Lower Serum Triglyceride Level Is Associated With Increased Stroke Severity

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Background and Purpose—A previous study showed that low triglyceride concentration predicts higher mortality after stroke. The aim of our study was to determine whether serum triglyceride level is associated with stroke severity on admission.

Methods—863 consecutive patients with acute ischemic stroke were included. Serum triglyceride level was measured within 36 hours after stroke onset. Stroke severity on admission was assessed using Scandinavian Stroke Scale (SSS). The patients were divided into 2 groups: those with severe stroke (SSS ≤25) and those with mild/moderate stroke (SSS >25).

Results—Patients with severe stroke had significantly lower serum triglyceride level than patients with mild/moderate stroke (1.4±0.6 versus 1.7±1.3 mmol/L). After adjusting for age, sex, atrial fibrillation, diabetes mellitus, obesity, and ischemic heart disease, patients with triglyceride ≥2.3 mmol/L had lower risk of severe stroke than those with triglyceride ≤2.3 mmol/L (OR: 0.58; 95% CI: 0.35 to 0.95).

Conclusions—Our results suggest that lower level of triglyceride is associated with the more severe stroke. (Stroke. 2004; 35:e151-e152.)

Key Words: stroke ■ cholesterol

The results of 2 independent studies suggested an association between poor outcome and lower serum total cholesterol (TC) level.1,2 Recently, Weir et al showed that low triglyceride (TG), not low TC concentration, independently predicts poor outcome after acute stroke.3

The aim of our study was to investigate if serum TG level predicts stroke severity on admission.

Materials and Methods

We analyzed retrospectively our stroke database. Patients in this study were recruited from 955 consecutive patients with first-ever ischemic stroke admitted to our stroke unit between May 2000 and April 2003. Patients admitted to the hospital >24 hours after stroke onset were excluded from the study.

Arterial hypertension was diagnosed when its presence was documented in medical records or when at least 2 readings of blood pressure were ≥140 mm Hg (systolic) or ≥90 mm Hg (diastolic) after the acute phase of stroke. Ischemic heart disease was diagnosed when there was a history of angina pectoris or myocardial infarction. Diabetes mellitus was diagnosed if its presence was documented in medical records or the patient was taking insulin or oral hypoglycemic agents. A patient was defined as a smoker if there was a history of cigarette smoking during the past 5 years. Hypercholesterolemia was diagnosed if TC >6.2 mmol/L and hypertriglyceridemia was diagnosed if TG >2.3 mmol/L. Abdominal obesity was diagnosed if a waist circumference was >102 cm in men and >88 cm in women.

All patients underwent head CT scan within 24 hours after stroke onset.

Stoke severity on admission was assessed using Scandinavian Stroke Scale (SSS).4 The patients were divided into 2 groups: those with severe stroke (SSS ≤25) and those with mild/moderate stroke (SSS >25).

Fasting serum TC and TG levels were measured between 12 and 36 hours after stroke onset using commercially available enzymatic kits (Boehringer Mannheim).

The χ² test was used to compare proportions and Student t test to compare continuous variables between groups. Logistic regression analysis was used to assess the independent contribution of variables statistically significant on univariate analysis in the prediction of stroke severity. Backward logistic regression including only variables with P<0.1 was followed by a forward logistic regression including the same variables. Odds ratios (OR) were calculated for severe stroke. Mild/moderate stroke was coded as 0 and severe stroke was coded as 1. Values of P<0.05 were considered statistically significant.

Results

From 955 patients with first-ever ischemic stroke, 863 patients (mean age: 68.3±13.0; 49.1% men) were included into the study. We excluded 92 patients who were admitted to the hospital >24 hours after stroke onset.

The characteristics of patients with severe stroke and those with mild/moderate stroke are shown in Table.

Patients with severe stroke were significantly older, more frequently had ischemic heart disease and atrial fibrillation, and had significantly lower serum TG level.
The following variables were put into logistic regression model: age (continuous variable), sex, ischemic heart disease, atrial fibrillation, diabetes mellitus, obesity, and TG level (continuous variable). On logistic regression analysis age (OR: 1.01; 95% CI: 1.00 to 1.03), atrial fibrillation (OR: 1.80; 95% CI: 1.26 to 2.58), and TG level (OR: 0.69; 95% CI: 0.55 to 0.87) independently predicted severe stroke. In the next model, we used dichotomized variables: age (65 years or older versus younger than 65 years), sex, ischemic heart disease, atrial fibrillation, diabetes mellitus, obesity, and TG level (>2.3 versus ≤2.3). In that model, atrial fibrillation (OR: 1.87; 95% CI: 1.28 to 2.71) and TG level (OR: 0.58; 95% CI: 0.35 to 0.95) independently predicted severe stroke. Finally, TC level (>6.2 versus ≤6.2) was added to the model, but it did not change significantly the results of analysis.

Three-month mortality rates did not differ between patients with TG >2.3 mmol/L and those with TG ≤2.3 mmol/L (10.9% and 13.5%, respectively; P = 0.53).

Discussion

In contrast to TC, serum TG level does not change in acute stroke and its measurement within first 48 hours seems to be a good reflection of usual TG concentrations in individual patients.5–7

Previous study showed that low TG concentration strongly and independently predicts higher mortality 6 months after stroke.3 Results of our study suggest that lower TG level is associated with more severe stroke as measured by SSS on admission. We did not find a significant difference in 3-month mortality between patients with TG >2.3 mmol/L and those with TG ≤2.3 mmol/L.

The potential biological mechanism responsible for association between TG level and stroke severity is unknown. Low TG level can reflect poor nutritional status. Although malnutrition after acute stroke is a risk factor for poor outcome,6 it does not explain stroke severity on admission. Therefore, we believe that alternative explanations focusing on potentially neuroprotective properties of cholesterol should be considered. It was speculated that high cholesterol may be protective through increasing gamma-glutamyltransferase. This enzyme plays a role in amino acid uptake and transport and could reduce the neurotoxic effects of amino acids.9 Cholesterol can also provide antioxidant protection.10 In the experimental model of myocardial ischemia, mice fed the high-cholesterol diet for 12 weeks showed a significantly lower area of myocardial infarction compared with mice fed a normal diet.11

The shortcomings of our study should be recognized. First, our study has all limitations of retrospective study. Second, TG level can be affected by fasting. The patients more severely affected by stroke may have been less likely to eat after stroke onset and therefore may have had lower TG level. However, Weir et al examined the effect of time from stroke onset on TG concentration and did not find any substantial difference between quartiles of TG concentration.3 Third, although existing data did not show significant changes in TG concentrations during first days after stroke, we cannot exclude that acute phase reaction accompanying stroke can at some degree influence TG level. Taking into account that TG level was measured in 85% of our patients within 24 hours after stroke onset, it seems unlikely that small changes in TC could significantly influence our results.

In summary, our results suggest that lower level of TG is associated with the more severe stroke.

References

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