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Homocysteine and its potential in acute myocardial infarction diagnosis

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ABSTRACT

Cardiovascular disorders are the main cause of death in the world. These include various incidents like cerebral or myocardial infarction. Such an important issue necessitates finding a marker in order to predict mentioned events. Recent studies suggest that homocysteine, an amino acid involved in the homocysteine-methionine cycle, may serve this role, as its elevated levels are common in patients with cardiovascular conditions. This review aims to assess its predictive value. For this review, studies published in English from January 2017 to July 2025 were selected from the PubMed database. In conclusion, the association between homocysteine and cardiovascular disorders is complex and requires additional investigation. However, high blood homocysteine levels are independently a risk factor for numerous cardiovascular events, such as acute myocardial infarction, stroke, coronary artery disease, and thrombotic episodes. Such a correlation provides a marker that can be used to monitor, evaluate risk, and predict the aforementioned events.

Keywords: homocysteine, myocardial infarction

1. INTRODUCTION

One of the most urgent problems of today's medicine is mortality, its most common reasons and how to decrease it. Myocardial infarction and other cardiovascular events tend to be major causes of death in the elderly. Throughout years it used to be a topic of multiple research trying to clarify and solve the problem.

Recent studies have shown that patients who experience these incidents often exhibit elevated blood homocysteine levels. Homocysteine, a metabolite in the homocysteine-methionine cycle, is commonly corelated with arteriosclerosis, a key factor to cardiovascular disease (Jakubowski & Witucki, 2025; ALSolami et al., 2023). Such a connection itself is a vast discovery that can have a great potential. Based on relationships between homocysteine and cardiovascular incidents, it is possible to gain a new metabolic marker that can play a crucial role in prevention of one of the most common causes of death in the world.

This review aims to explore further the correlation between higher plasma homocysteine levels and myocardial infarcts, and to assess whether homocysteine can be used as a predictive marker for cardiovascular events.

2. REVIEW METHODS

The study is based on the PubMed database. Publications analyzed for that purpose were from January 2017 to July 2025. The articles were in English, with no other languages included. Search strategy used a mix of keywords: "homocysteine", "cardiovascular incidents", and "infarction".

