Acute mountain sickness without headache at low altitude

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Acute mountain sickness (AMS) may occur at altitudes below 3000 m and may be triggered by chronic stress and over-exercise.

Case report

A 45-year-old tourist developed AMS three hours after starting to ski in a glacier skiing-region in Austria. His history was noteworthy for chronic stress, coffee-overuse, chronic sleep-deprivation, and previous AMS, 14 years ago at ~3000 m and six years ago at ~5100 m. He occasionally trekked to altitudes of 1500–2500 m without problems. The night before suffering AMS he had slept for four hours at an altitude of 760 m, and had drunk 1 L of coffee in the morning but less during previous days. He did not take any azetazolamide prophylaxis before travelling to the glacier.

After a 30-minute drive to the skiing-arena, he took the first cable car and went skiing for three hours without any problems (Table 1). After a strenuous ride off the regular slope he became tired and movements slowed down but recovered during ascent to 2678 m (Table 1). He skied down to 2450 m and although he needed a rest, he ascended to 2656 m. During this ascent at ~11 h he recognized intense tiredness and progressive impairment of breathing. He developed nausea and had the urgent need to descend to lower altitudes. He skied down to 2450 m with an increasing number of stop-overs. On arrival he was dyspnoeic with a breathing rate of ~50/min, panted for air, longed for oxygen, was tachycardious, recognized paresthesias of both hands and feet, and no longer able to recover despite resting. He managed to get on a cable car on his own, with slow motions, and descend to 1979 m. Despite this descent within four minutes and lying flat in the cabin, tachypnoea and tachycardia persisted, he became dizzy and feared he would immediately faint. When arriving at 1979 m he had difficulty leaving the cabin, to stay upright and to move. He was hardly able to lean on his sticks and took every effort not to faint. An employee of the lift-company helped the patient into a cable car and accompanied him on his further descent, during which tachypnoea and tachycardia did not resolve. He asked the employee to call the emergency services to give him oxygen and to transfer him to a hospital on arrival.

After delayed arrival of the doctor at the valley station, he measured a blood pressure of 120/60 mmHg, rhythmic tachycardia (116 beats/min) and a breathing rate of 42/min. He interpreted the condition as psychogenic hyperventilation, and vigorously prompted the patient to voluntarily stop tachypnoea and tried to infuse 500 mL of sodium-chloride plus diazepam (10 mg), which the patient vehemently refused. During transport to the hospital, tachypnoea and tachycardia resolved, and on admission auscultation and blood pressure were normal, electrocardiography showed sinus-rhythm (87/min), normal breathing rate, and normal creatine kinase and D-dimer. Blood gas analysis and X-ray of the lung were normal. He was not given oxygen at any time when symptoms evolved. He dismissed himself against medical advice and despite some concern about the incident, went skiing the same day. He ascended to maximally 2000 m during the next two hours, but tolerated the strain with some indisposition and weird feeling. After sufficient sleep, he skied during the whole next day to a maximal altitude of 2074 m, without complaints. Two days after AMS, he went to the same glacier skiing-region, where he had experienced AMS, even to an

Table 1
Lift rides before and after AMS in the presented patient

Time	*AVS*	*ATS*	*DR*	*SAMS*	*ADS*
08:05	911	1979	8	No	0
08:15	1978	2450	4	No	15 (slope)
08:40	1978	2450	4	No	0
08:45	2450	2656	8.5	No	10 (slope)
09:00	2450	2656	8.5	No	0
09:10	2629	2936	5.5	No	15 (slope)
09:35	2530	2842	4	No	30 (off slope)
10:10	2414	2611	6	No	15 (slope)
10:35	2265	2678	NP	No	10 (slope)
10:55	2450	2656	8.5	Onset	20 (slope)
11:30	2450	1978	4	Yes	Descent with lift
11:45	1979	911	8	Yes	Descent with lift
12:30	NA	NA	NA	Resolution	NA

AMS, acute mountain sickness; AVS, altitude at valley-station; ATS, altitude at top-station; DR, duration of ride (in minutes); SAMS, symptoms of AMS; ADS, approximate duration of skiing (in minutes); NP, not provided; NA, not applicable

altitude above 3000 m, and stayed there for the whole day, without any cardiac or respiratory problems.

Discussion

Altitude illness (AI) results from hypobaric hypoxia and comprises AMS and chronic mountain sickness, which may deteriorate to high-altitude pulmonary oedema (HAPE) or high-altitude cerebral edema (HACE).[1,2] AMS is the most frequent type of AI and characterized by headache, loss of appetite, nausea, anorexia, fatigue, sleep disturbance or lassitude within 6–12 h of gaining altitude.[3] In non-acclimatized patients and patients at risk for AI, AI may occur already below 3000 m.

