Open Access Emergency Medicine

Dovepress
open access to scientific and medical research

Open Access Full Text Article

CASE REPORT

A case of cerebral aneurysm rupture and subarachnoid hemorrhage associated with air travel

This article was published in the following Dove Press journal:
Open Access Emergency Medicine
5 April 2014
Number of times this article has been viewed

Victoria Cui[1,2]
Timur Kouliev[1]
Jason Wood[1]

[1]Beijing United Family Hospital, Beijing, People's Republic of China; [2]Department of Biological Sciences, Columbia University, New York, NY, USA

Abstract: During air travel, passengers are exposed to unique conditions such as rapid ascent and descent that can trigger significant physiological changes. In addition, the cabins of commercial aircraft are only partially pressured to 552–632 mmHg or the equivalent terrestrial altitudes of 1,500–2,500 m (5,000–8,000 feet) above sea level. While studies in high-altitude medicine have shown that all individuals experience some degree of hypoxia, cerebral edema, and increased cerebral blood flow, the neurological effects that accompany these changes are otherwise poorly understood. In this study, we report a case of acute subarachnoid hemorrhage from a ruptured cerebral aneurysm associated with travel on commercial aircraft. We then review relevant cases of neurological incidents with possible air travel-related etiology and discuss the physiological factors that may have contributed to the patient's acute subarachnoid hemorrhage. In the future, this report may serve as reference for more detailed and conservative medical guidelines and recommendations regarding air travel.

Keywords: high-altitude, cabin pressure, emergency, cerebral edema, triage, neurological

Introduction

More than 2.5 billion individual flights are taken by passengers on commercial airlines each year, with the US market expected to double in the next 2 decades.[1,2] In recent years, a number of cardio, pulmonary, and neurological cases and syndromes have been reported with air travel-associated etiology.[3–5] However, the exact causes of these incidents have not been elucidated because the physiological changes associated with air travel, especially those affecting the brain, are not well understood. Conditions aboard commercial aircraft also present considerable challenges to both emergency treatment and research, including – but not limited to – cramped and unfamiliar spaces, the lack of proper medical equipment, and the need to avoid disruption or distraction of other passengers and flight crew.[4] As a result, inferences in many studies including this one are made from research relating to high-altitude illness and simulations performed using hypobaric chambers.[6–8]

Rather than attaining sea level values of around 760 mmHg, the cabins of passenger aircraft cruising at 9,100–12,200 m (30,000–40,000 feet) are only partially pressurized to 552–632 mmHg, or the equivalent moderate terrestrial altitudes of 1,500–2,500 m (5,000–8,000 feet) above sea level.[9] This difference in cabin pressure reflects a tradeoff with fuel efficiency, energy diversion from other aircraft systems, and operational wear on the plane's aluminum airframe.[10] By Dalton's law, the absolute partial pressure of O_2 decreases in this situation while its proportion of air composition remains constant, resulting in lower hemoglobin saturation in all airline passengers,

Correspondence: Victoria Cui
Columbia University, 116th Street and Broadway, New York, NY 10027, USA
Tel +1 512 289 3718
Email cui.victoria@gmail.com

with blood oxygen saturation (SpO_2) falling to 85%–91% of normal values.[4,11,12] This hypoxia-inducing change can present problems to those with preexisting conditions such as anemia and chronic obstructive pulmonary disease.[13]

Hypoxia due to decreased cabin pressures is not the only significant physiological change induced by the aircraft environment. Mild cerebral edema also occurs in hypobaric and hypoxemic conditions, although the exact mechanism is unknown.[8,14] In addition, airline passengers are also subject to the stresses of prolonged sitting, noise, claustrophobia, exposure to infectious agents, dehydration, and sleep deprivation. Some illnesses stemming from these conditions, such as deep vein thrombosis and jet lag, are well-characterized.[13]

In this report, we present a case of acute intracranial subarachnoid hemorrhage (SAH) following a commercial airline flight and compare it to other neurological incidents that have been reported as occurring during or after air travel. We also suggest a pathogenesis for our case by considering the physiological changes that occur at high altitude and onboard aircraft, with the continued goals of improving health guidelines and recommendations and preventing medical emergencies.

