

A case of mountain sickness with premature ventricular contraction improving while descending a mountain

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Abstract: A 26-year-old Korean male started to climb Mt. Fuji, the highest mountain in Japan (3776 m above sea level [ASL]). He began coughing at the seventh station of the mountain (2700 m ASL) but continued to climb. When he reached the top of the mountain, he felt dyspnea, chest pain and headache. Even after descending the mountain, these symptoms did not improve. On arriving back at the seventh station, he could not stand and called a mountain rescue. When the members of the mountain rescue team checked him, he had clear consciousness, heart rate was 110 beats per minute (BPM) and his SpO₂ level was 75% under room air. When the medical staff checked him at the fifth station (2400 m ASL), he still felt chest pain and had a dry cough. Electrocardiogram showed approximate 10 premature ventricular contractions (PVCs) per minute. He was transported to our hospital (17 m ASL). During the transportation, his symptoms improved, and the frequency of the PVCs decreased. Electrocardiogram, chest roentgen and cardiac sonography findings were negative. Chest computed tomography depicted a ground glass appearance in the right lung field. After receiving 3 L/min of oxygen, all of his symptoms subsided the next day, and he was discharged. Given that hypoxia at high altitude can cause arrhythmia, physicians should consider the occurrence of life-threatening arrhythmia when an armature climber tries to climb a high mountain.

Keywords: mountain sickness; premature ventricular contraction; armature climber.

INTRODUCTION

During ascent from sea level, the atmospheric pressure and oxygen partial pressure, humidity and temperature decrease. High altitude is a challenge for the human body due to the progressive reduction of barometric pressure and subsequent reduction of oxygen pressure, leading to a series of physiologic responses that enable individuals to tolerate hypoxia and secure the oxygen supply to tissues[1]. Most of these adaptations are observed from 2000 m above sea level (ASL). The lung response to acute altitude exposure is mainly hyperventilation, which, together with an elevated heart rate, aims to deliver an adequate supply of oxygen to the tissues[1]. At rest, ventilation increases by first increasing the tidal volume, at least up to 3500 m[1]. Above this altitude, the breathing rate also significantly increases. Besides the compensatory response, other mechanisms affect the lung physiology during hypoxic exposure. Impaired pulmonary endothelial and epithelial nitric oxide synthesis and/or bioavailability may represent a central underlying defect predisposing individuals to exaggerated hypoxic pulmonary vasoconstriction, capillary stress failure and alveolar fluid flooding, which cause pulmonary edema[2]. While hypoxia induces a variety of arrhythmic events, few reports have described arrhythmia at high altitude[3-5]. We herein report a case of mountain sickness with premature ventricular

contraction that improved while descending the mountain.

CASE PRESENTATION

A 26-year-old Korean male started to climb Mt. Fuji, the highest mountain in Japan (3776 m ASL), from the Gotemba new fifth station (2400 m ASL) at 8 PM. He had no significant medical history, and this was his first time climbing such a high mountain. He began coughing at the seventh station of the mountain (2700 m ASL) but continued to climb. When he reached the top of the mountain at 5 AM the next day, he felt dyspnea, chest pain and headache in addition to a dry cough. Even after descending the mountain, these symptoms did not improve. On arriving back at the seventh station, he could not stand and called a mountain rescue. When the members of the mountain rescue team checked him at 3:43 PM, he had clear consciousness, his systolic blood pressure was 120/76 mmHg, heart rate was 110 beats per minute (BPM) and his SpO₂ level was 75% under room air. When the medical staff, transported via helicopter and ambulance,[6] checked him at the fifth station (2400 m ASL) after the mountain rescue team brought him down from the mountain at 5:55 PM, he still felt chest pain and had a dry cough. He had clear consciousness, his systolic blood pressure was 136/98 mmHg, heart rate was 86 beats per minute (BPM) and his SpO₂ level was

85% under room air, which increased to 100% on supplying 100% oxygen with a reservoir mask. Electrocardiogram showed approximate 10 premature ventricular contractions (PVCs) per minute, corresponding to grade 2 under the Lown criteria (Figure 1). [7] He was transported to our hospital (17 m ASL) at 9:10 PM via ambulance escorted by a doctor. During the transportation, his symptoms improved, and the frequency of the PVCs decreased. Electrocardiogram, chest roentgen and cardiac

sonography findings were negative (Figure 2). Chest computed tomography (CT) depicted a ground glass appearance in the right lung field and atelectasis in both dorsal lungs (Figure 3). The main results of a blood analysis were white blood cell count of $10,900/\text{mm}^3$ and N-terminal fragment of the type-B natriuretic peptide (NT-proBNP) of 1290 (<125) pg/ml. There were no elevated cardiac enzyme levels. After receiving 3 L/min of oxygen, all of his symptoms subsided the next day, and he was discharged.

