



## “Nesfatin-1: Opposing role with Angiopoietin -2 in Myocardial Infarction”

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### Abstract

**Background:** One of the main causes of morbidity and death in the world is myocardial infarction. Nesfatin-1, angiopoietin-2, and lipid abnormalities are risk factors linked to endothelial dysfunction and inflammation. **Objective:** The purpose of this study was to compare the levels of specific lipids in heart patients with those in healthy people. **Methodology:** From January 15, 2026 to May 31, 2026, this case-control study was carried out at the Maysan Center for Heart Diseases and Surgery. It comprised eighty cases, forty of which were myocardial infarction patients and forty of which were healthy controls participated in the study. Nesfatin-1 and angiopoietin-2 levels were measured along with body mass index, TC, TG, HDL-C, LDL-C, and VLDL-C. Patients with myocardial infarction had significantly higher BMI, TC, TG, LDL-C, and VLDL-C than the control group, but their HDL-C levels were significantly lower. Patients had higher levels of angiopoietin-2 and lower levels of nesfatin-1 when compared to healthy people. **Results:** According to ROC analysis, certain biomarkers have a high diagnostic power in distinguishing between patients and healthy people. **Conclusion:** In conclusion, lipid abnormalities are associated with myocardial infarction, especially elevated Angiopoietin-2 and decreased Nesfatin-1, suggesting their potential role in the pathophysiological processes underlying the disease's progression. This lends credence to the potential use of these biomarkers in early diagnosis and risk assessment for cardiovascular disease. [Bangladesh Journal of Infectious Diseases, June 2026;13(1):271-277]

**Keywords:** ROC curve; Myocardial infarction; Blood Lipids; Body Mass Index; Nesfatin-1; Angiopoietin-2

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### Introduction

Myocardial infarction (MI) is a serious cardiovascular disorder that increases death rates worldwide and is a significant cause of ischemic heart disease. Acute coronary artery blockage mostly results in myocardial ischemia and eventual myocardial tissue necrosis<sup>1</sup>. According to contemporary research on cardiovascular disorders, myocardial infarction (heart attack) is triggered by a complex interplay of mechanisms, including endothelial dysfunction, oxidative stress, and neurohormonal imbalances, rather than only coronary artery obstruction. These factors lead to

plaque instability, which in turn results in the formation of thrombus and consequent cardiac issues<sup>2,3</sup>.

Current research has demonstrated that oxidative stress, immune cell activity, microvascular dysfunction, and molecular inflammatory pathways are additional factors that worsen cardiomyopathy. Cardiomyopathy can be caused by any of these factors, and acute myocardial infarction can develop from chronic conditions alone<sup>3,4</sup>. The gold standard for diagnosing heart conditions is highly sensitive cardiac troponins<sup>5,6</sup>. Nevertheless, additional diagnostic biomarkers have been found in recent

studies. Thus, the purpose of this study was to examine how specific clinical factors might be used to predict cardiomyopathy prior to its onset<sup>2,7</sup>. Neural tissue, peripheral blood vessels, and the heart expressed the novel biomarker Nesfatin-1, which was derived from nucleobendine-2 (NUCB2)<sup>8,9</sup>. Recent research has shown how oxidative stress and endothelial function are important mechanisms in the development of coronary artery disease, atherosclerosis, and cardiovascular disease<sup>10</sup>. It has also been linked to myocardial infarction; patients with cardiomyopathy have different levels of this peptide in their blood. There may be a link between Nesfatin-1 and myocardial infarction because heart patients and some individuals with inflammatory disorders have decreased levels of Nesfatin-1<sup>11</sup>. Consequently, this new biomarker is considered a promising diagnostic for the identification of cardiac disease<sup>12,13</sup>.

However, further research is needed to confirm these findings. Other research has demonstrated that nesfatin-1 functions as a cardioprotective agent by reducing oxidative stress and cell death<sup>14</sup>. Recent studies have clarified the role of angiopoietin-2 (Ang-2) as a vascular growth factor. Angiogenesis, endothelial cell activity, and blood vessel remodeling all depend on it. It primarily aids in the synthesis of Tie2 receptors, which lead to vascular instability, increased endothelial permeability, and inflammation. Because of these vital roles, recent research has highlighted its involvement in inflammatory cardiovascular disorders<sup>15,16</sup>.

