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ORIGINAL ARTICLE.

The Relationship between Glycosylated Hemoglobin and Cognitive Dysfunction after Acute Ischemic Stroke

Ghada Abd El Wahab Khalil Ibrahim[1], Shaimaa A Elaidy[1]*, Mohamed Hanafy Aly Ghonemy[1]
[1] Neurology Department, Faculty of medicine, Zagazig university, Zagazig, Egypt

Corresponding Author
Shaimaa A Elaidy

Email address:
shaimaaelaidy@yahoo.com

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ABSTRACT

Background: Stroke takes the second place among worldwide causes of morbidity and mortality. Significant attention was paid to the relationship between Glycosylated hemoglobin (HbA1c) and cognitive functions in patients with acute ischemic stroke (AIS). **Aim**: This study aims at assess the relationship between post stroke cognitive impairment (PSCI) and HbA1c levels as well as other factors that may predict cognitive decline in those patients. **Methods:** 110 patients with a diagnosis of AIS were included in this study. Cognitive functions were evaluated after 3 months of the onset using the Montreal Cognitive Assessment scale (MoCA). Demographic, clinical features, laboratory parameters including HbA1C level as well as imaging findings were analyzed and relationship with PSCI were determined.

Results: PSCI was significantly related to Type 2 diabetes mellitus (P < 0.001) and to Sphincter dysfunction (P 0.04). Elevated HbA1c level and lower levels of high density lipoprotein (HDL) were significant in the group of PSCI (P value <0.001 and 0.021 respectively), as well as increased carotid intima media thickness (CIMT) on Carotid Doppler examination (P value 0.013). The increase of CIMT and higher levels of HbA1c were independent risk factors for cognitive dysfunction in acute ischemic stroke patients, with odds ratio of 0.002 (0, 0.145), 2.088 (1.601, 2.723) respectively, (95% confidence interval).

Conclusion: The occurrence of PSCI was independently related to higher Glycosylated hemoglobin levels and increased CIMT. Careful evaluation of these factors help clinician to intervene early and develop better treatment modalities.

Keywords: Acute ischemic stroke; Cognitive impairment; Glycosylated hemoglobin; MoCA scale.

INTRODUCTION

Stroke is the second worldwide leading cause of functional disability and mortality **[1]**. Cognitive impairment (CI) after stroke represents a heavy load on both individual families and society **[2]**. Even in cases with mild strokes, patients frequently complain from cognitive problems in addition to the motor deficit **[3]**. Therefore, early recognition of patients at risk for developing post stroke cognitive impairment (PSCI) might help clinicians plan better treatment strategies and to intervene early to prevent cognitive dysfunction.

The Montreal Cognitive Assessment (MoCA) **[4]** is a sensitive screening scale which seems reliable and valid for identification of cognitive functions that represent a crucial functional outcome in stroke patients **[5].**When compared to the Mini-Mental State Examination (MMSE), the former was a rapid and easy tool offering targeted executive functions and visual construction tasks evaluation **[6].**

Glycosylated hemoglobin (HbA1c) represents the average level of serum glucose over the prior three months **[7].** There has been prior research on HbA1c and CI, however the findings were inconsistent. Some studies showed that HbA1c has been linked to CI risk in diabetic patients **[8,9].** However, the relationship between HbA1c and CI has not been thoroughly examined in patients with

acute ischemic stroke (AIS), neither was the possibility of correlation between them **[10,11].**

Therefore, this study examined the link between the HbA1c and the CI occurring after AIS, and identified other factors that may lead to the development of PSCI

METHODS

This is an observational, prospective cohort study including a total of 110 adult patients with confirmed diagnosis of acute ischemic stroke (AIS). The study was approved by the local Ethic Committee (ZU-IRB#11042). Written informed consent was obtained from all subjects or their relatives. Ethical guidelines for human experimentation were observed in line with the Helsinki Declaration of the World Medical Association.

