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A Case of Acute Myocardial Infarction During Rapid Ascent to High Altitude

Haiying Xian[1]*, Xuewen Huang[1], Lin Ma[1], Yuanjing Ma[2], Qinghua He[1], Xiangyong Yu[1]

1.Sichuan Electric Power Hospital, Chengdu City, Sichuan 610065, China

2.North Sichuan Medical College, Nanchong City, Sichuan 637007, China

Abstract: Medical history: Male, 46 years old, driver. A long-term resident of the plains with no history of hypertension or altitude-related hypertension. High-altitude BP at 110/70-120/80 mmHg, fasting blood glucose at 6.6 mmol/L (health check indicated elevated fasting blood glucose; no further examinations conducted), previously undiagnosed diabetes, no thyroid-related disorders. Pre-altitude health assessment: BMI: 27.27, uric acid: 446 µmol/L (2023), 457 µmol/L (2024). No smoking history; occasional alcohol consumption in small amounts (approximately 50-150 g each time, 1-2 times per month). Symptoms developed about 12 h after driving from the plains to an average altitude of 2,580 m.Symptoms and signs: Sudden onset of squeezing pain behind the sternum after breakfast. Accompanied by palpitations, fatigue, numbness in the fingertips, and soreness in the jaw, the patient is observed sweating profusely with a distressed expression. BP 141/89 mmHg, HR 110 beats/min, SpO2 91% (not on supplemental oxygen), R 28 breaths/min. Consciousness is clear, no cough or pink frothy sputum, no significant respiratory distress, and no obvious abnormalities in urination or defecation. Physical examination: Bilateral pupils are equal and round, no neck stiffness, no dry or wet rales heard in both lungs, normal muscle strength and tone in the limbs, physiological reflexes present, and pathological reflexes not elicited. The patient developed upper back pain 0.5 h after symptom onset.Diagnostic methods: Bedside electrocardiogram shows ST segment elevation in leads V1-V5 and frequent premature ventricular contractions (PVCs). A complete myocardial enzyme profile, prothrombin time (PT), D-dimer, C-reactive protein (CRP), biochemical tests, chest CT, and other relevant examinations were conducted. A repeat electrocardiogram was performed.Treatment methods: Cardiac monitoring and vital sign observation were administered, and venous access was established. Due to limitations in field medical care, percutaneous coronary intervention (PCI) could not be performed. According to the “Guideline for rational medication of ST-segment elevation myocardial infarction in primary care,”[1] and considering the unique circumstances of high altitude, the following treatment plan was implemented on-site: (1) Clopidogrel loading dose of 300 mg and aspirin 300 mg (chewed for rapid absorption) for antithrombotic therapy; (2) 2 million units of Urokinase dissolved in 100 ml of normal saline (administered intravenously over 45 min) for thrombolysis (1.5 h after symptom onset). During thrombolysis, cardiac monitoring showed BP 110-140/60-86 mmHg, HR 30-80 beats/min, and R 24 breaths/min. (3) Emergency oxygen therapy. The oxygen flow rate is set at 4 L/min via nasal cannula for continuous delivery and results in oxygen saturation levels of 95-97%.Clinical outcome: After thrombolysis, the chest pain decreased, and a repeat electrocardiogram still indicated the presence of PVCs, with ST-segment elevation reduced compared to before. Following the administration of morphine for pain relief, the patient was transferred to the Chest Pain Center at West China Hospital, Sichuan University, approximately 4 h after symptom onset. During transport, the patient remained strictly in bed and received continuous oxygen at 2-4 L/min. Throughout the transfer, the chest pain intensity gradually reduced. Cardiac monitoring during transport showed BP 120/60-140/89 mmHg, heart rate 50-90 beats/min, respiration rate 21-24 breaths/min, and SpO2 91%-97%; there were no instances of urination or defecation. The patient was admitted to West China Hospital, Sichuan University, 16 h after symptom onset, and received PCI. After one day of post-operative observation, the patient was discharged and returned home without any discomfort.

