An Unusual Complication of Barotrauma at Altitude

Marion R. Powell; Leo D. Hurley; Tighe C. Richardson

BACKGROUND: A case of unilateral optic neuropathy secondary to barotrauma following a medial orbital wall fracture is presented.

CASE REPORT: A 41-yr-old U.S. Air Force aviator presented for a routine periodic health assessment. Evaluation uncovered a suspected acquired color deficiency in the right eye (OD). Subsequent discussions with the patient revealed a history of eye pain, redness, and proptosis during a flight overseas several years earlier. Local ocular examination demonstrated asymmetric optic nerve cupping, optic nerve pallor OD, a mild asymmetric color deficit, and a significant visual field defect OD. Evaluation with magnetic resonance imaging revealed findings consistent with an old medial orbital wall fracture OD and optic nerve findings consistent with optic neuropathy. Follow-up evaluation by neurology and otorhinolaryngology demonstrated only extensive sinus pathology. Based upon these findings, it is postulated that the member suffered a medial orbital wall fracture at altitude during ascent caused by expanding ethmoid sinus gases due to abnormal sinus anatomy with subsequent right optic nerve injury leading to an optic neuropathy and subsequent visual sequelae.

DISCUSSION: This case demonstrates one possible complication of active sinus disease while working in the aerospace environment. Several case reports have been published demonstrating the potential link between eye injuries and working in an environment with fluctuating atmospheric pressure. However, literature addressing the specific in-flight environment causing such complications is lacking. Furthermore, the case supports the need for versatility and a broad knowledge base in practicing flight surgeons to evaluate ocular pathology.

KEYWORDS: optic neuropathy, optic nerve, color vision, barotrauma, altitude.

Powell MR, Hurley LD, Richardson TC. *An unusual complication of barotrauma at altitude.* Aerosp Med Hum Perform. 2015; 86(11):994–998.

Sinus block is a routine phenomenon encountered in the aerospace environment. Sinus block occurs when air within the sinus cavity expands or contracts as the atmospheric pressure changes. As atmospheric pressure decreases, air is typically forced out of the sinus cavity as it expands, causing equilibration between the two environments. The opposite holds true during descent, with increasing atmospheric pressure causing a potential negative pressure differential within the sinuses. With abnormal sinus anatomy or function, this movement of gas can be hindered, leading to a sinus block and expansion/contraction of the trapped gas. This block can be seen clinically as the sensation of pain, pressure, dulled hearing, and potentially tympanic membrane rupture. Typically, these events are treated conservatively with no clinically significant outcomes. This case is presented as an unusual and rare potential complication from sinus block at altitude. It serves as a reminder of the difficulties facing local flight surgeons in evaluating aircrew members with what seem like innocuous complaints.

CASE REPORT

A 41-yr-old U.S. Air Force KC-10 boom operator presented for a routine periodic health assessment to his local flight surgeon's office. Color vision testing with the cone contrast test (CCT) revealed borderline green/blue defects in his right eye with significant asymmetry as compared to the left. Review of the patient's medical record revealed the patient had presented with complaints of blurry night vision in his right eye in 2009 with no known cause identified. Also, review disclosed a

From the San Antonio Uniformed Services Health Education Consortium, Ophthalmology Residency, Joint-Base San Antonio, TX, and the U.S. Air Force School of Aerospace Medicine, Aeromedical Consultation Service, Ophthalmology Branch, Wright-Patterson AFB, OH.

This manuscript was received for review in May 2015. It was accepted for publication in August 2015.

Address correspondence to: Marion R. Powell, M.D., 2200 Bergquist Dr., Lackland AFB, TX 78236; fincobbler@hotmail.com.

