



## High Altitude Retinopathy Following Mount Everest (8848.86m) Ascend without Supplementary Oxygen: A Case Report

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### Abstract

High altitude retinopathy is an ocular condition resulting as a manifestation of high altitude sickness mostly due to hypoxia. It is a rare presentation mostly seen in mountaineers and high altitude trekkers. We document the case of 36-years old male from Kuwait who successfully ascended the highest mountain in the world (Mt. Everest) without supplementary oxygen but suffered significant loss of central vision in both of his eyes.

**Keywords:** Retina; Retinopathy; High Altitude; Vision Loss; Hypoxia

### Abbreviations

HAR: High Altitude Retinopathy; HAI: High Altitude Illness; IOP: Intra Ocular Pressure; AMS: Acute Mountain Sickness; OCT: Optical Coherence Tomography; HIV: Human Immunodeficiency Virus; HCV: Hepatitis C Virus; VDRL: Venereal Disease Research Laboratory; HBsAG: Hepatitis B Surface Antigen; CRP: C-Reactive Protein

### Introduction

High Altitude Retinopathy (HAR) is considered to be one of the four clinical manifestations of High Altitude Illness (HAI) which includes acute mountain sickness, high altitude cerebral edema and high altitude pulmonary edema. HAI usually occurs with travel to elevations above 2500m (~8200 feet) and it is generally manifested by symptoms of headache, nausea, blurry vision and it may affect the lungs, brain and the eyes [1]. The acute ocular signs of HAR involve intra and pre-retinal hemorrhages, peripapillary hyperemia and dilated vessels generally occurring in individuals who are exposed to high altitudes above 3,000 meters [2].

According to the severity of ocular complications, Weidman and Tabin [3] have classified HAR in four grades. Grade I involves dilated retinal veins with hemorrhages within one disc diameter area. Grade II is characterized by moderate dilated retinal veins with

hemorrhages in 2 disc diameter areas. Grade III includes advanced dilated retinal veins with hemorrhage in 3 disc diameter area and paramacular and vitreous hemorrhage. Grade IV constitutes engorged retinal veins with hemorrhages greater than three disc diameter areas, macular and vitreous hemorrhage or papilloedema.

The etiopathogenesis of HAR is unknown. However, it is suggested that, like other oxygen-dependent retinal diseases (e.g., von Hippel-Lindau disease, retinopathy of prematurity, proliferative diabetic retinopathy, and glaucoma), factors such as heterodimeric transcription and hypoxia-inducible factor could be the mediator for the changes in HAR [4].

### Case Report

We report a case of bilateral High Altitude Retinopathy (HAR) with Pre-retinal hemorrhage and macular edema of a 36-year-old male who successfully ascended the highest mountain in the world (Mt. Everest 8,848.86 meters) without supplementary oxygen. He presented to us with a complaint of loss of central vision associated with floaters in both of his eyes. On detailed history taking, he revealed that he joined the record list for climbing Mt. Everest without supplementary oxygen. He denied the history of using any medications for any other conditions and he had no any past his-

tory of ocular interventions. He didn't have systemic symptoms like headache, nausea, vomiting or shortness of breath.

On examination, the best corrected visual acuity was 20/80 in the right eye and 20/200 in the left eye. Intra Ocular Pressure (IOP) was 15mmHg in both eyes. Anterior segment findings of both eyes were within normal limits while examining with slit-lamp biomicroscopy.

Color fundus photographs were taken which showed bilateral pre-macular hemorrhages, Roth spots and peri-papillary hemorrhages as shown in Figure 1A and 1B. Macular Optical Coherence Tomography (OCT) was performed and it revealed macular edema in both eyes as shown in Figure 2A and 2B.



Figure 1a: Fundus Photography of Right Eye.



Figure 1b: Fundus Photography of Left Eye.

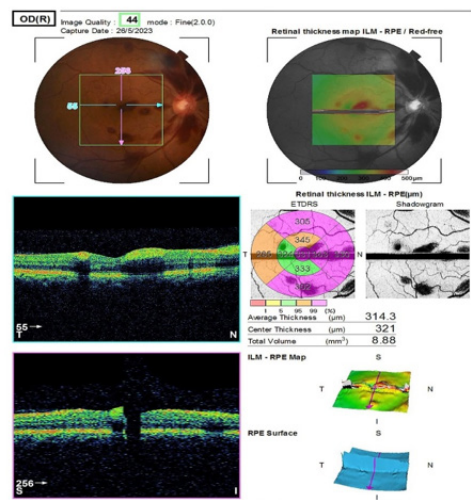


Figure 2a: Macula OCT RE.

