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Research Article

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In-Hospital Acute Ischemic Stroke Case Fatality: Experience from a University Hospital in Bogota, Colombia (2018-2019).

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Abstract

Introduction

Until 2020, stroke was the second cause of death in Colombia and worldwide. In-hospital case fatality accounts for the majority of deaths. Multiple risk factors for in-hospital mortality have been described in the stroke population and their recognition could reduce stroke-related death.

Aims

To describe a single center experience of in-hospital acute ischemic stroke case fatality and its risk factors.

Methodology

Single center, retrospective, cross-sectional study between august 2018 and august 2019. Admitted consecutive patients with acute ischemic stroke brain infarct type were included. Admission sociodemographic, clinical, laboratory and imaging studies variables were drawn out. Quantitative variables were summarized as means, medians and interquartile ranges. Bivariate analysis was used to evaluate associations of in-hospital stroke case fatality and imputed variables.

Results

402 stroke brain infarct type patients were included, mean age 73,28 years (SD±14,53), 50,4% men. Prevalent vascular risks factors were hypertension (70,90%), diabetes mellitus type 2 (24,13%) and previous stroke (20,90%). There were 13 (3,2%) in-hospital case fatalities. Mean time from admission to death was 11.38 days (SD±10,8). Lower hemoglobin levels (p=0.002), lower total, LDL and HDL serum cholesterol levels (p=0.02), internal carotid artery stenosis >50% by duplex ultrasound (p=0.01) and stroke severity as measured by NIHSS score (p<0,0001) showed association with in-hospital stroke case fatality.

Conclusions

NIHSS score, hemoglobin level, and cholesterol (total, LDL, HDL) levels upon admission may be used by clinicians to make the appropriate early transfer decisions to facilities with the capacity to offer advance in-hospital stroke care. The cholesterol paradox in acute stroke fatality warrants further studies.

Key Words: Stroke, In-Hospital, Fatality, Risk Factors.

1. Introduction

Stroke is a leading cause of mortality and disability worldwide [1]. In 2016, 5.5 million of deaths, accounting for 16.8% of the global burden, were attributed to stroke [2]. Until 2020, it was the second cause of mortality after cardiovascular disease and it is the first among all causes of death by neurological disorders. If this tendency continues, by 2030, 12 million deaths from stroke can be expected. Although mortality rates are declining, with the aging of the population and the epidemiological transition from the infectious to non-communicable diseases, the absolute numbers in mortality from stroke are expected to rise in low and middle income countries [3]. Around 8-12% of ischemic stroke are fatal with the highest percentage in the first month after the event. Case fatality rate in the first 30 days after acute ischemic stroke range from 8% (Dijon, France 2002-04) to 49% (Tartu, Estonia 1970-73) depending of the study period and country [4,5].

In Colombian adult population stroke became the second cause of death after cardiovascular disease in 2017. It accounts for 13.000 to 15.000 deaths per year, representing 6.8% up to 7.4% of all-cause mortality (2000-2015). With the increase in population and the demographic transition the number of deaths from stroke went from 12.785 in 1995 to 14.739 among adults in 2015. In patients older than 75 years, it increased from 5,739 in 1995 to 8,825 in 2015. Roughly 65% of strokes are ischemic

with a reported in-hospital case fatality rate of 9.4% [6,7].

Worldwide in-hospital case fatality for acute ischemic stroke has been reported between 3-18%, being higher in women. Among the described predictors from in-hospital fatality after acute ischemic stroke are time to presentation to the hospital, day and time of admission, ethnic group, hyperthermia (>99F), low diastolic pressure, hypoxia (<94% oxygen saturation), stroke severity, high National Institute of Health Stroke Scale (NIHSS),previous modified Rankin scale >3, Glasgow Coma scale <8, hyperglycemia (>200mg/dl), total leukocyte count, high sensitive C-reactive protein >10mg/dl, anemia, hyponatremia, dyslipidemia, history of heart disease, presence of atrial fibrillation, socioeconomic status, premorbid conditions, hospital in-patients volume, availability of stroke unit, respiratory infections and dysphagia [6-10]. The main causes of in-hospital case fatality in patients with ischemic stroke are sepsis, herniation from stroke, basilar artery occlusion and heart disease [6,11]. Knowledge of the high incidence of early in-hospital stroke case fatality, risk factors and causes of death allows clinicians to implement preventive measures, indicate appropriate studies and direct resources in an efficient manner.

