Early hyperbaric oxygen therapy for cerebral air embolism during atrial fibrillation ablation

Taner Ulus[1], Erdi Babayiğit[1], Ezgi Çamlı[1], Özlem Aykaç[2], Zehra Uysal Kocabaş[2], Atilla Özcan Özdemir[2], Erdinç Ercan[3]

[1] Department of Cardiology, Faculty of Medicine, Eskisehir Osmangazi University, Eskisehir, Turkey

[2] Department of Neurology, Faculty of Medicine, Eskisehir Osmangazi University, Eskisehir, Turkey

[3] Department of Aerospace Medicine, University of Health Sciences, Eskisehir, Turkey

Running title: Cerebral air embolism during ablation

Corresponding Author

Prof. Dr. Taner Ulus

Eskişehir Osmangazi Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı, Meşelik Kampüsü, 26480, Odunpazarı, Eskişehir, Türkiye

Tel: +90 505 748 1950

E-mail: tanerulusbuca@gmail.com

Abstract

This article has been accepted for publication and undergone full peer review but has not been through the copyediting, typesetting, pagination and proofreading process, which may lead to differences between this version and the Version of Record. Please cite this article as doi: 10.1111/pace.13475.

Cerebral air embolism is a potentially life threatening complication of left-sided ablation procedures. We present a 51-year-old woman with cerebral air embolism during atrial fibrillation cryoballoon ablation. Taking a deep breath while removing the dilatator was the most likely mechanism in our case. The patient was successfully treated with hyperbaric oxygen therapy at early stage and was discharged without any neurological sequelae.

Key words: Ablation, atrial fibrillation.

Introduction

Air embolism is a rarely seen during surgical and invasive procedures, but it is a potentially lethal complication. Air embolism may cause end-organ ischemia or infarction if there is insufficient collateral supply (1). Air emboli to the coronary or cerebral circulation can have major adverse consequences, even when the volume of air is small (1). Coronary artery air embolism during AF ablation has been reported previously (2,3). We present a case with cerebral air emboli during atrial fibrillation (AF) cryoballoon ablation. The patient was successfully treated with with hyperbaric oxygen (HBO) therapy and was discharged without any neurological sequelae.

Case report

A 51-year-old woman was referred to our clinic for AF ablation. The patient suffered from paroxysmal AF attacks resistant to antiarrhythmic therapy including amiodarone and propafenone for two years. In addition, she had histories of systemic hypertension and Parkinson's disease. Medications were metoprolol 100 mg twice a day, rivaroxaban 20 mg once a day, candesartan cilexetil/hydrochlorothiazide 16/12.5 mg once a day, venlafaxine

hydrochloride 150 mg once a day and madopar 125 mg three times a day. The patient was taken to the electrophysiology laboratory after taking her approval. The procedure was performed under conscious sedation. Sedation was provided with dexmedetomidine hydrochloride and midazolam. 6F and 8F sheaths into the right femoral vein and 6F sheath into the right femoral artery were inserted. A decapolar catheter was inserted into the coronary sinus and a pigtail catheter was placed in the aortic root. Transseptal puncture was performed and a transseptal sheath (8F SL1™, St. Jude Medical) was inserted into the left atrium. Intravenous unfractionated heparin (100 U/kg) was administered and the pigtail catheter was withdrawn. The transseptal sheath was replaced with a steerable sheath (FlexCath Advance™, Medtronic, Minneapolis, MN, USA). Dilatator and guidewire were removed. At the same time, the patient took a deep breath after a short period of apnea. At this time, air was seen in the side lumen of the Flexcath. The three-way tap immediately opened and the air was taken out to a large extent. However, the patient developed sudden respiratory distress. The patient had a generalized tonic seizure. She had neurological examination revealed dysarthria and left-sided hemiparesis. Hemodynamic findings were stable. The patient was conscious and she had spontaneous respiration. The Flexcath was removed and the procedure was stopped. 100% oxygen was administered, dexmedetomidine hydrochloride was stopped, and midazolam sedation was reversed with flumazenil. A transthoracic echocardiography and electrocardiogram did not show any abnormality. The neurology department immediately evaluated the patient. A computed tomography (CT) scan of the cerebrum showed several intracranial air embolic bubbles in the right sided parietal area (Figure 1). The bubbles were located in the distal branches of the cerebral arteries. For this reason, mechanical aspiration or thrombectomy the air was not performed. A computed

tomography (CT) scan of the cerebrum showed several intracranial air embolic bubbles in the right-sided parietal area (Figure 1). The bubbles were located in the distal branches of the cerebral arteries. For this reason, mechanical aspiration of the air was not applied.

