

Original Research Article

SERUM ZINC AND SELENIUM STATUS AND LIPID PROFILE ABNORMALITIES IN CRITICALLY ILL PATIENTS WITH ACUTE MYOCARDIAL INFARCTION: A CROSS-SECTIONAL OBSERVATIONAL STUDY

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ABSTRACT

Background: Myocardial infarction (MI) remains a leading cause of morbidity and mortality worldwide, with a rising burden in low- and middle-income countries such as India. Beyond traditional cardiovascular risk factors, oxidative stress and systemic inflammation play key roles in the pathogenesis and severity of MI. Essential trace elements such as zinc and selenium are critical components of antioxidant defense mechanisms, and their deficiency may aggravate myocardial injury. However, data evaluating trace element status and its association with lipid abnormalities in critically ill MI patients from the Indian population are limited. This study assessed serum zinc and selenium levels in critically ill patients with acute myocardial infarction, to compare lipid profile parameters between MI patients and healthy controls, and to evaluate the relationship between trace element levels and lipid profile abnormalities.

Materials and Methods: This hospital-based cross-sectional observational study included 50 critically ill patients diagnosed with acute myocardial infarction admitted to the intensive care unit and 50 age- and sex-matched healthy controls. Serum zinc and selenium levels were measured using atomic absorption spectrophotometry, and lipid profile parameters were estimated using standard enzymatic methods. Data were expressed as mean \pm standard deviation, and appropriate statistical tests were applied. Correlation analysis was performed to assess relationships between trace elements and lipid parameters.

Results: The mean age of MI patients was 56.08 ± 10.18 years, with a male predominance (60%). MI patients demonstrated significant dyslipidemia, with higher total cholesterol, LDL cholesterol, and triglyceride levels compared to controls ($p < 0.05$). Serum selenium ($77.46 \pm 13.46 \mu\text{g/L}$) and zinc ($53.79 \pm 25.08 \mu\text{g/dL}$) levels were significantly lower in MI patients than in controls ($p < 0.01$). Selenium deficiency was observed in 76% and zinc deficiency in 86% of MI patients. No significant correlations were found between serum zinc or selenium levels and lipid profile parameters, while strong positive correlations were observed among lipid parameters themselves.

Conclusion: Critically ill patients with acute myocardial infarction exhibit significant dyslipidemia along with profound deficiencies of zinc and selenium. These abnormalities appear to coexist rather than demonstrate a direct interaction, suggesting that micronutrient deficiency may contribute to myocardial injury primarily through oxidative and inflammatory pathways. Routine assessment of trace element status in critically ill MI patients may provide additional insights into disease severity and guide future interventional strategies.

Keywords: Myocardial infarction; Zinc; Selenium; Dyslipidemia; Trace elements; Oxidative stress; Critically ill patients; Lipid profile.

INTRODUCTION

Myocardial infarction (MI) remains one of the leading causes of morbidity and mortality worldwide and represents the most severe clinical manifestation of coronary artery disease.^[1] Although advances in diagnostic modalities and therapeutic interventions have contributed to improved outcomes in several high-income countries, the burden of myocardial infarction continues to rise in low- and middle-income countries such as India.^[2,3] Rapid urbanization, lifestyle transitions, altered dietary patterns, physical inactivity, and an increasing prevalence of metabolic risk factors have significantly contributed to this growing public health problem. In the Indian population, myocardial infarction often occurs at a younger age and is associated with increased disease severity and adverse outcomes.^[2,3]

Myocardial infarction occurs primarily as a result of acute interruption of coronary blood flow following rupture or erosion of an atherosclerotic plaque and subsequent thrombus formation.^[1] Atherosclerosis is now recognized as a chronic inflammatory disease characterized by lipid accumulation, endothelial dysfunction, oxidative stress, and immune-mediated vascular injury.^[4-6] These interrelated mechanisms play a crucial role in determining the extent of myocardial damage, clinical severity, and prognosis following infarction.

