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Research Article

Neurological Deterioration in the Acute Phase of Ischemic Stroke in Ouagadougou, Burkina Faso: Frequency, Causes and Risk Factors

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Abstract

Introduction: Neurological deterioration in the acute phase of ischemic stroke is associated with a poor prognosis. The aim of the present study was to determine the frequency, causes and risk factors of neurological deterioration in the acute phase of ischemic stroke at the Tingandogo University Hospital, in Ouagadougou, Burkina Faso, in order to help improve the prognosis after stroke.

Patients and Methods: This was a hospital, prospective, cross-sectional, descriptive and analytical study of patients hospitalized from January 2015 to December 2018 for ischemic stroke at the Tingandogo University Hospital. The general characteristics of patients on admission, the occurrence and characteristics of neurological deterioration, other intra-hospital complications, and the clinical outcome of patients at the end of hospitalization were analyzed. Univariate and then multivariate statistical analyzes according to the Cox proportional hazards model between the general characteristics of patients on admission and the occurrence of neurological deterioration, made it possible to identify the independent risk factors associated with the occurrence of neurological deterioration.

Results: A total of 110 patients were included, with an average age of 64.4 years and predominantly male (66.4%). Neurological deterioration occurred in 23 patients (20.9%) within an average of 4 days after admission. Progression and/or recurrence of ischemic stroke (17.4%) was the most common cause. After hospitalization, 22 patients died (20%) and 47 patients (42.7%) were still dependent. Age ≥ 65 years, impaired alertness on admission, hyperglycemia on admission, and cardioembolic ischemic stroke, were independent risk factors associated with the occurrence of neurological deterioration.

Conclusion: Neurological deterioration affects approximately one in 5 patients in the acute phase of ischemic stroke. The main cause is progression and/or recurrence of the cerebral infarction. Early admission of patients, implementation of stroke units and fibrinolysis, increased availability of efficient MRI, in our hospitals, will contribute to a significant improvement in the prognosis of stroke.

Keywords: *Ischemic Stroke; Neurological Deterioration; Intra-Hospital; Progression/Recurrence of the Infraction; Initial Hyperglycemia; Age ≥ 65 Years; Impaired Alertness; Cardio-Embolic; Ouagadougou*

Introduction

Neurological deterioration (ND) in the acute phase of ischemic stroke (IS) is a rapid or sudden and persistent clinical worsening of the basic neurological deficit, variously defined, most often corresponding to an increase of at least 4 points or at least 2 points on the National Institute of Healt Stroke Scale (NIHSS). The incidence of ND varies considerably, from 5% to 40% [1,2], depending on the definition criteria and the time frames used [1-4]. ND tends to occur during the first days after stroke and depends on various underlying causes, including progression of the infarction, malignant cerebral edema, symptomatic intra-infarct hemorrhagic rearrangement, increased intracranial pressure [1,2]; some authors associate various systemic diseases such as infections or metabolic or hemodynamic imbalances [2,3,5,6]. Whatever the definition criteria used, ND is systematically and strongly associated with an unfavorable vital and functional prognosis after stroke [2,3,6].

Given the still high rates of early mortality of IS in sub-Saharan Africa in general (17.9 - 38%) and in Burkina Faso (17.9%) in particular [7], given the absence of a Stroke Units (SU) and fibrinolysis, however proven to be effective in reducing post-stroke mortality and disability, a good understanding of intra-hospital ND and its risk factors for occurrence in the acute phase of IS, could help improve prognosis of stroke patients in our context, hence the interest of our study.

Objective of the Study

The objective of this work was to determine the frequency, causes and identify the risk factors of ND in the acute phase of IS, at the Tingandogo University Hospital (TUH) in Ouagadougou in Burkina Faso, through a prospective cross-sectional study.

Patients and Methods

This was a prospective cross-sectional and descriptive study that concerned all adult patients hospitalized at the TUH, in Ouagadougou (Burkina Faso) for IS from 01/01/2015 to 12/31/2018. Were included in the study, any patient aged > 15 years, hospitalized in said hospital for IS less than 72 hours since installation, during the study period, documented by Brain CT and/or MRI and for which consent to the study has been obtained.

