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The effect of metabolic syndrome and obesity on outcomes of acute ischemic stroke patients treated with systemic thrombolysis☆

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ABSTRACT

Metabolic syndrome (MetS) is associated with increased risk of ischemic stroke; while central obesity has controversial effects on ischemic stroke. We investigated effects of MetS and obesity on clinical courses and outcomes of patients treated with intravenous recombinant tissue-type plasminogen activator (iv rt-PA). 319 patients treated with intravenous thrombolysis were included to our study. Metabolic syndrome was determined if ≥3 of following criteria are present: elevated waist circumference; elevated triglycerides; reduced high density-lipoprotein cholesterol (HDL-C); elevated blood pressure; elevated fasting glucose. Obesity was defined as BMI ≥ 30. Clinical features at baseline, 24th hour and 3rd month were examined. Computed tomography (CT) findings for ASPECT scores and hemorrhagic transformation were analyzed. 182 patients were MetS +; they were older ($p = 0.035$), had similar ASPECT scores ($p = 0.477$) and NIHSS scores ($p = 0.167$) at admission; had significantly higher NIHSS scores at 24th hour ($p < 0.001$) and worse outcome at 3rd month ($p < 0.001$). Logistic regression analysis showed that either MetS, obesity or age were not independent predictors of poor outcome. Obese patients (n:72) had slight but significantly lower NIHSS scores at admission ($p = 0.049$) compared to non-obese patients; meanwhile there was no significant difference between NIHSS scores at 24th hour ($p = 0.736$) and 3rd month mRS scores ($p = 0.145$). Hemorrhagic transformation and mortality rates were not affected with MetS or obesity. MetS is not an independent factor on clinical outcome but its presence may have a relationship with poor outcome; but obesity was not found to have any significant role on clinical course and outcome of patients treated with iv rt-PA.

1. Introduction

The metabolic syndrome (MetS) is a complex disorder comprising vascular risk factors; dysglycemia, raised blood pressure, elevated triglyceride levels, low high density-lipoprotein cholesterol (HDL-C) levels and central obesity [1]. Inflammatory state and coagulation system activation seen in MetS constitute a risk for thromboischemic events [2]. MetS has been found to increase the risk of cardiovascular diseases and ischemic stroke [3–5].

Even though the central obesity is a component of MetS, obesity and stroke relation has not been clearly identified yet. Obesity was shown to be associated with good outcome and reduction in mortality in stroke, known as “obesity paradox” [6–8], and hemorrhagic transformation occurrence was found to be decreased with obesity [9]; while some other studies showed no significant favorable effect of obesity on mortality [10]. There are challenging studies concerning intravenous thrombolysis in stroke and obesity; that obesity can be an predictor of unfavorable outcome [11] versus no effect on outcome or symptomatic intracerebral hemorrhage (sICH) [12].

In our study we aimed to evaluate the individual and intimate effects of MetS and obesity on clinical presentations, courses and outcomes of patients treated with intravenous recombinant tissue-type plasminogen activator (iv rt-PA).

2. Materials and methods

Consecutive patients with acute ischemic stroke treated with only intravenous recombinant tissue-type plasminogen activator (iv rt-PA) within 4.5 h of symptom onset at our hospital between November/2008 and April/2015. Patients receiving further intra-arterial thrombolysis or mechanical thrombectomy were not included in this study. Patient data at baseline including demographics, stroke risk factors, pre-stroke medications, admission blood pressure and plasma glucose levels and chronology (time of symptom onset, baseline CT and administration of

☆ We declare that this study has been approved by the local ethics committee (Eskisehir Osmangazi University) and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments.

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iv rtPA) were documented. The local ethics committee approved the study protocol.

