

Cerebral Infarction in Diabetes Mellitus: A Comparative Study of Diabetic and Non-Diabetic Ischemic Stroke

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ABSTRACT

Background and purpose: Although diabetes is strong risk factor for stroke, it is still unclear whether stroke subtypes, severity, and prognosis are different in diabetic and non-diabetic patients. The present study was carried out to evaluate distinctive features of diabetic and non-diabetic ischemic stroke and to compare short-term prognosis between two groups. **Methods:** Fifty ischemic stroke cases categorized into group I of 25 known diabetic and 25 non-diabetics were included in this study. Patients were evaluated clinically at admission using Canadian Neurological Scale (CNS) and after one month by Barthel Activity of Daily Living (ADL). Risk factors were evaluated and laboratory investigations included admission glucose level (AGL), glycosylated hemoglobin (HbA1c), cholesterol and triglyceride serum levels were assessed. CT scan was done to all patients at admission and a repeat one when needed. **Results:** The present study showed that diabetic patients with stroke were, on average 3.2 years, younger than non diabetic stroke patients. Smoking either current or previous was less common in diabetic than non diabetic stroke patients (36% Vs, 40%) respectively. Transient ischemic attacks (TIA) were more frequent among patients with diabetes than without (40% vs 28% respectively). Hypertension was more frequent in our diabetic patients than in non diabetic stroke patients (60% vs 48% respectively). In our study, the levels of cholesterol and triglyceride were higher in stroke diabetic patients than non diabetics which were significant in triglyceride. Regarding the clinical characteristics of our stroke patients, dysarthria was more common in diabetic patients than non diabetics (32% Vs 20% respectively) and aphasia was more common in non diabetic stroke patients than diabetics (20% Vs 8% respectively). Furthermore, the distributed of clinical subtypes of stroke in our diabetic group was different, with more lacunar syndromes than non-diabetic (60% vs 36% respectively). We found that stroke patients with diabetes had significantly worse Canadian scale and non significantly worse outcome by ADL than those without diabetes. As regards the CT findings in our stroke patients we found non statistically significant differences between stroke patients with and without diabetes regarding site and size of cerebral infarcts and the presence of brain edema. However, subcortical infarcts were more common and cortical infarcts were less common in our diabetic patients. We found that urinary incontinence, hypertension, and glycosylated hemoglobin >6.2% had significant predictive values for poor ADL score outcome in diabetic patients [odd ratio (OR) =13.33 (95% confidence interval [CI], 1.1 to 364.1) for urinary incontinence, OR=12.8 for hypertension (95% CI, 1.04 to 333.6), and OR= 11.9 for HbA1c>6.2%, (95% CI, 1.4 to 102)]. **Conclusion:** Stroke in diabetic patients has a specific clinical pattern and a poor prognosis, which emphasizes the need for early diagnosis and treatment of every case of diabetes. (Egypt J. Neurol. Psychiat. Neurosurg., 2006, 43(1): 167-177)

INTRODUCTION

Though there are extensive data on diabetic retinopathy and peripheral vascular disease, there are not enough data on diabetic stroke¹. Diabetes mellitus (DM) is a well-established independent risk factor for stroke^{2,3,4}, and this increased risk

has been linked to the pathophysiological changes seen in the cerebral vessels of patients with diabetes³. However, it is still unclear whether stroke features, severity, and prognosis differ in diabetic and non-diabetics^{5,6}. Several studies showed that patients with diabetes who develop stroke have a less favorable outcome than those

without^{7,8,9,5}; however, a few other studies did not confirm these findings^{10,11}.

The aim of this study was to compare prospectively the characteristics of stroke patterns and severity, and to estimate recovery and short-term prognosis in diabetes and non-diabetes patients.

SUBJECTS AND METHODS

Fifty patients presenting within 24 hours of ischemic stroke according to the world Health organization (WHO)¹², with brain imaging confirmation were recruited prospectively from the Stroke Care Units of Neurology and Internal Medicine Departments, Zagazig University Hospitals. Patients were evaluated in the acute phase and one month after stroke.