In correspondence with the above there is an increase interest in determining the risk factors associated with early in-hospital acute ischemic stroke case fatality. In line with this, the paper intends to present the experience of a university hospital in Bogota, Colombia, with early acute brain infarction case fatality after admission and its predictors.

2. Patients and Methods

2.1 Study Design

This is a one center, retrospective, cross-sectional study with review of hospital records through august 1st 2018 to august 1st 2019 in patients with acute ischemic stroke. The inclusion criteria comprised patients older than 18 years of age, admitted to the neurology service with a diagnosis of acute ischemic stroke, brain infarction type, confirmed by clinical and neuroimaging criteria (Computed Tomography (CT) brain scan and/or brain magnetic resonance imaging (MRI)) according to the WHO (World Health Organization) definition. The primary outcome was in-hospital stroke case fatality after admission. The institution is a 349-bed university hospital located at north-end of Bogota, Colombia´s capital city. It does not have a stroke unit or an exclusive neurology ward.

Sociodemographic variables, past medical history, admission clinical information, reperfusion interventions and diagnostic studies were drawn out. Sociodemographic data included age, gender and educational status. Past medical history included information relevant to vascular disease: previous stroke, past or active cancer, high blood pressure, diabetes mellitus, cigarette smoking, dyslipidemia, heart failure, atrial fibrillation, heart arrhythmias, coronary artery disease and coagulation disorders. Admission clinical data comprised blood pressure, body mass index (BMI), blood glucose, sodium, creatinine, hemoglobin, hematocrit, leucocyte count, hemoglobinA1C, total serum cholesterol, low density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, VDRL (Venereal Disease Research Laboratory) and NIHSS (National Institute of Health Stroke Scale) score.

Reperfusion interventions recorded were limited to intravenous thrombolysis. Diagnostic studies results included brain CT scan and/or MRI, with stroke site and presence and type of previous lesions imputed. Head and neck vascular studies (Duplex and/or CT angiography) were analyzed for the presence of dissection, atherosclerotic disease and grade of stenosis. Echocardiograms (transthoracic and/or trans esophageal) were analyzed for the presence and type of structural abnormalities and classified based on risk of cardio embolism: high risk (intramural thrombus), mild to moderate risk (apical aneurism, left auricular dilation, left ventricular/global , hypokinesia, patent foramen oval, moderate/severe valve dysfunction) and unknown risk of cardio embolism (left ventricular dysfunction, left ventricular concentric hypertrophy and mild valve dysfunction). Also, an analysis based on a classification of risk of adverse cardiac outcome was performed: high risk (left ventricular/global hypokinesia, left ventricular dysfunction, severe valve dysfunction) and no risk (apical aneurism, left auricular dilation, intramural thrombus, concentric hypertrophy, patent foramen oval, mild/moderate valve dysfunction and normal echocardiogram).

Cardiac rhythm monitoring studies were reviewed for any abnormality and the presence of atrial fibrillation. The TOAST (Trial of Org 10172 in Acute Stroke Treatment) classification for stroke mechanism of each patient was also extracted from the clinical charts [12]. The cause of death and time from admission for each case-fatality was analyzed and recorded.

The protocol was revised and approved by the institutional scientific and ethics committee. All authors vouch for the completeness and accuracy of the data and analysis and for the fidelity of the study to the protocol.

3. Statistical Analysis

All variables were reported as frequencies and percentages. Quantitative variables were summarized as means and standard deviation for normally distributed data. For not normally distributed variables the data were summarized as medians and interquartile ranges. Bivariate analysis to evaluate associations of in-hospital stroke case fatality and clinical and demographic variables was done using the Mann-Whitney test and Chi-square test. All tests are two-sided with the statistical significance level set to 0.05. Statistical analysis was performed using R Software [13].