Were not included in the study, patients who did not perform brain CT, those admitted more than 72 hours after the onset of IS, patients who died on arrival, patients for whom consent participation in the study was not obtained.

For all patients, upon admission, measurement of vital constants, initial clinical neurological and general evaluation by a senior neurologist, standard ECG, brain CT and/or MRI interpreted by a radiologist, the initial biological blood test standard, including lipid balance, were performed. Transthoracic cardiac ultrasound and 24-hour holter ECG were also performed, if necessary. Standard chest radiography was performed in case of suspicion of bronchopulmonary infection, certain laboratory tests for monitoring metabolic disorders, venous ultrasound and thoracic CT angiography, respectively in case of suspicion of venous thrombosis of the limbs or of pulmonary embolism; thick gout, cytobacteriological examination of urine, blood cultures, respectively in case of suspected malaria, urinary tract infection, sepsis. Control brain CT was not routine; it was performed in the event of neurological deterioration and/or headache. On discharge from hospital, patients were subdivided according to discharge status, into surviving or deceased patients. In the event of death occurring during hospitalization, the immediate causes of death were determined by staff of senior neurologists.

Patient management was carried out according to the recommendations of the European Stroke Organization (ESO 2008). The neurology department does not yet have a SU, and intravenous fibrinolysis is not yet performed.

The variables studied were: socio-demographic data, time to admission, time to perform cerebral CT, vascular risk factors (VRF) [hypertension, diabetes mellitus, smoking, dyslipidemia (total cholesterol, HDL (High Density Lipoprotein) cholesterol, LDL (Low Density Lipoprotein) cholesterol, triglycerides), alcohol consumption (> 3 drinks per day), sedentary lifestyle (patients whose physical activity did not exceed 30 minutes during most days of the week), obesity (BMI > 30 kg/m^2), history of stroke, coronary artery disease, atherosclerosis of the supra-aortic trunks, cardiac arrhythmia due to atrial fibrillation (AF), heart failure, arteriopathy obliterans of the lower limbs, migraine, sickle cell anemia, hormonal contraception], comorbidities, constants on admission (BP, temperature, oxygen saturation), state

of vigilance according to the Glasgow score on admission and current hospitalization, neurological deficit according to NIHSS on admission and during hospitalization, the radiological characteristics of the cerebral infarction on admission [(old scarring lesions, early signs of cerebral ischemia, arterial territories, associated neuroradiological signs (cerebral edema, mass effect, cerebral engagement, hemorrhagic transformation, hydrocephalus)], qualitative biological data on admission and during hospitalization (glycemia, glomerular filtration rate, hemoglobin level, leukocytosis, natremia, serum potassium, protidemia), general medical complications present on admission or appearing during hospitalization, clinical neurological deterioration (ND) during hospitalization (occurrence, delay, severity, suspected cause), epileptic seizures, the modified Rankin score (mRS) at discharge, the duration of hospitalization.

The consent of the patients or that of their legal representatives, as well as the anonymous nature of the survey, were guaranteed before recruitment. The study protocol had been approved by the national ethics committee of Burkina Faso, then authorized by the General Management of Tingandogo University Hospital.

Statistical analyzes were carried out using the Epi info 7.2.26 software. Student's t-test was used to compare means and Pearson's Chi-square test to compare percentages; the p-value ≤ 0.05 was considered to be the threshold of statistical significance. Univariate analyzes between the independent variables (general characteristics of patients on admission) and the dependent variable (ND) made it possible to select the variables significantly associated with the occurrence of ND. Finally, we carried out a multiple logistic regression analysis (with calculation of odds ratios) using the Cox proportional hazards model, to identify the independent risk factors associated with the occurrence of ND. Only variables with a p-value < 0.20 in univariate analysis were taken into account for multivariate analysis.