Keywords: Rapid Ascent to High Altitude; Acute Myocardial Infarction; Clinical Outcome

Clinically, acute coronary syndrome (ACS) is not uncommon and is often observed in older patients with comorbidities such as hypertension, diabetes, coronary heart disease, and dyslipidemia. Most myocardial infarctions (MI) are caused by coronary artery atherosclerosis. However, non-atherosclerotic pathology can occasionally affect the coronary arteries and lead to acute myocardial infarction. Individuals from low altitudes who rapidly ascend to high-altitude areas or move from lower-altitude plateaus to higher regions may experience exacerbated hypoxia due to low oxygen levels, low atmospheric pressure, cold temperatures, respiratory infections, or excessive physical exertion, resulting in acute altitude sickness.[3] Among these, acute and severe altitude sickness is the leading cause of non-traumatic deaths under conditions of acute exposure to altitude.[4] Acute myocardial infarction (AMI) refers to a series of physiological and pathological changes that occur due to atherosclerosis and narrowing of the coronary arteries, leading to myocardial ischemia and hypoxia, ultimately resulting in myocardial necrosis.[5] Risk factors and past medical history related to coronary heart disease aid in the diagnosis, including a history of coronary heart disease (angina, MI, history of CABG or PCI treatment), hypertension, diabetes, peripheral artery disease, cerebrovascular disease (ischemic stroke, intracranial hemorrhage, or subarachnoid hemorrhage), dyslipidemia, and smoking.[6] According to the Interpretation of Report on Cardiovascular Health and Diseases in China 2021[7], the overall mortality rate for AMI in China is on the rise, while early recurrence rates of MI after discharge are relatively high among AMI patients, which poses health risks and increases economic burdens. Upon entering high altitudes, hypoxia occurs, and anaerobic metabolism increases, thus leading to the production of large amounts of lactic acid, decreased pH, and increased H+ concentration, which reduces hemoglobin's affinity for oxygen. There is also a redistribution of blood throughout the body, an increased stress response, and elevated cardiac load (increased heart rate, higher blood pressure, pulmonary hypertension). A series of physiological changes can significantly impact individuals rapidly ascending to high altitudes[8]. Thus, rapid ascent is an important and special trigger for AMI. This study reports a case of AMI following rapid ascent to high altitude and discusses the risk factors for MI in this context to provide insights for prevention and treatment.

1 Clinical data

1.1 General information

Patient: Male, 46 years old, driver, admitted to Batang County People's Hospital due to "sudden squeezing pain behind the sternum for over 90 min after breakfast." More than 90 min prior to admission, the patient experienced sudden squeezing pain behind the sternum after breakfast. The patient reported accompanying symptoms of palpitations, fatigue, numbness in the fingertips, and soreness in the jaw, with no significant shoulder or back pain, dizziness, visual disturbances, acid reflux, or heartburn at that time. The patient was sweating profusely with a distressed expression, and an electrocardiogram showed ST-segment elevation in leads V1-V3 and ST-T abnormalities in leads II/III/AVF. Continuous oxygen was administered via nasal cannula at a flow rate of 4 L/min, resulting in SpO2 of 95-97%. After sublingual administration of Danshen dripping pills and quick-acting heart-rescue pills, these symptoms were slightly alleviated. Approximately 30+ min after taking the quick-acting heart-rescue pills, the patient reported left shoulder and back pain, with persistent chest pain but reduced pressure sensation in the precordial area. Therefore, the patient sought medical attention at Batang County People's Hospital. The emergency diagnosis was "suspected AMI." The patient had been feeling unwell since the onset of symptoms, with no abnormalities in urination or defecation. There was no change in weight. Having lived long-term in the plains, the patient drove intermittently for more than 12 h from Chengdu Plain (average altitude of 500 m) to Batang County, Sichuan Province (altitude of 2,580 m) just prior to admission. There was no history of hypertension or altitude-related hypertension, no history of infectious diseases, surgeries, major trauma, or blood transfusions. The patient denied any food or drug allergies. He has lived in Chengdu, Sichuan, for a long time, has no smoking history, and drinks alcohol occasionally. There is no family history of hereditary or familial diseases. Upon admission, physical examination revealed: BP 141/89 mmHg, HR 110 beats/min, SpO2 91% (not on supplemental oxygen), R 28 breaths/min. Consciousness was clear; bilateral pupils were equal and round; no neck stiffness was noted; no significant dry or wet rales were heard in both lungs; muscle strength and tone in the limbs were normal; physiological reflexes were present; pathological reflexes were not elicited.

