



Flying with Colloid Cyst: A Cautionary Note

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Key words

- Air travel
- Colloid cyst
- Obstructive hydrocephalus

Abbreviations and Acronyms

AMS: Acute mountain sickness
CSF: Cerebrospinal fluid
CT: Computed tomography
HACE: High-altitude cerebral edema
ICP: Intracranial pressure
MRI: Magnetic resonance imaging
VEGF: Vascular endothelial growth factor
VPS: Ventriculoperitoneal shunt

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Citation: *World Neurosurg.* (2020) 138:84-88.

<https://doi.org/10.1016/j.wneu.2020.01.124>

Journal homepage: www.journals.elsevier.com/world-neurosurgery

Available online: www.sciencedirect.com

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INTRODUCTION

Colloid cysts are rare lesions with a yearly incidence of 3.2 per 1,000,000 people.¹ The growth patterns of these benign lesions are unpredictable. Studies reviewing the natural history of colloid cysts suggest that these lesions may remain stable over the years without producing any symptoms, or sometimes may present with life-threatening hydrocephalus due to a sudden increase in their size.¹⁻⁴ Only rarely do they exhibit the phenomenon of spontaneous regression.^{5,6} The attributing factors for sudden presentations are diverse. Younger age, cyst size, and hyperintensity on T2-weighted magnetic resonance imaging (MRI) have been correlated to growth and symptomatic presentations.² Air travel as an inciting factor for symptomatic presentation in patients harboring colloid cyst is rare. There have been only a few reports that describe such patients, all of

■ **BACKGROUND:** Colloid cysts are benign and rare tumors of the brain. The growth rates of these tumors are unpredictable. These cysts can increase in size and obstruct the cerebrospinal fluid pathways producing obstructive hydrocephalus. Consequently, this can manifest as acute severe headaches followed by deterioration in consciousness, or even sudden death in patients. Such remarkable episodes occurring in patients during air travel have been reported sparsely in the literature.

■ **CASE DESCRIPTION:** In this report, we narrate the ordeal of a patient who had severe headache followed by loss of consciousness during his air travel. After his arrival, he was taken to a referral center where the diagnosis of a colloid cyst obstructing the cerebrospinal fluid pathway resulting in acute obstructive hydrocephalus was revealed. We analyze the physiologic effects of cabin pressure and high altitude on the intracranial pressure and present a brief review of the literature.

■ **CONCLUSIONS:** Changes in cabin pressure during flight may play a role in worsening of intracranial pressure in patients with colloid cyst with marginal brain compliance.

whom have had a fatal outcome. In this case report, we describe the first surviving patient with a colloid cyst who had worsening of symptoms during air travel. We analyze the possible mechanisms of alterations in intracranial pressure (ICP) experienced during air travel and present a review of literature of similar cases.

CASE DESCRIPTION

A man aged 45 years had intermittent episodes of holocranial headache for 4 months. These episodes of headache gradually progressed in frequency and intensity, after which he sought medical attention. He was examined by a local physician who prescribed him analgesics, which gave him some relief. He was a manual laborer who had been recently recruited to join a company in the Middle East. Two days before his journey, he had an episode of severe headache. He again consulted the local physician who prescribed analgesics, which relieved his headache partially. This mild headache

lasted for the next 2 days but was ignored, considering the importance of traveling to the Middle East. He boarded the flight to the Middle East with mild headache. An hour and a half into his travel, his headache worsened, and he began to vomit. He was attended by the air hostess who found him to be delirious. There were no doctors on board this flight. Toward the end of his journey, as the flight landed at its destination, he became unconscious. He was taken to the nearby referral center in a comatose state where he underwent emergency imaging of the brain. An MRI of the brain was taken, which revealed a colloid cyst of size 3 x 4 cm in the region of foramen of Monro, causing obstructive hydrocephalus. Following this finding, he was taken up for emergency cerebrospinal fluid (CSF) diversion surgery in the form of a ventriculoperitoneal shunt (VPS). He was ventilated postoperatively for 24 hours, after which he regained consciousness. His condition was explained to him, and he was also given a choice to undergo a second surgery for the colloid cyst at the same hospital. However, he was

not keen on being operated on in a foreign land and expressed his desire to be flown back to India. At discharge, he was fully conscious and oriented without any focal neurologic deficit.

