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Lower Fasting Serum Triglyceride Levels on Admission are Associated with Increased Risks of 30-Day and 1-Year Mortality in Patients with Ischemic Stroke

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Abstract

Objective: This study was to assess the 30-day and 1-year mortality associated with serum triglyceride levels on admission for patients with acute ischemic stroke using the Taiwan Stroke registry (TSR) data.

Patients and methods: From the TSR database, we identified 45931 acute ischemic stroke patients from April 2006 to April 2014 and stratified them into 3 groups by the serum triglyceride level measured on admission: <150 mg/dL, 150 mg/dL -199 mg/dL, and ≥ 200 mg/dL. They were prospectively followed to measure the 30-day and 1-year mortality after acute ischemic stroke.

Result: The mortality rate after acute ischemic stroke decreased as the triglyceride level increased. The 30-day mortality rate was 2.3-fold greater in patients with triglyceride levels <150 mg/dL than patients with triglyceride levels >200 mg/dL (1.62 vs.0.72 per 1000 person-days), with an adjusted hazard ratio (HR) of 1.25 (95% confidence interval (CI)=1.06-1.46). The 1-year mortality rate was also greater in patients with lower triglyceride levels than in patients with higher levels (0.47 vs. 0.22 per 1000 person-days), with an adjusted HR of 1.21 (95% CI=1.09-1.34). Compared to patients with triglyceride levels >200 mg/dL, those with triglyceride levels <150 mg/dL had 2.2% lower 30-day survival rate and 6.3% lower 1-year survival rate after acute ischemic stroke.

Conclusion: Acute ischemic stroke patients with lower fasting serum triglyceride levels on admission are at a higher risk of both 30-day mortality and 1-year mortality.

Introduction

Stroke is the third leading cause of death after heart disease and cancer, and the major cause of serious long-term disability in the Unites States [1]. This disorder has a huge impact on the quality of life, productivity, and the cost of health care. It is crucial to identify risk factors associated with deaths from stroke. Identifying patients at a higher risk might help medical staffs to care these patients. We found previously that cholesterol levels and renal function are able to predict short-term and long-term mortality [2,3]. Ischemic stroke patients with an estimated glomerular filtration rate (GFR) <15 mL/min/1.73 m^2 are over 5-fold more likely to die within one month than those with a GFR ≥ 90 mL/min/1.73 m^2 [3]. Dialysis patients with the serum total cholesterol levels of ≥ 160 mg/dl or <120 mg/dl on admission for ischemic stroke are also at an elevated risk of death within one month [2].

Some observational studies have identified hypertriglyceridemia as a risk factor for ischemic stroke [4,5]. However, studies also found that low serum triglyceride levels measured in the early period after stroke are predictive of higher infarct volume [6], more severity [7,8], and both elevated short-term mortality and long-term mortality [9-13]. On the other hand, a Korean study found a J-shape relation between triglyceride levels and early neurological deterioration and mortality [14]. Despite of inconsistent results, these findings suggest that serum triglyceride levels might be associated with the prognosis of acute ischemic stroke.

The Taiwan Stroke Registry (TSR) is a government-funded program documenting medical information for acute stroke patients admitted at 59 major hospitals in Taiwan [15]. The aim of this study was to investigate the association between fasting serum triglyceride levels and 30-day and 1-year mortality rates after ischemic stroke using the TSR data.

Patients and Methods

Data source

The Taiwan Stroke Registry (TSR) program was established in 2006 with the participation of 59 academic medical centers and community hospitals. Details of the program have been described elsewhere [15]. Information collected for each patient included preadmission data, inpatient data, discharge information, and follow-up information assessed 1, 3, 6 and 12 months after stroke. This study used the medical data collected from April 2006 to April 2014 consisting of 91300 patients with acute stroke registered on the TSR program.