Inclusion criteria included age more than 18 years old, patients with their first ever AIS. The diagnosis of ischemic stroke was confirmed by computed tomography scanning of the head. Exclusion criteria included history of any psychiatric illness, thyroid disorders, any pre stroke cognitive deficits.

All patients were subjected to complete history taking with special attention for sex, age, body mass index (BMI), smoking status and presence of comorbidities such as hypertension, diabetes, dyslipidemia, cardiac problems. Complete laboratory investigations were performed including Glycosylated hemoglobin (HbA1c) and lipid profile. The National Institute of Health Stroke Scale (NIHSS) was used on admission to identify the severity of the stroke **[12]** beside the modifed Rankin scale (mRS) which determined the functional outcome. Post stroke cognitive impairment (PSCI) was assessed 3 months after stroke using the validated Arabic version of the Montreal Cognitive Assessment MoCA **[13].** The MoCA score is categorized into 6 domains evaluating memory problems, executive functioning deficits, attention, language, visuospatial and orientation, with a maximum total scoreof 30. Impairments in attention and executive functions were prominent after stroke, but memory, language, and perceptual–motor functioning deficits seem to be common as well **[14].** We used the initially suggested cut-off point of 25 to define PSCI. This cutoff value had proven good sensitivity for mild cognitive impairment (MCI) (i.e., ≥ 83%) **[4].** The relationship between PSCI and HbA1c levels as well as other demographic, clinical, laboratory and imaging parameters were studied.

RESULTS

Our study included 110 patients with their first diagnosis of ischemic stroke. Mean age of our patients was 63.13±10.19. Male represented 50.9% of our patients. Hypertension and obesity were present in 71.8% and 69.1% of patients respectively. Stroke severity was assessed using the NIHSS with a range of 5-30. Small and medium sized lesions represented 20% and 53.6% of patients respectively.

According to the presence of CI, patients were subdivided into two groups, those with PSCI (63, 57.2%) and those without PSCI (47, 42.7%). As shown in **table (1,2),** there was statistically significant association between CI and comorbid type 2 diabetes mellitus (type 2 DM) (82.9% of those with DM had CI). 68.9% cognitively impaired patients had significant sphincter dysfunction (P value 0.04). There was not statistically significant relation between CI and other baseline or clinical parameters.

Higher levels of cholesterol, low density lipoprotein (LDL), HbA1c and lower level of high density lipoprotein (HDL) were prevalent in the cognitively impaired group but only HbA1c and HDL levels reached a significant difference (P value <0.001 and 0.021 respectively).

On doing multivariate regression analysis of factors associated with CI among studied patients and after controlling for age, sex, and lipid profile, we found that increase in HbA1c and in the Carotid intima media thickness (CIMT) independently increased risk of CI **(Table 3).**

Using correlation analysis **(Table 4),** a highly significant negative correlation was found between HbA1c levels and MoCA scores (R -0.58, P value <0.001).

Table (1): Relationship between demographic, clinical characteristics and post stroke cognitive impairment in the studied patients.