DOI: 10.3357/AMHP.4359.2015

marginal passing score on the CCT in 2012, again with significant asymmetry between eyes. Following these insights, the member was deemed not fit for flying status and referred to local optometry for further evaluation. Optometry noted a cup-to-disc (C/D) ratio of 0.55 in the right eye (OD) and 0.40 in the left eye (OS) with pigmentary changes in the macula OD. Pupils were documented as normal with no afferent pupillary defect. The remainder of the exam was normal as well. Due to the lack of an etiology for the color vision defect and questionable macular changes seen on exam, a referral was placed for a retina specialist. Recommendation was made for a visual field test to be completed within a month, but the patient moved overseas at that time and was lost to follow-up.

Subsequent examination by ophthalmology disclosed a history of a painful red eye approximately 13 yr prior. The patient recalled the sudden onset of sharp pain and pressure behind his right eye during ascent on a mission overseas. He could not recall the exact altitude when this occurred but believed it to be around FL 250 (25,000 ft/7620 m). The pain peaked at approximately 15 min into the flight and resolved slowly over an hour after lowering the cabin altitude slightly. Following the flight, the pilot stated the patient's right eye appeared red and "looked like it was going to pop out of his head" earlier in the flight. The patient stated that he was seen on the ground following the flight with no abnormal findings and was returned to flying status. He denied any changes in his vision during the episode. The patient denied any chronic medical conditions, although review of his record did show borderline hypertension through the years. He denied any family history of eye disease or blindness. He also denied any history of head trauma or significant facial injuries/fractures. Record review disclosed normal color vision results on admission to the military in 1995 and on several repeat tests using color plates. An optometry exam prior to commissioning in 1998 demonstrated normal results with a C/D ratio of 0.4 bilaterally.

Complete examination by the local ophthalmologist, and later by the ophthalmologist at the Aeromedical Consultation Service, revealed numerous abnormalities. Multiple assessments were made of color vision, with the member passing pseudoisochromatic plate I and II color plate tests. CCT demonstrated a failing blue score OD and FM-100 showed a tritanomalous crossing pattern. These results, in addition to expanded matching ranges on the Moreland anomaloscope, indicated an acquired color deficiency OD. Diurnal intraocular pressures, gonioscopy, contrast sensitivity, and ultrasound pachymetry were normal. The patient's manifest refraction was recorded as $-1.00 +0.50 \times 010$ OD and $-1.00 +0.50 \times 170$ OS, correctable to 20/15 in both eyes. Optical coherence tomography demonstrated significant retinal nerve fiber layer thinning OD with sharp asymmetry compared to the left eye (average retinal nerve fiber layer thickness 64 μm OD and 107 μm OS). Extraocular movements were unrestricted. Pupil exam found a trace afferent pupillary defect OD; otherwise, external and slit lamp exams were normal. Gonioscopy was wide open and normal in both eyes. Automated perimetry showed an infero-nasal visual field defect OD (**Fig. 1**).

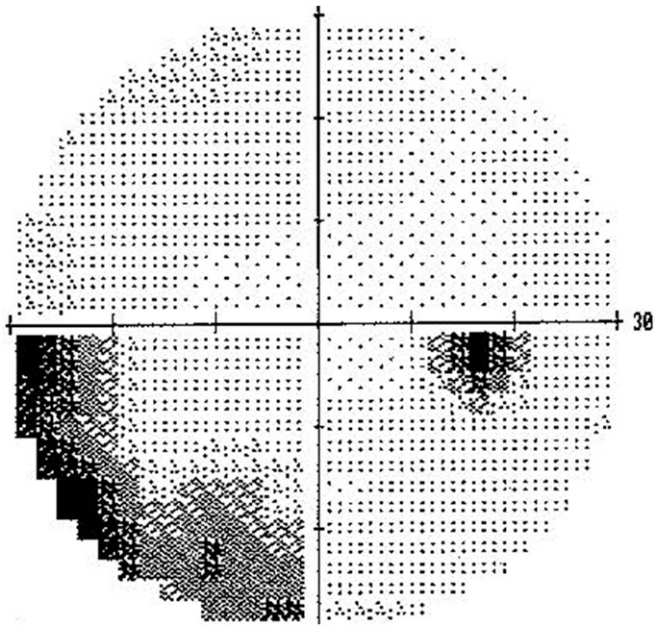

Fig. 1. Humphrey visual field 30-2 OD demonstrating infero-nasal visual defect and normal blind spot.

