Risk Factors of Cognitive Impairment in Ischemic Stroke Patients

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Abstract: Background: Cognitive impairment represents one of the most common causes of specific and overall functional disability of patients suffering from stroke. **Objective**: to evaluate the progression of cognitive impairment in ischemic stroke patients and to assess for the influencing and associated factors. **Patients and methods**: The present study prospectively recruited 50 patients and other 20 healthy subjects as a control group, matched for both age and sex. Strict inclusion and exclusion criteria were applied for patients presented with acute ischemic stroke according to the WHO (2010) stroke definition. All patients were evaluated for ischemic stroke risk factors, radiological investigations, stroke severity using the National Institute of Health Stroke Scale score (NIHSS), cognitive state using the Cognitive Abilities Screening Instruments (CASI) and Mini Mental State Examination (MMSE). Both were applied at baseline, 3 and 6 months follow up. Control group underwent evaluation using both MMSE and CASI. **Results:** Post stroke dementia affected up to 22% of stroke survivors and another 24% suffered from cognitive impairment without dementia. The maximum significant changes of cognitive function were seen at 3 months with less remarkable changes at 6 months follow up. The following factors were significantly associated with increased incidence of post stroke cognitive impairment: (1) demographically related factors; elderly patients and high body mass index (BMI) (2) stroke risk factors; family history of dementia, high pack-year smoking index, hypertension, low hemoglobin level, high cholesterol level, high C reactive protein level, presence of >30% carotid stenosis, atrial fibrillation and ischemic heart disease, (3) stroke related factors; high stroke severity, large size of infarction, presence of silent infarctions and left sided brain infarction. **Conclusion:** Adverse cognitive decline is a common consequence in patients with vascular ischemic stroke with many influencing associated factors. Better awareness about these factors should increase the awareness for better management and effectiveness of preventive strategies with subsequent improvement of quality of life outcome in patients with ischemic stroke.

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1. Introduction

Stroke is the world's third leading cause of death, with a high incidence of morbidity in surviving patients **(Woodruff *et al.*, 2011)** and can impact many aspects of a person's life **(Cumming *et al.*, 2014)**. People with stroke are likely to demonstrate significant decrements in motor performance only (cognitive-related motor interference), or decrements in both motor and cognitive performance **(Plummer *et al.*, 2013)**. Assessment of cognitive functions is often quite difficult because of lack of generally accepted definition of cognitive impairment, information about the pre-stroke cognitive state of the patients and the heterogeneity of procedures and parameters used in evaluation of cognitive state **(Maya *et al.*, 2012)**. The frequency of cognitive impairment after CVS varies between studies. A study reported that the incidence of cognitive impairment three months after stroke was 35%–37% **(Jonkman *et al.*, 1998)**. The most common types of cognitive deficits arising from stroke include impairment of attention, language syntax, delayed recall and executive dysfunction affecting the ability to analyze, interpret, plan, organize and execute complex information **(Jokinen *et al.*, 2006)**. The risk of vascular cognitive impairment and dementia as well as the rate of cognitive decline in cerebrovascular disease is highly dependent upon the control of the underlying risk factors for stroke **(Dufouil *et al.*, 2005)**. It is, therefore, important during post stroke rehabilitation to focus on reducing maladaptive changes and on promoting adaptive illness cognitions in order to improve overall quality of life **(Mierlo *et al.*, 2015)**.

2. Patients and Methods

The present study included 50 patients and other 20 healthy control subjects, matched for age, sex, social and educational level from the Neurology Department of Al-Azhar University Hospitals. The inclusion criteria comprised patients presented with acute ischemic stroke according to **WHO (2010)** definition of stroke. Exclusion criteria included presence of previous hemorrhagic or ischemic stroke, patients with any systemic, psychiatric or other