Exclusion criteria:

Patients were excluded if they had intracerebral or subarachnoid hemorrhage, posterior circulation infarction, history of previous stroke that would hamper interpretation of clinical or radiological data, or patients with Canadian Neurological Scale (CNS) score lower than 1.5. In addition, we excluded those patients with subsequent resolution of neurological symptoms and absence of lesion on acute and repeated CT scan. Also patients who initially in deep coma or died during the follow up period were excluded.

Diagnosis of diabetes:

Patients were divided into 2 groups according to patient recall or medical records: group [I]; including 25 patients with known diabetes, treated with either insulin therapy or oral hypoglycemic therapy or not treated, whatever the plasma glucose level at stroke onset; and group [II], composed of 25 patients with no past history of diabetes. In keeping with current diagnostic criteria for the definition of diabetes, we defined repeated fasting plasma glucose level of 126 mg/dL as diagnostic criteria for diabetes¹³. Besides, glycosylated hemoglobin (HbA1c) levels were obtained, and a raised HbA1c was defined as 6.2%¹³.

Vascular risk factors:

Including hypertension (previous diagnosis, current treatment or values > 160/95 mmHg), atrial fibrillation (history of chronic atrial fibrillation, confirmed by at least ECG, or presence of the arrhythmia during hospitalization); previous myocardial infarction, transient ischemic attack (TIA), lipid profiles and smoking (current or former habit).

Clinical assessment:

Baseline characteristic included age, sex, medication before stroke (e.g. anti hypertensive) were determined. Clinical state was assessed and clinical severity of stroke was rated according to Canadian Neurological Scale (CNS)¹⁴. This standardized neurological scale was used for evaluation of the level of consciousness, orientation, speech, facial and limb strength, with a global score ranges from 1.5 (maximum deficit) to 10 (absence of deficit). Since all patients were non comatosed and have moderate to severe neurological deficit at hospitalization, the initial score was never lower than 1.5 or higher than 5.8.

Clinical subtypes of stroke.

Clinical subtypes of ischemic stroke were rated according to the Oxford shire community stroke project criteria¹⁵, as total anterior circulation infarct (TACI), partial anterior infarct (PACI), and lacunar infarct (LACI).

Brain CT scan:

CT scans were performed to all patients at admission utilizing a general electric system SRI tomoscan with tissue matrix = 265x265 one second scanning time and 9mm slice thickness. In cases with absent lesion at the first CT scan, a repeated one after 48 hours was done. The size of infarcts was estimated according to the following formula; size = $0.5 \times a \times b \times c$; where a, b= largest perpendicular diameter measured on CT Scan, and c= slice thickness. Mean values for the size of the lesion were calculated and the lesions were classified according to their size into; small, when size was less than half of the calculated

mean values; moderate, when size was less than the calculated mean values but more than its halves; and large, when size exceeded the mean values. The presence or absence of brain edema was also evaluated. The site of infarct was defined as follow: [1] subcortical involvement of deep branch of middle cerebral artery (MCA) or internal border zone area; [2] cortical involvement of the territory of the superficial branches of the MCA; and [3] corticosubcortical, with concomitant involvement of the deep and superficial territories of the MCA¹⁶.

Outcome data after one month:

Short-term outcome data included disability using Barthel activity of daily living (ADL)¹⁷. The functional abilities of patients were measured using this index one month after stroke onset. This scale gives score between 0 and 20 in one point increments. The top score of 20 implies functional independence (not necessarily normality). Information about function was taken from the most available source (e.g. nurses, relatives, or the patients). There is a clear hierarchy among items of the scale (bowel, bladder, grooming, toilet use, feeding, transfer "bed-chair", walking, dressing, stairs and bathing). As bathing alone is the most difficult scores, disability at one month was defined as ADL score of < 12.

Statistical analysis:

Data were collected and statistically processed using Epi Info software package. Descriptive statistics was presented as means±SD. The relationships between baseline and clinical variables and diabetes were analyzed with the Chi square test and t test for categorical and continuous variables, respectively. For the assessment of associated factors with disability after one month, Odd Ratio (OR) (95% CI) was calculated.

