

Cerebral air embolization in the Electrophysiology Laboratory during Transseptal Catheterization: Curative treatment of acute left hemiparesis with prompt hyperbaric oxygen therapy

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Received: 13 April 2006 / Accepted: 9 June 2006 / Published online: 11 October 2006
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Abstract We present a case of a 44 year-old man with medically refractory symptomatic paroxysmal atrial fibrillation in whom the initial attempt at left atrial catheter ablation was complicated by coronary and cerebral arterial air embolization during transseptal catheter exchange. The manifestations, management, and long term outcomes are detailed. Following the case report is a review of published reports and contemporary management strategies for treatment of acute air embolization. Dramatic clinical consequences can be aborted by prompt intervention including volume loading, oxygenation, lidocaine, and hyperbaric oxygen therapy.

Keywords Transseptal · Left atrial catheter ablation · Atrial fibrillation · Air embolism · Complication · Hyperbaric oxygen

1 Introduction

More patients are undergoing ablation procedures as treatment for atrial fibrillation. During the development of

left atrial catheter ablation (LACA) procedures, several unexpected complications have been identified such as pulmonary vein stenosis and esophageal perforation or fistula [1, 2]. The anatomic approach to LACA mandates left atrial access through the interatrial septum utilizing transseptal catheterization (TC). While TC has been described since 1958, and used by electrophysiologists for many years, the prevalence of atrial fibrillation and the potentially curative LACA have resulted in a significant increase in the number of patients undergoing TC procedures [3, 4]. Potential complications include perforation of the cardiac tissues, perforation of the aorta, thromboembolism and air embolization, which may occur despite technique modifications such as intracardiac echocardiography and continuous sheath perfusion [5, 6]. A recently published voluntary multicenter European survey of 5,520 TC cases estimated a 0.79% complication rate [5]. One rare, but potentially devastating, complication is cerebral air embolization (AE). Curative treatment of cerebral AE with hyperbaric oxygen therapy (HBOT) is well described, but has not been addressed in the electrophysiology literature [7, 8]. Volume loading, oxygenation, and lidocaine may also play a role in treating AE [9, 10]. We describe a case in which AE occurred during TC for LACA, including the manifestations, management, and long term outcomes. Contemporary management strategies for treatment of AE are reviewed.

2 Case report

The patient is a 44 year-old man without structural heart disease or other medical problems, who suffered from medically refractory symptomatic paroxysmal atrial fibrillation (PAF). He presented as a self-referral for catheter

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based ablation of atrial fibrillation, having had prior successful ablation for sustained atrial flutter at an out-of-state institution. Despite therapy with flecainide, he had recurrent symptoms lasting from several minutes to several hours, requiring hospitalization. He consumes 1–2 alcoholic drinks daily, does not smoke, and is an experienced scuba diver. On physical examination, he was normotensive, athletic appearing and mildly overweight. There was an irregularly irregular rhythm with no other abnormalities. CBC, creatinine, electrolytes, INR, and TSH were normal. ECG showed sustained atrial fibrillation at 84 bpm. QRS axis and morphology were normal. Pre-ablation transesophageal echocardiography showed normal left ventricular function, right and left atrium at upper limits of normal size, no atrial septal defect, no smoke, and no thrombi.

The patient was taken off antiarrhythmic therapy for 72 h prior to the procedure. Written informed consent was obtained. Electrophysiologist directed intravenous conscious sedation utilizing midazolam, fentanyl, and phenergan maintained patient comfort. After sterile prep and drape, the right and left femoral veins were accessed with side-port sheaths, through which three quadripolar 4 French Medtronic diagnostic catheters were positioned into the high right atrium, His bundle region, and the right ventricular apex. Baseline rhythm was atrial fibrillation, with normal HV interval of 45 ms, and normal QRS duration of 80 ms. Left atrial access by TC was easily completed using biplane fluoroscopy facilitated with a modified technique after Swartz et al. A flushed Daig 8-french SL-2 sheath was advanced over the dilator after the septum was punctured with a Daig BRK-1 needle. There was no evidence of acute complication. The dilator and needle were removed, then the sheath was carefully aspirated, flushed, and 5,000 U of heparin were administered intravenously. After initial pulmonary vein potential mapping, the sheath was removed over a wire. A flushed Daig SL-1 guiding sheath with dilator was advanced over the wire to allow better catheter manipulation for ablation. The dilator was removed, and the sheath was carefully aspirated and flushed. During this sheath exchange fluoroscopic contrast changes consistent with intracardiac air were noted. Continuous sheath irrigation had not yet been initiated. Density changes were noted over the left atrial area and over the left ventricular apex. Co-incident to this, the patient developed acute inferior ST elevation, associated with bradycardia and hypotension, without chest pain. Attempted aspiration using the transseptal sheath reduced the amount of visible intracardiac air. The right femoral artery was accessed, and immediate right coronary angiography demonstrated a mid vessel lucency, which resolved after a second injection of contrast. The ST segments, blood pressure, and heart rate normalized. The patient was pain-free, conscious and conversant, but he now had a persistent

left hemiparesis. In hopes of minimizing any acute neurologic injury, 120 mg of methylprednisolone was given intravenously. After the catheters were withdrawn, he was directly transported, sheaths sutured in place, to the hyperbaric oxygen chamber for treatment.

