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

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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. In recent years, a number of cardio, pulmonary, and neurological cases and syndromes have been reported with air travel-associated etiology. 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. 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.

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. 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. By Dalton’s law, the absolute partial pressure of O2 decreases in this situation while its proportion of air composition remains constant, resulting in lower hemoglobin saturation in all airline passengers,
with blood oxygen saturation (SpO_2) falling to 85%-91% of normal values. This hypoxia-inducing change can present problems to those with preexisting conditions such as anemia and chronic obstructive pulmonary disease.\textsuperscript{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 hypoxicem conditions, although the exact mechanism is unknown.\textsuperscript{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.\textsuperscript{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.\textsuperscript{13} 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.”\textsuperscript{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.\textsuperscript{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.\textsuperscript{17,18} A history of smoking represented a
risk factor of aneurysm rupture for our patient, but the stronger
predictors of aneurysm size—a history of smoking habit and age—
did not apply. 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. 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. In a magnetic reso-
nance imaging study using hypobaric chambers to simulate con-
ditions 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. 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. 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.

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.

In evaluating two cases of hemorrhage at the site of menin-
gioma occurring 1–2 hours after aircraft descent, Goldberg et al suggest that cranial hypoxemia can cause necrosis and hemorrhage because the blood vessels of menin-
giomas lack the autoregulatory mechanisms of normal
tissue. Meningiomas are otherwise unlikely to begin bleed-
ing compared to other types of cranial tumors. 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.
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 appear-
ance 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 rep-
resent 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 recommenda-
tions 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.

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