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A 34-Year-Old Man With Exercise-Induced ST-Segment Elevation Myocardial Infarction 36 Hours After Descent From High Altitude: A Case Report

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABCDEF 1,2 **Zhiling He**
CDEF 2 **Luxun Tang**
B 2 **Jingtang Hu**
B 3 **Jiajie Liao**
B 2 **Peng Wang**
ABCDEFG 2 **Shuang Li**

1 Department of Clinical Medicine, North Sichuan Medical College, Nanchong, Sichuan, PR China
2 Department of Cardiology, The General Hospital of Western Theater Command, Chengdu, Sichuan, PR China
3 Department of Clinical Medicine, Chengdu Medical College, Chengdu, Sichuan, PR China

Corresponding Author: Shuang Li, e-mail: louis_1014@163.com
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Patient: Male, 34-year-old
Final Diagnosis: Acute anterior myocardial infarction • coronary atherosclerotic heart disease • hyperlipidaemia • Killip class II
Symptoms: Sudden chest pain presenting as a crushing sensation in the precordial region, radiating to both scapular areas • profuse sweating • nausea • vomiting of gastric contents
Clinical Procedure: —
Specialty: Cardiology
Objective: Unusual clinical course
Background: Prolonged exposure to high altitude can increase the risk of coronary artery disease, acute coronary syndrome, and other ischemic cardiovascular events. This report describes the case of a 34-year-old man with exercise-induced ST-segment elevation myocardial infarction (STEMI) 36 hours after descent from high altitude.
Case Report: A previously healthy 34-year-old man presented with 9 hours of crushing substernal chest pain. Electrocardiogram (ECG) demonstrated anteroseptal ST-segment elevation in leads V1-V3 with reciprocal changes in inferior leads. Emergency coronary angiography (CAG) revealed total occlusion with a high thrombus burden of the proximal left anterior descending (LAD) artery with TIMI 0 flow, accompanied by diffuse non-culprit coronary plaques (30-40% stenosis). Primary percutaneous coronary intervention (PCI) with drug-eluting stent implantation restored TIMI 3 flow. The event occurred 36 hours after descent from 4200 m altitude, immediately following intensive exercise, suggesting a temporal association between early de-acclimatization and acute coronary events.
Conclusions: Multiple factors, including dyslipidemia, residual hemorheological changes after high-altitude exposure, and vigorous exercise, may be associated with the occurrence of acute myocardial infarction (AMI) during high-altitude de-acclimatization. This report highlights the importance of recognizing that patients with a history of living at high altitude can be at increased risk of acute coronary syndromes, particularly ST-segment elevation myocardial infarction, when undertaking exercise.
Keywords: Acute Myocardial Infarction • High Altitude • Coronary Angiography • Exercise • Case Report
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Introduction

High-altitude environments, typically defined as elevations above 2500 meters, expose individuals to hypobaric hypoxia, low temperatures, and increased sympathetic activation. These conditions induce physiological adaptations such as erythrocytosis, increased blood viscosity, endothelial dysfunction, and alterations in coagulation pathways [1]. Previous studies have demonstrated that individuals residing at high altitude have a higher incidence of acute coronary syndrome (ACS), particularly among young and middle-aged populations [2]. While the cardiovascular effects of acute ascent and chronic exposure have been widely studied, the de-acclimatization phase, defined as the period following descent to low altitude, remains less well understood. Emerging evidence suggests that residual hematological and endothelial changes may persist during early de-acclimatization, potentially predisposing individuals to thrombotic events and acute coronary events [3]. However, reports of acute myocardial infarction occurring during this phase are limited. This report describes a 34-year-old man who developed exercise-induced STEMI 36 hours after descent from high altitude.

Case Report

A 34-year-old man presented with persistent chest pain for 9 hours. He had worked continuously at an altitude of approximately 4200 m for 4 months and then traveled by train (without stopping) to return to the plains (≈ 520 m elevation). During descent and travel, he experienced fatigue. Two days after arrival, he participated in a marathon. Approximately 36 hours after descent, and 2 minutes after completing the race, he developed crushing retrosternal chest pain radiating to both scapulae, accompanied by diaphoresis and nausea. He denied smoking, alcohol abuse, illicit drug use, or stimulant exposure. There was no known family history of premature coronary artery disease, and he had no prior diagnoses of hypertension, diabetes mellitus, or hyperuricemia. At the first medical contact, ECG (Figure 1A) demonstrated sinus rhythm (71 bpm), ST-segment depression in II, III, aVF and QS patterns in V1-V2. Cardiac troponin I (cTnI) was 0.93 ng/mL (reference range <0.10 ng/mL), consistent with acute myocardial infarction. He received dual antiplatelet loading (aspirin 300 mg+clopidogrel 300 mg) and was transferred to the General Hospital of the Western Theater Command for further care.