Dilated exam revealed a nerve fiber layer defect associated with supero-temporal optic nerve pallor, C/D ratio of 0.6 OD, and neural rim thinning/notching OD. Additional electrodiagnostic testing revealed significantly delayed latency, low amplitude, and poor waveform morphology on the visually evoked potential OD versus OS. To further elucidate the etiology of the optic nerve changes, a magnetic resonance imaging (MRI) of the orbits and brain was ordered. The MRI resulted in findings of patchy, nonenhancing T2 signal abnormality in the intraorbital segment of the right optic nerve (**Fig. 2**). Additionally, it identified an old right medial orbital wall blowout fracture with prolapse of the right medial rectus and intraorbital fat into the ethmoid sinus (**Fig. 3** and **Fig. 4**). Finally, multiple nonenhancing mucous retention cysts with inflammation in the paranasal sinuses were also identified.

Referral to neurology showed a normal exam with concern for a possible optic neuropathy as the potential cause for his optic nerve findings. Lab work was ordered, including angiotensin converting enzyme, antinuclear antibody panel, neocomplete paraneoplastic panel, Lyme disease antibody, bartonella antibody, human immunodeficiency virus, vitamin B1, and vitamin B12/folate. All results returned normal. The patient continued to complain of intermittent sinus block on flights and concern for ongoing sinus squeeze warranted a referral to otorhinolaryngology. Ear, nose, and throat examination and sinus computed tomography revealed uncontrolled chronic sinusitis with multiple sinus cysts. Recommendation was made to start maximal medical therapy to control symptoms with the possibility of the need for surgery in the future. Due to these recommendations and the presence of chronic sinusitis with symptoms, the member was returned to his home station and remained off of official flying status pending further management.

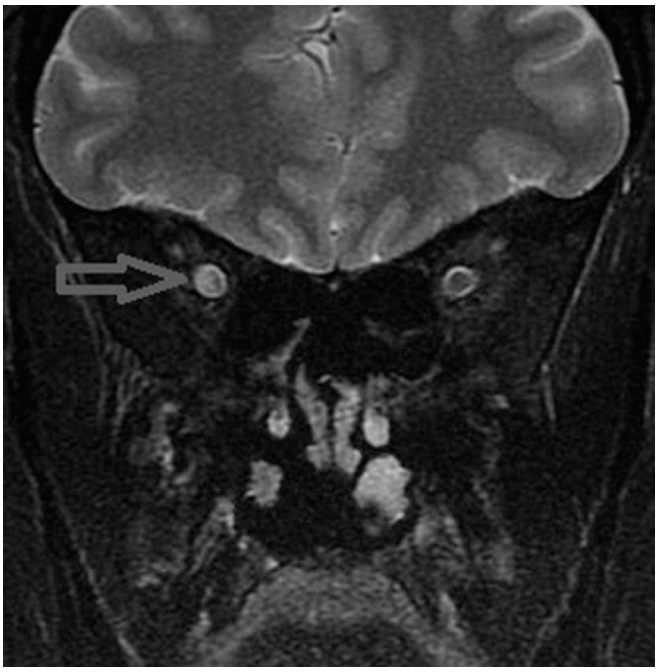

Fig. 2. Coronal T2 MRI image demonstrating hyperintensity in the right optic nerve at the intracanalicular segment.

