

LETTER TO THE EDITOR

Risk assessment for a high-altitude alpinist with coronary artery disease

Dear Editor,

we report the case of a 60-year-old man, professional climber, who was attending a mountaineering expedition in Annapurna. At an altitude of about 5500 m the patient felt sudden onset of dyspnea, out of proportion to the effort he was making, for which he was trained. After coming back to Italy, the patient underwent cardiologic examination. A 12-lead electrocardiogram (ECG) was performed: no signs of ischemia were present but a significant increase of troponin I was reported. Transthoracic echocardiography showed no alterations in left ventricular regional kinetics and a normal ejection fraction. However, it was decided to carry out a coronary angiography, which documented a stenotic lesion in a single vessel occluding the mid anterior descending artery, treated by percutaneous coronary intervention and bare metal stenting. Thereafter, the patient performed regular cardiologic follow-ups (including ECG, echocardiography and exercise stress test), always resulting negative for inducible myocardial ischemia. 18 months later, the

patient expressed the desire to continue performing high-altitude mountaineering, so he was directed to our centre for advice. In order to have an assessment as faithful as possible to the conditions that he would meet at high altitude, the patient underwent cardiopulmonary exercise testing with cycle ergometer breathing a hypoxic gas mixture (oxygen at 12%), simulating an altitude of 4800 m. The patient remained asymptomatic for the whole duration of the test. There were no ECG changes indicative of inducible myocardial ischemia. The oxygen consumption (VO_2) at anaerobic threshold was 13.6 mL/kg/min, while the peak VO_2 was 26.5 mL/kg/min (equal to 116% of the predicted maximum that is 22.8 mL/kg/min) and the indexes of cardio-respiratory functional capacity were greater than normal. Comparing the values of arterial oxygen saturation (SaO_2) at baseline and during hypoxia, the difference in SaO_2 ($\Delta\text{SaO}_2\%$) at rest was 8.32% (normal value $<21\%$), with a value of minimum SaO_2 at rest in hypoxia of 85%, while the $\Delta\text{SaO}_2\%$ during exercise was 17.5% (normal value $<33\%$), with a value of minimum SaO_2 during exercise in hypoxia of 65%. Furthermore, the patient showed a good cardio-respiratory response to hypoxia (Figure 1). Considering these data,

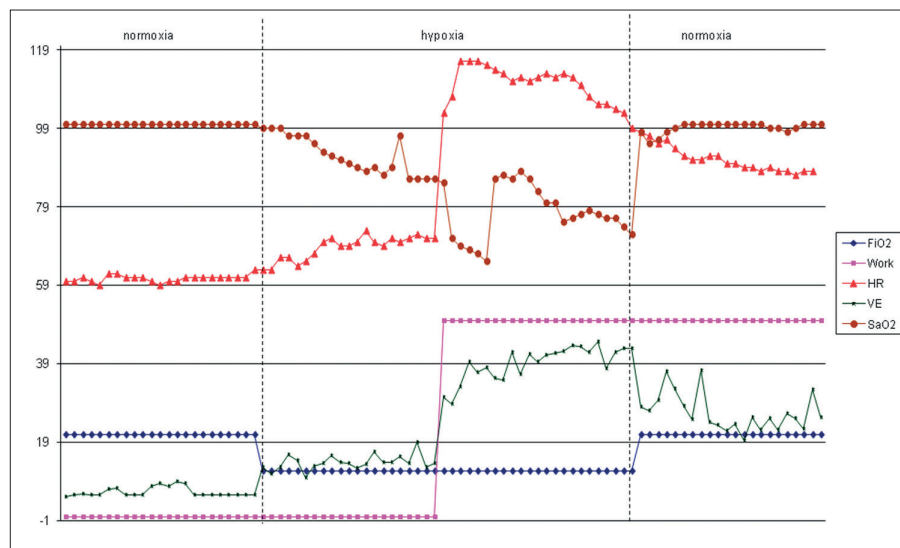


Figure 1 - Cardio-respiratory response to cardiopulmonary exercise test with hypoxic gas mixture. FiO_2 = fraction of inspired oxygen; HR = heart rate; VE = expired volume; SaO_2 = arterial oxygen saturation.

namely the constant asymptomatic state, the absence of residual coronary disease at control coronary angiography, the negativity of ergometric tests, and the excellent functional capacity demonstrated in cardiopulmonary exercise testing with hypoxic mixtures, the patient went back to high mountain at an altitude of about 5500 m. During the stay and mountain climbing, he did not feel symptoms. Subsequent follow-up showed clinical stability.