After flying to India, he arrived at our hospital. He did not have any symptoms of raised ICP, such as intense headache or vomiting, on his journey back to India. On examination, he was fully conscious and oriented. There were no signs of raised ICP. There was a scar of the VPS surgery over the scalp, as well as over the abdomen. A computed tomography (CT) scan was available (Figure 1A and B), which showed a hypodense lesion in the region of foramen of Monro, producing obstructive hydrocephalus. The ventricular end of the VPS was visualized in the right occipital horn of the lateral ventricle. He underwent an MRI scan of the brain, which confirmed the presence of the colloid cyst (Figure 2A–F). The need for excision of the cyst was explained to him, and he was taken up for surgery. He underwent excision of the colloid cyst by an interhemispheric transcalsal approach at our center. The cyst was totally excised, and he recovered well from the surgery. A postoperative scan (Figure 3A) was taken 4 days after the surgery showed subacute subdural hematoma in the right fronto-temporo-parietal region with mass effect and

midline shift. As there were no signs or symptoms of raised ICP, he was treated conservatively with antiedema measures. A CT scan was repeated after 2 weeks (Figure 3C and D), which showed total resolution of the subacute subdural hematoma. Histopathologic examination of the excision specimen revealed findings that were consistent with colloid cyst. Until his last follow-up at 1 year, he remained asymptomatic without any focal neurologic deficits.

DISCUSSION

The ICP and its alterations due to cabin pressure inside commercial flights have not been well documented. However, there have been studies documenting the potential of residual trapped air following craniotomy to expand at high altitudes and cause raised ICP.^{7,8} Phillips et al.⁹ examined a cohort of patients with brain tumors who had been flown to their center for surgical treatment. They noted that there was a worsening of symptoms in 24.4% of patients. The symptoms ranged from headache, fatigue, ear pain, and new-onset seizures. Reports of severe symptomatic presentation in patients with different brain pathologies during and immediately after air travel have emerged consistently in the literature.^{10–16} Moreover, such instances involving

patients with a colloid cyst has been reported 3 times.^{17–19} These 3 reports narrate the ordeal of 4 persons in whom a colloid cyst was diagnosed after the worsening of symptoms while flying.

In the earliest report by Nelson and Haymaker,¹⁷ they described 3 fatal cases of colloid cyst in flyers. Among them, 2 persons developed symptoms during their air travel. The first person was an airline pilot who developed severe headache followed by deterioration in consciousness during one of his flights. Following this, the copilot took over the plane and landed safely, but by that time, the pilot had died. The second person was a civilian passenger who experienced worsening of headache during his travel but recovered soon after landing only to become symptomatic again in the following week. A month later, his headaches progressed, followed by sudden death. The authors proposed that negative and positive gravitational forces during flying influence intracranial hypertension, and may have been the cause of aggravation of the symptoms in them.¹⁷ In another report, Büttner et al.¹⁸ narrated a case of a woman who suffered intense headache followed by loss of consciousness experienced during her intercontinental flight. CT scan did not reveal any intracranial pathology. She rapidly deteriorated after this and died. An autopsy was performed, which revealed the diagnosis of a colloid cyst of the third ventricle.¹⁸ Ter Meulen et al.¹⁹ reported another woman who had worsening of headache and sensorium during a transcontinental flight. She rapidly deteriorated and became comatose. CT scan revealed biventricular hydrocephalus. An external ventricular drain was inserted to relieve the hydrocephalus, but the patient did not show any improvement, and she finally died of her illness.¹⁹ An autopsy was conducted, which revealed a colloid cyst was obstructing the foramen of Monro.

Our patient is only the fifth reported case who had worsening of headache and deterioration of consciousness during his air travel. He is also the only person alive so far to survive such an incident. In our patient, the colloid cyst was obstructing the foramen of Monro, producing obstructive hydrocephalus. This was the most likely cause of the headaches that he

