

Hyperhomocysteinemia Causing Myocardial Infarction in a Young Patient: A Case Report

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Abstract

Hyperhomocysteinemia is an independent risk factor for acute myocardial infarction (MI). This case report describes a 19-year-old male with a marfanoid phenotype and no conventional risk factors presenting with acute MI. It highlights the significance of acknowledging hyperhomocysteinemia as a potential risk factor for MI, especially in young patients.

Categories: Cardiology

Keywords: acute myocardial infarction, case report, homocysteine, hyperhomocysteinemia, myocardial infarction

Introduction

An association between increased homocysteine levels and coronary heart disease has already been established [1]. Several case reports have been published on this condition [2-6]. All the cases included patients above 20 years of age. This case report identifies increased homocysteine levels as a cause of acute myocardial infarction (MI) in a patient of less than 20 years of age.

Case Presentation

A 19-year-old young man, with marfanoid phenotype, was brought to the emergency room of a tertiary care hospital in Pakistan complaining of central chest pain. The pain was crushing in nature, progressive, radiating into the back and left arm for six hours, accompanied by shortness of breath and apprehensions. At the time of presentation, the patient had a blood pressure of 100/75 mmHg, respiratory rate of 25 breaths/min, pulse rate of 63 beats/min, and temperature of 98-degree Fahrenheit. On general physical examination, he had a depressed nasal bridge, cleft lip, high-arched palate, pectus excavatum, and disproportionately long hands (Figure 1). Systemic examination was unremarkable. The patient consented to the disclosure of his identity in the form of images and the journal has received the signed consent form.

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FIGURE 1: Patient characteristics

Patient with depressed nasal bridge, cleft lip and high-arched palate (A), pectus excavatum (B), and disproportionately long hands (C).

Past medical history

He had no family history of ischemic heart disease or sudden cardiac death. He was a non-smoker and denied having a similar pain in the past or use of any drugs.

Differential diagnosis

Based on his symptoms, acute coronary syndrome, acute myocarditis, and aortic dissection were included in his differential diagnosis.

Investigations

Laboratory investigations showed mild leukocytosis, along with elevated cardiac enzyme and homocysteine levels (Table 1).

Parameters	Result	Normal Range
Complete Blood Count (CBC)		
Hemoglobin (Hb)	14 g/dL	12.0-15.0
Total Leukocyte Count (TLC)	$14 \times 10^9 /L$	4.0-10.0
Platelets (Plt)	$278 \times 10^9 /L$	150-400
Cardiac Enzymes		
Cardiac Troponin I	0.5	0-0.04 ng/mL
Creatine Kinase-MB (CK-MB)	232	5-25 IU/L
Creatine Phosphokinase (CPK)	2365	55-170 IU/L
Lactate Dehydrogenase (LDH)	557	140-280 IU/L
Homocysteine Levels	85	5-15 μ mol/L
Liver Function Tests (LFTs)		
Albumin	4.5	3.4-5.4 g/dL
Bilirubin	0.6	0.1-1.2 mg/dL
Aspartate Aminotransferase (AST)	212	5-40 IU/L
Prothrombin Time (PT)	12	11-13.5 s
Activated Partial Thromboplastin Time (aPTT)	-	21-35 s
Renal Function Tests (RFTs)		
Urea	12	5-20 mg/dL
Creatinine	0.9	0.7-1.3 mg/dL
Lipid Profile		
Cholesterol	101	<200 mg/dL
Triglycerides	84	<150 mg/dL
HDL Cholesterol	62	>60 mg/dL
LDL Cholesterol	85	<100 mg/dL
Thyroid Stimulating Hormone (TSH)	3.5	0.4-4.5 mU/L
Protein C	Normal	-
Protein S	Normal	-
ANA	Negative	-

TABLE 1: Laboratory investigations

ANA, antinuclear antibody; HDL; high-density lipoprotein; LDL, high-density lipoprotein

Polymerase chain reaction (PCR) for prothrombin gene mutation was negative. Cytogenetic analysis of the bone marrow showed no growth.

An electrocardiogram (ECG) showed ST elevation in limb leads II, III, and augmented vector foot (aVF) (Figure 2).

