EARLY NEUROLOGICAL DETERIORATION IN ACUTE ISCHEMIC STROKE: POTENTIAL PREDICTORS, CAUSES AND RELATION TO INFARCT GROWTH

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ABSTRACT:

Background: *Worsening of acute stroke early in its course (within 48–72 h of its onset) is a common occurrence and has potentially serious short term and long term consequences. The incidence of early neurological deterioration (END) among hospitalized patients varies widely in different studies between 13% and 38%.*

Aim of the Work: *To define incidence and timing of END in relation to acute ischemic stroke (AIS) onset. To identify possible causes and predictors associated with END. To assess the relation between END and the patient functional level at three months post stroke.*

Patients and Methods: *Three hundred patients were recruited into this hospital prospective comparative study. Clinical history, laboratory indices, structural brain imaging, Magnetic Resonance Angiography (MRA) and Carotid Duplex ultrasonography were done. Patients were examined on NIHSS and Glasgow Coma Scale (GCS) in day 1, 2, 3 and patients with END did a follow up (MRI diffusion film or CT brain) at day4 or 5 and all patients were followed up by Modified Rankin scale (MRs) at three-month post stroke.*

Results: *Of the Three hundred patients included in the study, the incidence of END was 16.7%. The median NIHSS on admission was 9.25. END was associated with long duration of DM (P 0.012), IHD (p 0.015), AF (p 0.048), severe stroke (p 0.0044), low blood pressure on admission (p 0.0079), high uric acid (p 0.033) and MCA occlusion (p 0.0007). END was associated with significant increase in MRS at 3 month ($p<0.0001$) and mortality rate (44% vs 4.4). Patients with END are more prone for aspiration pneumonia (p 0.0001) and hemorrhagic transformation.*

Conclusion: *Early neurological deterioration is a frequent complication after acute stroke, with a poor short-term prognosis. This study provides that hyperglycemia, hyperuricemia and cardiac disease (IHD and AF) may increase the risk of END.*

Keywords: Early Neurological Deterioration, Acute Ischemic Stroke.

INTRODUCTION:

Worsening of acute stroke early in its course (within 48–72 h of its onset) is common and has potentially serious short term and long term consequences for the patient[(1)]. Various terms, such as ‘‘progressive stroke’’, ‘‘stroke in evolution’’, and ‘‘stroke in progression’’ have been used to describe this worsening[(2)]. Now most

commonly termed early neurological deterioration (END)[3].

END was defined in the European progressing stroke study (EPSS) as any significant neurological deterioration from baseline to 72 hours[3].

A significant neurological deterioration was primarily defined as a decrease in the Scandinavian stroke scale (SSS) items score for consciousness, speech, gaze, arm, or leg by at least 2 points. Consciousness was given precedence over the other signs[3].

Many studies considered increase in the total national institute of health stroke scale (NIHSS) score by at least 2 points a significant neurological deterioration[3,4]. Others considered increase in the total (NIHSS) score by at least 4 points [5]**.** Bathia and his colleague considered increase in the total (NIHSS) score by at least 3 points [6]**.** Siegler and his colleague compared between the increase in the NIHSS score by ≥2 or ≥4 points and founded that A ≥2 point neurological deterioration is a sensitive indicator of poor outcome and in hospital mortality[7]**.**

The incidence of END in ischaemic stroke among hospitalized patients varies widely in different studies. 19% of acute stroke patients had END[8], 28%[9], 37.5%[10]. In the Harvard Cooperative Stroke Registry that included haemorrhagic and non haemorrhagic strokes, early worsening was noted in 20% of patients[11]. In the Barcelona Stroke Registry of 3577 consecutive patients hospitalized with stroke (all types), 37% showed END[12]. Rates of 29% and 25%, respectively, were reported for all acute strokes in Swiss[13] and Japanese[14] studies.

The differences in the time scale of assessments after acute stroke, diagnostic criteria used for END, and the case mix of stroke patients could account for the wide variations reported in the studies. Even among ischaemic strokes, subtypes differ in the rates of END[15]**.**

END after acute ischemic stroke (AIS) has been found to be related to case fatality and reduced functional outcome[16]**.** Various mechanisms and predictors have been postulated to explain END[3].

