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RESEARCH ARTICLE **ÖZGÜN ARAŞTIRMA**

HOW DO HYPERTRIGLYCERIDEMIA AND HYPOTRIGLYCERIDEMIA AFFECT PROGNOSIS IN ACUTE ISCHEMIC STROKE AND WHAT IS THEIR PREVALENCE?

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ABSTRACT

INTRODUCTION: Many studies are examining the relationship between cardiovascular diseases and triglyceride levels, and it has been shown that hypertriglyceridemia significantly increases the risk independently. In this study, we wanted to investigate whether there is a similar relationship for stroke.
METHODS: A total of 619 patients hospitalized with a diagnosis of acute ischemic stroke in our stroke center were included in the study. Demographic information, accompanying risk factors, hemoglobin level, thrombocyte count, triglyceride level, and C reactive protein level at the time of admission, and the National Institutes of Health Stroke Scale (NIHSS) scores in the first evaluation were recorded. Also, information about mortality in hospital, recurrent stroke, and the modified Rankin Scale (mRS) scores in the follow-up period was entered in our local database.
RESULTS: While 182 patients (32.3%) (78 females (42.9%) and 104 males (57.1%); mean age 64.5±12.01 years) were diagnosed with hypertriglyceridemia, this number was 25 (4.4%) patients (13 females (52%) and 12 males (48%); mean age 70.9±17.4 years) for hypotriglyceridemia. Among the risk factors studied, only diabetes mellitus was found to be associated with hypertriglyceridemia in patients with acute ischemic stroke ($P<0.05$).
DISCUSSION AND CONCLUSION: In this study conducted in our stroke center, hypertriglyceridemia and hypotriglyceridemia prevalences were found as 32.3% and 4.4%, respectively. HyperTG or hypoTG did not significantly affect on stroke severity and prognosis. We found out that DM is the major risk factor for acute ischemic stroke patients with hyperTG.
Keywords: Hypertriglyceridemia, hypotriglyceridemia, ischemic stroke.

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HİPERTRİGLİSERİDEMİ VE HİPOTRİGLİSERİDEMİ AKUT İSKEMİK İNMEDE PROGNOZU NASIL ETKİLER VE PREVALANSI NEDİR?

ÖZ

GİRİŞ VE AMAÇ: Kardiyovasküler hastalıkların trigliserid düzeyi ile ilişkisini araştıran bir çok çalışma mevcuttur ve hipertrigliserideminin riski bağımsız olarak belirgin düzeyde artırdığı gösterilmiştir. Biz bu çalışmada benzer bir ilişkinin inme için var olup olmadığını göstermek istedik.
GEREÇ VE YÖNTEM: Akut iskemik inmeli 619 hastanın verileri değerlendirildi. Demografik veriler, risk faktörleri, başvuru sırasındaki hemoglobin düzeyi, trombosit sayısı ve C reaktif protein düzeyi ve ilk değerlendirmedeki NIH İnme Ölçeği (National Institutes of Health Stroke Scale) puanları kayıt altına alındı. Ayrıca hastanede ölüm, tekrarlayan inme ve takiplerdeki modifiye Rankin Ölçeği (mRS) puanları veritabanına kayıt edildi.
BULGULAR: Toplamda 182 hastanın (32.3%) (104 erkek (57.1%) ve 78 kadın (42.9%); ortalama yaş 64.5 ± 12.01 yıl) hipertrigliseridemisi vardı. Diğer yandan ise 25 hastanın (4.4%) (12 erkek (48%) ve 13 kadın (52%); ortalama yaş 70.9± 17.4 years) verileri hipotrigliseridemi ile uyumlu idi. Logistik regresyon analizinde sadece diabetes mellitus (DM) akut iskemik inmeli hastalardaki hipertrigliseridemi durumu ile belirgin ilişkili bulundu (P<0.05).
TARTIŞMA VE SONUÇLAR: Hipertrigliseridemi bizim merkezimizde akut iskemik inmelerin 32.3% sinde görüldü. Diğer taraftan hipotrigliseridemi için bu oran 4.4% olarak saptandı. Hipertrigliseridemi veya hipotrigliserideminin olmasının inme şiddeti ve prognozuna etkisi olmadığı görüldü. Edinilen bilgiler ışığında hipertrigliseridemisi olan akut iskemik inmeli olgularda DM major risk faktörü gibi görünmektedir.

