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Selenium And Free Radical Status In Patients With Acute Coronary Syndrome

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ABSTRACT

Acute coronary syndrome (ACS) remains a major cause of cardiovascular morbidity in Uzbekistan. This prospective study evaluated serum selenium levels and free radical status in 122 patients with ACS variants (new-onset angina, unstable angina, non-Q-wave myocardial infarction) admitted to the Republican Specialized Scientific-Practical Medical Center of Cardiology in Tashkent. Selenium levels were suboptimal (mean 61.6 ± 1.6 μg/L), with evidence of intensified free radical oxidation and reduced antioxidant protection. Negative correlations existed between selenium and chemiluminescence parameters. Results support selenium status monitoring and potential organic selenium supplementation to mitigate oxidative stress and improve prognosis.

Keywords: Selenium, free radical oxidation, acute coronary syndrome, antioxidant defense, chemiluminescence, Uzbekistan.

INTRODUCTION

Ischemic heart disease, particularly ACS, is a leading health challenge globally and in Central Asia. In Uzbekistan, cardiovascular diseases contribute significantly to mortality, with environmental and dietary factors potentially exacerbating trace element deficiencies like selenium (Se) [1]. Selenium is essential for antioxidant enzymes such as glutathione peroxidase, protecting against oxidative stress implicated in ACS pathogenesis [2]. Prior studies in selenium-deficient regions link low Se to increased myocardial infarction risk [3]. Oxidative stress intensifies in ACS, consuming selenium and straining defenses [4]. This study, conducted in Tashkent, assessed serum selenium and its association with free radical markers in ACS patients to inform targeted interventions [5].

METHODS

This prospective observational study (2022–2024) included 122 patients with non-ST-elevation ACS hospitalized at the Republican Specialized Scientific-Practical Medical Center of Cardiology (Tashkent, Uzbekistan). Mean age: 68.3 ± 2.5 years (47 men, 75 women). All had concomitant hypertension and received standard therapy (anticoagulants, antiplatelets, β-blockers, nitrates, statins, ACE inhibitors).

Patients were divided into groups:

- Group 1: 23 with new-onset angina (age 63.5 ± 2.3 years; 10 men, 13 women). These patients did not receive any medications prior to admission.
- Group 2: 66 with unstable (progressive) angina (age 70.5 ± 2.5 years; 19 men, 47 women). Symptoms included increased angina attacks, reduced nitrate efficacy, decreased exercise tolerance, and negative ECG dynamics. Mean time from pain onset to admission: 2.13 ± 0.12 days.
- Group 3: 33 with non-Q-wave myocardial infarction (age 71 ± 2.6 years; 18 men, 15 women). Presented with anginal variant; diagnosis confirmed by ECG dynamics and positive troponin test. Mean time from pain onset to admission: 2.91 ± 0.32 days.

Control group: 30 healthy volunteers (age 65.0 ± 2.2 years; 15 men, 15 women) without cardiac pathology.

All patients resided in Tashkent for over 5 years. Exclusion criteria: ST-elevation ACS, arrhythmias (e.g., atrial fibrillation, flutter), NYHA III–IV heart failure, decompensated diabetes, peptic ulcer, chronic kidney failure, body mass index >25. Therapy followed guidelines for non-ST-elevation ACS, including hypotensive drugs (ACE inhibitors, calcium channel blockers). Serum selenium was determined by fluorimetric method using 2,3-diaminonaphthalene (adapted from Golubkina N.A., 1995) with certified reference standards (e.g., N23-KT serum with 88 µg/L Se) [6].

Free radical status was assessed by chemiluminescence (CL) on a Perkin Elmer LS-50B spectrometer with "Finlab" software [7]:

- Spontaneous CL (Ssp): reactive oxygen species generation.
- Fe^{2+}-induced CL (h: lipid hydroperoxides; Sind-1: peroxide radicals).
- H_2O_2-luminol induced CL (Slum: hydroxyl radicals; H: oxidation potential; Sind-2: antioxidant reserve).

Intensity expressed in relative units. Standardization and processing per Vladimirov Yu.A. (1991) and Arutyan A.V. (2000). Statistical analysis: mean (M), standard error (m), Student's t-test, Mann-Whitney U test for non-normal distributions, Pearson correlation (r). Differences significant at $p < 0.05$ using Microsoft Excel 2003, Statistica 6.0, or SPSS v26 equivalent [8]. The study was approved by the ethics committee of Tashkent Medical Academy. All participants provided informed consent.