AIM OF THE WORK:

- To define the incidence, severity and timing of END in relation to AIS onset
- To identify factors and possible causes and predictors associated with END after AIS
- To assess the relation between END after AIS and the patient functional level at three months post stroke including case fatality

PATIENTS AND METHODS:

Methods:

A hospital based prospective observational study carried out during the period from January 2017 to June 2018 at Ain Shams University Hospitals.

The definition of END was (increase in the National Institute of Health Stroke Score (NIHSS) by two or more points (or stroke related death) between admission and day 3).

Patients included in the study subjected to:

Clinical Data

Full clinical history and examination was obtained by a trained neurologist. The following data were expressly recorded for analysis:

Age, Sex, Smoking status, Diabetes Mellitus, hypertension, hyperlipidemia, heart disease (ischemic and otherwise) and History of previous Transient Ischemic Attacks (TIA) and/or stroke.

Laboratory Indices

The patients had the following laboratory tests done (venous blood samples):

Complete Blood Count, Fasting and glycosylated hemoglobin (HBA1c), Serum lipid profile, Serum Uric Acid (Hyperuricaemia was defined as serum uric acid above 7.0 in men or above 6 in women), Renal function tests (serum creatine and blood urea nitrogen), Liver function tests (liver enzymes, serum albumin and coagulation profile and Electrolytes (sodium, potassium and calcium).

Structural Brain Imaging:

Magnetic Resonance Imaging:

Magnetic Resonance Imaging (MRI) of the brain was done on a 1.5 tesla General Electric system (Signa Prospeed LX). The imaging protocol included the following sequences: Fast Spin Echo (FSE) Axial T1-weighted imaging, Axial T2-weighted imaging, Axial Fluid Attenuated Inversion Recovery (FLAIR), axial single-shot multi-slice diffusion-weighted echo-planar imaging pulse sequence (DWI-EPI) and Axial EPI Gradient Echo T2*. MR angiography was performed during the same session when available as detailed below.

Vascular Imaging

Magnetic Resonance Angiography

Three-dimensional Time-of-flight (TOF) Magnetic Resonance Angiography of the cerebral vessels was done on a 1.5 tesla General Electric machine (Signa Prospeed LX). The images were calibrated and visually inspected for presence of stenosis or occlusion of the major arteries on both sides (ICA, M1of MCA, P1 of PCA and BA).

Carotid Doppler Ultra sonography

This was done on by a trained radiologist using a 7.5 MHz transducer on a Philips ultrasound machine. Peak Systolic Flow and Mean Flow Velocities were measured for the common carotid (CCA), the internal carotid and the external carotid arteries on both sides.

Significant stenosis of the carotid arteries was defined as >70% narrowing.

Echo Cardiography:

To detect cardiac source of emboli and manifestations of long standing hypertension (HTN).

Trans thoracic Echo was done for all patients (trans esophegeal for selected patients) to detect: Atrial diameter, ejection fraction, cardiomyopathy and intracardiac thrombus.

ECG: To detect AF.

- All patients were examined on NIHSS and Glasgow Coma Scale (GCS) in the 1-2-3 day.
- All patients with END did a follow up MRI diffusion film or CT at day 5.
- All patients were followed up by Modified Rankin scale (MRs) at three-month post stroke.

Statistical Analysis:

Data entry, processing and statistical analysis was carried out using Med Calc ver. 18.2.1 (Med Calc, Ostend, Belgium). Tests of significance (Mann-Whitney's, and Chi square tests, logistic regression analysis, and Spearman's correlation) were used. Data were presented and suitable analysis was done according to the type of data (parametric and non-parametric) obtained for each variable. P-values less than 0.05 (5%) was considered to be statistically significant.

RESULTS:

Baseline data:

The mean age of all patients was (61.83 ± 11.5) years. Regarding gender of the patients, the majority (66.7%) of patients were males, while (33.3%) were females.

HTN was prevalent among study population (67.9%) with average duration of (11.2 ± 6.78) years, (50%) of patients had DM with average duration of (14.58 ± 8.6) years, (13.3%) had AF and (35%) had ischemic heart disease (IHD).

Table 1: Co -morbidities among 300 AIS patients:

Variables	Frequency (%)
HTN	203 (67.9%)
DM	150 (50%)
AF	40 (13.3%)
IHD	105 (35%)
Hyperlipidemia	23 (7.7%)
Collagen disease	2 (0.7%)
Previous Stroke	62 (20.7%)
TIA	15 (5%)
Variable	Mean ± SD
Duration of HTN	11.2± 6.78 y
Duration of DM	14.58± 8.6 y

HTN: hypertension, DM: diabetes mellitus, IHD: ischemic heart disease. AF: atrial fibrillation. TIA: transient ischemic attacks.

