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International Journal of Clinical Case Reports and Reviews
Aloy-Amadi Oluchi Chinwe *

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Research Article

Haemostatic Alterations in Patients with Myocardial Ischemia in Owerri, Nigeria

Wealthy-Emmanuel Enajite [1], Ebiliekwe Chisom Vivian [2], Aloy-Amadi Oluchi Chinwe [1*]

[1]Department of Hematology and Blood Transfusion Science, Faculty of Medical Laboratory Science, Federal University, Otuoke, Nigeria

[2]Department of Medical Laboratory Science, Imo State University, Owerri, Nigeria

***Corresponding Author:** Aloy-Amadi Oluchi Chinwe, Department of Hematology and Blood Transfusion Science, Faculty of Medical Laboratory Science, Federal University, Otuoke, Nigeria.

Received Date: July 17, 2026 | **Accepted Date:** July 28, 2026 | **Published Date:** August 06, 2026

Citation: Wealthy E. Enajite, Ebiliekwe C. Vivian, Oluchi Chinwe AA, (2026), Haemostatic Alterations in Patients with Myocardial Ischemia in Owerri, Nigeria, *International Journal of Clinical Case Reports and Reviews*, 36(5); **DOI:10.31579/2690-4861/1145**

Abstract:

Myocardial ischemia is associated with inflammation and coagulation abnormalities, yet routine hemostatic profiling is infrequently incorporated into early assessment in many low-resource settings. This cross-sectional study evaluated prothrombin time (PT), activated partial thromboplastin time (APTT), and fibrinogen in 30 adults with clinically and biochemically confirmed myocardial ischemia and 30 age-matched apparently healthy controls at a tertiary hospital in Owerri, Nigeria. Venous blood was collected into trisodium citrate, and plasma PT, APTT, and fibrinogen were determined using standard coagulometric methods and the Clauss assay. Data were analyzed using independent-samples t test and Pearson correlation at $p < 0.05$. Patients with myocardial ischemia had significantly higher mean PT, APTT, and fibrinogen than controls: PT (13.13 ± 1.34 vs 12.11 ± 0.63 s), APTT (33.39 ± 4.23 vs 30.61 ± 1.87 s), and fibrinogen (380.47 ± 87.41 vs 325.44 ± 39.40 mg/dL). No significant sex-based differences were observed among cases. Fibrinogen showed strong positive correlations with PT and APTT. These findings indicate a measurable hemostatic disturbance in myocardial ischemia and support further evaluation of routine coagulation testing in cardiovascular risk assessment.

Key words: APTT; fibrinogen; myocardial ischemia; Nigeria; prothrombin time

1.Introduction

Myocardial ischemia results from an imbalance between myocardial oxygen supply and demand and remains a major contributor to cardiovascular morbidity and mortality worldwide [6, 13].

It commonly develops in the setting of coronary atherosclerosis and thrombotic obstruction, which impair coronary blood flow and compromise myocardial perfusion [15, 21, 26].

When ischemia is sustained, irreversible myocardial injury may follow. [4, 9, 13]

The pathogenesis of myocardial ischemia involves endothelial dysfunction, inflammation, platelet activation, and activation of the coagulation cascade. [15, 20,25]

These processes promote thrombus formation and contribute to both acute and chronic ischemic syndromes. [3, 11,12]

Fibrinogen, prothrombin time (PT), and activated partial thromboplastin time (APTT) are practical laboratory markers that may reflect hemostatic disturbance and inflammatory activation. [14, 18, 21, 25]

In many low-resource settings, coagulation screening is not routinely integrated into the clinical evaluation of patients with myocardial ischemia, limiting recognition of hemostatic abnormalities that may have diagnostic or prognostic value. [4, 13,24]

The present study therefore assessed PT, APTT, and fibrinogen in patients with myocardial ischemia in Owerri, Nigeria, compared with apparently healthy controls, and examined whether these parameters differed by sex and whether fibrinogen correlated with clotting indices.

2.0 Materials and Methods

2.1 Study design and setting

This was a cross-sectional study conducted at the Cardiology Clinic of Imo State Specialist Hospital, Umuguma, Owerri, Imo State, southeastern Nigeria, between March and April 2025.

2.2 Study population

The study included 60 participants: 30 patients with myocardial ischemia and 30 apparently healthy age-matched controls. Patients were recruited consecutively from the Cardiology Clinic and Medical Wards. Controls

were selected from hospital staff and accompanying relatives of non-cardiac patients after clinical screening.

2.3 Eligibility criteria

Inclusion criteria were adults aged 18 years and above, clinically and biochemically diagnosed with myocardial ischemia, and apparently healthy controls without known cardiovascular disease. Exclusion criteria were history of liver disease, renal disorder, coagulopathy, recent major surgery, active malignancy, pregnancy, lactation, or use of anticoagulant or antiplatelet drugs within the preceding two weeks. Participants who declined consent were excluded.

2.4 Sample size

Sample size was determined using the formula described by Araoye 1 with a prevalence of 1.3%, a 95% confidence level, and a precision of 5%. The minimum sample size obtained was 30 participants per group, giving a total of 60 participants.

2.5 Sample collection and processing

Five milliliters of venous blood was collected aseptically from the antecubital vein of each participant. Four and a half milliliters was transferred into trisodium citrate anticoagulant tubes at a 9:1 blood-to-anticoagulant ratio. Samples were gently mixed and transported in a cold box to the laboratory. Plasma was separated by centrifugation at 3000 rpm for 15 minutes and stored at −20°C until analysis. All analyses were completed within 48 hours of collection.