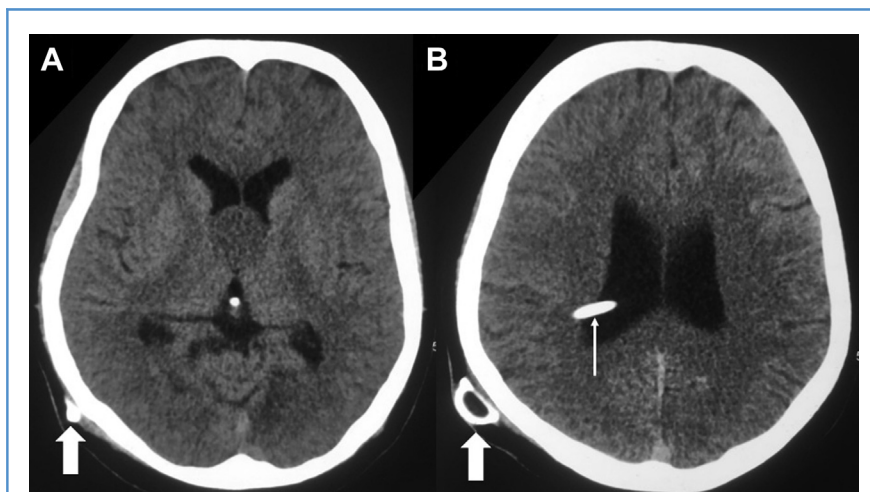


Figure 1. (A and B) CT brain plain post ventriculo-peritoneal shunt image 1. (A) A well defined hypodense spherical lesion is seen in the region of Foramen of Munro. Note the shunt chamber (Thick white arrow) in the right posterior parietal region (A and B). (B) The ventricular end of the shunt catheter is seen in the right lateral ventricle.