ND was defined as a sudden or rapid and persistent clinical worsening of the baseline neurological state, corresponding to a loss of at least 2 points in the Glasgow score and/or an increase of at least 4 points in the NIHSS total except in its items 1a, 1b or 1c (impaired alertness) where an increase of at least 2 points was sufficient; occurring between admission and the first two weeks after stroke installation. Depending on its time to onset after the installation of the IS, ND was subdivided into early ND (END) occurring in the first 24 hours, the intermediate ND between the 24th and the 72nd hours and the late ND occurring between the 4th and 14th day.

The NIHSS was used for the assessment of clinical severity in the acute phase of stroke. The neurological deficit was estimated: slight for an NIHSS ≤ 5; moderate for an NIHSS between 6 and 16; severe for an NIHSS between 17 and 25; very severe for NIHSS > 25.

The mRS was used for the evaluation of functional autonomy after stroke at discharge from hospital.

The Glasgow score was used for the assessment of alertness. Vigilance was considered normal for a Glasgow score of 15, impaired for a Glasgow score between 14 and 9; coma was defined for a Glasgow score ≤ 8.

Malignant cerebral edema was retained as the presumed cause of Clinical ND, when ND was consecutive to cerebral edema occurring in a cerebral infarction occupying at least 2/3 of the territory of the middle cerebral artery (MCA) and/or all MCA territory and/or an entire cerebellar hemisphere on neuroimaging, in the absence of other known causes of ND.

A symptomatic intra-infarct hemorrhagic transformation was retained as the cause of ND when the occurrence of ND was consecutive to the appearance of a hemorrhagic change; practically it is the *de novo* discovery of haematic hyperdensity within the CF focus on control brain CT motivated by the occurrence of ND, in the absence of other known causes of ND.

A recurrence or extension of the infarction was retained as the presumed cause of ND in the event of an occurrence of ND following the appearance of a new focus of infarction in an area free from ischemic lesion and/or the increase. The size of the base of the infarction; practically it is a question of the *de novo* discovery of a new infarction and/or of a significant increase in the size of the hearth of the IS on the cerebral CT of control motivated by the occurrence of the ND, without other presumed causes of ND.

A presumed hemodynamic or functional cause was selected as the probable cause of ND, in the event of ND occurring in the absence of all the above-mentioned presumed structural causes of ND, in the context of obvious metabolic and/or hemodynamic disorders.

Results

Descriptive study

During the study period, 165 patients were consecutively hospitalized for IS, among which 110 cases were selected according to our inclusion criteria.

The mean age was 64.4 years (± 13.8 years) (range 27 years and 94 years). The modal age group was [60 - 70] years with 36 patients (32.7%). The mean time to admission was 18.7 hours (± 14.21) (range 1-72 hours). The mean time to perform brain CT was 29.1 hours (± 29.31) (range 3 and 240 hours).

Male patients, those with an admission time of 4.5 hours to 24 hours, and those with a post-admission CT scan time of 12 hours to 24 hours, were most represented with 73 cases (66.4%), 58 cases (52.7%) and 78 cases (70.9%) respectively. The arterial hypertension with 82 patients (74.5%) and history of stroke with 18 cases (16.4%) were the most prevalent VRFs. Cardiac and neurological comorbidities with 14 cases (12.7%) and 12 cases (10.9%), respectively, were the most frequently found (Table 1).

Age (years)	Number (n = 110)	Frequencies (%)
< 50	16	14.55
[50 - 60]	19	17.27
[60 – 70]	36	32.73
[70 - 80]	26	23.63
> 80	13	11.82
Emergency admission delay (hours)		
≤ 4.5	4	3.6
[4,5 - 24]	58	52.7
[24 - 72]	48	43.6
Time to perform brain CT		
≤ 12	18	16.4
[12 - 24]	78	70.9
[24 - 72]	12	10.9
RVFs		
Arterial hypertension	82	74.5
History of stroke	18	16.4
Hypercholesterolemia	13	11.8
Alcohol	8	7.3
Smoking	8	7.3
Obesity	5	4.5
Sedentary lifestyle	2	1.8
Atrial fibrillation/Atrial flutter	1	0.9
Oral contraception	1	0.9
Comorbidities		
Cardiac	14	12.7
Chronic obstructive pulmonary disease	5	4.5
Neurological sequelae of stroke	12	10.9
Chronic renal failure	5	4.5
Spondylodiscopathy	4	3.6
Peptic ulcer	2	1.8
Other comorbidities	3	2.7