4. Results

During the studied period 554 stroke patients were admitted to the hospital. Patients with intracranial hemorrhage (33) and transient ischemic attacks (119) were excluded. A total of 402 acute ischemic stroke brain infarction type patients were included. The median age was 73.28 years (SD±14,53), 50,4% were men. 37,24% had only elementary school, 35,2% high school and the remainder had a technical/university education level and above. Table 1shows a summary of the main sociodemographic, and past medical history characteristics. The most prevalent vascular risks factors were history of hypertension (70,90%), diabetes mellitus type 2 (24,13%) and previous stroke (20,90%).

The clinical variables at admission are shown in table 2. Echocardiography was performed in 91.04% (366) of the patients. The main findings in the diagnostic echocardiograms included mild valve dysfunction in 40.88% (166), left auricular dilation in 37.19% (151), concentric ventricular hypertrophy in 22.16 % (90), and left ventricular systolic dysfunction in 17.98% (73) of patients. Atrial fibrillation was diagnosed in 31.59% (127), in 13.18% (53) previous to admission and in 18.41% (74) during hospitalization. In the head and neck vascular ultrasound and/or CT angiography 12.8% (52) had evidence of atherosclerotic disease of the internal carotid artery with a stenosis of <50%, and 3.69% (13) with more than 50%.

	N	%	Missing
Sex			
Male	203	50,5	-
Female	199	49,5	-
Age	73,28 (±14,53) *	-	1
Body mass index	25,47 (±4,26) *	-	77
Past Medical History			
Previous stroke	84	20,90	2
Cancer	25	6,22	1
Hypertension	285	70,90	2
Diabetes mellitus type 2	97	24,13	0
Tobacco	42	10,45	0
Dyslipidemia	75	18,66	0
Heart failure	88	21,89	0

Table 1: Gender, Age, BMI and Past Medical History of the Studied Population.

	Mean /Median	Standard Deviation/ Quartile 25-75	Minimum	Maximum	Missing
Systolic BP (mmHg)	141	127-160	79	233	5
Diastolic BP (mmHg)	80	70-90	35	136	5
Glycemia (mg%)	110	96-136,5	3	553	191
	176	140,5-207	55	482	63
LDL cholesterol(mg%)	141	127-160	15	355	88
HDL cholesterol(mg%)	39	32-47	12	313	71
Sodium level(mEq/L)	137	135-139	123	163	26
Glycated Hemoglobin(%)	5,7	5,4-6,3	0,7	104	61
Hemoglobin level(%)	14,45	13,1-15,9	7,28	20,2	10
Leucocyte count (10x3mm3) Hematocrit	8,2 44,5	6,53-10,6 39,47-47,92	3,4 12,9	40,6 60,6	10 10
Creatinine level (mg%)	0,9	0,8-1,2	0,5	11,3	17
*Variables with normal distribution were presented with mean and SD and no normal distribution were presented with median QR(quartile range) 25-75. BP: Blood Pressure, LDL: Low Density Lipoprotein, HDL: High Density Lipoprotein.					

Table 2: Admission Blood Pressure and Laboratory Results of the Studied Population

Forty-seven patients (11.7%) were administered intravenous thrombolysis with recombinant tissue plasminogen activator (rtPA) at a dose of 0.9mg/kg.

Table 3 shows the main brain infarction characteristics. The most frequent location was non-lacunar stroke in the anterior vascular territory (47.51%). Based on NIHSS score, 72.14% of acute ischemic strokes were considered mild to moderate. According to the TOAST stroke classification the most common mechanisms was undetermined (41.04%) and cardioembolic (28.61%). The former included patients with more than one mechanism, insufficient work up and unknown mechanism.