It has been demonstrated that elevated Ang-2 levels are significantly linked to a higher risk of cardiovascular conditions such as heart failure and coronary artery disease, which can lead to atherosclerosis and eventually cardiomyopathy<sup>17</sup>. Patients with myocardial infarction have been observed to have elevated angiopoietin levels, which exacerbate myocardial infarction symptoms and impair myocardial perfusion due to increased capillary leakage, increased vascular permeability, and increased endothelial dysfunction<sup>18</sup>. Angiopoietin is an important biomarker in the diagnosis of myocardial infarction because it indicates the extent of endothelial inflammation and vascular damage<sup>19</sup>.

**Angiopoietin-2** A may promote vascular remodeling by raising hypoxia, which may worsen heart failure and lead to the formation of new blood vessels, according to some study<sup>20</sup>. As a result, angiopoietin-2 is a promising biomarker for heart disease prediction and a possible therapeutic

target<sup>21</sup>. The goals of this study are to determine the complementary role of angiopoietin-2 and nesfatin-1 in regulating inflammatory and vascular responses, to demonstrate their correlation with myocardial infarction patients, and to predict their potential use as clinical markers and biomarkers in disease diagnosis.

## Methodology

**Study Settings and Population:** This case-control study was conducted at the Maysan Center for Cardiac Diseases and Surgery between January 15, 2026, and May 31, 2026. A total of 80 cases were included: 40 patients with myocardial infarction and 40 healthy individuals as a control group. Patients with diabetes, smoking, atherosclerosis, and cardiovascular disease were excluded from the study, with the exception of the myocardial infarction patients included in the study.

**Study Procedure:** A laboratory kit made by BIOLABO SAS France was used to measure blood lipid profiles, including cholesterol, triglycerides, and high-density lipoprotein (HDL) cholesterol. High-sensitivity troponin levels were assessed, and the Friedewald equation was used to determine very low-density lipoprotein (VLDL) and Low density lipoprotein (LDL). The body mass index was calculated for patients and control groups so age range of the study group was established as 45 to 55 years. Both groups' systolic and diastolic blood pressure as well as their body mass index were also measured. Nesfatin-1 and angiopoietin-2 were measured by sandwich Enzyme-linked Immunosorbent Assay (ELISA) using kits provided by Bioassay Technology Laboratory for nesfatin-1 and Abcam UK for angiopoietin-2.

**Statistical Analysis:** All statistical analyses were conducted using Graph Pad Prism version 6 and SPSS version 25.0. Depending on the data being studied, different analysis methods were used. The t-test was used to compare two sets of data based on whether the distribution was normal or skewed. \* =  $p < 0.05$ , =  $p < 0.01$ , and \* =  $p < 0.001$  were used in tables to indicate statistical significance for so that (P values less than 0.05) consider significant analysis.

## Ethical Consideration:

After receiving ethical approval from the University of Maysan's College of Dentistry's Institutional Committee for Scientific Ethics, this study was carried out. Samples were taken at the the Maysan Center for Cardiac Diseases and Surgery between January 15, 2026, and May 31, 2026

## Results

From table (1) we found no statistically significant differences were when the ages of patients and healthy individuals were compared using the t-test ( $P=0.553$ ). However, there was a highly significant difference ( $p=0.002$ ) in body mass index between patients and healthy individuals. In contrast, we found highly significant differences ( $p<0.001$ ) in systolic and diastolic blood pressure, cholesterol levels, triglycerides, high-density lipoprotein and low-density lipoprotein levels, Nesfatin-1 levels, and angiopoietin-2 levels between patients and healthy individuals.