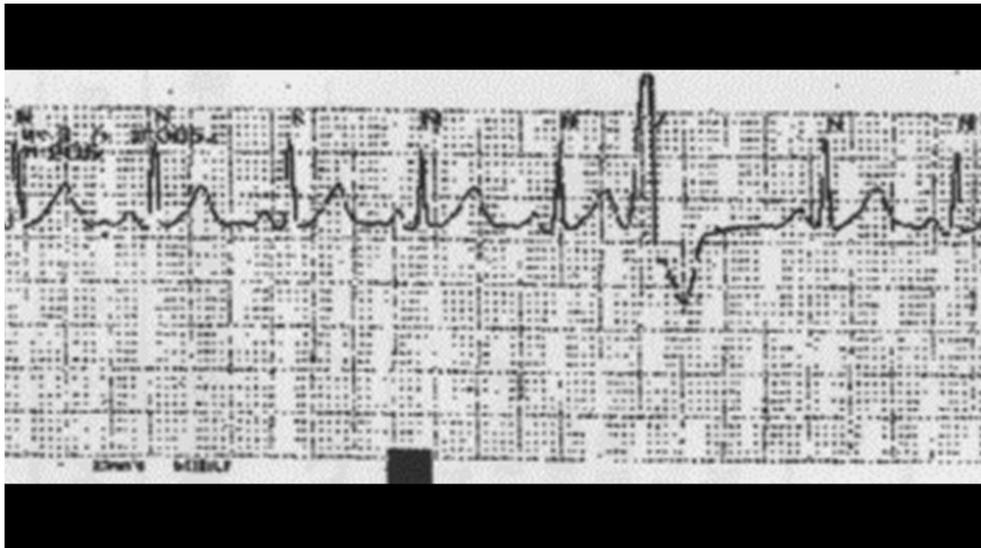


Fig-1: The electrocardiogram findings while ascending a mountain. The image shows premature ventricular contractions.

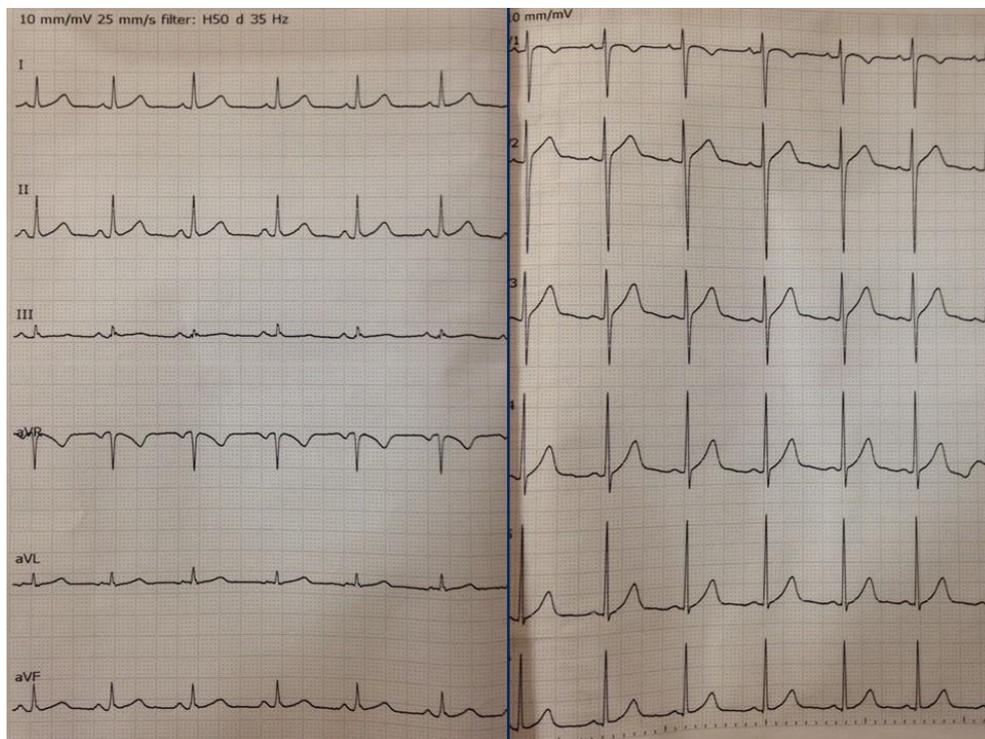


Fig-2: The electrocardiogram findings on arrival. The electrocardiogram shows no premature ventricular contractions.

