# Biochemical Evaluation of Cardiac Markers and Electrolyte Profiles in Patients with Myocardial Infarction and Cardiogenic Shock

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**Abstract.** Cardiovascular emergencies such as Myocardial Infarction (MI) and Cardiogenic Shock (CS) are leading causes of morbidity and mortality worldwide. Accurate biochemical assessment of cardiac injury and electrolyte imbalance is essential for diagnosis, prognosis, and therapeutic decision-making. This study aimed to evaluate and compare the levels of selected biochemical markers among patients with MI, CS, and healthy controls to identify significant diagnostic indicators and correlations relevant to cardiac pathophysiology. A total of 120 blood samples were collected and categorized into three groups: 40 patients with MI, 40 with CS, and 40 healthy individuals. Serum concentrations of Troponin, CK-MB, Calcium (Ca), Sodium (Na), Potassium (K), 25-OH-Vitamin D (D3), Total Serum Bilirubin (TSB), and Red Blood Cells (RBCs) were analyzed. Statistical analysis was conducted using one-way ANOVA to determine the significance of differences among groups. Significantly elevated levels of Troponin and CK-MB were observed in both MI and CS patients compared to controls ($p < 0.05$), with the highest values in the CS group. Electrolyte imbalances were evident, particularly decreased Vitamin D and Calcium levels in pathological groups. Elevated TSB and altered RBCs counts were also noted, indicating systemic physiological stress associated with cardiac dysfunction. The findings support the utility of biochemical markers such as Troponin, CK-MB, D3, and Ca in distinguishing between acute cardiac events. These parameters can aid in early detection and clinical differentiation between MI and CS, contributing to improved patient outcomes and management strategies.

**Keywords. Troponin, CK-MB, Electrolytes, Vitamin D, Myocardial Infarction, Cardiogenic Shock, Biochemical markers, Serum analysis.**

## 1. Introduction

Cardiovascular diseases, including Myocardial Infarction (MI) and Cardiogenic Shock (CS), represent major clinical challenges requiring rapid diagnosis and intervention. Vitamin D deficiency is a widespread public health issue with implications beyond bone health [1]. In addition to its classical role in calcium–phosphate homeostasis, 25-hydroxyvitamin $D_3$ exerts immunomodulatory, anti-inflammatory, and vascular-protective effects that may influence cardiovascular outcomes [2,3]. Obesity has been shown to decrease hepatic 25-hydroxylase activity, leading to lower circulating 25-hydroxyvitamin D levels [4]. DeLuca reviews the endocrine regulation and pleiotropic functions of vitamin D, underscoring its systemic importance [5].

Experimental and clinical studies demonstrate that vitamin D modulates the gut microbiota and systemic inflammation, highlighting its broad physiological impact [6]. The Endocrine Society's scientific statement details numerous nonskeletal actions of vitamin D, including cardiovascular modulation [7]. Genetic and lifestyle determinants significantly influence vitamin D status in Arab populations, pointing to interindividual variability in deficiency risk [8]. Globally, vitamin D sufficiency correlates with reduced cardiovascular morbidity, reinforcing the need for adequate intake [9]. Key enzymes such as CYP2R1 catalyze 25-hydroxyvitamin D synthesis; mutations in CYP2R1 impair this process and contribute to deficiency states [10]. Common polymorphisms in the vitamin D receptor (VDR) gene further modify individual responses and cardiovascular risk profiles [11].

Dietary sources and supplementation can correct deficiency, with guidelines evolved from nutritional studies on intake requirements [12,13]. Finally, vitamin D–binding protein variants critically determine bioavailable 25-hydroxyvitamin D levels across ethnic groups, impacting cardiovascular health disparities [14].

This study evaluates serum 25-hydroxyvitamin D levels in MI and CS patients versus healthy controls to assess diagnostic and prognostic utility

## 2. Materials and Methods

### 2.1. Study Design and Population:

This cross-sectional study was conducted between October 1, 2023, and June 1, 2024, at Tikrit Teaching Hospital, Iraq. A total of 120 blood samples were collected and divided into three groups: 40 patients with myocardial infarction (MI), 40 patients with cardiogenic shock (CS), and 40 healthy individuals as controls. The control group was confirmed to be free of cardiovascular and hematological conditions through clinical screening and laboratory evaluation.

### 2.2. *Sample Collection and Processing:*

Venous blood samples (5 mL) were obtained under aseptic conditions and distributed into:

EDTA tubes for hematological tests (e.g., RBCs count).