	N (%)	Missing (%)
Stroke location *		
Anterior territory	191(47,51)	106 (26,37)
Posterior territory	82 (20,40)	
Lacunar stroke	24(5,97)	
Multiple stroke	63(15,67)	
NIHSS*		
Mild (0-5)	207 (51,49)	64(15,92)
Moderate (6-15)	83(20,65)	
Moderately severe (16-20)	21(5,22)	
Severe (21-42)	27(6,72)	
TOAST		
Large artery	19 (4,73)	51 (12,69)
Small artery	31(7,71)	
Cardio embolism	115 (28,61)	
Undetermined	165 (41,04)	
Other	21 (5,22)	
*Some patients presented more than one stroke location.		

Table 3: Stroke Characteristics of the Studied Population (Stroke Location, NIHSS and TOAST)

	N (%) of deaths	N (%) alive	P
Sex	**N=13**	**N=389**	**0.54**
Male	5 (38.46)	198(50.90)	
Female	8 (61.54)	191(49.10)	
Stroke location	**N=13**	**N=389**	
Anterior territory	9(69,23)	183(46,8)	0,45
Posterior territory	2(15,38)	80(20,46)	0,91
Lacunar stroke	0	24(6,13)	0,74
Multiple stroke	0	63(16,11)	0,23
Past Medical History	**N=13**	**N=389**	
Previous stroke	4(30,77)	80(20,67)	0,59
Cancer	0	25(6,44)	0,71
High blood pressure	11(84,61)	276(70,80)	0,44
Diabetes mellitus type 2	2(15,38)	95(24,42)	0,67
Tobacco	2(15,38)	40(10,28)	0,88
Dyslipidemia	4(30,77)	71(18,25)	0,43
Heart failure	5 (38,46)	306(78,66)	0,25
Atrial fibrillation	3(23,08)	50(12,99)	0,52
Echocardiogram*	**N=12**	**N=354**	
Normal	1(8,33)	46(12,96)	0,55
Classification based on risk of cardio embolism*			
	N=12	N=354	
High risk of cardio embolism Mild to moderate risk of cardio embolism Unknown risk of cardio Embolism	1(8,33) 6(50) 4(33,33)	8(2,25) 197(55,493) 104(29,29)	0,55 0,55 0,55
Classification risk of adverse cardiac outcome			
	N=12	N=354	
High risk of adverse cardiac outcome No risk of cardiac adverse Outcome	4(33,33) 8(61,53)	122(34,37) 233(59,59)	1 1
Carotid Duplex ultrasound	**N=4**	**N=132**	
Internal carotid artery stenosis <50%	2(66,67))	50(33,56)	0,56

Internal carotid artery stenosis >50%	2(66,67)	13(87,25)	0,01
Neck computerized tomography angiography*			
	N=3	N=156	
Internal carotid artery stenosi <50%	0	30(7,71)	0.61
Internal carotid artery stenosis >50%	1(7,70)	20(5,14)	1.0
NIHSS	N=12	N=326	**<0,0001**
Mild (0-5)	1 (8,33)	206(63,19)	
Moderate (6-15)	3 (25.0)	80(24,54)	
Mildly moderate (16-20)	3 (25,0)	18(5,52)	
Severe (21-42)	5 (41,67)	22(6,75)	
TOAST	N=11	N=340	0.44
Large artery	0	19(5,59)	
Small artery	0	31(9,12)	
Cardioembolism	6 (54,54)	109(32,06)	
Undetermined	4 (36,36)	161(47,35)	
Other	1 (9,10)	20(5,88)	
*Not all patients had information from these variables.			

Table 4: Bivariate Analysis for In-Hospital Case Fatality in Brain Infarct and Qualitative Clinical Variables.

For two cases of in-hospital stroke case fatalities data were missing for stroke location and echocardiographic results.