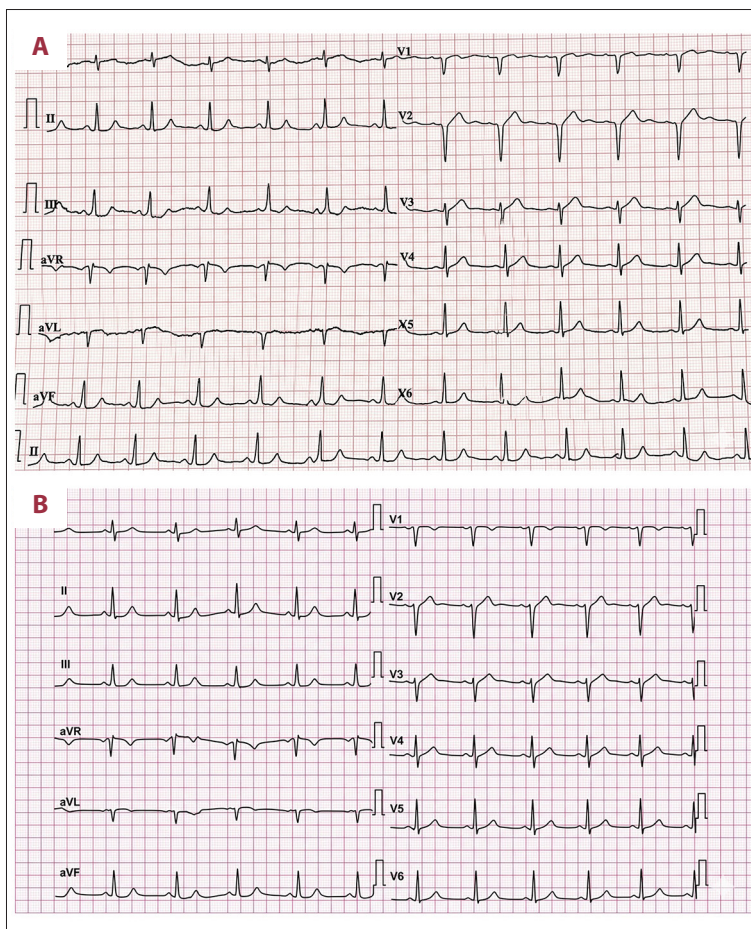
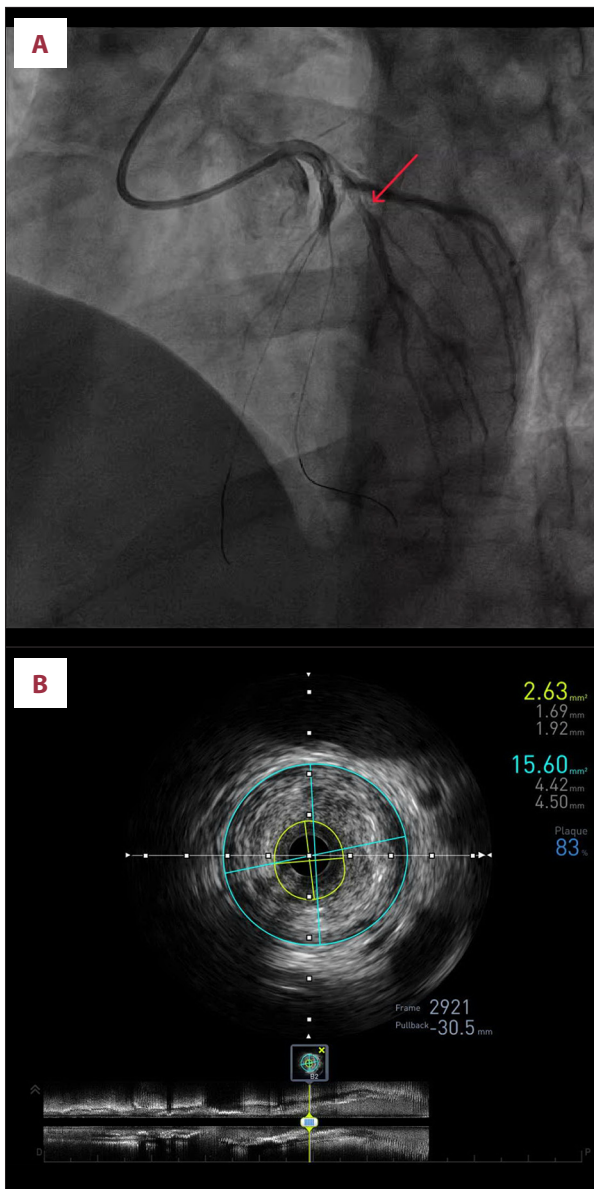