neurological disorder affecting cognition, e.g. hepatic patients, patients with aphasia, dense hemiplegia in the dominant upper limb or severe apraxia, persistent altered level of consciousness and patients having blindness or hearing impairment. Upon admission, all patients were evaluated and investigated for: (A) Stroke risk factors including demographic variables, cardio-vascular and hematologic risk factors. The following were done for all patients: full history and clinical neurological evaluation, the pack-year smoking index, body mass index (BMI),all appropriate laboratory investigations, electrocardiography (ECG), echocardiography, carotid and vertebra-basilar duplex, CT and/or MRI of the brain. The following radiological data were collected: presence of infarct subtypes, number, size, location (deep versus superficial location), laterality of lesions and cerebral circulation involvement and the presence of silent infarcts. (B) Stroke severity using the National Institute of Health Stroke Scale Score (NIHSS), (C) Cognitive state assessment at baseline, 3 and 6 months follow up applying the following 2 scales: (1) the Cognitive Abilities Screening Instruments (CASI), that consisted of 25 test items and provided quantitative assessment on attention, concentration, orientation, memory, language abilities, drawing and writing abilities, list generating abilities, abstract thinking and judgment. The cutoff point equals or less than 67 points for dementia, (2) the Mini Mental State Examination (MMSE). A score of 23 or less has generally been accepted as indicating the presence of cognitive impairment. The control subjects were recruited from the hospital staff and local volunteers. They were 20 healthy persons matched with the same age and sex and were subjected to both MMSE and CASI. All patients have given informed written consents for study participation.

Statistical analysis:

Descriptive statistics (proportions/percentage, mean, standard deviations), parametric (student-test) and non-parametric (Chi-square) statistical tests were applied as appropriate for the variables of interest using Statistical Package for the Social Sciences (SPSS-V17). The probability used to decide significance was set at $p<0.05$.

3. Results

1-Demographic characteristics for both patient and control groups:

Patients' mean age was 56.3 ± 8.4 with males represented 66% of patients. The control subjects' mean age was 54.4 ±8.5 years and 60% of them were males. There were no significant difference between patients and control as regard age, sex, level of education, residency and BMI (Table 1). All 50 patients were presented and evaluated for cognitive functions after 3 months follow up. At 6 months follow up, the number of patients became 41 (3 patients died and 6 did not attend the second follow up).

Table (1): Demographic characteristics for both patient and control groups

Groups \ Variables		Patients (N=50)		Controls (N=20)		Chi-X^2/ t-test	*P*-value
		N (%)	Mean ± SD	N (%)	Mean ± SD		
Age (years)	<55	23 (46)	56.3 ± 8.4	10 (50)	54.4 ±8.5	0.834	0.204
	55-65	19 (38)		6 (30)			
	>65	8 (16)		4 (20)			
Sex	Female	17 (34)	-	8 (40)	-	0.224	0.636
	Male	33 (66)		12 (60)			
Level of education	Illiterate	31 (62)	-	12 (60)	-	0.073	0.964
	Less than 8 years	16 (32)		7 (35)			
	More than 8 years	3 (6)		1 (5)			
Residency	Urban	39 (78)	-	14 (70)	-	0.497	0.481
	Rural	11 (22)		6 (30)			
BMI*	Normal	18 (36)	17±2.4	8 (40)	16±2.1	1.376	0.503
	Over weight	21 (42)	27±3.6	10 (50)	25±2.3		
	Obese	11 (22)	33±5.4	2 (10)	31±4.8		

*BMI: Body mass index

2-Cognitive state in patients and control groups:

There was significant difference ($p<0.01$) between patients and controls regarding to functional status at both 3 and 6 months (Figure 1). Cognitive status showed deterioration at 3 months follow up when 22% showed evidence of dementia while other 14% showed cognitive impairment only without dementia. At 6 months follow up the percentage of demented patients was 24% and 12 percent showed cognitive impairment (Table 2).

