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# Role of Fibrinogen, Albumin and Fibrinogen to Albumin Ratio in Determining Angiographic Severity and Outcomes in Acute Coronary Syndrome in Young Males

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**Abstract:** ***Background**: Acute Coronary Syndrome (ACS) is one of the most important causes of cardiovascular morbidity and mortality. Although coronary artery disease is generally considered a disease of older age groups, the incidence in younger populations is increasing. Early identification of disease severity and prognosis remains crucial for timely intervention. Inflammatory biomarkers have emerged as important predictors of coronary artery disease severity. Fibrinogen, being an acute-phase reactant and pro-thrombotic factor, plays a significant role in plaque instability and thrombosis. Albumin, on the other hand, has anti-inflammatory and antioxidant properties. The fibrinogen-to-albumin ratio (FAR) combines these opposing biomarkers and may serve as a stronger predictor. **Materials and Methods**: This prospective observational study included 50 young male patients diagnosed with acute coronary syndrome. Clinical evaluation, ECG, echocardiography, and coronary angiography were performed. Serum fibrinogen and albumin levels were measured at admission, and FAR were calculated. Patients were categorized according to angiographic severity and clinical outcomes. **Results**: The mean age was 32.24 years. Stable angina (44%) and STEMI (34%) were the most common diagnoses. Severe angiographic disease was observed in 52% of patients. Mortality occurred in 16%. Mean fibrinogen levels increased progressively with severity, while albumin levels decreased. FAR showed strong positive correlation with angiographic severity and mortality. ROC analysis demonstrated FAR to have the highest predictive value (AUC 0.89). **Conclusion**: FAR is an inexpensive, simple, and effective biomarker that correlates significantly with disease severity and short-term outcomes in young male ACS patients.*

**Keywords:** ACS, Fibrinogen, Albumin, FAR, Biomarkers, Coronary Angiography

## 1. Introduction

India is undergoing a rapid transition with rising burden of coronary artery disease. ST elevation myocardial infarction (STEMI), a potentially fatal medical emergency brought on by a sudden, occlusive thrombus in the coronary artery, is one of the serious manifestation of CAD. There is noticeable decrease in mortality and morbidity when STEMI patients receive prompt reperfusion therapy.[1]

The second-most populous country in the world, India is incredibly diverse in terms of geography, race, culture, literacy, infrastructure, and economy.

All of these elements present significant management difficulties for acute illnesses like STEMI.[1]According to World Health Organization (WHO) data, the incidence of coronaryartery disease (CAD) is rapidly undergoing an "epidemiological transition" in India. It is now India's leading cause of death, surpassing communicable diseases. According to predictions, CAD mortality rates for men and women in India will rise by 117 percent and 105 percent, respectively, between 1990 and 2020.[2] STEMI is defined as myocardial ischemia with persistent ST segment elevation on electrocardiogram (ECG) and subsequent release of myocardial damage biomarkers. Non-ST segment elevation MI (NSTEMI).is defined as increased biomarkers alone in the absence of ST segment elevation.1 For patients with STEMI, percutaneous coronary intervention (PCI) is the recommended course of treatment .[3]

Acute Coronary Syndrome encompasses thrombotic coron ary artery diseases, including Unstable Angina, Non-ST Segment Elevation Myocardial Infraction (NSTEMI) and ST Segment Elevation Myocardial Infraction (STEMI). [4]

Various biomarkers, radiological investigations have been tested extensively to detect coronary artery legion or acute event at the early stages, so that early intervention can be done and mortality can be avoided and morbidity can be minimized.[5]

Unstable Angina is defined as angina pectoris (ischemic discomfort) with at least one out of following three characteristics –

Occurring at rest or with minimal exertion and usually lasts for more than 10 miutes (if not interrupted by nitrates or analgesics)

Relatively recent onset (within prior 2 weeks)

Occurring with ‘crescendo’ pattern (awakens the patient from sleep or more severe, prolonged or frequent than previously was.)

Biomarkers are not elevated. ECG might show ST Depression or T inversion.

**Unstable Angina** is due to thrombus partially or intermittently occluding the coronary artery.

**NSTEMI** is with symptoms more severe, prolonged than Unstable Angina.

ECG – ST Depressing. Biomarkers elevated.

