High Altitude Retinopathy in Mountain Climbers

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Retinal hemorrhages appear to be a frequent, though often unappreciated, occurrence in unacclimated climbers experiencing prolonged exposure to altitudes above approximately 3,658 meters (12,000 ft), heights frequently attained by American mountaineers. This condition has not received attention in the ophthalmologic literature, though several reports of retinal and vitreous hemorrhages have appeared in nonophthalmologic journals.

Of six surviving members of a climbing expedition of Mt. Aconcagua, four had retinal hemorrhages. Two had permanently disturbed vision with paracentral scotomas plotted on a tangent screen.

Mountain climbing is one of man's more hazardous pursuits. While certain medical problems were recognized early, the full scope of high-altitude abnormalities has been disclosed only in recent years. Though occurring less frequently, pulmonary edema and cerebral edema are not rare disorders in mountaineers.

We wish to call attention to an infrequently cited, but commonly occurring, ocular insult resulting from ascent to high altitude, retinal hemorrhages. We also report that although the hemorrhages clear, this retinopathy may not be transient and innocuous. Scotomas may persist with permanent impairment of vision. Six survivors of an ill-fated climb of Mt. Aconcagua (Argentina) were examined. Four survivors had retinal hemorrhages. In this communication, we will tell the story of the climb, demonstrate the characteristics of the hemorrhages, and discuss the pathogenetic mechanisms that might produce such an altitude-induced retinopathy.

REPORT OF CASES

Eight members of an Oregon-based mountaineering club planned to climb Mt. Aconcagua in the Argentine Andes. This mountain, 6,921 meters (22,834 ft) in height, is the tallest in the western hemisphere. Though of varied occupational and geographic backgrounds (Table), the climbers were experienced, each having climbed peaks taller than 5,488 meters (18,000 ft) without complications. All were in excellent physical condition, without
known illnesses or history of migraine.

The party flew from the United States on Jan 13, 1973, arriving the same day in Buenos Aires, Argentina. One of their 26 pieces of baggage did not arrive with them—the oxygen. Undaunted, as the oxygen was intended for emergency use only, the group flew to Mendoza (elevation, 762 meters [2,500 ft]) on Jan 15, 1973. Two days were spent clearing procedural aspects and making arrangements for mules, supplies, and an Argentine guide. While in Mendoza, all climbers began a three-day course of acetazolamide (250 mg every eight hours), and then traveled by bus to Punta de Vacas (elevation, 2,332 meters [7,650 ft]) where the following day was spent sightseeing on the Chile-Argentina border (elevation, 3,201 meters [10,500 ft]).

On Jan 20, 1973, eight days after leaving the United States and six days after leaving lower altitudes, the actual climb began. The route paralleled that successfully negotiated by a Polish group in 1934[10] (Fig 1). A two-day, 65-km (40-mile) hike with light packs followed, at the end of which the climbers established a base camp (elevation, 4,115 meters [13,500 ft]) at the foot of the east face of Mt. Aconcagua.

Days 10 through 12 were spent packaging supplies and climbing to camp 1 at 4,725 meters (15,500 ft). Climbing was particularly difficult in this section because of fields of "nieves penitentes," stalagmite-like snow formations, often shoulder-deep and unyielding.

On days 13 through 14, the party ascended to camp 2 (5,366 meters [17,600 ft]). At this point, three climbers dropped out, one with pulmonary edema (a lawyer), one with suspected cerebral edema (a student), and one of the physician-climbers (an internist) to look after the two stricken members.

The five remaining climbers and the Argentine guide ascended to camp 3 (5,915 meters [19,400 ft]). The assault on the summit began on the 17th day, but one climber (a psychiatrist) soon was in difficulty. He was unable to apply his crampons and was grossly disoriented. As he descended with the guide to base camp, he developed a mild hemiparesis on the right and ataxia, which cleared at lower altitude.

The four remaining climbers (a teacher, a NASA engineer, a farmer, and a policeman) attempted the ascent to the summit. That night they bivouaced at 6,402 meters (21,000 ft). What transpired above this altitude is open to doubt, as only two of the climbers survived and their recall of the events is fragmentary.

The next morning (day 18), when he began to experience symptoms of acute altitude sickness, the NASA engineer elected to return to camp 3. To his companions, he seemed oriented and capable of making the descent unaided, but his body was later found a short distance above camp 3.

The remaining three climbers continued. After surmounting an ice fall above 6,402 meters, they unroped to ascend the final dome in deep snow. As night came, they were still 60 meters (200 ft) shy of the summit. The climb was abandoned because the teacher was lethargic, hallucinating, and refusing to go further. An attempt was made to descend immediately but they became disoriented and instead bivouaced at 6,890 meters (22,600 ft).