Anahtar Sözcükler: Hipertriglseridemi, hipotrigliseridemi, iskemik inme.

INTRODUCTION

Hypertriglyceridemia (hyperTG) is identified in 17.96 % of patients with ischemic stroke and transient ischemic attack (1). Many studies are examining the relationship between cardiovascular diseases and triglyceride levels, and it has been shown that hyperTG significantly increases the risk independently (2). Data that can fully explain the relationship between stroke and triglyceride is still insufficient in current sources (3-13). A triglyceride level of 100 mg/dl for fasting is the optimal value, while it is also acceptable for it to be below 150 mg/dl (3). In a study conducted in the United States, the prevalence of hyperTG in adults was studied and it was found that those over 150 mg/dl, > 200 mg/dl, > 500mg/dl, and >1000 mg/dl respectively, constitute the 33, 18, 1.7, and 0.4 percent (4). Some studies are adjusting similar risk of elevated triglyceride levels for both stroke and ischemic heart diseases. There are few studies about the relationship between subtypes of ischemic stroke and hyperTG that incompatible with each other (5,6,7).

Fasting triglyceride less than 50 mg/dl is accepted as hypotriglyceridemia (hypoTG). There has not been any data for hypoTG prevalence in patients with ischemic stroke. In a study, it has been concluded that hypoTG increases in-hospital mortality in acute ischemic stroke and is associated with severe stroke (14).

Bharosay et al.'s study showed that the risk of hemorrhagic stroke increases with hypoTG and that hypoTG is associated with neurological deterioration (15). In another study, it was shown that being hypertensive, and especially a man with low cholesterol levels, increased hemorrhagic stroke, while hypoTG decreased the risk of ischemic stroke (16).

Also, some studies suggest that both hyperTG and hypoTG are poor prognostic factors in ischemic stroke. We aimed to get answers to the questions about the prevalence of hyperTG and hypoTG in our stroke center and how they affect the prognosis of stroke.

METHODS

The study was performed in accordance with Helsinki Declaration. Ethics committee approval was obtained in advance of the study from Ankara University Human Researches Ethics Committee (Number: 2021000110-2, Date: 21.05.2021). Informed consent was received from participants included in the study. In case of having a problem with consciousness, it was obtained from legal guardians.

A total of 619 patients with acute ischemic stroke who applied to our clinic between January 2012 and October 2014 were evaluated. A total of 564 patients who were adequately tested within

the first 48 hours of serum lipid profile were included in the study. Triglyceride (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) analyses were performed in our local laboratory. Samples were drawn early in the morning after 12 hours of fasting the day after admission to the hospital.

In line with the recommendations in the National Cholesterol Education Program (NCEP) III categorization guidelines, values higher than 149 mg/dl were accepted for hyperTG and lower than 50 mg/dl for hypoTG (Table 1) (9).

Demographic information, accompanying risk factors hypertension, DM, hyperlipidemia, previous stroke, coronary artery disease (CAD), congestive heart failure (CHF), atrial fibrillation (AF)), hemoglobin level, thrombocyte count, and C reactive protein level at the time of admission, the National Institutes of Health Stroke Scale (NIHSS) scores in the first evaluation were recorded. Also, information about mortality in hospital, recurrent stroke, and the modified Rankin Scale (mRS) scores in the follow-up period was entered in our local database.

