Case reports

Secondary deterioration in a patient with cerebral and coronary arterial gas embolism after brief symptom resolution: a case report

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Keywords
Air embolism; Case reports; Hyperbaric oxygen treatment

Abstract
(Tsushima R, Mori K, Imaki S. Secondary deterioration in a patient with cerebral and coronary arterial gas embolism after brief symptom resolution: a case report. Diving and Hyperbaric Medicine. 2024 31 March;54(1):61–64. doi: 10.28920/dhm54.1.61-64. PMID: 38507911.)
Introduction: Hyperbaric oxygen treatment (HBOT) is recommended for arterial gas embolism (AGE) with severe symptoms. However, once symptoms subside, there may be a dilemma to treat or not.
Case presentation: A 71-year-old man was noted to have a mass shadow in his left lung, and a transbronchial biopsy was performed with sedation. Flumazenil was intravenously administered at the end of the procedure. However, the patient remained comatose and developed bradycardia, hypotension, and ST-segment elevation in lead II. Although the ST changes spontaneously resolved, the patient had prolonged disorientation. Whole- body computed tomography revealed several black rounded lucencies in the left ventricle and brain, confirming AGE. The patient received oxygen and remained supine. His neurological symptoms gradually improved but worsened again, necessitating HBOT. HBOT was performed seven times, after which neurological symptoms resolved almost completely.
Conclusions: AGE can secondarily deteriorate after symptoms have subsided. We recommend that HBOT be performed promptly once severe symptoms appear, even if they resolve spontaneously.

Introduction

Arterial gas embolism (AGE) is an arterial occlusion caused by bubbles that results in organ ischaemia. Most cases occur in compressed gas diving or medical procedures, including arterial catheterisation, cardiovascular surgery, positive pressure ventilation, and bronchoscopy. The incidence rate of AGE during bronchoscopy is 0.00096% for all bronchoscopic procedures.[1]

Hyperbaric oxygen treatment (HBOT) and normobaric oxygen (NBO)[2] are recommended as treatments. Among the types of AGE, HBOT is recommended for treating coronary and cerebral artery embolisms.[3] Most experts recommend HBOT even if initial symptoms are mild or improving because of the possibility of secondary deterioration. However, reports of secondary deterioration in humans are very limited.[4] Herein, we report a case of cerebral and coronary AGE with deterioration after brief symptom resolution.

Case presentation

This patient was lost to follow-up, but consent for publication of his anonymised case details were obtained from an immediate family member.

A 71-year-old man with a history of chronic obstructive pulmonary disease and pulmonary fibrosis presented with a mass shadow in the S6 lower lobe of the left lung detected on a chest radiograph. The patient was referred to our respiratory medicine department for bronchoscopy.

Endobronchial ultrasound-guided sheath transbronchial biopsy was performed with mild sedation using intravenous midazolam (3 mg), and oxygen was administered via a nasal cannula at a rate of 2 L·min^{-1}. Flumazenil (0.5 mg) was intravenously administered 27 min after the beginning of the procedure; nevertheless, the patient remained comatose, with a Glasgow coma scale (GCS) score of 3 (E1 V1 M1). A few minutes later, the patient developed bradycardia,

