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Association Between Plasma Fibrinogen Levels and Carotid Intima–Media Thickness in Acute Ischaemic Stroke Patients: *A Hospital-Based Cross-Sectional Study in Myanmar*

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Abstract

Background: *Acute ischaemic stroke (AIS) is a major cause of mortality and long term disability worldwide. Atherosclerosis is a key underlying mechanism, influenced by traditional vascular risk factors and inflammatory processes. Plasma fibrinogen, an acute phase reactant, and carotid artery intima–media thickness (CIMT), a surrogate marker of subclinical atherosclerosis, have both been associated with cerebrovascular disease. However, data from Myanmar populations remain limited.*

Objective: *To evaluate the association between plasma fibrinogen levels and CIMT in patients with acute ischaemic stroke.*

Methods: *This hospital based cross sectional descriptive study was conducted at No. (1) Defence Services General Hospital (1000 bedded) and No. (2)Military Hospital (500 bedded), Yangon, from October 2010 to September 2012. Fifty five patients with CT confirmed acute ischaemic stroke were enrolled. Demographic data, body mass index (BMI), and vascular risk factors were recorded. Plasma fibrinogen was measured within 72 hours of stroke onset. CIMT was assessed bilaterally using B mode carotid Doppler ultrasonography. Associations were analysed using Fisher's Exact Test and chi square test.*

Results: *The mean age was 53.02 ± 9.18 years; 72.7% were male. Elevated plasma fibrinogen (>4.0 g/L) was observed in 92.7% of patients (mean 5.09 g/L). Increased CIMT (>0.7 mm) was present in 81.8%. A significant positive association was found between elevated plasma fibrinogen and increased CIMT ($p < 0.001$). Age, male sex, obesity, smoking, hypertension, diabetes mellitus, and hypercholesterolaemia were all significantly associated with increased CIMT ($p < 0.05$).*

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Conclusion: *Higher plasma fibrinogen levels are significantly associated with increased CIMT in acute ischaemic stroke patients, supporting the role of inflammation in atherosclerotic stroke. CIMT also correlates strongly with traditional cerebrovascular risk factors. Plasma fibrinogen and CIMT may serve as useful markers for vascular risk stratification in AIS.*

Keywords: *Acute ischaemic stroke; plasma fibrinogen; carotid intima–media thickness; atherosclerosis; inflammation.*

INTRODUCTION

Stroke is a leading cause of death and disability worldwide, with ischaemic stroke accounting for approximately 85% of all cases. Atherosclerosis of large and medium-sized arteries remains a major pathogenic mechanism. Traditional vascular risk factors—age, male sex, obesity, hypertension, diabetes mellitus, hypercholesterolaemia, and smoking—contribute significantly to disease burden. In addition, inflammation plays a critical role in atherogenesis, plaque destabilization, and thrombosis (Harlan, 2004).

Plasma fibrinogen is a key coagulation factor and inflammatory marker associated with increased blood viscosity, platelet aggregation, and endothelial dysfunction. Elevated fibrinogen levels have been linked to both cardiovascular and cerebrovascular events (Tint lwin ,2006). Carotid artery intima–media thickness, measured non-invasively by ultrasonography, is a validated marker of subclinical atherosclerosis and predictor of future stroke and myocardial infarction.

Although studies from Western and Asian populations suggest a relationship between fibrinogen levels and CIMT, data from Myanmar are scarce. Understanding this relationship may help refine vascular risk assessment and preventive strategies in local populations.

AIM AND OBJECTIVES

Aim

To determine the association between plasma fibrinogen levels and carotid artery intima–media thickness in acute ischaemic stroke patients.

Objectives

1. To measure plasma fibrinogen concentrations in acute ischaemic stroke patients.
2. To assess the extent of carotid atherosclerosis using CIMT.
3. To analyze the association between plasma fibrinogen, CIMT, and cerebrovascular risk factors.

RESEARCH METHODOLOGY

Study Design : Hospital based cross-sectional descriptive study

Sample size: Total 55 acute ischaemic stroke patients were studied.

Study Period : October, 2010 to September, 2012

Study Site : Medical wards and Neuromedical wards of No (1) Defence Services General Hospital, 1000 bedded, Mingalardon and No (2) Military Hospital, 500 bedded, Yangon

Sample Size

Minimum of (55) patients

By using sample size determination in health studies, WHO Geneva 1991

$$N = \frac{z^{2\times p\times q}}{d^2}$$

Where

z = confidence interval = 1.96 (95 %)

p = proportion or prevalence or a best guess about the value of interest = 0.4

q = 1 – p

d = precision or margin of error = 0. 13(%)

$$N = \frac{1.96^2\times 0.6}{0.13^2} = 54.555\sim 55$$

Ethical Consideration: Ethical clearance was obtained by Ethical committee of Defence Services Medical Academy.