Table 1: *Distribution of patients according to general characteristics.*

On admission, the Glasgow score averaged 13.9 +/- 2.7 (range 7-15); a coma was present on admission in 4 patients (3.6%). The NIHSS averaged 12.4 (± 5.8) (range 0 and 26) on admission; 21 patients (19.1%) had severe to very severe neurological deficit on admission.

Hypertension found on admission in 85 patients (78.7%) was the main abnormality of the constants. Hyperglycemia in 43 patients (39.1%), hypokalaemia in 36 patients (32.7%) and renal failure in 29 patients (26.4%) were the most common laboratory abnormalities. Old scarring lesions were present in 26 patients (23.6%), early signs of cerebral ischemia in 7 patients (6.2%) with erase of cortical sulcus in four patients and spontaneously hyperdense arteries in three patients, ie 3.63% and 2.73% respectively, were found. The MCA territory with 65 cases (59.1%) was most frequently affected. Atherosclerosis of the cervico-encephalic arteries and lacunar infarctions, found respectively in 29 cases (26.4%) and 28 patients (25.4%), were the most frequently encountered causes of IS (Table 2).

Clinical and paraclinical characteristics on admission	Number (n)	Frequencies (%)
State of vigilance		
Normal vigilance	85	77.3
Altered vigilance	21	19.1
Coma	4	3.6
Severity of neurological deficit (NIHSS)		
NIHSS ≤ 5	18	16.4
6≤ NIHSS ≤ 16	71	64.6
17 ≤ NIHSS ≤ 25	20	18.2
NIHSS> 25	1	0.9
Anomalies of constants		
Arterial hypertension	85	78.7
Arterial hypotention	4	3.6
Fever	16	14.5
Desaturation (SPO_2 < 95%)	14	12.7
Biological abnormalities		
Hyperglycemia	43	39.1
Hypokalaemia	36	32.7
Renal failure	29	26.4
Anemia	21	19.1
Hyperleukocytosis	18	16.4
Hyponatremia	18	16.4
Hypoprotidemia	11	10.0
Leukopenia	7	6.4
Hypoglycemia	5	4.5
Hyperkalaemia	3	2.7

Table 2: *Distribution of patients according to clinical and paraclinical characteristics.*

Intra-hospital ND was observed in 23 patients, i.e. 20.9%, including 12 cases (10.9%) with associated impairment of vigilance and 11 cases (10%) with an increase in neurological deficit without impairment of vigilance. The mean time to onset of ND was 4 days (± 4) (range 1 to 17 days); the maximum frequency of occurrence of ND (13.6%) was observed during the first 72 hours after patient admission (Figure 1).

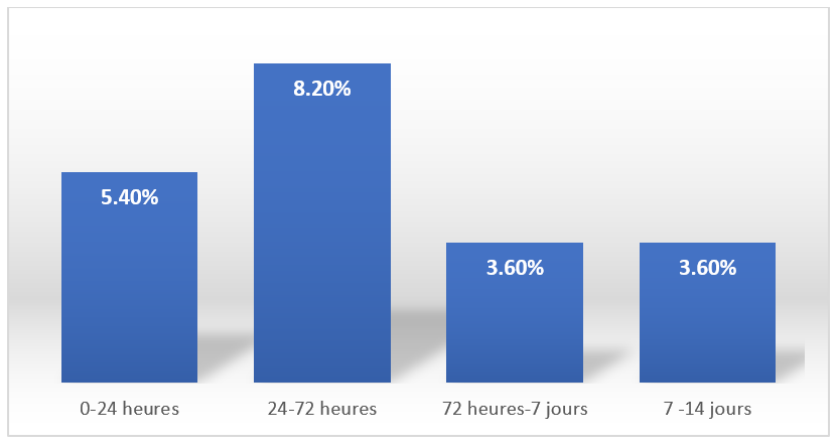

Figure 1: *Distribution of patients by time to onset of ND since admission.*

The increase in the neurological deficit corresponded on average to an increase of 8.6 points (± 4.9) (range 4 and 19 points) in the NIHSS; the majority of patients, or 16 patients (69.6%) experienced an increase in neurological deficit of at least 5 points on the NIHSS score during their hospitalization. The average number of points lost on the Glasgow scale was 4.4 points (± 2.3) points (range 2 and 7 points); altered alertness plunged 5 patients (21.7%) into a coma.