Figure 1. Electrocardiogram (ECG) Examinations. (A) ECG from Chongzhou People's Hospital showed that 1. Sinus rhythm with a heart rate of 71 beats per minute. 2. ST-segment depression of 0.05-0.1mV in leads II, III, aVF. 3. QS pattern in leads V1 and V2. (B) ECG from the General Hospital of Western Theater showed that 1. Sinus rhythm with a heart rate of 63 beats per minute. 2. ST-segment elevation with pathological Q waves in leads V1-V3.



Upon admission, the patient's cTnI was 0.34 ng/mL (reference range <0.10 ng/mL). Repeat ECG (Figure 1B) revealed sinus rhythm with ST-segment elevation and pathological Q waves in V1-V3, consistent with STEMI. Emergency CAG (Figure 2A) showed total occlusion of the proximal LAD with TIMI 0 flow, while the left main, circumflex, and right coronary arteries exhibited no significant obstructive stenosis (TIMI 3). After guidewire crossing and balloon pre-dilation, diffuse plaques (30-40% stenosis) were observed in the LAD. Intravascular ultrasound (IVUS) demonstrated (Figure 2B) diffuse calcified plaques with a large thrombus burden (minimum lumen area 2.63 mm²; plaque burden 83%), findings suggestive of plaque disruption with superimposed thrombus, although definitive evidence of plaque rupture was not established. A drug-eluting stent (3.5×22 mm) was implanted in the LAD, achieving adequate expansion and restoration of



Figure 2. Coronary angiography (CAG) and intravascular ultrasound (IVUS) Examinations. (A) Emergency CAG at 20: 45 on October 1, 2024. Proximal occlusion of the left anterior descending artery (LAD) with thrombolysis in myocardial infarction (TIMI) 0 flow, and diffuse plaques throughout the LAD (red arrow). (B) IVUS revealed diffuse calcified plaque with substantial thrombus burden, minimum lumen area 2.63 mm² plaque burden 83%. (C) A follow-up CAG was performed at 9: 02 on October 8, 2024. No stenosis in the left main coronary artery, 30% stenosis with plaque in the mid-to-distal segments of the LAD with TIMI 3 flow (green arrow).

TIMI 3 flow. Postoperatively, a tirofiban 12.5 mg infusion was administered due to heavy thrombus burden, followed by transfer to the coronary care unit (CCU) for further monitoring and treatment. Long-term therapy included aspirin (100 mg once daily), clopidogrel (75 mg once daily), rosuvastatin (10 mg once daily at night), and perindopril (4 mg once daily). Follow-up CAG 1 week later (Figure 2C) showed mild residual plaques (30-50% stenosis) with preserved TIMI 3 flow. Echocardiography demonstrated normal left ventricular function (LVEF 62%). The patient remained asymptomatic after reperfusion, with no chest pain recurrence during the 2-month follow-up. The chronological sequence of clinical events is summarized in Table 1.

Discussion

This case report describes a young patient who developed STEMI shortly after intense exercise during early high-altitude de-acclimatization. The findings suggest a potential interaction between environmental, physiological, and behavioral factors.

Table 1. Timeline of clinical events.