RESEARCH PROCEDURE

Acute ischaemic stroke patients were recruited from Medical wards & Neuro-medical wards within 72 hours

↓

History, physical examination & basic investigations (CP, ESR, blood sugar, cholesterol, CXR, ECG etc) and CT head scan

↓

Exclusion Criteria

- Recent MI, major surgery or trauma in previous month and history of previous stroke
- Chronic inflammatory condition, acute or chronic infection like TB, malignancy and liver disease
- History of taking hormone replacement therapy and birth controlling oestrogen pills

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The quantitative determination of fasting plasma fibrinogen within 48- 72 hours- fasting 3.6 ml venous blood and 0.6 ml of sodium citrated ,centrifuged took out plasma within 1 hr by PT drives fibrinogen assay. normal - 1.5-4.0 g/l)

Carotid intima media thickness (CIMT) - the mean of the maximum wall thickness for far walls on both the left and right sides, and measured at 1.5 cm proximal to the carotid bulb, by using the high resolution B-mode ultrasonography

Data analysis

RESULT

- **Most of patients were Age ≥ 50 years – 33 patients (60%) and < 50 years old- 22 patients (40%) , and 40 (72.7%) male and 15(27.3 %) female**
- **Obese – 33 (60.0%), severe obese patients – 16 (29.09%) , and normal body weight – 6 (10.9%)**
- **Smoker 36 (65.5%) and non – smoker 19 (34.5%)**
- **Diabetics Mellitus 24 (43.6%) and Non- DM 31 (51.4%)**
- **Hypertension 41 (74.5%) and Non- Hypertensive 14(25.5%)**
- **Hypercholesterolaemia 23(41.8%) and normal cholesterol 32(58.2%)**

Table 1. Measurement of plasma fibrinogen level in acute ischemic stroke Patients

Plasma Fibrinogen Level	Acute Ischaemic Stroke Patients	
	No. of patients	Percentage(%)
Normal (1.5 - 4g / l)	4	7.27
Increased (< 4g / L)	51	92.7

Minimal Fibrinogen level - 3.8g / l maximal - 5.9g / l Mean -5.09g/1

Table 2. Measurement of Carotid IMT in acute ischaemic stroke patients

Carotid Intima Media Thickness (IMT)	Acute Ischemic Stroke Patients	
	No. of patients	Percentage(%)
≤ 0.7 mm	10	18.1
> 0.7 mm	45	81.8

Minimal carotid intima media thickness (IMT) -0.7 mm, Maximum – 0.9 mm. Mean- 0.83mm

Table 3. Comparison of CVD risk (BMI) in extent of Carotid artery IMT

BMI (kg/m2)	IMT ≤0.7 mm		IMT >0.7 mm		P value
	Number	(%)	Number	(%)	< 0.002
18- 23	3	50	3	50	
24-29	7	21.2	26	78.8	
>30	0		16	100	

This table shows that all severe obese patients had increased carotid IMT. And among 33 obese patients, 26 (78.8%) patients had greater carotid IMT and the rest were normal carotid IMT. In normal body weight patients with acute ischaemic stroke, it was same percentage in carotid IMT. So, the greater body weight, the thicker carotid IMT in acute ischaemic stroke patients. Significant P value was <0.002.

Table 4. Comparison of CVD risk (Smoking, Hypertension, Diabetic Mellitus and Hypercholesterolemia) in extent of Carotid artery IMT

CVD Risks		IMT≤ 0.7 mm		IMT >0.7 mm		P values
Smoking	+	9	25%	27	75%	<0.003
	-	10	55.5%	9	44.4%	
Hypertension	+	5	12.2%	36	87.8%	<0.002
	-	5	35.7%	9	64.3%	
Diabetic Mellitus	+	0		24	100%	<0.003
	-	10	32.3%	21	67.7%	
Hypercholesterolemia	+	0		23	100%	<0.003
	-	10	31.2%	22	68.8%	

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This table shows that carotid IMT was greater in acute ischemic stroke patients with history of smoking than patients without smoking history. Significant P value was <0.003. And carotid IMT was thicker in patients with history of hypertension than non - hypertensive patients. It also had significant P value was <0.002. Moreover, diabetic mellitus, one of risk factors of CVD, was associated with increased carotid IMT. All diabetic patients had greater carotid IMT in this study. P value was <0.003. And then all patients with hypercholesterolemia had greater carotid IMT and significant P value was <0.003.

Table 5. Relationship between plasma fibrinogen level and extent of Carotid Artery IMT in acute ischaemic stroke patients

Plasma Fibrinogen Level	Carotid IMT		P value
	≤0.7mm	> 0 ,7 mm	< 0.001
Normal (1.5 - 4g / l)	0	4(100%)	
Increased (> 4g / l)	10(19.6%)	41 (80.4%)	

All AIS patients with normal plasma fibrinogen level had greater carotid IMT. And normal carotid IMT was also found in AIS patients with raised plasma fibrinogen level. Elevated plasma fibrinogen (>4.0 g/L) was detected in 51 patients (92.7%). Increased CIMT (>0.7 mm) was observed in 45 patients (81.8%). The relationship between plasma fibrinogen and carotid artery IMT was calculated by using Fisher's Exact Test method. P value was < 0.001 was considered significant. Age, male sex, obesity, smoking, hypertension, diabetes mellitus, and hypercholesterolaemia were all significantly associated with increased CIMT.