Case report

A 48-year-old Japanese man was brought to our Emergency Department at Beijing United Family Hospital unconscious and in respiratory arrest shortly after his plane landed in Beijing. The patient had taken a 3-hour morning flight from Guangzhou and complained of a sudden headache after the plane began its descent. While deplaning, the patient collapsed, and an ambulance was called. He vomited once and experienced gradual neurological and respiratory deterioration on the way to the Emergency Department.

Upon arrival, the patient was unresponsive with a Glasgow Coma Score of 3. The initial rhythm was atrial fibrillation, and there was no palpable pulse. Cardiopulmonary resuscitation was initiated, and the patient was intubated and ventilated following the American Heart Association's Advanced Cardiac Life Support procedure.[15] There was restoration of spontaneous circulation 2 minutes later with: heart rate, 119 bpm; blood pressure, 150/102; SpO_2, 98% on 100% oxygen bagging; and temperature, 37°C. The patient remained in atrial fibrillation with multiple premature ventricular contractions. Physical examination revealed a well-developed male with no signs of trauma. He remained unresponsive without sedation, and his pupils were of slightly unequal size with left $<$ right; $2<3$ mm.

Computed tomography (CT) scan of the head showed an acute SAH with blood extending to the lateral fissures, cisternae, and ventricles. Thoracic imaging indicated no signs of cardiopulmonary disease. The patient was a frequent business traveler, had a history of migraines, had previously stopped smoking, and was not a heavy drinker. The patient's mother died from acute myocardial infarction. He had no history of hypertension or other relevant medical conditions.

Subsequent angiography showed a fusiform aneurysm at the origin of the left-posterior inferior cerebellar artery (L-PICA). A thrombus had formed at the site of the aneurysm, partially occluding the vessel. The anterior inferior cerebellar artery, right vertebral artery, and both central carotid arteries were normal and showed no signs of stenosis or aneurysm. His left lateral ventricle was punctured and drained. The L-PICA had been occluded naturally by the expanding thrombus at the aneurysm site, and two smaller branches originating from the left vertebral artery appeared to be supplying the area.

Head CT angiography confirmed a 4 mm diameter aneurysm in the proximal part of the L-PICA, 5 mm from its origin in the left vertebral artery. The magnetic resonance imaging was consistent with these results, and an additional magnetic resonance angiogram showed a cessation of blood flow at the L-PICA. On day 5, the affected segment of the L-PICA was embolized with coils. The patient opened his eyes on day 2 and was taken off mechanical ventilation on day 6. At discharge on day 8, he was alert but had slurred speech. The patient was allowed to fly back to Japan that day accompanied by paramedics and was recommended for neurological follow-up. He has since recovered and returned to work with no reported neurological deficits.

Discussion

This patient was brought to our attention due to similarities with a case at our hospital previously reported in, "Intracranial hemorrhage during aeromedical transport and correlation with high altitude adaptations in the brain."[16]

That paper describes a patient who experienced two spontaneous cerebral hemorrhages; the second occurred in the same location after treatment, discharge, and aeromedical transport following the first incident.[16] However, the rupture of a relatively small cerebral aneurysm was determined to be the immediate cause of hemorrhage in our patient. Large cohort studies have estimated the likelihood of rupture for a cerebral aneurysm of less than 10 mm in diameter to be as low as 0.05% to at most 0.7% a year.[17,18] A history of smoking represented a

risk factor of aneurysm rupture for our patient, but the stronger predictors of aneurysm size –active smoking habit and age – did not apply.[19] In active smokers, it has been shown that the risk of SAH and aneurysm rupture is greatest in the 3 hours immediately following a cigarette due to an acute increase in blood pressure.[20] With these considerations, it is plausible that the likelihood of aneurysm rupture increased with the physiological changes experienced during air travel.