Time point	Clinical event
2024-09-24 to 2024-09-29	The patient returned from a high-altitude region to the plains by train, completing the de-acclimatization period
2024-10-01, 07: 30 AM	The patient participated in a marathon race
2024-10-01, ~09: 30 AM	After approximately 2 hours of running (total distance ~21 km), the patient developed sudden-onset compressive chest pain, radiating to both scapular regions, accompanied by nausea, vomiting, and profuse diaphoresis
2024-10-01, 09: 30-10: 00 AM	Chest pain persisted despite 30 minutes of rest, without symptom relief
2024-10-01, 10: 00 AM	Emergency medical services were contacted, and the patient was transported to Chongzhou People's Hospital
2024-10-01, daytime	Initial evaluation, including ECG and cardiac biomarkers, supported a diagnosis of AMI. Dual antiplatelet therapy was initiated
2024-10-01, 20: 22 PM	The patient was transferred to General Hospital of Western Theater Command for definitive management
2024-10-01, 20: 45 PM	PCI was performed
2024-10-01, 21: 55 PM	PCI was completed after 1 hour and 10 minutes, followed by transfer to the CCU for further monitoring and treatment
1 week later	Follow-up CAG showed patent stent and mild residual stenosis
2-month follow-up	Patient remained asymptomatic without recurrence of chest pain

Previous studies have reported that the risk factors for STEMI in young individuals in high-altitude areas include diabetes, smoking history, total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), direct bilirubin, and high-sensitivity C-reactive protein [4]. In this patient, elevated LDL-C and TC were documented. Dietary inquiry revealed that during high-altitude residence, his diet was relatively high in salt and fat, whereas no structured dietary intervention had yet been implemented after descent. The patient was a young man with only dyslipidemia and no other risk factors such as smoking, hypertension, diabetes, obesity, or family history. However, CAG and IVUS indicated a heavy plaque burden, and the onset of this STEMI was acute and severe. To further explore the causes of the disease, this patient's case is discussed in depth.

Permanent high-altitude residence induces systemic pathophysiological adaptations. Upon returning to lower elevations, these changes may not immediately normalize, potentially leading to transient increases in blood viscosity and microcirculatory dysfunction [5,6]. Although this patient did not meet the diagnostic criteria for high-altitude polycythemia, his hemoglobin level was relatively elevated, which may have contributed to increased shear stress and impaired microcirculation. Studies have demonstrated persistent cardiac impairment and autonomic imbalance, evidenced by sympathetic suppression and parasympathetic dominance-during early de-acclimatization [7,8]. Elevated myocardial enzymes further suggest delayed recovery of myocardial

tissue [3]. This patient, with prior acute high-altitude reaction symptoms, engaged in intense exercise while still in this vulnerable state of incomplete cardiac and autonomic recovery. This mismatch between cardiac capacity and metabolic demand likely contributed to his event. Therefore, individuals recently descending from high altitudes should avoid strenuous exercise, and those with residual symptoms require stratified management based on cardiac function and hemodynamic status.

The temporal proximity between marathon running and symptom onset suggests that vigorous exercise have acted as an acute trigger. However, exercise-triggered acute thrombotic events are rare in otherwise healthy individuals. This is because during intense physical activity, coagulation, anticoagulation, and fibrinolysis are simultaneously activated, reaching a balanced state that reduces the risk of thrombotic events. While intense exercise generally activates both procoagulant and fibrinolytic pathways, in susceptible individuals it can transiently tilt the balance toward thrombosis. Therefore, for individuals with latent coagulation dysfunction, vigorous exercise can serve as a trigger for thrombotic events [9]. Studies have also shown that acute sessions of high-intensity interval rowing increase the production of plasma thrombin after exercise, but these differences are resolved within 16 to 24 hours after exercise [10]. Our patient was treated with low-molecular-weight heparin for anticoagulation after the onset of the disease, although direct coagulation markers could not

be reliably assessed due to early anticoagulant therapy. CAG and IVUS revealed a high thrombus burden against a background of non-occlusive plaques, supporting the hypothesis that acute thrombus formation played a role in this event. However, as comprehensive coagulation testing was not performed prior to anticoagulant therapy, a definitive causal relationship cannot be established.

Previous reports have described increased cardiovascular risk at high altitude, but cases occurring during de-acclimatization remain rare. A recent case report described multiple thrombotic events, including cerebral and pulmonary thrombosis, in a young individual exposed to high altitude, suggesting increased thrombotic susceptibility under hypoxic conditions [11]. Another study reported acute coronary syndrome in young individuals following high-altitude exposure [4]. Compared with these reports, the present case highlights the potential role of early descent combined with intense exercise as a triggering factor.