Patients were evaluated for metabolic syndrome based on presence of ≥ 3 of following criteria: elevated waist circumference (≥ 94 cm for male; ≥ 80 cm for female); elevated triglycerides (≥ 150 mg/dL) (drug treatment for elevated triglycerides is an alternate indicator); reduced HDL-C (< 40 mg/dL in males and < 50 mg/dL in females) (drug treatment for reduced HDL-C is an alternate indicator); elevated blood pressure (≥ 130 mm Hg and/or diastolic ≥ 85 mm Hg) (antihypertensive drug treatment in a patient with a history of hypertension is an alternate indicator); elevated fasting glucose (≥ 100 mg/dL) (drug treatment of elevated glucose is an alternate indicator) [1]. Serum fasting glucose, HDL-C and triglyceride levels measured at the third day of admission. Elevated blood pressure level criteria was evaluated at least 24th hour after the symptom onset.

Body mass index was calculated in kg/m^2; patients' weight (kg) and height (m) data were obtained either by measuring or from patients themselves or their relatives. Obesity was defined as BMI ≥ 30 and BMI < 30 defined as non-obese [13].

Neurological assessments were made using the National Institutes of Health Stroke Scale (NIHSS) scores at the time of presentation, 24 h later and at the time of discharge. A NIHSS score of ≥ 10 was defined as severe neurological deficit. At 24 h, ≥ 4 points of remission on the NIHSS was defined as good response, and a NIHSS of 0–1 or ≥ 8 points of remission on the NIHSS was defined as a dramatic neurological improvement. Clinical outcome at 90 days was evaluated using a modified Rankin Scale (mRS) score where 3–6 was defined as a poor outcome, 0–2 was defined as a favorable outcome and mRS score 0–1 as favorable functional recovery. Stroke etiology was classified according to the Trial of Org 10,172 in Acute Stroke Treatment (TOAST) trial criteria after diagnostic tests were completed [14].

Alberta Stroke Program Early CT Score Study (ASPECTS) scala was used for scoring early ischemic changes in baseline CT scans. Follow up CT scans at 24 h were evaluated for infarct area; and hemorrhagic transformation (HT) graded as hemorrhagic infarction (HI) or parenchymal hemorrhage (PH) based upon the European-Australasian Acute Stroke Study (ECASS) classification [15]. Symptomatic hemorrhage was defined using Safe Implementation of Thrombolysis in Stroke Monitoring Study (SITS-MOST) as local or remote PH2 at the 22 to 36 h CT scan combined with a neurological deterioration of ≥ 4 points on the NIHSS from baseline or leading to death [16].

Statistical analyses were performed using Statistical Package for the Social Sciences (SPSS) version 21. For comparison of categorical variables, the x^2-test was performed. For non-categorical variables, the Mann-Whitney *U* Test was performed. For analyses between three groups, the Independent-Samples Kruskal-Wallis Test was performed, and for risk identification, logistic regression was performed. We initially performed univariate analysis to detect the clinical; laboratory and demographic parameters affecting poor outcome (mRS 3–6); logistic regression analysis was used for multivariate analysis with stepwise enter model. The goodness-of-fit of models was evaluated with Hosmer and Lemeshow test x^2 test.

3. Results

319 patients (mean age 65 ± 11; 56.4% male) were included to this study. 182 patients (57.1%) have been diagnosed metabolic syndrome (mean age 66 ± 10; 48.4% male). Demographic features and clinical properties of patients were shown in Table 1.

MetS + patients were significantly older ($p = 0.035$) and had higher female ratio ($p = 0.001$) than MetS − patients. There was no difference between their admission ASPECT scores ($p = 0.477$), and presentation to treatment times ($p = 0.411$). Their admission NIHSS scores were similar ($p = 0.167$). There was no difference in stroke etiology frequencies between groups ($p = 0.385$).

MetS + patients had significantly higher NIHSS scores at 24th hour compared to MetS − patients ($p < 0.001$). There was no difference in symptomatic ($p = 0.251$) or asymptomatic ($p = 0.317$) hemorrhage prevalances. In multivariate logistic regression analysis we found that only diabetes mellitus ($p = 0.006$) and admission NIHSS score ($p = 0.003$) are independent predictors of early clinical response (i.e. at 24th hour NIHSS score = 0–1 or ≥ 8 points decrease in admission NIHSS score). Metabolic syndrome was not found to be an independent predictor. Clinical progressions and outcomes were shown in Table 2.