All individuals experience hypoxemia due to the hypobaric conditions of commercial aircraft as well as some degree of edematous swelling in the brain, although in the majority of cases these changes are asymptomatic.[11] In a magnetic resonance imaging study using hypobaric chambers to simulate conditions at 4,572 m (15,000 feet), Mórocz et al found a significant increase in brain volume of 36.2±19.6 mL after 32 hours of exposure.[8] It is reasonable to expect these effects to be present but less pronounced following air travel, due to shorter duration and lower equivalent altitudes. Studies on high-altitude illness propose that a combination of hypoxia-induced increases in cerebral blood flow, capillary pressure, and blood–brain barrier permeability led to this cerebral edema. In the model, patients with high-altitude illness do not have sufficient cerebrospinal capacity to buffer changes in cerebrospinal fluid volume and suffer from the symptoms of acute mountain sickness and its more severe form, high-altitude cerebral edema.[7] Changes in cabin pressure have also been proposed as one of many causes for the condition known as "airplane headache," with two subjects in a case series having similar presentation during high mountain descent.[5]

For patients with headache during or after air travel that is particularly severe or of unusual presentation, clinicians may consider performing additional screening for preexisting aneurysms and other intracranial lesions. Relevant conditions include meningiomas, colloid cysts, and sites of previous acute neurological events, such as SAH.[16,21]

In evaluating two cases of hemorrhage at the site of meningioma occurring 1–2 hours after aircraft descent, Goldberg et al suggest that cranial hypoxemia can cause necrosis and hemorrhage because the blood vessels of meningiomas lack the autoregulatory mechanisms of normal tissue.[22] Meningiomas are otherwise unlikely to begin bleeding compared to other types of cranial tumors.[22] Similarly, epithelial tissue at the aneurysm site may be more sensitive to blood pressure changes and therefore vulnerable to rupture. In the rare cases that intracranial pressure (ICP) has been measured at high altitudes, it does not appear to increase in tandem with cerebral blood flow or cerebral edema.[23] A constant ICP and increased blood pressure contribute to a rapid net increase in central perfusion pressure, which can lead to greater tension on the aneurysm wall and serve as a proximate physical mechanism of damage and aneurysm rupture following airplane takeoff and during the sustained high-altitude flight conditions.

Based on the similarities to previous cases and the appearance of positive symptoms within the patient's time on the airplane, it appears unlikely that the occurrence of SAH was purely coincidental. It is not yet apparent if this incident was triggered by the patient's most recent trip or if his frequent business flights progressively increased the likelihood of aneurysm rupture by gradually weakening the arterial wall. Repeated exposure to depressurizing conditions may represent the greater risk factor, with the patient's individual flight, which was unremarkable in terms of duration and flying conditions, contributing incrementally to reaching a physical threshold for aneurysm rupture. Comparison with similar cases of SAH and aneurysm rupture should yield additional insights.

Conclusion

The need for detailed and verified medical guidelines for air travel with preexisting neurological conditions remains. In addition to providing more conservative recommendations for those with unruptured cranial aneurysms, future criteria may need to cover other types of intracranial lesions. Providing emergency aeromedical transport via medical assistance services for patients with these conditions may also require reconsideration.

Studies of high-altitude sickness have thus far served as the closest analogies toward understanding the neurological effects and medical incidents associated with air travel. In this report, we make the preliminary conclusion that exposure to the conditions aboard commercial aircraft may increase the likelihood of intracranial aneurysm rupture, resulting in SAH. Continued reporting of similar neurological incidents and systematic study of physiological effects is necessary to understand the risks and prevent the complications of air travel with preexisting medical conditions.

Disclosure

The authors report no conflicts of interest in this work.

References

1. Price HJ. US Department of Transportation. Airline Passenger Travel to Nearly Double in Two Decades [press release]. Washington DC: US Department of Transportation; Mar 2012. Available from: http://www.faa.gov/news/press_releases/news_story.cfm?newsId=13394. Accessed August 1, 2013.

