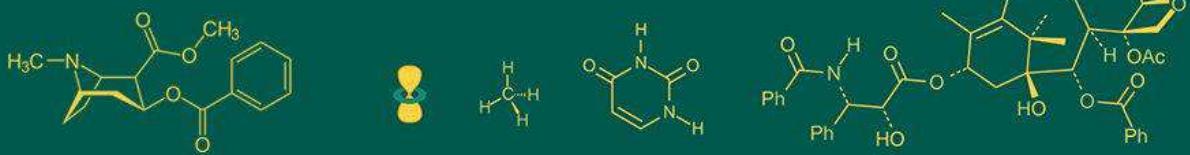


# International Journal of Advanced Biochemistry Research



**ISSN Print:** 2617-4693  
**ISSN Online:** 2617-4707  
**IJABR 2019; 3(2):** 21-25  
[www.biochemjournal.com](http://www.biochemjournal.com)  
**Received:** 14-09-2019  
**Accepted:** 19-10-2019

**Dr. Mohd Azam Hyder**  
Assistant Professor,  
Department of Biochemistry,  
Bhaskar Medical College &  
General Hospital, Ranga  
Reddy, Telangana, India

## A study on correlation between fasting & post-prandial serum triglycerides and severity of stroke-at a tertiary care hospital

### Dr. Mohd Azam Hyder

#### Abstract

**Background:** Although dyslipidemia is a well-established risk factor for coronary artery disease, its relationship to ischemic cerebrovascular disease has remained unclear, perhaps because of the heterogeneous nature of stroke. Recent studies indicate postprandial triglyceride (TG) had a better association with cardiovascular events and metabolic syndrome than fasting TG.

**Aim & Objective of the study:** The aim of this study is to investigate if serum TG levels predict stroke severity on admission with correlation of fasting and post-prandial serum TG levels

**Methods:** 80 patients of acute ischemic stroke who were admitted to Bhaskar Medical College within 24 hours of development of symptoms were included in this study in which fasting TG levels & post prandial TG levels were done. Stroke severity on admission was assessed using Scandinavian Stroke Scale (SSS). The patients were divided into 2 groups: those with severe stroke (SSS  $\leq$ 25) and those with mild/moderate stroke (SSS >25).

**Results:** Of the 80 patients studied, 54 were males and 26 were females. Among the males, 42 had FTG  $\leq$ 2.3mmol/l and 12 had FTG levels >2.3mmol/l & 27 had PPTG  $\leq$ 2.3mmol/l and 27 had PPTG levels >2.3mmol/l. Of the 26 females 18 had FTG  $\leq$ 2.3mmol/l and 8 patients had FTG levels >2.3mmol/l & 15 had PPTG  $\leq$ 2.3mmol/l and 11 had PPTG levels >2.3mmol/l. In this study the number of patients with severe stroke (SSS  $\leq$ 25) were 20 of which 15 had F TG  $\leq$ 2.3mmol/l and only 5 patient had F TG >2.3mmol/l & 9 had PPTG  $\leq$ 2.3mmol/l and 12 had PPTG >2.3mmol/l. Out of 80 patients, 44 were hypertensive and 32 had FTG  $\leq$ 2.3mmol/l and 12 had FTG >2.3mmol/l & 19 had PPTG  $\leq$ 2.3mmol/l and 25 had PPTG >2.3mmol/l of the remaining 36 non hypertensive patients, 30 had FTG  $\leq$ 2.3mmol/l and 6 had FTG >2.3mmol/l & 25 had PPTG  $\leq$ 2.3mmol/l and 11 had PPTG >2.3mmol/l. 16 were IHD patients of which 12 had FTG  $\leq$ 2.3mmol/l and 4 had FTG >2.3mmol/l & 8 had PPTG  $\leq$ 2.3mmol/l and 8 had PPTG >2.3mmol/l & of the remaining 64 patients, 50 had FTG  $\leq$ 2.3mmol/l and 14 had FTG >2.3mmol/l & 30 had PPTG  $\leq$ 2.3mmol/l and 34 had PPTG >2.3mmol/l. The levels of serum FTG were not influenced by hypertension, IHD, age and sex of an individual but the level of serum PPTG were influenced by HTN.