hypotension, and ST-segment elevation in lead II, with a heart rate of 46 beats·min^{-1} and blood pressure of 90/70 mmHg. Atropine (0.5 mg) was intravenously administered for symptomatic bradycardia. Subsequently, haemodynamics and consciousness improved slightly. The heart rate was 106 beats·min^{-1}, blood pressure was 142/98 mmHg, and the GCS score was 7 (E2 V1 M4). A 12-lead electrocardiogram (ECG) was obtained, which showed ST-segment elevation at II, III, and ST depression at I and aVL (Figure 1A). Therefore, myocardial infarction was diagnosed. However, the ST changes spontaneously resolved approximately 8 min later (Figure 1B). Although the ECG changes disappeared, the patient had prolonged disorientation with a GCS score of 9 (E4 V1 M4), complete left hemiplegia (manual muscle testing [MMT] scale score of 1), and total aphasia. Thus, we performed whole-body computed tomography (CT), which revealed several black rounded lucencies in the left ventricle and left cerebral hemispheres, confirming AGE (Figure 2A). While the patient received NBO and remained in the supine position the neurological symptoms gradually improved, evidenced by a GCS score of 14 (E4 V4 M6), improvement in left hemiparesis (MMT scale score of 3), and improvement in aphasia. A CT scan performed again after 90 min showed oedematous changes in the right cerebral hemisphere, although the gas had disappeared (Figure 2B). The patient was then admitted to the intensive care unit for conservative treatment. However, shortly after admission, the patient became restless and neurological symptoms deteriorated again (GCS score 11 [E4 V2 M5]) along with worsened left hemiparesis (MMT scale score: 2), and HBOT was deemed necessary. The patient was transferred to a medical institution equipped with a multiplace HBOT chamber.

The first round of HBOT was initiated seven hours and 15 minutes after the onset of symptoms and was performed according to the US Navy Treatment Table 6. During treatment, tonic-clonic convulsions were observed three times. At each instance, diazepam (5 mg) was intravenously administered, and the convulsions were successfully controlled. On a once daily basis, the second and third round of HBOT were performed according to the US Navy Treatment Table 5, and an additional four treatments were administered at 152 kPa (1.5 atmospheres absolute) for 90 minutes.

The neurological findings improved over time, with a GCS score of 14 (E4 V4 M6), MMT scale score of 4 in the left upper limb, and MMT scale score of 5 in the left lower limb at the end of the seventh round of HBOT. Brain magnetic resonance imaging fluid-attenuated inversion recovery showed an enlarged high-signal intensity area in the right cerebral hemisphere (Figure 3). Eight days later, the patient was referred to our hospital for rehabilitation.

Neurological findings improved almost completely, except for a mild decrease in writing ability and mild paralysis of the left finger.

Figure 1

(A) A 12-lead electrocardiogram showing ST-segment elevation at II, III, and ST depression at I and aVL; (B) spontaneous ST change improvement noted approximately 8 min later

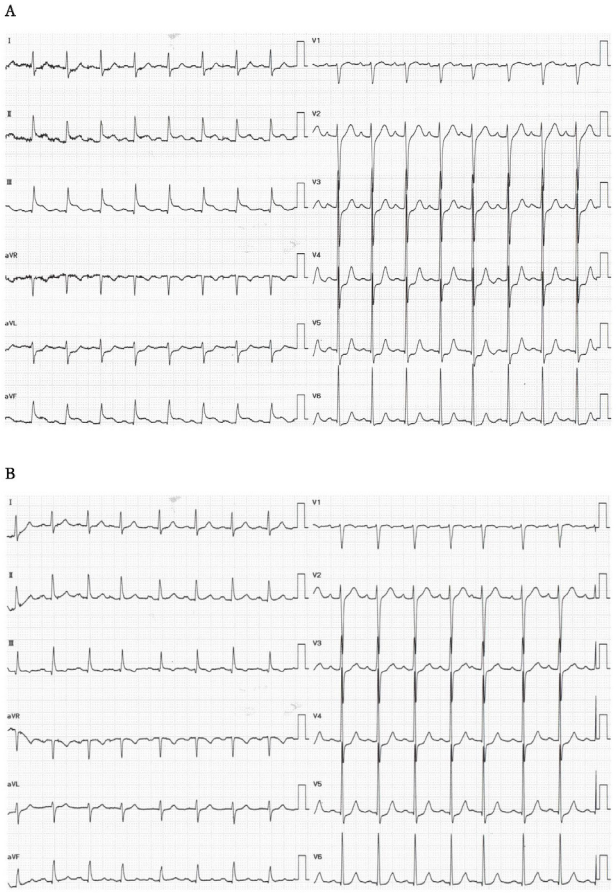


Figure 2

(A) Computed tomography (CT) scan showing rounded lucencies in the left ventricle and left cerebral hemisphere; (B) Repeat CT 90 min later showing bubble disappearance and new edematous changes in the right cerebral hemisphere