2. Air Transport, passengers carried. *Online: The World Bank*; 2013. Available from: http://data.worldbank.org/indicator/IS.AIR.PSGR. Accessed February 13, 2014.
3. Isayev Y, Chan RK, Pullicino PM. "Economy class" stroke syndrome? *Neurology*. 2002;58(6):960–961.
4. Hinkelbein J, Spelten O, Wetsch WA, Schier R, Neuhaus C. Emergencies in the sky: In-flight medical emergencies during commercial air transport. *Trends in Anaesthesia and Critical Care*. 2013;3(4):179–182.
5. Mainardi F, Lisotto C, Maggioni F, Zanchin G. Headache attributed to airplane travel ('airplane headache'): Clinical profile based on a large case series. *Cephalalgia*. 2012;32(8):592–599.
6. Hackett PH, Roach RC. High-altitude illness. *N Engl J Med*. 2001;345(2):107–114.
7. Basnyat B, Murdoch DR. High-altitude illness. *Lancet*. 2003;361(9373):1967–1974.
8. Mórocz IA, Zientara GP, Gudbjartsson H, et al. Volumetric quantification of brain swelling after hypobaric hypoxia exposure. *Exp Neurol*. 2001;168(1):96–104.
9. Medical aspects of transportation aboard commercial aircraft. AMA commission on emergency medical services. *JAMA*. 1982;247(7):1007–1011.
10. Muhm JM, Rock PB, McMullin DL, et al. Effect of aircraft-cabin altitude on passenger discomfort. *N Engl J Med*. 2007;357(1):18–27.
11. Gradwell DP, Risdall JE. Effect of high altitude commercial air travel on oxygen saturation. *Anaesthesia*. 2005;60(9):929; author reply 929–930.
12. Humphreys S, Deyermond R, Bali I, Stevenson M, Fee JP. The effect of high altitude commercial air travel on oxygen saturation. *Anaesthesia*. 2005;60(5):458–460.
13. Silverman D, Gendreau M. Medical issues associated with commercial flights. *Lancet*. 2009;373(9680):2067–2077.
14. Gallagher SA, Hackett PH. High-altitude illness. *Emerg Med Clin North Am*. 2004;22(2):329–355, viii.
15. Sinz E, Navarro K, Soderberg ES, Callaway CW, editors. *Advanced cardiovascular life support – Provider manual*. Texas: American Heart Association; 2011.
16. Kouliev T, Richardson A, Glushak C. Intracranial hemorrhage during aeromedical transport and correlation with high altitude adaptations in the brain. *Open Access Emergency Medicine*. 2012;4:93–95.
17. Rinkel GJ, Djibuti M, Algra A, van Gijn J. Prevalence and risk of rupture of intracranial aneurysms: a systematic revew. *Stroke*. 1998;29(1):251–256.
18. Wiebers D, Whisnant J, Forbes G, et al. Unruptured intracranial aneurysms – risk of rupture and risks of surgical intervention. *N Engl J Med*. 1998;339(24):1725–1733.
19. Juvela S, Porras M, Poussa K. Natural history of unruptured intracranial aneurysms: probability of and risk factors of aneurysm rupture. *J Neurosurg*. 2008;108(5):1052–1060.
20. Longstreth WT Jr, Nelson LM, Koepsell TD, van Belle G. Cigarette smoking, alcohol use, and subarachnoid hemorrhage. *Stroke*. 1992;23(9):1242–1249.
21. Perin A, Larosa F, Longatti P. Barometric changes in patients with intracranial lesions: can they dive and fly? *Surg Neurol*. 2009;71(3):368–371.
22. Goldberg CR, Hirschfeld A. Hemorrhage within brain tumors in association with long air travel. *Acta Neurochir (Wien)*. 2002;144(3):289–293.
23. Wilson MH, Newman S, Imray CH. The cerebral effects of ascent to high altitudes. *Lancet Neurol*. 2009;8(2):175–191.