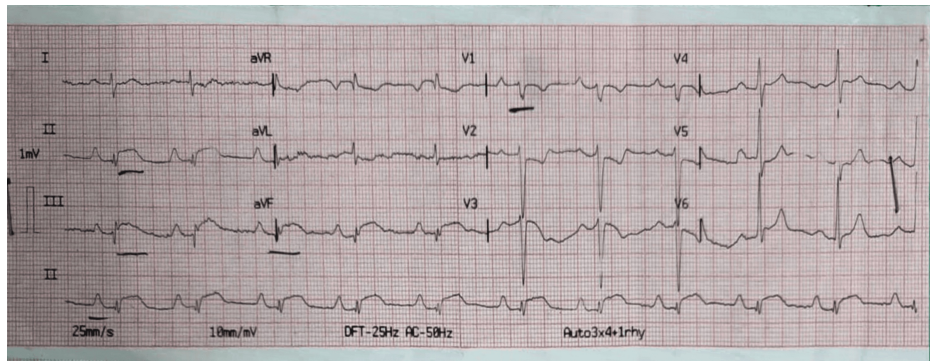


FIGURE 2: Electrocardiogram

Prominent ST elevation in limb leads II, III, and augmented vector foot (aVF).

Echocardiography showed an ejection fraction (EF) of 45-50%, with infero-posterior wall hypokinesia and jerk septal motion. Mid-to-distal right ventricular (RV) free wall hypokinesia with tricuspid annular plane systolic excursion (TAPSE) of 19 mm was observed (Figure 3). The right-sided chamber was prominent.

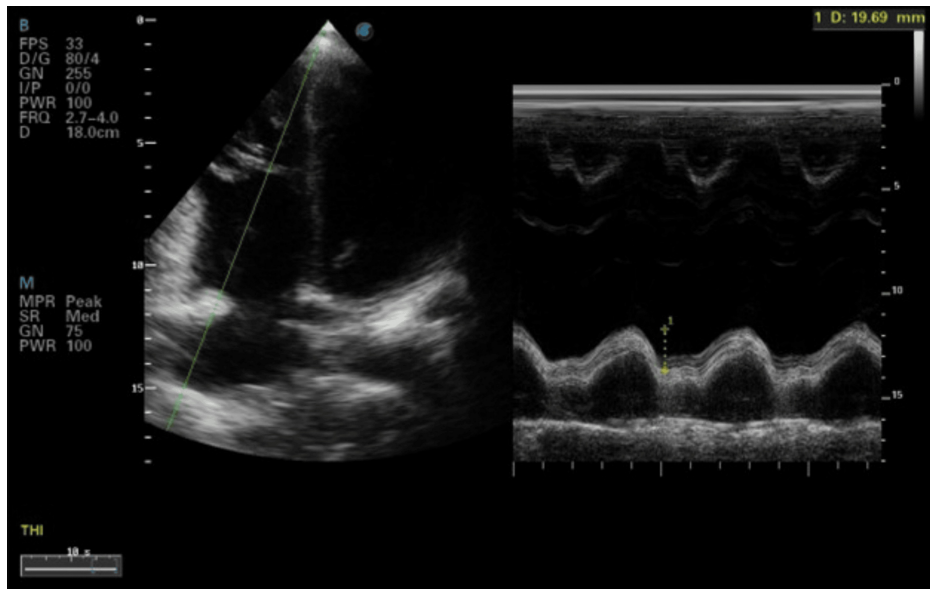


FIGURE 3: Echocardiography picture showing preserved RV systolic function with TAPSE of 19 mm

RV, right ventricular; TAPSE, tricuspid annular plane systolic excursion

The patient was diagnosed with hyperhomocysteinemia (HHCY), which caused inferior wall MI. He was treated with thrombolysis using streptokinase. Later, angiography revealed a recanalized right coronary artery (RCA) with good thrombolysis in myocardial infarction (TIMI) III flow (Figure 4).



FIGURE 4: Coronary angiography

Recanalized right coronary artery (RCA)

The patient was discharged on the fifth day with dual antiplatelet (aspirin 75 mg and clopidogrel 75 mg), anticoagulant (rivaroxaban 10 mg), beta-blocker (bisoprolol 2.5 mg), glyceryl trinitrate (2.6 mg), statin (rosuvastatin 20 mg), angiotensin-converting enzyme (ACE) inhibitor (enalapril 5 mg), folic acid, and multivitamins, with instructions to follow-up with a cardiologist within two weeks.

Discussion

Homocysteine is a sulfur-containing amino acid whose normal concentration ranges from 5 to 15 $\mu\text{mol/L}$ in the blood. However, when the levels exceed 15 $\mu\text{mol/L}$, it becomes HHCY [7]. HHCY is mainly caused by the dysfunction of enzymes and cofactors related to the biosynthesis of homocysteine. Other causes include vitamin B12, B6, and folate deficiency. Conditions like chronic renal insufficiency, hepatic dysfunction, Systemic Lupus Erythematosus, cancers, Marfan syndrome, hypothyroidism, and lifestyle factors, including smoking and alcohol abuse, can also lead to HHCY [8-9]. Traditional risk factors for MI include smoking, diabetes mellitus, hypertension, and hypercholesterolemia. Certain non-traditional risk factors are also known for MI in young adults, including HIV, systemic lupus erythematosus, obstructive sleep apnea, familial hypercholesterolemia, homocystinuria, antiphospholipid syndrome, and fibromuscular dysplasia [10].

