	100	.000
	Presence	53	1.000
Carotid stenosis	Absence	135	.000
	Presence	18	1.000
Atrial fibrillation	Absence	131	.000
	Presence	22	1.000
Diabetes	Absence	94	.000
	Presence	59	1.000

Omnibus Tests of Model Coefficients

		Chi-square	df	Sig.
Step 1	Step	40.534	1	.000
	Block	40.534	1	.000
	Model	40.534	1	.000
Step 2	Step	13.968	1	.000
	Block	54.502	2	.000
	Model	54.502	2	.000
Step 3	Step	4.905	1	.027
	Block	59.407	3	.000
	Model	59.407	3	.000

Model Summary

Step	-2 Log likelihood	Cox & Snell R Square	Nagelkerke R Square
1	98.936[a]	.233	.389
2	84.968[a]	.300	.501
3	80.062[a]	.322	.538

a. Estimation terminated at iteration number 6 because parameter estimates changed by less than .001.

Classification Table[a]

	Observed		Predicted		
			ED presence-post-stroke		Percentage Correct
			ED absence post stroke	ED presence post stroke	
Step 1	Presence-ED post-stroke	ED absence post stroke	10	16	38.5
		ED presence post stroke	5	122	96.1
	Overall Percentage				86.3
Step 2	Presence-ED post-stroke	ED absence post stroke	12	14	46.2
		ED presence post stroke	8	119	93.7
	Overall Percentage				85.6
Step 3	Presence-ED post-stroke	ED absence post stroke	13	13	50.0
		ED presence post stroke	5	122	96.1
	Overall Percentage				88.2

a. The cut value is .500

Variables in the Equation

		B	S.E.	Wald	Df	Sig.	Exp(B)	95% C.I.for EXP(B)	
								Lower	Upper
Step 1[a]	Age	.210	.039	29.775	1	.000	1.234	1.144	1.331
	Constant	-9.887	2.056	23.136	1	.000	.000		
Step 2[b]	Age	.183	.043	18.183	1	.000	1.201	1.104	1.306
	Hypercholesterolemia(1)	2.093	.599	12.228	1	.000	8.110	2.509	26.213
	Constant	-9.346	2.291	16.646	1	.000	.000		
Step 3[c]	Age	.160	.046	12.250	1	.000	1.174	1.073	1.284
	Hypercholesterolemia(1)	1.887	.616	9.378	1	.002	6.597	1.972	22.069
	HTA(1)	1.332	.596	4.994	1	.025	3.787	1.178	12.177
	Constant	-8.902	2.415	13.585	1	.000	.000		

a. Variable(s) entered on step 1: Age
b. Variable(s) entered on step 2: Hypercholesterolemia
c. Variable(s) entered on step 3: HTA.

The final significant model (p <0.001) obtained by which we determine whether a patient has or not post-stroke ED using significant predictors to the model is characterized in the table below (the results in table below shows the key results in the tables above, tables supplied by the statistic software SPSS 20). We note that the significant predictors for the model are patient age, hypercholesterolemia and hypertension.

Variable	Coeficient (B)	Eroarea Standard (ES)	Statistica Wald	Grade de libertate	p-valoare	Exp (B)	95% IC pentru Exp (B)
Patien age	0.160	0.046	12.250	1	<0.001	1.174	1.073 – 1.284
Hypercholesterolemia	1.887	0.616	9.378	1	0.002	6.597	1.972 – 22.069
HTA	1.332	0.596	4.994	1	0.025	3.787	1.178 – 12.177
Constant	-8.902	2.415	13.585	1	<0.001	<0.001	

The interpretation of variables in regression equation obtained for the determination of ED for a patient after stroke is:

- Age of the patient: For an increase by one unit of age (assuming other variables remain constant), the chance of the patient to have post-stroke ED is increased by a factor Exp (0.160) = 1.174 (thus the chances of having post-stroke ED are 1,174 higher). With an increase by k years of age, and accustom odds ratio has a value Exp (k * B). Thus, for k = 10 (value up 10 years of age) the chance to develop ED after stroke increases with Exp (10 * 0.160) = exp (1.6) = 4.953.
- Hypercholesterolemia: The presence of hypercholesterolemia (assuming other variables remain constant) increases the chance of the patient to have post-stroke ED by a factor Exp (1.887) = 6.597 (thus the chances of having post-stroke ED are 6.597 for a patient with hypercholesterolemia compare to a patient without hypercholesterolemia)
- HTA: The presence of hypertension (assuming other variables remain constant) increases the chance of the patient to have post-stroke ED by a factor Exp (1.332) = 3.787 (thus the chances of having post stroke ED are 3.787 for a patient with hypertension compared a patient without hypertension).

The logistic regression equation obtained is: **Logit (ED post-stroke) = -8.902 + 0.160 * (Age patient) + 1.887 + 1.332 * * Hypercholesterolemia HTA**

So how can we use this equation in medical practice? We can predict the probability of post-stroke ED for a patient knowing their age and whether or not he has hypercholesterolemia or hypertension. For example, if we consider patient 60 years old, hypercholesterolemia = 1 (presence of hypercholesterolemia) and HTA = 0 (absence of HTA) we obtain: Logit (ED post-stroke) = -8.902 + 0.160 + 1.887 * 60 = 2.585, and after transforming this value in probability, Prob = exp (2,585) / (1 + exp (2,585)) = 0.92989. Thus the probability that a patient 60 years old, with hypercholesterolemia and without HTA, to be diagnosed with ED post-stroke is 92.989%.

CONCLUSIONS

The results obtained in our study showed that the prevalence and severity of ED increase after stroke and that there is some association between the ED prevalence the infarction site location by affecting the autonomous central structures involved in sexual function. Given that most of the patients presented ischemic lesions in the MCA territory, and the number of patients with lesions in other territories was reduced, we can not determine a statistically significant correlation between ED and location of the ischemic lesion. Also, due to low number of patients we can not draw conclusions on the fact that ischemic lesions from certain cerebral territories present a higher or lower risk to develope an ED post-stroke.

To determine the exclusive anti-erectile effects of a stroke, a control group with the same risk factors and medicines used by the patients from the pre-stroke group would have been much more significant. However, this approach requires a study with a larger number of patients with the same risk factors and using the same medication, and that some patients will develop a stroke and others do not. Instead we tried to compare the results obtained in stroke patients and those in the control group with the average age about equal, and reflecting the risk of ED or stroke in elderly patients from the same community with those who have suffered a stroke.

In conclusion, studies on a larger number of stroke patients could more accurately determine the impact that the location and size of cerebral infarction has on erectile function and also help us to distinguish more accurately the anti-erectile effects of the infarction or medication introduced post-stroke.

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COMPLIANCE WITH ETHICAL STANDARDS

CONFLICT OF INTEREST None

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