Dyslipidemia is a well-established and major modifiable risk factor in the pathogenesis of atherosclerosis and coronary artery disease.^[4,5] Elevated levels of total cholesterol, low-density lipoprotein cholesterol (LDL-C), and triglycerides, along with reduced high-density lipoprotein cholesterol (HDL-C), contribute to lipid deposition within the arterial wall, plaque formation, and increased vulnerability to plaque rupture.^[5,6] Lipid abnormalities not only predispose individuals to myocardial infarction but also influence disease progression, recurrence, and long-term cardiovascular outcomes.^[6]

Oxidative stress and systemic inflammation play central roles in both the initiation and progression of atherosclerosis and the extent of myocardial injury during acute myocardial infarction.^[4,7] Excessive generation of reactive oxygen species during ischemia and reperfusion leads to lipid peroxidation, endothelial injury, and cardiomyocyte apoptosis. In recent years, increasing attention has been directed toward the role of trace elements in cardiovascular health and disease, particularly in relation to their antioxidant and immunomodulatory functions.^[8]

Zinc and selenium are essential trace elements that are critically involved in antioxidant defense mechanisms, immune regulation, and maintenance of cellular integrity.^[9,10] Zinc functions as a structural and catalytic component of numerous enzymes and transcription factors, contributes to stabilization of cellular membranes, and modulates inflammatory signaling pathways.^[9] Selenium is an integral

component of several selenoproteins, including glutathione peroxidase, which protects cells against oxidative damage by neutralizing reactive oxygen species and limiting lipid peroxidation.^[11,12] Deficiency of zinc and selenium can impair endogenous antioxidant defenses, increase oxidative stress, and promote inflammatory responses, thereby potentially aggravating myocardial injury.^[10,13]

Critically ill patients with acute myocardial infarction are particularly vulnerable to micronutrient imbalance due to acute stress, systemic inflammation, increased metabolic demands, redistribution of trace elements, reduced nutritional intake, and impaired gastrointestinal absorption.^[14,15] These alterations may occur independently of traditional cardiovascular risk factors and are often overlooked during routine clinical evaluation in intensive care settings.

Several studies have demonstrated altered serum zinc and selenium levels in patients with acute myocardial infarction, suggesting their involvement in disease pathophysiology and prognosis.^[16,17] However, the relationship between trace element status and lipid profile abnormalities in myocardial infarction remains incompletely understood. Available evidence suggests that dyslipidemia and micronutrient deficiency frequently coexist in myocardial infarction, likely reflecting parallel pathophysiological processes mediated by oxidative stress and inflammation rather than a direct causal interaction.^[5,17]

Furthermore, there is a paucity of data from the Indian population evaluating serum zinc and selenium levels specifically in critically ill myocardial infarction patients admitted to intensive care units.^[3] Understanding the magnitude of trace element deficiency and its association with lipid abnormalities in this high-risk group may provide valuable insights into disease mechanisms and help identify patients who may benefit from targeted nutritional assessment and future interventional strategies.

Therefore, the present study was undertaken to evaluate serum zinc and selenium levels, lipid profile abnormalities, and their interrelationships in critically ill patients with myocardial infarction and to compare these findings with age- and sex-matched healthy controls.

Aims and Objectives of the Study

Primary Objectives

1. To assess serum zinc and selenium levels in critically ill patients with acute myocardial infarction.
2. To compare lipid profile parameters between myocardial infarction patients and age- and sex-matched healthy controls.

Secondary Objectives

1. To evaluate the prevalence of zinc and selenium deficiency in critically ill myocardial infarction patients.

- To analyze the relationship between serum zinc, selenium levels, and lipid profile parameters in myocardial infarction patients.

MATERIALS AND METHODS

Study Design: A hospital-based, cross-sectional observational study.

Study Setting: The study was conducted in a tertiary care teaching hospital involving the Departments of Medicine and Biochemistry.

Study Duration: The study was carried out over a defined study period after obtaining institutional ethical clearance.

Sample Size Calculation: The sample size for the present study was calculated based on previous study Uddin et al,^[16] reporting significant differences in serum zinc and selenium levels between myocardial infarction patients and healthy controls. Assuming a confidence level of 95% and a power of 80%, the minimum required sample size was estimated using standard statistical formulae for comparison of means between two independent groups. Considering the expected mean difference in trace element levels, standard deviation from earlier published literature, and allowing for possible attrition or non-availability of complete data, a total sample size of 50 critically ill myocardial infarction patients was considered adequate for the study. An equal number of age- and sex-matched healthy individuals were included as controls to allow meaningful comparison and statistical validity.