RESULTS

The present study showed that diabetic patients with stroke were, on average 3.2 years, younger than non diabetic stroke patients. There

was no sex difference between the diabetic and non diabetic stroke patients. Smoking either current or previous was less common in diabetic than non diabetic stroke patients (36% Vs, 40%) respectively. Transient ischemic attacks (TIA) were more frequent among patients with diabetes than without (40% vs 28%, respectively). Atrial fibrillation was equally distributed in both groups whereas myocardial infarction was more common in diabetic group. Hypertension was more frequent in our diabetic patients than in non diabetic stroke patients (60% vs 48% respectively). Antihypertensive therapy was prescribed in a greater proportion of diabetic hypertensive patients (44%) than in non-diabetic patients (28%). Three out of 14 patients in diabetic group received more than one antihypertensive drug compared to two in non-diabetic group (Table 1).

In our study, the levels of cholesterol and triglyceride were higher in stroke diabetic patients than non diabetics which were significant in TG. The serum levels of admission glucose and HbA1c were significantly higher in stroke patients with diabetes than without.

Regarding the clinical characteristics of our stroke patients, dysarthria was more common in diabetic patients than non diabetics (32% Vs 20% respectively) and aphasia was more common in non diabetic stroke patients (20% Vs 8% respectively). Furthermore, the distributed of clinical subtypes of stroke in our diabetic group was different, with more lacunar syndromes than non-diabetic (60% vs 36% respectively). We found that stroke patients with diabetes had significantly worse Canadian scale and non significantly worse outcome by ADL than those without diabetes (Table 3).

As regards the CT findings, we found non statistically significant differences between stroke patients with and without diabetes regarding site and size of cerebral infarcts and the presence of brain edema (Table 4).

We found that urinary incontinence, hypertension, and glycosylated hemoglobin > 6.2% had significant predictive values for poor

ADL score in diabetic patients [odd ratio (OR) =13.33 (95% confidence interval [CI], 1.1 to 364.1) for urinary incontinence, OR=12.8 for

hypertension (95% CI, 1.04 to 333.6), and OR=11.9 for HbA1c>6.2%, (95% CI, 1.4 to 102)] (Table 5).

Table 1. Demographic data and risk factors of stroke patients with diabetes (group I) and those without diabetes (group II).

Variables	Group I	Group II	P
Age: in years (X±SD)	58.8±10.1	61.6±12.5	0.263
Sex: (No/%)			
Male	13(52)	15(60)	0.569
Female	12(48)	10(40)	
Risk factors: (No/%)			
- Smoking	8(36)	10(40)	0.77
- TIA	10(40)	7(28)	0.342
- AF	5(20)	5(20)	1.0
- MI	9(36)	7(28)	0.66
- Hypertension	15(60)	12(48)	0.39
SBP (X±SD)	156.8±23.3	143.2±14.0	0.015*
DBP (X±SD)	94.4±11.6	86.8±8.0	0.004*
Antihypertensive drugs(No/%)	14(56)	10(40)	0.25
Ca++ channel blockers	6(42.9)	3(30)	0.83
Beta blockers	8(57.1)	6(60)	0.76
AC inhibitors	3(21.4)	3(30)	0.66

* Significant

SBP: Systolic blood pressure.

DBP: Diastolic blood pressure.

TIA: Transient ischemic attacks

AF: Atrial fibrillation

MI: Myocardial infarction

Table 2. Laboratory data in patients with diabetes (group I) and those without diabetes (group II).

Variables: (X±SD)	Group I	Group II	P
Triglyceride	211.6±80.2	166.5±35.8	0.012*
Cholesterol	255.4±84	221.7±60	0.1
Admission glucose level	259.8±63.9	173.6±33.4	<0.001*
Glycosylated hemoglobin	8.4±1.3	5.2±0.86	<0.001*

* Significant

Table 3. Clinical characteristics and clinical stroke classification in patients with diabetes (group I) and those without diabetes (group II).