Echocardiography and carotid duplex parameters

Regarding baseline Echocardiography data; the mean LAD was (39.38 ± 5.1) mm; the mean EF was (59.7 ± 11.1) %, with (2.9%) had intra-arterial or ventricular thrombus, and (4.4%) had cardiomyopathy.

Regarding baseline Carotid Duplex data; (5.2%) had Lt-sided significant stenosis, and (4.2%) had Rt-sided significant stenosis.

Follow up data:

Regarding Follow up clinical outcomes: (8%) had chest complications, (1.7%) had abdomen complications (hematemesis), (2.7%) had cardiac complications, and (3.7%) had UTI complication.

Comparative studies:

The 300 AIS patients were classified according to (Early Neurological Deterioration) END outcomes into 2independent groups:

- **END group (50 patients)*based on NIHSS change ≥ 2 from baseline.**
- **No END group (250 patients)**

Table 2: Follow up clinical data among 300 AIS patients:

Variables	Mean ± SD
SBP-1 (mmHg)	155.78± 23.1
SBP-2	144.16± 20.1
SBP-3	136.5± 23.62
DBP-3	80.88± 12.7
GCS-1	14.72±0.94
GCS-2	14.62± 1.2
GCS-3	14.3± 2
NIHSS-1	9.15± 4.65
NIHSS-2	8.28± 4.73
NIHSS-3	7.74± 5.1
MRS (3-months)	2.12± 1.87
Variables	Frequency (%)
Chest complications	24 (8%)
Abdomen complications (hematemesis)	5 (1.7%)
Cardiac complications	8 (2.7%)
UTI	11 (3.7%)
DVT	0 (0%)

UTI: Urinary tract infection. DVT: deep venous thrombosis. SBP: systolic blood pressure (day- 1, 2, 3). DBP: diastolic blood pressure (day-2, 3). GCS: Glasgow coma scale (day-1, 2, 3). NIHSS: National institute of health stroke scale (day-1, 2, 3). MRS: Modified Rankin Scale

Comparative studies are shown in the following tables and figures;

Baseline data:

Comparative study between the 2 groups revealed non-significant difference as regards all basic demographic data ($p > 0.05$).

Table 3: Comparison between the 2 groups as regards basic demographic data using Mann-Whitney's U and Chi square tests:

Variable		END group (50)	No END group (250)	Mann-Whitney's U test
		Median (IQR)	Median (IQR)	P value
Age (years)		62.5 (57 – 66)	62 (55 – 70)	= 0.896
Variable		END group (50)	No END group (250)	Chi square test
				P value
Gender	Female Male	19 (38%) 31 (62%)	81 (32.4%) 169 (67.6%)	= 0.4440
Handiness	Lt Rt	3 (6%) 47 (94%)	9 (3.9%) 241 (96.4%)	= 0.4300
Smokers	+ve	8 (16%)	40 (16%)	= 1.000
Ex-smokers	+ve	1 (2%)	14 (5.6%)	= 0.2871

Follow up data:

Comparative study between the 2 groups revealed; highly significant decrease in day-1, day-2 and day-3 SBP and DBP, in END group; compared to non-END group; with highly significant statistical difference ($p < 0.05$), highly significant increase in chest, abdomen, cardiac complications, in END group; compared to non-END group; with highly significant statistical difference ($p < 0.05$) and non-significant difference as regards UTI and DVT complications ($p > 0.05$).