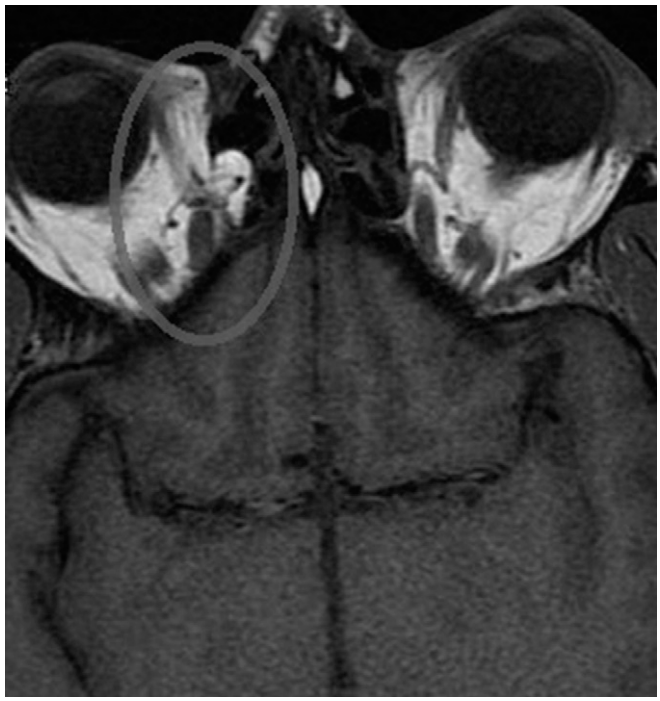

Fig. 4. Axial T1 MRI image demonstrating right medial wall defect and intraorbital contents within the ethmoid sinus.

DISCUSSION

This case presents several challenges in evaluation and diagnosis of a relatively common occurrence in flight medicine: the newly failed color vision test. It also is an example of the degree of knowledge a flight physician must have about presentations of eye diseases. Based upon initial findings, one may have erroneously concluded this patient suffered from early normal-tension glaucoma due to his visual field deficit, new asymmetric color deficiency, and optic nerve cupping. However, optic nerve pallor suggests a different etiology and must be investigated. This patient's complaints seem most likely related to a nonglaucomatous optic neuropathy. Although normal tension glaucoma can be considered in this case, it is less likely in light of disc pallor and the documented stability of cupping found on exam.

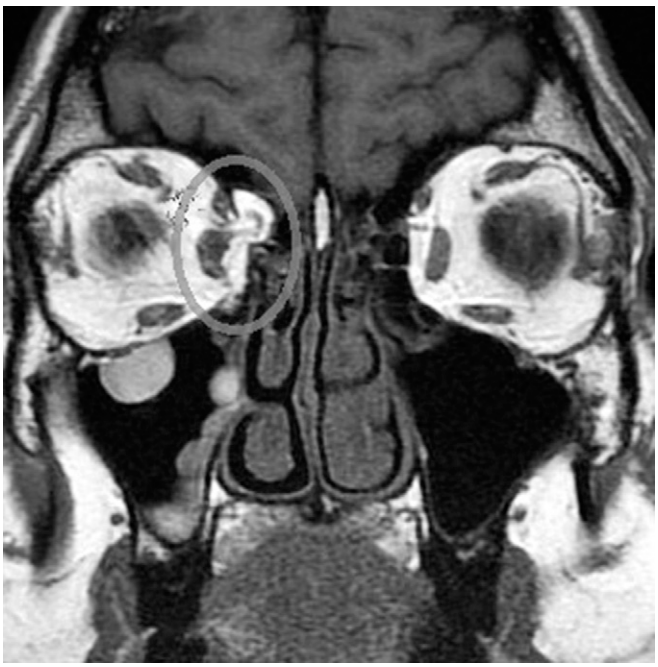

Fig. 3. Coronal T1 MRI imaging demonstrating right medial rectus and periorbital fat contained in the ethmoid sinus.