Over the past few decades, extensive research has been conducted to elucidate the pathogenesis of MI associated with HHCY. Several studies have demonstrated that HHCY is a risk factor for cardiovascular diseases independent of classic risk factors such as smoking, hypercholesterolemia, diabetes mellitus, and hypertension [1]. The “arteriosclerosis” theory, previously expressed by McCully in 1969, supports the pathophysiological basis of association between HHCY and MI [11]. According to a meta-analysis conducted in 2008, every 5 $\mu\text{mol/L}$ increase in homocysteine levels increased the risk of coronary heart disease by approximately 20% [12]. Similarly, another meta-analysis carried out in 2022 indicated that a reduction in homocysteine levels by 3 $\mu\text{mol/L}$ reduced the risk of ischemic heart disease, stroke, and deep venous thrombosis occurrences by 16 %, 24%, and 25%, respectively [13].

In patients with acute coronary syndrome, HHCY is linked to thrombin generation and coagulation system activation. HHCY accelerates atherosclerosis by increasing vascular wall absorption of low-density lipoprotein (LDL) cholesterol and stimulating the growth of vascular smooth muscle [4]. HHCY may promote blood clot formation by increasing platelet aggregation and activation, leading to the formation of thrombi

that block blood flow to the heart, causing MI. Elevated levels of homocysteine have also been shown to impair the production of nitric oxide, a molecule that helps maintain the normal functioning of the endothelium. This can lead to endothelial dysfunction, which is a precursor to the development of atherosclerosis and, ultimately, MI [14]. Thus, elevated total plasma homocysteine levels can cause vascular occlusion through thromboembolic events or endothelial dysfunction.

Treatment of HHCY depends on the underlying cause and severity of the condition. In cases where HHCY is caused by a deficiency in vitamin B12, folic acid, or vitamin B6, supplementation with these vitamins can effectively reduce homocysteine levels [15]. Lifestyle modifications, such as regular exercise, smoking cessation, and a healthy diet low in fat and cholesterol, can also help reduce the risk of cardiovascular disease associated with HHCY [16]. For patients with a history of MI, aggressive management of risk factors such as hypertension, diabetes, and dyslipidemia are crucial. This may involve the use of medications, such as ACE inhibitors or beta-blockers, to control blood pressure, cholesterol, and other risk factors [17]. Overall, the management of HHCY requires a multifaceted approach that addresses both the underlying cause of the condition and the associated risk factors for cardiovascular disease.

Follow-up

The patient attended the cardiology outpatient department after two weeks, and his symptoms have been under control since then. He is taking his medications regularly and is doing well.

Learning objectives

1. This case report helps to recognize HHCY as an independent risk factor for MI, especially in young patients who do not have traditional risk factors.
2. It also emphasizes the need to apply appropriate diagnostic and management strategies for MI caused by HHCY.
3. It focuses on how to timely integrate a multidisciplinary approach for managing patients with marfanoid phenotypes and elevated homocysteine levels to prevent cardiovascular complications.

Conclusions

This case emphasizes the importance of recognizing HHCY as a potential risk factor for MI, especially in young individuals. Early diagnosis and management of HHCY are crucial to prevent cardiovascular complications. Aggressive management of the risk factors for cardiovascular diseases is important for preventing further events.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Amna Akbar, Mamoon Qadir, Sarosh Khan Jadoon

Acquisition, analysis, or interpretation of data: Amna Akbar, Mamoon Qadir, Sabina Aslam, Bushra lail Shah

Drafting of the manuscript: Amna Akbar, Mamoon Qadir

Critical review of the manuscript for important intellectual content: Amna Akbar, Mamoon Qadir, Sabina Aslam, Bushra lail Shah, Sarosh Khan Jadoon

Supervision: Mamoon Qadir

Disclosures

Human subjects: Consent for treatment and open access publication was obtained or waived by all participants in this study. Ethical Committee of Federal Govt Polyclinic Hospital Islamabad issued approval 9866. After evaluating the case report, permission was granted to proceed with it. However, if reports regarding any risk to the study subject are received, the committee reserves the right to discontinue the case report. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no

other relationships or activities that could appear to have influenced the submitted work.