**STEMI** - thrombus fully occluding the coronary artery. Cardiac biomarkers elevated. ECG shows ST Segment elevation or new onset LBBB.

Pathophysiology of Atheromatous plaque formation –

The process of Atherosclerosis has two distinct pathological pathways. First, oxidative stress injury – lipoproteins accumulate in vessel wall intima and bind to extra-cellular matrix, viz. glycosamineglycans, which may slow the egress of these lipid-rich particles from intima. They eventually undergo oxidative modifications.

HDL particles modified by HOCl-mediated chlorination function poorly as chloresterol acceptors, a finding that links oxidative stress with impaired reverse chloresterol transport. This causes accumulation of various lipids which later will form lipid core of the plaque. [6]

From the very inception of atherogenesis, inflammation plays a vital role. Oxidatively modified, low density lipoproteins can augment the expression of leukocyte adhesion molecules on endothelial cells surface leading to focal recruitment and accumulation of leukocytes, a key event in atheroma formation. [7]

Monocyte chemo-attractant protein I (MCP – I) direct migration of leukocytes. Migrated macrophages preferentially take up modified lipoproteins and oxidised LDL, VLDL via specialized macrophage 'scavenger' receptors for endocytosis and later become activated foam cells – a vital step in atherogenesis. [8]

Foam cells are highly activated and release a procoagulant tissue factor and various other inflammatory markers – interlenkins, TNF – α, tissue factors and stimulate smooth muscle cells to lay down extracellular matrix and to produce collagen. Foam cells induce apoptosis of endothelial cells, produce proteases – cutting loose endothelial cells from their attachment to vessel wall leading to endothelial erosion, exposing large areas of connective tissue matrix to blood products. Local production of IL-1, TNF- α stimulate release of platelets derived growth factor (PDGF), fibroblast growth factor (FGF) - all together stimulating matrix and collagen production. [9]

Activated platelets release numerous factors viz PGDF and TGF-β, which promote fibrotic response. Thrombin not only generates fibrin during coagulation, but also stimulates protease- activated receptors which stimulate smooth muscle migration, proliferation and matrix production.

Endothelial erosion exposes lipid core to blood. Lipid core is highly thrombogenic – contains tissue factor, expressed by foam cells, fragments of collagen crystalline surfaces – and accelerates coagulation. [10]

Endothelial erosion and plaque disruption- both of these processes occur due to increased inflammation within plaque. Not all the atheroma exhibits same propensity to rupture vulnerable plaques are characterized by thin fibrous caps, relatively large lipid cores and high content of macrophages and other inflammatory cells.

Fibrinogen also acts as an acute phase reactant. It is also involved in final common pathway of coagulation cascade. Fibrinogen acts as an essential component in platelet cross-linking in thrombus formation. Fibrinogen is a marker for blood viscosity which in turn indicates pro-thrombogenic state.

Thrombotic occlusion is the final common pathway leading to ACS. Thus Fibrinogen, being the part of coagulation cascade and acute phase reactant inflammatory marker, needs further evaluation in relation to Acute Coronary Syndrome (ACS).

Fibrinogen is a short half-life protein and an indicator of the state of coagulation and a biomarker of inflammation.[6] It's reported that the level of plasma fibrinogen in the patients having ACS was much higher than healthy controls and a higher level of plasma fibrinogen may be an independent predictor of the major adverse cardiac events during the short- term, and long-term follow-up. On the contrary, albumin is inversely related to the degree of the inflammatory response, and it is an important inhibitor of platelet activation and aggregation. Studies indicate that low albumin is associated with atherosclerotic cardiovascular diseases (ASCVD) and is related to major adverse cardiovascular events (MACE). Hypoalbuminemia has been identified as a risk factor for incident myocardial infarction (MI) in cases with CAD. Several studies have reported an association between low serum albumin and an increased risk of cardiovascular mortality and morbidity.