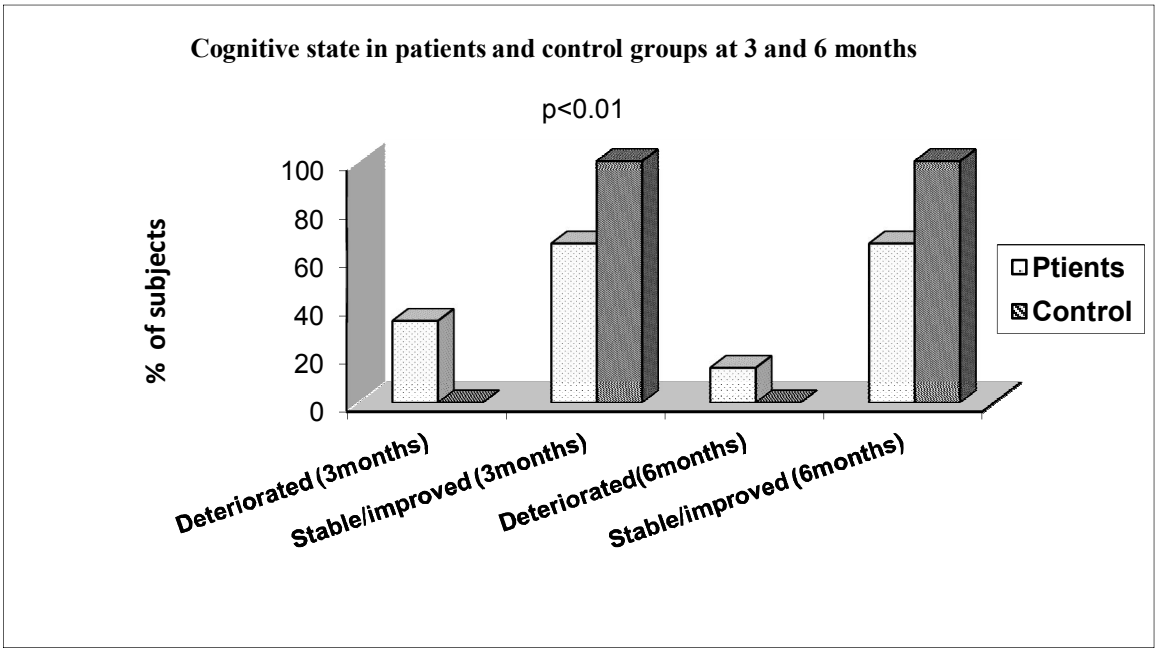

Figure (1): Comparison between cognitive state in patients and control groups at 3 and 6 months

Table (2): Functional status after 3 and 6 months in patients and control groups

Variables / Groups	At 3 months		X^2	*p*	At 6 months		X^2	*p*
	patients N (%)	control N (%)			patients N (%)	control N (%)		
Cognitively impaired	7 (14)	0(0)	14.4	0.001*	5(12)	0(0)	14.2	0.001*
Demented	11 (22)	0(0)			10 (24.4)	0(0)		
Cognitively intact	32 (64)	20(100)			26 (63.4)	20(100)		

3-Cognitive state in relation to demographic factors:

Compared to baseline values, there were significant differences in patient groups (deteriorated and stable/improved) after 3 months as regard age and BMI. However none of these factors remained significant at 6 months follow up when compared to 3 months follow up. On the other hand, there was no significant difference in patients groups as regard sex, level of education and residency at either follow up (Table 3).

4-Cognitive state in relation to stroke risk factors:

There were significant differences in patient groups (deteriorated and stable/improved) only after 3 months follow up regarding to the family history of dementia, but not the family history of stroke. Similarly, there were significant difference between deteriorated and stable/improved patients groups after 3 months as regard presence or absence of smoking, hypertension, diabetes, atrial fibrillation (AF), ischemic heart disease, more than >30% carotid stenosis but not TIAs (Figure 2). At 6 months, the only 3 risk factors remained significant were smoking, hypertension and presence of carotid stenosis. Alike, there were significant correlation between the cognitive state and the stroke severity at both 3 which has not significantly changed at 6 months (Table 3). Significant association was found between low hemoglobin, high cholesterol and positive CRP and vascular cognitive decline. No significant association found with other laboratory results (Table 4).

3-Neuro-imaging findings in relation to cognitive state:

After 3, there were significant relations between cognitive state and the neuroimaging findings regarding, size of infarction and left sided lateralization (Table 5). The relations were not significant as regard arterial territory (anterior or posterior circulation) or the level of infarction (superficial or deep). At 6 months follow up, none of these factors remained significant when compared to 6 months cognitive state.

Table (3): Relation between cognitive state and demographic and clinical variables in patient groups after 3 and 6 months.