Simultaneously, Table 2 show a non-significant negative correlation coefficient between Nesfatin-1 and age, with the corresponding correlation coefficient and p-values being ( $r=-0.125$   $p=0.448$ , Body mass index and nesfatin-1 showed a significant positive correlation ( $r=0.389$   $p=0.014$ ), Systolic and diastolic blood pressure and Nesfatin

-1 had a very weak inverse relationship, according to correlation analysis, with correlation coefficients of ( $r = -0.035$ ,  $r=-0.153$ ). There was no significant correlation between them, as this relationship was not statistically significant ( $P > 0.05$ ). So Total Cholesterol with Nesfatin-1 showed a significant positive correlation ( $r=0.369$   $p=0.021$ ), non-significant correlation between Triglyceride with Nesfatin -1 ( $r= 0.093$ )  $p>0.05$ , as well as we show a weak inverse relationship between HDL with Nesfatin -1 ( $r = -0.145$ ) according to correlation analysis, but this relationship was not statistically significant ( $P = 0.313$ ), suggesting that there was no meaningful correlation between them.

Similarly from table 2, which examines the correlations between, angiopoietin-2, and the other variables in the patient group, reveals a non-significant negative correlation coefficient between angiopoietin-2 and age, with the corresponding correlation coefficient and p-values

**Table (1) illustrated Mean $\pm$  SD for all parameters between control and case with MI T-TEST**

| Group Statistics |         |    |          |                |               |
|------------------|---------|----|----------|----------------|---------------|
|                  | Groups  | N  | Mean     | Std. Deviation | P value       |
| age              | Control | 40 | 48.5750  | 2.77246        | 0.553         |
|                  | Case    | 40 | 48.9744  | 3.17463        |               |
| weight           | Control | 40 | 74.5750  | 10.22788       | 0.075         |
|                  | Case    | 40 | 78.2821  | 7.85376        |               |
| high             | Control | 40 | 168.9750 | 5.10649        | 0.039*        |
|                  | Case    | 40 | 166.0513 | 7.13755        |               |
| BMI              | Control | 40 | 26.1019  | 3.26654        | 0.002*        |
|                  | Case    | 40 | 28.3949  | 2.92394        |               |
| highpre          | Control | 40 | 12.40    | .496           | $P < 0.001^*$ |
|                  | Case    | 40 | 14.18    | 1.805          |               |
| lowpre           | Control | 40 | 8.0000   | .00000         | $P < 0.001^*$ |
|                  | Case    | 40 | 8.3590   | .48597         |               |
| TC               | Control | 40 | 157.2500 | 27.36622       | $P < 0.001^*$ |
|                  | Case    | 40 | 324.9231 | 34.00726       |               |
| TG               | Control | 40 | 93.8000  | 8.25616        | $P < 0.001^*$ |
|                  | Case    | 40 | 252.6923 | 48.86725       |               |
| HDL              | Control | 40 | 52.3250  | 5.31272        | $P < 0.001^*$ |
|                  | Case    | 40 | 36.8974  | 3.23464        |               |
| LDL              | Control | 40 | 86.1650  | 27.34853       | $P < 0.001^*$ |
|                  | Case    | 40 | 238.6872 | 35.84754       |               |
| VLDL             | Control | 40 | 18.7150  | 1.64668        | $P < 0.001^*$ |
|                  | Case    | 40 | 50.6256  | 9.71577        |               |
| nesfatin         | Control | 40 | 5.6325   | .59974         | $P < 0.001^*$ |
|                  | Case    | 40 | 2.7718   | .49839         |               |
| Aang             | Control | 40 | 27.8350  | 2.35498        | $P < 0.001^*$ |
|                  | Case    | 40 | 37.2692  | 2.43128        |               |