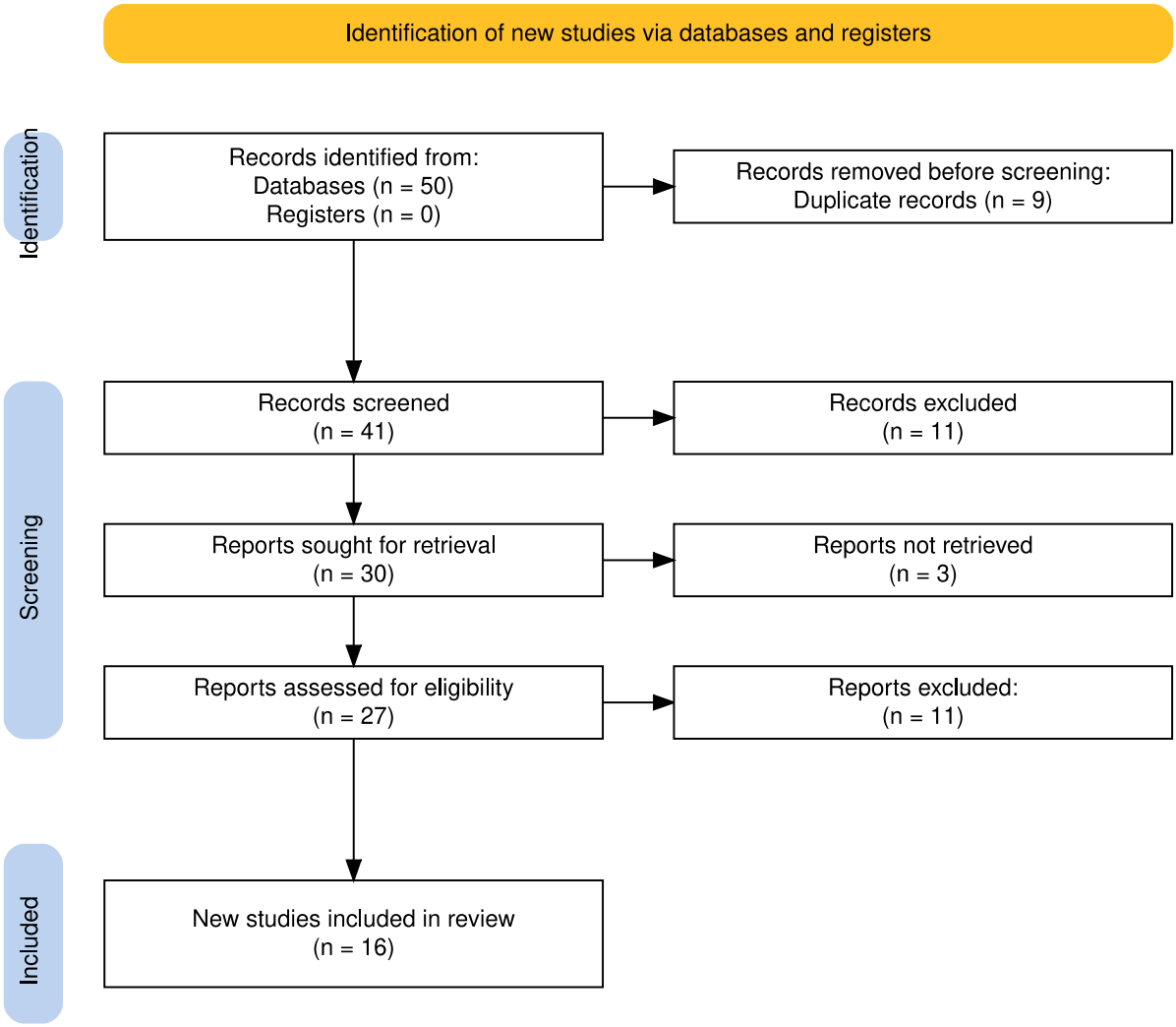

Figure 1. PRISMA flow diagram.

The inclusion criteria required that studies must show straight connection between metabolic disorders, it's effect on serum homocysteine and impact on cardiovascular system. A clear and vast perspective with an accurate description of all the processes involved in homocysteine interaction was crucial to prepare a meritorious review. After the search, screening and exclusions, 16 articles met the criteria and joined in the analysis (Figure 1).

3. RESULTS & DISCUSSION

Cardiovascular incidents

Currently, cardiovascular diseases (CVD), particularly ischemic heart and brain diseases, are the leading causes of mortality and disability. The Global Burden of Disease Study reveals that the prevalence of all CVD nearly doubled from 271 to 523 million in years 1990-2019. Likewise, deaths attributable to these diseases increased from 12.1 million to 18.6 million, and might possibly reach 23 million by 2030 (Roth et al., 2020).

Nearly every country is seeing an increase in the burden of CVD, and those highly developed nations that had previously seen a drop in CVD prevalence are also seeing an increase (Bajic et al., 2022). Family history of CVD, smoking, sedentary lifestyle, obesity, alcohol consumption, diabetes, dyslipidemia, and hypertension are the primary cardiovascular risk factors.

All of these promote the uptake of low-density lipoprotein (LDL) by the vascular wall, leading to lipid accumulation in the subendothelial layer and atherogenesis. Atherogenesis causes the development of foam cells and the activation of endothelial cells by monocytes are caused. The accumulation of smooth muscle cells in artery walls exacerbates atherosclerosis, which might result in the formation of a fibrous atheroma cap. All the mentioned processes, combined with elevated blood pressure and cholesterol levels, worsens atherosclerosis.

Dysregulation of the methionine cycle, resulting in elevated homocysteine levels, is strongly associated with hypertension. Elevated homocysteine can damage the vascular endothelium and contribute to the development of cardiovascular disease (Bajic et al., 2022; Paganelli et al., 2021).

Acute coronary syndrome encompasses several clinical presentations, including unstable angina pectoris, acute myocardial infarction (AMI) with ST-segment elevation, acute myocardial infarction (AMI) without ST-segment elevation, and sudden cardiac death (Aydin et al., 2019). One of the leading causes of death and morbidity in the world is acute myocardial infarction (AMI). A heart attack diagnosis is in about 10% of patients admitted to emergency rooms with chest pain every year. AMI occurs when an atherosclerotic plaque ruptures, leading to a growing thrombus that partially or completely blocks the coronary artery and limits blood flow to the heart. It can result from either ischemic heart disease or coronary artery disease in combination.

Due to the low sensitivity and specificity of ECG in diagnosing AMI, the European Society of Cardiology (ESC) together with the American College of Cardiology (ACC) established criteria for AMI. At least two of the following must be met by patient: characteristic symptoms, a visible rise or fall in cardiac biomarkers (such as CK-MB), preferably serum troponins (cTnI or TnT), or a specific ECG pattern with Q waves indicative of AMI (Aydin et al., 2019).