Cognitive state / Variables		After 3 months (50 patients)				After 6 months (41 patients)			
		Deteriorated N (%)	Stable/ Improved N (%)	X^2	*p*-value	Deteriorated N (%)	Stable/ Improved N (%)	X^2	*p*-value
Age	<55	3(17.7)	20(60.6)	20.1	0.000*	2(33.3)	18(51.4)	3.1	0.21
	55-65	6(35.3)	13(39.4)			4(66.7)	11(31.4)		
	>65	8(47.1)	0(0)			0(0)	6(17.1)		
Sex	Female	7(41.2)	10(30.3)	0.6	0.44	1(16.7)	13(37.1)	0.9	0.328
	Male	10(58.8)	23(69.7)			5(83.3)	22(62.9)		
Level of education	Illiterate	11(64.7)	20(60.6)	1.66	0.435	3(50)	23(65.7)	1.28	0.526
	> 8 ys	6(35.3)	10(30.3)			3(50)	10(28.6)		
	< 8 yrs	0(0)	3(9.1)			0(0)	2(5.7)		
Residency	Urban	13(76.5)	26(78)	0.04	0.84	5(83.3)	27(77.1)	0.1	0.735
	Rural	4(23.5)	7(21.2)			1(16.7)	8(22.7)		
BMI*	Normal	4(23.5)	14(42.4)	9.4	0.009*	3(50)	13(37.1)	1.98	0.372
	Obese	8(47.1)	3(9.1)			0(0)	9(25.7)		
	Over weight	5(29.4)	16(48.5)			3(50)	13(37.1)		
Family history of dementia	Negative	12(70.6)	32(96.9)	7.4	0.007*	6(100)	30(85.7)	0.976	0.323
	Positive	5(29.4)	1(3)			0(0)	5(14.3)		
Family history of stroke	Negative	14(82.4)	24(72.7)	0.6	0.450	3(50)	27(77.1)	1.9	0.166
	Positive	3(17.7)	9(27.3)			3(50)	8(22.9)		
NIHSS*	Mild	8(47.1)	33(100)	14.3	0.000*	6(100)	28(80)	1.04	0.307
	Moderate	5(29.41)	0(0)			0(0)	5(14.3)		
	Severe	4(23.53)	0(0)			0(0)	2(5.7)		

*BMI: Body mass index
*NIHSS: National institute of health stroke scale

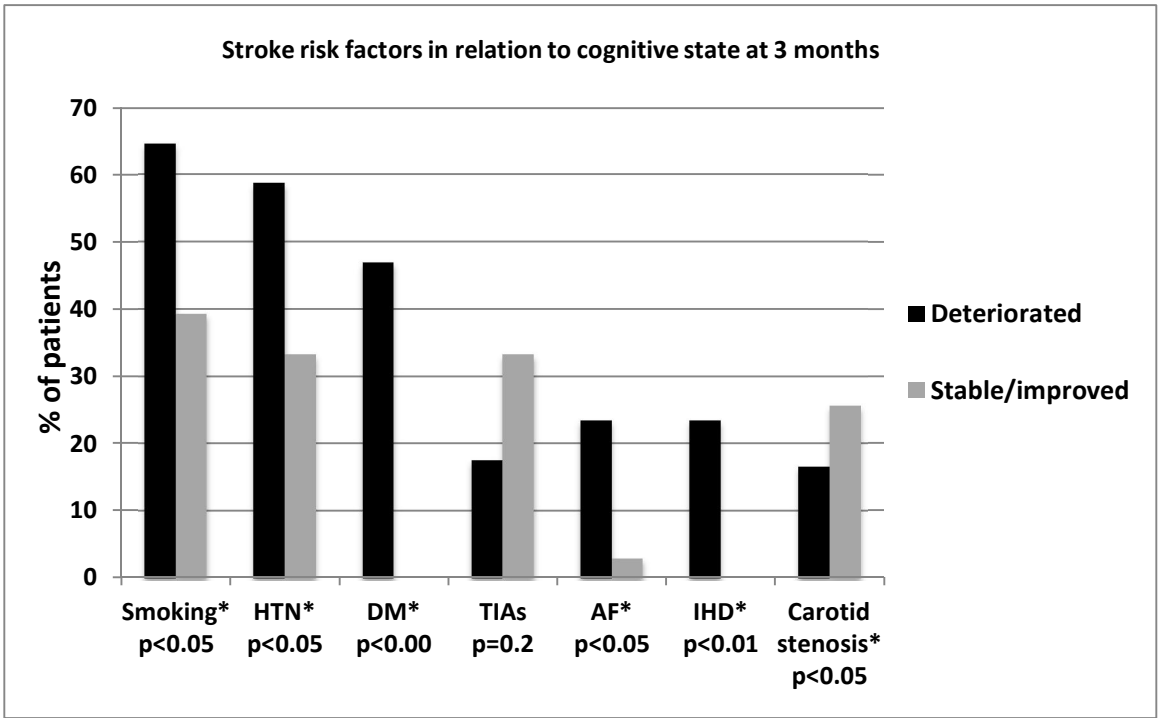

Figure (2): Relation between stroke risk factors and cognitive state at 3 months follow up