**Conclusion:** In this study, mean serum fasting & post prandial serum triglyceride levels were not significantly associated with the severity of the stroke. As per this study hypertension, IHD, age and sex of an individual do not influence levels of serum fasting triglyceride levels but the serum post prandial triglyceride level were influenced by HTN.

**Keywords:** Stroke, triglycerides, Hypertension

#### Introduction

Among all the neurologic diseases of adult life, the stroke clearly rank first in frequency and importance accounting upto 20% of all central nervous system disorders, in the urban sectors of India. Stroke is defined as an abrupt neurologic deficit that is attributable to focal vascular cause. Risk factors for stroke include hypertension, carotid stenosis, atrial myxomas, smoking, hyperlipidemia, diabetes, myocardial infarction and atrial fibrillation.

The link between hyperlipidemia and stroke, was more difficult to establish. The difficulty arose in part because of the heterogeneous nature of stroke. To investigate a possible etiologic relationship between hyperlipidemia and stroke, it became essential to distinguish ischemic from hemorrhagic stroke. It now appears likely that hyperlipidemia is an independent risk factor for ischemic stroke.

Humans spend most of their time in a postprandial state, not in a fasting state, and their arteries are thus exposed to postprandial plasma most of the time. One would thus surmise that postprandial lipid values would correlate better with prognosis than fasting values.

**Corresponding Author:**  
**Dr. Mohd Azam Hyder**  
Assistant Professor,  
Department of Biochemistry,  
Bhaskar Medical College &  
General Hospital, Ranga  
Reddy, Telangana, India

Postprandial TG has been suggested as an independent risk factor for cardiovascular disease (CVD) in healthy subjects [1-3]. Several studies support the hypothesis postprandial TG is correlated with carotid IMT and waist circumference (WC), and furthermore superior to fasting TG in association with metabolic syndrome [4-6]. However, the potential effects of postprandial hypertriglyceridemia on the development of atherosclerosis are still controversial in subjects with type 2 diabetes (T2D) [7].

This study is undertaken to correlate fasting and postprandial serum triglyceride levels with severity of stroke as measured by Scandinavian stroke scale.

## Materials and Methods

### Source of Data

Eighty consecutive patients presenting with acute ischemic stroke, occurring within 24 hours, confirmed by CT scan admitted in Bhaskar Medical College & General Hospital from January 2018 to June 2019 were included in this study and in each patient fasting and post prandial serum TG levels were estimated.

### Inclusion Criteria

Patients with first attack of ischemic stroke occurring within 24 hours, confirmed by CT scan, admitted to Bhaskar Medical College, RR District, Telangana, India.

### Exclusion Criteria

Patients admitted to the hospital >24 hours after stroke onset.

Previous history of stroke.

Previous history of transient ischemic attack.

Haemorrhagic stroke.

Patients with space occupying lesions.

Patients with CVT.

### Method of Collection of Data

A CT scan head (MRI brain where required) was taken within 24 hours after stroke onset for all patients with first ever stroke admitted in Bhaskar Medical College and hospital.

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).

Arterial hypertension was diagnosed when at least 2 readings of blood pressure were ≥ 140 mm Hg (systolic) or ≥ 90mm Hg (diastolic) after the acute phase of stroke. Ischemic heart disease was diagnosed when a history of angina pectoris or myocardial infarction was present.

In addition to routine investigations as per standard protocol in the evaluation of stroke patient, fasting serum triglyceride level and post prandial serum triglyceride level were measured between 12 and 36 hours after stroke onset using commercially available kits.

Hypertriglyceridemia was diagnosed if TG >2.3mmol/l.

Patients were followed up till they were discharged from the hospital.