Myocardial necrosis and a decline in ventricular function are the results of ischemia brought on by reduced coronary artery flow. In addition, enzymes like ALT, AST, LDH, CK, and troponins have long usage as markers for an AMI diagnosis, since their levels were usually high because of the necrosis. Therefore, in our study, cardiac markers (cTnI and cTnT), CK-MB, and myoglobin, which are standard in diagnosing AMI and determining prognosis, are mentioned in the review. We intend to discuss some other important candidate biomarkers, such as copeptin and irisin, which may be relevant in diagnosing AMI and determining prognosis (Aydin et al., 2019).

Homocysteine

Homocysteine (Hcy) is a non-essential amino acid, that through its interaction with folic acid and vitamin B12, plays a crucial role in the Hcy-methionine cycle (Gospodarczyk et al., 2022). The sulfur amino acid Hcy is an intermediary metabolite in the metabolic pathways of methionine (Met) and cysteine (Cys), two classical sulfur-containing amino acids involved in the genetic code.

Methionine produced in the gut from dietary protein is the only source of Hcy in the human body. It (Met) participates in the synthesis of new proteins as well as the universal methyl donor S-adenosylmethionine (AdoMet), which is transformed into S-adenosylhomocysteine, the immediate precursor of Hcy by cellular methylating enzymes. The two primary reactions that regulate Hcy levels are transsulfuration to cysteine (reaction (II)), which is catalyzed by CBS and cystathionine γ-lyase (CSE), and remethylation back to Met (reaction (I)), which is catalyzed by Met synthase (with methyltetrahydrofolate cofactor supplied by the MTHFR enzyme) and betaine-Hcy methyltransferase. Despite not being involved in protein production, the sulfur component of Hcy is also incorporated into proteins by post-translational processes (Jakubowski & Witucki, 2025).

Disruption of these metabolic processes may result in Hcy accumulation, leading to clinically significant outcomes such as calcified vascular tissues, atherothrombosis, cardiac vasculopathy, and impaired cognition. The suggested mechanisms involved in homocysteine-induced atherosclerosis include: a) endothelial injury, which appears to be mediated by oxidative stress; b) smooth muscle proliferation; c) oxidative modification of low-density lipoproteins; d) endothelial-leukocyte interactions; e) reduced fibrinolytic activity, and f) direct effects on platelets. The precise weight of each of these pathways, however, remains unknown, particularly since many findings have come from in vitro research using homocysteine concentrations significantly higher than those found in humans (Miñana et al., 2021; Esse et al., 2019).

In the past, the majority of clinical research measured plasma tHcy to assess the relationships between HHcy and stroke and CVD. Major oxidized Hcy forms, including Hcy bound by disulfide linkages to plasma protein thiols (S-Hcy-protein) and to small molecular

weight thiols (Hcy-S-S-Cys and Hcy-S-S-Hcy), are included in the composite marker known as plasma tHcy. One to two percent of plasma tHcy consists of free reduced Hcy, a small component. Nevertheless, a number of other significant Hcy metabolites, including Hcy-thiolactone, N-Hcy-protein, cystathionine, and AdoHcy, are absent from tHcy. It is difficult to definitively link the observed pathology to a specific metabolite in HHcy, as changes occur not only in plasma tHcy levels but also in levels of Hcy-related metabolites (Mundi et al., 2018).

Some studies though have evaluated specific relationships between cardiovascular incidents like myocardial infarction, stroke, mortality not only with certain Hcy metabolites, including AdoHcy, cystathionine and Hcy-thiolactone, but also with anti-N-Hcy-protein autoantibodies (Jakubowski & Witucki, 2025). Increased levels of Hcy in plasma, or hyperhomocysteinemia (HHcy), are categorized as moderate (15–30 μmol/L), medium (30–100 μmol/L), or severe (greater than 100 μmol/L). The following five categories comprise the causes of HHcy: (i) enzyme disorders; (ii) cofactor deficiencies; (iii) excessive methionine intake; (iv) certain diseases like hypothyroidism, anemia, malignant tumors, or chronic renal failure; and (v) the use of certain medications like methotrexate, cholestyramine, oral contraceptives, phenytoin, carbamazepine, or metformin.

Enzyme problems, such as deficiencies or hereditary influences, are a common cause of HHcy in Hcy metabolism, as mentioned above. For instance, because CBS is essential for Hcy to cystathionine conversion, its absence may result in increased Hcy concentrations, since insufficient amounts of Hcy are converted to cystathionine in the transsulfuration process (Bajic et al., 2022; Smith & Refsum, 2021).