Progression and/or recurrence of HF with 4 cases (17.4%) was the most common cause, however with a high frequency (56.5%) of unknown causes (Figure 2).

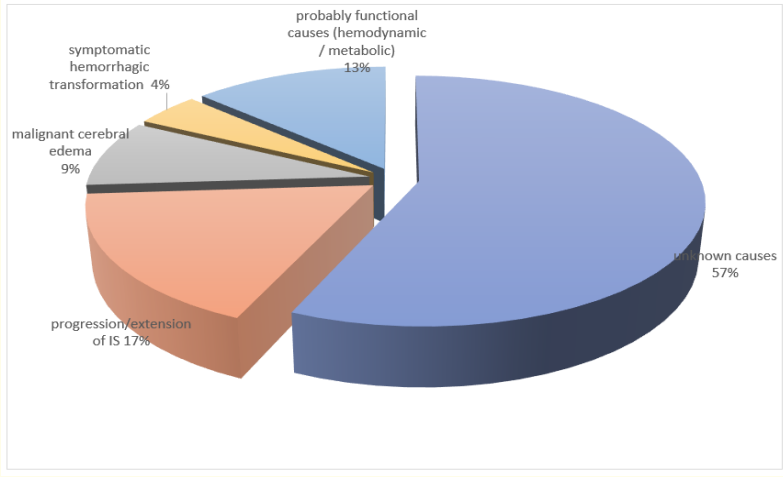

Figure 2: *Distribution of patients according to the causes of ND.*

At least one epileptic seizure was noted in 5 patients (4.5%). Intra-hospital extra neurological medical complications dominated by infectious complications and malnutrition respectively in 24 patients (21.8%) and 18 patients (16.4%), were recorded (Table 3).

Intra-Hospital Complications	Number (N = 24)	Frequencies (%)
Infections	24	21.8
Bronchopulmonary infection	14	12.7
Urinary tract infection	5	4.5
Sepsis	5	4.5
Undernutrition	18	16.4
Cardiac complications	7	6.4
Urinary incontinence	6	5.4
Pressure ulcers	4	3, 6
Deep vein thrombosis of limb	3	2.7
Respiratory distress	3	2.7

Table 3: *Distribution of patients according to intra-hospital medical complications.*

The median length of hospital stay was 7 days; the mean hospital stay was 11.4 days (± 7.97) (range 2-47 days). A total of 22 patients died during hospitalization, for an intra-hospital mortality rate of 20%; The 88 surviving patients were divided into 41 independent or autonomous (mRS 0-2) (37.3%) and 47 patients (42.7%) still dependent (mRS 3-5). At the end of hospitalization, the neurological deficit was marked by worsening in 23 patients (20.9%), a significant improvement in 34 patients (30.9%) and remained stationary in 53 patients (48.2%) (Figure 3).