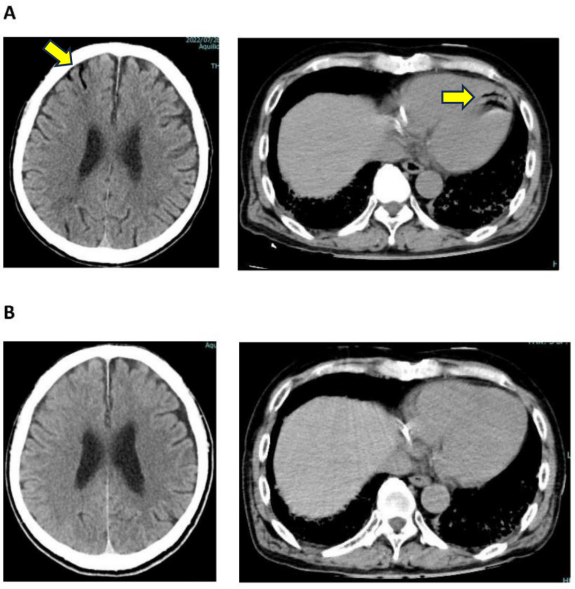
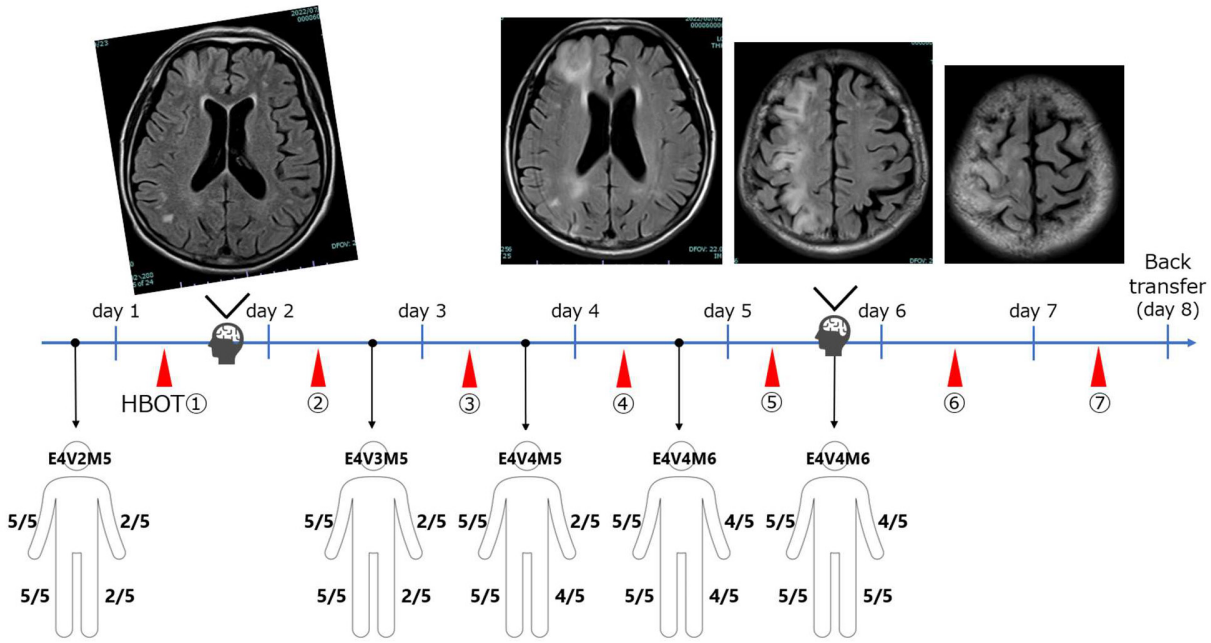


Figure 3

Magnetic resonance imaging signal-change transitions and changes in the manual muscle testing (MMT) score over the course of the patient's treatment with hyperbaric oxygen (HBOT)



The patient was discharged on day 30, with subsequent planned outpatient rehabilitation.