**Table 2: Correlation between Nesfatin -1 and angiopoietin-2 with all parameters for the patient group with MI**

| Group Statistics |   |       |         |         |         |         |        |        |         |       |        |         |          |        |
|------------------|---|-------|---------|---------|---------|---------|--------|--------|---------|-------|--------|---------|----------|--------|
|                  |   | age   | weight  | high    | BMI     | highpre | lowpre | TC     | TG      | HDL   | LDL    | VLDL    | nesfatin | Aang   |
| age              | r | 1     | -.027   | -.095   | .078    | -.142   | .091   | -.034  | -.125   | .207  | .297   | .292    | -.125    | -.073  |
|                  | P |       | .870    | .564    | .636    | .390    | .580   | .837   | .447    | .205  | .067   | .071    | .448     | .660   |
| weight           | r | -.027 | 1       | .404*   | .636**  | -.225   | -.193  | .036   | -.444** | -.109 | .202   | -.513** | .227     | .188   |
|                  | P | .870  |         | .011    | .000    | .169    | .240   | .828   | .005    | .510  | .218   | .001    | .164     | .252   |
| high             | r | -.095 | .404*   | 1       | -.446** | .106    | -.104  | -.343* | -.255   | -.117 | -.269  | -.251   | -.193    | .055   |
|                  | P | .564  | .011    |         | .004    | .523    | .528   | .032   | .117    | .477  | .098   | .123    | .240     | .739   |
| BMI              | r | .078  | .636**  | -.446** | 1       | -.317*  | -.084  | .343*  | -.204   | -.013 | .421** | -.279   | .389*    | .133   |
|                  | P | .636  | .000    | .004    |         | .050    | .612   | .032   | .213    | .939  | .008   | .085    | .014     | .421   |
| highpre          | r | -.142 | -.225   | .106    | -.317*  | 1       | -.075  | -.029  | .120    | .116  | -.096  | .058    | -.035    | .019   |
|                  | P | .390  | .169    | .523    | .050    |         | .648   | .859   | .467    | .482  | .560   | .727    | .832     | .910   |
| lowpre           | r | .091  | -.193   | -.104   | -.084   | -.075   | 1      | -.022  | -.133   | -.143 | .109   | -.115   | -.153    | -.402* |
|                  | P | .580  | .240    | .528    | .612    | .648    |        | .893   | .421    | .384  | .509   | .487    | .353     | .011   |
| TC               | r | -.034 | .036    | -.343*  | .343*   | -.029   | -.022  | 1      | .175    | -.163 | .155   | -.045   | .369*    | .035   |
|                  | P | .837  | .828    | .032    | .032    | .859    | .893   |        | .288    | .322  | .347   | .785    | .021     | .833   |
| TG               | r | -.125 | -.444** | -.255   | -.204   | .120    | -.133  | .175   | 1       | .158  | -.182  | .666**  | .093     | -.042  |
|                  | P | .447  | .005    | .117    | .213    | .467    | .421   | .288   |         | .337  | .266   | .000    | .572     | .800   |
| HDL              | r | .207  | -.109   | -.117   | -.013   | .116    | -.143  | -.163  | .158    | 1     | .074   | .203    | -.145    | .313   |
|                  | P | .205  | .510    | .477    | .939    | .482    | .384   | .322   | .337    |       | .653   | .214    | .377     | .053   |
| LDL              | r | .297  | .202    | -.269   | .421**  | -.096   | .109   | .155   | -.182   | .074  | 1      | -.134   | .199     | -.111  |
|                  | P | .067  | .218    | .098    | .008    | .560    | .509   | .347   | .266    | .653  |        | .415    | .225     | .502   |
| VLDL             | r | .292  | -.513** | -.251   | -.279   | .058    | -.115  | -.045  | .666**  | .203  | -.134  | 1       | -.043    | -.181  |
|                  | P | .071  | .001    | .123    | .085    | .727    | .487   | .785   | .000    | .214  | .415   |         | .794     | .270   |
| nesfatin         | r | -.125 | .227    | -.193   | .389*   | -.035   | -.153  | .369*  | .093    | -.145 | .199   | -.043   | 1        | .116   |
|                  | P | .448  | .164    | .240    | .014    | .832    | .353   | .021   | .572    | .377  | .225   | .794    |          | .483   |
| Aang             | r | -.073 | .188    | .055    | .133    | .019    | -.402* | .035   | -.042   | .313  | -.111  | -.181   | .116     | 1      |
|                  | P | .660  | .252    | .739    | .421    | .910    | .011   | .833   | .800    | .053  | .502   | .270    | .483     |        |

\*. Correlation is significant at the 0.05 level (2-tailed).; \*\*. Correlation is significant at the 0.01 level (2-tailed).

