CASE REPORT

Painless acute myocardial infarction on Mount Kilimanjaro

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SUMMARY

An individual experiencing dyspnoea or syncope at high altitude is commonly diagnosed to have high-altitude pulmonary edema or cerebral edema. Acute myocardial infarction (AMI) is generally not considered in the differential diagnosis. There have been very rare cases of AMI reported only from Mount Everest. We report a case of painless ST segment elevation myocardial infarction (STEMI) that occurred while climbing Mount Kilimanjaro. A 51-year-old man suffered dyspnoea and loss of consciousness near the mountain peak, at about 5600 m. At a nearby hospital, he was treated as a case of high-altitude pulmonary edema. ECG was not obtained. Two days after the incident, he presented to our institution with continued symptoms of dyspnoea, light-headedness and weakness, but no pain. He was found to have inferior wall and right ventricular STEMI complicated by complete heart block. He was successfully managed with coronary angioplasty, with good recovery.

BACKGROUND

Mountaineering and travelling to high altitudes are becoming increasingly popular, and over the past few decades, the number of individuals undertaking these activities has increased significantly. There is also increased prevalence of cardiovascular disease among mountain climbers, as shown by a study from the Austrian Alps, where 12.7% of the hikers were afflicted with at least one type of cardiovascular disease.¹ High-altitude exposure can lead to a variety of well-described clinical syndromes including acute mountain sickness (AMS), high-altitude pulmonary edema (HAPE), high-altitude cerebral edema (HACE), high-altitude retinal haemorrhage, etc.² Acute myocardial infarction (AMI) is not a commonly described entity in mountain climbers. Although sudden cardiac deaths have been reported to account for 30% of mountain sporting fatalities,³ very few cases of AMI have been reported; the reported cases have been related to climbing Mount Everest.^{4 5} One of the cases was a 29-year-old athletic man without any known risk factors for coronary artery disease.⁴ The other was a middle-aged man experiencing chest pain and dyspnoea, which was diagnosed as HAPE.⁵ We report a case of AMI occurring while climbing Mount Kilimanjaro, and this was also initially diagnosed as HAPE. Although the patient had cardiovascular symptoms, there was no chest, neck or arm pain. In the National Registry of Myocardial Infarction 2, there was 33% incidence of painless myocardial infarction.⁶ Chest pain is the usual

manifestation of a myocardial infarction, but atypical presentation without chest pain has been known to occur, especially in patients with diabetes, those of older age, female patients, those of non-white race and in patients with previous history of congestive heart failure or stroke.⁶ An increased awareness to consider AMI as a possibility in a mountain climber presenting with cardiovascular symptoms or atypical symptoms is warranted, especially since rapid revascularisation of coronary occlusion is the accepted best modality of treatment.

CASE PRESENTATION

A 51-year-old Norwegian man was climbing Mount Kilimanjaro in Tanzania with his wife. At about 5600 m, before reaching the peak at 5895 m, he experienced acute onset of dyspnoea associated with excessive sweating and loss of consciousness. Brief cardiopulmonary resuscitation (CPR) was performed. Since there was no stretcher available, he was quickly dragged down the mountain to about 4700 m, where he had a brief episode of tonic-clonic seizure; the exact duration of the seizure was not documented. There was no associated urine or faecal incontinence, and no postictal phase. One of his travelling companions gave him acetazolamide, with diclofenac to reduce the low back pain caused by being pulled down the mountain. The dosage of medications is not known. Later he was brought down the mountain by a stretcher and taken to Moshi hospital, where he was diagnosed to have AMS and HAPE. ECG was not performed. He was discharged home after administration of acetazolamide 250 mg orally, furosemide 40 mg orally and diclofenac 75 mg intramuscularly. He continued to feel weak and experienced dyspnoea and dizziness on walking, but had no chest, neck or arm discomfort. Owing to his continued symptoms he was brought by a commercial flight to Dar es Salaam, Tanzania, and reported at the Aga Khan Hospital 2 days after the incident. The patient's symptoms were dyspnoea, fatigue, light-headedness and low back pain. He denied any history of hypertension, diabetes mellitus or hyperlipidaemia. There was no family history of premature coronary artery disease; his father had a myocardial infarction and coronary stenting in his seventies. The patient, however, had been smoking one to two packs of cigarettes a day for about 30 years. In the emergency room, the examination showed heart rate of 56 bpm and blood pressure of 108/75 mm Hg. The patient was awake and oriented, but appeared weak and slightly drowsy. Cardiovascular examination