	Patients with PSCI (n=63)	Patients without PSCI (n= 47)	P value
Age, mean±SD	63.37 ± 10.97	62.87 ± 9.15	0.803
Sex, male, n (%)	34 (60.7%)	22 (39.3%)	0.457
Vascular risk factors			
Hypertension, n (%)	44 (55.7%)	35 (44.3%)	0.99
Diabetes, n (%)	63 (82.9%)	13 (17.1%)	**<0.001****
Obesity, n (%)	43 (54.4%)	36 (45.6%)	0.336
Dyslipidemia, n (%)	19 (57.6%)	14 (42.4%)	0.966
Smoking, n (%)	28 (60.9%)	18 (39.1%)	0.518
Atrial fibrillation, n (%)	8 (72.7%)	3 (27.3%)	0.347
Rheumatic heart disease, n (%)	1 (100%)	0 (0%)	>0.999
Ischemic heart disease, n (%)	7 (53.8%)	6 (46.2%)	0.79
Cardiomyopathy, n (%)	3 (50%)	3 (50%)	>0.999
Systolic BP, mean±SD	146.83 ± 27.11	144.68 ± 27.34	0.683
Diastolic BP, mean±SD	88.41 ± 9.54	89.57 ± 11.41	0.563
Admission GCS, mean±SD	9.71 ± 2.63	10.23 ± 2.94	0.332
Admission NIHSS, median (IQR)	17.5(12 – 24)	15(10 – 24.25)	0.628
Admission mRS, median (IQR)	3(2 – 5)	3(2 – 5)	0.741
Degree of weakness, n (%)			
Absent	2 (66.7%)	1 (33.3%)	
Paresis	27 (54%)	23 (46%)	
Paralysis	34 (59.6%)	23 (40.4%)	0.795
Speech defect, n (%)			
Absent	26 (57.8%)	19 (42.2%)	
Dysarthria	22 (57.9%)	16 (42.1%)	0.979
Dysphasia	15 (55.6%)	12 (44.4%)	
Cranial nerve deficit, n (%)	61 (58.1%)	44 (41.9%)	0.424
Sensory deficit, n (%)	14 (60.9%)	9 (39.1%)	0.695
Visual field defect, n (%)	28 (51.9%)	26 (48.1%)	0.259
Incoordination, n (%)	4 (44.4%)	5 (55.6%)	0.493
Sphincter dysfunction, n (%)	31 (68.9%)	14 (31.1%)	**0.04***

Data represented as mean ± SD and compared using independent sample t test
Data represented as median (IQR) and compared using Mann Whitney test
Categoricaldata is represented by n (%) and compared using Chi square test
*p<0.05 is statistically significant
**p≤0.001 is statistically highly significant
TOAST, Trial of Org 10172 in Acute Stroke Treatment
GCS, Glasgow Coma Scale
NIHSS, National Institute of Health Stroke Scale
mRS, Modified Rankin Scale

Table (2): Relation between radiological and laboratory parameters in the studied patients and post stroke cognitive impairment.

	Patients with PSCI (n=63)	Patients without PSCI (n= 47)	p
Side of brain lesion			
Left	26 (51%)	25 (49%)	
Right	37 (62.7%)	22 (37.3%)	0.215
Size of lesion, n (%)			
Large	18 (62.1%)	11 (37.9%)	
Medium	33 (55.9%)	26 (44.1%)	
Small	12 (54.5%)	10 (45.5%)	0.573

	Patients with PSCI (n=63)	Patients without PSCI (n= 47)	p
Site of lesion, n (%)			
Brainstem Cerebellar Lobar Lobar& subcortical Subcortical	4 (66.7%) 1 (50%) 25 (52.1%) 12 (54.5%) 21 (65.6%)	2 (33.3%) 1 (50%) 23 (47.9%) 10 (45.5%) 11 (34.4%)	0.775
TOAST classification, n (%)			
Cardioembolic Lacunar Large artery Undetermined	23 (56.1%) 9 (50%) 15 (57.7%) 16 (64%)	18 (43.9%) 9 (50%) 11 (42.3%) 9 (36%)	0.83
CIMT	1.11 ± 0.14	1.05 ± 0.12	**0.013***
Carotid plaques, n (%)	56 (57.1%)	42 (42.9%)	0.937
Degree of stenosis, n (%)			
<50% 50 – 69% 70 – 89% >90%	6 (100%) 32 (57.1%) 12 (40%) 13 (72.2%)	0 (0%) 24 (42.9%) 18 (60%) 5 (27.8%)	**0.021***
D dimer, mean±SD	287.46 ± 78.8	274.78 ± 76.33	0.402
HbA1c, n (%)	10.2 ± 2.12	6.46 ± 2.55	**<0.001****
WBCs, mean±SD	10.28 ± 3.06	9.65 ± 2.06	0.201
Hemoglobin (g/dl), mean±SD	11.79 ± 0.81	11.91 ± 1.17	0.542
Platelet (10^3/mm^3), mean±SD	198.7 ± 51.55	200.28 ± 53.53	0.876
ESR (mm/hr), mean±SD	19(10 – 30)	20(14.25 – 30)	0.536
CRP (mg/L), mean±SD	10.5(5 – 21.25)	12.5(5.75 – 21)	0.489
BUN (mg/dl), mean±SD	14.5(14 – 16.25)	14.5(12.83 – 19.08)	0.77
Creatinine (mg/dl), mean±SD	1.08 ± 0.65	1.15 ± 0.56	0.573
Albumin (g/dl), mean±SD	3.72 ± 0.5	3.74 ± 0.42	0.801
ALT, mean±SD	14.5(12 – 20)	13.5(12 – 15.5)	0.11
AST, mean±SD	15(14 – 26.4)	15(13 – 21)	0.256
Cholesterol,mean±SD	219.7 ± 42.83	206.75 ± 49.99	0.156
Triglycerides,mean±SD	125.83 ± 47.73	128.4 ± 38.43	0.762
LDL cholesterol (mg/dl), mean±SD	119.75 ± 33.13	115.85 ± 22.3	0.463
HDL cholesterol (mg/dl), mean±SD	43.36 ± 10.9	48.25 ± 10.8	**0.021***
PT (second),mean±SD	14.95 ± 3.18	14.34 ± 2.58	0.268
PTT (second),mean±SD	106.3 ± 7.37	104.77 ± 4.84	0.217
INR, mean±SD	1.17 ± 0.22	1.2 ± 0.27	0.566