Severe AMS (Lake Louis score 7) was diagnosed in the presented patient despite the absence of headache, since no other causes of his complaints were detected (Table 2).[1,2] Tachypnoea was regarded as a manifestation of AMS, because it has been previously reported during AMS[4] and is regarded as part of the cardiovascular response to high altitude (increase in cardiac output, pulmonary artery pressure and blood pressure, tachypnoea and tachycardia).[5] Tachypnoea may even be periodic.[6] The diagnosis was supported by the fact that the condition started three hours after ascent and that the abnormalities immediately disappeared upon descent to <1000 m. An argument against AMS could be the early onset after ascent (3 h contrary to 6–12 h per definition).[3] Differential diagnoses excluded were pulmonary embolism, myocardial infarction, viral infection, cardiac arrhythmia or inhalation of toxic gases. Mild pulmonary embolism cannot be completely excluded, since no computed tomography scans of the lung were performed.

The presented case is interesting for several points. Firstly, AMS developed at relatively low altitude. In most cases, AMS developed at altitudes >3000 m.[1–3] Only rarely does AMS develop at altitudes <3000 m.[4] Low-altitude AMS in the presented patient may be due to refraining from

Table 2
Assessment of severity of AMS in the described patient according to the Lake Louise Score

	Rating
Headache	
None	**0**
Mild	1
Moderate	2
Severe, incapacitating	3
Gastrointestinal symptoms	
None	0
Poor appetite or nausea	1
Moderate nausea or vomiting	**2**
Severe nausea or vomiting	3
Fatigue or weakness	
Not tired or weak	0
Mild fatigue/weakness	1
Moderate fatigue/weakness	2
Severe fatigue/weakness	**3**
Dizziness/lightheadedness	
Not dizzy	0
Mild dizziness	1
Moderate dizziness	**2**
Severe dizziness, incapacitating	3
Difficulty sleeping	
Slept as well as usual	**0**
Did not sleep as well as usual	1
Woke many times, poor sleep	2
Could not sleep at all	3
Total score	7

AMS, acute mountain sickness
A total score of 3–5 is interpreted as mild AMS, a score of >5 as severe AMS

acclimatization, chronic stress, chronic sleep deprivation or the history of previous AMS. Usually, pulmonary function decreases with increasing altitude.[7] Risk factors, which generally predispose for AMS include rapid ascent, strenuous physical activity, young age, living at low altitude and a history of AI[8], of which four were the case in the presented patient.

Secondly, triggers of AMS include hypobaric hypoxia, dehydration, increased physical exertion (over-exertion), infection or exsiccosis.[9] Triggers in the presented patient may be dehydration, excessive exercise, chronic stress, excessive consumption of coffee, sleep deprivation, non-acclimatization and the history of previous AMSs. However, he had climbed to such altitudes under similar conditions repeatedly before without complaints.

Thirdly, the patient had already experienced two previous episodes of AMS. A general disposition for AI is rather unlikely, since he had repeatedly climbed to high altitudes without problems between the three episodes. Whether patients with a positive history for AMS carry an increased risk to experience relapsing AMS is unknown, but some authors regard previous AMS as a risk factor for a relapse.[8]

Fourthly, the patient had not developed headache or a visual problem at any time during AMS, which is usually part of the clinical spectrum of AMS. Whether headache is truly a prerequisite for diagnosing AMS is under debate, since it was absent even in patients with HAPE.[10] Absence of headache could be explained by the absence of increased cerebral pressure, which is believed to cause headache in AI. The absence of headache was most likely due to the relatively low altitude at which microgravity is not large enough to result in an inflow/outflow mismatch of cerebral perfusion or hypoxia-induced pulmonary vasoconstriction and consecutive compromise of cerebral venous outflow.[11]

In conclusion, AMS may occur at altitudes <3000 m and be triggered by chronic stress or over-exercise. Appropriate acclimatization even for altitudes <3000 m is recommended to prevent stress-triggered AMS. Oxygen should be available in emergency rooms of lift stations in high-altitude skiing areas. Absence of headache may strengthen the need to broaden the lower end of the clinical spectrum of AMS and to also include cases of pre-AMS, which generally are self-resolving and require only minimal intervention. Outcome of AMS is favourable upon immediate descent to low altitude.

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