The etiological stroke subtype was classified based on the automated Causative Classification System (CCS, available at https://ccs.mgh.harvard.edu) (17). This system is an automated version developed to increase the reliability of the SSS-TOAST classification, and it concludes by evaluating the diagnostic tests for the etiology of stroke among multiple causes, based on evidence. The CCS subtypes are classified as supra-aortic large artery atherosclerosis (LAA), cardio-aortic embolism, small artery occlusion, other causes, and undetermined causes. Vascular imaging studies used for etiological classification consisted of carotid Doppler ultrasonography, computerized tomography angiography (CTA), magnetic resonance angiography (MRA), or digital subtraction angiography (DSA). Related laboratory tests were used to determine causes such as hypercoagulability and vasculitis and transthoracic or transesophageal echocardiography, 24 hours cardiac rhythm monitoring to identify cardiac causes.

Statistics: Data were analyzed by using The Statistical Package for the Social Sciences (SPSS) 16.0 version (SPSS Inc. Chicago, Illinois, USA). Normally distributed continuous variables are presented as mean and standard deviation, while non-normally distributed variables are presented as a median and interquartile range, respectively. Student's t-test for comparing means and Chi-squared test for group rates were used. Data obtained from logistic regression analysis were used to determine predictor variables of hyperTG and hypoTG. Statistically, a significant p-value was considered to be less than 0.005.

Table 1. Classification of serum triglyceride levels, according to the National Cholesterol Education Program (NCEP) III categorization (9).

Class	Serum triglyceride level
Normal	<150 mg/dl (1.7 mmol/l)
Borderline high	150–199 mg/dl (1.7–2.2 mmol/l)
High	200–499 mg/dl (2.2–5.6 mmol/l)
Very high	≥500 mg/dl (≥5.6 mmol/l)

RESULTS

HyperTG was detected in 182 patients (32.3%) (78 females (42.9%) and 104 males (57.1%)) with mean age of 64.5±12.01 years (Table 2).

On logistic regression analysis, it was seen that the presence of hypertension, hyperlipidemia, CAD, transient ischemic attack, and stroke in the history were statistically similar for the groups (P>0.05). Among the risk factors studied, only diabetes mellitus was found to be associated with hyperTG in patients with acute ischemic stroke (P=0.001) (Table 3). Otherwise, CHF and AF were more in the group without hyperTG than in the group with hyperTG (P<0.05) (Table 2). NIHSS score, thrombocyte count, and CRP levels at admission were similar in groups (P>0.05). The mean hemoglobin level at admission was significantly higher in the hyperTG group compared to others (P=0.018). Stroke subtypes were etiologically similar for groups (p>0.05) (Table 2).

In-hospital mortality rates in patients followed for an average of 9 (1-32) months were similar for the groups (P=0.703). 72 of the hyperTG patients and 171 of the non-hyperTG patients had follow-up data. The mean mRS was 1.9±2.2 (0-6) and 2.4±2.2 (0-6) for the hyperTG group and non-hyperTG group during follow–up, respectively (P=0.437). Stroke recurrence was high in the hyperTG group, with insignificant statistics (P=0.365) (Table 2).

On the other hand, hypoTG was detected in 25 (4.4%) patients (13 females (52%) and 12

Table 2. Epidemiologic and clinical characteristics of patients with hyperTG and without hyperTG.