Data is represented as mean ± SD and compared using independent sample t test
Data is represented as median (IQR) and compared using Mann Whitney test
Data is represented by n (%) and compared using Chi square test
*p<0.05 is statistically significant
**p≤0.001 is statistically highly significant
TOAST, Trial of Org 10172 in Acute Stroke Treatment
CIMT, carotid intima media thickness
HBA1c, Glycated hemoglobin
WBCs, white blood cells
ESR, erythrocyte sedimentation rate
CRP, C reactive protein
BUN, blood urea nitrogen
ALT, alanine transaminase
AST, aspartate transaminase
LDL, low density lipoprotein
HDL, high density lipoprotein

PT, prothrombin time
PTT, partial thromboplastin time
INR, international normalized ratio

Table (3): Predictors of cognitive impairment among studied patients.

	β	p	AOR	95% C.I.	
				Lower	Upper
IMT of carotid artery	-6.471	0.005*	0.002	0	0.145
HbA1C	0.736	<0.001**	2.088	1.601	2.723

AOR adjusted odds ratio **p≤0.001 is statistically highly significant CI Confidence interval

Table (4): Correlation between HbA1c and MoCA score (after controlling for age, sex and lipid profile).

	R	p
HbA1c	**-0.58**	**<0.001****

r Pearson correlation coefficient
** highly significant

DISCUSSION

Stroke is the second leading cause of morbidity and mortality **[1].** In the first year following stroke, the estimated prevalence of CI ranged from 17.5% to 54.9% with detrimental impact on the quality of life and represent an economic burden **[15].**

The goal of the current study was to investigate the relationship between HbA1c levels and cognitive deficits which occur after AIS. We included 110 patients in our study with definite diagnosis of AIS of which 50.9% were males and 49.1 % were females. Their mean age was 63.15 ±10.19 years.

In our study, CI was assessed 3 months after stroke onset using MoCA test with a < 25 score consistent with PSCI. Our results showed a significant positive association between elevated levels of HbA1c and PSCI at P value <0.001. In disagreement with our work, previous studies conducted on this relationship had shown mixed results. Two earlier studies did not find an association between HbA1c and PSCI **[16,17].** Also this was documented by two further studies that assessed CI three months after the onset **[10,11].** However other studies go in hand with our work, **Xu L and colleagues 2023** studied acute mild ischemic stroke and found that 38.59% of patients had cognitive dysfunction 3-6 months after stroke onset **[18]**. **Gong et al., 2021** found that 53.5% of mild stroke patients developed cognitive decline 6-12 months after onset using a cutoff value less than 22 to define cognitive dysfunction **[19].** In another study done in Korea 2021, they found that 21.6% of patients developed CI after 3 months of stroke **[11].** The reason below these differences may be related to the difference in the neuropsychological tests used in the evaluation of PSCI and different cutoff values for the same test.