2 Auxiliary examinations

(1) Electrocardiogram: Short PR interval / extensive ST-T changes / frequent PVCs (Fig. 1);

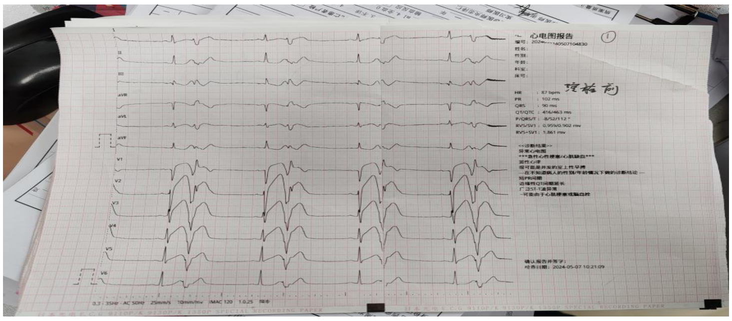


Fig. 1 Electrocardiogram

(2) Cardiac enzyme test upon admission: CK isoenzyme<2.5 U/L, troponin <0.01, myoglobin 33.70 ng/ml (Fig. 2);

序号	中文名称	结果	单位	参考值
1	肌酸激酶同工酶	<2.5	U/L	0-24
2	肌钙蛋白	<0.01		<0.5
3	肌红蛋白	33.70	ng/ml	<70

Fig. 2 Cardiac enzyme test upon admission

(3) Chest CT: Possible chronic infections in the lower lobes of both lungs, slight thickening of the pleura bilaterally, and CBC showed no significant abnormalities;

(4) HIV test (negative);

(5) Biochemical tests: Total bilirubin 20.91 μmol/L, uric acid 437.19 μmol/L, glucose 11.08 mmol/L (random), triglycerides 2.42 mmol/L, low-density lipoprotein cholesterol 4.2 mmol/L (Fig. 3);

序号	中文名称	结果		单位	参考值
1	谷丙转氨酶	37		U/L	0-41
2	谷草转氨酶	23		U/L	0-37
3	碱性磷酸酶	63.95		U/L	53-128
4	总蛋白	79.35		g/L	66-87
5	白蛋白	52.95		g/L	40-55
6	球蛋白	26.40		g/L	20-40
7	白/球比例	2.01			1.2-2.4
8	γ-谷酰转肽酶	51.11		U/L	11-61
9	总胆红素	20.91	↑	umol/L	5.1-19
10	直接胆红素	5.80		umol/L	1.7-6.8
11	谷草/谷丙	0.62	↓		1.2-2.5
12	总胆汁酸	3.45		umol/L	0-10
13	肌酐	88.11		umol/L	男:70-115;女:44-80
14	尿酸	437.19	↑	umol/L	202-416
15	尿素	6.27		mmol/l	1.7-8.3
16	葡萄糖	11.08	↑	mmol/L	3.33-6.11
17	甘油三酯	2.42	↑	mmol/L	0.00-2.30
18	总胆固醇	5.58		mmol/L	0-5.6
19	高密度脂蛋白胆固醇	0.95		mmol/L	>0.9
20	低密度脂蛋白胆固醇	4.20	↑	mmol/L	0-4.11
21	间接胆红素	15.11	↑	umol/L	3.4-12.2
22	乳酸脱氢酶	138.92		U/L	135-225
23	肌酸激酶	75.84		U/L	24-190
24	钾	4.25		mmol/L	3.50-5.30
25	钠	135.50		mmol/L	135-145
26	氯	101.20		mmol/L	99-110