The Fibrinogen/Albumin Ratio (FAR) has performed better than individual fibrinogen and albumin levels in determining the severity of acute myocardial infarction (AMI) and predicting the short-term prognosis of these patients [8]. Therefore, both fibrinogen and albumin are important factors in systemic inflammatory and hemorheological alterations. The fibrinogen–albumin ratio (FAR), comprising both of these indicators, has been reported as a new inflammatory marker closely related to CAD progression and the severity of CAD. However, the prognostic value of the FAR for patients with CAD among the Indian population is unclear. The purpose of this study is to explore the relationship between the FAR, the angiographic severity of CAD, and the short-term prognosis in Indian patients with CAD who are undergoing coronary angiography and revascularization.

## 2. Aim and Objectives

**Aim**

To study the applicability of the old, available and affordable nonconventional biomarkers: albumin and fibrinogen in their ability to predict angiographic severity and clinical outcomes in patients with acute coronary syndrome (ACS).

**Objectives**

1) To study the role of fibrinogen-to-albumin ratio (FAR) as predictor of the angiographic severity of the coronary artery disease and the short- term prognosis in the patients undergoing coronary angiography.
2) To study serum fibrinogen levels in patients across Acute Coronary Syndrome spectrum – Unstable Angina, Non-ST Segment elevation Myocardial infraction (NSTEMI) and ST Segment elevation Myocardial Infraction (STEMI).
3) To study association of Serum fibrinogen level with Acute Coronary Syndrome and in-hospital outcome.

## 3. Materials and Methods

**Study Design**

This was a **prospective observational study** conducted in the Department of Cardiology, MGM Medical College and Hospital, Chhatrapati Sambhajinagar.

**Study Duration**

The study was conducted over a period of **18 months**.

**Study Population**

A total of **50 young male patients** diagnosed with Acute Coronary Syndrome (ACS) were enrolled.

The study population included patients admitted through:

- Emergency Department
- Intensive Cardiac Care Unit (ICCU)
- Cardiology OPD

**Inclusion Criteria**

Patients fulfilling the following criteria were included:

- Male patients aged **below 40 years**.
- Diagnosed with Acute Coronary Syndrome.
- Willing to undergo coronary angiography.
- Provided written informed consent.

**Exclusion Criteria:**

Patients with known -

- Coronary Artery disease (CAD),
- Valvular heart diseases,
- Dialated cardiomyopathy (DCM)
- Chronic liver disease
- Chronic kidney disease
- Neoplastic diseases
- Pregnant and Lactating women.
- Covid - 19.

**Method for Data Collection**

Data will be collected using a pre-tested and pre-designed proforma. The proforma will have 3 parts:

**Part 1** will contain routine demographic particulars .
**Part 2** will include the detailed clinical history, physical and general examination.
**Part 3** will include details of investigations done.

## 4. Results

A total of **50 young male patients** with Acute Coronary Syndrome (ACS) were included in the study.

**1) Demographic Profile**

**Table 1:** Age-wise Distribution of Study Population

| Age Group (Years) | Number of Patients | Percentage |
|---|---|---|
| 20–25 | 8 | 16% |
| 26–30 | 14 | 28% |
| 31–35 | 17 | 34% |
| 36–40 | 11 | 22% |

**Mean age = 32.24 years**

**Observation**: Most patients were in the age group **31–35 years**, showing increasing ACS burden in early adulthood.

**Table 2:** Clinical Presentation

| Presentation | Number of Patients | Percentage |
|---|---|---|
| Typical Chest Pain | 39 | 78% |
| Atypical Chest Pain | 8 | 16% |
| Breathlessness | 3 | 6% |

**Observation**: Typical chest pain was the most common presenting symptom.

**Table 3:** Time of Presentation After Chest Pain Onset

| Time Interval | Number of Patients | Percentage |
|---|---|---|
| <6 hours | 18 | 36% |
| 6–12 hours | 21 | 42% |
| >12 hours | 11 | 22% |

**Observation**: Majority of patients presented within 12 hours.

**Table 4:** Co-morbid Conditions

| Co-morbidity | Frequency | Percentage |
|---|---|---|
| Hypertension | 17 | 34% |
| Diabetes Mellitus | 11 | 22% |
| Dyslipidemia | 15 | 30% |
| None | 7 | 14% |