After hospital admission there were thirteen deaths registered. The in-hospital case fatality rate for acute ischemic stroke brain infarct type was 3.2%. The median time from admission to death were 11.38 days (SD±10,84). The cause of death was due to brain herniation in five patients, respiratory insufficiency in one, undetermined vascular cause in two, airway tract sepsis in two, cardiogenic shock in one, urinary tract sepsis in one and in one patient due to hydroelectrolitic disorder. The main clinical and paraclinical characteristics of the deceased are shown in table 4. In the bivariate analysis admission lower hemoglobin, total serum cholesterol, LDL cholesterol and HDL cholesterol levels were associated with in-hospital case fatality in patients with an acute brain infarction. (Tables 4 and 5). Internal carotid stenosis >50% measured by duplex ultrasound was also associated with case fatality. The highest association was with severe acute brain infarct as determined by a stratified NIHSS score (Mild 0-5, moderate 6-15, moderately severe 16-20 and severe >20) (Fig 1).

With regards to the TOAST stroke classification, 4 of the deceased were classified as undetermined and 6 as cardioembolic, with the result not statistically significant (P=0.44). Based on our classification on echocardiographic risk for cardioembolism and cardiac adverse outcome, there was no association with in-hospital stroke case fatality.

Mediana (RQ 25-75))			
	Death	**Alive**	**P**
Age BMI	79 (69-82) 24,75 (22,98-27,34)	76(65-83) 25,23 (22,58-28)	0,72 0,83
Systolic BP(mm Hg)	140 (116-160)	141(127-160)	0,81
Diastolic BP(mm Hg)	89 (60-94)	80 (70-90)	0,9
Laboratory			
Leucocyte count(10x3mm3)	8,34 (7,25-12,22)	8,2(6,5-10,51)	0,41
Hematocrit	39,05 (36,27-42,15)	44,1(39,7-48)	0,07
Sodium level (mEq/L)	136,5 (135-138)	137(135-139)	0,69
Hemoglobin level (%)	12,1 (11,77-13,25)	14,5(13,17-15,9)	0,002
Glycemia (mg%)	134 (125,5-146)	109(96-136)	0,17
Creatinine level(mg%)	1,05 (0,77-1,9)	0,9(0,8-1,2)	0,31
Total Cholesterol(mg%)	133,5(126,5-136,75)	177(143,5-208)	0,02
HDL cholesterol (mg%)	31,5(27,5-33,75)	39(32-48)	0,02
LDL cholesterol (mg%)	75(71,5-91)	108(77,5-136)	0,02
BP: Blood pressure, HDL: High Density Lipoprotein, LDL: Low Density Lipoprotein.			

Table 5: Bivariate Analysis for In Hospital Case Fatality in Brain Infarct and Quantitative Clinical Variables.

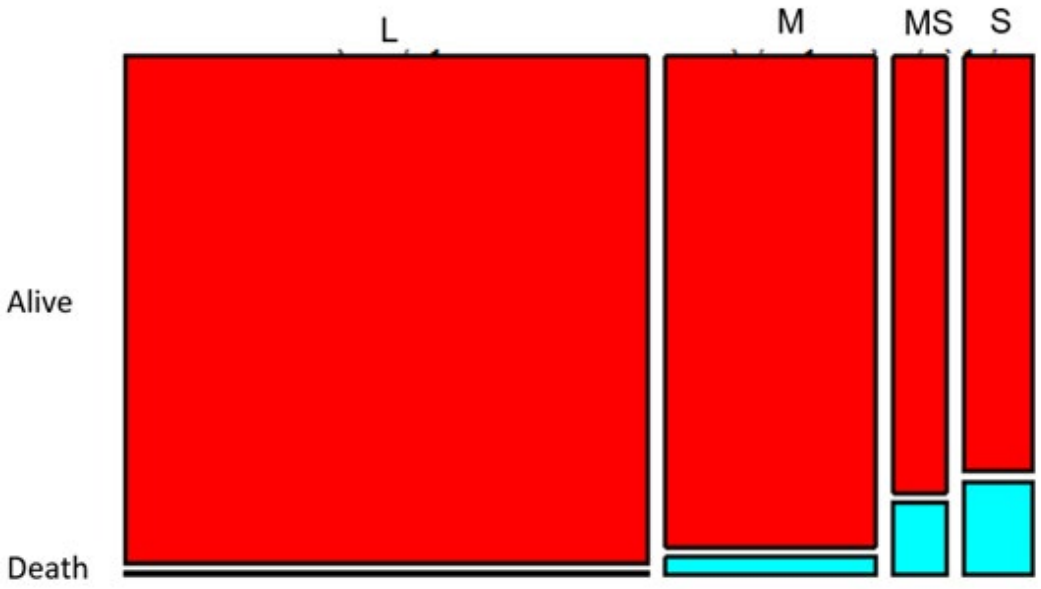

Figure 1: Distribution of NIHSS Categories in Live and Death Patients After Brain Infarct