Deficits in vitamins B2, B6, and B12, which are cofactors involved in the metabolism of Hcy, can also result in HHcy. Mentioned vitamins are readily eliminated in the urine since they are soluble in water, what can result in their shortage. Older adults are frequently deficient in vitamins B12 and B2, which are involved in the remethylation process, and vitamin B6, which is a cofactor in the transsulfuration process. HHcy is therefore more prevalent in older people (Djurovic et al., 2020).

As an essential amino acid, methionine is the only food source of Hcy. Methionine-rich diet increases Hcy production in the body. In comparison to mice on a balanced diet, animals fed diets high in methionine, or Hcy, showed greater urine amounts of homocysteine-thiolactone.

Reduced renal excretion and glomerular filtration may be the cause of HHcy in renal failure. Additionally, kidney tissue contains the enzymes involved in transsulfuration and remethylation. Nevertheless, these enzymes are dormant in chronic renal failure. Impaired folate metabolism is one of the non-renal causes of HHcy in renal failure (ALSolami et al., 2023).

Patients with both clinical and subclinical types of hypothyroidism have elevated Hcy values. Consistent information about the HHcy mechanism for this illness is lacking. Elevated Hcy levels links to Hashimoto thyroiditis. Hcy concentrations are higher in patients with iatrogenic hypothyroidism than in those without the condition. Difference in Hcy levels implies that Hcy levels may rise due to immunological inflammatory disorders.

Also, tumor cells release significant amounts of Hcy. Rapidly multiplying tumor cells consume folate for their metabolism and inactivate the remethylation pathway mediated by MS, producing HHcy.

Increased expression of the N-methyl-D-aspartate (NMDA) receptor in HHcy links to neurotoxicity and DNA damage. Additionally, HHcy raises the risk of bone fracture since it modifies the microarchitecture of the bone. Not only that, according to an experimental study, Hcy can change heart function by lowering cardiac acetylcholinesterase activity, leading to bradycardia (Bajic et al., 2022).

In recent years, more and more clinical studies on homocysteine have been published. Some of their results gave homocysteine a new role, as a potential marker, since its multiple connections with metabolic diseases were discovered (Table 1). Patients with CAD, stroke, peripheral artery disease, and venous thrombosis have a high prevalence of increased homocysteine plasma levels, according to several epidemiological studies. Prospective investigations associated blood homocysteine levels with long-term outcomes in patients with STEMI, NSTACS, and without prior CAD. More recently, a greater risk of short-term death and nonfatal ischemic events occurred in individuals with NSTEACS and STEMI. In these investigations, the risk of new recurrent coronary ischemia events during long-term follow-up was not addressed (Miñana et al., 2021; Fan et al., 2017).

A different study from 2023 showed a strong correlation between serum homocysteine level and elevated lipid levels, inflammation, infarct size, and major adverse cardiac event risk in AMI patients. Serum Hcy levels among AMI patients were significantly higher compared to angina pectoris patients (Ren et al., 2023). Likewise, conclusion on homocysteine and MI connection was stated in a study from Romania published in 2020. It focused on the correlation between plasma homocysteine level and the first myocardial infarct in young patients. 61 patients took part, of whom 28 were in the study group, and 33 were in the control group.

Results of that case pointed out a strong correlation between a higher level of serum homocysteine and the appearance of first in life event of acute MI in patients younger age (Nedelcu et al., 2021).

In a retrospective cohort study published in January 2017 to July 2025, where the correlation between homocysteine level and new in-hospital new-onset atrial fibrillation among patients experiencing acute myocardial infarction was analyzed, results were nonlinear. The association was significantly higher among patients with smaller left atrial anterior-posterior diameter, possible sign of left ventricular systolic dysfunction (Jin et al., 2025).

A different study published in 2021 evaluated higher homocysteine levels as a predictor of ischemic cardiac events after an acute coronary syndrome. The connection between homocysteine at discharge and recurrent MI was investigated by bivariate negative binomial regression. Mortality was accounted as a competing event to myocardial infarction. At a median of 9.7 years, 709 (54.3%) fatalities occurred and 779 recurrent MIs in 478 (36.6%) cases. Patients in the upper quartiles of homocysteine had a higher incidence of recurrent MI ($p < 0.001$). After multivariate adjustment, homocysteine levels demonstrated an approximately linear association with increased risk of all-cause mortality ($p < 0.001$) and recurrent myocardial infarction (MI) ($p = 0.001$). These findings indicate a significant relationship between elevated homocysteine concentrations and the risk of recurrent MI in patients with acute coronary syndrome (ACS) during very-long-term follow-up (Miñana et al., 2020).

