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Stress hyperglycemia and acute ischemic stroke in-hospital outcome

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ABSTRACT

Background and Aims. Stress hyperglycemia is frequent in patients with acute ischemic stroke. However, it is unclear whether stress hyperglycemia only reflects stroke severity or if it is directly associated with adverse outcome. We aimed to evaluate the prognostic significance of stress hyperglycemia in acute ischemic stroke.

Methods. We prospectively studied 790 consecutive patients who were admitted with acute ischemic stroke (41.0% males, age 79.4 ± 6.8 years). The severity of stroke was assessed at admission with the National Institutes of Health Stroke Scale (NIHSS). Stress hyperglycemia was defined as fasting serum glucose levels at the second day after admission ≥126 mg/dl in patients without type 2 diabetes mellitus (T2DM). The outcome was assessed with adverse outcome rates at discharge (modified Rankin scale between 2 and 6) and with in-hospital mortality.

Results. In the total study population, 8.6% had stress hyperglycemia. Patients with stress hyperglycemia had more severe stroke. Independent predictors of adverse outcome at discharge were age, prior ischemic stroke and NIHSS at admission whereas treatment with statins prior to stroke was associated with favorable outcome. When the NIHSS was removed from the multivariate model, independent predictors of adverse outcome were age, heart rate at admission, prior ischemic stroke, log-triglyceride (TG) levels and stress hyperglycemia, whereas treatment with statins prior to stroke was associated with favorable outcome. Independent predictors of in-hospital mortality were atrial fibrillation (AF), diastolic blood pressure (DBP), serum log-TG levels and NIHSS at admission. When the NIHSS was removed from the multivariate model, independent predictors of in-hospital mortality were age, AF, DBP, log-TG levels and stress hyperglycemia.

Conclusion. Stress hyperglycemia does not appear to be directly associated with the outcome of acute ischemic stroke. However, given that patients with stress hyperglycemia had higher prevalence of cardiovascular risk factors than patients with normoglycemia and that glucose tolerance was not evaluated, more studies are needed to validate our findings.

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1. Introduction

Ischemic stroke is a leading cause of death and long-term disability worldwide [1,2]. The presence of type 2 diabetes mellitus (T2DM) more than doubles the risk of ischemic stroke [3–6]. Furthermore, ischemic stroke is more severe in patients with T2DM and is also associated with poorer functional outcome and higher risk of death [7–9]. Moreover, preliminary data suggest that antidiabetic treatment might ameliorate stroke severity and improve the outcome of acute ischemic stroke [10–13].

In contrast with the established adverse effects of T2DM on stroke severity and outcome, it is unclear whether acute elevations in glucose levels during acute ischemic stroke adversely affect the outcome of non-diabetic patients. During acute ischemic stroke, stress stimulates the hypothalamus–pituitary–adrenal axis and the sympathetic nervous system leading to release of stress hormones, including cortisol and catecholamines, which increase glucose levels [4]. This phenomenon, coined stress hyperglycemia, is present in approximately 8–35% of non-diabetic patients with acute ischemic stroke [4,14,15]. Several studies reported worse outcomes in patients with acute ischemic stroke who exhibit stress hyperglycemia [15–17]. However, stress hyperglycemia is more frequent in patients with more severe stroke [18–21]. Therefore, it is unclear whether the association between stress hyperglycemia and stroke outcome is causal or due to a greater stroke severity in patients with stress hyperglycemia. Moreover, many studies did not differentiate between patients with stress hyperglycemia and those with established T2DM [22–25].

The aim of the present study was to evaluate whether stress hyperglycemia is associated with the functional outcome at discharge and the in-hospital mortality of non-diabetic patients with acute ischemic stroke.

2. Patients and Methods

We prospectively studied all patients who were admitted in our Department with acute ischemic stroke between September 2010 and March 2015 (n = 790; 41.0% males, age 79.4 ± 6.8 years).

At admission, demographic data (age, sex), history of cardiovascular risk factors [hypertension, T2DM, atrial fibrillation (AF), smoking, alcohol consumption, family history of cardiovascular disease (CVD)], history of concomitant CVD (coronary heart disease (CHD), previous ischemic stroke, heart failure) and pharmacological treatment were recorded. Smoking status and alcohol consumption were self-reported. Systolic and diastolic blood pressure (DBP) was measured at the Emergency Department. Anthropometric parameters (weight, height, waist and hip circumference) were also measured. The severity of stroke was assessed at admission with the National Institutes of Health Stroke Scale (NIHSS).

Routine laboratory investigations were performed after overnight fasting at the first day after admission and included serum levels of glucose, total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), triglycerides (TG), creatinine and uric acid. Low-density lipoprotein cholesterol (LDL-C) levels were calculated using Friedewald's formula [26]. Glomerular filtration rate (GFR) was estimated using the Modification of Diet in Renal Disease (MDRD) equation [27]. Chronic kidney disease was defined as estimated GFR (eGFR) < 60 ml/min/1.73 m^2.

T2DM was defined as a history of physician-diagnosed T2DM or treatment with antidiabetic agents. Stress hyperglycemia was defined as fasting serum glucose levels at the first day after admission ≥126 mg/dl in patients without T2DM.

All patients underwent brain computed tomography at admission and a second brain computed tomography was performed if clinically indicated.

The outcome was assessed with adverse outcome rates at discharge and with in-hospital mortality. Adverse outcome was defined as a modified Rankin scale at discharge between 2 and 6. Outcome data were collected during hospitalization.

2.1. Statistical Analysis

All data were analyzed with the statistical package SPSS (version 17.0; SPSS, Chicago, IL, USA). Data are presented as percentages for categorical variables and as mean and standard deviation for continuous variables. Serum TG levels were log-transformed to normalize their distribution. Differences in categorical variables between groups were assessed with the chi-square test. Differences in continuous variables between groups were assessed with one-way analysis of variance and pair-wise post-hoc comparisons between groups were performed with the Holm–Sidak test. Binary logistic regression analysis was used to identify independent predictors of adverse outcome at discharge and of in-hospital mortality. In all cases, a two-tailed $p < 0.05$ was considered significant.

3. Results

At admission, 32.0% of the total study population had T2DM. At the second day after admission, 8.6% of the total study population had stress hyperglycemia (i.e. 12.7% of the non-diabetic patients). Characteristics of patients with stress hyperglycemia, T2DM and neither stress hyperglycemia nor T2DM are shown in Table 1. Patients with stress hyperglycemia were older than patients with T2DM, had higher HDL-C levels, were less likely to receive statins and antiplatelet agents prior to stroke and had more severe stroke. Patients with stress hyperglycemia also had higher prevalence of AF, higher heart rate and more severe stroke than patients with neither stress hyperglycemia nor T2DM. On the other hand, patients with T2DM had higher body mass index and waist circumference, higher prevalence of CHD and heart failure, lower HDL-C levels and higher TG levels than patients with neither stress hyperglycemia nor T2DM, but had lower LDL-C levels, were more likely to receive statins, antiplatelet and antihypertensive agents prior to stroke and had similar stroke severity compared with the former.

At discharge, 64.3% of patients had adverse outcome. Average discharge time was 6.5 ± 3.9 days. Patients with stress hyperglycemia had higher rates of adverse outcome than patients with T2DM and patients with neither stress

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