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Quality indicators and patients' characteristics in relation to early outcome of Kasr-al-ainy stroke unit

Maha Atef Zaki¹, Ahmed Mohamed Abdelalim¹, Husam Salah Mourad¹, Abdallah Adel Saad¹ and Amr Mohamed Fouad^{1*}¹

Abstract

Background A wide variety of factors have been reported to influence stroke prognosis. Quality indicators serve as standards of care. The aim of this study is to assess the clinical and demographic characteristics of patients with stroke and to measure quality indicators in relation to stroke early outcome. We analyzed a prospective hospital-based data. All patients with acute stroke admitted to Kasr-al-ainy stroke unit over a 1-year period were included. Clinical variables and quality indicators were studied in relation to early outcome which was measured by both disability at discharge assessed by modified Rankin score (mRS) and in-hospital death.

Results We studied 242 patients with acute stroke, 145 patients (59.9%) had poor outcome at discharge [mRS 3–6]. There were 36 (14.9%) in-hospital deaths. High mRS and National institute of health stroke scale (NIHSS) at stroke onset, low Gugging swallowing scale (GUSS) score and hypertension (HTN) were independent predictors for more disability on discharge. Chest infection, bed sores, high mRS at stroke onset and hemorrhagic transformation were independent predictors for in-hospital mortality. Receiving recombinant tissue plasminogen activator (rtPA) showed significant association with good outcome. Mean door to needle time (DNT) was 50.6 min. Low GUSS score was associated with increased risk of chest infection.

Conclusions Stroke severity, potentially modifiable risk factors and complications are associated with an increased risk of poor early outcome. Dysphagia screening using GUSS scale can predict patients with higher risk of aspiration pneumonia after stroke. DNT needs to be improved.

Keywords Acute stroke, Patients' characteristics, Early outcome, Predictors, Quality indicators

Background

After stroke, Clinicians are often asked to predict outcome by the patient, family, other healthcare workers, and insurance providers. A wide variety of factors have been reported to influence stroke prognosis, including age, stroke severity, stroke mechanism, infarct location,

Amr Mohamed Fouad

amro.fouad@kasralainy.edu.eg

¹ Neurology Department, Faculty of Medicine, Cairo University, 17 Lewaa Farouk El Sawy St, El Haram, Giza, Egypt comorbid conditions, clinical findings, and related complications. In addition, interventions such as thrombolysis, stroke unit care, can play a major role in the outcome of stroke [1].

Clinical guidelines were written to promote diagnostic or therapeutic interventions applicable to the majority of patients in most circumstances. However, the use of guideline recommendations for individual patients has traditionally been left to the discretion of individual clinicians [2].

A recognized approach to assist the translation of research evidence into clinical practice is to monitor the key performance indicators (KPIs) which are standards of



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^{*}Correspondence:

care implying that health care professionals are providing inadequate care if eligible patients do not receive that standard of care [2].

The mRS is a clinician-reported measure of global disability after stroke and used as a primary end point in randomized clinical trials (RCTs) of stroke treatments. Extensive evidence on the validity, reliability and sensitivity of the mRS exists across literature [3].

Determining an individual patient's risk of mortality at admission could aid clinical care by identifying those at high risk for poor outcomes who may require more intensive resources [4].

The aims of this study are measuring quality indicators together with identification of characteristics of stroke patients and the relation of both to early outcome of stroke units.

Methods

This was an observational prospective cohort hospitalbased study involving all acute stroke patients (242) of both sexes aging above 12 years who were admitted to the stroke unit in Kasr-al-ainy hospital through 1 year from 1st of October 2019 to 30th of September 2020.

All Patients were subjected to medical history (including detailed history of possible stroke risk factors), neurological and neurovascular examination including dysphagia screening using GUSS scale [5], computed tomography (CT) brain using 16-slice CT scanner (Siemens, Somatom go.Top, Germany) and \or Magnetic resonance imaging (MRI) brain using MRI scanner (Philips, achieva 1.5T, Netherland), laboratory investigations including workup for stroke risk factors, $Electrocardiogram(ECG) \pm Echocardiography$ using ultrasound machine (Philips, HDI 5000, USA) ± Carotid and vertebrobasilar duplex ± transcranial duplex using ultrasound machine (philips, IU22, USA) ± CT angiography using the above mentioned CT scanner or Magnetic resonance angiography (MRA) using the above mentioned MRI scanner.

Clinical evaluation: we used Oxford community stroke project classification of clinically identifiable cerebral stroke subtypes to classify our patients [6]. Regarding stroke etiology: we used TOAST (trial of ORG 10172 in acute stroke treatment) classification [7] to classify ischemic patients. Regarding hemorrhagic stroke, it includes parenchymal hemorrhage, subarachnoid hemorrhage, intraventricular hemorrhage. Other etiological types include transient ischemic attack, sinus thrombosis and venous infarctions. NIHSS [8] and mRS [9] were used for assessment of stroke severity.

We have used a previously designed quality indicators to measure performance of management of stroke patients. We selected these indicators from last version of Get With The Guidelines (GWTG) Stroke, a performance improvement program for hospitals that use a stroke registry to support its aims [10].

Both mRS on discharge and in-hospital mortality were used to assess early stroke outcome. We grouped patients regarding mRS on discharge into 2 categories: good outcome: mRS 0–2 and poor outcome: mRS 3–6.

The association of demographics, clinical characteristics, complications, management and compliance to quality indicators with early outcome was assessed.

Data were coded and entered using the statistical package for the Social Sciences (SPSS) version 26. Data were summarized using mean, standard deviation, median, minimum and maximum in quantitative data and using frequency (count) and relative frequency (percentage) for categorical data. Comparisons between quantitative variables were done using the non-parametric Kruskal– Wallis and Mann–Whitney tests for non-normally distributed date and t test for normally distributed data. For comparing categorical data, Chi square (χ^2) test was performed. Exact test was used instead when the expected frequency is less than 5. Multivariate logistic regression was done to detect independent predictors of mortality and bad mRS on discharge. *P* values less than 0.05 were considered as statistically significant.

The institutional review board of faculty of medicine, Cairo University approved this study. All methods were carried out in accordance with relevant guidelines and Declaration of Helsinki.

Results

Two hundred and forty-two acute stroke patients were enrolled in our study; out of them, 186 had ischemic stroke. Age of patients ranges from 25 to 87 years with mean of 59.3 ± 13.1 , 155 (64%) were males, while 87 (36%) were females. The prevalence of stroke risk factors, stroke subtypes, etiology and complications are illustrated in Table 1.