Gel tubes for serum separation and biochemical assays.

Samples were centrifuged at 4000 rpm for 5 minutes, and serum was stored at −4°C until analysis.

### 2.3. *Biochemical Parameters:*

The following biomarkers were evaluated using standard commercial kits (Mindray, Getein, Abbott, Agappe):

1. Cardiac markers: Troponin, CK-MB
2. Electrolytes: Calcium (Ca), Sodium (Na), Potassium (K)
3. Other markers: 25-OH Vitamin D (D3), Total Serum Bilirubin (TSB), Red Blood Cells (RBs)

All assays were conducted following the manufacturer's protocols using semi-automated analyzers.

2.4. *Statistical Analysis:*

Data were analyzed using SPSS v25. Descriptive statistics (mean ± SD) were calculated for each variable. Comparisons among groups were performed using one-way ANOVA, followed by Post-hoc (LSD) tests to assess pairwise differences. Statistical significance was set at $p < 0.05$.

## 3. Results and Discussion

The analysis of biochemical markers revealed significant differences among the three study groups (Control, MI, CS). Serum troponin was markedly elevated in MI and CS patients versus controls (Table 1). Mean troponin rose from 0.010 ng/mL in controls to 7.34 ng/mL in MI and 12.61 ng/mL in CS, reflecting progressive cardiomyocyte necrosis [1,3].

**Table 1.** Troponin levels among Control, MI, and CS groups

Troponin - Descriptives

| Group | N | Mean | Std. Deviation | Std. Error |
|---|---|---|---|---|
| Control | 40 | c.0100 | 0 | 0 |
| Myocardial Infarction (MI) | 40 | b7.3375 | 2.01206 | 0.31813 |
| Cardiogenic Shock | 40 | a12.6093 | 1.86712 | 0.29522 |
| Total | 120 | 6.6523 | 5.42081 | 0.49485 |

Troponin - ANOVA

| Reference | Sum of Squares | df | Mean Square | F | Sig. |
|---|---|---|---|---|---|
| Between Groups | 3202.996 | 2 | 1601.498 | 637.665 | 0 |
| Within Groups | 293.846 | 117 | 2.512 | | |
| Total | 3496.842 | 119 | nan | | |

CK-MB activity increased from 2.55 U/L in controls to 29.72 U/L in MI and 16.01 U/L in CS (Table 2). Although less specific than troponin, CK-MB remains a reliable early marker of myocardial injury [4].

**Table 2.** CK-MB enzyme levels among Control, MI, and CS groups

CK-MB - Descriptives

| Group | N | Mean | Std. Deviation | Std. Error |
|---|---|---|---|---|
| Control | 40 | c2.5553 | 0.34943 | 0.05525 |
| Myocardial Infarction (MI) | 40 | a29.7205 | 25.54252 | 4.03863 |
| Cardiogenic Shock | 40 | b16.0117 | 22.59911 | 3.57323 |
| Total | 120 | 16.0958 | 22.47812 | 2.05196 |

CK-MB - ANOVA

| Reference | Sum of Squares | df | Mean Square | F | Sig. |
|---|---|---|---|---|---|
| Between Groups | 14759.44 | 2 | 7379.72 | 19.032 | 0 |
| Within Groups | 45367.213 | 117 | 387.754 | | |
| Total | 60126.653 | 119 | nan | | |

Serum Calcium and Potassium were modestly reduced in MI and CS, whereas Sodium levels were variable. Hypocalcemia and hyperkalemia in acute ischemia may stem from altered cell membrane permeability and activation of neurohormonal pathways [5,6]. And these are the details, as shown in table (3).

Mean $Na^+$ fell from 138.25 mmol/L (Control) to 134.78 mmol/L (MI) and 135.10 mmol/L (CS) but did not reach significance ($F = 2.559$, $p = 0.082$). Although a trend toward dilutional hyponatremia is apparent—particularly given the wide variability in CS (SD = 11.64)—these differences may reflect individual fluid shifts or therapeutic interventions rather than a consistent clinical effect.

Potassium means were 4.2425 mmol/L (Control), 4.5300 mmol/L (MI) and 4.3450 mmol/L (CS), with no significant group differences ($F = 0.999$, $p = 0.372$). Slight elevations in MI likely arise from ischemia-induced $K^+$ leak, but robust renal and hormonal regulation maintains overall homeostasis across all groups.

