ISSN: 3067-2376
Research Article
Journal of Comparative Medicine Research Reviews and Reports

# Association Between Urinary Escherichia Coli and Plasma NT-proBNP Levels in Patients with Post-Infarction Cardiosclerosis

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**Submitted:** 13 October 2025 **Accepted:** 21 October 2025 **Published:** 26 October 2025

**https://doi.org/10.63620/MK.JCMRRR.2025.1026**

***Citation:*** *Aliyeva, S., Hajiyev, A., Dadashova, G., Rakhmanov, Z., & Hajizada, S. (2025). Association Between Urinary Escherichia Coli and Plasma NT-proBNP Levels in Patients with Post-Infarction Cardiosclerosis. J of Comp Med Res Rev Rep, 2(5), 01-03.*

***Abstract***

***Background:*** *Recent studies suggest that bacterial endotoxins may influence plasma NT-proBNP levels. This study aimed to evaluate the impact of Escherichia coli bacteriuria on NT-proBNP levels in patients with post-myocardial infarction cardiosclerosis (CVS).*

***Methods:*** *Fifty-six patients diagnosed with post-infarction cardiosclerosis were enrolled. Urine samples were cultured to detect E. coli using the VITEK 2 system, and plasma NT-proBNP levels were measured by fluorescence immunoassay (AFIAS 10, Boditech, Korea). Based on NT-proBNP levels, patients were divided into two groups: NT-proBNP > 500 pg/mL (n=35) and NT-proBNP < 500 pg/mL (n=21) accordingly the existing recommendations. Data were analyzed using SPSS 21.0.*

***Results:*** *E. coli bacteriuria was detected in 67.9% (38/56) of patients. The median [min–max] NT-proBNP level in the E. coli-positive group was 3081.1 (38.1–16083.0) pg/mL, while in patients without bacteriuria it was 348.0 (24.7–7208.0) pg/mL (p=0.003). The frequency of NT-proBNP > 500 pg/mL was significantly higher in the E. coli group (73.68%) compared to patients without bacteriuria (38.89%, p=0.018). The odds ratio (OR) was 0.23 (95% CI: 0.069–0.748, p=0.028), with a number needed to treat (NNT) of 2.87 (95% CI: 1.61–13.39).*

***Conclusion:*** *The presence of E. coli bacteriuria is associated with significantly elevated NT-proBNP levels in patients with post-infarction cardiosclerosis, suggesting that bacterial infection may partly contribute in the biomarker elevation before cardiac dysfunction development.*

**Keywords:** The Next-Generation Medical Infrastructure Act, The Personal Information Protection Act (PIPA), Virtual Desktop Infrastructure (VDI), European Health Data Space (EHDS), Certified Anonymous Processed Medical Information Creation Provider.

## Introduction

Cardiosclerosis following myocardial infarction (MI) is often associated with chronic neurohumoral activation and subclinical inflammation, both of which can influence the release of natriuretic peptides. Recent studies, however, have indicated that non-cardiac factors—particularly systemic inflammation and infection—can also influence circulating NT-proBNP levels [1]. Among these biomarkers, N-terminal pro–B-type natriuretic peptide (NT-proBNP) is widely recognized as a sensitive indicator of myocardial wall stress and ventricular dysfunction. However, recent evidence suggests that non-cardiac factors, including systemic infections and inflammatory stimuli, may also contribute to increased NT-proBNP levels [2]. Bacterial infections, particularly those caused by Escherichia coli, can induce the release of endotoxins such as lipopolysaccharides (LPS), which activate the immune system and promote cytokine production. These endotoxins activate Toll-like receptor pathways and promote the release of proinflammatory cytokines (TNF-α,

IL-6), which can impair myocardial contractility and endothelial function [3]. This inflammatory cascade has been linked to endothelial dysfunction, oxidative stress, and transient myocardial impairment—mechanisms that may secondarily elevate circulating NT-proBNP concentrations even in the absence of acute cardiac decompensation. In patients with post-myocardial infarction cardiosclerosis, who already exhibit compromised myocardial compliance and persistent neurohumoral activation, the additional burden of bacteriuria may further modulate NT-proBNP expression. Despite this potential interaction, limited data exist regarding the direct relationship between E. coli bacteriuria and NT-proBNP levels in this patient population [4].

Therefore, this study aimed to evaluate the impact of Escherichia coli bacteriuria on plasma NT-proBNP levels in patients with post-myocardial infarction cardiosclerosis, in order to clarify whether infectious and inflammatory factors may influence this key cardiac biomarker before traditional hemodynamic deteriorations development.

## Materials and Methods

### Study Population

Fifty-six patients with clinically stable post-myocardial infarction cardiosclerosis were included. Exclusion criteria were acute coronary syndrome, chronic renal failure (eGFR <45 mL/min/1.73m²), severe valvular disease, or systemic infection other than bacteriuria.

Venous blood was collected and centrifuged to obtain serum. NT-proBNP levels were analyzed using the AFIAS 10 analyzer (Boditech, Korea), which operates on the fluorescence immunoassay (FIA) principle. In this method, antigen–antibody binding produces fluorescence signals proportional to the analyte concentration. The system performs automatic calibration and internal quality checks before each run, providing results within 15 minutes.