The patient was admitted to the intensive care unit. Physiological saline 100 ml per hour, and antiedema therapy including mannitol and dexamethasone 4 mg three times a day was started. Intravenous levetiracetam was loaded (1500 mg). After stabilization, 4 h after the air embolization, the patient was treated with HBO therapy according to "US Navy Treatment of Arterial Gas Embolism or Serious Decompression Sickness Protocol" and we decided to continue HBO treatment with US Navy Treatment Table 6 (4), and followed by 2.4 ATA (atmospheres absolute) 120 minutes HBO therapy twice a day for 2 days. On the second day of HBO therapy, dysarthria and left-sided hemiplegia were completely resolved and the appearance of intracranial air was completely lost in the second CT (Figure 2). The patient was discharged from the intensive care unit and received 2.4 ATA 120 minutes HBO therapy for another 5 days. Patient received total 11 HBO therapies in consecutive seven days. Flecainide 100 mg and levetiracetam 500 mg twice daily were added. Dexamethasone was reduced on the third day of the event and discontinued on the fifth day. The patient was discharged on the eighth day of the event without any neurological sequelae.

Discussion

Arterial air embolism may be due to direct instillation of air into the arterial tree (2,3) or may be associated with atrial-esophageal fistula (5) during and/or after AF ablation. Catheters are the reason for the vast majority of air embolisms in the acute phase. Possible mechanisms causing air embolism from introducing catheters are rapid removal of catheters and dilators,

multiple catheter exchanges, deep sedation and prolonged apnea periods with deep breaths, and air-opened or loosened hemostasis valves (3,6). In our case, the most likely cause for cerebral air embolism was air being suctioned into the Flexcath. Taking a deep breath while removing the dilatator was the most likely mechanism for air entry. Deep breathing was probably related to a short period of apnea induced by sedation. Preventive measures to avoid aeroembolism are a sedative use with less depressant effect on the respiratory system, avoiding the rapid withdrawal of the dilator, continuous flushing with heparinized saline through a haemostatic valve (6).

When air enters the circulation, it forms bubbles, thereby impairing the circulation. The clinical signs and symptoms related to the mechanism of air entrapment and the location of the air embolism (1). Neurological symptoms may occur as it is in our case. 100% oxygen treatment administered by a non-rebreather mask maximizes end-organ oxygenation (7). High flow oxygen may also aid the reabsorption of nitrogen gas from the bubble into the blood, reducing the size of the air embolus (7). Air emboli may be treated by several approaches, such as aspiration of the air or HBO therapy (8,9). Air aspiration was not applied in our case because the bubbles were in the distal branches of the cerebral arteries.

HBO therapy plays a key role in the treatment of air embolism (1). It acts immediately on the air bubbles by increasing the ambient pressure and the oxygen tension in the blood (10-13). The high blood oxygen tension provides an oxygen diffusion gradient forcing oxygen into and nitrogen out of the bubble, reducing the size of the bubble (10-13). The bubble also seems to form an inflammatory response, which can be reduced by a rapid reduction of the volume of the bubble (10,13). HBO therapy also downregulates adhesion between the

endothelium and leucocytes in inflammated tissue (14). Evidence suggests that when indicated, it must be stressed that HBO therapy is started within the first four to six hours after onset of neurologic symptoms, and also at any manifestation of end-organ damage, cardiopulmonary, or hemodynamic compromise (15,16). In our case, HBO therapy was started early and neurological findings were completely resolved on the second day of treatment.

Conclusion

Cerebral air embolism is a potentially life threatening complication of left-sided ablation procedures. Preventive measures should be taken to avoid this complication. Operators should maintain high degree of suspicion for air emboli. After stabilization, advanced managements, including HBO therapy should be considered at early stage.

References

1. McCarthy CJ, Behravesh S, Naidu SG, Oklu R. Air Embolism: Diagnosis, Clinical Management and Outcomes. Diagnostics (Basel). 2017;7. pii: E5.

2. Cay S, Ozeke O, Ozcan F, Topaloglu S, Aras D. Coronary Air Embolism during Cryoablation of Atrial Fibrillation: A Catastrophic Complication and Its Management. J Atr Fibrillation. 2017; 10(3): 1728.