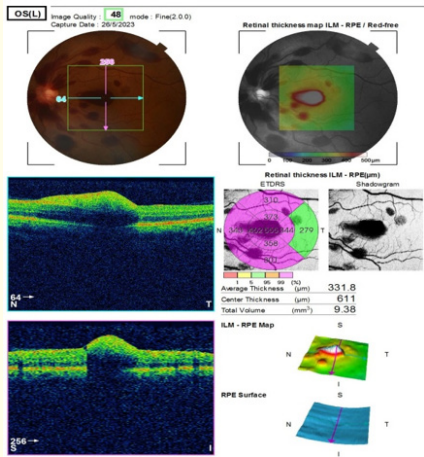


Figure 2b: Macula OCT LE.

Blood pressure at the time of presentation was 130/90mmHg. Systemic investigations such as complete blood count, haemoglobin, sugar, creatinine, urea, lipids, CRP and serology for HIV, HCV, VDRL and HBsAg were within normal limits. The tuberculin test and chest X-ray also appeared to be within normal limits. Physician consultation didn't reveal any abnormalities.

Conservative treatment with Carboxy Methyl Cellulose eye drops (Evatears- Everest Parenterals, Nepal) was given and follow-up visit after 3 months was advised. On examination after 3 months, the patient had the visual acuity of 20/20 in both the eyes and posterior segment examination revealed no traces of hemorrhage.

## Discussion

Various vascular compensatory mechanisms and pathological responses induced by hypobaric hypoxia, and potentially exacerbated by physical exertion and Valsalva effects, are believed to cause physiological and pathological retinal changes observed in individuals who ascend to high altitudes [5]. The signs of HAR were first described by Singh [6] in 1969 which includes dilated retinal veins, pre and intra-retinal hemorrhage, papillary hemorrhage, peri-papillary hyperemia and papilloedema. Cotton wool spots, Roth spots and retinal vein occlusions have also been reported [7].

Barthelemy, *et al.* [8] published a case series of 28 patients, who underwent fundus photography along their ascent and descent of Mt. Austagh Ata (24, 751 ft). Few retinal hemorrhages were noted during the ascent. However, retinal hemorrhages increased during the descent. In this study, 79% of patients exhibited retinal hemorrhages, most of which were detected after return to base camp from a high altitude. Significant correlations with decreased oxygen saturation and increased hematocrit were noted. Due to the delayed appearance, the authors mentioned that retinal hemorrhages should not be considered a warning sign of impending high-altitude illness. This finding is consistent with Singh, *et al.* observational study, which demonstrated a time lag of around six to 96 hours between arrival and onset of symptoms of Acute Mountain Sickness (AMS) [6]. The time lag between arrival of high altitude and onset of signs and/or symptoms suggest HAR or AMS may not be directly or solely related to hypoxia.

The majority of cases of HAR resolve spontaneously with no long-term adverse visual outcomes [7,8]. No specific therapies for HAR have been proven, although it may be necessary to treat the acute manifestations of altitude illness or the chronic ocular complications of the condition. Medications such as NSAIDs, corticosteroids and acetazolamide have been shown to have no impact on retinal hemorrhages [7]. The use of furosemide is inconclusive [9].

Frayser, *et al.* [9] delineated retinal hemorrhages in nine out of twenty-five climbers ascending 5,335 meters on Mt. Logan. However, there was no relation between retinal hemorrhage and speed of ascent. In another study, retinal hemorrhages accounted for 36% of 39 individuals climbing above 4,329 m on Mt. McKinley [10]. Here, they found a positive correlation between the occurrence of retinal hemorrhage and the severity of headache. They also discovered that individuals with a history of migraine, increased physical exertion, and rapid ascent are more prone to developing retinal hemorrhages.

Retinal hemorrhages from capillary bursts or leakage from the arterial side of the retinal vascular bed are a predominant pathological finding in HAR [8]. These hemorrhages may be punctate or diffuse, intra-retinal or pre-retinal, and peripheral or central. The cause of the bleeding is still not clear but one consideration is that low oxygen from shunting of blood or hypoperfused capillary beds stimulates expression of factors that increase vascular permeability, such as VEGF.

## Conclusion

Individuals willing to ascend high altitudes have a high risk of developing HAR. Necessary precautions have to be taken prior to climbing higher altitudes. Detailed eye examination should be carried out if any vision related problem arises.

## Conflict of Interest

None.

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