The following morning, the farmer descended to camp 3, leaving the teacher and policeman to follow at a more cautious pace. During descent, the latter two fell down a slope 300 vertical meters (1,000 ft) coming to rest about a half kilometer above camp 3. The policeman continued the descent. The teacher was to follow but never arrived.

The two survivors of the summit attempt arrived in base camp, still hallucinating. They had seen pieces of highway equipment on the summit ridge, dead mules at camp 3, skiers at 6,400 meters, and trees high on the mountain. In fact, both were unaware of their companions' fates, having presumed that both had been rescued by the Argentine mountain patrol, whose presence and voices they had hallucinated.

As he flew back to the United States, the internist noted paracentral scotomas while reading. Soon after his return, examina-

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**Data in Eight American Climbers of Mount Aconcagua**

<table>
<thead>
<tr>
<th>Patient/ Age, yr/ Sex</th>
<th>Occupation</th>
<th>Home City</th>
<th>Maximum Altitude Attained, Meters (ft)</th>
<th>Hemor- rages</th>
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<td>Denver, Colo</td>
<td>6,890 (22,600)</td>
<td>Died</td>
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</tbody>
</table>

* Before fundi could be inspected for hemorrhages.

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**Fig 1.—Selected route of climb followed by which had been successfully negotiated by Polish expedition in 1934.**
tion by Felix Sabates, MD, disclosed a visual acuity of 20/15 in each eye. Scattered intraretinal and nerve fiber-layer hemorrhages were present in both eyes (Fig 2). Though the retinal hemorrhages cleared over several months, small bilateral paracentral scotomas were plotted on a tangent-screen examination more than one year after descent.

We examined all of the remaining survivors within two weeks of their return to this country. All had visual acuities correctable to 20/15 in each eye. Three had retinal hemorrhages, and two of those with retinopathy had paracentral scotomas on Amsler-grid and tangent-screen testing. The psychiatrist experienced the most severe retinopathy (Fig 3).

In addition to the flame-shaped and thumb-print hemorrhages seen in the internist, he had preretal hemorrhages. No definite perivascular distribution was discernible. The policeman and dairy farmer had ascended to the greatest altitude, but showed a less severe hemorrhagic retinopathy (Fig 4, left eye) than either of the physicians (Table). No nerve fiber layer infarcts or vitreous hemorrhages were noted in any of the climbers, nor was papilledema present at the time of our examinations. The hemorrhages cleared in all individuals within two to three months; however, the psychiatrist still has dense paracentral scotomas in the right eye that have persisted unchanged for over one year.

**COMMENT**

Acute mountain sickness ("puna" or "soroche") is becoming more prevalent in today's mobile society. Rapid ascent from low to high altitude is commonplace among skiers and mountaineers and has introduced many of them to the symptoms of headache, lassitude, insomnia, and gastrointestinal upset that characterize this entity. Some persons are affected at altitudes as low as 2,439 meters (8,000 ft) and most will show at least some symptoms by 4,575 meters (15,000 ft) of elevation. The symptoms usually begin within 8 to 24 hours of arrival at the altitude, and in most cases, clear over a four- to eight-day period. Occasionally, progression to pulmonary edema, cerebral edema, and death occurs. In the more severe cases, a timely descent to a lower altitude is curative.

Though theories abound, the pathophysiological nature of this disease is largely unknown. Slow ascent, allowing adequate time for acclimatization, is the best preventive. Physical conditioning does not seem to offer protection, although acetazolamide taken for a 48-hour period prior to making a climb appears to be beneficial.

Ocular hemorrhages appear to be a frequent but often unrecognized accompaniment of acute mountain sickness. Four previous reports of retinal hemorrhages and two of vitreous hemorrhages at high altitude have appeared. Frayer et al. found retinal hemorrhages in nine of 25 individuals who ascended to 5,385 meters (17,500 ft) on Mt. Logan in the Yukon territory. No relationship between retinal hemorrhage and concomitant symptoms of acute mountain sickness or speed of ascent was noted. Houston reported retinal hemorrhages in two of 99 climbers on Mt. Rainier (elevation, 4,393 meters [14,410 ft]). In his study, 0.6% of climbers had severe eye symptoms (type unspecified), while 1.8% had some eye "troubles."

A very thorough study of high-altitude retinopathy is that of Schumacher and Petajan, who found retinal hemorrhages in 36% of 39 subjects at altitudes above 4,329 meters (14,200 ft) on Mt. McKinley. They discovered a positive correlation between the incidence and severity of altitude headache and the incidence of retinal hemorrhage. A prior history of migraine was found to place a climber at higher risk of developing retinal hemorrhages. In their experience, rapid ascent and increased physical exertion appeared to enhance the chance of developing a hemorrhagic retinopathy.