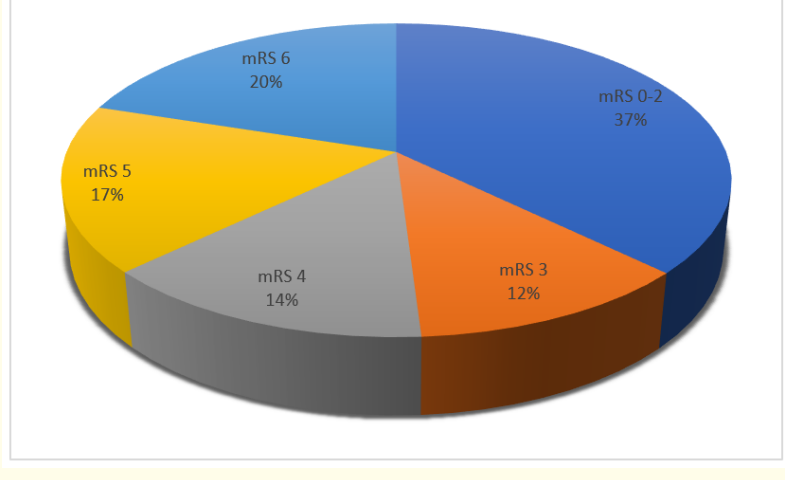

Figure 3: *Distribution of patients according to clinical outcome at the hospital discharge.*

Analytical study

In univariate analysis, the following variables were significantly associated with the occurrence of ND: age ≥ 65 years (p = 0.008), renal comorbidities (p = 0.005), impaired vigilance on admission (p = 0.002), hyperglycemia on admission (p = 0.006), a cardio-embolic cause of cerebral infarction (p = 0.02).

In multivariate analysis, according to the step-by-step logistic regression model, age ≥ 65 years (OR = 3.51; 95% CI [1.19 - 10.32]; p = 0.008), impaired alertness on admission (OR 6.81; 95% CI [2.45 - 18.91] ; p = 0.002), hyperglycemia on admission (OR 3.69; 95% CI [1.36 - 10.04]; p = 0.006) and cardio-embolic IS (OR 4, 91; 95% CI [1.13 - 21.27]; p = 0.02) were independent risk factors of ND occurrence (Table 4).

Variables	Modalities	Univaried analysis			Multivariate analysis		
		DN		P	GOLD	95% CI	p value
		Yes	No				
Age	≥ 65 ≤64	18 5	44 43	0.008	3.51	1.19-10.32	0.008
Sex	M F	8 15	29 58	0.44			
Educational level	No Primary Secondary Superior	15 1 7 0	56 5 19 7	0.56			
Reference mode	Residence CHU Private clinic CSPS/Paramedical practice CM	10 1 3 0 9	37 10 9 2 29	0.87			
Place of residence	Ouaga Other localities	17 6	53 34	0.36			
HTA	Yes No	16 7	66 21	0.27			
Smoking	Yes No	0 23	8 79	0.28			
Hypercholesterol-emia	Yes No	0 23	1 86	0.38			
Diabetic sugar	Yes No	1 22	12 75	0.11			
Oral contraception	Yes No	0 23	1 86	0.39			
ACFA/Atrial flutter	Yes No	1 22	0 87	0.10			
Alcohol consump-tion	Yes No	0 23	8 79	0.07			
Comorbidities	Yes No	4 19	10 77	0.23			
Glasgow score	≤ 14 ≥ 15	12 11	12 75	0.002	6.81	2.45-18.91	0.002
NHISS Score	≤ 16 ≥ 17	13 10	65 22	0.14			
Hyperglycemia	Yes No	10 12	16 72	0.006	3.69	1.36-10.04	0.006
Renal failure	Yes No	11 9	26 42	0.09	1.97	0.72-5.40	0.09
Hyperleukocytosis	Yes No	6 11	10 54	0.04	6.94	0.88-9.79	0.12
Hypokaiemia	Yes No	8 20	28 54	0.13			

Hyponatremia	Yes No	2 19	16 45	0.05	0.29	0.06-1.41	0.45
Early signs of cerebral ischemia	Yes No	3 20	4 83	0.09	3.11	0.64-15.02	0.89
Stroke territory	TO THAT ACM ACP Others	6 13 4 0	13 52 14 8	0.34			
Brain engagement	Yes No	3 20	7 80	0.2			
Brain edema	Yes No	2 21	8 79	0.49			
Hemorrhagic transformation	Yes No	1 22	3 84	0.4			
Embologenic heart disease	Yes No	4 6	8 59	0.02	4.91	1.13-21.27	0.02
Cervico-encephalic atherosclerosis	Yes No	2 8	27 39	0.11			

Table 4: *Results of univariate and multivariate analysis of risk factors associated with the occurrence of ND.*

In univariate analysis, ND was significantly associated with intra-hospital mortality (OR 8.29; 95% CI 2.89 - 23.71; p 0.00001) and unfavorable clinical outcome (mRS 3-6) (OR 2, 30 95% CI 1.23 - 4.48; p 0.001) (Table 5).