Analysis of variance showed a significant, stepwise decrease in mean serum calcium from Control (10.9923 mg/dL) to MI (10.1057 mg/dL) to CS (9.8881 mg/dL) ($F = 5.313$, $p = 0.006$). This progressive hypocalcemia parallels worsening cardiac injury, reflecting both ischemia-induced impairment of $Ca^{2+}$ membrane pumps and reduced intestinal absorption in the context of low vitamin $D_3$.

Mean $Cl^-$ was 105.00 mmol/L (Control), 104.05 mmol/L (MI) and 107.38 mmol/L (CS), also non-significant (F = 2.540, p = 0.083). The low SD in controls (1.95) versus CS (10.91) suggests greater intra-group variability under shock conditions, though overall chloride balance remains preserved.

$D_3$ levels were significantly lower in MI (mean 18.2 ng/mL) and CS (14.7 ng/mL) versus controls (28.4 ng/mL). Vitamin D deficiency promotes endothelial dysfunction, inflammation, and plaque instability [15–17,22]. Obesity and CYP2R1 polymorphisms further impair $D_3$ synthesis and elevation in cardiovascular risk [16,18–20].

**Table 3.** Serum Electrolyte Levels (Na, K, $Ca^2$, $Cl^-$) Among Control, MI, and CS Groups

$Na^+$- Descriptives

| Group | N | Mean | Std. Deviation | Std. Error |
|---|---|---|---|---|
| Control | 40 | a138.2500 | 1.97094 | 0.31163 |
| Myocardial Infarction (MI) | 40 | a134.7750 | 5.77123 | 0.91251 |
| Cardiogenic Shock | 40 | a135.1000 | 11.64166 | 1.84071 |
| Total | 120 | 136.0417 | 7.6865 | 0.70168 |

Na- ANOVA

| Reference | Sum of Squares | df | Mean Square | F | Sig. |
|---|---|---|---|---|---|
| Between Groups | 294.717 | 2 | 147.358 | 2.559 | 0.082 |
| Within Groups | 6736.075 | 117 | 57.573 | | |
| Total | 7030.792 | 119 | nan | | |

$K^+$- Descriptives

| Group | N | Mean | Std. Deviation | Std. Error |
|---|---|---|---|---|
| Control | 40 | a4.2425 | 0.34928 | 0.05523 |
| Myocardial Infarction (MI) | 40 | a4.5300 | 1.10249 | 0.17432 |
| Cardiogenic Shock | 40 | a4.3450 | 1.10174 | 0.1742 |
| Total | 120 | 4.3725 | 0.92218 | 0.08418 |

$K^+$- ANOVA

| Reference | Sum of Squares | df | Mean Square | F | Sig. |
|---|---|---|---|---|---|

| | | | | | |
|---|---|---|---|---|---|
| Between Groups | 1.698 | 2 | 0.849 | 0.999 | 0.372 |
| Within Groups | 99.501 | 117 | 0.85 | | |
| Total | 101.199 | 119 | nan | | |

$Ca^{2}$- Descriptives

| Group | N | Mean | Std. Deviation | Std. Error |
|---|---|---|---|---|
| Control | 40 | a10.9923 | 1.59832 | 0.25272 |
| Myocardial Infarction (MI) | 40 | b10.1057 | 1.57442 | 0.24894 |
| Cardiogenic Shock | 40 | b9.8881 | 1.64079 | 0.25943 |
| Total | 120 | 10.3287 | 1.66189 | 0.15171 |

$Ca^{2}$- ANOVA

| Reference | Sum of Squares | df | Mean Square | F | Sig. |
|---|---|---|---|---|---|
| Between Groups | 27.365 | 2 | 13.683 | 5.313 | 0.006 |
| Within Groups | 301.3 | 117 | 2.575 | | |
| Total | 328.665 | 119 | nan | | |

Cl- Descriptives

| Group | N | Mean | Std. Deviation | Std. Error |
|---|---|---|---|---|
| Control | 40 | ab105.000 | 1.94804 | 0.30801 |
| Myocardial Infarction (MI) | 40 | b104.050 | 3.9804 | 0.62936 |
| Cardiogenic Shock | 40 | a107.3750 | 10.9068 | 1.72452 |
| Total | 120 | 105.475 | 6.88435 | 0.62845 |

Cl - ANOVA

| Reference | Sum of Squares | df | Mean Square | F | Sig. |
|---|---|---|---|---|---|
| Between Groups | 234.65 | 2 | 117.325 | 2.54 | 0.083 |
| Within Groups | 5405.275 | 117 | 46.199 | | |
| Total | 5639.925 | 119 | nan | | |

Mean RBC count rose from 108×10^4/μL in controls to 167×10^4/μL in MI and CS (Table 4), suggesting compensatory erythropoiesis secondary to hypoxia. Wide interpatient variability likely reflects comorbidities and therapeutic interventions [26].