### Bacteriological Analysis

Midstream urine samples were collected under sterile conditions. Urinary sediment was first examined microscopically using a light microscope (Olympus CX43, Japan) to detect leukocytes and bacteria. Samples were then inoculated onto MacConkey agar and 5% blood agar and incubated at 37°C for 18–24 hours. Bacterial colonies identified as E. coli were confirmed by the VITEK 2 automated system (BioMérieux, France), which performs species-level identification and antibiotic susceptibility testing using standardized biochemical and kinetic growth profiles in accordance with CLSI guidelines.

### Echocardiography

All patients underwent transthoracic echocardiography using a Sonoscape (China) system. Standard 2D and M-mode imaging was performed to measure left ventricular ejection fraction (LVEF) and other parameters. Patients with LVEF <40% or significant structural abnormalities were excluded to avoid confounding effects on NT-proBNP levels.

### Statistical Analysis

Statistical analysis was performed using IBM SPSS Statistics v21.0 (IBM Corp., USA). Continuous variables were expressed as median (min–max) and compared using the Mann–Whitney U test. Categorical variables were compared using Chi-square or Fisher's exact tests. A p-value <0.05 was considered statistically significant.

## Results

Of the 56 patients, E. coli bacteriuria was present in 38 (67.9%). Prior to culture, urine sediments were evaluated microscopically, and samples without evidence of leukocyturia were excluded from further bacteriological analysis to ensure diagnostic accuracy. Median NT-proBNP levels were significantly higher in E. coli-positive patients compared to those without bacteriuria [3081.1 (38.1–16083.0) vs 348.0 (24.7–7208.0) pg/mL, p=0.003]. The frequency of NT-proBNP > 500 pg/mL was 73.68% in the E. coli group and 38.89% in the bacteriuria-negative group (p=0.018). The calculated OR was 0.23 (95% CI: 0.069–0.748, p=0.028), indicating a strong association between E. coli infection and NT-proBNP elevation. However, no significant differences in echocardiographic parameters (LVEF, wall stress, chamber size) were found between groups.

The chart below (fig. 1) provides a visual comparison of NT-proBNP levels and the frequency of NT-proBNP exceeding 500 pg/ml between the E. coli group and the group without bacteriuria: these visualizations underscore the statistically significant differences, with the E. coli group showing much higher NT-proBNP levels and a higher frequency of elevated NT-proBNP (>500 pg/ml).

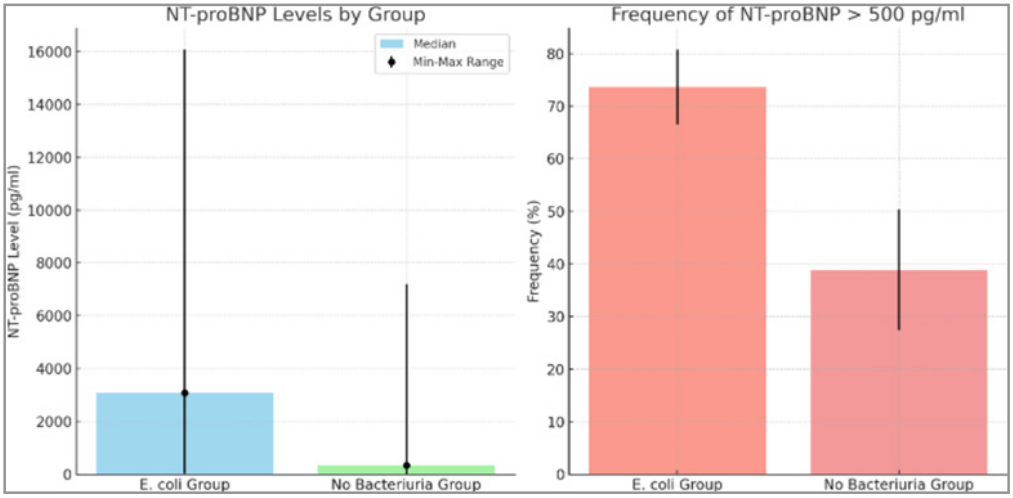


**Figure 1:** NT-pro BNP levels in E. coli positive and negative patients' groups

## Discussion

This study results demonstrate the evidence of E. coli bacteriuria is associated with elevated NT-proBNP levels in patients with post-myocardial infarction cardiosclerosis. The obtained findings align with previous evidence showing that bacterial endotoxins can stimulate cardiac peptide secretion through cytokine-mediated mechanisms [1]. Notably, NT-proBNP elevation occurred independently of echocardiographic dysfunction, suggesting that infection-induced systemic inflammation or renal handling alterations might influence peptide clearance or synthesis, as was mentioned previously [5]. These results highlight the importance of evaluating infection status when interpreting elevated NT-proBNP values in cardiac patients to avoid misdiagnosis of decompensated heart failure [6-19].

## Conclusion

In patients with post-infarction cardiosclerosis, urinary E. coli infection was linked to a significant elevation in plasma NT-proBNP levels. This relationship underscores the need for careful interpretation of cardiac biomarkers in the context of bacterial infections. Routine microbiological screening may enhance diagnostic accuracy and prevent unnecessary therapeutic escalation.


## Acknowledgements

The authors thank the staff of the J.M. Abdullayev Scientific Research Institute of Cardiology for their technical and clinical support.


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