	HyperTG n = 182	**Non-hyperTG n = 382**	**P**
Age, year, *Mean±SD*	64.5±12.0	71.3±13.1	0.4
Sex, *n (%)*			
Female	78 (42.9)	182 (47.6)	0.29
Male	104 (57.1)	200 (52.6)	
Medical history			
- Hypertension, *n (%)*	123 (67.6)	270 (70.7)	0.45
- Diabetes mellitus, *n (%)*	70 (38.5)	95 (24.9)	**0.001**
- Atrial fibrillation, *n (%)*	16 (8.8)	94 (24.6)	**<0.001**
- Hyperlipidemia, *n (%)*	57 (31.3)	99 (25.9)	0.18
- CAD, *n (%)*	35 (19.2)	93 (24.3)	0.17
- CHF, *n(%)*	9 (4.9)	53 (13.9)	**0.002**
- Previous TIA history, *n (%)*	12 (6.6)	33 (8.6)	0.40
- Previous stroke history, *n (%)*	28 (15.4)	71 (18.6)	0.35
CCS classification, n *(%)*			
- Large-artery atherosclerosis	51 (28)	94 (24.6)	0.49
- Cardio-aortic embolism	67 (36.8)	167 (43.7)	
- Small artery occlusion	13 (7.1)	18 (4.7)	
- Other causes	10 (5.5)	22 (5.8)	
- Undetermined causes	41 (22.5)	81 (21.2)	
CRP, mg / l, *Mean±SD*	20.3±41.3	23.4±38.3	0.61
Hemoglobin, g/dl, *Mean±SD*	13.6±2.2	13.1±1.9	**0.018**
Thrombocyte, X10^9/L, *Mean±SD*	250±86.9	237±84.9	0.24
Admission NIHSS, *Mean±SD*	5.18±4.18	5.98±4.31	0.29
(Min-Max)	(0-26)	(0-26)	
Mortality in hospital, *n (%)*	15 (8.2)	28 (7.3)	0.70
Follow up mRS, *Mean±SD*	1.9±2.2	2.4±2.2	0.44
(Min-Max)	(0-6)	(0-6)	
Follow up months, *Mean±SD*	10.3±8.4	8.9±7.7	0.17
Recurrent stroke, *n (%)*	3 (1.6)	10 (2.6)	0.47

SD; Standard Deviation, CAD; Coronary Artery Disease, CHF; Congestive heart failure, TIA; transient ischemic attack, mRS: modified Rankin Scale, NIHSS; National Institutes of Health Stroke Scale scores, CRP; C reactive protein.

Table 3. Logistic regression analysis of HyperTG.

	OR	**95 %, CI**	**p**
Diabetes mellitus, *n(%)*	3.449	1.113 - 4.133	0.001

males (48%); and mean age 70.9±17.4 (23-98) years with acute ischemic stroke (Table 4).

The prevalence of transient ischemic attack history in the hypoTG group was statistically higher than in the non- hypoTG group (P=0.023). The mean CRP level of the hypo TG group at admission was significantly lower than those in the other groups (P=0.012). Other accompanying risk factors, NIHSS score, thrombocyte count, and hemoglobin levels at admission were similar in the groups (P>0.05). Stroke subtypes were etiologically similar for groups (p>0.05) (Table 2).

In-hospital mortality rates in patients followed for an average of 9 (1-32) months were similar for the groups (P=0.39). 10 of the hypoTG patients and 264 of the non-hyperTG patients had follow-up data. The mean mRS was 3.1±2.5 (0-6) and 2.2±2.2 (0-6) for the hypoTG group and non-hypoTG group in clinical follow-ups, respectively (P=0.437). While stroke recurrence was observed in any patient in the hypoTG group, 13 (2.4%) patients in the non-hypoTG group developed recurrent stroke (P=0.43) (Table 4).

As a result of the logistic regression analysis, no variables were found to be associated with hypoTG in acute ischemic stroke patients (P<0.05).

DISCUSSION AND CONCLUSION

Prevalence: HyperTG was detected in 182 patients (32.3%) in our study (78 females (42.9%) and 104 males (57.1%); mean age 64.5±12.01 years) (Table 2).

The previous studies have not reported any data for hypoTG prevalence. In our study, we evaluated the prevalence of hypoTG. HypoTG was detected in 25 (4.4%) patients (13 females (52%) and 12 males (48%); mean age 70.9±17.4 years).

In the National Health and Nutrition Examination Survey (NHANES), the prevalence of having serum triglyceride level ≥150 mg/dl was 33% (37% of men and 30% of women) (11).

Table 4. Epidemiologic and clinical characteristics of patients with hypoTG and without hypoTG.

