Lipid Subhyaloid Maculopathy and Exposure to High Altitude

Roberta Rosas Petrocinio; Elga Dias Gomes

BACKGROUND: High altitude retinopathy (HAR) includes a number of diseases related to high altitude such as acute mountain sickness (AMS), high altitude pulmonary edema (HAPE) and high altitude cerebral edema (HACE). High altitude retinopathy is mainly characterized by retinal hemorrhages, usually sparing the macular region, a condition specifically known as high altitude retinal hemorrhages (HARH). The pathogenesis of HARH is unclear. Many studies show that lack of oxygen causes an inadequate autoregulation of retinal circulation, causing vascular incompetence. Other retinal changes described in HAR have been reported, such as optical disk edema, optic disc hyperemia, cotton wool exudates, venous occlusions, and macular edema.

CASE REPORT: In this paper we present a case of an aviator who developed a unilateral maculopathy through subhyaloid lipid accumulation on a climb to the top of Mt. Everest. The clinical findings are suggestive of an apparent case of temporary altitude-induced visual disruption maybe by the same presumable pathogenesis of HARH. Right eye visual loss was perceived at 5150 m when he was trying to take a photograph 40 d into the expedition.

DISCUSSION: The maculopathy developed by this patient adds to the discussion on the pathogenesis of HARH, especially the aspect of this maculopathy and its complete resolution. It seems that autoregulation failure could lead to exudation and lipid deposits in the foveal area. Although macular damage is not a common signal in HARH, checking visual acuity during high altitude expeditions remains an important procedure to avoid late diagnosis as unilateral blindness may not be detected early.

KEYWORDS: retinal diseases, hypoxia, retinal hemorrhage, altitude sickness.

Rosas Petrocinio R, Gomes ED. *Lipid subhyaloid maculopathy and exposure to high altitude.* Aerosp Med Hum Perform. 2016; 87(10):898–900.

High altitude retinopathy (HAR) refers to a broad spectrum of retinal disorders, including dilated retinal veins and arteries, increased tortuosity of the retinal vessels, venous occlusions, diffuse or punctate retinal hemorrhages, vitreous hemorrhages, papillary hemorrhages, cotton wool spots, papillary hyperemia, and papilledema associated with exposure to prolonged hypobaric hypoxia at high altitude.[6,8,10,12] It is mainly characterized by retinal hemorrhages, usually sparing the macular region, a condition specifically known as high altitude retinal hemorrhages (HARH).[8] HAR was first described by Singh et al. in 1969 as papilledema and vitreous hemorrhages with other clinical findings as part of acute mountain sickness (AMS) and, since then, various articles and case reports have described the presentation of this disease.[9] Apart from HAR there are other diseases related to high altitude such as: AMS, snow blindness caused by photo keratitis, high altitude pulmonary edema (HAPE), and high altitude cerebral edema (HACE).[10]

High altitude retinal hemorrhages appear as "splinter-type" and "flame-type" hemorrhages in the superficial layers of the retina, but hemorrhages in the deeper layers appear dark and rounded or as what are known as "dot-and-blot-type" hemorrhages.[7,11] The changes observed in HAR disappear with reduction in altitude and are normally without sequelae.[6,10,12]

The pathogenesis of HARH is unclear. It is believed that lack of oxygen causes an inadequate blood flow and then incompetence of retinal vascular autoregulation.[1,5] The increase in retinal blood flow at high altitude is hypoxia-induced and it

From the Brazilian Air Force Institute of Aerospace Medicine (IMAE), Rio de Janeiro, Brazil.

This manuscript was received for review in February 2016. It was accepted for publication in June 2016.

Address correspondence to: Roberta Rosas Petrocinio, M.D., Brazilian Air Force Institute of Aerospace Medicine (IMAE), 1200 Marechal Fontenelle Ave., E-019, Campo dos Afonsos, 21740-002 Rio de Janeiro RJ, Brazil; petrociniorrp@fab.mil.br.

DOI: 10.3357/AMHP.4578.2016

is accompanied by retinal vascular dilation. The vessels' dilation leads to a decrease in linear velocity of the blood flow and to an increase of lateral pressure, causing more mechanical stress on the vessel walls. It is possible that the increase in venous back pressure may cause damage to fragile vessel walls due to this vascular distension.[8]

Also, there are some risk factors that increase the chances of HAR: rapid rise, climbing to higher altitudes, sleeping at higher altitudes, continuing to climb even with AMS symptoms, previous history of HAR, history of infection of the upper respiratory tract, chronic bronchitis or asthma, older than 50 yr, individual susceptibility, cervical surgery or irradiation history, and diabetic or hypertensive retinopathy.[10] In this paper we present a case of an aviator who developed a unilateral maculopathy through subhyaloid lipid accumulation on his climb to the top of Mt. Everest. The clinical findings are suggestive of an apparent case of temporary altitude-induced visual disruption, maybe by the same presumable pathogenesis of HARH.

CASE REPORT

A 31-yr-old male aviator, white, and a beginner mountaineer, reported a history of temporary and central visual loss in the right eye (OD) during an expedition through the north side to the top of Mt. Everest during April and May of 2014. The patient identified visual loss at 16,896 ft (5150 m) of level sea and after 40 d of exposure to different levels of altitude during the expedition.

