INTRODUCTION

High altitude retinopathy (HAR) is an acquired vascular retinopathy, characterised by dilated veins, intraretinal and preretinal hemorrhages, usually not affecting the vision until and unless macula is involved. Sometimes cotton wool spots, optic disc edema and hyperemia may also be associated. Travelling to altitudes of 2500m or more put people at risk of HAR but typical picture appears usually above 16000 ft. (approximately 4900m).

High altitude illness apart from HAR comprises of wider range of diseases like acute mountain sickness (AMS), snow blindness (photo keratitis), high altitude pulmonary edema (HAPE) and high altitude cerebral edema (HACE). The last two conditions are potentially life threatening. Before we discuss about HAR a brief mention of these conditions becomes important.

Acute mountain sickness (AMS) is similar to a hangover that resolves usually within 12-15 hrs. of stopping ascent or maximum of 4 days. The climber feels symptoms of headache, fatigue, nausea vomiting, anorexia, dizziness, decrease in night vision and sleep disturbances. Mild tachycardia and peripheral edema may be associated. On the basis of these signs and symptoms, Lake Louise Score of AMS has been made: A total score of 3 or more indicates mild AMS while more than 6 is severe type. AMS usually precede HACE which occurs due to cerebral edema resulting in symptoms of focal neurological deficits, confusion, ataxia, seizures diplopia, hallucinations etc. and if descent is not stopped immediately then patient might land up in coma and might as well die. Another illness is HAPE which may not be preceded by AMS which occurs due to extravasation of fluid from intravascular to extravascular spaces in lungs. It is characterised by dyspnea, dry cough, hemoptysis and decreased exercise tolerance. Sometimes fever, tachycardia tachypnea and crepts are present. Apart from these conditions, high altitude pharyngitis and bronchitis have also been reported.

HIGH ALTITUDE RETINOPATHY (HAR)

Etiopathogenesis

The mechanism of HAR remains largely unknown, though it has been hypothesized that decreased arterial oxygen results in inadequate autoregulation of retinal circulation eventually causing vascular incompetence.

Dr Gabriel William and colleagues, university of Tubingen, Germany have reported that widespread retinal capillary leakage plays important role in pathogenesis of HAR. A Fundus Fluorescein Angiography (FFA) study of 14 healthy, unacclimatised participants with average age of 35 yrs. was done at 1119ft as baseline and after ascent to 14957 ft. within 24 hrs. FFA was repeated more than 2 weeks after return as a part of Tubingen High Altitude Ophthalmology Study (THAO). Researchers found no abnormality at baseline, however marked leakage of peripheral retinal vessels in 50% of cases which reversed after descent.

Given time, humans are able to acclimatise to higher altitudes as high as 4000 -5000m by adapting following mechanisms -

1. Hyperventilation (via carotid body hypoxic ventilator response)

2. Increased red blood cell production (via erythropoietin).

3. Increased vascularity of lungs and other tissues.

4. Increased tissue mitochondria

5. Suppression of ADH and Aldosterone

These parameters of acclimatization are compromised in ascending to extended heights like above 5000 m resulting in high altitude sickness. The range of height above 8000m is considered as death zone.
The chances of HAR increase with following risk factors:

1. Rapid ascent
2. Climbing to higher altitudes
3. Sleeping at higher altitudes
4. Continued ascent even after symptoms of AMS appear
5. Past history of HAR
6. History of upper respiratory tract infection, chronic bronchitis, asthma etc.
7. Age more than 50 years
8. Individual susceptibility
9. Past history of neck surgery or irradiation.
10. Any associated diabetic or hypertensive retinopathy

Snow blindness occurs due to UV light exposure at high altitude as there is around 5% increase in UV rays per 300 m gain in height. The symptoms of corneal burns are noted within 6-12 hrs. in form of pain photophobia, watering and blepharospasm.

**Clinical picture**

HAR is characterised by dilated and tortuous veins, widespread retinal hemorrhages (FIG.1 and FIG 2), cottonwool spots (cws) and sometimes disc edema & hyperemia. These features disappear automatically after descent and do not cause any visual symptoms until fovea is involved. The intelligent patient may appreciate central or paracentral scotoma. Though the retinopathy runs a benign course and carries good prognosis its presence should alert the physician the possibility of cerebral edema.

HARH is both a significant component and predictor of high altitude illness. Studies have shown a significant correlation exists between HARH and symptoms of AMS. HARH resulting in diminution of vision should be a contraindication to further ascent as the climber might enter into more severe conditions like full blown HAR involving the macula threatening vision or potentially fatal HACE.

Optic disc edema (ODE) results due to hypoxia induced brain volume increase, presents as reaction of body to high altitude exposure, fully reversible and has no correlation with AMS.

**INVESTIGATIONS**

FFA shows bilateral leakage of peripheral retinal vessels and blocked fluorescence in areas of hemorrhages. OCT reveals subtle increase in peripapillary RNFL thickness due to optic disc edema quantification of which has no correlation with AMS. Central retinal thickness subject to minor yet statistically significant changes, however again these changes do not correlate with central retinal thickness and AMS.

THAO study has concluded that ERG shows an altered pattern of implicit times of a & b wave of combined rod and cone response.
TREATMENT
As already mentioned HAR resolves spontaneously and does not require any treatment, however systemic altitude illness needs to be treated as per the symptoms. Severe AMS, HAPE and HACE warn of immediate descent and hospitalization. Management includes oxygen, hyperbaric therapy, diuretics, acetazolamide and dexamethasone. Role of nifedipine as a vasodilator has been proven. Ginkgo biloba extract given to climbers before rising to high ascent reduce chances of AMS as compared to acetazolamide.

References
1  Arch Ophthalmol. 2008 May; 126(5):644-50
2  Weidman M, Tabin GC. High Altitude Retinopathy and Altitude Illness Ophthalmology 1999; 106(10). 1924-26