Regarding stroke severity on admission, mRS ranged from 0 to 5 with mean of 3.5 ± 1 , while NIHSS ranged from zero to 31 with a median of 10. Thirteen (5.4%) patients had NIHSS zero, 33 patients (13.6%) had minor stroke (NIHSS 1–4), 166 (68.6%) had moderate stroke (NIHSS 5–15) and 30 patients (12.4%) had severe stroke (NIHSS > 15).

Patients' hospital stays ranged from 1 day to 105 days, with a median of 8 days.

Regarding Quality indicators, out of 77 patients who arrived at the hospital within 4.5 h, 75 received rtPA, while two patients did not; one received low molecular weight heparin before presentation to us, while the other was subjected to mechanical thrombectomy directly. GUSS score was done for all patients on admission

Table 1 Descriptive results of our cohort

Descriptive results	Itoms	n (%)	
	items	H (70)	
1) Clinical stroke	TAC I	51 (21.1)	
subtypes (Oxford clas- sification)	TAC H	1 (0.4)	
Sincationy	PAC I	53 (21.9)	
	PAC H	23 (9.5)	
	POCI	19 (7.9)	
	POC H	2 (0.8)	
	LAC I	67 (27.7)	
	LAC H	14 (5.8)	
	TIA	3 (1.2)	
	Others	9 (3.8)	
2) Risk factors	HTN	167 (69)	
	Diabetes	85 (35.1)	
	Dyslipidaemia	111 (45.9)	
	Obesity	65 (26.9)	
	Overweight	98 (40.5)	
	Current smoker	85 (35.1)	
	Hold smoking	25 (10.3)	
	Transient ischemic attack	30 (12.4)	
	Atrial fibrillation	47 (19.4)	
	Valve replacement	7 (2.9)	
	Congestive heart failure	30 (12.4)	
	Substance abuse	6 (2.5)	
	Oral contraceptive pills (OCP)	3 (3.45) of all female patients	
3) Stroke subtype	l arge artery	51 (21.1)	
s, science subtype	Cardio-embolic	42 (174)	
	Small artery	51 (21 1)	
	Other determined	5 (2 1)	
	Non-determined	37 (153)	
	TIA	3 (1 2)	
	Haemorrhage including SAH	46 (19 1)	
	Sinus thrombosis	5 (2 1)	
	Venous infarction	2 (0.8)	
Complications	Chest infection	2 (0.0) //8 (10.8)	
complications		2/ (0 0)	
	Sonsis	24 (9.9) 18 (7.4)	
	Bod soros	16 (6.6)	
		TO (0.0)	
	DVI Pleadings other than corobral	J(2.1)	
		+(1./) 2 (0.0)	
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		レビ (1.ツ) 4 (1.フ)	
	FILS	4 (1./)	
	Allergy from rtpa (of total 75)	I (I.3)	

TACI: total anterior circulation infarction; HTN: hypertension; TACH: total anterior circulation hemorrhage; PACI: partial anterior circulation infarction; PACH: partial anterior circulation hemorrhage; POCI: posterior circulation infarction; POCH: posterior circulation hemorrhage; LACI: lacunar infarction; LACH: lacunar hemorrhage; TIA: transient ischemic attack

Table 2 Quality indicators

	n (%)
Proportion of all ischemic stroke patients who receive treatment with rtPA	75/186 (40.3)
IV rt-PA arrive by 2 h, treat by 3 h	28/28 (100)
IV rt-PA arrive by 3.5 h, treat by 4.5 h	72/72 (100)
IV rt-PA arrive by 3 h, treat by 3 h	31/57 (54.3)
Onset to door (min)	
<60	5 (6.66)
61–120	23 (30.66)
121–180	29 (38.66)
181–240	18 (24)
241–270	0 (0)
Door to needle(min)≤60	75/75
Door to needle(min)≤45	22/75
Onset to needle (min)	
≤60	0 (0)
61–120	5 (6.7)
121–180	26 (34.6)
181–240	30 (40)
241–270	14 (18.7)
mRs at discharge	
0	35 (14.4)
1	11 (4.5)
2	50 (20.6)
3	29 (11.9)
4	80 (33)
5	1 (0.41)
6	36 (14.9)

rtPA: recombinant tissue plasminogen activator, min: minute, mRs: modified Rankin scale

(n = 242), ranging 0 to 20 with a mean of 14.62 ± 7.1 . It was done also for all patients on discharge apart from those who died at hospital (n=206), ranging from 2 to 20 with a mean of 18.69 ± 3.8 . Patients complicated with chest infection had significantly lower GUSS scores at stroke onset (median of 6, range from 0 to 20) compared to those not complicated with chest infection (median of 20, range from 0 to 20), P < 0.001. The mean onset-todoor time ranged from 60 to 230 min, with a median of 150 and a mean of 151.93 ± 45.43 min, while the mean door-to-needle time ranged from 20 to 60 min, with a median of 50 and a mean of 50.6 ± 9.04 min. Regarding onset-to-needle time, it ranged from 110 to 270 min, with a median of 210 and a mean of 202.5 ± 42.4 min. The mRS at discharge (including patients who died in hospital) ranged from 0 to 6 with a median of 3. Out of 242 patients, 36 died in hospital (14.9%) due to stroke and its complications. Other quality indicators are summarized in Table 2.

On comparing outcome groups depending on mRS on discharge; the presence of HTN, stroke complicated by chest infection, bed sores, or sepsis, posterior circulation infarct stroke, cardioembolic stroke, large artery stroke, not receiving rtPA, older age, higher NIHSS, and lower GUSS score on admission are significantly associated with bad mRS on discharge, as illustrated in Tables 3 and 4.

Results of multivariate logistic regression analysis for prediction of having bad mRS on discharge (utilizing significant variables in Tables 3 and 4) showed a significant regression, P < 0.001 and R2 = 0.38. The risk of having bad mRS on discharge is more than the double when the patient is hypertensive (OR = 2.6, CI 1.24–5.44, P value = 0.01). Similar results were found regarding lower GUSS on admission and higher both mRS on and NIHSS on admission (Table 5).

On comparing between patients discharged alive and dead groups: older age, posterior circulation infarction (POCI) stroke, cardio embolic stroke, large artery stroke, stroke complicated by hemorrhagic transformation, chest infection, bed sores or sepsis, admission in ICU, higher NIHSS and mRS and lower GUSS scores on admission are associated with higher in-hospital mortality, while small artery infarction was associated with lower mortality as shown in Tables 3 and 4.