Table (4): Relation between cognitive state and laboratory findings in patient groups after 3 and 6 months

Variables \ Cognitive state		After 3 months (50 patients)				After 6 months (41 patients)			
		Deteriorated N (%)	Stable/ improved N (%)	X^2	*p*-value	Deteriorated N (%)	Stable/ improved N (%)	X^2	*p*-value
Liver function tests	Normal	16 (94.1)	31 (94)	0.001	0.980	6 (100)	32 (91.4)	0.555	0.456
	Elevated	1 (5.9)	2 (6.1)			0 (0)	3 (8.6)		
Hemoglobin	Normal	6 (35.3)	30 (91)	17.2	0.00*	7 (31.8)	16(84.2)	11.4	0.001*
	Low	11 (64.7)	3(9)			15 (68.2)	3(15.8)		
ESR*	Normal	11 (64.7)	18 (54.6)	0.475	0.490	3 (83.3)	21 (60)	1.202	0.273
	Elevated	6 (35.3)	15 (45.5)			1 (16.7)	14 (40)		
CRP*	Negative	6 (35.3)	33 (100)	27.38	0.00*	1 (11.1)	16 (50)	4.4	0.043*
	Positive	11 (64.7)	0 (0)			8 (88.9)	16 (50)		
Cholesterol	Normal	6 (26.1)	18 (66.7)	8.24	0.004*	1(12.5)	17 (51.5)	4	0.046*
	High	17 (73.9)	9 (33.3)			7 (87.5)	16 (49.5f)		
Triglycerides	Normal	9 (53)	11 (33.3)	1.797	0.180	2 (33.3)	15 (42.9)	0.191	0.662
	High	8 (47.1)	22(66.7)			4 (66.7)	20 (57.1)		
Renal function tests	Normal	15 (88.2)	32(97)	1.518	0.218	5 (83.3)	34 (97.1)	2.105	0.147
	High	2 (11.8)	1(3)			1 (16.7)	1 (2.9)		

*ESR: erythrocytic sedemintation rate, CRP: C-reactive protein

Table (5): Relation between cognitive state and radiological findings in patient groups after 3 and 6 months.

Variables \ Cognitive state		After 3 months (50 patients)				After 6 months (41 patients)			
		Deteriorated N (%)	Stable/ improved N (%)	X^2	*p*-value	Deteriorated N(%)	Stable/ improved N(%)	X^2	*p*-value
Silent infarctions	Negative	7(41.2)	33(100)	24.2	0.000*	5(83.3)	27(77.1)	0.115	0.735
	Positive	10(58.8)	0(0)			1(16.7)	8(22.9)		
Size of infarction (mm)	Small	6(35.3)	18(54.6)	12.1	0.002*	5(83.3)	16(45.7)	3.07	0.213
	Large	7(41.2)	1(3)			0(0)	6(17.1)		
	Lacunar	4(23.5)	14(42.4)			1(16.7)	13(37.1)		
Level of infarction	Deep	10(58.8)	28(84.9)	1.1	0.141	6(100)	27(77.1)	1.7	0.192
	Superficial	7(41.2)	5(15.2)			0(0)	8(22.7)		
Lateralization	Right	10(58.8)	24(72.7)	7.2	0.027*	3(50)	24(68.6)	0.798	0.671
	Left	7(41.2)	4(12.1)			2(33.3)	7(20)		
	Unclassified	0(0)	5(15.2)			1(16.7)	4(11.4)		
Arterial territory	Anterior	15(88.2)	25(75.7)	1.1	0.296	5(83.8)	26(74.3)	0.2	0.633
	Posterior	2(11.8)	8(24.2)			1(16.7)	9(25.7)		