### Parameters

The following parameters were tested

- 1) Age
- 2) Sex

3) Hypertension

4) IHD

### Statistical Methods

1. Descriptive statistical analysis has been carried out in the present study. Significance is assessed at 5% level of significance.

### Procedure

A detailed history, clinical examination and relevant laboratory investigations were done as per proforma.

Fasting serum triglyceride levels and post prandial serum triglyceride levels were estimated in patients, quantitatively by GPO- TRINDER method based on the method of Wako and the modifications by McGOWAN *et al.* [8].

The kit used was Triglycerides Kit: Ortho Clinical Diagnostics Rochester, NY, USA.

### Results

The following tables show association between the fasting & post prandial triglyceride levels and severity of stroke based on SSS scale and their influence based on risk factors-age, sex, hypertension, IHD.

**Table 1:** Association between Age and SSS

		SSS			X <sup>2</sup> Value
AGE		≤25	>25	Total	
	≤65	15	40	55	0.133
AGE	>65	13	12	25	
	Total	28	52	80	

**Table 2:** Association between FTG and SSS

		SSS			X <sup>2</sup> Value
FTG		≤25	>25	Total	
	≤2.3	15	50	79	0.647
	>2.3	5	10	21	
	Total	20	60	80	

**Table 3:** Association between PPTG and SSS

		SSS			X <sup>2</sup> Value
PPTG		≤25	>25	Total	
	≤2.3	9	34	43	0.548
	>2.3	12	25	37	
	Total	21	59	80	

In this study the number of patients with severe stroke (SSS≤25) were 20 of which 15 had F TG≤2.3mmol/l and only 5 patient had F TG>2.3mmol/l & 9 had PPTG≤2.3mmol/l and 12 had PPTG>2.3mmol/l.

The number of patients with mild to moderate stroke (SSS>25) were 60 of which 50 had F TG≤2.3mmol/l and 10 patients had F TG>2.3mmol/l & 34 had PPTG≤2.3mmol/l and 25 had PPTG>2.3mmol/l. In the present study, there is no association between FTG & PPTG Level and SSS at 5% level of significance.

**Table 4:** Association between FTG Level and age

		FTG Level (mmol/l)			X <sup>2</sup> Value
Age		<2.3	>2.3	Total	
	<65	42	10	52	0.827
	>65	23	5	28	
	Total	65	15	80	

**Table 5:** Association between PPTG Level and age

PPTG Level (mmol/l)					
Age		<2.3	>2.3	Total	X <sup>2</sup> Value
	<65	25	28	53	0.231
	>65	15	12	27	
	Total	40	40	80	

The number of patients <65 years were 52 of which 42 had FTG≤2.3mmol/l and 10 patients had F TG levels >2.3mmol/l & 25 patients had PPTG<2.3mmol/l and 28 had PPTG>2.3mmol/l.

The total number of patients in the age group ≥65 were 28 of which 23 had FTG≤2.3mmol/l and 5 patients had TG levels >2.3mmol/l & 15 patients had PPTG<2.3mmol/l and 12 had PPTG>2.3mmol/l. From the results there is no association between FTG & PPTG Level and age at 5% level of significance.

**Table 6:** Association between SEX and SSS

SSS					
Sex		<25	>25	Total	X <sup>2</sup> Value
	Male	20	34	54	0.196
	Female	6	20	26	
	Total	26	54	80	

**Table 7:** Association between F TG Level and Sex of an individual

FTG Level (mmol/l)					
Sex		<2.3	>2.3	Total	X <sup>2</sup> Value
	Male	42	12	54	0.857
	Female	18	8	26	
	Total	60	20	80	

**Table 8:** Association between PP TG Level and Sex of an individual

PPTG Level (mmol/l)					
Sex		<2.3	>2.3	Total	X <sup>2</sup> Value
	Male	27	27	54	0.857
	Female	15	11	26	
	Total	42	38	80	

Of the 80 patients studied, 54 were males and 26 were females. Among the males, 42 had FTG≤2.3mmol/l and 12 had FTG levels >2.3mmol/l & 27 had PPTG≤2.3mmol/l and 27 had PPTG levels >2.3mmol/l. Of the 26 females 18 had FTG≤2.3mmol/l and 8 patients had FTG levels >2.3mmol/l & 15 had PPTG≤2.3mmol/l and 11 had PPTG levels >2.3mmol/l. In the present study, there is no association between FTG & PPTG Level and Sex of an individual at 5% level of significance.