Singh et al. reported three instances of vitreous hemorrhage among a group of 1,925 army troops in the Himalayas working at altitudes of 3,353 to 5,488 meters (11,000 to 18,000 ft). No retinal hemorrhages were seen, although retinal-vein distention and papilledema were noted in some individuals. Wilson, in a recent review of high-altitude illness, cited three anecdotal reports of climbers who had experienced perivascular retinal hemorrhages; one also had a vitreous hemorrhage.

Of our six patients, four had a hemorrhagic retinopathy. In two, it was quite pronounced (Fig 2 and 3) and accompanied by subjective symptoms...
of visual dysfunction. We were unable to define a relationship between severity of altitude headache or prior history of migraine and the incidence of retinopathy. However, our sample is too small to draw any useful conclusion regarding this reported correlation. The hemorrhages were not distributed in any clear-cut perivascular pattern. In fact, some intraretinal hemorrhages occupied areas that were devoid of ophthalmoscopically visible retinal vasculature, suggesting a capillary origin.

Hemorrhages represent one of many ocular effects of high altitude. Additional retinal structural changes include increases in the diameter, tortuosity, and cyanosis of the retinal arteries and veins, as well as cyanosis of the disc. The hypoxia of high altitude induces changes in retinal physiology as well. Retinal blood flow and retinal blood volume are increased, while the mean retinal circulation time decreases.

In addition to changes in retinal hemodynamics, hypoxia produces numerous adjustments in systemic respiratory and circulatory physiology. Cerebral blood flow and cerebral venous pressure increase, the latter effect producing a decrease in cerebrospinal fluid absorption. When coupled with the cerebral edema from hypoxia-induced capillary permeability, increased CSF pressure and papilledema result. Increased retinal venous pressure follows, and the stage is set for a hemorrhagic retinopathy.

Intracocular pressure has been shown to decrease with exercise, potentially increasing the propensity for intraocular bleeding. The extreme physical exertion and concomitant Valsalva maneuvers required in mountain climbing may also play a role. A hypoxic retinal capillary bed already exposed to increased venous pressure and increased blood flow would likely be intolerant of a further Valsalva-induced increase in venous pressure. The clinical appearance of Valsalva hemorrhagic retinopathy shares certain common features with the altitude-induced variety.

Another pathogenetic aspect is the possible effect of blood hyperviscosity. The retinal changes of increased vessel diameter and tortuosity, as well as retinal hemorrhages, have long been known to occur in such states as macroglobulinemia, in which serum viscosity is increased. It has been shown in man that exposure to altitude produces a rather rapid 20% decrease in intravascular serum volume as fluid moves out of the intravascular space into the intracellular space. This results in an increase in hematocrit and blood viscosity that occurs much sooner than that which is due to the hematopoietic response to hypoxia. Whether such an increase in blood viscosity has importance in high-altitude retinopathy is speculative, but it merits consideration as a possible factor.

In trying to define the reasons for the high incidence of hemorrhagic retinopathy seen in our patients, the roles of ozone and rapid ascent must be considered. Conditions in the Andes in January are ideal for the production of relatively high levels of atmospheric ozone. This would serve to further decrease oxygen uptake and potentiate the already existent tissue hypoxia and the effects thereof. Houston has recommended a rate of ascent not exceeding 305 meters (1,000 ft) per day below elevations of 4,268 meters (14,000 ft) and 152 meters (500 ft) per day when climbing from 4,268 to 5,488 meters (14,000 to...
18,000 ft). Above heights of 5,488 meters (18,000 ft), no firm guidelines exist. Our patients greatly exceeded these recommendations, often ascending more than 610 meters (2,000 ft) in a single day. Such a rapid climb did not allow adequate time for acclimatization and enhanced the likelihood of damage from hypoxia.

Unquestionably, man is capable of adapting to great heights. Some permanent Andean mining villages are situated above 5,183 meters (17,000 ft). Himalayan expeditions have climbed above 8,537 meters (28,000 ft) without the use of supplemental oxygen. However, such feats require prolonged acclimatization periods at moderately high levels (4,573 to 5,183 meters [15,000 to 17,000 ft]). The possible protective effect of supplemental oxygen on climbs of the magnitude considered here has not been explored, and one can only speculate on how its use might have altered the outcome.

The factors that might lead to the production of high-altitude retinopathy are numerous and complex (Fig 5), and our knowledge is presently incomplete. However, with the increasing popularity of high-altitude pursuits, the prevalence of this aspect of ascent to high-altitude pursuits, the prevalence of this aspect of ascent to high places is certain to increase, and with it, our understanding of its pathogenesis and prevention. Certainly, ophthalmologists should be aware of this condition, which is capable of producing a substantial retinopathy in healthy, young, athletic people.

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References


Arch Environ Health, to be published.