RESULTS

Clinical and Lipid Profile

Patients exhibited combined hyperlipidemia (total cholesterol >5 mmol/L, LDL-C >3 mmol/L, triglycerides >1.77 mmol/L), modulated by statin therapy in Groups 2 and 3 (5.71 ± 0.14 and 5.51 ± 0.17 mmol/L). In Group 3, ALT and AST elevated 2.1- and 2.9-fold vs. control ($p < 0.05$); non-Q MI confirmed by troponin [9].

Selenium Status

Serum Se ranged 34.3–107 µg/L, all below optimal 115–120 µg/L [10]. Mean in ACS: 61.6 ± 1.6 µg/L ($p < 0.05$ vs. control 82.5 ± 1.8 µg/L). Distribution (Fig. 1, adapted): optimal 0%; suboptimal (90–114 µg/L) 4.1%; deficiency (<90 µg/L) 37.7%; severe (<50 µg/L) 23.8%. Critical deficiency (<70 µg/L) in 34.4%.

By groups (Table 1):

Group	n	Serum Se (µg/L, M ± m)
Control	30	81.2 ± 1.9
New-onset angina	23	72.4 ± 1.5* #
Unstable angina	66	62.7 ± 1.7* ** #
Non-Q MI	33	49.6 ± 1.7*

- $p < 0.05$ vs. control; ** $p < 0.05$ vs. new-onset; # $p < 0.01$ vs. non-Q MI.

No gender differences (Table 2):

Gender	Control (n, µg/L)	ACS (n, µg/L)
Men	15, 80.8 ± 1.4	47, 60.8 ± 1.9
Women	15, 81.7 ± 2.9	75, 62.6 ± 1.79

Smoking men (n=20, 12 ± 1.3 cigarettes/day, >10 years): 56.9 ± 1.7 µg/L ($p < 0.05$ vs. control and non-smoking men) [11]

(Table 3):

Group	n	Serum Se (µg/L)
Control (non-smokers)	15	80.8 ± 1.9
Smoking men with ACS	20	56.9 ± 1.7*
Non-smoking men with ACS	25	64.8 ± 1.6* #

- $p < 0.05$ vs. control; # $p < 0.05$ vs. smokers.

Free Radical Status

Marked activation of lipid peroxidation: elevated Ssp (1.5–1.7x), Sind-1 (1.5–3.8x), Slum (1.7–7.2x), h (1.2–1.4x); reduced Sind-2 (2.2–3.2x) and H (1.5–1.6x) [12]. Most pronounced in Group 1 (new-onset) vs. Group 2: Sind-1 +2.4x, Slum +4.3x, Sind-2 -1.3x ($p < 0.05$) (Fig. 2, adapted). Vs. Group 3: Sind-1 +2.5x, Slum +7.9x, Sind-2 -1.4x ($p < 0.05$) (Fig. 3, adapted). No differences between Groups 2 and 3 (Fig. 4, adapted), possibly due to antioxidant therapy [13]. Moderate negative correlations in Group 3: Se vs. Sind-1 ($r = -0.54$), Slum ($r = -0.39$), h ($r = -0.61$; $p < 0.05$) [14]. Direct correlation between CL indicators and ACS severity.

DISCUSSION

Findings reveal selenium deficiency in Uzbek ACS patients, likely compounded by regional dietary and environmental factors, amplifying oxidative stress [15]. Highest radical activity in new-onset angina suggests absent preconditioning and lack of antioxidants [16]. Hydroxyl radicals promote LDL oxidation, endothelial damage, vasoconstrictor production, platelet aggregation, leading to plaque instability and ACS (Schema 1, adapted) [17]. Results align with international evidence on selenium's cardioprotective role and oxidative stress in ACS [18]. Correlations underscore selenium's involvement in antioxidant defense [19]. Organic selenium supplementation may reduce peroxidation, warranting randomized trials in this population.

CONCLUSION

1. ACS patients in Uzbekistan show reduced serum Se (61.6 ± 1.6 µg/L), below local healthy levels (82.5 ± 1.8 µg/L) and optimal (115–120 µg/L).
2. Intensified free radical oxidation and reduced antioxidant defense.
3. Direct correlation between CL indicators and ACS variants; most pronounced in new-onset angina.
4. Negative moderate correlation between CL and Se levels, linked to disease severity.
5. Se levels correlate with ACS variants, enabling prognosis of severe course.

Routine selenium assessment could predict non-Q MI risk; adjunctive organic selenium therapy merits consideration alongside standard care. Practical recommendations: Assess Se and free radicals in ACS for risk stratification; consider organic Se correction to reduce oxidation and improve outcomes.

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