**Table 4.** Red Blood Cells (RBCs) counts among Control, MI, and CS groups

RBCs - Descriptives

| Group | N | Mean | Std. Deviation | Std. Error |
|---|---|---|---|---|
| Control | 40 | b108.0500 | 49.90861 | 7.89124 |
| Myocardial Infarction (MI) | 40 | a166.8125 | 77.74133 | 12.29198 |
| Cardiogenic Shock | 40 | a167.2000 | 42.20354 | 6.67296 |
| Total | 120 | 147.3542 | 64.49579 | 5.88763 |

RBCs - ANOVA

| Reference | Sum of Squares | df | Mean Square | F | Sig. |
|---|---|---|---|---|---|
| Between Groups | 92692.054 | 2 | 46346.027 | 13.478 | 0 |
| Within Groups | 402313.144 | 117 | 3438.574 | | |
| Total | 495005.198 | 119 | nan | | |

TSB increased from 0.88 mg/dL in controls to 1.05 mg/dL in MI and 1.49 mg/dL in CS (Table 5), indicating oxidative stress and transient hepatic hypoperfusion [24,25]. Elevated bilirubin may confer antioxidant protection but also signals severe systemic insult.

**Table 5.** Total Serum Bilirubin (TSB) levels among Control, MI, and CS groups

TSB - Descriptives

| Group | N | Mean | Std. Deviation | Std. Error |
|---|---|---|---|---|
| Control | 40 | b.8825 | 0.47818 | 0.07561 |
| Myocardial Infarction (MI) | 40 | b1.0550 | 0.49869 | 0.07885 |
| Cardiogenic Shock | 40 | a1.4925 | 0.31654 | 0.05005 |
| Total | 120 | 1.1433 | 0.50572 | **0.04617** |

TSB - ANOVA

| Reference | Sum of Squares | df | Mean Square | F | Sig. |
|---|---|---|---|---|---|
| Between Groups | 7.91 | 2 | 3.955 | 20.455 | 0 |
| Within Groups | 22.525 | 117 | 0.193 | | |
| Total | 30.435 | 119 | nan | | |

Taken together, troponin, CK-MB, $D_3$, calcium, and bilirubin are the most consistent and clinically significant markers for distinguishing MI and CS from normal physiology. Their combined use enhances early diagnosis, risk stratification, and monitoring in acute cardiac care [27].

## Conclusions

This study highlighted significant differences in biochemical marker levels among patients with Myocardial Infarction (MI), Cardiogenic Shock (CS), and healthy controls. Elevated Troponin and CK-MB were strong indicators of cardiac injury severity, whereas reduced Vitamin D and altered electrolyte levels (Ca, Na, K) reflected broader systemic implications. Additionally, elevated Total Serum Bilirubin (TSB) and Red Blood Cell (RBC) counts provided supplementary insights into oxidative stress and physiological response to cardiac damage. These biochemical parameters collectively enhance clinical diagnosis, differentiation, and management strategies in acute cardiac conditions. Further research is recommended to explore the therapeutic implications and prognostic value of these biomarkers in larger patient populations.

## References

[1] Holick MF. Vitamin D deficiency. N Engl J Med. 2007;357(3):266–281.

[2] Hollis BW. Circulating 25-hydroxyvitamin D levels indicative of vitamin D sufficiency: implications for establishing a new effective dietary intake recommendation for vitamin D. J Nutr. 2005;135:317–22.

[3] Bouillon R, Marcocci C, Carmeliet G, Bikle D, Dawson-Hughes B, Lips P, et al. Skeletal and extraskeletal actions of vitamin D: current evidence and outstanding questions. Endocr Rev. 2019;40(4):1109–1151.

[4] Reijven P, Soeters P. Vitamin D: a magic bullet or a myth? Clin Nutr. 2020;39:2663–2674.

[5] DeLuca HF. Vitamin D. Vitam Horm. 2016;100:1–20.

[6] Battistini C, Ballan R, Herkenhoff ME, Saad SMI, Sun J. Vitamin D modulates intestinal microbiota in inflammatory bowel diseases. Int J Mol Sci. 2020;22:362.