**Table 9:** Association between HTN and SSS

SSS					
HTN		<25	>25	Total	X <sup>2</sup> Value
	No	12	24	36	0.648
	Yes	13	31	44	
	Total	25	55	80	

**Table 10:** Association between FTG Level and HTN

FTG Level (mmol/l)					
HTN		<2.3	>2.3	Total	X <sup>2</sup> Value
	No	30	6	36	0.645
	Yes	32	12	44	
	Total	62	18	80	

**Table 11:** Association between PPTG Level and HTN

PPTG Level (mmol/l)					
HTN		<2.3	>2.3	Total	X <sup>2</sup> Value
	No	25	11	36	0.031
	Yes	19	25	44	
	Total	44	36	80	

Out of 80 patients, 44 were hypertensive and 32 had FTG≤2.3mmol/l and 12 had FTG>2.3mmol/l & 19 had PPTG≤2.3mmol/l and 25 had PPTG>2.3mmol/l of the remaining 36 non hypertensive patients, 30 had FTG≤2.3mmol/l and 6 had FTG>2.3mmol/l & 25 had PPTG≤2.3mmol/l and 11 had PPTG>2.3mmol/l. There is an association between PPTG Level and hypertension (HTN) at 5% level of significance.

**Table 12:** Association between IHD and SSS

SSS					
IHD		<25	>25	Total	X <sup>2</sup> Value
	No	18	46	64	0.925
	Yes	6	10	16	
	Total	24	56	80	

**Table 13:** Association between FTG Level and IHD

FTG Level (mmol/l)					
IHD		<2.3	>2.3	Total	X <sup>2</sup> Value
	No	50	14	64	0.245
	Yes	12	4	16	
	Total	62	18	80	

**Table 14:** Association between PPTG Level and IHD

PPTG Level (mmol/l)					
IHD		<2.3	>2.3	Total	X <sup>2</sup> Value
	No	30	34	64	0.645
	Yes	8	8	16	
	Total	38	42	80	

Out of 80 patients, 16 were IHD patients of which 12 had FTG≤2.3mmol/l and 4 had FTG>2.3mmol/l & 8 had PPTG≤2.3mmol/l and 8 had PPTG>2.3mmol/l & of the remaining 64 patients, 50 had FTG≤2.3mmol/l and 14 had FTG>2.3mmol/l & 30 had PPTG≤2.3mmol/l and 34 had PPTG>2.3mmol/l.

## Discussion

### Fasting & post prandial triglycerides levels and severity of stroke

The present study involved 80 patients of acute ischemic stroke. Serum triglyceride levels are generally increased for 3–6 h after a meal [9]. Once postprandial hypertriglyceridemia occurs, it is exacerbated by the next meal and persists for the entire day. The most common pattern of dyslipidemia in type 2 diabetic patients is elevation of triglyceride levels and a decrease in HDL cholesterol levels. Therefore, measuring postprandial values for evaluation of hypertriglyceridemia in patients with diabetes is important. However, to our knowledge, the association between postprandial triglyceride (PPTG) levels and atherosclerosis has not been investigated.

One would thus surmise that postprandial lipid values would correlate better with Prognosis than fasting values. This has indeed been shown in recent epidemiologic studies. Bansal and colleagues [10] analyzed the prognostic value of fasting and nonfasting triglyceride levels in a cohort of 26,509

healthy women enrolled in the Women's Health Study. After 11.4 years of follow-up, PPTG but not fasting Triglycerides in the upper tertile of the distribution were associated with an almost 2-fold increase in cardiovascular disease even after adjustment for age, blood pressure, smoking, use of hormone therapy, total and high-density lipoprotein cholesterol, diabetes mellitus, body mass index, and high-sensitivity C-reactive protein.