Variables	Deceased	Living	OR	95% CI	P
ND+	12	11	8.29	2.89-23.71	0.00001
ND -	10	77			
	mRS 0-2	mRS 3-6	OR	95% CI	
ND +	8	15	2.30	1.23-4.48	0.001
ND -	48	39			

Table 5: *Results of the univariate analysis between DN and intra-hospital death or clinical outcome at the end of hospitalization.*

Discussion

Most of the studies were limited to END, that is to say that occurring within the first 24 hours after the IS and the overall incidences described varied widely from 5% to 40% [1,8-10], depending on the definition used (the type and scale Δ of stroke) and the time period used for deterioration [2-4], the period of the study, the use or not of fibrinolysis, admission or not of patients to SU.

Regarding the studies that used the ΔNIHSS ≥ 4 endpoint, the incidence of END was 8% within 9 hours of onset [4]; the combined incidence of END within 24 hours of onset of infarction was 13.8% (range 2.2% to 37.5%) in a systematic review [1]; in fact this incidence varied from 8.1 to 28.1% and from 16.3 to 17.6%, respectively after fibrinolysis and in non-thrombolysed patients; this incidence was 15.5% at 24 hours from 15.5% in a Chinese study [11]. In a large multicenter German study, a 13% incidence of ND was reported within 72 hours of hospitalization [12]. These results are generally similar to ours with similar definition criteria. We found a frequency of intra-hospital ND of 20.9%, which became sudivised in END at 24 hours (5.4%), ND at 72 hours (13.6%) and late ND after 72 hours (7.3%).

In studies using the ΔNIHSS ≥ 2 endpoint, the incidence or prevalence of reported ND was 8.2% in a Norwegian study [6]; 25.4% in a Chinese study [11]; and 34% in an American study [3]. A recent multicenter study in Korea reported an incidence of ND that sharply decreased over time after stroke onset, from 18.3% at the 6th hour and to 6.4% between the 7th and 72 hours [2]. In this latest study, the incidence was 3 times higher in patients who went to hospital within 6 hours of stroke. Also in our study ND was variable over time, but was more frequent after the 24th hour. However in our context the END could have been underestimated because a significant proportion of cases were hospitalized between the 24th and 72nd hours following the onset of the stroke; thus possible cases of END would probably have gone unnoticed. This suggests that the varying incidences reported by previous studies are largely due to differences in observation duration.

While END occurred in 23 to 43% of patients with IS according to previous studies [13-15], some recent studies have shown a tendency to reduce the incidence of END (in 8 at 18%) [1,2,11,12], in particular due to the combined effect of SU and fibrinolysis [1,6,15]. Thus the early admission of our patients to SU and the administration of fibrinolysis to those with IS, will contribute to a significant improvement in their prognosis, through a reduction in the incidence of post-stroke ND.

Throughout the literature, the causes of ND commonly identified in order of decreasing frequency are i) progression or extension of the cerebral infarction in nearly 50% of patients after intravenous fibrinolysis [1], or even more in non-fibrinolyzed patients [1-3,6,11], approximately 40% of the causes of ND in all IS [3]; ii) symptomatic hemorrhagic transformation in 21.4% of fibrinolyzed infarcts and about 3 times less in non-fibrinolysed infarcts [1,3,11]; iii) malignant cerebral edema in 10.5% to 27% of cases [1,3,11]; iv) and widely varying recurrent cerebral infarction in less than 1% to 29% of cases [1-3,11].