Study subjects

Figure 1 shows the process to identify study population. We identified 45931 ischemic stroke patients for this study after excluding patients with following characteristics: (1) haemorrhagic stroke or missing stroke type (N=24682); (2) missing triglyceride laboratory data (N=7778); (3) less than 18 years (N=285); (4) patients without information of the Trial of Org 10172 in Acute Stroke Treatment (TOAST) criteria (N=3258); (5) missing information on body mass index (BMI), systolic blood pressure, haemoglobin levels, or serum creatinine levels (N=9160); (6) patients without dialysis status (N=206). The 30-day and 1-year mortality rates after acute ischemic stroke were analysed by fasting serum triglyceride levels on admission to hospital.
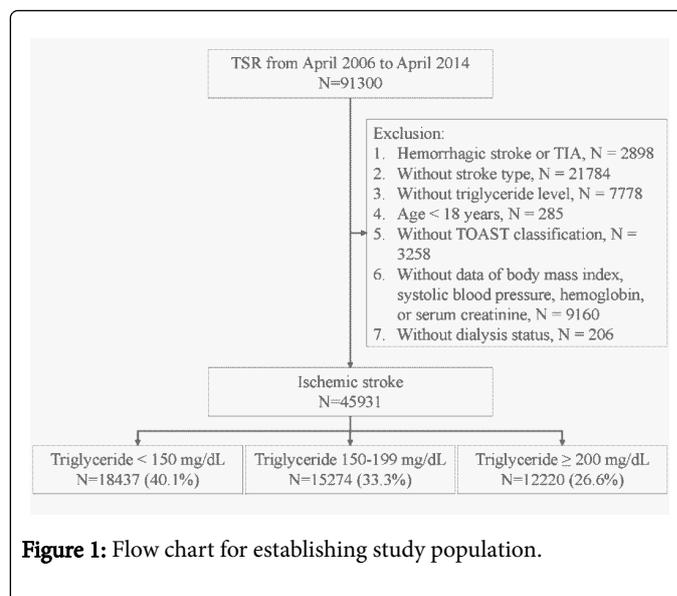

Figure 1: Flow chart for establishing study population.

Statistical analysis

Acute ischemic stroke patients were stratified into 3 groups by the serum triglyceride levels: <150 mg/dL, 150-199 mg/dL, and ≥ 200 mg/dL. Distributions of demographic status, comorbidities and treatment were expressed as frequencies and percentages for categorical variables and were compared among the three groups. Median, 25th percentile and 75th percentile values were estimated for continuous variables. The Chi-square and Mann-Whitney U tests were used to test differences for category variables and continuous variables, respectively, among the three groups. Patients were followed prospectively until death or were censored for loss to follow-up. The 1-month and 1-year mortality rates were calculated. The Cox hazards regression analysis was used to assess the hazard ratios (HRs) for mortality and 95% confidence intervals (CI). Adjusted HR was calculated after controlling for age, gender, TOAST, location, body mass index, smoking, atrial fibrillation, previous stroke history, ischemic heart disease, congestive heart failure, diabetes mellitus, systolic blood pressure, haemoglobin, estimated glomerular filtration rate (eGFR) levels, National Institutes of Health Stroke Scale (NIHSS) score on admission and medicine use. The eGFR was calculated using the CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration) equation [16]. All statistical analyses were performed by using SAS software (version 9.4 for Windows; SAS Institute, Cary, NC). A two-tailed p-value below 0.05 was considered as significant.

Results

The median age was 69.6 years in our study population, with 61.2% patients were men. Among the 45931 ischemic stroke cases, 40.1% had triglyceride <150 mg/dL and 26.6% had triglyceride ≥ 200 mg/dL (Table 1). Patients were prevalent with small vessel occlusion (40.8%), and the prevalence increased with the triglyceride level. Compared with patients with higher triglyceride, patients with triglyceride <150 mg/dL were older, and more prevalent with anterior circulation stroke, low BMI, heart disorders, hypertension, low HB, higher NIHSS, low eGFR and medication use before admission, but were less prevalent with diabetes.

