Splinter Haemorrhages at High Altitude— A Case Report

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Abstract

HAEMORRHAGES and thrombotic phenomenon have been reported to occur at high altitudes. Though a larger number of cases have been reported on oral, retinal and gastric haemorrhages and thrombotic episodes, splinter haemorrhages in the nail beds have not been reported frequently. A case of splinter haemorrhage occurring in a mountaineer at altitude between 18,000 feet and 20,000 feet has been presented in this paper. Various possible explanations like alterations in clotting mechanism and haematocrit values, effects of cold, hypoxia, etc. for causation of such splinter haemorrhages have been discussed. The frequency of such haemorrhages in various groups of people at high altitude suggests that their aetiology and clinical significance need further investigation.

Introduction

Haemorrhages and thrombotic phenomenon have been reported to occur at high altitude. A large number of cases have been reported of oral, retinal and gastric haemorrhages. Splinter haemorrhages in nail beds have not been reported frequently.

These splinter haemorrhages have been associated with many pathological conditions. Horder[4] drew attention to their association with subacute bacterial endocarditis (SBE). Two alternative hypothesis have been advanced to explain their appearance. Lewis[6] and White[10] stated that their presence is due to an increased capillary fragility in SBE. Others[3] believe them to be embolic in origin. By 1950 Wood[11] had come to the conclusion that these are not diagnostic of bacterial endocarditis. Platt and Greaves[8] found them in 44% of patients with uncomplicated mitral stenosis, 18% of other patients and with some frequency in apparently healthy individuals and suggested that their presence may be due to factors unconnected with the disease. Gross and Tall[3] maintain that they may be associated but are not diagnostic of the SBE as they are seen secondary to trauma, trichinosis, rheumatic fever and infectious mononucleosis. Donald and William[2] reported their presence in Indian population in the Andes and more frequently in patients with Monge's disease. Rennie[9] reported appearance of splinter haemorrhages in him and 15 other climbers at 5880 M during an expedition to Dhaaulagiri in Nepal. They appeared spontaneously without any associated symptoms. This case report refers to splinter haemorrhages in a climber between 15,000 ft and 20,000 ft.

Case Report

The subject (author) was a member of the Indian Air Force Expedition to Bharte Khunta (21,580 ft) in the Garhwal Himalayas in 1978. He was fully acclimatised to altitude having spent four weeks in Sikkim Himalayas training at altitudes upto 17,500 ft. Thereafter he had spent 10 days in the plains making pre-expedition preparations.

The ascent to altitude was gradual, reaching 6500 ft by road in 3 days. Rest of the climb was on foot; from 6500 ft to 10,300 ft (21 Km) in one day; from 10,300 ft to 12,700 ft (17 Km) in one day; stay at 12,700 feet for four days doing two ferries upto base camp (BC) at 14,280 feet for acclimatisation; stay at BC for two days doing one ferry to Advance Base Camp (ABC) at 15,500 ft; stay at ABC for three days doing one ferry with 30 lb load to Camp I

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(19,500 ft); move from Camp I to Camp II (20,500 ft) in one day; assault on peak (21,580 ft) next morning and return to Camp I after packing up Camp II the same day. Splinter haemorrhages were noticed in both the hands at ABC (15,500 ft) after a stay of two days. These were linear dark red in colour at the distal part of the nails and the nails were tender on pressure. A further crop of such haemorrhages occurred at Camp II (20,500 ft). There was no echymosis or haemorrhages on the mucous membranes of the oral cavity or the conjunctivae. Hess test was done at ABC and it did not show any evidence of increased capillary fragility. General clinical examination did not reveal any abnormality. Blood was examined a week later which showed Hb 17.0 gm%. Cell counts were normal. By about two to three weeks the haemorrhages grew out of the nails. Separation of nails from the nail bed was seen involving ⅓ of the distal part of the nail. The nails became normal by about eight weeks.

Discussion

The causation of splinter haemorrhages at high altitude is not very clear. Many explanations have been offered. Physical exertion, increased haematocrit value, cold, malnutrition and hypoxia have been suggested but all are disputable.

In the present case haemorrhages occurred after six weeks of heavy exertion and four weeks of this was above 12,000 ft. In cases reported by Rennie[9] haemorrhages occurred five weeks after heavy exertion a month of which was above 3660 M. The haemorrhages were not seen in other climbers and porters who accompanied the team. They were carrying more load (upto 40 lbs) and went along the climbers upto an altitude of 20,000 ft.

Malnutrition has been suggested as a cause by Rennie[9], but it may be discounted as the team was well supported by porters and helicopter supplies upto ABC (15,500 ft). Anorexia due to mountain sickness was of too short a duration to affect nutrition.

Polycythemia has also been blamed as shown by increased frequency of these haemorrhages in Andean Indians and more so in patients with Monge's disease where a haemoglobin level of 22–27 gm% and haematocrit values of 73–86% have been seen[2].

Increased viscosity of blood due to increased haematocrit values and dehydration due to low atmospheric humidity and enforced immobilisation due to bad weather etc. may increase the chances of thrombosis in large vessels. Microemboli from these may get detached and lodge themselves at the distal part of the nails where the capillaries form the loops. This may be facilitated due to exposure to cold which may further slow down the circulation in the region.

No definite single factor can be pin-pointed as the cause of such splinter haemorrhages. More than one might have been operative in their appearance. The frequency of such haemorrhages in various groups of people at high altitude suggest that their aetiology and clinical significance need further investigation.

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