As well known, exposure to high altitude may exacerbate cardiac and pulmonary diseases. Particularly, cardiovascular adaptation to it implies hypoxemia, pulmonary hypertension, sympathetic activation and subsequent reduction in plasma volume with increased peripheral resistances (1, 2). Moreover, it was recently demonstrated by coronary angiography that diseased coronaries do not dilate with increasing hypoxemia as normal ones do (3). All these phenomena may severely deteriorate coronary perfusion in patients with overt or unknown coronary artery disease (CAD).

Few studies exist on the effects, in term of safety, of exposure to high altitudes in patients with stable CAD. Some studies debated on the safety of high altitude after coronary bypass surgery (4, 5). Although some authors recommend caution, or even discourage physical activity at high altitude in patients with CAD, especially in those with impaired coronary flow reserve (6, 7), there is no evidence on its contraindication in stable patients. Several authors suggest that for asymptomatic patients with good exercise tolerance, high altitude exposure represents an acceptable risk (5, 8, 9). Among these, Rimoldi et al. (10) state that in patients with stable CAD, if exercise testing is clinically and/or electrically negative, then high-altitude exposure can be considered safely. Schmid et al. (9) administered to 15 patients after ST-elevation myocardial infarction and 7 patients after a non-ST-elevation myocardial infarction (12 months after the acute event) a maximal, symptom-limited exercise stress test at 540 m (in Bern) and after a rapid ascent to the Jungfrauoch, Switzerland

(3454 m). After maximal exercise stress test, no ECG signs of myocardial ischemia or significant arrhythmias were noted. The authors concluded that rapid ascent and submaximal exercise can be considered safe for low risk patients 6 months after revascularisation for an acute coronary event and a normal exercise stress test at low altitude. De Vries et al. (11) performed an exercise test and echocardiography at sea level and at base camp (4200 m) during an expedition at Aconcagua in 8 patients with a history of acute myocardial infarction and a low risk score compared with 7 healthy controls. Symptoms and echocardiographic signs of myocardial ischemia were absent in both groups. Wu et al. (12) described the case of a patient post-coronary stenting who intermittently ascended to and worked at an altitude near 5000 m 2 years after stenting procedure, without significant alterations at regular follow-up. One of the major question is how can these patients be evaluated. In this field, Marticorena et al. studied the impact of the simulated hypoxia technique into hypobaric chamber to rehabilitate CAD patients (13).

This case highlights the lack of indications in current guidelines on the recovery to work activity or sports at high altitude after an ischemic episode. According to our knowledge, there are no trials exploring this issue, but only clinical cases in which patients were taken at high altitudes and an exercise stress test or an evaluation of inducible ischemia are performed. We think that a return to mountain climbing at high altitude may be safe if a cardiologist and a pulmonologist specialized on high-mountains diseases evaluate together such cases. As shown in our case, a crucial role should be played by cardiopulmonary exercise test with hypoxic gas mixtures. In clinical practice it is not always possible to directly assess exercise stress test at high altitude, and certainly after an ischemic episode having an assessment of inducible ischemia before rising again at high altitude is more correct. Evaluating the patient on the stratification of cardiovascular risk and recurrent ischemia with various test of inducible ischemia, namely

exercise stress test, scintigraphy, stress echo, performed at low altitude, could be a mistake as they are carried out in completely different conditions of hemodynamic and gas exchange comparing to those in which the patient will be in high mountain.

Our opinion is that highly specialized centres with experience on high mountain diseases, involving both pulmonologists and cardiologists, should evaluate patients such as the one described in this case, in order to provide an integrated assessment of the cardiovascular and pulmonary risk by non-routine tests (e.g., cardiopulmonary exercise test with hypoxic mixtures).

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Enrico Vizzardi¹, Marialma Berlendis²,
Edoardo Sciatti¹, Ivano Bonadei¹,
Filippo Quinzani¹, Gian F. Tassi², Marco Metra¹

¹Section of Cardiovascular Diseases,
Department of Medical and Surgical Specialties,
Radiological Sciences and Public Health,
University of Brescia, Italy;
²Pneumologic Unit, Spedali Civili of Brescia, Italy

Corresponding author:
Dr. Enrico Vizzardi
Piazzale Spedali Civili 1
25123 Brescia, Italy
E-mail: enrico.vizzardi@tin.it

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