From Kathmandu [4265 ft (1300 m)] in Nepal to Camp 2 [24,606 ft (7500 m)], to avoid altitude illness and ensure acclimatization, a climbing regime was completed, including an overnight stay at the cities and camps reached. The patient set the following ascent schedule: basecamp [16,896 ft (5150 m)], intermediate camp [19,029 ft (5800 m)], advanced camp [20,997 ft (6400 m)], Camp 1 [23,294 ft (7100 m)], and Camp 2 [24,606 ft (7500 m)]. According to the patient, from Camp 1, 90% of the participants were using oxygen, while he just used oxygen against strong winds to avoid frostbite. They then waited for an opportunity to attack the summit from Camp 2. They were predicting a window on May 25, but their sherpas declined to make a new attempt because strong winds compromised their departure.

On May 28, at the basecamp, the patient realized that the vision in OD was blurry when trying to take a picture and returned to Brazil. On June 3, in Brazil, the patient's retinography and fluorescein angiography suggested macular lesion in OD due to subhyaloid lipid accumulation (**Fig. 1A** and **B**). Optical coherence tomography (OCT), done in the same day, showed a superficial hyperechoic lesion in the OD foveal region. It was not possible to assess the underlying retinal lesion, as described on the medical report. The left eye (OS) had normal results in both exams. His visual acuity (VA) at that time was worse than 20/400 in OD and 20/20 in OS.

On June 10, a new OCT showed significant reduction of accumulation. In the next day after a medical examination the patient was restricted from duty involving flying and physical exercises. Visual acuity was unchanged from the last exam. On July 1 new tests found complete recovery of VA in OD (20/20) followed by regression of retinal pathology (**Fig. 2**) and the patient returned to flying without restrictions.

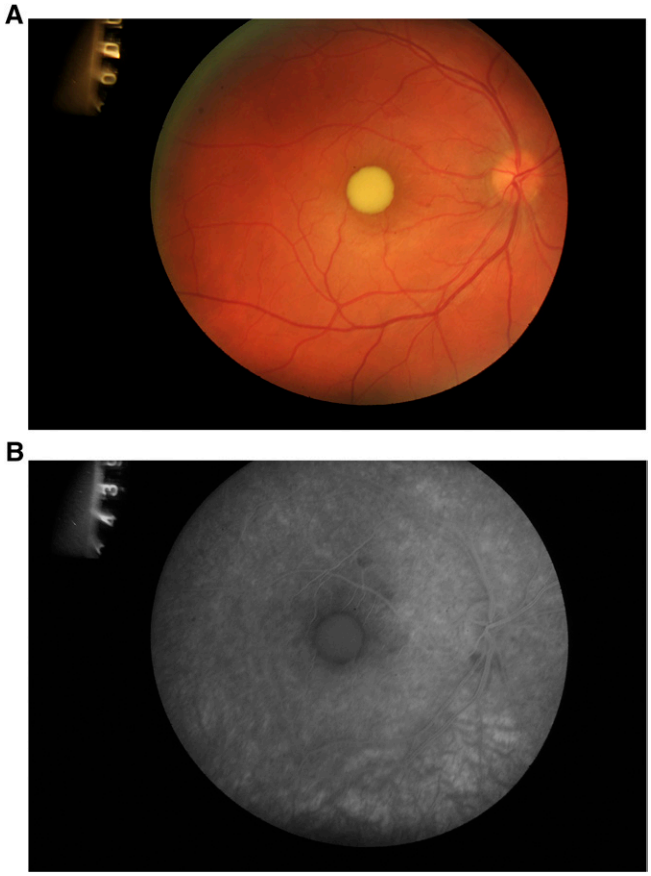

Fig. 1. Retinography showing A) maculopathy in OD suggestive of subhyaloid lipid accumulation in the foveal region (see the online version for color figure: DOI:10.3357/AMHP.4578.2016) and B) fluorescein angiography showing stable fluorescence in OD.

DISCUSSION

The patient is a Captain from the Brazilian Air Force, a helicopter pilot, and has 1500 h of experience and 16 yr on duty. He had already attempted to climb to high altitude two times. In 2013, exactly on February 20, he reached the Lobuche East summit in Nepal. The other one was the attempted climb to the summit of Mt. Everest on April and May of 2014. He was focused on aerobic training, including running and cycling.

The patient presented no common HAR signs as described by earlier works, despite having been exposed to high altitude and long periods without oxygen supplementation. The patient's past medical history was unremarkable. The image led to a differential diagnosis including some retinopathies such as Best disease. Even though they can look similar, the patient's maculopathy disappeared after just a month, while Best disease would progressively develop. Additionally, candidates undergo

Fig. 2. Retinography showing resolution of lipid accumulation in the foveal region in OD (see the online version for color figure: DOI:10.3357/AMHP.4578.2016).

a very difficult selection process for the Brazilian Air Force Academy that includes intellectual, physical, psychological, and medical evaluations.