Study Population: The study included 50 critically ill patients diagnosed with myocardial infarction, admitted to the intensive care unit. Age- and sex-matched healthy individuals served as controls.

Inclusion Criteria

- Patients diagnosed with acute myocardial infarction based on clinical features, ECG changes, and cardiac biomarkers
- Critically ill patients admitted to ICU

- Age above 18 years
- Patients willing to participate in the study

Exclusion Criteria

- Patients with chronic liver disease, chronic kidney disease, or malignancy
- Patients on zinc or selenium supplementation
- Patients with acute or chronic inflammatory diseases other than MI
- Pregnant and lactating women

Sample Collection: Venous blood samples were collected under aseptic precautions after stabilization of the patient. Serum was separated and used for biochemical analysis.

Biochemical Analysis: Lipid profile parameters (Total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides) were estimated using standard enzymatic methods.

Serum zinc and selenium levels were measured using atomic absorption spectrophotometry (AAS), a sensitive and specific technique suitable for trace element estimation. All analyses were performed using calibrated instruments, and appropriate standards and controls were employed to ensure accuracy and reproducibility

Statistical Analysis: Data were expressed as mean \pm standard deviation. Comparison between groups was performed using appropriate statistical tests. Correlation analysis was carried out to evaluate relationships between zinc, selenium, and lipid profile parameters. A p value <0.05 was considered statistically significant.

RESULTS

A total of 50 critically ill subjects diagnosed with myocardial infarction (MI) were included in the present study. These subjects were evaluated for demographic parameters, lipid profile abnormalities, serum zinc and selenium levels, and inter-parameter correlations. The findings were compared with age-matched healthy controls.

Table 1: Age-wise Distribution of Critically Ill MI Subjects

Age Group (Years)	Number of Subjects (n)	Mean Age \pm SD (Years)
< 40	3	36.00 \pm 1.00
40–55	23	49.87 \pm 4.85
> 55	24	64.54 \pm 5.69
Total	50	56.08 \pm 10.18

Most MI subjects belonged to the 40–55 years (46%) and >55 years (48%) age groups. The mean age of MI subjects was 56.08 \pm 10.18 years, indicating a higher prevalence of myocardial infarction among older individuals

Table 2: Gender-wise Distribution of Critically Ill MI Subjects

Gender	Number (n)	Percentage (%)
Male	30	60
Female	20	40
Total	50	100

A male predominance was observed among MI subjects, with 60% males and 40% females, suggesting increased susceptibility of males to myocardial infarction

Table 3: Comparison of Lipid Profile Parameters in MI Subjects and Controls Across Age Groups

Parameter	Age Group	Control (mg/dl)	MI (mg/dl)	P Value
Total Cholesterol	<40	167 ± 41	153 ± 128	<0.01
	40–55	166 ± 47	170 ± 80	<0.01
	>55	170 ± 54	199 ± 82	0.054
	All	167 ± 46	183 ± 84	<0.05
LDL Cholesterol	<40	86 ± 31	94 ± 80	<0.01
	40–55	77 ± 38	89 ± 67	<0.01
	>55	75 ± 39	73 ± 66	<0.05
	All	81 ± 35	82 ± 67	<0.01
Triglycerides	<40	150 ± 88	75 ± 57	0.68
	40–55	142 ± 58	169 ± 103	<0.01
	>55	124 ± 47	186 ± 80	<0.05
	All	143 ± 72	171 ± 93	<0.05
HDL Cholesterol	<40	39 ± 17	45 ± 36	<0.05
	40–55	39 ± 13	50 ± 27	<0.01
	>55	39 ± 13	51 ± 24	<0.01
	All	39 ± 15	50 ± 25	<0.01

The lipid profile comparison revealed significant dyslipidemia among myocardial infarction (MI) subjects compared to controls. Overall, MI patients demonstrated significantly higher total cholesterol, LDL cholesterol, and triglyceride levels, particularly in the middle-aged and older groups, supporting their established role in atherogenesis and coronary artery disease. HDL cholesterol levels were also

significantly higher in MI subjects across all age groups, which may reflect acute-phase reactions, treatment effects, or compensatory metabolic responses following myocardial injury. These findings highlight the importance of comprehensive lipid profiling in the evaluation and management of MI patients.