The differential diagnosis for an acute optic neuropathy is robust, but can be viewed as stemming from one of only a few categorical etiologies. These categories include vascular, inflammatory, compressive, infectious, nutritional, or idiopathic/other. The differential includes diagnoses such as tumor, ischemic optic neuropathy, multiple sclerosis, and Lyme disease, in addition to many other etiologies. Typically, rapidity of onset and the time course of symptoms are useful in determining the likely etiology of an optic neuropathy; however, this case seemed more difficult due to the lack of patient complaints or obvious vision deficits once he was examined. Even so, reviewing this patient's presentation and exam findings help make many etiologies of optic neuropathy less likely.

The patient's age at the time of presentation is a significant factor that can exclude many causes of optic neuropathy. Arteritic anterior ischemic optic neuropathy has an average age of presentation of 70 and is typically associated with giant cell arteritis.[1] The patient demonstrated no evidence of significant hypertensive disease or diabetes on exam. No evidence of a compressive etiology due to tumor or aneurysm was found on current MRI. Additionally, MRI found no indications of

ongoing inflammation, including no indication of white-matter disease. However, the MRI did show some increased signal in the intraorbital/intracanalicular segment of the right optic nerve consistent with gliosis. The posterior location of this finding makes an old case of nonarteritic anterior ischemic optic neuropathy less likely. Because the patient presented years after his initial injury with only minimal visual complaints (halos and perceived decrease in night vision capabilities), this leads to the exclusion of Leber's hereditary optic neuropathy. Leber's is usually a bilateral disease presenting earlier in life that goes on to progress to blindness and is extremely rare. Laboratory examination made several of the remaining diagnoses less likely as well.

Based upon findings during the exam and review of the patient's history, the differential diagnosis can be tailored to include decompression sickness (DCS), posterior ischemic optic neuropathy, or sinusitis. Any time an optic neuropathy is encountered in the aerospace or dive environment, DCS must be considered. At least two case reports have been published demonstrating a risk for the development of optic neuropathy in patients exposed to a hypobaric environment.[4,8] This patient was exposed to altitude, but the cabin environment reportedly never exceeded 8000 ft (2438 m) above sea level, making DCS somewhat less likely. Additionally, the member's reported pain, redness, and proptosis resolved during cruise altitude and did not require treatment. Also, no recurrence of symptoms has occurred over the following 12 yr even with numerous flights. Sinusitis has also been reported to be a potential cause of optic neuropathy in rare cases, although this is still disputed.[5] This patient does have an extensive history of recurrent allergic rhinitis and sinusitis in the past. Additionally, the most recent exam and imaging studies indicated ongoing inflammation with a recommendation by otorhinolaryngology for treatment. In spite of these findings, the clinical picture of acute onset retrobulbar pain, redness, and proptosis with resolution within minutes to hours does not lean toward a sinusitis etiology.

We believe this patient suffered from a sinus block on ascent that caused an abrupt, traumatic fracture of the medial orbital wall. This fracture allowed the escape of air into the orbit (orbital emphysema), causing an orbital compartment syndrome as evidenced by the proptosis, pain, and redness from venous congestion. This compartment syndrome either led to direct nerve injury from compression, stretching/traction on the nerve, or a posterior ischemic optic neuropathy. As the cabin altitude was lowered and the atmospheric pressure increased, the volume of the trapped air decreased secondary to Boyle's law. This allowed resolution of symptoms and is why the patient did not present with more severe vision compromise on the ground.