	HypoTG n = 25	**Non-hypoTG n = 539**	**P**
Age, year, *Mean ± SD*	70.9 ± 17.4	69.0 ± 12.9	0.14
Sex, *n(%)*			
Female	13(52)	247(45.8)	0.55
Male	12(48)	292(54.2)	
Medical history			
- Hypertension,*n(%)*	18(72)	375 (69.6)	0.79
- Diabetes mellitus, *n(%)*	4(16)	161(29.9)	0.14
- Atrial fibrillation, *n(%)*	7(28)	103(19.1)	0.27
- Hyperlipidemia, *n(%)*	5(20)	151(28)	0.38
- CAD, *n(%)*	3(12)	125(23.2)	0.19
- CHF, *n(%)*	3 (12)	53(13.9)	0.87
- Previous TIA history, *n(%)*	5 (20)	40(7.4)	**0.023**
- Previous stroke history, *n(%)*	5 (20)	94(17.4)	0.74
CCS classification, n*(%)*			
- Large-artery atherosclerosis	4 (16)	141 (26.2)	0.39
- Cardio-aortic embolism	12 (48)	222 (41.2)	
- Small artery occlusion	1 (4)	30 (5.6)	
- Other causes	0 (0)	32 (5.9)	
- Undetermined causes	8 (32)	114 (21.2)	
CRP, mg / l, *Mean ± SD*	7.8 ± 8.5	23.3 ± 40.1	**0.012**
Hemoglobin, g/dl, *Mean ± SD*	13.2 ± 1.5	13.3 ± 2.1	0.15
Thrombocyte, X10^9/L, *Mean ± SD*	238±93.2	242 ± 85.5	0.65
Admission NIHSS, *Mean ± SD*	7.48±5.35	5.65±4.22	0.83
(Min - Max)	(0 - 20)	(0 - 26)	
Mortality in hospital, *n(%)*	3(12)	40 (7.4)	0.39
Follow up mRS, *Mean ± SD*	3.1 ± 2.5	2.2 ± 2.2	0.62
(Min - Max)	(0 - 6)	(0 - 6)	
Follow up months, *Mean ± SD*	6.4 ± 8.2	9.4 ± 7.9	0.47
Recurrent stroke, *n(%)*	0 (0)	13 (2.4)	0.43

SD; Standard Deviation, CAD; Coronary Artery Disease, CHF; Congestive heart failure, TIA; transient ischemic attack, mRS: modified Rankin Scale, NIHSS; National Institutes of Health Stroke Scale scores, CRP; C reactive protein.

The percentage was 42% in 60 years old and/or over (11). The study of Christian et al. showed that the prevalence of hyperTG in US adults is 32.2%, and a very high TG level (>500 mg/dl) is 1.7 % (12).

In a prospective study, 6323 patients showed a similar relative risk of elevated triglyceride levels for stroke and ischemic heart diseases (13). In a study associated with the prevalence of severe hyperTG in adults in the USA, the mean TG was found to be 179.9 ± 62.8 mg/dl and 148.0 ± 51.9 mg/dl respectively in the cases and control group, significantly higher in the case group. ($p<0.05$) (12). And also Austin et al. and Tanne et al. reported a similar result (18,19). Austin et al. showed that baseline triglyceride levels are associated with CVD mortality in familial combined hyperTG patients (19). In a prospective study involving 11177 hyperTG patients, hyperTG (particularly fasting triglyceride level> 200 mg/dl) was reported to be an important factor increasing the initial stroke risk. (20).In a retrospective cohort study of 3249 elderly hypertensive Chinese patients by Huang et al. Data was obtained indicating that high triglyceride level is an independent high-risk factor for the first stroke (21). Also in two studies conducted on pediatric stroke cases, data were obtained indicating that hypertriglyceridemia increases the risk of ischemic stroke (22,23). The Copenhagen study has also shown a strong linear relationship of nonfasting triglyceride level with ischemic stroke (24).

Risk factors: In our study, AF and CHF were detected less in the group with hyperTG than in the group without hyperTG. ($P<0.05$). On logistic regression analysis, among the risk factors studied, only diabetes mellitus was found to be associated with hyperTG in patients with acute ischemic stroke ($P=0.01$).