Table 4: Serum Selenium Levels in MI Subjects Compared with Controls

Age Group	Control (µg/L)	MI (µg/L)	P Value
<40	143.35 ± 15.59	90.93 ± 10.78	<0.01
40–55	148.31 ± 22.72	76.76 ± 14.48	<0.01
>55	159.05 ± 26.76	76.44 ± 12.25	<0.01
All	147.74 ± 21.13	77.46 ± 13.46	<0.01

Serum selenium levels were significantly reduced in MI subjects across all age groups, indicating selenium deficiency.

Table 5: Distribution of Selenium Status in MI Subjects

Selenium Level (µg/L)	Number (n)	Percentage (%)
<70	38	76
70–95	10	20
95–125	2	4
>125	0	0
Total	50	100

A majority (76%) of MI subjects had selenium levels below the physiological range.

Table 6: Serum Zinc Levels in MI Subjects Compared with Controls

Age Group	Control (µg/dl)	MI (µg/dl)	P Value
<40	106.16 ± 63.88	37.33 ± 20.49	<0.05
40–55	109.94 ± 74.79	51.27 ± 26.42	<0.01
>55	76.20 ± 53.70	58.26 ± 23.94	<0.01
All	103.08 ± 67.70	53.79 ± 25.08	<0.01

MI subjects demonstrated significantly lower zinc levels compared to controls, indicating widespread zinc deficiency.

Table 7: Distribution of Zinc Status in MI Subjects

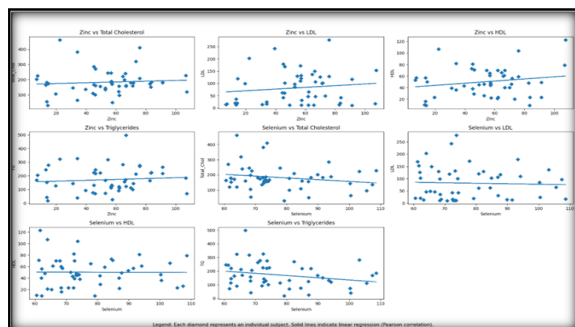
Zinc Level (µg/dl)	Number (n)	Percentage (%)
<80	43	86
80–105	5	10
105–135	2	4
>135	0	0
Total	50	100

A high proportion (86%) of MI subjects had zinc levels below the normal range.

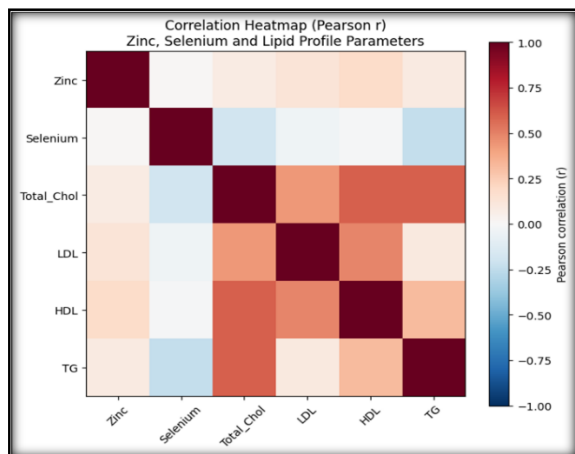
Table 8: Pairwise Correlation of Zinc, Selenium and Lipid Profile Parameters in MI Subjects

Parameter	Zinc	Selenium	Total Cholesterol	LDL	HDL	Triglycerides
Zinc	1	0.009 (0.950)	0.076 (0.598)	0.132 (0.360)	0.184 (0.201)	0.087 (0.549)
Selenium		1	-0.187 (0.192)	-0.041 (0.776)	-0.008 (0.958)	-0.235 (0.100)
Total Cholesterol			1	0.435 (0.002)	0.599 (0.000)	0.603 (0.000)
LDL				1	0.492 (0.000)	0.103 (0.477)
HDL					1	0.321 (0.023)
Triglycerides						1

Significant positive correlations were observed among lipid parameters, whereas zinc and selenium showed no significant correlation with lipid profile parameters in MI subjects.