According to the literature reviewed, HARH occasionally affects the macular region and in most cases there are no complaints of impaired vision.[1,4] It was also noted that the macular edema as a result of exposure to high altitude is a rare condition and was found in two cases in the medical literature: a case of macular edema due to occlusion of a venous branch and a case of cystoid macular edema.[1,6] High altitude retinal hemorrhages are rare below 9843 ft (3000 m). High altitude retinal hemorrhages occur in up to 30% of soldiers at 13,999 ft (4267 m), in 50 to 60% at 17,999 ft (5486 m), and probably in 100% above 22,310 ft (6800 m).[11]

In addition to vascular changes found in hypobaric hypoxia, studies show that physical exercises for acclimatization can overwhelm these retinal vessels, which are already dilated, contributing to the emergence of HARH. Young individuals with excellent physical conditioning as this patient appear to be more susceptible to HARH because of their higher cardiac output and the systemic circulatory strain imposed on the retina during exercise.[2,3] Vigorous exercise and blood pressure surges caused by forced Valsalva maneuvers during active climbing or defecating can also be considered risk factors.[11]

In this case, perhaps retinal vascular autoregulation failure and higher cardiac output from those exercises could have led to exudation from the loss of endothelial integrity and later to lipid deposits in the foveal area of the patient's OD. Although the foveal area has an avascular zone, it is rounded by a very delicate capillary grid and maybe their exudates drifted over the foveal region, causing this maculopathy.

As a Brazilian Air Force pilot, the patient must be submitted to an annual periodic health assessment and, besides that, all Brazilian Air Force pilots undergo physiological training at the Brazilian Air Force Institute of Aerospace Medicine every 4 yr, including a hypoxia test at the hypobaric chamber. In order to prevent further damage and maintain pilot skills, the patient was instructed to obtain regular eye evaluation, as well as the mandatory periodic health assessment, and to avoid exposure to high altitude while practicing mountaineering, especially without oxygen supplementation, because of the risk of developing HAR, HACE, HAPE, or AMS.

The maculopathy developed by this patient contributes to the discussion on the pathogenesis of HARH and will probably bring many questions to the scientific community about the aspect of this maculopathy and its complete resolution. Although macular damage is not a common signal in HARH, checking visual acuity during a high altitude expedition remains an important procedure to avoid late diagnosis, as unilateral blindness may not be detected early.

ACKNOWLEDGMENTS

The authors wish to thank Col. Carlos Frederico Grave Schonhardt for all his guidance and support.

Authors and affiliation: Roberta Rosas Petrocinio, M.D., Ophthalmologist, Aerospace Medicine Specialist, and Elga Dias Gomes, M.D., Aerospace Medicine Specialist, Brazilian Air Force Institute of Aerospace Medicine (IMAE), Rio de Janeiro, Brazil.

REFERENCES

1. Arora R, Jha KN, Sathian B. Retinal changes in various altitude illnesses. Singapore Med J. 2011; 52(9):685–688.
2. Brinchmann-Hansen O, Myhre K, Sandvik L. Retinal vessel responses to exercise and hypoxia before and after high altitude acclimatisation. Eye (Lond). 1989; 3(6):768–776.
3. Brinchmann-Hansen O, Myhre K. Blood pressure, intraocular pressure, and retinal vessels after high altitude mountain exposure. Aviat Space Environ Med. 1989; 60(10, Pt. 1):970–976.
4. Clarke C. Acute mountain sickness: medical problems associated with acute and subacute exposure to hypobaric hypoxia. Postgrad Med J. 2006; 82(973):748–753.
5. Lee KM, Yoo SJ, Woo SJ. Central retinal vein occlusion following hypobaric chamber exposure. Aviat Space Environ Med. 2013; 84(9):986–989.
6. Mishra A, Luthra S, Baranwal VK, Shyamsunder K. Bilateral cystoid macular oedema due to high altitude exposure: an unusual clinical presentation. Med J Armed Forces India. 2013; 69(4):394–397.
7. Müllner-Eidenböck A, Rainer G, Strenn K, Zidek T. High-altitude retinopathy and retinal vascular dysregulation. Eye (Lond). 2000; 14(5): 724–729.
8. Rock PB, Mader TH. Additional medical problems in mountain environments. In: Lounsbury DE, Bellamy RF, Zajtchuk R, editors. Textbooks of military medicine: medical aspects of harsh environments, vol. 2. Washington (DC): Office of The Surgeon General, Borden Institute; 2002:815–853.
9. Singh I, Khanna PK, Srivastava MC, Lal M, Roy SB, Subramanyam CS. Acute mountain sickness. N Engl J Med. 1969; 280(4):175–184.
10. Tyagi R, Mittal S, Gupta M, Arora D. High altitude retinopathy. Uttaranchal Journal of Ophthalmology (UJO). 2013; 8(1):12–14.
11. U.S. Department of the Army Headquarters. Altitude acclimatization and illness management. Washington (DC): Department of the Army; 2010. Bulletin No.: 505.
12. Willmann G, Fischer MD, Schatz A, Schommer K, Gekeler F. Retinal vessel leakage at high altitude [letter]. JAMA. 2013; 309(21):2210–2212.