	Triglyceride level (mg/dl)								p-value
	Total		<150		150-199		≥ 200		
	N=45931	N=18437 (40.1%)		N=15274 (33.3%)		N=12220 (26.6%)			
Variable	n	%	n	%	n	%	n	%	
Men	28105	61.2	11581	62.8	9605	42.9	6919	56.6	<0.0001
Age, year									<0.0001
<60	12204	26.6	3869	21	4092	26.8	4243	34.7	
60-69	11351	24.7	4156	22.5	3881	25.4	3314	27.1	
70-79	13520	29.4	5699	30.9	4636	30.4	3185	26.1	
≥ 80	8856	19.3	4713	25.6	2665	17.5	1478	12.1	
Median (Q1, Q3)	69.6	(59.3, 77.9)	72.3	(61.9, 80.2)	69.3	(59.2, 77.4)	65.9	(56.7, 74.5)	
TOAST									<0.0001
Large artery atherosclerosis	13263	28.9	4977	27	4507	29.5	3779	30.9	
Small vessel occlusion	18727	40.8	6828	37	6281	41.1	5618	46	
Cardioembolism	730	1.59	372	2.02	226	1.48	132	1.08	
Determined and undetermined etiology	13211	28.8	6260	34	4260	27.9	2691	22.02	
Location									<0.0001
No significant finding	8105	17.7	2686	14.6	3137	20.5	2282	18.7	
Anterior circulation	22327	48.6	9542	51.8	7175	47	5610	45.9	
Posterior circulation	11159	24.3	4146	22.5	3680	24.1	3333	27.3	
Both	2471	5.38	1143	6.2	762	4.99	566	4.63	
Others	1869	4.07	920	4.99	520	3.4	429	3.51	
BMI									<0.0001
≤ 20	4883	10.6	2590	14.1	1454	9.52	839	6.87	
>20, ≤ 24	16152	35.2	4943	37.7	5325	34.9	3884	31.8	
>24	24896	54.2	8904	48.3	8495	55.6	7497	61.4	
Median (Q1, Q3)	24.3	(22.0, 26.9)	23.9	(21.5, 26.4)	24.5	(22.2, 27.1)	25	(22.7, 27.5)	
Smoking									<0.0001
Current	11192	24.4	3759	20.4	4110	26.9	3323	27.2	
Past	5355	11.7	2751	14.9	1496	9.73	1118	9.15	
AF	3027	6.59	-	-	-	-	-	-	
Previous stroke history	11461	25	4982	27	3759	24.6	2720	22.3	<0.0001
Ischemic heart disease	5729	12.5	2516	13.7	1941	12.7	1272	10.4	<0.0001
Congestive heart failure	932	2.03	707	3.83	104	0.68	121	0.99	<0.0001
Diabetes mellitus	17826	38.8	6579	35.7	5831	38.2	5416	44.3	<0.0001
Systolic blood pressure, mmHg									<0.0001