Variables	Group I	Group II	P
Clinical characteristics: (No/%)			
Dysarthria	8(32)	5(20)	0.33
Dysphasia	7(28)	9(36)	0.66
Confusion	11(44)	7(28)	0.67
Urinary incontinence	10(40)	8(32)	0.77
Clinical classification: (No/%)			
TACI	4(16)	4(16)	0.17
PACI	6(24)	12(48)	
LACI	15(60)	9(36)	
CNS: (X±SD)	6.5±1.1	5.3±1.7	0.008*
ADL: (X±SD)	8.4±4.0	9.4±3.3	0.64

TACI: Total anterior circulation infarction.

PACI: Partial anterior circulation infarction

LACI: Lacunar infarction.

CNS: Canadian Neurological Scale.

ADL: Barthel Index of daily activity

* Significant

Table 4. CT findings in stroke patients with diabetes (group I) and those without diabetes (group II).

Infarcts	Group I		Group II		X ²	P
	No	%	No	%		
Size:						
Small	14	56	10	40	2.13	0.34
Medium	4	16	3	12		
Large	7	28	12	48		
Site:						
Cortical	4	16	6	24	1.35	0.5
Subcortical	13	52	9	36		
Corticocortical	8	32	10	40		
Edema:						
Present	10	40	12	48	0.32	0.56
Absent	15	60	13	52		

Table 5. Association between baseline and clinical variables and disability defined as ADL (0-12).

Variables	Total No=50 OR (95% CI)	Group I No=25 OR (95% CI)	Group II No=25 OR (95% CI)
Gender:			
Male	1	1	1
Female	1.26(0.32-5.14)	5.83(0.69-60.17)	0.23(0.02-2.18)
Age:			
≤55	1	1	1
>55	2.19(0.61-0.39)	1.25(0.18-8.78)	0.09(0.01-1.05)
No confusion	1	1	1
Confusion	1.59(0.4-6.47)	2.08(0.3-15.33)	1.45(0.16-15.3)
No urinary incontinence	1	1	1
urinary incontinence	5.33(1.1-29.22)*	13.33(1.1-364.1)*	2.22(0.24-23.4)
No hypertension	1	1	1
Hypertension	4.65(1.05-22.1)*	12.8(1.04-333.6)*	2.1(0.27-17.63)
HbA1c < 6.2%	1	1	1
HbA1c > 6.2%	2.83(1.25-6.43)*	11.9(1.4-102)*	2.08(0.86-5.01)

* Significant

HbA1c= Glycosylated hemoglobin

DISCUSSION

The main purpose of our study is to evaluate the impact of diabetes mellitus (DM) on initial stroke type patterns and outcome in diabetic patients.

The present study showed that diabetic patients with stroke were, on average 3.2 years, younger than non diabetic stroke patients. This is in accordance with Sarkar et al.¹, who found that average age of diabetic stroke was lower than non diabetic. The younger age of diabetic stroke patients could be explained as hyperglycemia accelerate and aggravate cerebral ischemia¹⁸. Also premature onset and rapid progression of atherosclerosis are characteristic features of diabetes¹⁹. On the contrary, Devuyst et al.⁶, found that diabetic stroke patients were older than the non diabetic stroke patients. Jorgensen et al.³ and Megherbi et al.⁵, found that diabetic patients with stroke were the same age as non diabetic patients. This difference was probably caused by difference in the selection of patients, in the size of cohort,

and in the definition of diabetes and methods of measuring hyperglycemia.

Regarding the sex, there was no difference between the diabetic and non diabetic stroke patients. This is in agreement with Tuomilehto et al.⁹; Currie et al.²⁰, and Magherebi et al.⁵, and the last authors explained this on the ground that diabetes has the same impact on cerebral vessels in both sexes. Lehto et al.²¹ and Sarkar et al.¹ found a female preponderance in diabetic stroke patients. On the contrary, Davis et al.²², demonstrated that men had a relative risk of stroke more than 1.5 times that of women in patients with DM and they explained this on the ground that women with diabetes probably had less cerebrovascular "protection" associated with sex.

Unexpectedly, smoking either current or previous was less common in diabetic than non diabetic stroke patients (36% Vs, 40%) respectively. This in accordance with Abbott et al.²³, who found smoking in 38.8% and 44.4% respectively in diabetic and non diabetic subjects. In support, Tuomilehto et al.⁹ in their study about DM as a risk factor for death from stroke, found

that the effect of smoking on risk of death from stroke was more pronounced in non-diabetic than diabetic subjects. Megherbi et al.⁵ found that smoking was distributed equally in both diabetic and non diabetic stroke patients.