Discussion

We encountered a case of cerebral and coronary AGE during bronchoscopy. After secondary deterioration, the patient was treated with HBOT, resulting in a good outcome. To the best of our knowledge, reports of secondary deterioration in AGE are limited.[4]

There are four possible mechanisms involved in secondary deterioration: first, there may be re-embolisation by gas trapped in one of the heart chambers or pulmonary veins that has not yet passed into the systemic circulation;[5] second, minute gas bubbles undetectable on CT may cause progression of organ ischaemia; third, inflammatory changes in the cerebral blood vessels incited by the passage of bubbles may cause a progressive reduction of cerebral blood flow;[6,7] and fourth, reperfusion may induce inflammation. In cases of AGE, it is recommended that the patient is kept in a supine position,[8] and NBO (as first aid) and HBOT are recommended. There are four potential mechanisms by which HBOT helps in AGE: first, it encourages bubble redistribution and reduces vascular occlusion by decreasing bubble volume;[9] second, it markedly increases the partial pressure of dissolved oxygen in plasma, increases oxygen delivery to ischaemic tissues, and inhibits cellular damage;[10] third, it counters vasodilation of the capillaries within hypoxic tissues, thereby minimising collection of extravascular fluids, reducing brain vasogenic oedema and potentially reducing intracranial pressure;[11] and fourth, it suppresses leukocyte β2 integrin function, inhibiting inflammatory cell adhesion to vascular endothelial cells after reperfusion, and suppresses consequent inflammatory damage in adjacent tissue.[12]

Early administration of HBOT, especially within 6–8 h, is associated with improved neurological prognosis. A systematic review and meta-analysis published in 2023 showed that increased time-to-HBOT is associated with decreased probability of favorable outcome in iatrogenic gas embolism.[13] Although adverse events such as lung injury, pneumothorax, and tympanic trauma have been reported, all are rare and manageable,[14] and HBOT should be performed promptly after the onset of serious symptoms. In this case, when the symptoms initially resolved the patient was treated with supine positioning and NBO, but later experienced secondary deterioration and was treated with HBOT. With the benefit of hindsight, HBOT should have been performed at the time the symptoms first appeared, with the possibility of secondary deterioration in mind.[15]

Conclusions

This case is a contemporary reminder that AGE can secondarily deteriorate after apparent spontaneous recovery. We acknowledge that close observation without HBOT has been successfully employed after spontaneous recovery from AGE where compression was considered risky (because of concomitant pneumothoraces).[16] However, unless (as in that case) there are other complicating factors, we recommend that HBOT be provided promptly after diagnosis of AGE, irrespective of any spontaneous recovery.

References

1 Maemura K, Kage H, Isago H, Takeshima H, Makita K, Amano Y, et al. Cerebral arterial air embolism after diagnostic flexible fiberoptic bronchoscopy: A case report and review of the literature. Case Rep Pulmonol. 2018;2018:7108215. doi: 10.1155/2018/7108215. PMID: 29854532.

2 Brull SJ, Prielipp RC. Vascular air embolism: A silent hazard to patient safety. J Crit Care. 2017;42:255–63. doi: 10.1016/j.jcrc.2017.08.010. PMID: 28802790.

3 Closon M, Vivier E, Breynaert C, Duperret S, Branche P, Coulon A, et al. Air embolism during an aircraft flight in a passenger with a pulmonary cyst: A favorable outcome with hyperbaric therapy. Anesthesiology. 2004;101:539–42. doi: 10.1097/00000542-200408000-00037. PMID: 15277939.

4 Leitch DR, Green RD. Pulmonary barotrauma in divers and the treatment of cerebral arterial gas embolism. Aviat Space Environ Med. 1986;57:931–8. PMID 3778391.