Table 1. Summary of most important studies and its findings in connection between homocysteine and cardiovascular incidents

Study	Year	Cardiovascular incident	Main findings
Fan et al.,	2017	STEMI	Elevated serum Hcy level, equally useful as GRACE score
Ren et al.,	2023	AMI	Elevated serum Hcy level in comparison to Angina pectoris
Nedelcu et al.,	2023	MI among young patients	Elevated serum Hcy levels
Jin et al.,	2025	NOAF in MI patients	Elevates serum Hcy levels in NOAF patients, nonlinear association
Miñana et al.,	2020	Reccurent MI among patients after ACS	Elevated serum Hcy levels in patients with reccurent MI

STEMI- ST-elevation myocardial infarction, Hcy- homocysteine, AMI- acute myocardial infarction, MI- myocardial infarction, NOAF- new-onset atrial fibrillation, ACS- acute coronary syndrome

4. CONCLUSION

Cardiovascular incidents are a very serious global problem. Since they are a major cause of death, they need special attention. The possibility of early detection would definitely contribute to reducing mortality caused by cardiovascular events, thanks to earlier detection and prevention. Such a potential has homocysteine. Being involved in pathological endothelium eruption mechanisms, which are further responsible for atherosclerosis and thrombotic events. As many studies have shown, it has a vast correlation with various cardiovascular incidents, including acute myocardial infarction. However, since many of homocysteine aspects remain unclear, its role as a potential marker of myocardial infarction still needs to be investigated. With further studies and determination of all its attachments, it is possible to receive a highly related and easy-to-use marker that can be used not only to monitor but also to prevent many cardiovascular events. That would be a massive improvement in treating not only cardiologically but also metabolically ill patients, and it would possibly help to reduce the number of deaths caused by acute cardiovascular incidents.

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Authors' Contributions

Marcin Kaniewski - Conceptualization, review and editing, investigation, methodology

Maja Kondratowicz- Methodology, investigation, visualization, supervision

Kamila Kałamarz- Conceptualization, visualization, resources
Kinga Żmuda- Review, data curation, investigation
Maciej Świerczyna- Resources, writing- rough preparation, data curation
Maja Czerniachowska- Visualization, data curation, investigation
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Michał Grabek- Resources, writing- rough preparation, formal analis
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Data and materials availability
All data associated with this study will be available based on the reasonable request to corresponding author.

REFERENCES

1. ALSolami AA, Almalki AA, Alhedyan SY, Alghamdi A, Alzahrani SM, Dause WR, Hamdi FA, Howladar MT, Ibrahim IA. Plasma Homocysteine Levels and Cardiovascular Events in Patients with End-Stage Renal Disease: A Systematic Review. Cureus 2023;15(6):e40357. doi: 10.7759/cureus.40357.
2. Aydin S, Ugur K, Aydin S, Sahin İ, Yardim M. Biomarkers in acute myocardial infarction: current perspectives. Vasc Health Risk Manag 2019;15:1-10. doi: 10.2147/VHRM.S166157.
3. Bajic Z, Sobot T, Skrbic R, Stojiljkovic MP, Ponorac N, Matavulj A, Djuric DM. Homocysteine, Vitamins B6 and Folic Acid in Experimental Models of Myocardial Infarction and Heart Failure-How Strong Is That Link? Biomolecules 2022;12(4):536. doi: 10.3390/biom12040536.
4. Djurovic Z, Jovanovic V, Obrenovic R, Djurovic B, Soldatovic I, Vranic A, Jakovljevic V, Djuric D, Zivkovic V. The importance of the blood levels of homocysteine, folate and vitamin B12 in patients with primary malignant brain tumors. J BUON 2020;25(6):2600-2607.
5. Esse R, Barroso M, Tavares de Almeida I, Castro R. The Contribution of Homocysteine Metabolism Disruption to Endothelial Dysfunction: State-of-the-Art. Int J Mol Sci 2019; 20(4):867. doi: 10.3390/ijms20040867.
6. Fan Y, Wang J, Zhang S, Wan Z, Zhou D, Ding Y, He Q, Xie P. Homocysteine enhances the predictive value of the GRACE risk score in patients with ST-elevation myocardial infarction. Anatol J Cardiol 2017;18(3):182-193. doi: 10.14744/AnatolJ Cardiol.2017.7798.
7. Gospodarczyk A, Marczewski K, Gospodarczyk N, Widuch M, Tkocz M, Zalejska-Fiolka J. Homocysteine and cardiovascular disease - a current review. Wiad Lek 2022;75:2862-2866. doi: 10.36740/WLek202211224.
8. Jakubowski H, Witucki Ł. Homocysteine Metabolites, Endothelial Dysfunction, and cardiovascular disease. Int J Mol Sci 2025;26(2):746. doi: 10.3390/ijms26020746.
9. Jin P, Wu P, Ma J, Bian Y, Kou H, Ma X, Jia S, Zheng Q. The relationship between homocysteine and new-onset atrial fibrillation in patients with acute myocardial infarction. Ann Med 2025;57(1):2530222. doi: 10.1080/07853890.2025.2530222.
10. Miñana G, Gil-Cayuela C, Fácila L, Bodi V, Valero E, Mollar A, Marco M, García-Ballester T, Zorio B, Martí-Cervera J,