In the current study, transient ischemic attacks (TIA) were more frequent among patients with diabetes than without (40% vs 28%, respectively). This coincides with Alter et al.²⁴ (22.2% vs 16.6%) and Sarkar et al.¹ (22.8% vs 7.5%) for diabetic and non diabetic stroke patients respectively and in the last study it was highly significant. Nevertheless, the proportion of previous TIA was similar in both groups in the study of Megherbi et al.⁵ (12.6% vs 12.3% in diabetic stroke patients Vs non diabetic stroke patients respectively). The last authors suggested that the effects of hypertension (which was more frequent in diabetic stroke patients) and atrial fibrillation (which was more frequent in non diabetic stroke patients) are balanced equally in the 2 groups. In our study hypertension was more frequent in diabetic stroke patients and AF was equal and these could explain why TIA was more frequent in that group.

Hypertension, which is an important risk factor for stroke, was more frequent in our diabetic patients than in non diabetic stroke patients (60% vs 48% respectively). This is in agreement with Sarkar et al.¹, who found hypertension in 70.9% of their stroke patients with diabetes compared to 47.6% in stroke patients without diabetes. Furthermore, the mean values of systolic and diastolic blood pressures were significantly higher in diabetic than non-diabetic patients. Hypertension that is more frequent in diabetic patients were observed in other previous studies^{9,21,24,22,5} emphasizing the fact that diabetes and hypertension are associated²⁵. Furthermore, Tuomilehto et al.⁹ mentioned that it is possible that some of the effect of hypertension on the risk of stroke also can be attributed to glucose intolerance rather than blood pressure. It has been shown that diabetes results in an increased permeability of vessel walls, particularly in patients with proteinuria or microalbuminuria.

Microalbuminuria is an independent determinant of coronary heart disease in diabetic subjects²⁶. With the same mechanism, diabetes may cause cerebrovascular disease as well⁹.

Antihypertensive therapy was prescribed in a greater proportion of diabetic hypertensive patients (44%) than in non-diabetic patients (28%), which in accordance with Lehto et al.²¹ and Megherbi et al.⁵. In contrast, antihypertensive drug treatment was not found to be independently associated with increased risk of death from stroke in either diabetic or non-diabetic cohort⁹. In addition, there was no difference in types of antihypertensive therapy in both diabetic and non-diabetic patients in our study. Nevertheless, in one study, effective antihypertensive drug treatment seemed to reduce the risk of stroke similarly in both diabetic and non-diabetic subjects²⁷.

Hypertriglyceridemia is the most common lipid abnormality in patients with DM although lipid abnormalities have been shown to be associated with cerebral atherosclerosis data on the relationship between dyslipidemia and stroke is limited²¹. In our study, the levels of cholesterol and triglyceride were higher in stroke diabetic patients than non diabetics which were significant in TG. Sarkar et al.¹, found that hypercholesterolaemia was an important risk factor in 30.9% and in 21.1% in stroke patients with diabetes than without diabetes respectively which was significant ($P < 0.001$). Furthermore, Arboix et al.²⁸ found that hyperlipidemia was independently associated with ischemic stroke in patients with diabetes. What is the explanation for the association of hyperlipidemia and risk of stroke? Impaired fibrinolysis has emerged as a new risk factor for ischemic heart disease²¹. Fibrinolytic activity in blood is regulated mainly by plasma plasminogen activator inhibitor (PAI₁). Moreover, increased levels of PAI-1 have been demonstrated in patients with DM and in patients with coronary heart disease and stroke²⁹. Furthermore, triglyceride level has been shown to correlate with PAI-1.