Table 4: Comparison between the 2 groups as regards Follow up clinical outcomes:

Variable	END group (50)	No END group (250)	Mann-Whitney's U test
	Median (IQR)	Median (IQR)	P value
SBP-1	140(130-160)	155(135-170)	0.0079**
DBP-1	90(80-90)	90(80-100)	0.0083**
SBP-2	130 (120 – 150)	150 (130 – 160)	= 0.0036**
DBP-2	80 (70 – 90)	80 (80 – 90)	= 0.0036**
SBP-3	130 (120 – 150)	140 (130 – 150)	= 0.0078**
DBP-3	80 (70 – 90)	80 (80 – 90)	= 0.044*
Variable	END group (50)	No END group (250)	Chi square test
			P value
Chest complications	13 (26%)	11 (4.4%)	< 0.0001**
Hematemesis	3 (6%)	2 (0.8%)	= 0.0089**
Cardiac complications	6 (12%)	2 (0.8%)	< 0.0001**
UTI complication	3 (6%)	8 (3.2%)	= 0.3370
DVT complication	0 (0%)	0 (0%)	= 1.000
Seizure	1 (2%)	0 (0%)	= 0.025*

*significant. **highly significant. UTI: Urinary tract infection. DVT: deep venous thrombosis. SBP: systolic blood pressure (day-1, 2, 3). DBP: diastolic blood pressure (day-1, 2, 3).

Comparative study between the 2 groups revealed; highly significant increase in day-1, 2 and 3 NIHSS scores, in END group; compared to non-END group; with highly significant statistical difference ($p < 0.05$).

Comparative study between the 2 groups revealed; highly significant increase in NIHSS change, in END group; compared to non-END group; with highly significant statistical difference ($p < 0.01$).

Comparative study between the 2 groups revealed; highly significant increase in MRS (3-months), in END group; compared to non-END group; with highly significant statistical difference ($p < 0.01$).

Table 5: Comparison between the 2 groups as regards Follow up neurological outcomes using Mann-Whitney's U test:

Variable		END group (50)	No END group (250)	Mann-Whitney's U test
		Median (IQR)	Median (IQR)	P value
Neurological Outcomes	GCS-1	15 (13-15)	15 (15-15)	0.000013**
	GCS-2	15 (13 – 15)	15 (15 – 15)	0.000013**
	GCS-3	13 (11 – 15)	15 (15 – 15)	< 0.0001**
	NIHSS-1	12.5 (9-15)	7.5 (4-11)	<0.0001**
	NIHSS-2	12.5 (9 – 15)	7 (4 – 10)	< 0.0001**
	NIHSS-3	14 (10 – 17)	6 (3 – 9)	< 0.0001**
	NIHSS change	3 (3 – 4)	-2 (-4 – -1)	< 0.0001**
	MRS (3-m)	4 (2 – 6)	2 (0 – 3)	< 0.0001**

GCS: Glasgow coma scale (day-1, 2, 3). NIHSS: National institute of health stroke scale (day-1, 2, 3). MRS: Modified Rankin Scale.

Table 6: Follow up imaging for patients with END

Variables	Frequency (%)
Hemorrhagic transformation	14 (28%)
Brain edema	9 (18%)
New stroke (Progression)	4 (8%)

Logistic regression analysis shows that; after applying (Backward method) and entering some predictor variables; the increase in age, HTN duration, NIHSS-1, DWSI, MCA, and Hemorrhagic transformation; had an independent effect on increasing the probability of END occurrence; with significant statistical difference ($p < 0.05$ respectively.

Table 7: Logistic regression model for the Factors affecting END occurrence:

Predictor Factor	Coefficient	Std. Error	P value
(Constant)			
Age	0.23907	0.10120	0.018*
HTN duration	0.72233	0.27350	0.0083**
NIHSS-b	1.00310	0.35256	0.0044**
RBS	0.019366	0.011106	0.0812
DWSI	11.62603	4.75236	0.0144*
MCA	6.65628	2.44499	0.0065**
Hemorrhagic transformation	11.56131	4.00902	0.0039**
Brainedema	7.50333	3.93371	0.0565

Excluded from the model if (p value > 0.1). NIHSS: National institute of health stroke scale (baseline). HTN: hypertension. DWSI: Deep watershed infarction. MCA: Middle cerebral artery.

DISCUSSION:

Acute ischemic stroke (AIS) is a common and frequently occurring disease among middle aged and elderly population with severe disability and high mortality rate. In this prospective comparative study we evaluated early neurological deterioration (END) in AIS, its potential predictors, causes and assessed the relationship between END after AIS and the patient's functional level at three months post stroke including case fatality.

In our study, the incidence rate of END after AIS was 16.7% (50 patients). This was consistent with the overall incidence of END (10-40%) in different studies[17]. Similar

results reported by lee and lee, 2017 (19%) who studied 516 patients. END was defined as a ≥ 2 point increase in the National Institutes of Health Stroke Scale (NIHSS) during the first 72 hours of hospitalization.