Results of multivariate logistic regression analysis for prediction of in-hospital mortality (utilizing significant variables in Tables 3 and 4) showed a significant regression, P < 0.001 and R2 = 0.34. The risk of in-hospital mortality is more than seven times higher when the patient developed hemorrhagic transformation (OR=7.39, CI 1.97–27.7, P value=0.003). Similar results were found regarding chest infection, bed sores, mRS on admission (Table 5).

Discussion

The current study stated that advanced age correlates significantly with poor mRS on discharge and in-hospital mortality, but not an independent predictor of either in multivariate analysis. These findings are consistent with other studies, which stated that the association between age and early stroke disability and in-hospital mortality may be better explained by the coexistence of other factors, such as the severity of neurological impairment on admission [11–13]. However, other studies have found that age is a predictor for disability and mortality [14–16]. The population of these studies is only of ischemic stroke.

In the present work, no statistically significant difference was found between males and females regarding mortality or disability. The existence of gender differences in stroke outcome is controversial in previous studies; some previous studies reported that females had worse functional outcome and more in-hospital mortality after stroke [14, 17, 18], while other studies have stated that male sex is associated with more in-hospital mortality [19, 20]. Some more studies argued that gender has no effect on stroke outcome [21–23].

The present results did not show any significant relation between any of the studied risk factors and outcome, except for hypertension which was associated with disability and found to be a predictor of it. This comes in agreement with previous studies [17, 24, 25]. Other studies have found that, in addition to hypertension, diabetes, atrial fibrillation, history of previous stroke, and ischemic heart disease are associated with an increased risk of poor outcome and mortality following stroke [14, 15, 26]. On the other hand, other studies have stated that the presence of common stroke risk factors was not associated with poor functional outcome or mortality [18, 27].

In our study, stroke initial clinical severity was significantly associated with disability and mortality; however, it was an independent predictor only for disability. Previous studies reported that stroke severity as measured by the NIHSS is an important factor determining outcome after stroke [1, 14] and predicting in-hospital mortality [17, 28]; these studies used NIHSS categorization in relation to mortality. The non-categorization of NIHSS in our study may explain why NIHSS is not a predictor for mortality.

The current work showed that large artery and cardioembolic strokes were associated with poorer mRS on discharge compared to other stroke subtypes; however, neither was an independent predictor for it. This agrees with previous studies [29, 30]. Other studies showed that they were associated with higher disability and in-hospital mortality [15, 26].

Our study showed that partial anterior circulation infarction (PACI) was associated with poorer mRS on discharge compared to other stroke subtypes, but was not an independent predictor for it. On the other hand, POCI was associated with significantly higher in-hospital mortality, while lacunar infarction (LACI) was associated with lower in-hospital mortality, but not an independent predictor for it. Previous studies had found similar results [12, 15, 28]. Other studies has reported no significant difference in outcome between anterior and posterior circulation strokes [22].

Medical and neurological complications occur after stroke and can affect outcome. Our study found that chest infection and bed sores were associated with poor outcome and were independent predictors for mortality. The same was found in previous studies [27, 28, 31]. On the other hand, another study has stated that poststroke complications, including bronchopneumonia, urinary tract infection, bedsore, deep venous thrombosis,