Fig. 3 Biochemical tests

(6) CRP 5.3 mg/L (Fig. 4);

序号	中文名称	结果	单位	参考值
1	全程C-反应蛋白	5.30	mg/L	<10

Fig. 4 CRP

A. Coagulation tests: prothrombin time (PT) 12.1 s, international normalized ratio (INR) 1.00, activated partial thromboplastin time (APTT) 30.52 s, fibrinogen (FIB) 4.120 g/L, thrombin time (TT) 18.00 s, D-dimer 0.26 mg/L (Fig. 5);

序号	中文名称	项目	结果	单位	状态	参考值
1	血浆凝血酶原时间(PT)	PT	12.10	s		10-15
2	国际标准化比值	INR	1.00			0.80-1.50
3	活化部分凝血活酶时间	APTT	30.52	s		22.00-38.00
4	纤维蛋白原	FIB	4.120	g/L	↑	2.00-4.00
5	凝血酶时间测定(TT)	TT	18.00	s		14-21
6	D-D二聚体	D-D二聚	0.26	mg/L		0-0.5

Fig. 5 A.Coagulation tests

3 Diagnosis and differential diagnosis

3.1 Diagnosis upon admission: AMI

The diagnostic basis is as follows: The patient entered a high-altitude area from a low-altitude region 1 d prior, experiencing sudden squeezing pain behind the sternum after breakfast. This was accompanied by palpitations, fatigue, numbness in the fingertips, and soreness in the jaw. The patient also reported difficulty breathing at rest and a pressure sensation in the chest. Physical examination revealed arrhythmia. The bedside electrocardiogram showed short PR interval / extensive ST-T changes / frequent PVCs.

3.2 Differential diagnosis

(1) High altitude pulmonary edema (HAPE): The patient entered a high-altitude area from a low-altitude region 1 d prior, with no upper respiratory infection before the onset of symptoms. The patient may experience difficulty breathing at rest and a pressure sensation of chest, which are similar to the symptoms of AMI. The patient does not exhibit cough or pink frothy sputum. Physical examination did not reveal significant cyanosis of the lips or nail beds, and no notable dry or wet rales were heard in both lungs. Chest CT indicated possible chronic infections in the lower lobes of both lungs and slight thickening of the pleura bilaterally.

(2) Heart failure: Commonly associated with left heart failure, this condition often has a history and signs of hypertension, coronary heart disease, or rheumatic heart disease with mitral stenosis. Symptoms may include paroxysmal cough, often producing pink frothy sputum, widespread wet rales and wheezing in both lungs, an enlarged left heart border, increased heart rate, and X-ray findings may show cardiac enlargement and pulmonary congestion. This patient has no clear cardiovascular risk factors, no exertional dyspnea, no cough with pink frothy sputum, and auscultation revealed no wheezing. Therefore, this diagnosis is not currently considered.

4 Treatment

(1) Upon admission, the patient was immediately placed under cardiac monitoring, vital signs were assessed, and venous access was established. Strict bed rest was enforced, and oxygen was administered at a rate of 4 L/min.

(2) Pharmacological treatment:

Before thrombolysis, the patient chewed and took enteric-coated aspirin 300 mg and clopidogrel 300 mg. Urokinase thrombolysis was performed with the following medication regimen: Urokinase 2 million units dissolved in 100 ml of normal saline (administered intravenously over 45 min) for thrombolysis (1.5 h after symptom onset).