The relationship between hyperglycemia and stroke has been previously reported^{30,31,18,32}. Thus

poor metabolic control, reflected by high blood glucose and glycosylated hemoglobin (HbA1c), is a major risk factor for stroke independent of age²¹. However, the test of (HbA1c) reflects exposure of individual's red blood cells to the mean blood glucose level during the proceeding 2 to 3 months. It is widely considered to be the best and most sensitive measure of control of blood glucose currently available²⁴. In our study, the serum levels of admission glucose and HbA1c were significantly higher in stroke patients with diabetes than without. The same results were obtained by other authors^{21,24,1}. Poor metabolic control accelerates diabetic microvascular disease, but the importance of hyperglycemia with respect to macrovascular disease in non insulin dependent diabetes mellitus (NIDDM), is still controversial. Hyperglycemia is related to atherogenic lipoproteins changes and is also a procoagulant state. Hyperglycemia can decrease prostacyclin synthesis, increase thrombosis formation, and cause glycosylation of proteins in the artery wall. These conditions, which are related to poor metabolic control, can cause atherosclerosis, which could contribute to increase the risk of stroke³⁰.

Regarding the clinical characteristics of our stroke patients, dysarthria was more common in diabetic patients than non diabetics (32% Vs 20% respectively). However aphasia was more common in non diabetic stroke patients (20% Vs 8% respectively). This is in agreement with Kiers et al.⁸, Jorgensen et al.³ and Megherebi et al.⁵, and the last ones postulated that dysarthria may be interpreted as the consequence of bilateral small lesions affecting pyramidal corticonuclear tracts by means of lacunar lesions. In support in our diabetic group, the distributed of clinical subtypes of stroke was different, with more lacunar syndromes than non-diabetic (60% vs 36% respectively). This result is keeping with Megherebi et al.⁵, Sarkar et al.¹ and Arboix et al.²⁸, who found that people with diabetes compared to people without diabetes presented more frequently lacunar infarcts. The fact that we observed more lacunar infarct syndromes in

diabetic patients has not been reported in other study⁶ which raises the problem of biases induced by small series, the definition of diabetes, and other methodological aspects³³. The influences of diabetes on various vascular lesion that cause brain ischemia was postulated by Caplan³⁴: (1) Atherosclerosis of the large intracranial arterial; (2) intracranial atheromatous branch disease of macroscopically visible branches of the intracranial arteries; (3) degenerative abnormalities such as lipohyalinosis and fibrinoid changes within penetrating artery branches visible only microscopically. These three types of disorders all can cause deep subcortical brain infarcts, the predominant type of brain infarction in diabetic patients.

As regards the effects of DM on the clinical severity, we found that stroke patients with diabetes had significantly worse Canadian scale than those without diabetes. Some clinical series support the possibility that diabetes leads to greater severity rather than greater frequency. For example, in one case series of patients with ischemic stroke clinical severity was significantly greater in diabetics than non diabetics³⁵. In another case series of patients coming to non invasive testing for carotid artery disease, patients with diabetes had significantly more irreversible neurologic deficits, than patients without diabetes³⁶. Barrett-connor and Khaw², stated that it is possible that diabetes predisposes not to stroke per se but more irreversible brain damage during ischemia. On the contrary, Devuyt et al.⁶, found that stroke severity did not differ between diabetics and non diabetics and this may be due to inclusion of subjects with cerebral hemorrhage.

As regards the CT findings in our stroke patients we found non statistically significant differences between stroke patients with and without diabetes in site and size of cerebral infarcts and the presence of brain edema. However, we found that subcortical infarcts were more common (52%) and cortical infarcts were less common (16%) in diabetic group. This result go in hand with Devuyt et al.⁶, who found that superficial infarcts were less frequent in diabetics,

while subcortical infarcts were more common in diabetics, irrespective of concomitant hypertension. Analysis of a large cohort by Toni et al.³⁷, initially indicated no association between admission glucose within 12 hours of hemispheric stroke and outcome CT lesion size. Other groups using conventional MRI did not show an association between the presence of diabetes and the size of ischemic lesion³⁸. Dora et al.³², found no correlation between the mean of glycemic regulation (MGR) for 10 days and the lesion size in CT scan, despite the patients with marked cerebral edema had a significantly higher MGR compared to patients with lesser edema. On contrary, Horowitz et al.³⁹ using CT found that admission glucose levels correlated with infarct size and hemorrhagic transformation. Lastly, Baird et al.⁴⁰, postulated that persistent hyperglycemia on serial glucose monitoring is an independent determinant of infarct expansion and is associated with worse functional outcome. Differences between our results and others may reflect in part differences in the definition of diabetes, size of samples, the timing of hyperglycemia which can not readily controlled for, and the definition of hyperglycemia with a single measure of blood glucose at a variable time period after the ictus is problematic⁴⁰.