DISCUSSION

This study demonstrates a significant and robust positive association between plasma fibrinogen levels and carotid intima–media thickness (CIMT) in patients with acute ischaemic stroke. Our findings support the hypothesis that elevated fibrinogen reflects an underlying pro-inflammatory and prothrombotic state that contributes to the development and progression of carotid atherosclerosis, thereby increasing the risk of acute cerebral ischaemic events.

Plasma fibrinogen is a well-established acute-phase reactant and plays a central role in thrombogenesis, blood viscosity, platelet aggregation, and endothelial dysfunction. Elevated fibrinogen levels may accelerate atherosclerotic plaque formation and instability, particularly in the carotid arteries, which are a major source of embolic stroke. The high prevalence of raised fibrinogen observed in our acute stroke cohort underscores its importance as a biological marker linking systemic inflammation to vascular pathology. These results are consistent with large population-based studies, including the Atherosclerosis Risk in Communities (ARIC) study, which demonstrated fibrinogen as an independent predictor of carotid atherosclerosis and stroke risk (Chambless LE et al., 2000).

In this study, patients with increased CIMT had significantly higher mean fibrinogen levels compared with those with lower CIMT values, with a strong statistical association ($p < 0.001$). This finding suggests a dose–response relationship between systemic inflammation and structural arterial wall changes. Increased CIMT reflects early and advanced atherosclerosis and has been validated as a surrogate marker for generalized vascular disease. The observed association supports the concept that fibrinogen not only reflects inflammatory burden but may actively participate in the atherothrombotic process leading to acute ischaemic stroke.

Traditional vascular risk factors, including increasing age, male sex, smoking, hypertension, diabetes mellitus, obesity, and hypercholesterolaemia, were also significantly associated with increased CIMT in our cohort. These findings are in agreement with previous regional and international studies and reinforce the multifactorial nature of atherosclerosis (Ohmar Saw, 2011). The interaction between conventional risk factors and inflammatory biomarkers such as fibrinogen likely amplifies vascular injury and accelerates carotid artery disease.

The CIMT cut-off value used in this study was comparatively lower than that reported in Western populations; however, this reflects population-specific reference values derived from Myanmar-based studies. Previous local research has identified a CIMT value greater than 0.7 mm as an independent risk factor for acute ischaemic stroke (Tin Htar Nwe, 2005). Our findings align with this evidence and highlight the importance of using ethnicity- and population-appropriate CIMT thresholds when assessing stroke risk in different regions.

From a clinical perspective, the demonstrated association between elevated fibrinogen and increased CIMT suggests that fibrinogen may serve as a useful adjunctive biomarker for identifying high-risk individuals with subclinical or established carotid atherosclerosis. Early detection of elevated fibrinogen levels, in conjunction with CIMT assessment, may aid in improved risk stratification and targeted preventive strategies, particularly in resource-limited settings where advanced imaging modalities are not widely available.

Several limitations of this study should be acknowledged. The relatively small sample size and cross-sectional design limit the generalizability of the findings and preclude definitive conclusions regarding causality. Additionally, fibrinogen and CIMT were measured at a single time point during the acute phase of stroke, which may be influenced by the acute inflammatory response. Longitudinal studies with repeated measurements and larger sample sizes are needed to determine whether elevated fibrinogen precedes CIMT progression or simply reflects acute vascular injury.

Despite these limitations, this study provides important regional data supporting the role of plasma fibrinogen as a marker of carotid atherosclerosis in acute ischaemic stroke patients. Future prospective studies are warranted to explore the prognostic value of fibrinogen and its potential role as a therapeutic target in stroke prevention.

CONCLUSION

This study demonstrates a significant association between elevated plasma fibrinogen levels and increased carotid intima–media thickness in patients with acute ischaemic stroke, highlighting the close interplay between systemic inflammation, thrombogenesis, and carotid atherosclerosis. Increased CIMT was also strongly associated with established cerebrovascular risk factors, underscoring the multifactorial nature of stroke pathophysiology. Elevated plasma fibrinogen levels are significantly associated with increased carotid intima–media thickness in acute ischaemic stroke patients. CIMT is also strongly related to conventional

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cerebrovascular risk factors. Combined assessment of plasma fibrinogen and CIMT may improve vascular risk stratification and guide preventive strategies in acute ischaemic stroke. The combined assessment of plasma fibrinogen and CIMT may provide incremental value in vascular risk stratification and facilitate early identification of high-risk individuals. These findings support the potential utility of integrating inflammatory biomarkers with non-invasive vascular imaging to inform targeted preventive and secondary prevention strategies in acute ischaemic stroke. Further large-scale prospective studies are warranted to confirm these observations and clarify their prognostic and therapeutic implications.

RECOMMENDATIONS

Larger prospective studies incorporating multivariate and regression analyses are recommended to determine the independent role of fibrinogen in carotid atherosclerosis. Evaluation of additional inflammatory biomarkers and interventional strategies targeting inflammation may further enhance stroke prevention.

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