<120	3518	7.66	1683	9.13.	1168	7.65	667	5.46	
120-139	7859	17.1	3509	19	2586	16.9	1764	14.4	
140-159	12386	27	5120	27.8	4168	27.3	3098	25.4	
160-179	10356	22.6	3885	21.1	3532	23.1	2939	24.1	
180-199	6652	14.5	2491	13.5	2171	14.2	1990	16.3	
≥ 200	5160	11.2	1749	9.49	1649	10.8	1762	14.4	
Median (Q1, Q3)	158	(140, 180)	155	(137, 177)	158	(140, 180)	163	(144, 185)	
Hb, g/dL									<0.0001
<10	2272	4.95	1290	7	623	4.08	359	2.94	
10-Nov	6075	13.2	2895	15.7	1924	12.6	1256	10.3	
Dec-13	15684	34.2	6403	34..7	5346	35	3935	32.2	
14-15	16380	35.7	6056	32.9	5525	36.2	4799	39.3	
≥ 16	5520	12	1793	9.73	1856	12.2	1871	15.3	
Median (Q1, Q3)	13.8	(12.5, 15.1)	13.6	(12.1, 14.9)	13.9	(12.6, 15.1)	14.2	(12.9, 15.3)	
NIHSS score at admission									<0.0001
≤ 5	27737	60.4	10090	54.7	9567	62.6	8080	66.1	
Jun-20	14684	32	6395	34.7	4748	31.1	3541	29	
>20	3510	7.64	1952	10.6	959	6.28	599	4.9	
Median (Q1, Q3)	4	(2, 9)	5	(2, 11)	4	(2, 8)	4	(2, 7)	
eGFR, mL/min/1.73 m^2									0.0006
≥ 90	8706	19	3433	18.6	2808	18.4	2465	20.2	
60-89	19548	42.6	7825	42.4	6597	43.2	5126	42	
30-59	13835	30.1	5565	30.2	4659	30.5	3611	29.6	
<30	2856	6.22	1181	6.41	901	5.9	774	6.33	
Dialysis	986	2.15	433	2.35	309	2.02	244	2	
Medication									
Antiplatelet drugs	8903	19.4	4109	22.3	2848	18.7	1946	15.9	<0.0001
Warfarin	1158	2.52	637	3.46	315	2.06	206	1.69	<0.0001
Lipid lowering drug	3675	8	1588	8.61	1048	6.86	1039	8.5	<0.0001
tPA	1919	4.18	1095	5.94	482	3.16	342	2.8	<0.0001

Q1, 25th percentile; Q3, 75th percentile; SD, standard deviation; TOAST, Trial of Org 10172 in Acute Stroke Treatment; BMI, body mass index; AF, atrial fibrillation; Hb, hemoglobin; NIHSS, National Institutes of Stroke Scale; eGFR, estimated glomerular filtration rate; tPA, tissue plasminogen activator.

Table 1: The baseline characteristics of ischemic stroke patients by the triglyceride level.

The 30-day survival curves estimated by the Kaplan-Meier analysis showed that patients with triglyceride levels <150 mg/dL had 2.2% lower survival probability than those with the level >200 mg/dL (p<0.001) (Figure 2) (p<0.001). Table 2 shows that there were 1261 patients (2.75%) died within 30 days after ischemic stroke and over half (54.3%) of deceased cases were patients with triglyceride levels <150 mg/dL. The mortality rate declined as the triglyceride level increased. The mortality rate was 2.3-fold greater for patients with triglyceride levels <150 mg/dL than those with triglyceride levels of 200 mg/dL and above (1.62 vs. 0.72 per 1000 person-days), with an adjusted HR of 1.25 (95% CI=1.06-1.46).

Triglyceride (mg/dl)	Death (n)	Person-days	Mortality‡	Crude HR (95% CI)	Adjusted HR (95% CI)
<150	685	421939	1.62	2.2	1.25
				(1.90-2.56)***	(1.06-1.46)**
150-199	347	403879	0.86	1.21	0.94
				(1.02-1.42)*	(0.79-1.11)
≥ 200	229	320272	0.72	1.00 (reference)	1.00 (reference)
Overall	1261	1146090	1.1	-	-
Adjusted for age, gender, TOAST, location, body mass index, smoking, atrial fibrillation, previous stroke history, ischemic heart disease, congestive heart failure, diabetes mellitus, systolic blood pressure, hemoglobin, eGFR levels, NIHSS score at admission and medicine use.					
‡per 1000 person-days.					
*p<0.05, **p<0.01, ***p<0.001.					

Table 2: Mortality and estimated hazard ratio within 30 days after acute ischemic stroke by serum total triglyceride level on admission.