**Figure 1: Correlation Analysis of Serum Micronutrients and Lipid Parameters in MI Patients**

[Figure 1] demonstrates that while serum zinc shows no significant correlation with lipid parameters, serum selenium exhibits a weak negative association with total cholesterol and triglycerides. These findings suggest that micronutrient alterations in critically ill myocardial infarction patients may coexist with dyslipidemia but are not strong independent determinants of lipid abnormalities.

**Heat map 1: Correlation Heatmap of Serum Zinc, Selenium, and Lipid Profile Parameters in Critically Ill Myocardial Infarction Patients**

The heatmap depicts Pearson's correlation coefficients (r) between serum zinc, selenium, and lipid profile parameters in critically ill myocardial infarction patients. Red shades indicate positive correlations, blue shades indicate negative correlations, and lighter colours represent weak or no correlation.

DISCUSSION

Myocardial infarction (MI) remains a leading cause of morbidity and mortality worldwide, particularly among middle-aged and elderly populations. The present study evaluated the demographic profile, lipid abnormalities, and trace element status, specifically zinc and selenium, in critically ill MI patients and compared the findings with healthy controls. The results provide important insights into the role of micronutrient imbalance and dyslipidemia in the pathophysiology of MI.

Age and Gender Distribution: In the present study, the mean age of critically ill MI patients was 56.08 ± 10.18 years, with the majority of subjects belonging to the 40–55 years (46%) and >55 years (48%) age groups. This finding highlights the increasing burden of MI with advancing age and reflects the cumulative effect of cardiovascular risk factors over time. Similar age distributions have been reported by Ali Kelani et al,^[18] who observed a mean age of 55.9 ± 13.9 years, and by Tanita et al,^[16] who reported a higher mean age of 67 ± 15 years among acute MI patients. These observations collectively support age as a major non-modifiable risk factor for myocardial infarction.

A male predominance was observed in the present study, with 60% males and 40% females. This finding is consistent with previous studies by Ali Kelani et al,^[18] and Tanita et al,^[16] who reported male proportions of 70% and 74%, respectively. The higher prevalence of MI in males may be attributed to greater exposure to modifiable risk factors such as smoking, dyslipidemia, and occupational stress, as well as the protective effects of estrogen in premenopausal females.

Lipid Profile Abnormalities: The present study demonstrated significant dyslipidemia among MI patients when compared to controls. Overall, total cholesterol (183 ± 84 mg/dL), LDL cholesterol (82 ± 67 mg/dL), and triglycerides (171 ± 93 mg/dL) were significantly higher in MI subjects ($P < 0.05$). These abnormalities were particularly pronounced in the 40–55 years and >55 years age groups, supporting the established role of lipid derangements in atherogenesis and coronary artery disease.

Interestingly, HDL cholesterol levels were also significantly higher in MI subjects (50 ± 25 mg/dL) compared to controls (39 ± 15 mg/dL). Although traditionally considered cardioprotective, elevated HDL levels in acute MI may reflect acute-phase

metabolic changes, statin therapy, hemodilution, or compensatory lipid remodeling following myocardial injury. Similar observations have been noted in earlier studies, suggesting that HDL behavior during acute illness may differ from that seen in stable cardiovascular states.^[19]

Serum Selenium Levels: A key finding of the present study was the significant reduction in serum selenium levels across all age groups in MI patients. The overall mean selenium level in MI subjects was $77.46 \pm 13.46 \mu\text{g/L}$, compared to $147.74 \pm 21.13 \mu\text{g/L}$ in controls ($P < 0.01$). Furthermore, 76% of MI patients had selenium levels below $70 \mu\text{g/L}$, indicating widespread selenium deficiency.