Good (mf8 ≤ 2) $n = 95$ Bad $n = 15$ PvalueAlive $n = 206$ Dead $n = 36$ PvalueRote (mf8 ≥ 2)Count (%)Count (%)Count (%)Count (%)Count (%)Count (%)Count (%)Risk factorsSacMale67 (69.1)88 (60.7)0.1813 (64.6)29 (60.8)0.16Penale30 (30.9)57 (39.9)73 (35.4)14 (58.9)0.16Diaberes31 (82.7)54 (32.7)0.3969 (85.1)16 (40.4)0.2Dyslipidemia38 (40.2)72 (42.7)0.1539 (85.1)16 (40.4)0.3Overwoight38 (39.2)60 (41.4)0.7314 (49.9)0.93347 (18.9)10 (22.8)0.11Th10 (0.3)20 (13.8)0.4224 (13.6)12 (28.9)12 (28.9)17 (28.9)17 (28.9)17 (28.9)Valvereplacement4 (4.1)3 (21.9)0.445 (2.9)11 (28.9)17 (28.9)16 (28.7)18 (41.9)18 (41.9) </th <th></th> <th colspan="3">mRs on discharge</th> <th colspan="3">Mortality</th>		mRs on discharge			Mortality			
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Obesity 26 (26.3) 39 (26.9) 0.98 56 (27.2) 9 (25) 0.78 Overweight 38 (39.2) 60 (41.4) 0.73 84 (40.8) 14 (38.9) 0.83 Arial fibrillation 14 (14.4) 33 (22.8) 0.11 37 (18.0) 10.77.8) 0.17 Valve replacement 4 (4.1) 3 (2.1) 0.44* 6 (2.9) 1 (2.8) 1* TA 10 (10.3) 20 (13.8) 0.42 28 (13.6) 2.6 (3.7) 18 (3.7) 0.43 1.3 0.59 74 (35.9) 11 (3.6) 0.53 Substance abuse 1 (1) 5 (3.4) 0.41* 5 (2.4) 1 (2.8) 1* Heart failure 15 (15.5) 15 (10.3) 0.24 25 (12.1) 5 (13.9) 0.78 Complications	Dyslipidemia	39 (40.2)	72 (49.7)	0.15	93 (45.1)	18 (50)	0.6	
Overwight 38 (39.2) 60 (41.4) 0.73 84 (40.8) 14 (38.9) 0.83 Atrial fibrillation 14 (14.4) 33 (22.8) 0.11 37 (18.0) 10 (27.8) 0.17 Valve replacement 44.1) 32 (2.8) 0.44* 6 (2.9) 12.8) 0.2 Old CVS 7.7.2 18 (12.4) 0.19 22 (10.7) 3 (8.3) 1* Smoker 36 (37.1) 49 (38.8) 0.59 74 (35.9) 11 (30.6) 0.53 Substance abuse 1 (1) 5 (3.4) 0.41* 5 (2.4) 1 (2.8) 1* OCP 3 (3.1) 0 (0) 0.66* 3 (1.5) 0 (0.0) 1.78 Complications 1 15 (10.5) 15 (10.3) C.000* 3 (1.1) 25 (69.4) -0.00 Bed sore 1 (1) 15 (10.3) 0.000* 4 (1.9) 12.8 0.001* UT 7 (7.2) 17 (1.7) 0.2 18 (8.7) 6 (16.7) 0.44 DVT 0 (0) 5 (3.4) <	Obesity	26 (26.8)	39 (26.9)	0.98	56 (27.2)	9 (25)	0.78	
Arial fibrillation 14 (14.4) 33 (22.8) 0.11 37 (18.0) 10 (27.8) 0.17 Valve replacement 4 (4.1) 3.2.1) 0.44* 6.2.9 1.2.8) 1* TA 10 (10.3) 20 (13.8) 0.42 28 (13.6) 2.5.6) 0.27* Old CVS 7 (7.2) 18 (12.4) 0.19 22 (10.7) 3 (8.3) 1* Smoker 36 (37.1) 49 (33.8) 0.59 74 (35.9) 11 (30.6) 0.53 Substance abuse 1 (1) 5 (13.9) 0.00 16* 0.000 1* Hear failure 15 (15.5) 15 (10.3) 0.24 25 (12.1) 5 (13.9) 0.78 Complications	Overweight	38 (39.2)	60 (41.4)	0.73	84 (40.8)	14 (38.9)	0.83	
Valve replacement 4 (4.1) 3 (2.1) 0.44* 6 (2.9) 1 (2.8) 1* TA 10 (10.3) 20 (13.8) 0.42 28 (15.6) 2.6 (5.6) 0.27* Old CVS 7 (7.2) 18 (12.4) 0.19 22 (10.7) 3 (8.3) 1* Smoker 3 (3.1) 0 (0) 0.65* 3 (1.5) 0 (0) 13 (3.5) 0 (0) 1* OCP 3 (3.1) 0 (0) 0.66* 3 (1.5) 0 (0) 1* Heart failure 15 (15.5) 15 (15.3) 0.24 25 (12.1) 5 (16.7) 0.78 Complications 1 15 (10.3) 0.004* 4 (1.9) 12 (3.3) <0.001*	Atrial fibrillation	14 (14.4)	33 (22.8)	0.11	37 (18.0)	10 (27.8)	0.17	
TA 10 (10.3) 20 (13.8) 0.42 28 (13.6) 2 (5.6) 0.27* Old CVS 7 (7.2) 18 (12.4) 0.19 22 (10.7) 3 (8.3) 1* Smoker 36 (37.1) 49 (33.8) 0.59 74 (35.9) 11 (30.6) 0.53 Substance abuse 11 5 (3.4) 0.41* 5 (2.4) 1 (2.8) 1* OCP 3 (3.1) 0 (0) 0.06* 3 (1.5) 0 (0.0) 1* Heart failure 5 (5.2) 14 (9.7) 0.24 13 (6.3) 6 (16.7) 0.045 Complications - - - 0.001* 24 (1.9) 12 (3.3) <0.001*	Valve replacement	4 (4.1)	3 (2.1)	0.44*	6 (2.9)	1 (2.8)	1*	
Old CVS 7 (7.2) 18 (12.4) 0.19 2 (10.7) 3 (8.3) 1* Smoker 36 (3.7) 49 (3.3) 0.59 74 (35.9) 11 (30.6) 0.53 Substance abuse 1 (1) 5 (3.0) 0.41* 5 (2.4) 1 (2.8) 1* OCP 3 (3.1) 0 (0) 0.66* 3 (1.5) 0 (0.0) 1* Heart failure 15 (15.5) 15 (10.3) 0.24 25 (12.1) 5 (13.9) 0.68 Complications	TIA	10 (10.3)	20 (13.8)	0.42	28 (13.6)	2 (5.6)	0.27*	
Smoker 36 (37.1) 49 (33.8) 0.59 74 (35.9) 11 (30.6) 0.53 Substance abuse 1 (1) 5 (3.4) 0.41* 5 (2.4) 1 (2.8) 1* OCP 3 (3.1) 0 (0) 0.66* 3 (1.5) 0 (0.0) 1* Heart failure 15 (15.5) 15 (10.3) 0.24 25 (2.1) 5 (13.9) 0.78 Complications	Old CVS	7 (7.2)	18 (12.4)	0.19	22 (10.7)	3 (8.3)	1*	
Substance abuse 1 (1) 5 (3.4) 0.41* 5 (2.4) 1 (2.8) 1* OCP 3 (3.1) 0 (0) 0.06* 3 (1.5) 0 (0.0) 1* Heart failure 15 (15.5) 15 (10.3) 0.24 25 (12.1) 5 (13.9) 0.78 Complications 4 (4.1) 44 (30.3) <0.001*	Smoker	36 (37.1)	49 (33.8)	0.59	74 (35.9)	11 (30.6)	0.53	
OCP 3 (3.1) 0 (0) 0.06* 3 (1.5) 0 (00) 1* Heart failure 15 (15.5) 15 (10.3) 0.24 25 (12.1) 5 (13.9) 0.78 Complications	Substance abuse	1 (1)	5 (3.4)	0.41*	5 (2.4)	1 (2.8)	1*	
Heart Failure 15 (15.5) 15 (10.3) 0.24 25 (12.1) 5 (13.9) 0.78 Complications	OCP	3 (3.1)	0 (0)	0.06*	3 (1.5)	0 (0.0)	1*	
Complications Hemorrhagic transformation 5 (5.2) 14 (9.7) 0.2 13 (6.3) 6 (16.7) 0.04s Bed sores 1 (1) 44 (30.3) < 0.001*	Heart failure	15 (15.5)	15 (10.3)	0.24	25 (12.1)	5 (13.9)	0.78	
Hemorrhagic transformation 5 (5.2) 14 (9.7) 0.2 13 (6.3) 6 (16.7) 0.04s Chest infection 4 (4.1) 44 (30.3) < 0.001*	Complications							