(3) Changes in symptoms and biomarkers after thrombolysis:

Following thrombolysis, the patient's chest pain decreased, and the electrocardiogram showed the presence of PVCs, with ST-segment elevation reduced (Fig. 6).

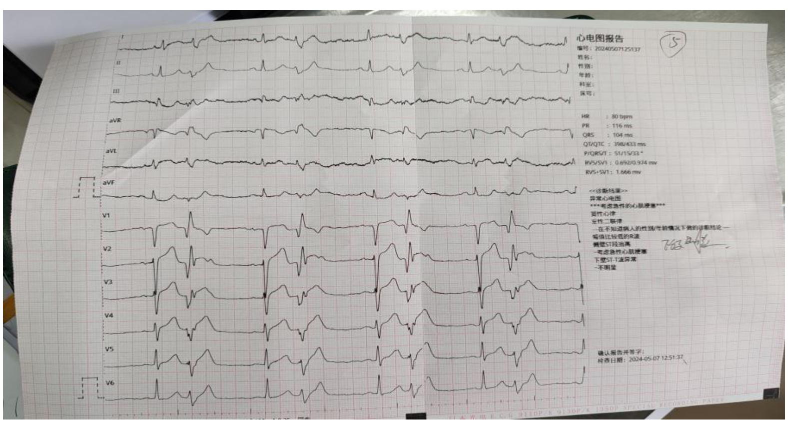
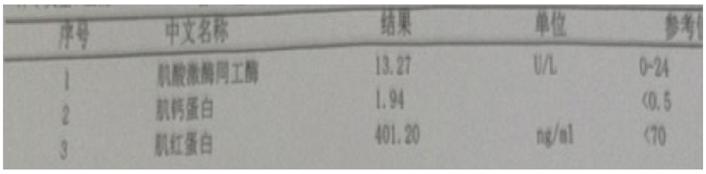


Fig. 6 Changes in symptoms and biomarkers after thrombolysis

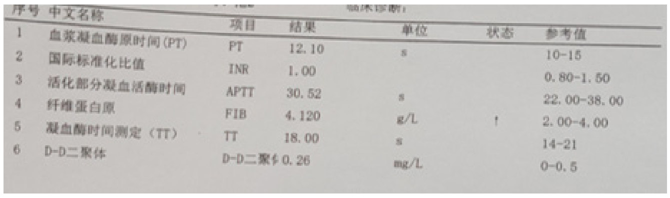
Cardiac enzyme tests (Fig. 7)



序号	中文名称	结果	单位	参考
1	肌酸激酶同工酶	13.27	U/L	0-24
2	肌钙蛋白	1.94		<0.5
3	肌红蛋白	401.20	ng/ml	<70

Fig. 7 Cardiac enzyme tests

Coagulation tests (Fig. 8):



序号	中文名称	项目	结果	单位	状态	参考值
1	血浆凝血酶原时间(PT)	PT	12.10	s		10-15
2	国际标准化比值	INR	1.00			0.80-1.50
3	活化部分凝血活酶时间	APTT	30.52	s		22.00-38.00
4	纤维蛋白原	FIB	4.120	g/L	↑	2.00-4.00
5	凝血酶时间测定（TT）	TT	18.00	s		14-21
6	D-D二聚体	D-D二聚体	0.26	mg/L		0-0.5

Fig. 8 Coagulation tests

5 Treatment outcomes, follow-up, and prognosis

After thrombolysis, the patient was transferred to West China Hospital, Sichuan University. The patient was admitted 16 h after symptom onset and underwent PCI, during which a coronary stent was placed. After one day of post-operative observation, the patient was discharged and returned home without any discomfort.