NIHSS. L: mild (0-5), M: moderate (6-15), MS: moderately severe (16-20), S: severe (>20)
Each column represents the number of patients within each category of stroke severity. In each column the area in red represent the stroke survivors and the area in blue the stroke fatalities.

5. Discussion

This retrospective, single-center study evaluated in-hospital stroke (brain infarct type) mortality. In a one-year period (august 2018-august 2019), 402 consecutively acute ischemic stroke patients admitted at our university hospital were included. Although other studies in Colombia have presented stroke related mortality, this one includes a more comprehensive study of variables upon admission.

Stroke patient's characteristics were similar to other described populations around the world. Mean age of studied patients (73.28 years SD±14.53) are well within range of other described cohorts. From 61 years (SD ±24.6) in Tanzania (1) to 71.1(SD±13.5) in Germany. Stroke was equally distributed among men and women (50.4%:49.6%). The most prevalent vascular risk factor, high blood pressure found in 70.9%, is in the range of 30.7% to 86.1% described in other cohorts. Diabetes mellitus, found in 24.13% in our studied patients, is within the range described in other studies (10.9%-30.5%).

Our in-hospital case fatality rate for acute brain infarct (3.2%) was toward the lower end described in the literature. Similar to a rate in a large population study from Taiwan (3.1%)(6) but lower to rates described in Peru (13,6%) (5), Spain (7.13%) (7) , Germany (4.9%)(8) and Ethiopia (23,6%). And one third of the rate of 9.4% found in a previous study in Colombia. Although our hospital does not have a stroke unit and a strokologist on call, this low rate of in-hospital stroke case fatality may be attributed to less severe stroke as defined by NIHSS (51.49% mild strokes), dysphagia screening of all stroke patients and the hospital's policy on infection control and antithrombotic prophylaxis. Death, as commonly described, occurred within the first 30 days of hospital admission (11.38 days SD±10.84) and the causes were the previously found in the literature (sepsis, herniation and cardiovascular) [6] .

Eleven of the thirteen fatalities had comorbid clinical conditions, including high blood pressure (n=11), heart failure (n=5), previous stroke (n=4) and diabetes mellitus type 2 (n=2). But, contrary to other studies where heart disease(6) dyslipidemia (7),atrial fibrillation, diabetes mellitus and previous stroke (8) were associated with higher mortality rates, none of these comorbidities showed statistical significance. On bivariate analysis, only lower hemoglobin levels, lower total, LDL and HDL serum cholesterol levels, internal carotid artery stenosis >50% measured by duplex ultrasound and severe stroke measured by NIHSS were associated in-hospital stroke case fatality.

Lower hemoglobin levels showed a statistically significant association with fatality. Deceased patients had mean hemoglobin levels of 12,1 (11,77-13,25) mg% versus 14,5(13,17-15,9) mg% among survivors. Previous studies have shown an association between mortality in stroke patients and anemia. The ASTRAL stroke registry evidenced an OR 2,51(1,83-3,45) for stroke mortality and anemia (28). Fabjan et al described a statistically significant association between anemia and mortality in stroke patients ($p<0.001$) [10]. A meta-analysis showed an OR 1.39 (CI 95% 1.22-1.58) for short and long term stroke mortality and anemia It is known that mild anemia is a prognostic factor for all-cause mortality in elderly patients, probably in relation to chronic kidney disease or inflammatory state [12,11]. Anemia has been defined as a new vascular risk factor, specially for thrombotic disease [13]. Anemia is a hyperdinamic vascular state that alter endothelial adhesion molecule genes and may lead to thrombus formation [14]. Anemia stimulates production of erythropoietin and a proinflammatory state activating the coagulation cascade,

increasing the risk of infarction [15]. Anemia is risk factor for stroke and a prognostic marker for stroke mortality. In the latter, probably as a sign of chronic disease, malnutrition, and a clinical state of diminished response to infections and vascular injury [15]. Anemia or lower hemoglobin levels of stroke patients upon admission could be a modifiable factor to prevent in-hospital stroke fatality. Studies of anemia treatment and interventions to augment lower hemoglobin levels in acute brain infarct patients are probably warranted.

