

# Deep Vein Thrombosis at High Altitude

Acute mountain sickness high altitude pulmonary oedema (HAPE) and high altitude cerebral oedema (HACE) are common at high altitude but just a few cases of coronary and cerebral thrombosis and phlebitis of limbs have been reported<sup>1</sup>.

A young man who was performing his duties at 17000 ft in northern areas of Pakistan developed progressive painless swelling of his right leg. He was a chronic smoker and did not have any trauma, fever and chest pain. Examination revealed signs of deep vein thrombosis in his right leg. Rest of systemic examination was unremarkable.

He was descended within next forty-eight hours of incident to Rawalpindi at 2000 ft. above sea level. The investigations revealed following:

Haemoglobin- 14.8g/dl, platelets - 190x 10<sup>9</sup> per liter while total leukocytes count, ECG, X-rays chest, bleeding time, clotting time, PT, PTTK were within normal limits. Lung scan was also normal. Doppler duplex ultrasound and radioisotope scanning of right leg revealed a large thrombus in popliteal artery extending up to femoral artery. Blood lupus anticoagulant was positive while anticardiolipin antibody, factor V Leiden and protein C levels were within normal limits. Inj. heparin 1000 unit per hour was given by continuous infusion for 4 days. Concurrently oral warfarin 10 mg per day was started before stopping heparin. Within one week swelling of the leg resolved and he became hilly mobile. He was advised to continue warfarin for next four months.

The patient was healthy but developed deep vein thrombosis when he went to high altitude. Various factors attribute to the hypercoagulability at high altitude, In this case smoking, decreased physical activity, coagulation disparities and probably dehydration are important. Decreased physical activity and dehydration lead to stasis of blood and hence cause tendency to coagulate. Coagulation enhancement may also result from abnormal platelets with increased adhesiveness<sup>2</sup>, red cell anisocytosis<sup>3</sup> and polythermia<sup>4</sup> leading to microthrombi which initiate progressive coagulation cascade. Thromboembolism manifests itself in various ways like cerebrovascular accident<sup>5</sup> pulmonary embolism<sup>6</sup> and deep vein thrombosis. The role of microthrombi in acute mountain sickness has also been theoretically suggested. The successful medical treatment results from the fact that all disorders of coagulation return to normal on descent to lower heights i.e. less than 10000 ft (ideally sea levels) and early anticoagulant therapy with adequate mobilization.

This is to highlight the role of high altitude as a factor leading to tendency towards hypercoagulability. Management of cases of thrombosis occurring at high altitude depends on rapid evacuation to lower heights and early anticoagulant therapy.

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## References

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