Triglyceride (mg/dl)	Death (n)	Person-days	Mortality‡	Crude HR (95% CI)	Adjusted HR (95% CI)
<150	1449	3053158	0.47	2.05	1.21
				(1.86-2.26)***	(1.09-1.34)***
150-199	867	3273126	0.26	1.2	0.96
				(1.08-1.33)***	(0.86-1.07)
≥ 200	578	2621759	0.22	1.00 (reference)	1.00 (reference)
Overall	2894	8948043	0.32	-	-
Adjusted for age, gender, TOAST, location, body mass index, smoking, atrial fibrillation, previous stroke history, ischemic heart disease, congestive heart failure, diabetes mellitus, systolic blood pressure, hemoglobin, eGFR levels, NIHSS score at admission and medicine use.					
‡per 1000 person-days.					
***p<0.001.					

Table 3: Mortality and estimated hazard ratio within one year after acute ischemic stroke by serum total triglyceride level on admission.
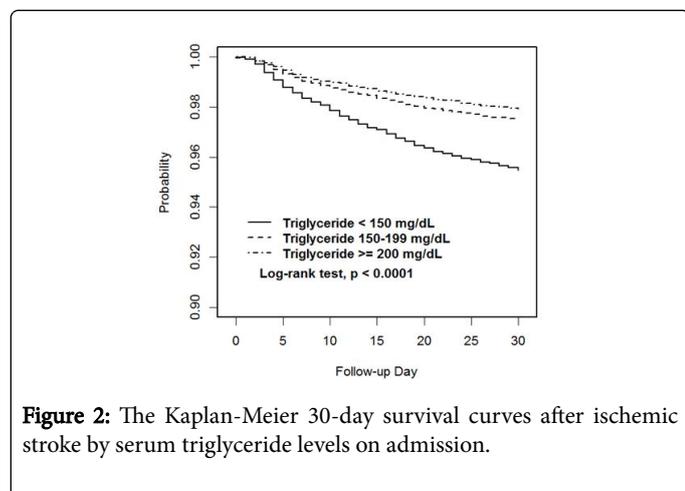

Figure 2: The Kaplan-Meier 30-day survival curves after ischemic stroke by serum triglyceride levels on admission.

The number of the stroke patients died within one year after ischemic stroke increased to 2894 and one half of deceased cases were patients with low triglyceride levels (Table 3). The mortality rate declined as the triglyceride level increased with 2.1-fold greater for patients with triglyceride levels <150 mg/dL than those with triglyceride levels of 200 mg/dL and above (0.47 vs. 0.22 per 1000 person-days), with an adjusted HR of 1.21 (95% CI=1.09-1.34). Patients with triglyceride levels <150 mg/dL (p<0.001) had 6.3% lower survival proportion than those with the level of >200 mg/dL (Figure 3).

Discussion

Previous studies using clinical indicators on the admission for acute stroke to evaluate death were rarely based on a large sample. The present study evaluated the impact of triglyceride level on mortality using one of the largest stroke registry data in the world. Our study demonstrated that as many as 40.1% of acute ischemic stroke patients had lower fasting serum triglyceride levels on admission. These patients were at an over 2-fold greater risk than those with higher triglyceride levels for both short-term mortality (30-day) and long-term (1-year) mortality. The serum triglyceride level is thus an important factor associated with death for patients with an acute ischemic stroke, independent of stroke severity. Findings based on this large stroke database confirm the results of previous smaller studies.