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showed cannon A waves in the jugular veins and variable intensity of first heart sound. His lungs were clear with equal air entry bilaterally. ECG showed normal sinus rhythm, complete heart block with junctional escape rhythm, small Q waves in inferior leads, and inferior wall ST segment elevation myocardial infarction (STEMI) with significant ST depression in the precordial and lateral leads (figure 1). The right-sided chest leads ECG showed right ventricular injury as well (figure 2). Complete blood count, serum electrolytes, lipid profile and serum creatinine were essentially normal. Total serum creatine kinase (CK) was 1047 IU/L, with CK-MB of 64 IU/L (normal values of up to 171 and 25 IU/L, respectively). Qualitative troponin T was positive.

Although the patient was pain free, he had other symptoms together with ECG evidence of complete heart block as well as continued ischaemia and injury. It was decided to proceed with emergency cardiac catheterisation with possible angioplasty. Preprocedure, he was administered oral aspirin 300 mg, clopidogrel 600 mg and atorvastatin 80 mg. Owing to the complete heart block, a temporary transvenous pacemaker was placed prophylactically via the right femoral vein at the beginning of the procedure. It was removed at the end of the procedure, since the junctional escape rhythm remained stable.

Coronary angiogram performed via the right femoral artery revealed total occlusion of the dominant right coronary artery (RCA) within a few millimetres of its origin (figure 3), with thrombolysis in myocardial infarction (TIMI) grade 0 flow. There was mild disease of the left coronary artery with faint collateral filling of the distal RCA and mid-RCA. The RCA total occlusion was crossed with a 014 Rinato wire^R (Abbott lab) and after predilation with a 2 mm balloon, the area of total occlusion was stented with a 3 mm×26 mm Resolute drug eluting stent (DES)^R (Medtronic). Repeat angiogram showed another 70% lesion in the mid-RCA, which was treated with an overlapping 3 mm×26 mm Resolute DES. Both the stents were postdilated with a 3.5 mm non-compliant NC Quantum Apex balloon^R (Boston Scientific) at high pressure. Final right coronary angiogram showed restoration of TIMI grade 3 flow in the RCA (figure 4).

Echocardiogram performed the day after the angioplasty showed moderate right ventricular and right atrial dilation, normal left ventricular chamber size and akinesia of basal

inferior septum with overall good systolic function. Estimated left ventricular ejection fraction was 65%. There was borderline septal hypertrophy. On the second day after the procedure, the complete heart block had resolved. The patient was ambulated without symptoms and was discharged home on the third day on oral medications of daily aspirin 75 mg, prasugrel 10 mg and atorvastatin 80 mg.

DIFFERENTIAL DIAGNOSIS

At very high altitude, if an individual experiences acute onset of dyspnoea associated with excessive sweating and loss of consciousness requiring brief CPR, the differential diagnoses to consider are:

1. AMS: This is a mild form of high-altitude sickness usually presenting with headache plus one or more of the following: gastrointestinal symptoms (anorexia, nausea or vomiting), insomnia, dizziness and lassitude or fatigue. The symptoms in this case were much more severe.
2. HAPE: Dyspnoea with cough and pink frothy sputum are the usual symptoms. Excessive diaphoresis, loss of consciousness and need for brief CPR would favour looking for an alternative or additional diagnosis.
3. HACE: Usual symptoms are severe headache, change in mentation, ataxia, photophobia, nausea, vomiting, etc. Acute dyspnoea and excessive sweating are not present.
4. AMI: Dyspnoea, excessive diaphoresis, loss of consciousness and brief CPR would favour this possibility. Usually, patients with AMI have chest pain, but in about a third of patients it is absent.

OUTCOME AND FOLLOW-UP

The patient flew back home to Norway 2 days after discharge. He is back to full activity including bike riding in the Norwegian mountains 1000 m above sea level.

DISCUSSION

High mountain climbing is a very strenuous activity that is carried out in a cold environment at a low ambient oxygen level. It is believed that altitude exposure is generally safe in healthy subjects but could pose a risk in patients with previous cardiovascular conditions. At high altitude, hypoxia, physical activity and dehydration cause sympathetic nervous system

Figure 1 Initial ECG showing acute inferior wall injury.