An essential straightforward test for assessing CI is the MoCA exam **[4].** It covers most of the cognitive domains included in the fifth edition of Diagnostic and Statistical Manual (DSM-5), and correlates favorably with other batteries of neuropsychological assessments **[22].** However, cutoff values are still variable. In multiple studies**,** the previous initial cut-off point of 25/26 **[4]** had shown good sensitivity of equal or more than 83% and a specificity of 66% or less **[23,24].** Consequently, new cut-offs have been put to meet different populations and languages **[25].** The best cut-offs should be verified in separate samples since they are probably sample-dependent and unique to each study **[26,27].**

In the present study, on doing multivariate regression analysis of factors associated with CI among studied patients, we found that the increase in both the carotid intimal thickness and the HbA1c levels significantly independently increases risk of CI by 0.002 and 2.088 folds respectively. This result comes in agreement with **[28]** and **[29]** regarding carotid intimal thickness.

We noticed that 68.9% of our stroke patients who had sphincter dysfunction had CI. To the best of our knowledge, this result has never appeared in any other study.

As regards relation between CI and different comorbid chronic diseases, we found that there was statistically significant relation between CI and DM (82.9% of those with DM had CI). Diabetic patients have CI as early as 1week post-stroke **[30].** Previous old studies in 1990 had shown conflicting resultsin regards to cognitive function within 3 months. They reported that diabetes mellitus is the only risk factor for post stroke dementia (PSD) **[31,32].** A finding that was not duplicated in a later study with the same follow up period **[33]**. Diabetes mellitus impaired cognitive status of stroke patients and increased the occurrence of PSD 2-6 months following the event **[34].** These data were the same even after adjusting for stroke severity and the risk was more significant in men than in women **[35].** Variations between studies in the design, the demographic data, the ethnicity, the cognitive tests used and most notably the definition of hyperglycemia and glycemic control status could all be contributing factors to the discrepancies in these results.

Acute hyperglycemia following stroke could be a consequence of elevation of acute stress hormones like cortisol**,** or it can be the consequence of pre-existing diabetes mellitus and prediabetes **[36].** In cases of brain injury, several pathophysiological pathways have been noted. Basically, hyperglycemia may increase lactate levels in the ischemic brain, which may result in intracellular acidosis **[37].** The consequent acidity increases lipid peroxidation, production of reactive oxygen species and mitochondrial dysfunction, aggravating subsequent insult to the ischemic brain **[38].** Moreover, people who have hyperglycemia but have never been diagnosed with diabetes are more likely to have impaired glucose tolerance or insulin resistance **[36].** This condition is also associated with reduced peripheral glucose uptake and endothelial dysfunction, both of which are known to exacerbate secondary neuronal injury after an ischemic stroke **[39].**

Furthermore, we found that CI negatively correlated to HDL levels. Our findings go in hand with Wang et al., 2024 who described the same finding **[40].** Another study showed that low HDL levels is associated with poor memory and memory decline in middle-aged adults **[41].**

Our study has several limitations. First, we did not consider control of blood glucose levels with antidiabetic medications during hospitalization. Second, we assessed cognition 3 months after onset of stroke, which is a relatively short duration. Long term follow up of cognitive functions needs to be assessed in future research. Third, we included in our cohort all types of stroke whatever the severity, which might be a confounding factor as severe motor disability might influence the cognitive assessment. Further studies focusing on mild ischemic stroke patients should be considered.

Conclusion: Our study found the occurrence of PSCI to be independently related to higher Glycosylated hemoglobin levels and increased thickness of intima media of the carotid arteries. Careful evaluation of these factors help clinician to intervene early and develop better treatment modalities.

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