Figure 3: The Kaplan-Meier 1-year survival curves after ischemic stroke by serum total triglyceride levels on admission.
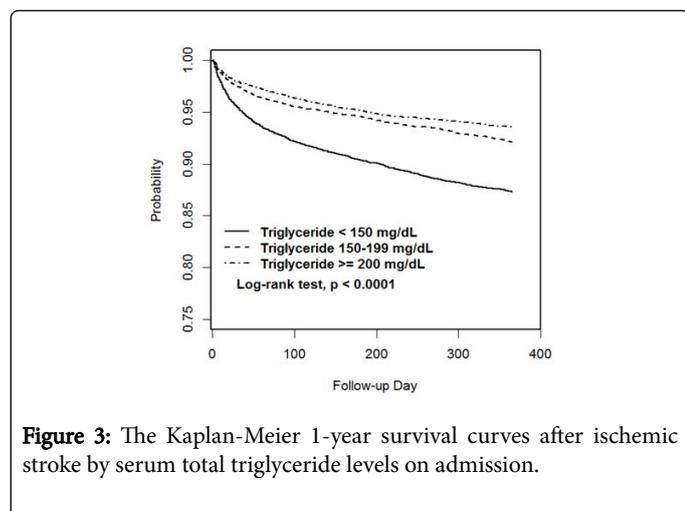
Previous studies have shown patients with serum triglyceride levels below 150 mg/dL on admission after acute stroke are associated with stroke severity and worse prognosis [6-13,17]. A retrospectively study found a higher serum triglyceride level (≥ 150 mg/dL) within 24 h after admission is associated with a lower infarct volume on CT brain scans [6]. Dziedzic et al. [7] have shown that a higher level of serum triglyceride (>2.3 mmol/L) is associated with 42% reduced stroke severity [7]. Weir et al. [17] studied 1310 non-diabetic patients admitted for acute stroke and found that low serum triglyceride levels could independently predict poor 6-month survival [17]. Li et al. [11] reported that low levels of serum triglyceride and total cholesterol, and high-density lipoprotein cholesterol are strong independent predictors of 3-month poor outcome (modified Rankin Scale (mRS) ≥ 3) in patients with acute stroke [11]. A Korean prospective cohort study also showed that low serum triglyceride is associated with an adjusted HR of 3.6 for mortality after acute non-cardio embolic ischemic stroke [13]. On the other hand, Choi et al. [14] reported that serum triglyceride levels had a nonlinear, J-shaped association with early poor outcome including neurological deterioration and mortality, and a reverse J-shaped association with early clinical improvement [14].

Triglyceride levels are associated with nutrition status [18]. The low triglyceride level is an indicator of malnutrition. Malnutrition after acute stroke is a poor prognostic factor [19,20]. The FOOD Trial Collaboration study found malnourished patients had an odds ratio of 2:3 for death after stroke. In addition, lipids are essential for cell membrane function and maintaining homeostasis [21]. Although triglyceride is harmful to vascular function, it has been shown to be able to protect against fatty acid-induced neurotoxicity [22]. Unsaturated fatty acids promote triglyceride accumulation and prevent lipotoxicity. The above mentioned mechanism may explain why lower triglyceride levels are associated with worse prognosis after ischemic stroke.

This study is a real world study using a large longitudinal stroke registry data to investigate the relationships between fasting serum triglyceride levels on admission and short-term and long-term mortality risks after ischemic stroke. However, the present study has several limitations. The information on serum triglyceride levels prior to stroke was unavailable for patients. It is not clear whether the triglyceride levels on admission related to the levels before stroke. Information on low density lipoprotein cholesterol and high density lipoprotein cholesterol was unavailable for patients. Thus, we were unable to investigate the specific role of these 2 types of lipoprotein cholesterol in this study. Laboratory data of serum albumin and C-reactive protein were also missing for some patients, limiting our ability to control these factors in the data analysis.

Conclusion

Our study demonstrated that lower fasting serum triglyceride levels on admission are predictive of short-term mortality (30-day) and long-term (1-year) mortality for patients with an acute ischemic stroke, independently of stroke severity. These findings confirm the results of previous smaller studies. The triglyceride level can be used as a marker to identify patients with high mortality risk. Patients with these conditions deserve greater cautiousness for the post-stroke care.

Acknowledgements

Authors' Contributions

I-Kuan Wang designed the study and drafted the manuscript. I-Ju Tsai designed the study and conducted the statistical analysis. Fung-Chang Sung designed the study and revised the manuscript. Other authors participated in data collection, manuscript development and final approval of the manuscript.

Conflict of Interests

The authors declare no conflicts of interest.

Financial Disclosure

No conflict of financial interest is declared.

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