Several studies showed that patients with diabetes who develop stroke have a less favorable outcome than those without^{7,8,9,20,5}. In accordance with the previous reports, our study showed that stroke patients with diabetes had worse clinical outcome, though non significant, measured by ADL than those without diabetes. However, a few other studies did not confirm these findings^{11,6}. Interestingly, most studies have shown that admission hyperglycemia is a risk factor for poor outcome following local and global cerebral ischemia^{41,42,43,40,32,44}. In the present study, in agreement with those studies, we found that admission glucose and HbA1c were significantly higher in stroke diabetic patients, who had worse outcome than non diabetics. In addition, we found that HbA1c > 6.2% had OR of 11.9, which was significant, for poor prognosis measured by ADL

in our diabetic patients. Furthermore, hypertension and urinary incontinence had significant predictive value for a poor Barthel Index score in diabetic patients. This is in agreement with Megherbi et al.⁵, who found that female sex, old age, dysphagia, confusion, and urinary incontinence are factors that increase the risk of disability measured by the Barthel Index. Urinary incontinence as a predictive value for poor outcome in our diabetic patients could be attributed to autonomic neuropathy which through several mechanisms could markedly increase the risk of stroke in NIDDM⁴⁵. Thus, as suggested by ADL scores, diabetic stroke patients recovered more slowly than those without diabetes. Various mechanisms may account for poorer outcome in diabetic stroke patients, including more comorbidities, more prestroke disability, more ischemic lacunas, more motor problems, and diabetic neuropathy⁵.

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الملخص العربي

الجلطة المخية في مرض السكر: دراسة مقارنة بين الجلطة المخية في مرضى السكر و غير مرضى السكر

بالرغم من أن مرض السكر يعتبر عامل من عوامل الخطورة في حدوث الجلطة المخية إلا أنه إلى الآن لم يتضح إذا ما كانت جلطة المخ في مرضى السكر تختلف عنها في غير مرضى السكر سواء في أنواعها أو درجة شدتها أو مدى تطور المرض ولهذا تم عمل هذه الدراسة لكي نستطيع التفريق بين الجلطة المخية في مرضى السكر وغير مرضى السكر.

وقد أجرى البحث على 50 مريضاً بالجلطة المخية تم تقسيمهم تبعاً إلى مجموعتين:

1- المجموعة الأولى: وتضم 25 مريضاً بالجلطة المخية بالإضافة إلى مرض السكر

2- المجموعة الثانية: وتضم 25 مريضاً بالجلطة المخية بدون مرض السكر

وقد تم تقييم المرضى إكلينيكيًا بالمقياس الكندي مع قياس عوامل الخطورة للجلطة المخية. كما تم عمل أشعة مقطعية على المخ لجميع المرضى. وقد تم إعادة تقييم للمرضى بمقياس بارثل لقياس مدى تطور المرض.

وقد أظهرت نتائج البحث أن متوسط أعمار مرضى السكر بالجلطة المخية كانت أصغر بحوالي 3.2 سنة من هؤلاء من دون مرضى السكر. وبينما كانت نسبة التدخين أقل في المجموعة الأولى كان ضغط الدم المرتفع أكثر في ذات المجموعة.

كما وجد أن المقياس الكندي الإكلينيكي أسوأ في المجموعة الأولى عن الثانية بالإضافة إلى أن مقياس بارثل لقياس مصير المرض كان أسوأ أيضاً في ذات المجموعة. أما بخصوص نتائج الأشعة المقطعية على المخ لم نستطع إيجاد فرق يذكر بين المجموعتين.

مما سبق نستخلص أن صورة الجلطة المخية في مرضى السكر تختلف عنها في غير مرضى السكر. حيث أن الجلطة المخية في مرضى السكر أشد إكلينيكيًا وأسوأ مصيراً عن دون مرضى السكر.