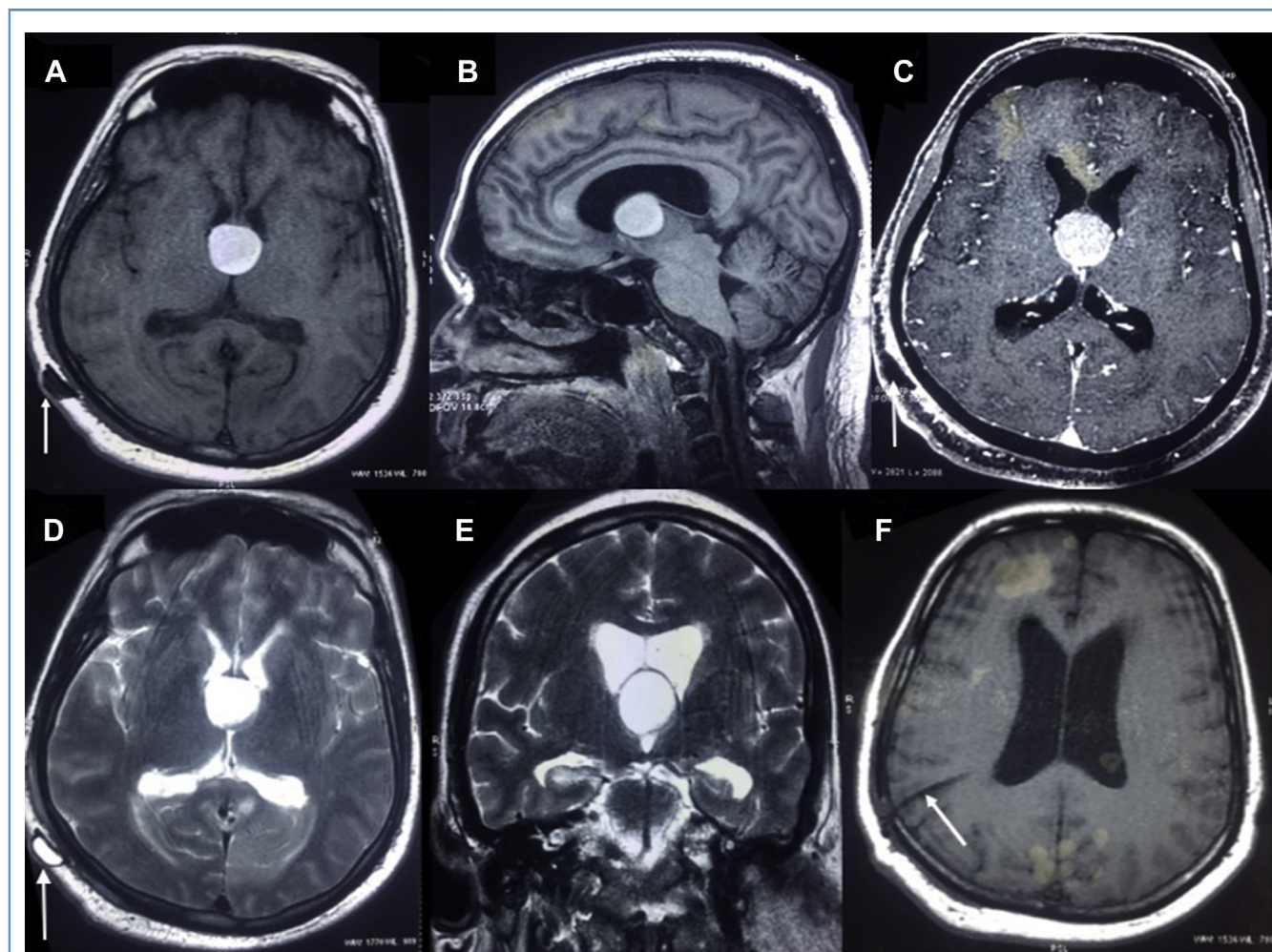


Figure 2. MRI Brain: (A) T1 Axial, (B) T1 Sagittal, (C) SPGR with contrast, (D) T2 Axial, (E) T2 Coronal, (F) T1 Axial. A well defined hyperintense spherical lesion is seen in the region of Foramen of Munro obstructing the

CSF pathway (A-E). The shunt chamber is seen in the right posterior parietal region of the scalp (A, C, D) and the ventricular catheter is seen traversing through the right parietal lobe towards the lateral ventricle (F).

experienced over 4 months before his travel. However, because imaging of the brain was not done, this pathology remained concealed. His symptoms worsened owing to raised ICP, which was further aggravated during his flight, possibly owing to alterations in altitudinal pressures.

The United States Federal Aviation Administration and the rest of the world follow regulations that require commercial jets to maintain a pressure equivalent to being at an altitude of no more than 2440 m.²⁰ They also have recommended the pressure equivalent rate of ascent to be limited to not more than 5 m per second. These altitudes have been correlated with symptoms of acute mountain sickness

(AMS), and high-altitude cerebral edema (HACE) experienced in unacclimatized mountaineers. The exact pathophysiology of HACE has not been deciphered yet. In the descriptions of altitudinal medicine, the deleterious effects of altitude on the brain have been described as a spectrum ranging from a less severe form of high altitudinal headache, acute motion sickness, and to more severe HACE. Ross²¹ described the “tight fit hypothesis” as an explanation for the development of AMS. According to them, the symptomatology of the patients in AMS varies and is dependent on the differential compliance of their own brain. Patients with large CSF spaces and ventricles have minimal or no symptoms, whereas patients with tight

CSF spaces and smaller ventricles exhibit severe symptoms. This hypothesis has been corroborated with the nearly insane experiment unlikely to be repeated in the realm of neurosurgery by Cummins et al., the findings of which were published later by Wilson and Milledge.²² In this study, 10 climbers of the British expedition to the Himalayas were subjected to CT scans before the commencement of their journey. Among the subjects, 3 climbers (1 including Cummins himself) were subjected to burr hole and placement of a telemetric neuromonitoring device. ICP recordings, as well as symptoms of AMS, were noted during the ascend. Although the ICP values remained in the upper limit of normal at high altitudes, severe