This patient was a young man who resides permanently in a high-altitude area, and the high-altitude environment may be a significant factor contributing to the AMI. Residents of high-altitude regions experience compensatory increases in red blood cells (RBCs) and hemoglobin (HB), leading to elevated blood viscosity. In China, HB concentrations ≥ 210 g/L in males are indicative of high-altitude polycythemia. Although this patient's HB level was elevated but did not meet diagnostic criteria for polycythemia, further investigation is needed to determine whether the increased RBCs played a role in the development of this AMI. A retrospective cohort study suggested that subjects with HB levels ≥ 15.0 g/dL in females and ≥ 16.5 g/dL in males were associated with an increased risk of major adverse cardiovascular events (a composite of cardiovascular mortality, new-onset myocardial infarction, and stroke) [12]. Similarly, studies have shown that higher HB levels in anemic STEMI patients are associated with a reduced risk of 1-year mortality, while higher HB levels in patients with polycythemia vera (PV) are associated with an increased risk of 1-year mortality [13]. PV, a category of myeloid proliferative neoplasms, is characterized by increased RBCs and HB, with manifestations of bleeding and thrombosis. Studies have indicated that asymptomatic PV patients exhibit coronary microvascular dysfunction even in the absence of clinical indicators suggesting coronary atherosclerotic heart disease [14]. For individuals residing in high-altitude areas, especially those with high-risk factors, regular blood tests to assess indicators such as HB, RBCs, and hematocrit are recommended to potentially prevent and control the occurrence of AMI.

The laboratory test results for our patient indicated a transient elevation in serum uric acid. Hyperuricemia has become a common metabolic disorder in China, following diabetes, and is recognized as one of the risk factors for cardiovascular

diseases. Elevated blood uric acid levels can cause oxidative stress and endothelial dysfunction, while the body's high-inflammatory state can influence the occurrence and progression of atherosclerotic cardiovascular diseases [15]. Uric acid is associated with a prothrombotic state through 2 mechanisms: first, reactive oxygen species (ROS)-mediated vascular endothelial inflammation, smooth muscle cell proliferation, and interference with the coagulation system; second, vascular endothelial injury-induced oxidative stress, reduced nitric oxide bioavailability, and endothelial dysfunction (ED), which subsequently promote arterial and venous thrombosis [16]. Additionally, hyperuricemia can reduce myocardial microcirculation perfusion and promote plaque progression, thereby exacerbating myocardial infarction. A study combining serum uric acid levels with IVUS revealed that hyperuricemia serves as an independent predictor of plaque lipid content, suggesting an association between serum uric acid levels and plaque stability [17]. Studies have also reported that serum uric acid levels are a strong and independent predictor of poor coronary blood flow in patients with STEMI [18]. Blood uric acid is a readily available biomarker that can assist in the risk stratification of AMI patients in the near and short term [19]. Studies have suggested that blood uric acid levels are associated with the severity and number of coronary artery lesions in young and middle-aged patients with acute STEMI at high altitudes, and patients with hyperuricemia have a higher risk of recent heart failure [20]. As mentioned above, long-term hyperuricemia can lead to a series of changes affecting the cardiovascular system, while the hypoperfusion and hypoxia caused by AMI can further elevate blood uric acid levels. However, as a non-specific metabolic indicator, blood uric acid is susceptible to factors such as exercise, diet, and medication. Our patient had no history of hyperuricemia, no significant abnormalities in uric acid levels were observed in patients during both initial and follow-up examinations, and elevated uric acid was detected only during the acute phase. Therefore, it remains unclear whether this represented a chronic metabolic risk factor or an acute stress response to acute coronary events. Further consideration is needed to determine whether blood uric acid levels can be used to assess the risk and severity of coronary artery lesions in such patients from high-altitude areas who were previously healthy and had no related risk factors.

This case report has several limitations. As a single-case observation, causal inference cannot be established. In addition, detailed longitudinal hematological and coagulation data were not available.

Conclusions

AMI can occur during early high-altitude de-acclimatization, particularly when combined with strenuous physical activity.

Clinicians should be aware of this potential risk and consider advising temporary restriction of intense exercise in individuals returning from high altitude.

Institution Where Work Was Done

The General Hospital of Western Theater Command, Chengdu, Sichuan, PR China.

Patient Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. All identifying information has been removed to protect patient privacy.

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During the preparation of this manuscript, the authors used [ChatGPT/Gemini/DeepL] to translate parts of the initial draft and polish the English language to improve readability. After using this tool, the authors comprehensively reviewed and edited the content as needed and take full responsibility for the final content of the publication.

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