**Table 3: ROC Analysis for Case and Control Groups**

| Parameter | AUC   | SE    | P value | Cut-off value | Sensitivity | Specificity |
|-----------|-------|-------|---------|---------------|-------------|-------------|
| Age       | 0.533 | 0.066 | 0.617   | 47.5          | 60.0%       | 40.0%       |
| Weight    | 0.615 | 0.065 | 0.078   | 75.5          | 69.0%       | 60.0%       |
| High      | 0.350 | 0.063 | 0.022   | 167.5         | 38.5%       | 60.0%       |
| BMI       | 0.699 | 0.059 | 0.002   | 27.87         | 64.0%       | 67.5%       |
| Hp        | 0.969 | 0.026 | 0.000   | 13.5          | 95.0%       | 100.0%      |
| Lp        | 0.679 | 0.061 | 0.006   | 8             | 75.0%       | 90.0%       |
| TC        | 1.000 | 0.000 | 0.000   | 231           | 100.0%      | 100.0%      |
| TG        | 1.000 | 0.000 | 0.000   | 157.5         | 100.0%      | 100.0%      |
| HDL       | 0.004 | 0.004 | 0.000   | 45.5          | 97.5%       | 92.5%       |
| LDL       | 1.000 | 0.000 | 0.000   | 147.9         | 100.0%      | 100.0%      |
| VLDL      | 1.000 | 0.000 | 0.000   | 31.5          | 100.0%      | 100.0%      |
| Nesfatin  | 0.000 | 0.000 | 0.000   | 4.35          | 100.0%      | 100.0%      |
| Ang-2     | 1.000 | 0.000 | 0.000   | 33.05         | 100.0%      | 100.0%      |

**FiFigure I: ROC analysis for patients and control groups**

being  $r=-0.073$   $p=0.660$ ). whereas body mass index and angiotensin-2 showed a positive but non-significant correlation ( $r=0.133$   $p=0.421$ ). there was no statistically significant correlation ( $r=0.019$ ) between angiotensin-2 and systolic blood pressure. Diastolic blood pressure and angiotensin-2 did, however, show a moderate inverse correlation coefficient of  $-0.402$ , which was not statistically significant  $p>0.05$ . Also no statistically significant correlation ( $r=0.035$ ) between angiotensin-2 and Total Cholesterol  $p>0.05$ , non-significant correlation between Triglyceride with Angiotensin-2 ( $r=-0.042$ )  $p>0.05$ . High-density lipoprotein and angiotensin-2 showed a positive correlation ( $r=0.313$ ). Still, this correlation was not statistically significant ( $P=0.053$ ), suggesting a potential trend in the relationship without definitive statistical confirmation. Since the correlation coefficient values  $-0.502$  ultimately there was positive correlation but non-significant between nesfatin-1 and angiotensin-2 ( $r=0.116$ ,  $p=0.483$ )

From table (3) and figure (1) we show AUC for nesfatin-1 = 0.000 while cut-off = 4.35, similarly the AUC for angiotensin-2 = 1.000 while cut-off = 33.05.

## Discussion

A disorder in lipid metabolism brought on by obesity linked to heart disease can be the cause of low levels of HDL. A lipid profile marked by atherosclerosis, high levels of hardened fats, and low levels of good HDL cholesterol-which carries cholesterol from cells to the liver to remove its detrimental effects by changing it into a form the body can use it frequently brought on by high body mass index, total cholesterol, triglycerides, and low-density lipoprotein. Consequently, coronary heart disease is caused by low HDL levels<sup>22</sup>.

Taking the graph into consideration, variables like weight, TC, and BMI appear to perform better because their curves are higher and closer to the upper-left corner<sup>23</sup>. Because their curves remain closer to the reference line, variables like HDL appear weaker.