HyperTG might be due to excessive synthesis, defective procession, and cleaning, or both of them. It might be both because of primary or secondary factors such as obesity, diabetes, hypothyroidism, sedentary life, drugs, and sepsis (25).

In a study investigating the relationship between metabolic syndrome and stroke, it was shown that all components were independently associated with stroke, although hyperTG was one of the most associated (26). In a case-control study by Jeetendrakumar et al., in which they compared lipid profile results in diabetics and non-diabetic stroke patients, concluded that diabetic patients had higher triglyceride levels (27). The data of 3216 participants were evaluated in the "The Strong Heart Study", which investigated the relationship between triglyceride and HDL-C dyslipidemia in the case of glycemic dysregulation with CAD and ischemic stroke (28). In this study, it was shown that the risk of CAD and ischemic stroke increased in participants with high fasting triglyceride levels in combination with low HDL-C, regardless of other atherosclerotic risk factors, especially if the participant was diabetic or accompanied by high LDL-C (≥130 mg/dL) (28). Multivariate analyzes have shown that this increased risk is associated with being diabetic independent of HbA1c level (28). Shin et al. showed that hyperTG with diabetes was related to lacunar stroke (5). Also, some studies are showing that poorly controlled DM had higher triglyceride levels and prolonged postprandial triglyceride values than the well-controlled one (8).

A population-based study designed by Njolstad et al. showed an independent relationship with nonfasting triglyceride only for women (29). In a meta-analysis by Leonards C. et al., in which they reviewed the results of twenty-five studies to evaluate the relationship between fasting and postprandial triglyceride levels and stroke, the results were contradictory (30). Researchers concluded that this may be related to fact that triglyceride measurements were not made under standardized conditions, and they think that doing it this way based on oral triglyceride tolerance test (OTTT) will give more accurate results (30). Choi et al. reported that groups hypoTG, normoTG, and hyperTG were similar in terms of gender, smoking, alcohol, DM, hypertension, previous stroke history (31). They reported that having histories of dyslipidemia and statin drug usage are associated with current hyperTG (31).

We couldn't evaluate our patients for genetic predisposition. However, there is a 20-year observational study showing a 70% increased risk of cardiovascular disease mortality in first-degree relatives of familial hyperTG compared to controls (19).

In our study, the mean CRP levels at admission in patients with hypoTG were significantly lower compared to other patients (P=0.012), while not related to hyperTG (Table 3). Okada et al. showed that increased triglyceride level was associated with high sensitive CRP level and it might be an indicator of chronic inflammation (32). It was known that hs-CRP independently predicted future cardiovascular disease and, could be used as a marker for the progression of atherosclerosis (32). Since the CRP level could not be measured with high-sensitivity kits in our study, a similar result could not be obtained in the hyperTG group and it would be appropriate to evaluate the subject in future studies.

Stroke subtypes: In our study, we found that TG levels and etiologic stroke subtypes had no significant association (p>0.05) (Table 2, Table 3). As a matter of fact, there are a limited number of studies reviewing the relationship between hypo TG and ischemic stroke (14,15,16), few studies are evaluating the relationship between hyperTG and ischemic stroke subtypes. It was shown that 3 stroke subtypes (LAA, small-vessel event, cardioembolic event) are associated with higher triglyceride levels, with not any significant difference in stroke subtypes, in a case-control study (n=240) (6). In another study, it was stated that large artery disease is associated with increased TG, but the small-vessel disease is not (7). In a retrospective study, the TG level of 2141 patients was evaluated for stroke subtypes of patients with diabetes; it was significantly and consistently associated with lacunar infarction rather than LAA and cardioembolic disease (8).

In a study using Mendelian randomization on how genetic hyperlipidemias affect stroke and stroke subtypes, it was found that there was no relationship of genetically high triglycerides with stroke and stroke subtypes (33).

Since there are a limited number of studies reviewing the relationship between hypo TG and ischemic stroke (14,15,16), we couldn't find any for ischemic stroke subtypes.