5 J Tingleff, F S Joyce, G Pettersson. Intraoperative echocardiographic study of air embolism during cardiac operations. Ann Thorac Surg. 1995;60:673–7. doi: 10.1016/0003-4975(95)00577-8. PMID: 7677498.

6 Helps SC, Parsons DW, Reilly PL, Gorman DF. The effect of gas emboli on rabbit cerebral blood flow. Stroke. 1990;21:94–9. doi: 10.1161/01.str.21.1.94. PMID: 2300997.

7 Helps SC, Gorman DF. Air embolism of the brain in rabbits pretreated with mechlorethamine. Stroke. 1991;22:351–4. doi: 10.1161/01.str.22.3.351. PMID: 2003304.

8 Banham NDG, Saw J, Hankey GJ, Ghia D. Cerebral arterial gas embolism proven by computed tomography following transthoracic echocardiography using bubble contrast. Diving Hyperb Med. 2020;50:300–2. doi: 10.28920/dhm50.3.300-302. PMID: 32957135. PMCID: PMC7819724.

9 Kindwall EP, Whelan HT, editors. Hyperbaric medicine practice. 2nd ed. Flagstaff (AZ): Best Publishing Company; 2002.

10 Sunami K, Takeda Y, Hashimoto M, Hirakawa M. Hyperbaric oxygen reduces infarct volume in rats by increasing oxygen supply to the ischemic periphery. Crit Care Med. 2000;28:2831–6. doi: 10.1097/00003246-200008000-00025. PMID: 10966258.

11 Lei Huang, Andre Obenaus. Hyperbaric oxygen therapy for traumatic brain injury. Med Gas Res. 2011;1:21. doi: 10.1186/2045-9912-1-21. PMID: 22146562. PMCID: PMC3231802.

12 Thom SR, Mendiguren I, Hardy K, Bolotin T, Fisher D, Nebolon M, et al. Inhibition of human neutrophil β2-integrin-dependent adherence by hyperbaric O_2. Am J Physiol. 1997;272:C770–7. doi: 10.1152/ajpcell.1997.272.3.C770. PMID: 9124510.

13 Fakkert RA, Karlas N, Schober P, Weber NC, Preckel B, van Hulst RA, Weenink RP. Early hyperbaric oxygen therapy is associated with favorable outcome in patients with iatrogenic cerebral arterial gas embolism: systematic review and individual patient data meta-analysis of observational studies. Crit Care. 2023;27(1):282. doi: 10.1186/s13054-023-04563-x. PMID: 37434172. PMCID: PMC10337083.

14 Hadanny A, Meir O, Bechor Y, Fishlev G, Bergan J, Efrati S. The safety of hyperbaric oxygen treatment – retrospective analysis in 2,334 patients. Undersea Hyperb Med. 2016;43:113–22. PMID: 27265988.

15 Mitchell SJ, Bennett MH, Moon RE. Decompression sickness and arterial gas embolism. N Engl J Med. 2022;386:1254–64. doi: 10.1056/NEJMra2116554. PMID: 35353963.

16 Banham N, da Silva E, Lippmann J. Cerebral arterial gas embolism (CAGE) during open water scuba certification training whilst practising a controlled emergency swimming ascent. Diving Hyperb Med. 2023;53:345–50. doi: 10.28920/dhm53.4.345-350. PMID: 38091595.

Acknowledgments

We express our sincere gratitude to the staff of the Neurosurgery Department in Yokohama Rosai Hospital for their cooperation in treating the patient. We are grateful to K Miyazaki and H Okamoto, doctors of the Respiratory Department in Yokohama Municipal Citizen's Hospital for their support. We also thank H Yamagata, K Sato, and A Nogaki, doctors of the Emergency Department in Yokohama Municipal Citizen's Hospital for their valuable input.

Conflicts of interest and funding: nil

Submitted: 23 August 2023
Accepted after revision: 12 November 2023