Chest infection 4 (4.1) 44 (30.3) < 0.001* 23 (11.2) 25 (69.4) < 0.001 Bed sores 1 (1) 15 (10.3) 0.004* 4 (1.9) 12 (33.3) < 0.001*	Hemorrhagic transformation	5 (5.2)	14 (9.7)	0.2	13 (6.3)	6 (16.7)	0.045	
Bed sores 1 (1) 15 (10.3) 0.004* 4 (1.9) 12 (33.3) <0.001* UTI 7 (7.2) 17 (11.7) 0.25 18 (8.7) 6 (16.7) 0.14 DVT 0 (0) 5 (3.4) 0.08* 4 (1.9) 1 (2.8) 0.55* Pulmonary embolism 0 (0) 2 (1.4) 0.52* 1 (0.5) 1 (2.8) 0.27* Fits 2 (2.1) 2 (1.4) 1* 3 (1.5) 1 (2.8) 0.47* Sepsis 0 (0) 18 (12.4) <0.001*	Chest infection	4 (4.1)	44 (30.3)	< 0.001*	23 (11.2)	25 (69.4)	< 0.001	
UTI 7 (7.2) 1 (11.7) 0.25 18 (8.7) 6 (16.7) 0.14 DVT 0 (0) 5 (3.4) 0.08* 4 (1.9) 1 (2.8) 0.55* Pulmonary embolism 0 (0) 2 (1.4) 0.52* 1 (0.5) 1 (2.8) 0.27* Fits 2 (2.1) 2 (1.4) 1* 3 (1.5) 1 (2.8) 0.47* Sepsis 0 (0) 18 (12.4) <0.001*	Bed sores	1 (1)	15 (10.3)	0.004*	4 (1.9)	12 (33.3)	< 0.001*	
DVT 0 (0) 5 (3.4) 0.08* 4 (1.9) 1 (2.8) 0.55* Pulmonary embolism 0 (0) 2 (1.4) 0.52* 1 (0.5) 1 (2.8) 0.27* Fits 2 (2.1) 2 (1.4) 1* 3 (1.5) 1 (2.8) 0.47* Sepsis 0 (0) 18 (12.4) <0.001*	UTI	7 (7.2)	17 (11.7)	0.25	18 (8.7)	6 (16.7)	0.14	
Pulmonary embolism 0 (0) 2 (1.4) 0.52* 1 (0.5) 1 (2.8) 0.27* Fits 2 (2.1) 2 (1.4) 1* 3 (1.5) 1 (2.8) 0.47* Sepsis 0 (0) 18 (12.4) -<0.001*	DVT	0 (0)	5 (3.4)	0.08*	4 (1.9)	1 (2.8)	0.55*	
Fits 2 (2,1) 2 (1,4) 1* 3 (1,5) 1 (2,8) 0,47* Sepsis 0 (0) 18 (12.4) <0.001*	Pulmonary embolism	0 (0)	2 (1.4)	0.52*	1 (0.5)	1 (2.8)	0.27*	
Sepsis 0 (0) 18 (12.4) < 0.001* 0 (0) 18 (50) < 0.001* Bleedings other than cerebral 1 (1) 3 (2.1) 0.65* 2 (1) 2 (5.6) 0.106* Stroke subtypes (etiological classification) 2 (2.1) 0 (0) 0.15* 2 (1) 0 (0) 1* Sinus thrombosis 5 (5.2) 0 (0) 0.009* 5 (2.4) 0 (0) 1* Hemorrhage including SAH 20 (20.6) 26 (17.9) 0.15 40 (19.4) 6 (16.7) 0.74 Non-determined 17 (17.5) 20 (13.8) 0.4 28 (13.6) 9 (25) 0.07 Other determined 2 (2.1) 3 (2.1) 1* 4 (1.9) 1 (2.8) 0.5* Small artery 24 (24.7) 27 (18.6) 0.2 51 (24.8) 0 (0) 0.0001* Cardioembolic 11 (11.3) 31 (21.4) 0.04 32 (15.5) 10 (27.8) 0.2 TIA 3 (3.1) 0 (0) 0.03* 3 (1.4) 0 (0) 1* LACH 5 (Fits	2 (2.1)	2 (1.4)	1*	3 (1.5)	1 (2.8)	0.47*	
Bleedings other than cerebral 1 (1) 3 (2.1) 0.65* 2 (1) 2 (5.6) 0.106* Stroke subtypes (etiological classification)	Sepsis	0 (0)	18 (12.4)	< 0.001*	0 (0)	18 (50)	< 0.001*	
Stroke subtypes (etiological classification) 2 (2.1) 0 (0) 0.15* 2 (1) 0 (0) 1* Sinus thrombosis 5 (5.2) 0 (0) 0.009* 5 (2.4) 0 (0) 1* Hemorrhage including SAH 20 (20.6) 26 (17.9) 0.15 40 (19.4) 6 (16.7) 0.74 Non-determined 17 (17.5) 20 (13.8) 0.4 28 (13.6) 9 (25) 0.07 Other determined 2 (2.1) 3 (2.1) 1* 4 (1.9) 1 (2.8) 0.5* Small artery 24 (24.7) 27 (18.6) 0.2 51 (24.8) 0 (0) 0.0001* Cardioembolic 11 (11.3) 31 (21.4) 0.64 32 (15.5) 10 (27.8) 0.2 Large artery 13 (13.4) 38 (26.2) 0.01 41 (19.9) 10 (27.8) 0.2 TIA 3 (3.1) 0 (0) 0.03** 3 (1.4) 0 (0) 1* Stroke subtype (Oxford classification) T T 1 (5.3) 3 (8.3) 0.4* POCH 1 (1) 1 (0.7) 1* 2 (1) 0 (0) 1* PACH	Bleedings other than cerebral	1 (1)	3 (2.1)	0.65*	2 (1)	2 (5.6)	0.106*	
Venous infarction 2 (2.1) 0 (0) 0.15* 2 (1) 0 (0) 1* Sinus thrombosis 5 (5.2) 0 (0) 0.009* 5 (2.4) 0 (0) 1* Hemorrhage including SAH 20 (2.6) 26 (17.9) 0.15 40 (19.4) 6 (16.7) 0.74 Non-determined 17 (17.5) 20 (13.8) 0.4 28 (13.6) 9 (25) 0.07 Other determined 2 (2.1) 3 (2.1) 1* 4 (1.9) 1 (2.8) 0.5* Small artery 24 (24.7) 27 (18.6) 0.2 51 (24.8) 0 (0) 0.0001* Cardioembolic 11 (11.3) 31 (21.4) 0.04 32 (15.5) 10 (27.8) 0.2 TIA 3 (3.1) 0 (0) 0.03* 3 (1.4) 0 (0) 1* Stroke subtype (Oxford classification) T T 1 1* 1* POCH 1 (1) 1 (0.7) 1* 2 (1) 0 (0) 1* LACH 5 (5.2) 9 (6.2) 0.7 11 (5.3)	Stroke subtypes (etiological classifica	ation)				()		
Sinus thrombosis 5 (5.2) 0 (0) 0.009* 5 (2.4) 0 (0) 1* Hemorrhage including SAH 20 (20.6) 26 (17.9) 0.15 40 (19.4) 6 (16.7) 0.74 Non-determined 17 (17.5) 20 (13.8) 0.4 28 (13.6) 9 (25) 0.07 Other determined 2 (2.1) 3 (2.1) 1* 4 (1.9) 1 (2.8) 0.5* Small artery 24 (24.7) 27 (18.6) 0.2 51 (24.8) 0 (0) 0.0001* Cardioembolic 11 (11.3) 31 (21.4) 0.04 32 (15.5) 10 (27.8) 0.2 TIA 3 (3.1) 0 (0) 0.03* 3 (1.4) 0 (0) 1* Stroke subtype (Oxford classification) TIA 3 (3.1) 0 (0) 0.06* 3 (1.4) 0 (0) 1* LACH 5 (5.2) 9 (6.2) 0.7 11 (5.3) 3 (8.3) 0.4* POCH 1 (1) 1 (0.7) 1* 2 (1) 0 (0) 1* LACH 5 (5.2) 9 (6.2) 0.7 11 (5.3) 3 (8.3) 0.7* POCH	Venous infarction	2 (2 1)	0 (0)	0.15*	2 (1)	0 (0)	1*	
Hemorrhage including SAH 20 (20.6) 26 (17.9) 0.15 40 (19.4) 6 (16.7) 0.74 Non-determined 17 (17.5) 20 (13.8) 0.4 28 (13.6) 9 (25) 0.07 Other determined 2 (2.1) 3 (2.1) 1* 4 (1.9) 1 (2.8) 0.5* Small artery 24 (24.7) 27 (18.6) 0.2 51 (24.8) 0 (0) 0.0001* Cardioembolic 11 (11.3) 31 (21.4) 0.04 32 (15.5) 10 (27.8) 0.2 TIA 3 (3.1) 0 (0) 0.03* 3 (1.4) 0 (0) 1* Stroke subtype (Oxford classification) TIA 3 (3.1) 0 (0) 0.06* 3 (1.4) 0 (0) 1* LACH 5 (5.2) 9 (6.2) 0.7 11 (5.3) 3 (8.3) 0.4* POCH 1 (1) 1 (0.7) 1* 2 (1) 0 (0) 1* PACH 11 (11.3) 12 (8.2) 0.4 20 (9.7) 3 (8.3) 0.7* TACH 0 (0) 1 (0.7) 1* 1 (0.5) 0 (0) 1* LACI 32 (33	Sinus thrombosis	5 (5 2)	0 (0)	0.009*	5 (2 4)	0 (0)	1*	