2.6 Laboratory analysis

PT was determined by standard coagulometric method using citrated plasma and thromboplastin-calcium reagent. 8,23

APTT was measured using a standard activated partial thromboplastin reagent with calcium chloride. [8,13]

Fibrinogen concentration was determined by the Clauss method using diluted citrated plasma and thrombin reagent. [18, 2]

Each assay was performed in duplicate, and the mean value was recorded.

Reference ranges were 11–13.5 seconds for PT, 25–35 seconds for APTT, and 150–400 mg/dL for fibrinogen. [13,24]

2.7 Statistical analysis

Data were analyzed using SPSS version 21. Results were presented as mean ± standard deviation. Group differences were evaluated using the independent-samples t test, and associations were assessed using Pearson correlation coefficient. Statistical significance was set at $p < 0.05$. 22, 24

2.8 Ethical consideration

Ethical approval was obtained from the Ethics Committee of Imo State Specialist Hospital, Umuguma, Owerri. Written informed consent was obtained from all participants before enrolment.

3.0 Results

Patients with myocardial ischemia had higher mean PT, APTT, and fibrinogen levels than controls. PT was 13.13 ± 1.34 seconds in patients and 12.11 ± 0.63 seconds in controls. APTT was 33.39 ± 4.23 seconds in patients and 30.61 ± 1.87 seconds in controls. Fibrinogen was 380.47 ± 87.41 mg/dL in patients and 325.44 ± 39.40 mg/dL in controls.

Patients versus Controls (Mean ± SD)

Parameter	Test (n=30)	Control (n=30)	t-value	p-value0.003*
PT (seconds)	13.13 ± 1.34	12.11 ± 0.63	3.79	<0.0001*
APTT (seconds)	33.39 ± 4.23	30.61 ± 1.87	3.30	0.002*
Fibrinogen (mg/dL)	380.47 ± 87.41	325.44 ± 39.40	3.14	0.003*

Table 3.1: Mean Values of PT, APTT and Fibrinogen in Myocardial Ischemia

KEY* Significant at $p < 0.05$. PT: Prothrombin Time, APTT: Activated Partial Thromboplastin Time, SD: Standard Deviation

No significant sex-based differences were observed in PT, APTT, or fibrinogen among patients with myocardial ischemia. Male patients had mean PT of 12.74 ± 1.26 seconds compared with 13.52 ± 1.34 seconds in female patients. Mean APTT was 32.62 ± 4.42 seconds in males and 34.16 ± 4.03 seconds in females. Fibrinogen levels were 376.04 ± 93.76 mg/dL in males and 384.91 ± 83.63 mg/dL in females.

Patients Based on Gender (Mean ± SD)

Parameter	Male(n=15)	Female(n=15)	t-value	p-value
PT (seconds)	12.74 ± 1.26	13.52 ± 1.34	-1.64	0.112
APTT (seconds)	32.62 ± 4.42	34.16 ± 4.03	-1.00	0.326
Fibrinogen (mg/dL)	376.04 ± 93.76	384.91 ± 83.63	-0.27	0.787

Table 3.2: Mean Values of PT, APTT and Fibrinogen in Myocardial Ischemia

KEY: PT = prothrombin time; APTT = activated partial thromboplastin time; SD = standard deviation.

Fibrinogen showed a strong positive correlation with PT and APTT in patients with myocardial ischemia. The correlation between fibrinogen and PT was r = 0.794, and the correlation between fibrinogen and APTT was r = 0.677.

Dependent Variable	n	R-value	p-value
PT	30	0.794	<0.0001*
APTT	30	0.677	<0.0001*

Table 3.3: Pearson Correlation of Fibrinogen with PT and APTT in Myocardial Ischemia Patients

KEY: *: Significant p-value PT: Prothrombin Time APTT: Activated Partial Thromboplastin Time

4.0 Discussion

This study demonstrated that patients with myocardial ischemia had significantly higher PT, APTT, and fibrinogen levels than apparently healthy controls, suggesting a measurable hemostatic disturbance in this cohort. [13, 21, 24]

The absence of significant sex-based differences indicates that the observed coagulation changes were not modified by sex in this sample.

The elevated fibrinogen level is consistent with its role as an acute-phase reactant and a mediator of thrombosis. [18, 2, 21, 19]

Increased fibrinogen may enhance blood viscosity, promote platelet aggregation, and support fibrin clot formation, thereby contributing to a prothrombotic state. [18,26]

The significant prolongation of PT and APTT may reflect systemic inflammatory activity, altered clotting factor function, or consumption of coagulation factors in the setting of vascular injury. [9,17, 25]

The strong positive correlation between fibrinogen and both PT and APTT indicate that these variables changed in parallel in the patients studied.

Although fibrinogen is classically associated with procoagulant activity, the present findings suggest that hemostatic dysregulation in myocardial ischemia may be more complex and may involve concurrent inflammatory and coagulation pathway perturbations. [11, 12, 10, 20, 25]

This supports the view that simple coagulation assays may provide additional laboratory information in cardiovascular patients. [4,13,24]

From a clinical perspective, the findings support the potential value of routine hemostatic profiling in the assessment of patients with myocardial ischemia, particularly in resource-limited settings where advanced testing may not be readily available. [13, 24] however, because this was a cross-sectional study, causality cannot be inferred.

Larger prospective studies are required to determine whether these parameters have prognostic value or can guide treatment decisions. [7, 21]

5.0 Conclusion

Myocardial ischemia in this study was associated with prolonged PT and APTT and increased fibrinogen concentration compared with controls. No significant sex-related differences were observed among patients. The findings suggest that routine coagulation testing may be a useful adjunct in the laboratory evaluation of myocardial ischemia, although further prospective studies are needed to confirm its clinical value.

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DOI:10.31579/2690-4861/1145