

CASE REPORT

Deep Vein Thrombosis and Pulmonary Embolism in a Mountain Guide: Awareness, Diagnostic Challenges, and Management Considerations at Altitude

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High intensity exercise is associated with several potentially thrombogenic risk factors, including dehydration and hemoconcentration, vascular trauma, musculoskeletal injuries, inflammation, long-distance travel, and contraceptive usage. These are well documented in case reports of venous thrombosis in track and field athletes. For mountaineers and those working at high altitude, additional risks exist. However, despite there being a high degree of vigilance for “classic” conditions encountered at altitude (eg, acute mountain sickness, high altitude pulmonary edema, and high altitude cerebral edema), mainstream awareness regarding thrombotic conditions and their complications in mountain athletes is relatively low. This is significant because thromboembolic events (including deep vein thrombosis, pulmonary embolism, and cerebral vascular thrombosis) are not uncommon at altitude. We describe a case of deep vein thrombosis and pulmonary embolism in a male mountain guide and discuss the diagnostic issues encountered by his medical practitioners. Potential risk factors affecting blood circulation (eg, seated car travel and compression of popliteal vein) and blood hypercoagulability (eg, hypoxia, environmental and psychological stressors [avalanche risk, extreme cold]) relevant to the subject of this report and mountain athletes in general are identified. Considerations for mitigating and managing thrombosis in addition to personalized care planning at altitude are discussed. The prevalence of thrombosis in mountain athletes is uncharted, but lowlanders increasingly go to high altitude to trek, ski, or climb. Blood clots can and do occur in physically active people, and thrombosis prevention and recognition will demand heightened awareness among participants, healthcare practitioners, and the altitude sport/leisure industry at large.

Key words: altitude, deep vein thrombosis, exercise, mountain sport, pulmonary embolism, stress

Introduction

Mountain athletes and those who work or pursue leisure activities at high altitude are exposed to multiple, potentially thrombogenic risk factors (vascular damage, hypercoagulability and circulatory stasis) relating to Virchow’s triad (Figure 1).¹ However, despite there being a high degree of vigilance for “classic” conditions encountered at altitude (eg, acute mountain sickness [AMS], high altitude pulmonary edema, and cerebral edema), mainstream awareness about the risk factors for thrombotic conditions, including deep vein

thrombosis (DVT) and pulmonary embolism (PE), could be improved. We highlight a case of DVT and PE in an elite male mountain athlete who presented to healthcare practitioners over the course of 1 month and identify his risk factors (Figure 1), “easy to miss” signs and symptoms, and the differential diagnoses that were made before formal diagnosis.

Case Report

An otherwise healthy 42-year-old male mountain guide (73.8 kg; 178 cm; body mass index 23.29) first presented to medical practitioners at a ski resort (1000–2000 m above sea level) with physical fatigue and symptoms typical of a common cold. His heart rate was 60 beats/min and blood pressure within the normal range (120/80–140/90 mmHg). His peripheral capillary oxygen

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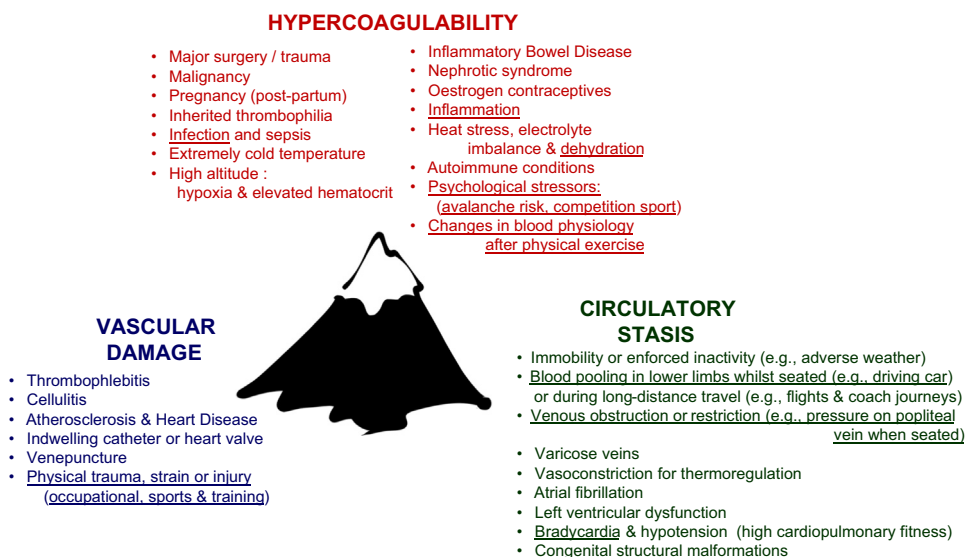