Bathia et al studied 114 patients with acute ischemic stroke and founded that 25 patients had END (21.9%). END was defined as a ≥ 3 point increase in the National Institutes of Health Stroke Scale (NIHSS) during the first 72 hours of hospitalization [(6)].

Percentage of END was variable between studies: Chu Huang et al studied 163 patient with anterior circulation stroke and reported 43 patients (26.4) with END[(4)], Hong Geng et al. reported 32% of 1064 patients had END (definition of END was the same of our study)[**(18)**], Zhang et al reported 23.6% of studied patients had END (extend the duration of END to 7 days)[**(5)**].

The difference in prevalence of END can be attributed to the differences in the time scale of assessments after acute stroke, diagnostic criteria used for END, and the case mix of stroke patients. Also, the implementation of stroke units specialized in treatment of AIS and the advance of new stroke management strategies (intravenous thrombolysis and mechanical thrombi-ctomy) could account for the wide variations reported in the studies.

Regarding baseline data, there was no significant difference as regards all basic demographic data (age, sex, smoking, DM, HTN, hyperlipidemia, collagen disease, previous stroke and TIA) between patients experienced END and others ($p > 0.05$) which came in agreement with Huang *et al.*[(4)], and Siegler *et al.*[(19)] (except for age), Kwan and Hand,[(8)] (except for smoking) and Davalos *et al.*[(10)] and disagreement with Geng *et al.*[(18)] for DM and HTN.

In our study, duration of DM was significantly higher in patients with END and the correlative study showed a highly significant positive correlation of HBA1c and RBS at admission with END ($p < 0.05$) which came in agreement with Bathia *et al.*[(6)] and Zhang *et al.*[(5)].

In our study, IHD was significantly higher in END group compared to non-END group, with significant statistical difference ($p < 0.05$) which came in agreement with Sumer *et al.*[(2)], and Zhang *et al.*[(5)] in (IV-rTPA group) and disagreement with Haung *et al.*[(4)], and Geng *et al.*[(18)].

IHD is often associated with a higher prevalence of severe extracranial and intracranial atherosclerotic disease which lead to further extension of penumbral tissue volumes and level and hence the infarction due to poor collaterals.

In our study, there was significant increase in AF in END group (p=0.048) which came in agreement with Kwan and Hand,[(8)] (33% vs 16%, p=0.039) and disagreement with Haung *et al.*[(4)]. In Echo parameters, there was no difference between END and non-END in LAD and EF ($p > 0.05$), but there was significant increase in cardiomyopathy in END group ($p = 0.019$) which came in disagreement with Aoki in 2010[(20)].

Aoki *et al.*[(20)] reported that non-significant statistical difference as regard cardiac disease, which included myocardial infarction, cardiac valve disease, cardio-myopathies, and congestive heart failure.

In our study, There was highly significant decrease in baseline SBP and DBP, in END group compared to non-END group with highly significant statistical difference ($p < 0.0079$, 0.0085 respectively) which came in agreement with Bathia *et al.* [(6)] and Kwan and Hand, [(8)] founded that systolic and diastolic blood pressure was lower in patients with END in comparison to non-END with no significant statistical difference disagreement with Zhang *et al.*[(5)] and Miyamoto *et al.* [(21)].

In our study, there was significant increase in uric acid in END group; compared to non-END group; with significant statistical difference (p = 0.033) which came in agreement with[4&22].

In our study, there was negative relationship between hemoglobin level and END. Which means that high serum hemoglobin is protective against END? Anemia was reported by several studies that it had strong association with poor outcome and mortality after acute ischemic stroke [23&24].

In our study, TLC was higher in END group but with no statistical difference which came in disagreement with Zhang *et al.*[5], Bathia *et al.*[6] and Kwan and Hand,[8].

There was no significant difference as regards all the remaining laboratory variables (p > 0.05) which came in agreement with Lord *et al.*[25].

In our study, there was no significant difference as regards Carotid Duplex data (p > 0.05) which came in agreement with Lord *et al.*[25].

The possible explanation that extracranial arterial stenosis (EAS) had little effect on END in comparison to intracranial stenosis is that brain region affected by IAS is more likely to have a limited collateral blood flow whereas in EAS, collaterals across the circle of Willis maybe preserved allowing the perfusion into a brain region distal to the occlusion.