These findings are in close agreement with Ali Kelani et al,^[18] who reported significantly lower selenium levels in MI patients ($80.3 \pm 20.5 \text{ ng/mL}$) compared to controls ($97.2 \pm 14.0 \text{ ng/mL}$), and identified low selenium as the strongest independent predictor of myocardial infarction. Similarly, Hameed et al,^[20] and Hassanzadeh M,^[21] reported significantly reduced selenium levels (in MI patients. Selenium deficiency may exacerbate myocardial injury by impairing selenoprotein-mediated redox balance, thereby amplifying lipid peroxidation and endothelial dysfunction.^[22,23]

Serum Zinc Levels: The present study also demonstrated a marked reduction in serum zinc levels among MI patients. The overall mean zinc concentration in MI subjects was $53.79 \pm 25.08 \mu\text{g/dL}$, significantly lower than that in controls ($103.08 \pm 67.70 \mu\text{g/dL}$, $P < 0.01$). Notably, 86% of MI patients had zinc levels below the normal range ($< 80 \mu\text{g/dL}$), highlighting the high prevalence of zinc deficiency in critically ill MI patients.

These findings are consistent with Uddin et al,^[16] who reported significantly lower zinc levels in MI patients ($0.26 \pm 0.01 \text{ mg/L}$) compared to controls ($0.82 \pm 0.04 \text{ mg/L}$), and with Hameed et al,^[20] who observed a similar reduction in serum zinc among MI patients. Moreover, Tanita et al,^[17] demonstrated that patients with low serum zinc levels ($< 66 \mu\text{g/dL}$) had a significantly increased risk of heart-failure hospitalization following MI, underscoring the prognostic significance of zinc deficiency.

Zinc plays a crucial role in modulating oxidative stress, inflammatory responses, and cellular repair mechanisms. Deficiency of zinc may therefore exacerbate myocardial injury, impair healing, and contribute to adverse outcomes in MI patients.^[24]

Distribution of Trace Element Deficiency: The high prevalence of trace element deficiency observed in the present study, with 76% of patients showing selenium deficiency and 86% showing zinc deficiency, suggests a profound disturbance in micronutrient homeostasis in critically ill MI patients. Similar trends have been reported in previous studies, indicating that trace element depletion is a common biochemical abnormality in myocardial infarction, particularly in resource-limited settings and in patients with severe disease.

Correlation between Trace Elements and Lipid Profile:

In the present study, no significant correlation was observed between serum zinc or selenium levels and lipid profile parameters, whereas strong positive correlations were noted among lipid parameters themselves. This observation is consistent with findings by Ali Kelani et al,^[18] who reported weak or absent correlations between selenium and lipid parameters but a strong negative correlation with inflammatory markers such as C-reactive protein. These findings suggest that The absence of significant correlations between trace elements and lipid parameters suggests that micronutrient deficiency in MI operates largely independent of lipid metabolism.

Overall Interpretation: In summary, the present study demonstrates that critically ill MI patients are characterized by advanced age, male predominance, significant dyslipidemia, and profound deficiencies of antioxidant trace elements such as zinc and selenium. The magnitude of trace element deficiency observed in this study is comparable to, and in some aspects more pronounced than, that reported in previous studies. These findings reinforce the hypothesis that micronutrient imbalance plays a significant role in the pathophysiology, severity, and prognosis of myocardial infarction and highlight the potential importance of incorporating micronutrient assessment and correction into comprehensive MI management strategies.

Limitations of the study: The present study has certain limitations. The cross-sectional observational design precludes the establishment of a causal relationship between serum zinc and selenium levels and lipid profile abnormalities in patients with myocardial infarction. The relatively small sample size and the single-center nature of the study may limit the generalizability of the findings to broader populations. Additionally, inflammatory markers and oxidative stress parameters were not assessed, which could have provided deeper mechanistic insights into the role of trace element imbalance in the pathophysiology of myocardial infarction. Baseline dietary intake of zinc and selenium was also not evaluated, which may have influenced serum micronutrient levels. Therefore, well-designed longitudinal and interventional studies with larger sample sizes are warranted to confirm these findings and to explore the potential therapeutic implications of targeted micronutrient supplementation.

CONCLUSION

Critically ill patients with myocardial infarction demonstrate significant dyslipidemia along with profound deficiencies of antioxidant trace elements such as zinc and selenium. These biochemical abnormalities appear to coexist in the setting of acute myocardial injury rather than exhibiting a direct interaction, suggesting that micronutrient deficiency may contribute to myocardial damage predominantly

through oxidative and inflammatory pathways. The findings underscore the importance of assessing trace element status as part of a comprehensive biochemical evaluation in critically ill myocardial infarction patients and provide a rationale for future studies evaluating the clinical impact of targeted micronutrient correction.

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