Núñez E, Chorro FJ, Sanchis J, Núñez J. Homocysteine and long-term recurrent infarction following an acute coronary syndrome. Cardiol J 2021;28(4):598-606. doi: 10.5603/CJ.a2020.0170.

11. Mundi S, Massaro M, Scoditti E, Carluccio MA, van Hinsbergh VWM, Iruela-Arispe ML, De Caterina R. Endothelial permeability, LDL deposition, and cardiovascular risk factors-a review. Cardiovasc Res 2018;114(1):35-52. doi: 10.1093/cvr/cvx226.
12. Nedelcu C, Ionescu M, Pantea-Stoian A, Niţă D, Petcu L, Mazilu L, Suceveanu AI, Tuţă LA, Parepa IR. Correlation between plasma homocysteine and first myocardial infarction in young patients: Case-control study in Constanta County, Romania. Exp Ther Med 2021;21(1):101. doi: 10.3892/etm.2020.9533.
13. Paganelli F, Mottola G, Fromonot J, Marlinge M, Deharo P, Guieu R, Ruf J. Hyperhomocysteinemia and Cardiovascular Disease: Is the Adenosinergic System the Missing Link? Int J Mol Sci 2021;22(4):1690. doi: 10.3390/ijms22041690.
14. Ren L, Guo J, Zhao W, Zuo R, Guo S, Jia C, Gao W. Serum homocysteine relates to elevated lipid level, inflammation and major adverse cardiac event risk in acute myocardial infarction patients. Biomark Med 2023;17(6):297–306.
15. Roth GA, Mensah GA, Johnson CO, Addolorato G, Ammirati E, Baddour LM, Barengo NC, Beaton AZ, Benjamin EJ, Benziger CP, Bonny A, Brauer M, Brodmann M, Cahill TJ, Carapetis J, Catapano AL, Chugh SS, Cooper LT, Coresh J, Criqui M, DeCleene N, Eagle KA, Emmons-Bell S, Feigin VL, Fernández-Solà J, Fowkes G, Gakidou E, Grundy SM, He FJ, Howard G, Hu F, Inker L, Karthikeyan G, Kassebaum N, Koroshetz W, Lavie C, Lloyd-Jones D, Lu HS, Mirijello A, Temesgen AM, Mokdad A, Moran AE, Muntner P, Narula J, Neal B, Ntsekhe M, Moraes de Oliveira G, Otto C, Owolabi M, Pratt M, Rajagopalan S, Reitsma M, Ribeiro ALP, Rigotti N, Rodgers A, Sable C, Shakil S, Sliwa-Hahnle K, Stark B, Sundström J, Timpel P, Tleyjeh IM, Valgimigli M, Vos T, Whelton PK, Yacoub M, Zuhlke L, Murray C, Fuster V; GBD-NHLBI-JACC Global Burden of Cardiovascular Diseases Writing Group. Global Burden of Cardiovascular Diseases and Risk Factors, 1990-2019: Update from the GBD 2019 Study. J Am Coll Cardiol 2020;76(25):2982-3021. doi: 10.1016/j.jacc.2020.11.010. Erratum in: J Am Coll Cardiol 2021;77(15):1958-1959. doi: 10.1016/j.jacc.2021.02.039.
16. Smith AD, Refsum H. Homocysteine - from disease biomarker to disease prevention. J Intern Med 2021;290(4):826-854. doi: 10.1111/joim.13279.