Figure 2 Initial ECG with right-sided chest leads showing right ventricular injury.

activation resulting in widespread vasoconstriction, with an increase in heart rate, blood pressure and cardiac output.⁷ To meet the increased demand, the myocardium has to rely almost exclusively on coronary vasodilatation.⁷ Individuals with coronary artery disease are particularly at increased risk of myocardial ischaemia because of their impaired endothelial vasomotor function. Moreover, the cold temperatures as well as the alkalosis caused by increased ventilation and sympathetic activity may also cause coronary vasoconstriction, reduced myocardial perfusion and platelet activation. All these factors could trigger acute coronary syndrome in individuals with coronary atherosclerosis.⁷ STEMI is a form of acute coronary syndrome that usually occurs from rupture of a vulnerable coronary plaque followed by occlusion of the artery by intracoronary thrombus.

Chest pain is the usual manifestation of a myocardial infarction but atypical presentation without chest pain is known to

occur, especially in patients with diabetes, those of older age, female patients, those of non-white race and patients with a history of congestive heart failure or stroke.⁶ Compared with myocardial infarction patients who present with chest pain, those without chest pain have higher mortality.⁶ The absence of pain in a mountain climber cannot be attributed to the ambient condition since at high altitude pain threshold has been reported to be decreased through improved sensory discrimination.⁸ In our patient, the presentation was with cardiovascular symptoms but without pain. An ECG was not obtained at the initial medical encounter, resulting in a missed diagnosis. Early coronary angiogram and angioplasty is the preferred recommended management of a STEMI.⁹ The use of non-steroidal anti-inflammatory drugs (NSAIDs) (such as diclofenac) for pain relief is common in mountain climbers. If myocardial infarction is suspected, the use of NSAIDs and selective cyclo-oxygenase-2 (COX-2) inhibitors are contraindicated because their use is

Figure 3 Dominant right coronary artery with proximal total occlusion (black arrow).

Figure 4 Dominant right coronary artery after angioplasty and stenting. Stents marked by white arrows.

associated with an increased risk of death, reinfarction, cardiac rupture, hypertension, renal insufficiency and heart failure.⁹

Finally, our patient also had seizure after his initial collapse. A seizure at high altitude can be confused with epilepsy or HACE.

Patient's perspective

- ▶ It was a tremendous ordeal getting sick on top of Mount Kilimanjaro. The distances are vast and communications poor. I was, however, never truly afraid because everybody told me it was probably only mountain sickness.
- ▶ Because nobody thought of heart attack, I retrospectively realise that it was a tremendous risk taking a commercial flight from Moshi to Dar es Salaam.
- ▶ I am extremely grateful for the excellent, professional and caring treatment I received in the hands of Dr Jamal and his team at the Aga Khan Hospital in Dar es Salaam.

Learning points

- ▶ Besides acute mountain sickness (AMS), high-altitude pulmonary edema (HAPE), high-altitude cerebral edema (HACE), etc, myocardial infarction should be in the differential diagnosis of an individual who is experiencing cardiovascular symptoms at high altitude.
- ▶ Recognise that myocardial infarction can present with other cardiovascular symptoms without chest pain even though there is a lowered pain threshold at high altitude.
- ▶ Non-steroidal anti-inflammatory drugs (NSAIDs) are frequently prescribed for pain relief in mountain climbers. If a myocardial infarction is suspected, one should avoid the use of NSAIDs and arrange for emergent management.
- ▶ An electrocardiogram should be obtained after successful cardiopulmonary resuscitation to rule out myocardial infarction, arrhythmia, etc.

Contrary to what might be expected, there is no evidence that altitude increases the risk of an epileptic seizure even in patients with known epilepsy that is well controlled with medication.¹⁰ In our patient, we believe that the seizure may have been caused from cerebral hypoperfusion due to bradycardia resulting from complete heart block.

Up to now, very few cases of myocardial infarction have been reported during mountain climbing. One wonders if some cases have been missed or misdiagnosed as mountain sickness or HAPE,⁵ as was the situation with our patient. It is important to have a high index of suspicion of myocardial infarction even in the absence of pain and not to attribute cardiorespiratory difficulty at high altitude to HAPE.

Competing interests None declared.

Patient consent Obtained.

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