It is important, however, not to dismiss triglyceride elevations in individuals who forget to fast for their regular blood draw. Such an elevation should prompt discussion with the patient about the link between such triglyceride elevations and insulin resistance, future cardiovascular disease, and the importance of lifestyle modifications (quality and quantity of food, weight management, physical activity) [11].

Postprandial remnant particles of triglyceride-rich lipoproteins have also been found to be an independent risk factor for early atherosclerosis and may be responsible for the increased rate of carotid intima-media thickness in these subjects. The difference in the results of these studies may be related to measurement of triglycerides in the fasting versus postprandial state.

Hypertriglyceridemia may also contribute to cerebrovascular disease through its effects on thrombosis. This effect is produced by thrombogenic alterations of the coagulation system as well as elevations in plasma viscosity. Previous study reported that 18 patients with severe hypertriglyceridemia (mean fasting plasma triglyceride level 504.4 mg/dl) had higher concentrations of plasma fibrinogen, lower fibrinolytic activity and higher levels of clotting factor Xc compared to normolipidemic controls [11]. Several studies have not found triglycerides to be an independent risk factor for IS [12]. For example, Bowman *et al.* [12] Conducted a prospective, randomized, nested case-control study among patients from the Physician Health Study, which included 296 fatal and nonfatal IS in white male physicians and an equal number of controls.

Another study consisting of 121 consecutive acute ischemic stroke patients showed that a higher ( $\geq 1.70$  mmol/l) fasting serum triglyceride level (within 24h after admission) was associated with a lower infarct volume ( $p = 0.014$ ) [13].

The biological mechanism responsible for association between TG level and stroke severity is unknown. Low TG level may reflect poor nutritional status of the patient.

**Table 15:** Shows the Study population and FTG

Author/study	Study population	FTG in mmol/l associated with SSS $\leq 25$ Mean $\pm$ SD	FTG in mmol/l associated with SSS $> 25$ Mean $\pm$ SD
Present study	80 patients	1.23 $\pm$ 0.50	1.28 $\pm$ 0.51
Tomasz Dziedzic [15] and others	863 patients	1.4 $\pm$ 0.6	1.7 $\pm$ 1.3

In both the studies, the level of serum triglyceride was lower in patients with severe stroke as compared to the levels in mild/moderate stroke patients, which was statistically not significant.

#### Age and fasting & post prandial triglyceride levels

Triglycerides increase gradually in men until about age 50 years and then decline slightly but in women, they continue to increase with age. The prevalence of mild

hypertriglyceridemia is slightly more in men beginning at age 30 years and women starting at age 60 years. Fasting triglycerides gradually increase in age  $< 65$  years and severity of stroke is less (SSS  $< 25$ ). Post-prandial triglycerides gradually increase in age  $< 65$  years and severity of stroke is more when compared to age  $> 65$  years.

The present study did not show any statistically significant association between FTG & PPTG Level and age. However, in the present study no statistically significant association between FTG & PPTG level and sex of an individual could be noticed.

#### IHD and Triglyceride Levels

The role of triglycerides as a risk factor of ischemic heart disease (IHD) remains controversial. In the Copenhagen Male Study on 2906 white men who were initially free of overt cardiovascular disease, an 8-year follow-up period showed that a clear gradient of risk of IHD was found with increasing triglyceride levels within each level of HDL cholesterol, including high HDL cholesterol levels, which are thought to provide protection against IHD. However, the present study did not show any significant association between FTG & PPTG Level and IHD.

#### Hypertension and Triglyceride Levels

Essential hypertension is frequently associated with metabolic abnormalities including glucose intolerance, hypertriglyceridemia and enhanced postprandial lipemia [14]. In this study there is statistically significant association between PPTG level and hypertension.