At 3rd month MetS − patients had better outcomes ($p < 0.001$) than MetS + patients. Mortality rates were not different between MetS + and MetS − groups ($p = 0.417$). MetS − patients significantly more frequently achieved favorable functional recovery (mRS 0–1) than MetS + patients ($p = 0.003$).

Obesity was found in 72 patients. In comparison of obese and non-obese group; obese patients had significantly lower admission NIHSS scores ($p = 0.049$); but severe neurologic deficit (NIHSS score ≥ 10) rates were similar between groups ($p = 0.318$). Obesity did not affect the NIHSS scores at 24th hour ($p = 0.736$); good outcome ($p = 0.145$), symptomatic hemorrhage ($p = 0.588$), asymptomatic hemorrhage ($p = 0.236$) or mortality ($p = 0.337$).

We made subgroup analysis in MetS + patients based on obesity; obese MetS + patients presented with lower NIHSS scores compared to non-obese MetS + patients ($p = 0.025$). NIHSS scores at 24th hour

Table 1
Demographic and clinical characteristics at presentation of groups.

	MetS + (n:182)	MetS − (n:137)	p-Value
Age, years (mean ± SD)	66.4 ± 9.6	63.1 ± 12.34	*0.035*
Male (n)	88 (48.4%)	92 (67.2%)	*0.001*
Admission NIHSS score (median)	16 (11–19)	15 (11–18)	*0.167*
Admission NIHSS score ≥ 10 (n)	158 (86.8%)	117 (85.4%)	*0.843*
Admission ASPECT score (median)	9	9	*0477*
Admission serum glucose (mg/dL) (median)	145.5 (113.8–187.0)	115.0 (100.0–140.0)	*< 0.001*
Admission BP > 185/110 mm Hg (n)	72	26	*< 0.001*
Diabetes mellitus (n)	89 (48.9%)	9 (6.6%)	*< 0.001*
Hypertension (n)	157 (86.3%)	50 (36.5%)	*< 0.001*
Hyperlipidemia (n)	106 (59.2%)	57 (42.5%)	*0.005*
Atrial fibrillation (n)	69 (39.2%)	46 (34.6%)	*0.476*
Previous stroke (n)	23 (12.8%)	18 (13.1%)	*0.924*
Stroke subtypes (n)			
Atherosclerosis	28 (15.4%)	30 (21.9%)	*0.385*
Cardioembolism	103 (56.6%)	76 (55.5%)	
Small vessel disease	10 (5.5%)	3 (2.2%)	
Other causes	5 (2.7%)	4 (2.9%)	
Cryptogenic	36 (19.8%)	24 (17.5%)	

Where indicated data are presented as mean ± SD, median (Q1 – Q3), or n.
NIHSS: National Institutes of Health Stroke Scale.

Table 2
Clinical course and outcome features of groups.

	MetS + (n:182)	MetS − (n:137)	p-Value
NIHSS score at 24 h (median)	10 (3.5–17)	5 (2 – 12)	*< 0.001*
Dramatic neurological improvement at 24 h (n)	71 (39%)	76 (55.5%)	*0.003*
Asymptomatic hemorrhage (n)	37 (20.3%)	21 (15.3%)	*0.317*
Symptomatic hemorrhage (n)	15 (8.2%)	6 (4.4%)	*0.251*
mRS score at day 90 (median)	3 (1–5)	1 (0–4)	< 0.001
Favorable outcome (mRS 0–2) (n)	73 (40.1%)	84 (61.3%)	*< 0.001*
Poor outcome (mRS 3–6) (n)	109 (59.9%)	53 (38.7%)	*< 0.001*
Mortality (n)	34 (18.7%)	20 (14.6%)	*0.417*

Where indicated data are presented as mean ± SD, median (Q1 – Q3), or n.
NIHSS: National Institutes of Health Stroke Scale; mRS: modified Rankin Scale.

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