In our study, the data on the causes of ND post IS should be interpreted with caution, due to the unavailability of MRI exams, the late admission of our patients and the absence of SU. In fact, in our work context, only CT is available and accessible for the progressive monitoring of patients, even though this examination is unsuitable for the detection of most of the causes and mechanisms of ND. Thus an implementation of SU and early revascularization therapies for IS (fibrinolysis and thrombectomy) in our hospitals, awareness of the population and health professionals on the therapeutic emergency of stroke, availability and increased accessibility of brain MRI with good resolution parenchymal and vascular sequences, would contribute to a better understanding of the causes and mechanisms of post-stroke ND and an improvement in the vital and functional prognosis of this condition in our context.

Several predictive factors of END present on admission, identified in the literature, were also found in our series: advanced age, hyperglycemia [1,16], initial severity of stroke [1,17-19], embolic heart disease [20]. On the other hand, other recognized predictive factors of END were not found in our study: history of diabetes, history of TIA, prior use of aspirin, a long delay in admission [1], large cerebral infarction [1,17], proximal arterial occlusion [1,21], etc. However, in the literature, the predictive factors of ND found appear to be very variable, and sometimes even contradictory, from one study to another. These discrepancies can be explained by differences in the definitions of ND and the methodologies used according to the studies. The initial clinical severity of the IS, represented clinically by a high NIHSS (> 17) and/or an alteration of alertness present on admission, most often reflects a neurological impairment that was severe from the outset, involving a risk significant symptomatic hemorrhagic transformation [1,22] and/or the onset of malignant cerebral edema [23]. However, the risk of END associated with the admission NIHSS appears to depend on the criteria for defining END, particularly whether absolute or relative changes are taken into account. Thus, less severe deficits predict END, which can be explained by a "ceiling effect", while higher admission scores are less likely to increase further [1].

Hyperglycemia promotes END by increasing the rupture of the blood-brain barrier and promoting the symptomatic hemorrhagic transformation of the infarction [1,24,25]; Hyperglycemia also facilitates neuronal damage, by producing lactate in the brain and accelerating the conversion of severely hypoperfused risk tissue into cerebral infarction [26]. Hyperglycemia also has prothrombotic effects, and may therefore facilitate the extension of the thrombus and therefore the progression of the infarction [27].

Advanced age and cardioembolic IS are associated with a greater risk of ND because cardioembolic IS is generally more severe with a greater risk of cerebral edema and hemorrhagic transformation and usually occurs in the group of older patients [20].

ND is associated with an unfavorable short- to medium-term vital and functional prognosis in almost all studies [1,2,6,28]. The same observation was made in our study, where ND significantly increased intra-hospital death by a factor of 8.2 (p = 0.00001) and the vital and functional prognosis by a factor of 2.3 (p = 0.001). The early admission and monitoring of patients suffering from stroke in SU and the use of early revascularization therapies (fibrinolysis, thrombectomy), which contribute to the reduction of the risk of occurrence of ND, therefore make it possible to reduce the négative impact of ND on the clinical vital and functional outcome of these patients [29,30].

Limits of Our Study

The small number of our patients due to the late admission beyond 72 hours for most of our patients, the admission of a good part of our patients beyond the 24th following the beginning of the stroke, the unavailability of SU and MRI, and the unsystematic nature of control brain CT scan, could have introduced bias on the frequency and causes of ND.

Conclusion

ND is a relatively frequent complication in the acute phase of IS, affecting globally one in 5 patients; in fact variable, from one in 20 patients during the first 24 hours, to just under one in 10 patients by the 72nd hour of hospitalization. Progression and/or recurrence of IS and metabolic and/or hemodynamic disorders are the main causes detected. Advanced age, initial impairment of alertness, hyperlycemia on admission and cardioembolic IS are its identified risk factors. ND significantly increases intra-hospital mortality and darkens the functional prognosis at the end of hospitalization. The early admission of patients, the implementation of SU and early revascularization therapies such as fibrinolysis and/or thrombectomy of IS, availability and increased accessibility of high-performance MRIs, in our hospitals, will contribute to a drastic reduction in the frequency and severity of ND in the acute phase of stroke through early diagnosis and management of the causes and risk factors of ND.

Conflict of Interest

The authors declare no conflict of interest.

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Personal.

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