#### Conclusion

In this study, based on our review of the existing evidence, we conclude that post prandial serum triglyceride level should also be measured to assess the severity of stroke. In this study patients with HTN have higher PPTG value and has increased risk for stroke. So dietary and lifestyle modifications should be encouraged. To assess the benefit of pharmacologic and dietary TG reduction with respect to primary and secondary prevention of ischemic stroke.

#### Acknowledgment

The author thankful to management of Bhaskar Medical College & General Hospital for providing all the facilities to carry out this work.

#### Conflict of Interest: None

#### References

1. Stalenhoef AF, De Graaf J. Association of fasting and nonfasting serum triglycerides with cardiovascular disease and the role of remnant-like lipoproteins and small dense LDL. *Curr Opin Lipidol.* 2008; 19:355-61.
2. Bansal S, Buring JE, Rifai N, Mora S, Sacks FM, Ridker PM. Fasting compared with nonfasting triglycerides and risk of cardiovascular events in women. *JAMA.* 2007; 298:309-16.
3. Nordestgaard BG, Benn M, Schnohr P, Tybjaerg-Hansen A. Nonfasting triglycerides and risk of myocardial infarction, ischemic heart disease, and death in men and women. *JAMA.* 2007; 298:299-308.
4. Ryu JE, Howard G, Craven TE, Bond MG, Hagaman AP, Crouse JR. 3rd. Postprandial triglyceridemia and

- carotid atherosclerosis in middle-aged subjects. *Stroke* 1992; 23:823-8.
- 5. Oka R, Kobayashi J, Miura K, Nagasawa S, Moriuchi T, Hifumi S *et al.* Difference between fasting and nonfasting triglyceridemia: the influence of waist circumference. *J Atheroscler Thromb.* 2009; 16:633-40.
  - 6. Rector RS, Linden MA, Zhang JQ, Warner SO, Altena TS, Smith BK *et al.* Predicting postprandial lipemia in healthy adults and in at-risk individuals with components of the cardio metabolic syndrome. *J Clin. Hypertens (Greenwich)*. 2009; 11:663-71.
  - 7. Enkhmaa B, Ozturk Z, Anuurad E, Berglund L. Postprandial lipoproteins and cardiovascular disease risk in diabetes mellitus. *Curr Diab Rep.* 2010; 10:61-9.
  - 8. Gowenlock AH. Lipids and lipoproteins; Varley's practical clinical biochemistry. 6th Edn. New Delhi: CBS publishers and distributors, 1988, 464-466.
  - 9. Bjorkgren J, Karpe F, Milne RW, Hamsten A. Differences in apolipoprotein and lipid composition between human chylomicron remnants and very low density lipoproteins isolated from fasting and postprandial plasma. *J Lipid Res.* 1998; 39:1412-1420.
  - 10. Bansal S, Buring JE, Rifai N, Mora S, Sacks FM, Ridker PM. Fasting compared with nonfasting triglycerides and risk of cardiovascular events in women. *JAMA.* 2007; 298:309-316.
  - 11. National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Third report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation.* 2002; 106:3143-3421.
  - 12. Hackman A, Abe Y, Insull W Jr, Pownall H, Smith L, Dunn K *et al.* Levels of soluble cell adhesion molecules in patients with dyslipidemia. *Circulation.* 1996; 93:1334-1338.
  - 13. Pikić S, Milević D, Trkulja V, Kidemet-Piskac S, Pavlicek I, Sokol N. Higher serum triglyceride level in patients with acute ischemic stroke is associated with lower infarct volume on CT brain scans. *Eur. Neurol.* 2006; 55(2):89-92.
  - 14. Venkatesan A, Hemalatha A, Zachariah B, Selvaraj N, Sathiyapriya. Effect of smoking on lipid profile and lipid peroxidation in normal subjects. *Indian J Physiol Pharmacol.* 2006; 50(3):273-278.
  - 15. Tomasz D, Agnieszka S, Elzbieta AG, Szczudlik A. Lower serum triglyceride level is associated with increased stroke severity. *Stroke.* 2004; 35:e151-e152.