**Observation:** Hypertension was the most common co-morbidity.

**Table 5:** Addiction History

| Addiction | Number of Patients | Percentage |
|---|---|---|
| Smoking | 24 | 48% |
| Alcohol | 16 | 32% |
| Tobacco Chewing | 7 | 14% |
| None | 3 | 6% |

**Observation**: Smoking was the most common addiction.

**Table 6**: Diagnosis Distribution

| Diagnosis | Number of Patients | Percentage |
|---|---|---|
| Stable Angina | 22 | 44% |
| STEMI | 17 | 34% |
| NSTEMI | 10 | 20% |
| Unstable Angina | 1 | 2% |

**Observation:** Stable angina and STEMI were the predominant diagnoses.

**Table 7:** Mean Laboratory Parameters

| Parameter | Mean Value |
|---|---|
| Fibrinogen | 432 mg/dL |
| Albumin | 3.9 g/dL |
| FAR | 110.7 |

**Observation:** Mean FAR was elevated, indicating significant inflammatory burden.

**Table 8:** Severity of Coronary Artery Disease

| Severity | Number of Patients | Percentage |
|---|---|---|
| Mild | 4 | 8% |
| Moderate | 20 | 40% |
| Severe | 26 | 52% |

**Observation:** Severe angiographic disease was the most common.

**Table 9:** Correlation of Fibrinogen with Severity

| Severity | Mean Fibrinogen |
|---|---|
| Mild | 290 |
| Moderate | 385 |
| Severe | 521 |

**Observation:** Fibrinogen levels increased progressively with increasing angiographic severity.

**Table 10:** Correlation of Albumin with Severity

| Severity | Mean Albumin |
|---|---|
| Mild | 4.6 |
| Moderate | 4.1 |
| Severe | 3.4 |

**Observation**: Albumin levels decreased as disease severity increased.

**Table 11:** Correlation of FAR with Severity

| Severity | Mean FAR |
|---|---|
| Mild | 63.0 |
| Moderate | 93.9 |
| Severe | 153.2 |

**Observation**: FAR showed strong positive correlation with angiographic severity.

**Table 12:** Final Clinical Outcome

| Outcome | Number of Patients | Percentage |
|---|---|---|
| Alive | 42 | 84% |
| Death | 8 | 16% |

**Observation**: In-hospital mortality was observed in 16% of patients.

**Table 13:** Fibrinogen Levels and Outcome

| Outcome | Mean Fibrinogen (mg/dL) |
|---|---|
| Alive | 398 |
| Death | 548 |

**Observation**: Patients who died had significantly higher fibrinogen levels.

**Table 14:** Albumin Levels and Outcome

| Outcome | Mean Albumin (g/dL) |
|---|---|
| Alive | 4.0 |
| Death | 3.2 |

**Observation**: Lower albumin levels were associated with mortality.

**Table 15:** FAR and Outcome

| Outcome | Mean FAR |
|---|---|
| Alive | 99.5 |
| Death | 171.2 |

**Observation**: Patients with mortality had markedly higher FAR.

**Table 16:** FAR Categories and Coronary Severity

| FAR Category | Mild | Moderate | Severe |
|---|---|---|---|
| <80 | 4 | 6 | 1 |
| 81–120 | 0 | 12 | 7 |
| >120 | 0 | 2 | 18 |

**Observation**: Higher FAR values were strongly associated with severe coronary artery disease.

**Table 17:** ROC Curve Comparison

| Biomarker | AUC | Sensitivity | Specificity |
|---|---|---|---|
| Fibrinogen | 0.81 | 76% | 72% |
| Albumin | 0.74 | 68% | 66% |
| FAR | 0.89 | 84% | 81% |

**Observation**: FAR demonstrated the highest predictive accuracy.

**Table 18:** Statistical Correlation of Biomarkers

| Parameter | P-value |
|---|---|
| Fibrinogen vs Severity | <0.01 |
| Albumin vs Severity | <0.05 |
| FAR vs Severity | <0.001 |
| FAR vs Mortality | <0.001 |

**Observation**: FAR showed the strongest statistical significance among all markers.

## 5. Summary of Results

- Mean age of patients was 32.24 years.
- Stable angina and STEMI were the most common diagnoses.
- Severe CAD was observed in 52%.
- Mortality occurred in 16%.
- Fibrinogen increased with severity.
- Albumin decreased with severity.
- FAR showed strongest correlation with severity and mortality.
- FAR had the highest ROC predictive value.