This came in disagreement with Siegler *et al.*[19] who reported that The presence of carotid stenosis was found to be a risk factor for ND (unadjusted OR 1.80; 95% CI 1.10-3.10; P = .0298), and interestingly, this relationship remained independent regardless of the side of the stenosis in relation to the side of the cerebral infarct.

Our results disagree with Weimar *et al.*[26] reported that patients with END had posterior circulation stenosis more than patients without END with highly significant statistical difference (VA (P=0.002), BA (P<0.001) and PCA (P<0.001) and anterior cerebral artery (p= 0.007).

Regarding follow up data: there was highly significant decrease in day-1,2 and day-3 SBP and DBPs, in END group; compared to non-END group; with highly significant statistical difference (p < 0.05 respectively) which came in agreement with Kang in 2017 **[27]**.

The role of BP becomes particularly important whenever a large volume of hypo perfused but still viable tissue, susceptible to changes in systemic BP is present. Indeed, in this case, any drop in BP cannot be compensated for by auto regulatory mechanisms, which can in turn lead to tissue infarction **[28].** When the changes in systolic BP during the first day were taken into account in a logistic regression, patients with a decrease in systolic BP 20 mm Hg showed significantly increased odds of END **[29].**

In our study, systemic complication (pneumonia, hematemesis, myocardial infarction) was more prevalent in END group; compared to non-END group; with highly significant statistical difference (p < 0.05 respectively) which came in disagreement with Kwan in 2006 Kwan and Hand,[8].

In our study, 17 patients with END (34%) had systemic complication. This was higher than reported by other studies**.** Siegler *et al.* [19] (using the same definition for END) reported 35 out of 323 patients had systemic complication (11%). Lord *et al.* [25] reported 5 out of 98 patients had systemic complication (5%).

In our sample 29.7% had territorial infarction (large stroke) and 37.8% (108 patients) had intracranial arterial stenosis and 22.4% (64 patients) had MCA disease. This means that our sample had large number of severe stroke which make them

more prone for complication like aspiration pneumonia.

There was no significant difference as regards UTI and DVT complications ($p > 0.05$) which came in agreement Kwan and Hand, [8].

In our study, we assessed neurological complication in 40 patients (80%) of END group. 27 (67.5%) of them had abnormality in follow up imaging (14 patients had hemorrhagic transformation (35%), 9 patients had malignant brain edema (22.5%) and 4 patients had stroke progression (10%).

Siegler *et al.* [19] studied causes of END in 323 patients within the first day after stroke and founded that 86 patients had progressive stroke (26.6%), 23 patients had hemorrhage (7%), 10 patients had new stroke (3%) and 34 patient had brain edema (10.5%). Among 231 patients with END within the first day after acute ischemic stroke studied by Davalos *et al.* [10] 61 patients had hemorrhage (27%) and 48 patients had malignant brain edema (21%). Weimar *et al.*[26] studied 256 patients with END and founded that 33.6% had progressive stroke, 27.3% had brain edema and 10.5 % had hemorrhage.

As regard GCSs, there was highly significant decrease in day-1,2 and 3 GCSs, in END group; compared to non-END group; with highly significant statistical difference ($p < 0.01$ respectively) which came in agreement with Lord in 2015[25].

Lord *et al.* [25] Reported that Compared with those with No END at any point during the study, patients with END were more likely to have lower GCS (END versus No END; 13 versus 15; $P<0.001$).

Logistic regression analysis showed that; after applying (Backward method) and entering some predictor variables; the increase in age, HTN duration, NIHSS-b, DWSI, MCA, and Hemorrhagic transformation; had an independent effect on increasing the probability of END occurrence; with significant statistical difference ($p < 0.05$ respectively) which came in agreement with Nacu *et al.*[17] as regard NIHSS-b and MCA occlusion, Bathia *et al.*[6] and Geng *et al.*[18] as regard baseline NIHSS and diabetes. Siegler *et al.*[19] as regard age baseline NIHSS. Weimar *et al.*[26] as regard MCA occlusion and diabetes.

Conclusion:

- Early neurological deterioration is a frequent complication after acute stroke, with a poor short-term prognosis. This study provides that hyperglycemia, hyperuricemia and cardiac disease (IHD and AF) may increase the risk of END.
- Careful attention should be paid for patients with severe stroke and those with intracranial arterial disease to prevent or minimize the risk of complication like aspiration pneumonia and hemorrhagic transformation.