Age, weight, body mass index (BMI), blood pressure, lipid profile markers, and biochemical indicators are among the anthropometric, clinical, and biochemical parameters whose diagnostic performance is assessed using the Receiver Operating Characteristic (ROC) curve analysis shown in Figure 1 and Table 4. A popular statistical method for evaluating the sensitivity and specificity of diagnostic tests at various

threshold values is the ROC curve<sup>24</sup>. Higher diagnostic accuracy is indicated by curves in ROC analysis that are closer to the upper-left corner, whereas a test with no discriminatory power (random classification) is represented by the diagonal reference line. Certain variables, like BMI, VLDL, and high blood pressure, demonstrated superior predictive performance in the current investigation since their ROC curves were farther from the reference line, indicating greater sensitivity and specificity. On the other hand, variables with curves close to the diagonal line showed less discriminative power. As a result, ROC curve analysis offered a useful comparison of the parameters' predictive ability in differentiating the groups under investigation<sup>25</sup>.

Nesfatin-1 levels were significantly lower in cardiac patients than in the control group, but this is because the peptide's protective anti-inflammatory and anti-oxidative stress effects are lost, which causes endothelial dysfunction and atherosclerosis. On the other hand, patients had significantly higher levels of Angiotensin-2, which was linked to the inflammatory response and increased activation of endothelial cells in cardiovascular diseases. Consequently, the development of coronary heart disease, which is associated with integrated mechanisms such as oxidative stress, chronic inflammation, and vascular instability, may be caused by a decrease in Nesfatin-1 and an increase in Angiotensin-2 level<sup>26,27</sup>.

According to the ROC curve, Nesfatin-1 had a sensitivity and specificity of 100% and an area under the curve (AUC) of 0.000. This is explained by the variable's inverse relationship between patients and the control group, where patient values fell rather than rose. This does not indicate subpar diagnostic performance; rather, it would have shown a 100% diagnostic capability if the values had been reversed and increased in patients compared to healthy individuals. However, angiotensin-2 demonstrated an AUC of 1.000 with a sensitivity and specificity of 100% when compared between patients and healthy individuals, indicating optimal diagnostic capability in differentiating between patients and healthy individuals.

## Conclusion

We draw the conclusion from this study that the significance of the findings is not restricted to the conventional biomarkers for determining the percentage of fats and their direct impact on the occurrence of heart diseases, particularly myocardial infarction. This conclusion is supported by the findings for both nesfatin-1 and angiotensin-2. As

significant biomarkers in the high diagnostic ability to predict myocardial infarction. Because there are few local studies that have examined these contemporary biomarkers in assessing the combined diagnostic ability of metabolic and biomarkers in patients with myocardial infarction, this study helps close the research gap that resulted in challenges in interpreting the results. This offers fresh evidence for the use of nesfatin-1 and angiopoietin -2 A as novel biomarkers to aid in early disease detection, evaluate the precision of the injury, and lessen disease complications. Therefore, this study may help improve diagnosis, treatment, and prevention methods for the condition's progression.

#### Acknowledgements

I would want to express my gratitude to everyone who helped to finish this study, to all the patients who took part in giving blood samples, and especially to the entire staff of the Maysan Center for Heart Diseases and Surgery. contributed blood samples, and we would especially want to thank the entire personnel at the Maysan Center for Heart Diseases and Surgery.

#### Conflict of interest:

As the researcher, I certify that I have no competing interests.

#### Financial Disclosure

This study was entirely self-funded and did not depend on any government or private support.

#### Authors' contributions

The author contribution in methodology, sample collection, and laboratory work and wrote the manuscript

#### Data Availability

Any questions regarding the availability of the study's supporting data should be addressed to the corresponding author, who can provide it upon justifiable request.

#### Ethics Approval and Consent to Participate

Ethical approval for this study was granted by the University of Misan Since this was a follow-up study, every study participant provided formal informed consent. Each method followed the appropriate rules and regulations.

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**How to cite this article:** Mohaisen IK. Nesfatin-1: Opposing role with Angiopoietin-2 in Myocardial Infarction. Bangladesh J Infect Dis 2026;13(1):271-277

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#### Article Info

Received on: 1 April 2026

Accepted on: 12 May 2026

Published on: 1 June 2026

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