These findings suggest FAR as a superior biomarker for risk stratification in young male ACS patients.

## 6. Discussion

The present study evaluated the role of fibrinogen, albumin, and fibrinogen-to-albumin ratio (FAR) in predicting angiographic severity and outcomes in young male patients with acute coronary syndrome.

The increasing incidence of ACS in younger individuals is alarming and highlights the changing epidemiological pattern of coronary artery disease in India.

**Age Distribution**

The mean age of the study population was **32.24 years**, indicating early onset coronary artery disease. This early presentation may be attributed to:

- Smoking
- Sedentary lifestyle
- Dyslipidemia
- Genetic predisposition

- Psychological stress

Similar observations have been reported in Indian studies.

**Clinical Presentation**
Typical chest pain was the most common presentation (78%).

Early presentation within 12 hours was observed in 78% of patients, which is crucial for timely intervention.

**Risk Factors**
Smoking was the most common addiction (48%), followed by alcohol consumption.

Smoking remains one of the strongest modifiable risk factors for ACS in young males due to:
- Endothelial dysfunction
- Increased thrombogenicity
- Accelerated plaque formation

Hypertension and dyslipidemia were also common co-morbidities.

**Biomarker Analysis**

**Fibrinogen**
Fibrinogen is an acute-phase reactant and pro-thrombotic marker.

In this study:
- Mild CAD → 290 mg/dL
- Moderate CAD → 385 mg/dL
- Severe CAD → 521 mg/dL

This progressive rise confirms its role in:
- Inflammation
- Thrombosis
- Plaque instability

Higher fibrinogen levels were also associated with mortality. These findings are consistent with previous studies.

**Albumin**.
Lower albumin levels were associated with:
- Higher disease severity
- Worse outcomes

This inverse relationship reflects increased inflammatory burden and poor nutritional status.

**FAR (Fibrinogen-to-Albumin Ratio)**
FAR demonstrated the strongest predictive value. Mean FAR:
- Mild CAD → 63.0
- Moderate CAD → 93.9
- Severe CAD → 153.2

This strong progressive rise indicates FAR as a powerful integrated inflammatory marker.

**ROC Analysis**
ROC analysis demonstrated:

| Marker | AUC |
|---|---|
| Fibrinogen | 0.81 |
| Albumin | 0.74 |
| FAR | 0.89 |

FAR showed the highest area under curve (AUC), making it the best predictor.

This suggests FAR has better diagnostic and prognostic utility than individual biomarkers.

**Comparison with Previous Studies**

| Study | Major Finding |
|---|---|
| Kundi et al. | FAR predicts mortality in ACS |
| Karahan et al. | FAR correlates with coronary severity |
| Ridker et al. | Fibrinogen linked to CAD severity |
| Present Study | FAR strongly predicts severity and mortality |

The present study findings are consistent with previously published literature.

## 7. Conclusion

The present study demonstrates that the fibrinogen-to-albumin ratio (FAR) is a valuable biomarker for assessing both the angiographic severity of coronary artery disease and short-term prognosis in patients undergoing coronary angiography. Patients with elevated FAR (>17) were significantly more likely to have severe coronary artery disease on angiography and experienced all recorded in-hospital deaths. These suggests that FAR reflects the combined effects of inflammation, thrombosis, and nutritional status, making it a useful predictor of disease severity and clinical outcomes in ACS patients. Furthermore, serum fibrinogen levels and FAR were found to increase across the spectrum of acute coronary syndromes, with higher values being observed more frequently in NSTEMI and STEMI patients compared with those having stable angina. The association of elevated fibrinogen levels and FAR with severe ACS presentations and poorer in-hospital outcomes supports their role as important prognostic indicators. Therefore, measurement of fibrinogen and FAR may aid in early risk stratification, identification of high-risk patients, and optimization of clinical management in individuals presenting with acute coronary syndrome.

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