Recommendation:

- Effective treatment strategies are urgently needed to reduce the occurrence of END and its impact on outcome.
- Identifying all people at risk and control their risk factor is very important to avoid hazards and complications of END.
- This study is small size (300 patients) one center study, so: large multicenter study with long term follow up are needed to explore causes and predictors of early and late neurological deterioration and their effect on short and long term outcome.

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تدهور الجهاز العصبي المبكر بعد السكتة الدماغية الحادة-المؤشرات والأسباب المحتملة وعلاقتها بزيادة حجم الجلطة الدماغية

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مقدمــة: تفاقم السكتة الدماغية الحادة في وقت مبكر في مسارها (في غضون يومين أو ثلاثة) أمر شائع وله عواقب على المدى القريب والبعيد. معدل انتشار التدهورات العصبية المبكرة متنوع بين الدراسات المختلفة فهو يتراوح ما بين ثلاثة عشر إلى ثمانية وثلاثين بالمأة.

هدف العمل:

١. تحديد مدى انتشار وشدة وتوقيت التدهورات العصبية المبكرة بعد السكتة الدماغية الحادة.
٢. تحديد العوامل والأسباب المحتملة للتدهورات العصبية المبكرة بعد السكتة الدماغية الحادة.
٣. تحديد تأثير التدهورات العصبية المبكرة بعد السكتة الدماغية الحادة على المستوى الوظيفي للمريض بعد ثلاثة أشهر.

المرضي وطريقة البحث :خضع المرضى الذين شملتهم الدراسة إلى:

١- التاريخ المرضي والفحص العام والعصبي لتحديد العوامل الخطرة التي تؤدى إلى السكتة الدماغية مثل ارتفاع ضغط الدم والسكر والرفة الأذينية وجلطة القلب.
٢- الاختبارات المعملية شملت قياس نسبة السكر بالدم وكذلك قياس نسبة الدهون بالدم
٣- عمل رسم القلب لاكتشاف الرفة الأذينية وعمل موجات صوتية علي القلب للكشف عن مصدر السدة الوعائية ومعرفة أثار ارتفاع ضغط الدم على القلب.
٤- عمل أشعة دوبلكس للكشف عن أمراض الشريان السباتي خارج الجمجمة.
٥- عمل رنين مغناطيسي على المخ عمل وعلى الأوعية الدموية المخية لتحديد ضيق الشرايين المخية.
٦- تم فحص جميع المرضى على مقياس السكتة الدماغية لدى المعهد الوطني للصحة ومقياس جلاسكو في اليوم الأول والثالث والخامس.
٧- خضع جميع المرضى الذين يعانون من التدهورات العصبية المبكرة لإعادة الفحص بالرنين المغناطيسي أو الأشعة المقطعية.
٨- جميع المرضى تم متابعتهم بعد ثلاثة أشهر بعمل مقياس رانكن المعدل.

النتائــج :من بين ثلاثمائة مريض شملتهم الدراسة, وجد التدهور المبكر في خمسين مريض (١٦.٧%). وأظهرت الدراسة وجود علاقة قوية بين التدهورات العصبية المبكرة بعد السكتة الدماغية الحادة وكلا من:

- ارتفاع نسبة السكر بالدم مع بداية السكتة الدماغية وكذلك الإصابة بداء السكري لمدة طويلة.
- الإصابة بقصور الشرايين التاجية والرفة الأذينية وارتخاء عضلة القلب.
- ارتفاع نسبة أملاح النقرس بالدم.
- انخفاض ضغط الدم في بداية السكتة الدماغية وكذلك في الأيام التالية.
- كبر حجم السكتة الدماغية من البداية وكذلك إصابة الشريان الأوسط من المخ.

كما أوضحت الدراسة أن التدهورات العصبية المبكرة بعد السكتة الدماغية الحادة يؤدي ارتفاع معدل الوفاة ونسبة العجز بعد السكتة الدماغية.

خلاصة البحث: التدهورات العصبية المبكرة أمر شائع وله عواقب علي المدي القريب. أوضحت الدراسة أن الاصابة بالسكري وارتفاع حمض اليوريك بالدم والرفة الاذنية وقصور الشرايين التاجية يؤدي الي زيادة معدل التدهورات العصبية المبكرة.