Non-determined 17 (17.5) 20 (13.8) 0.4 28 (13.6) 9 (25) 0.07 Other determined 2 (2.1) 3 (2.1) 1* 4 (1.9) 1 (2.8) 0.5* Small artery 24 (24.7) 27 (18.6) 0.2 51 (24.8) 0 (0) 0.0001* Cardioembolic 11 (11.3) 31 (21.4) 0.04 32 (15.5) 10 (27.8) 0.06 Large artery 13 (13.4) 38 (26.2) 0.01 41 (19.9) 10 (27.8) 0.2 TIA 3 (3.1) 0 (0) 0.03* 3 (1.4) 0 (0) 1* Stroke subtype (Oxford classification) T 1 1 0.7 11 (5.3) 3 (8.3) 0.4* POCH 1 (1) 1 (0.7) 1* 2 (0.97) 3 (8.3) 0.7* TACH 0 (0) 1 (0.7) 1* 2 (1) 0 (0) 1* PACH 11 (11.3) 12 (8.2) 0.4 20 (9.7) 3 (8.3) 0.7* TACH 0 (0) 1 (0.7) 1* 1 (0.5) 0 (0) 1* LACI 32 (33) <t< td=""><td>Hemorrhage including SAH</td><td>20 (20 6)</td><td>26 (17 9)</td><td>0.15</td><td>40 (19 4)</td><td>6 (16 7)</td><td>0.74</td></t<>	Hemorrhage including SAH	20 (20 6)	26 (17 9)	0.15	40 (19 4)	6 (16 7)	0.74	
Non-determined 2 (11.6) 2 (11.6) 0.1 1 (10.6) 1 (2.6) 0.1 Other determined 2 (2.1) 3 (2.1) 1* 4 (1.9) 1 (2.8) 0.5* Small artery 24 (24.7) 27 (18.6) 0.2 51 (24.8) 0 (0) 0.0001* Cardioembolic 11 (11.3) 31 (21.4) 0.04 32 (15.5) 10 (27.8) 0.6 Large artery 13 (13.4) 38 (26.2) 0.01 41 (19.9) 10 (27.8) 0.2 TIA 3 (3.1) 0 (0) 0.03* 3 (1.4) 0 (0) 1* Stroke subtype (Oxford classification) TIA 3 (3.1) 0 (0) 0.06* 3 (1.4) 0 (0) 1* LACH 5 (5.2) 9 (6.2) 0.7 11 (5.3) 3 (8.3) 0.4* POCH 1 (1) 1 (0.7) 1* 2 (1) 0 (0) 1* PACH 11 (11.3) 12 (8.2) 0.4 20 (9.7) 3 (8.3) 0.7* TACH 0 (0) 1 (0.7) 1* 1 (0.5) 0 (0) 1* LACI 32 (33) <t< td=""><td>Non-determined</td><td>17 (17 5)</td><td>20 (13.8)</td><td>0.4</td><td>28 (13.6)</td><td>9 (25)</td><td>0.07</td></t<>	Non-determined	17 (17 5)	20 (13.8)	0.4	28 (13.6)	9 (25)	0.07	
Sound occentinated 2 (2.17) 3 (2.17) 1 (1.13) 1 (1.13) 1 (1.13) 1 (1.13) 1 (1.13) 0.001* Small artery 24 (24.7) 27 (18.6) 0.2 51 (24.8) 0 (0) 0.0001* Cardioembolic 11 (11.3) 31 (21.4) 0.04 32 (15.5) 10 (27.8) 0.2 TIA 3 (3.1) 0 (0) 0.03* 3 (1.4) 0 (0) 1* Stroke subtype (Oxford classification) TIA 3 (3.1) 0 (0) 0.06* 3 (1.4) 0 (0) 1* LACH 5 (5.2) 9 (6.2) 0.7 11 (5.3) 3 (8.3) 0.4* POCH 1 (1) 1 (0.7) 1* 2 (1) 0 (0) 1* PACH 11 (11.3) 12 (8.2) 0.4 20 (9.7) 3 (8.3) 0.7* TACH 0 (0) 1 (0.7) 1* 1 (0.5) 0 (0) 1* LACI 32 (33) 35 (24.1) 0.13 66 (32.1) 1 (2.8) 0.0003* POCI 6 (62) 13 (9) 0.4 11 (5.3) 8 (22.2) 0.0003* <td>Other determined</td> <td>2 (2 1)</td> <td>3 (2 1)</td> <td>1*</td> <td>4 (1 9)</td> <td>1 (2.8)</td> <td>0.5*</td>	Other determined	2 (2 1)	3 (2 1)	1*	4 (1 9)	1 (2.8)	0.5*	
Sindicate(y) Er (E.B.) Er (E.B.) <td>Small artery</td> <td>2 (2.1)</td> <td>27 (18.6)</td> <td>0.2</td> <td>51 (24.8)</td> <td>0 (0)</td> <td>0.0001*</td>	Small artery	2 (2.1)	27 (18.6)	0.2	51 (24.8)	0 (0)	0.0001*	
Large artery 13 (13.4) 38 (26.2) 0.01 41 (19.9) 10 (27.8) 0.2 TIA 3 (3.1) 0 (0) 0.03* 3 (1.4) 0 (0) 1* Stroke subtype (Oxford classification) TIA 3 (3.1) 0 (0) 0.06* 3 (1.4) 0 (0) 1* LACH 5 (5.2) 9 (6.2) 0.7 11 (5.3) 3 (8.3) 0.4* POCH 1 (1) 1 (0.7) 1* 2 (1) 0 (0) 1* PACH 11 (11.3) 12 (8.2) 0.4 20 (9.7) 3 (8.3) 0.7* TACH 0 (0) 1 (0.7) 1* 1 (0.5) 0 (0) 1* LACI 32 (33) 35 (24.1) 0.13 66 (32.1) 1 (2.8) 0.0003*	Cardioembolic	11 (11 3)	31 (21.4)	0.04	32 (15 5)	10 (27.8)	0.06	
Large artery 15 (15.1) 50 (25.2) 0.01 11 (15.7) 10 (21.6) 0.2 TIA 3 (3.1) 0 (0) 0.03* 3 (1.4) 0 (0) 1* Stroke subtype (Oxford classification) TIA 3 (3.1) 0 (0) 0.06* 3 (1.4) 0 (0) 1* LACH 5 (5.2) 9 (6.2) 0.7 11 (5.3) 3 (8.3) 0.4* POCH 1 (1) 1 (0.7) 1* 2 (1) 0 (0) 1* PACH 11 (11.3) 12 (8.2) 0.4 20 (9.7) 3 (8.3) 0.7* TACH 0 (0) 1 (0.7) 1* 1 (0.5) 0 (0) 1* LACI 32 (33) 35 (24.1) 0.13 66 (32.1) 1 (2.8) 0.0003* POCI 6 (6 2) 13 (9) 0.4 11 (5.3) 8 (22.2) 0.0005*		13 (13.4)	38 (26 2)	0.01	41 (19.9)	10 (27.8)	0.00	
Inv 5 (0.1) 6 (0) 6 (0) 6 (0) 6 (0) 1 Stroke subtype (Oxford classification) TIA 3 (3.1) 0 (0) 0.06* 3 (1.4) 0 (0) 1* LACH 5 (5.2) 9 (6.2) 0.7 11 (5.3) 3 (8.3) 0.4* POCH 1 (1) 1 (0.7) 1* 2 (1) 0 (0) 1* PACH 11 (11.3) 12 (8.2) 0.4 20 (9.7) 3 (8.3) 0.7* TACH 0 (0) 1 (0.7) 1* 1 (0.5) 0 (0) 1* LACI 32 (33) 35 (24.1) 0.13 66 (32.1) 1 (2.8) 0.0003* POCI 6 (6 2) 13 (9) 0.4 11 (5.3) 8 (22.2) 0.0005*		3 (3 1)	0 (0)	0.03*	3 (1 /)	0 (0)	1*	
TIA 3 (3.1) 0 (0) 0.06* 3 (1.4) 0 (0) 1* LACH 5 (5.2) 9 (6.2) 0.7 11 (5.3) 3 (8.3) 0.4* POCH 1 (1) 1 (0.7) 1* 2 (1) 0 (0) 1* PACH 11 (11.3) 12 (8.2) 0.4 20 (9.7) 3 (8.3) 0.7* TACH 0 (0) 1 (0.7) 1* 1 (0.5) 0 (0) 1* LACI 32 (33) 35 (24.1) 0.13 66 (32.1) 1 (2.8) 0.0003* POCI 6 (6 2) 13 (9) 0.4 11 (5 3) 8 (22 2) 0.0005*	Stroke subtype (Oxford classification)	0 (0)	0.05	J (1.1)	0(0)	I	
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		52 (55)	13 (0)	0.15	11 (5 2)	r (∠.0) Q (フフ フ)	0.0005	