In our study we found that levels of total, LDL and HDL serum cholesterol were statistically significantly (p=0.02) lower in the in-hospital fatality group compared with the non-fatality group. Median total, LDL and HDL cholesterol serum levels in the fatality group were 133.5 mg/dl,75 mg/dl and 31.5mg/dl, respectively, compared to 177mg/dl, 108mg/dl and 39 mg/dl in the non-fatality group. This relationship was stronger in the cardioembolic stroke case fatalities. Studies recognize high total and LDL serum cholesterol levels as a risk factor for vascular disease. Guidelines recommend lipid lowering strategies for primary and secondary stroke prevention [16]. This cholesterol paradox for in-hospital acute stroke case fatality has been previously described. In a Japanese stroke study, the relative risk of mortality was 1.25 for low levels of cholesterol [17]. In a study from Slovenia, LDL cholesterol low levels showed an association with stroke fatality ($p<0.05$)(10). The explanations for this phenomenon (the cholesterol paradox) in acute ischemic stroke in-hospital case fatality can be multiple. It has been suggested that lower serum cholesterol levels are related with a decline in the general health status [18]. Also, lower levels of HDL and LDL serum cholesterol have been correlated with systemic inflammation [19,20]. And low total, LDL and HDL serum cholesterol levels are associated with lower capacity to buffer free oxygen radicals which may contribute to higher acute stroke fatality. The relation and pathophysiologic mechanisms of cholesterol levels and acute ischemic stroke outcomes deserve further research in different populations.

Two deceased patients showed stenosis of >50% of the internal carotid artery by duplex ultrasound. In the bivariate analysis this finding was statistically significant for in-hospital stroke case fatality (p=0.01). In a large population study of 274.998 stroke patients a stenosis >50% of internal carotid arteries was found to be significantly related to mortality (p>0.02) [9]. This finding may have different explanations including concomitant coronary disease, intracranial atherosclerotic lesions, larger strokes, higher burden of renal disease, older age, higher stroke recurrence and a chronic inflammatory state. But, the low number of patients with stenosis >50% of internal carotid arteries in both groups in our study precludes any firm conclusion.

Stroke severity measured by the NIHSS on admission had the strongest association with in-hospital case fatality ($p<0.0001$). This association between stroke severity and mortality is well described in literature and our findings are coherent with previous studies [6-8].

The study's strengths included the complete diagnostic work up of stroke patients, the thorough review of medical charts and the characteristics of the population. The patient's characteristics are those commonly found in general and university hospitals that treat stroke patients. Most of the analyzed information is easy to obtain, available on admission or within the first 24 to 48 hours. There are some limitations which include the retrospective cross-sectional nature of the study, a one center experience, variables not included (socioeconomic status, dysphagia screening, ethnic background, day and hour of admission, brain infarcted volume, hemorrhagic transformation, seizures, inflammatory biomarkers, endovascular thrombectomy, craniectomy, clot location and burden, collaterals among others), not accounting for changes in clinical and paraclinical status during hospitalization, low in-hospital stroke case fatality rate, and no information on pharmacological treatment given aside from intravenous thrombolysis.

6. Conclusion

Considering the strengths and limitation of the study, the NIHSS score, the hemoglobin level, and the cholesterol (total, LDL, HDL) levels upon admission may be used by clinicians to make the appropriate early transfer decisions to facilities with the capacity to offer advance in-hospital stroke care. More research is needed to understand the relation between low cholesterol levels and stroke fatality.

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