4. Discussion

The frequency of cognitive impairment and dementia after CVS varies between studies. Taken only development of dementia, the results of this study revealed that dementia affected up to 22% and 24 % of stroke survivors three and six months after stroke respectively. However 14% and 12% showed only cognitive impairment without dementia at three and six months respectively. One study reported that the incidence of dementia three months after stroke was 35%–37% **(Jonkman *et al.*, 1998)**. In another study, 50 % to 75 % of stroke patients are found to be affected by post stroke cognitive impairment depending on age **(Tay *et al.*, 2006)**. The differences of prevalence of post stroke dementia among various studies may be explained by the differences in the study design, demographic characteristics (as age, gender, and ethnicity), criteria used for the diagnosis of dementia, radiological chosen criteria, vascular risk factors, the time interval between the stroke and the neuropsychological assessment and length of follow-up **(Hénon *et al.*, 2006)**. The identified lower frequency of post stroke cognitive impairment or dementia in the current study may be related to using strict criteria with exclusion of patients with persistent aphasia, persistent altered level of consciousness and recurrent stroke. In the present study, demographic determinants for post stroke cognitive impairment included increasing age and family history of dementia. These findings were in agreement with **Gorelick, 1997, McDowell *et al.*, 2007** and **Stoquart *et al.*, 2007**. In concordance with **Tay *et al.*, 2006**, we found that the size of infarction significantly affecting post stroke cognitive impairment. Furthermore, the left brain infarction lateralization has significantly affecting post stroke cognitive impairment. These findings were comparable with **Ballard *et al.*, 2003**. Similar to **Madureira *et al.*, 2001,** we found that the

stroke severity significantly affected post stroke cognitive impairment. **Censori *et al.*, 1996**, explained that the degree of post stroke cognitive impairment did not always parallel that of neurological deficit. Another finding of the present study was that smoking significantly affected the post stroke cognitive state, which was in line with the results of **Ikeda *et al.*, 2008**. Another influencing risk factor that significantly associated with post stroke cognitive was hypertension. This was in concordance with the conclusions of **Harrington *et al.*, 2000**. Studies pointed out that blood pressure levels should be kept within a certain range (below 140/90 mmHg) for prevention of cardiovascular, cerebrovascular damage, and maintaining cerebral perfusion enough to preserve cognitive ability and prevention of some types of dementia **(Birkenhäger *et al.*, 2001)**. An important risk factor showed to negatively affect the post stroke cognitive state was atrial fibrillation (AF). **Ott *et al.* (1997)** clarified that there is an increased incidence of silent brain infarctions in patients having AF. As concluded by **Hemmingsson *et al.*, 2007**, our findings pointed out that there was significant association between IHD and post stroke cognitive impairment. **Komulainen *et al.* (2007)** have demonstrated that carotid stenosis was more frequent in patients with post stroke cognitive impairment and that common carotid artery intima-media thickness (CCA-IMT) predicts an increased risk for cognitive impairment in elderly patients. In consistent with **Rasquin *et al.* (2004)**, there was significant association between the presence of silent infarctions and post stroke cognitive impairment. They elucidated that "silent" infarctions could predict post-stroke dementia, especially in cases when the period between the stroke and the clinical manifestation of cognitive impairment is too long. Regarding laboratory findings that directly influencing the post stroke cognition, our data pointed out that there was significant association between low hemoglobin level and post stroke cognitive impairment which was similar to the conclusions of other authors **(Gottesman *et al.*, 2010)**. They mentioned that hemoglobin level seems to be an important factor in determining degree of specific cognitive impairment at the time of stroke. Both low and high hemoglobin levels are associated with worse performance on tests of neglect and with more frequent neglect, independent of infarct size and stroke severity. Concerning the body mass index, we found that higher body mass index significantly affecting post stroke cognitive impairment. This was in agreement with **Power *et al.* (2011),** but in disagreement with **Fitzpatrick *et al.* (2009),** who found that older people who are overweight or obese may actually have lower risk of developing dementia than those who have normal weight. Finally, significant association between high cholesterol and positive CRP and vascular cognitive decline has been concluded. These findings are consistent with those of other authors **(Exel *et al.*, 2002 and Maya *et al.*, 2012)**. For the overall cognitive influencing factors, the main association was found at three months follow up, with less important association noted at six months follow up which imply that the peak incidence cognitive impairment happened at third months post stroke and less significant deterioration occurred afterward. In conclusion, this study highlighted the common occurrence of adverse cognitive decline in patients with vascular ischemic stroke. Better awareness about the associated and risk factors of cognitive impairment should increase the effectiveness of management and preventive strategies with subsequent improvement of quality of life outcome in patients with ischemic stroke.

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