Table 3 Comparing between groups depending on mRS on discharge and mortality regarding various categorial parameters

	mRs on discharge			Mortality			
	Good (mRs≤2) n=97	Bad (mRs > 2) n = 145	P value	Alive n=206	Dead n=36	<i>P</i> value	
	Count (%)	Count (%)		Count (%)	Count (%)		
PACI	15 (15.5)	38 (26.2)	0.04	44 (21.4)	9 (25)	0.6	
TACI	17 (17.5)	34 (23.5)	0.2	39 (18.9)	12 (33.3)	0.051	
Others	7 (7.2)	2 (1.4)	0.03*	9 (4.4)	0 (0)	0.2	
rtPA							
rtPA	39 (58.2)	36 (30.3)	< 0.001*	64 (41)	11 (36.7)	0.65	
Place of admission							
Intermediate care unit	92	112	0.0002	183 (89.7)	21 (10.3)	< 0.001*	
Intensive care unit (ICU)	5	33		23 (60.5)	15 (39.5)		

*P value calculated from Fisher's exact test. Bold values indicate statistical significance

mRS: modified Rankin score; HTN: hypertension; CVS: cerebrovascular stroke; OCP: oral contraceptive pills; UTI: urinary tract infection; DVT: deep venous thrombosis; SAH: sub arachnoid hemorrhage; TIA: transient ischemic attack; LACH: lacunar hemorrhage; POCH: posterior circulation hemorrhage; PACH: partial anterior circulation hemorrhage; TACH: total anterior circulation hemorrhage; LACI: lacunar infarction; POCI: posterior circulation infarction; PACI: partial anterior circulation infarction; TACI: total anterior circulation infarction; rtPA: recombinant tissue plasminogen activator

Table 4 Comparing between groups depending on mRS on discharge and mortality regarding various numerical parameters

	Good outco (mRS≤2) n	me Bad outcome = 97 (mRS > 2) n = 14		ne <i>P</i> value = 145		Alive <i>n</i> = 206		Dead <i>n</i> =36		P value
	$Mean \pm sd$	Median (Range)	$Mean \pm sd$	Median Range		$Mean \pm sd$	Median (Range)	$Mean \pm sd$	Median Range	
Age	55.9 ± 13.8		61.7 ± 12.2		0.001	58.38 ± 13.34		64.81 ± 10.54		0.005
NIHSS on admission		6 (0–19)		12 (0–31)	<0.001		9 (0–22)		13.50 (2–31)	< 0.001
Onset to door(min)		150 (60–230)		150 (60–220)	0.4		150 (60–230)		180 (60–210)	0.55
Door to needle(min)	50.9 <u>+</u> 8.3		50.2 <u>+</u> 9.8		0.7	49.9 <u>+</u> 9.36		54.5 <u>+</u> 5.7		0.11
Onset to needle(min)		210 (110–270)		205 (115–260)	0.4		200 (110–270)		230 (120–260)	0.37
GUSS score on admission		20 (2–20)		12 (0–20)	< 0.001		20 (0–20)		4.5 (0–20)	< 0.001

mRS: modified Rankin score; NIHSS: national institute of health stroke scale; min: minute; GUSS: Gugging Swallowing Scale. Bold values indicate statistical significance

seizures, and others, were not associated with poor outcome at discharge. They considered all complications as one item and did not consider each as a separate entity. That could be an explanation for the discrepancy in results [32].