Stroke severity: There is not any statistical data was showing the significant relationship of the NIHSS with hyperTG and hypoTG in our study (p>0.05). Jain et al. showed that the decrease in

triglyceride levels caused an increase in NIHSS in the form of a graph matching the reverse linear relationship (P=0.0015), although there was no association between the level of cholesterol, HDL, and LDL and NIHSS (respectively, p=0.4575, p=0.1237, and p=0.1422) (29). They showed that high triglycerides had been associated with low NIHSS scores (34). In a prospective study, major lipids and their association with NIHSS at admission were evaluated. It was demonstrated that hypoTG had been associated with more severe stroke (14).

Prognosis, mortality in hospital, recurrent stroke rate, and disability of hyperTG: In our study, recurrent stroke occurred higher in the hyperTG group, but not significantly (3 (3.7%) patients with hyperTG and 10 (2.1%) patients without hyperTG, (P=0.365)). While stroke recurrence was observed in any patient in the hypoTG group, 13 (2.4%) patients in the non-hypoTG group developed recurrent stroke (P=0.43).

Choi et al. (n=736) reported a relationship between early neurological deterioration in ischemic stroke and serum triglyceride level, suggested a reverse J-shaped association with good prognosis and a nonlinear, J-shaped association with poor prognosis (31). Kwon et al. also reported hyperTG (>145 mg/dl) increased the risk of early neurological deterioration in lacunar stroke, independently (10). So this issue could be studied in more detail in future experiments for evaluating the effect of hypoTG on the prognosis of patients.

In our study, triglyceride levels didn't cause any change in mortality in-hospital statistically, for the hyperTG group (P = 0.703) and the hypoTG group (P = 0.39). Choi et al. reported similar results for acute ischemic stroke (31). In some studies both hyperTG and hypoTG have been shown to increase mortality independently in CAD (31). And also hyperTG was shown to be related to recurrent CAD (31). Although there have been a few studies about relation with triglyceride level and mortality, in a previous study it was reported that findings were indicating low TG levels cause a high mortality rate in stroke (35). Tziomalos et al. reported that hypoTG was a predictor for in-hospital mortality rates in acute ischemic stroke but not dependency at discharge (14).

Triglyceride-glucose(TyG) index has recently been used as a marker for insulin resistance and calculated as Ln (fasting blood glucose (mg/dL) × fasting triglycerides (mg/dL) ÷ 2) (36). Zhou Y et al. studied, how the course of stroke will be affected by the TyG index and they concluded that the TyG index is associated with recurrent stroke, mortality, and neurological deterioration in Ischemic stroke (37). As a limitation of our study, since the fasting blood glucose values of the patients were not recorded in our database, we could not provide statistical data on the effect of the TyG index on these conditions.

In our study, we didn't find any significant difference between having hyperTG or hypoTG, and mRS (p=0.437, p=0.437). Choi et al. reported that having low normal triglyceride levels (50 to 100 mg/dl) had better clinic healing and higher mRS 0–2 at 30 days (31). Also, in a prospective study, Jain et al. reached data suggesting that low triglyceride levels cause a worse prognosis in acute ischemic stroke (34).

Our study has some limitations since it is designed retrospective and carried out in a single medical center. Our patients had been evaluated for stroke subtype, as most of our patients had brain MR imaging, brain and cervical vascular imaging, echocardiography, and 24hour rhythm Holter.

In conclusion;

-In 619 patients included in the study, the hyperTG detection rate was 32.3 percent, while this rate was 4.4 percent for hypoTG.

- HyperTG or hypoTG did not significantly affect on stroke severity and prognosis.

- Among the risk factors studied, only diabetes mellitus was found to be associated with hyperTG in patients with acute ischemic stroke.

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Ethics

Ethics Committee Approval: The study was approved by Ankara University Human Researches Ethics Committee (Number: 2021000110-2, Date: 21.05.2021).

Informed Consent: The authors declared that informed consent was signed by the patients.

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