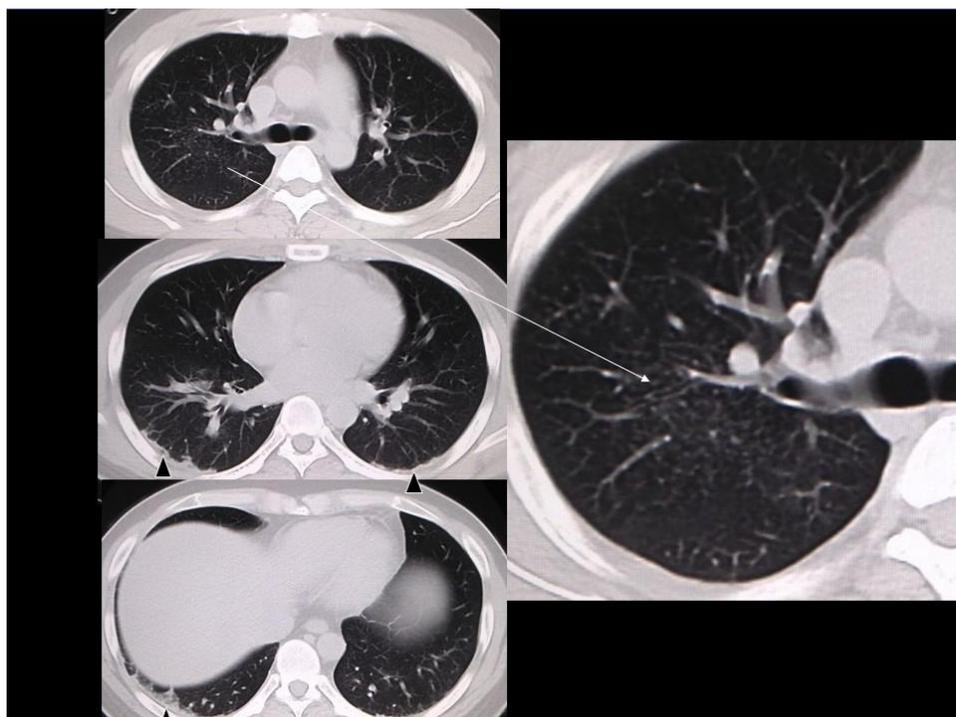


Fig-3: Chest computed tomography (CT) on arrival. CT depicted a ground glass appearance in the right lung field (white arrow) and atelectasis in both dorsal lungs (black triangle).

DISCUSSION

As the present case had no significant medical history but showed the triad (cough, chest pain, dyspnea) while climbing a high mountain and improvement in the triad by descending the mountain and receiving oxygen, along with mild lung edema on CT, he was diagnosed with mountain sickness including high-altitude pulmonary edema. As an increase in the NT-proBNP is observed in patients with high-altitude pulmonary edema induced by high pulmonary artery systolic pressure[10] the marked elevation of the NT-proBNP in this case was compatible with a diagnosis of mountain sickness. Climbing to the top of a high mountain without preparation is one cause mountain sickness[11].

The present case had also sinus tachycardia and hypoxia (SpO₂ 75%) at the seventh station (2700 m ASL), and PVCs and hypoxia (SPO₂ 85%) at the Gotemba new fifth station (2400 m ASL). While descending the mountain, the frequency of the PVCs decreased and disappeared by the time he reached ground level, so these PVCs were deemed to have been induced by hypoxia. At high altitude, the increase in the sympathetic tone triggered by hypoxia, hypothermia and exercise while climbing and respiratory alkalosis triggered by hyperventilation tends to induce arrhythmia[9]. The effect of hypoxia on the autonomic nervous system commonly results in an increase in the heart rate during rest and exercise, similar to the present case[12]. Given that hypoxia is known to trigger PVC even under normo- and hyperbaric conditions[13,14] this case was compatible with hypoxia-induced PVCs.

While, Woods *et al.* reported the results of implantable loop recorders inserted subcutaneously in the left pectoral region of 9 healthy male volunteers who were flown to Kathmandu (1,250 m) and then Lukla(2,800 m) before immediately commencing an identical ascent and descent profile to high altitude[15]. All subjects demonstrated sinus tachycardia and marked sinus arrhythmia; one subject demonstrated atrial flutter; one subject had non-conducted p waves and a further subject had marked ST segment depression. The authors therefore concluded the increased risk of sudden cardiac death at high altitude and noted that the elderly account for 15% of the 100 million visitors to altitude annually [15,16]. In animal study, the adaptation to 5000 m for 4 h/day decreased the total number of PVCs[17]. Fact that adaptation to intermittent high altitude hypoxia is associated with enhanced tolerance of the rat heart to ischemic arrhythmias, may explain dissociation of frequency of PVC between Woods and ours.

CONCLUSION

Given that hypoxia at high altitude can cause arrhythmia, physicians should consider the occurrence of life-threatening arrhythmia when an amateur climber tries to climb a high mountain.

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