Regarding treatment with rtPA, our results showed that it is significantly associated with better outcomes regarding disability at discharge, but is not an independent predictor. Regarding in-hospital mortality, our results showed no significant difference between rtPA and nonrtPA treated patients. This come in agreement with other results [22, 27].

Moreover, our results showed that time to treatment (onset to treatment) was not significantly related to outcomes.rtPA was associated with better outcome in a time-dependent pattern, while mortality did not vary up to 4.5 h in one study [33]. In addition, treatment within 60 min, compared with treatment within 61 to 270 min, was associated with increased odds of discharge to home, independent ambulation at discharge, and freedom from disability (mRS 0–1) on discharge, without increased hemorrhagic complications or in-hospital mortality in another study [34].

In agreement with our results, one study found that early treatment with rtPA was not associated with an increased probability of having major neurologic improvement at 24 h after IV rt-PA administration. The mean time from symptom onset to treatment was **Table 5** Results of multivariate logistic regression analysis (usingstepwise model) for prediction of having bad mRS on dischargeand in-hospital mortality (table showing only significant results)

	P value OR		95% Cl	
			Lower	Upper
Bad mRS				
HTN	0.011	2.6	1.242	5.440
GUSS on admission	0.001	0.899	0.845	0.957
mRS on admission	0.002	2.401	1.369	4.209
NIHSS on admission	0.003	1.175	1.056	1.308
Mortality				
Hemorrhagic transformation	0.003	7.392	1.971	27.719
Chest infection	< 0.001	12.125	4.539	32.383
Bed sores	< 0.001	17.551	3.542	86.958
mRS on admission	0.003	3.560	1.523	8.323

mRS: modified Rankin scale; GUSS: Gugging Swallowing Scale; NIHSS: national institute of health stroke scale. Bold font indicates statistical significance

157 min (median 160 min). In the overall sample, only 4.1% of patients were treated within 90 min of stroke onset [35].

In our study, the mean onset to needle time was 202.5 min, and with more than half of the patients (58.7%) being injected after 3 h from stroke onset, this may explain the negative association between time to treatment and outcome in our study.

Our results showed that lower GUSS was significantly associated with disability, mortality, and developing chest infections. Moreover, in multivariate logistic regression, the GUSS score appears to be an independent predictor for disability. The same result was found in previous works [36, 37].

Conclusions

At the end of our discussion, we have to note that there is wide variability between studies regarding different variables in relation to stroke outcome. This could be explained the by different scores and variable systems used to evaluate the outcome, as well as the different used functional outcome measures among studies.

There were some limitations in this study. First, this study measured a very short-term outcome of acute stroke patients during hospital stay. A long-term study is required for further comments. Second, the study was conducted in a university hospital, which may include more severe and complicated stroke patients, and this may affect our results. Finally, a significant portion of our study occurred during the corona virus disease 2019(COVID-19) era which might affected the rate of patients admitted to the stroke unit and the quality of management.

We recommend including quality indicators as part of our stroke unit program to regularly assess our performance, so that we can improve it, which will have a beneficial effect on patients. Special attention should be taken to reduce "door to needle" time for intravenous thrombolysis. Dysphagia screening using a valid, reliable, and easy tool such as the GUSS scale is beneficial in predicting and thus preventing and managing aspiration pneumonia.

Abbreviations

mRS	Modified Bankin scale
NILLCC	National institute of health stroke scale
	Guaging Swallowing Scale
UU33 ПТИ	Hyportonsion
rtDA	Pocombinant tissue plasminogen activator
	Door to poodlo
VDIc	Key performance indicators
	Rey performance indicators
ncis ct	
	Computed tomography
	Magnetic resonance imaging
ECG	Electrocardiogram
MRA	Magnetic resonance angiography
IOASI	Irial of ORG 10172 in acute stroke treatment
GWIG	Get With The Guidelines
SPSS	Statistical package for the Social Sciences
REC	Research Ethics Committee
TACI	Total anterior circulation infarction
TACH	Total anterior circulation hemorrhage
PACI	Partial anterior circulation infarction
PACH	Partial anterior circulation hemorrhage
POCI	Posterior circulation infarction
POCH	Posterior circulation hemorrhage
LACI	Lacunar infarction
LACH	Lacunar hemorrhage
TIA	Transient ischemic attack
OCP	Oral contraceptive pills
CVS	Cerebrovascular stroke
UTI	Urinary tract infection
DVT	Deep venous thrombosis
SAH	Sub arachnoid hemorrhage
ICU	Intensive care unit
COVID-19	Corona virus disease 2019

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Author contributions

MAZ was the idea founder, shared in the patient collection, and the supervisor in all the steps. AMA shared in the patient collection and supervision. HSM shared in the patient collection and supervision AAS shared in the patient collection, wrote and revised the manuscript. AMF did the data analysis, and he is the submitting and corresponding author. All authors read and approved the final manuscript.

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Availability of data and materials

The data sets generated and/or analyzed during the current study are not publicly available due to privacy and ethical restrictions but are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate.

The institutional review board of faculty of medicine, Cairo University approved this study in 17-4-2018. Informed written consent was obtained from each participant or their relatives if they were unable to give the consent owing to their medical condition. All methods were carried out in accordance with relevant guidelines and Declaration of Helsinki.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

Authors' information

Maha Atef Zaki is a professor of neurology at Cairo University. Ahmed Mohamed Abdelalim is a professor of neurology at Cairo University. Husam Salah Mourad is a professor of neurology at Cairo University. Abdallah Adel Saad is an Assistant lecturer of neurology at Cairo University. Amr Mohamed Fouad is a lecturer of neurology at Cairo University.

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