3. Ahmad K, Asirvatham S, Kamath S, Peck S, Liu X. Successful interventional management of catastrophic coronary arterial air embolism during atrial fibrillation ablation. HeartRhythm Case Rep. 2015;2:153-6.

4. Commander, Naval Sea Systems Command U.S. navy diving manual, (air diving). Chapter 8, Vol 1. Flagstaff, AZ: Best Publishing Company, 1993.

5. Wuestenberghs F, Pirson N, Bulpa P, Fervaille C, Dive A. Iatrogenic cerebral air embolism revealing an atrial-esophageal fistula. Intern Emerg Med 2017;12:715–6.

6. Kuwahara T, Takahashi A, Takahashi Y, Kobori A, Miyazaki S, Takei A, Fujii A, et al. Clinical characteristics of massive air embolism complicating left atrial ablation of atrial fibrillation: lessons from five cases. Europace. 2012 Feb;14(2):204-8.

7. Ie SR, Rozans MH, Szerlip HM. Air embolism after intravenous injection of contrast material. South Med J. 1999;92:930-3.

8. Patterson MS, Kiemeneij F. Coronary air embolism treated with aspiration catheter. Heart 2005;91:e36.

9. Khan M, Schmidt DH, Bajwa T, Shalev Y. Coronary air embolism: incidence, severity, and suggested approaches to treatment. Cathet Cardiovasc Diagn 1995;36:313–8.

10. Muth CM, Shank ES. Gas embolism. New Engl J Med 2000; 342 (7): 476–82.

11. Grim PS, Gottlieb LJ, Boddie A, Batson E. Hyperbaric oxygen therapy. JAMA 1990; 263 (16): 2216.

12. Van Hulst RA, Klein J, Lachmann B. Gas embolism: Pathophysiology and treatment. Clin Physiol Functional Imag 2003; 23: 237–46.

13. Shank ES, Muth CM. Decompression illness and iatrogenic gas embolism. Int Anesthesiol 2000; 38 (1): 111–38.

14. Buras JA, Stahl GL, Svoboda KK, Reenstra WR. Hyperbaric oxygen downregulates ICAM-1 expression induced by hypoxia and hypoglycemia: the role of NOS. Am J Physiol Cell Physiol 2000; 278: C292–302.

15. Leach RM, Rees PJ, Wilmshurst P. Hyperbaric oxygen therapy. BMJ. 1998;317:1140-3.

16. Closon M, Vivier E, Breynaert C, Duperret S, Branche P, Coulon A, De La Roche E, 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.

Figure legends

Figure 1. Axial non-contrast CT of the head. Arrows show intravascular pockets of air on several cut. CT: Computed tomography.

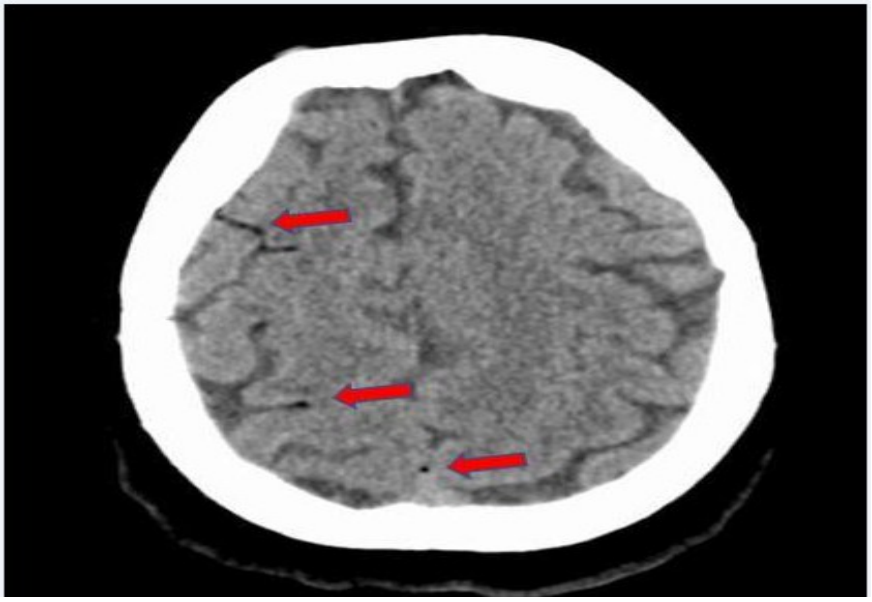

Figure 2. Axial non-contrast CT of the head. The appearance of intracranial air was completely lost in the second CT. CT: Computed tomography.