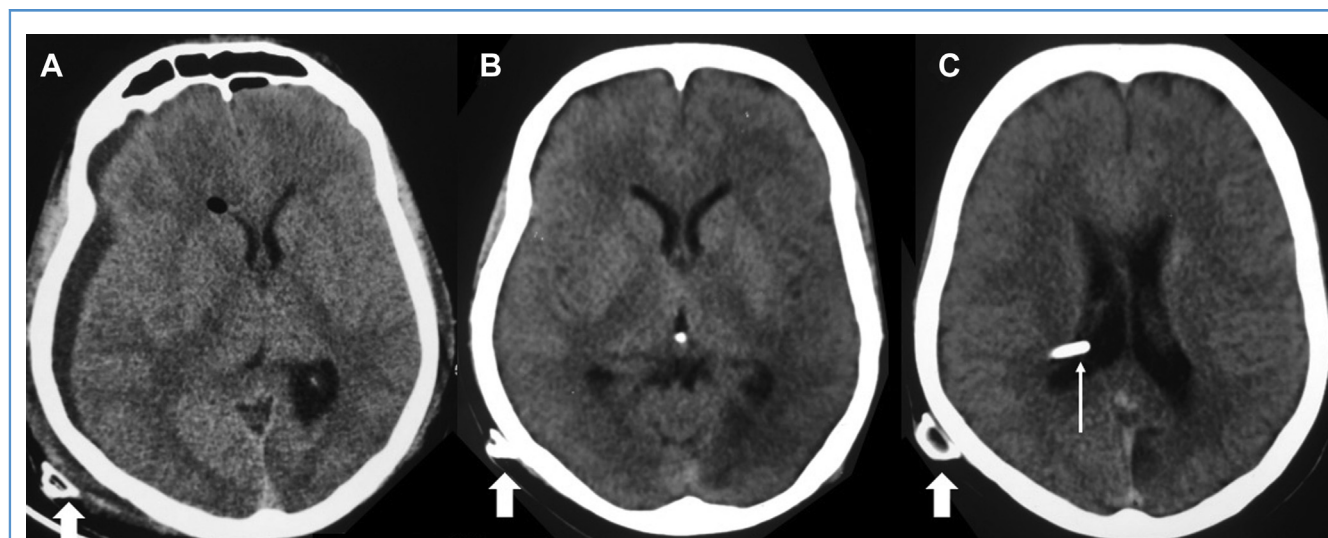


Figure 3. (A) CT brain plain post-operative scan 4 days after the surgery. Total removal of the colloid cyst is noted. There is minimal air in the frontal horn of the right lateral ventricle. There is a thin subdural hematoma on the right fronto-temporo-parietal region which is causing mass effect and

midline shift. (B and C) Post-operative CT Brain after 2 weeks. There is total resolution of the subdural hematoma. Note the shunt chamber (*thick white arrow*) in the posterior parietal region and shunt catheter (*thin white arrow*) in the right lateral ventricle.

symptoms were observed only in climbers with tight brains and smaller ventricles. Furthermore, experimental studies by Dipasquale et al.²³ have concluded that there is a state of hyperperfusion, increased ICP, and cerebral edema in AMS. Our patient had a tight brain, as evidenced by the MRI, which showed obstructive hydrocephalus. His noncompliant brain might have been subjected to iterative pressure changes during his flight, which further aggravated the ICP rendering him unconscious at the end of the journey. Such obstructive lesions of the CSF pathways producing hydrocephalus have been shown to deteriorate during air travel. Zrinzo et al.¹⁰ reported 2 cases with acute obstructive hydrocephalus who became severely symptomatic during their air travel. Among them was a case of choroid plexus xanthogranuloma occluding the ventricular system, and the other was a case of posterior fossa extra-axial lesion obstructing the aqueduct. The authors concluded that long air travel might have caused mild hypoxia and cerebral edema very much similar to AMS and HACE.¹⁰ These events are usually well-tolerated in normal individuals but may lead to grave consequences in patients harboring intracranial pathology, given the lack of brain reserve.

Different mechanisms interplay during commercial flights, which may additively result in brain edema. At high altitudes, there is an upregulation of vascular endothelial growth factor (VEGF) in response to hypoxia. VEGF, in turn, increases capillary permeability and consequent cerebral edema.²⁴ Dexamethasone is known to downregulate VEGF and has been shown to decrease cerebral edema.^{25,26} Because of this action, it has been extensively used in the management of HACE. In a review of the safety of commercial flight in patients with brain tumors, Phillips et al.⁹ noted that perflight administration of steroids lowered the incidence of symptomatic worsening than in the nonsteroidal group. Another mechanism that increases the ICP can be attributed to an increased fraction of inspired carbon dioxide in the inhaled air inside the cabin. This mild degree of hypercapnia is well documented to increase ICP.²⁷ These factors are additive in the worsening of patients with marginal cerebral compliance.

The postoperative period in our patient was complicated by the detection of sub-acute subdural hematoma. Although not common, it has been a described complication following the excision of colloid cyst.^{28,29} This is thought to occur because of the rupture of bridging veins following sudden decompression of the ventricles

during surgery. The VPS inserted in this patient for relief of hydrocephalus was retained and left in situ. This practice of leaving behind shunt catheters following definitive surgery for tumors/cysts causing obstructive hydrocephalus is common in this part of the world. This practice has been followed at our center for decades and has not been associated with increased complications.

CONCLUSIONS

The complications of air travel with brain pathologies, especially those lesions obstructing the CSF pathways, such as colloid cysts, should be appraised to the patients. The ICP changes, experienced during air travel, might become significantly perilous in patients whose ICP is in a partially compensated state. Immediate return to sea level altitude should be considered in such situations. Perflight administration of steroids may be helpful during the transportation of such patients.

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Conflict of interest statement: The authors declare that the article content was composed in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Received 3 October 2019; accepted 16 January 2020

Citation: World Neurosurg. (2020) 138:84-88.
<https://doi.org/10.1016/j.wneu.2020.01.124>

Journal homepage: www.journals.elsevier.com/world-neurosurgery

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