Figure 1. Virchow's triad (circa 1854) for thrombosis and thromboembolic conditions. Risk factors that are believed to have affected the subject of the present study are underlined. Public domain image.¹

saturation was normal (>95%), and he did not smoke, had no history of lung embolism or thrombosis, and did not experience angina pectoris, edema, or neoplasia. He was not known to have a hereditary thrombophilia or any other condition related to blood clotting. Auscultation indicated that his lungs were clear bilaterally (normal breath sounds over the dorsal pulmonary fields).

One week after first presentation, the mountain guide presented again after a skiing accident (a minor fall while breaking in new ski boots); he described having strained/pulled muscle tissue in his left leg but said that he had been continuing to work at high altitude (~3500 m) as part of an avalanche security team under conditions of extreme cold, psychological stress, and environmental risk (navigating an unmarked track). He commented on breathlessness, general (occupation-related) anxiety, and left calf discomfort that became more apparent when he removed his ski boot. At this point minor swelling (left calf <2 cm larger than asymptomatic right calf) was observed and the injury was diagnosed as a muscle tear; the mountain guide was advised to rest.

Five days later the mountain guide again presented to his physician with the same complaints (chronic calf pain and shortness of breath). He commented on feeling physically and psychologically exhausted and stated that he was planning to spend time with his family. Osteopathic articulations of the joints in his lower extremities and back, lymphatic drainage, and massage of the lower extremities were performed. After resting for 2 days after the osteopathic interventions, the mountain guide returned to work (altitudes of 2000–2500 m) for 1 more day before descending (seated car travel for a distance of

65 km) to a lowland area. After his descent from altitude, the mountain guide developed severe thoracic and pleuritic pain, a cough with bloody sputum, and cramp-like sensations in his left shoulder, left vertebral, and lumbar regions. He presented at his doctor's surgery and was immediately referred for a thoracic computed tomography scan, which revealed a paracentral and peripheral pulmonary embolism in the right and left lower lung lobes with infarction pneumonia (ground-glass infiltration). He was admitted to the hospital and initiated onto anticoagulation therapy (rivaroxaban) for a minimum of 6 months.

While in the hospital, the mountain guide experienced thoracic pain that worsened upon deep inhalation, hemoptysis, and low-grade fever (37.9°C). Standard blood profiling (full blood count, liver, renal and electrolytes, blood glucose, hemoglobin) and coagulation screens were unremarkable, but mild homocysteinemia (19.9 µmol/L) was noted. Subsequent tests revealed a raised inflammatory marker (C-reactive protein; 73 mg/L) and a significantly elevated D-dimer (ELISA test; 1.7 µg/mL [normal range 0.1–0.5 µg/mL]). Follow-up duplex ultrasonography of pelvic and leg veins revealed DVT of the left popliteal vein, left peroneal vein, and tibialis posterior vein. At this time, significant calf pain and calf swelling, often indicative of DVT (Table 1; Wells scoring criteria) were absent.² His heart rate was 90 beats/min, below the Wells score threshold (100 beats/min) for suspected PE (Table 2),³ and his blood pressure was normal (128/79 mmHg). Electrocardiography revealed a normal sinus rhythm and heart rate, unobtrusive de- and repolarisation, and normal conduction time; no signs of right heart strain or abnormal

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