References

1. Muzaffar R, Khan MA, Mushtaq MH, et al.: Hyperhomocysteinemia as an independent risk factor for coronary heart disease: Comparison with conventional risk factors. *Braz J Biol*. 2021, 83:e249104. [10.1590/1519-6984.249104](https://doi.org/10.1590/1519-6984.249104)
2. Varlamos C, Pappas C, Kiouri E, et al.: Hyperhomocysteinemia as the only risk factor in a young man presenting with ST-elevation myocardial infarction. *J Cardiol Cases*. 2020, 31:112-114. [10.1016/j.jccase.2020.10.004](https://doi.org/10.1016/j.jccase.2020.10.004)
3. Ren MY, Zhang CS, Zhang XJ, et al.: Acute myocardial infarction in a young man with hyperhomocysteinemia and pulmonary tuberculosis. *Intern Med*. 2016, 55:153-9. [10.2169/internalmedicine.55.4565](https://doi.org/10.2169/internalmedicine.55.4565)
4. Shamkani WA, Jafar NS, Narayanan SR, et al.: Acute myocardial infarction in a young lady due to vitamin B12 deficiency-induced hyperhomocysteinemia. *Heart Views*. 2015, 16:25-9. [10.4103/1995-705X.152998](https://doi.org/10.4103/1995-705X.152998)
5. Das D, Tutan D, Pramanik S: Hyperhomocysteinemia presenting as exclusive small vessel coronary artery disease (CAD) in a young. *J Family Med Prim Care*. 2022, 11:3298-301. [10.4103/jfmpc.jfmpc_1539_21](https://doi.org/10.4103/jfmpc.jfmpc_1539_21)
6. Dworakowska D, Kazimierska E, Weyer-Hepka J, et al.: Hyperhomocysteinemia complicated by myocardial infarction and portal vein thrombosis--case report. *Pol Arch Med Wewn*. 2006, 115:234-7.
7. Veeranki S, Gandhapudi SK, Tyagi SC, et al.: Interactions of hyperhomocysteinemia and T cell immunity in causation of hypertension. *Can J Physiol Pharmacol*. 2017, 95:239-46. [10.1139/cjpp-2015-0568](https://doi.org/10.1139/cjpp-2015-0568)
8. Son P, Lewis L: Hyperhomocysteinemia. *StatPearls [Internet]*. StatPearls Publishing, Treasure Island, FL; 2024.
9. Zhou S, Zhang Z, Xu G: Notable epigenetic role of hyperhomocysteinemia in atherogenesis. *Lipids Health Dis*. 2014, 13:134. [10.1186/1476-511X-13-134](https://doi.org/10.1186/1476-511X-13-134)
10. Krittanawong C, Khawaja M, Tamis-Holland JE, et al.: Acute myocardial infarction: etiologies and mimickers in young patients. *J Am Heart Assoc*. 2023, 12:e029971. [10.1161/JAHA.123.029971](https://doi.org/10.1161/JAHA.123.029971)
11. McCully KS: Vascular pathology of homocysteinemia: implications for the pathogenesis of arteriosclerosis. *Am J Pathol*. 1969, 56:111-28.
12. Humphrey LL, Fu R, Rogers K, Freeman M, Helfand M: Homocysteine level and coronary heart disease incidence: a systematic review and meta-analysis. *Mayo Clin Proc*. 2008, 83:1203-12. [10.4065/83.11.1203](https://doi.org/10.4065/83.11.1203)
13. Wald DS, Law M, Morris JK: Homocysteine and cardiovascular disease: evidence on causality from a meta-analysis. *BMJ*. 2002, 325:1202. [10.1136/bmj.325.7374.1202](https://doi.org/10.1136/bmj.325.7374.1202)
14. Coppola A, Davi G, De Stefano V, et al.: Homocysteine, coagulation, platelet function, and thrombosis. *Semin Thromb Hemost*. 2000, 26:243-54. [10.1055/s-2000-8469](https://doi.org/10.1055/s-2000-8469)
15. Maron BA, Loscalzo J: The treatment of hyperhomocysteinemia. *Annu Rev Med*. 2009, 60:39-54. [10.1146/annurev.med.60.041807.123308](https://doi.org/10.1146/annurev.med.60.041807.123308)
16. Rippe JM: Lifestyle strategies for risk factor reduction, prevention, and treatment of cardiovascular disease. *Am J Lifestyle Med*. 2019, 13:204-12. [10.1177/1559827618812395](https://doi.org/10.1177/1559827618812395)
17. Maxwell S, Waring WS: Drugs used in secondary prevention after myocardial infarction: case presentation. *Br J Clin Pharmacol*. 2000, 50:405-17. [10.1046/j.1365-2125.2000.00287.x](https://doi.org/10.1046/j.1365-2125.2000.00287.x)