Orbital barotrauma is a known complication of activities involving sudden changes in atmospheric pressure. Typically, these types of injuries are encountered in the diving world as divers commonly encounter rapidly changing pressures associated with the sport. Additionally, aircrew members are subjected to changes in atmospheric pressure in performance of their duties. The anatomy of the orbital wall is such that rapid changes in pressure could lead to a fracture if a sinus block is encountered. Fractures in the medial wall and/or floor of the orbit can lead to communication of the orbit with the respective sinus. One study of orbital blowout fractures demonstrated medial wall fractures were the most common fractures of the orbit in patients sustaining clinically suspicious blunt trauma to the eye.[3] This supports the clinical findings in this patient with history of a medial wall fracture, although we believe the fracture was not the result of blunt trauma, but was due to sinus block on ascent leading to a fracture. Sinus barotrauma has been reported in the literature as a potential nidus in ophthalmic pathology. Specifically, one case report demonstrated complete blindness following sinus barotrauma in a diver while ascending from depth.[2] This case was in contrast to our patient in that the diver's loss of vision was believed to be due to a subsequent infection following the barotrauma. An additional case has been reported in a scuba diver in which the patient's optic neuropathy was believed to be caused by barotrauma to the sphenoid sinus exerting pressure on the optic nerve.[6] In our case report, we postulate the patient's optic neuropathy to be the result of orbital compartment syndrome resulting from orbital emphysema following communication with the ethmoid sinus. Orbital compartment syndrome can be the result of traumatic orbital fractures in which a ball-valve effect occurs at the fracture site, allowing entrapment of air in the orbit, resulting in increased intraorbital pressures.[7] This trapped air can lead to direct compression of the optic nerve or compromise of the posterior circulation. This diagnosis is supported by the fact the patient has evidence of a medial orbital wall fracture and presented with sudden onset pain, redness, and proptosis. In addition, late sequelae of an optic neuropathy were discovered as evidenced above with no other underlying etiology identified.

This case encompasses the difficulty flight surgeons face evaluating aircrew. This member most likely presented with little to no external signs of disease and was nearly asymptomatic. It is also difficult to say what type of exam was completed, since no documentation is available for review. Since the patient had no visual complaints at the time of initial evaluation, it would have been difficult to justify a more through exam. With that said, if an adequate visual acuity, visual field, pupil, and dilated eye exam had been performed and abnormalities were noted, it may have been reasonable to treat this patient as a DCS case in hopes of improving oxygenation to the optic nerve, although DCS may not have been the underlying cause.

ACKNOWLEDGMENTS

The views expressed in this article are those of the authors and do not necessarily reflect the official policy or position of the Air Force, the Department of Defense, or the U.S. Government.

Authors and affiliations: Marion R. Powell, M.D., San Antonio Uniformed Services Health Education Consortium, Ophthalmology Residency, Joint-Base San Antonio, TX, and Leo D. Hurley, M.D., and Tighe C. Richardson, D.O., U.S. Air Force School of Aerospace Medicine, Aeromedical Consultation Service, Ophthamology Branch, Wright-Patterson AFB, OH.

REFERENCES

1. Arnold AC. Ischemic optic neuropathies. In: Yanoff M, Duker JS, editors. Ophthalmology, 3rd ed. Edinburgh: Mosby Elsevier; 2009:970–975.
2. Bellini MJ. Blindness in a diver following sinus barotrauma. J Laryngol Otol. 1987; 101(4):386–389.
3. Burm JS, Chung CH, Oh SJ. Pure orbital blowout fracture: new concepts and importance of medial orbital blowout fracture. Plast Reconstr Surg. 1999; 103(7):1839–1849.
4. Butler FK. Decompression sickness presenting as optic neuropathy. Aviat Space Environ Med. 1991; 62(4):346–350.
5. Čelakovský P, Vokurka J, Školoudík L, Růžička J. Optic neuritis and paranasal sinus diseases. Cent Eur J Med. 2010; 6(1):117–119.
6. Gunn DJ, O'Hagan S. Unilateral optic neuropathy from possible sphenoidal sinus barotrauma after recreational scuba diving: a case report. Undersea Hyperb Med. 2013; 40(1):81–86.
7. Lima V, Burt B, Leibovitch I, Prabhakaran V, Goldberg RA, Selva D. Orbital compartment syndrome: the ophthalmic surgical emergency. Surv Ophthalmol. 2009; 54(4):441–449.
8. Steigleman A, Butler F, Chhoeu A, O'Malley T, Bower E, Giebner S. Optic neuropathy following an altitude exposure. Aviat Space Environ Med. 2003; 74(9):985–989.