6 Discussion

As a unique environment, high altitude has its distinct climatic characteristics. As altitude increases, the partial pressure of oxygen in the air decreases, leading to a gradual decline in the partial pressure of oxygen in inhaled gases. This resulting hypoxemia can cause damage to the organ functions. Such conditions reduce the cardiopulmonary function of critically ill patients and increase the risk of circulatory and respiratory disorders.[9] Rapid ascent to high altitudes results in abrupt changes in atmospheric pressure and significant hypoxia, which can lead to intense stimulation of the sympathetic nervous system and coronary artery spasm,[10] thereby increasing the likelihood of cardiovascular-related diseases. Additionally, the low temperatures, hypoxia, and oxygen deficiency at high altitudes can cause endothelial injury, alterations in coagulation function, coagulopathy or fibrinolytic dysfunction,[11] hypoxic stress,[12] blood redistribution, and increased blood viscosity.[13, 14] Hypoxic pulmonary hypertension at high altitudes leads to increased anaerobic metabolism and lactic acid production, which raises myocardial oxygen consumption. As individuals ascend to high altitudes, the arterial partial pressure of oxygen decreases, making them more susceptible to MI due to various factors mentioned above.[15] Therefore, rapid ascent to high altitude is an important triggering factor for AMI.

Previous reports have indicated that the peak incidence of AMI occurs between 6:01 AM and 12:00 PM, with a secondary peak from 6:01 PM to 12:00 AM. The possible mechanism behind this pattern is a result of periodic activities of external environments and biological functions. The secretion of corticotropin-releasing hormone (CRH) and adrenal corticosteroid exhibits diurnal fluctuations, peaking before morning awakening, which subsequently causes corresponding fluctuations in the secretion of adrenal hormone. In the morning, levels of norepinephrine, plasma renin activity, cortisol, and platelet aggregation are all elevated. Increased hormone levels can make the epicardial vessels more sensitive to vasoconstrictive stimuli, leading to increased heart rate and elevated blood pressure. Rapid ascent to high altitudes results in abrupt changes in atmospheric pressure and significant hypoxia, causing patients to experience stress-induced hypertension, increased heart rate, and elevated blood pressure, all of which contribute to increased cardiac load. The rapid ascent also leads to blood redistribution and elevated pulmonary artery pressure, which together increase the risk of AMI. Furthermore, the prior undiagnosed glucose abnormalities may have already caused endothelial injury, thus leading to the formation of microthrombus upon entering high altitude. As the secretion of CRH and adrenal corticosteroid peaks before morning awakening, elevated levels of norepinephrine, plasma renin activity, cortisol, and platelet aggregation can lead to coronary artery spasm and contribute to the sudden onset of AMI. This suggests that undiagnosed glucose abnormalities, low atmospheric pressure at high altitudes, hypoxia, increased cardiac load, and microthrombus formation may all represent significant risks for AMI during rapid ascent. Given the unique high-altitude environment, individuals working outdoors who experience squeezing pain behind the sternum should be actively differentiated from conditions such as pulmonary embolism and acute pulmonary edema. Portable electrocardiograms can enhance early detection rates of AMI by providing timely electrocardiographic assessments. The special geographical environment at high altitudes alters the occurrence and progression of related diseases. This case provides important references for individuals rapidly ascending to high altitudes. Health education and screening for individuals preparing to enter high-altitude regions are crucial for alleviating psychological stress and reducing susceptibility to altitude-related illnesses among at-risk populations. This is significant for decreasing the incidence of altitude sickness among individuals rapidly ascending to high altitudes. Through this case study, we aim to provide an early warning mechanism for health issues in future populations ascending rapidly to high altitudes. Given the unique challenges posed by high-altitude environments, portable electrocardiogram examinations are essential for on-site diagnosis and differentiating between AMI, high-altitude pulmonary edema, and pulmonary embolism in individuals presenting with acute chest pain or difficulty breathing. Additionally, it underscores the necessity and specificity of maintaining oxygen saturation through supplemental oxygen in hypoxic environments following AMI.

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Corresponding author

Hai ying Xian (1988-), female, master’s degree, attending physician, research direction in high-altitude medicine.

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