[7] Rosen CJ, Adams JS, Bikle DD, Black DM, Demay MB, Manson JE, et al. The nonskeletal effects of vitamin D: an Endocrine Society scientific statement. Endocr Rev. 2012;33(4):456–492.

[8] Mezzavilla M, Tomei S, Alkayal F, Melhem M, Ali MM, Al-Arouj M, et al. Investigation of genetic variation and lifestyle determinants in vitamin D levels in Arab individuals. J Transl Med. 2018;16:20. doi:10.1186/s12967-018-1396-8

[9] Hossein-Nezhad A, Holick MF. Vitamin D for health: a global perspective. Mayo Clin Proc. 2013;88:720–755.

[10] Zhu JG, Ochalek JT, Kaufmann M, Jones G, DeLuca HF. CYP2R1 is a major, but not exclusive, contributor to 25-hydroxyvitamin D production in vivo. Proc Natl Acad Sci U S A. 2013;110(35):15650–5.
[11] Uitterlinden AG, Fang Y, van Meurs JB, Pols HA, van Leeuwen JP. Genetics and biology of vitamin D receptor polymorphisms. Gene. 2004;338:143–156.
[12] Lamberg-Allardt C. Vitamin D in foods and as supplements. Prog Biophys Mol Biol. 2006;92:33–38.
[13] Elsammak MY, Al-Wosaibi AA, Al-Howeish A, Alsaeed J. Vitamin D deficiency in Saudi Arabs. Horm Metab Res. 2010;42:364–368.
[14] Powe CE, Evans MK, Wenger J, Zonderman AB, Berg AH, Nalls M, et al. Vitamin D-binding protein and vitamin D status of black Americans and white Americans. N Engl J Med. 2013;369(21):1991–2000. doi:10.1056/NEJMoa1306357
[15] Roizen JD, Long C, Casella A, et al. Obesity decreases hepatic 25-hydroxylase activity causing low serum 25-hydroxyvitamin D. J Bone Miner Res. 2019;34:1068–1073.
[16] Powe CE, Evans MK, Wenger J, et al. Vitamin D-binding protein and vitamin D status of Black Americans and White Americans. N Engl J Med. 2013;369:1991–2000.
[17] Aloia JF. African Americans, 25-hydroxyvitamin D, and osteoporosis: a paradox. Am J Clin Nutr. 2008;88(2):545S–550S.
[18] Aloia JF, Patel M, Dimaano R, et al. Vitamin D intake to attain a desired serum 25-hydroxyvitamin D concentration. Am J Clin Nutr. 2008;87(6):1952–1958.
[19] Elsammak MY, Al-Wosaibi AA, Al-Howeish A, Alsaeed J. Vitamin D deficiency in Saudi Arabs. Horm Metab Res. 2010;42(5):364–368.
[20] Mezzavilla M, Tomei S, Alkayal F, et al. Investigation of genetic variation and lifestyle determinants in vitamin D levels in Arab individuals. J Transl Med. 2018;16:20. doi:10.1186/s12967-018-1396-8.
[21] Meirina F, Sari DK, Lubis IND, et al. Correlation between CYP2R1 rs10741657 and CYP27B1 rs10877012 polymorphisms and latent tuberculosis infection in vitamin D–deficient pregnant mothers. J Med Chem Sci. 2023;6:2922–2933.
[22] Manousaki D, Dudding T, Haworth S, et al. Low-frequency synonymous coding variation in CYP2R1 has large effects on vitamin D levels and risk of multiple sclerosis. Am J Hum Genet. 2017;101:227–238.

[23] Forrest KY, Stuhldreher WL. Prevalence and correlates of vitamin D deficiency in US adults. Nutr Res. 2011;31(1):48–54.
[24] Rosen CJ, Adams JS, Bikle DD, et al. The nonskeletal effects of vitamin D: an Endocrine Society scientific statement. Endocr Rev. 2012;33(3):456–492.
[25] DeLuca HF. Vitamin D. Vitam Horm. 2016;100:1–20.
[26] Battistini C, Ballan R, Herkenhoff ME, Saad SMI, Sun J. Vitamin D modulates intestinal microbiota in inflammatory bowel diseases. Int J Mol Sci. 2020;22(1):362.
[27] Bouillon R, Marcocci C, Carmeliet G, et al. Skeletal and extraskeletal actions of vitamin D: current evidence and outstanding questions. Endocr Rev. 2019;40(4):1109–1151.