Within 77 min of the recognition of AE, he was in the hyperbaric chamber (Sechrist Industries, Model 2500 Monoplace Hyperbaric Chamber) which was then brought to 3.0 atm of pressurization at a rate of 5.0 psig/min for 90 min, based on a modified US Navy Treatment Table 6 protocol. Less than 10 min after reaching 3.0 atm, use of the left hand returned. By 1 h gross motor skills returned to both left sided extremities. At the end of the initial treatment finer motor skills appeared, so that he was able to make a fist. Additional pressurization cycles were conducted up to 2.0 atm, for an additional 150 min on the initial day, and 90 min each on days two through four. The patient was hospitalized in the intensive care unit, placed onto amiodarone and given lovenox for persistent atrial fibrillation. Non-contrast head CT immediately after hyperbaric treatment showed no ischemia, no parenchymal hematoma, no midline shift, no hydrocephalus, and no extra-axial fluid collection. Initial cardiac enzymes showed acute elevation, with troponin-I at 1.24 $\mu\text{g/l}$, and total CPK at 108 U/l (5.3% MB), which all declined on subsequent tests. Three days after the AE induced myocardial infarction, an echocardiogram showed normal left ventricle size and systolic function with no pericardial effusion. He was discharged home on warfarin and amiodarone 4 days after the AE following electrical cardioversion to restore sinus rhythm and provide better rate control. At that point the patient felt he had essentially full neurologic recovery.

The patient has remained physically active, with complete resolution of any neurologic sequelae. Despite amiodarone, he continued to have breakthrough episodes of PAF, which made him lethargic and fatigued. He was referred to another center for ablation, but was turned down. Eight months after the AE he underwent a second attempt at LACA, with successful pulmonary vein entrance block of the left upper, left lower, and the right upper pulmonary veins. The right inferior was not isolated. Five months after LACA, he has now remained asymptomatic without clinical evidence of atrial fibrillation on flecainide therapy. At 12 months following the acute AE he has remained without residual neurologic sequelae.

3 Discussion

Almost five decades since TC was first described AE can still occur, despite improved strategies for prevention [6]. Meticulous attention to sheath management is paramount, with visualized aspiration and flushing, particularly when

not working under a carbon dioxide field. Continuous sheath perfusion, which is used to reduce thromboembolism, had not yet been initiated during this catheter exchange [4]. In less than a 3-year time span, three AE during TC for LACA were reported from one large United States tertiary referral institution [7, 8]. The number of events in lower volume settings is not known, however a voluntary survey of 33 Italian centers suggests a reported incidence of recognized AE below 0.1%, but not all TC procedures were for LACA [16]. Gas embolism, more specifically AE, have been reported by a myriad of medical specialties [11–13]. In the case presented here, the AE appeared to develop during catheter exchange. This risk might be further reduced by improved catheter technology, such as steerable sheaths obviating the need for exchange.

Arterial AE can distribute to virtually any organ, but have devastating clinical sequelae when they enter the end-arteries. This can lead to the hypoxic manifestations of myocardial injury and cerebrovascular accidents. In this case urgent coronary angiography documented right coronary AE, which resolved with repeated contrast injection. Routine diagnostic modalities to identify AE in the terminal arterial circulations lack sensitivity, and diagnosis is typically based on the appropriate clinical scenario with possible air identified in the antecedent cardiac chambers. Clinical diagnosis in our case was made by direct observation of neurologic deficits on physical exam coupled with the fluoroscopic evidence of intracardiac air contrast. Air rather than thrombus was the presumptive mechanism of the neurologic injury, and delaying treatment to obtain either CT or MRI was thought to be impractical and unnecessary. Other reported sequelae of procedure-related cerebral AE include agitation, hypotension, nausea, dizziness, headaches, seizures, and visual disturbances. Anesthesia and sedation may mask or alter the clinical symptoms, thus complicating the evaluation and delaying diagnosis of the patient's clinical condition [11]. Our protocol uses mild sedation, making prompt assessment possible.

Therapy for AE include intervention to prevent a recurrence of venous air entry; oxygen therapy for the hypoxia, and reduction in the size of the air embolus by establishing a diffusion gradient; cardiopulmonary support in cases of circulatory collapse; and airway protection in comatose patients [11, 14, 15]. Central venous catheter extraction of the residual AE if localized in the right atrium or right ventricle has been described [9]. In this case, the intravascular air could not be completely removed from the arterial circulation. Current management scenarios recommend against Trendelenberg positioning, given that the pressure of the arterial system is sufficient to override any buoyancy characteristics of the AE, and the head-down position could exacerbate cerebral edema. Recurrent small

AE may cause changing neurological symptoms. Keeping the patient supine, maintaining euglycemia, and volume loading with saline to maintain adequate cerebral blood flow have been advised [7, 8].

The acute use of anticoagulants is now generally discouraged given the risk of hemorrhage into infarcted tissue, however many LACA patients will receive heparin during the procedure. Magnetic resonance imaging has demonstrated asymptomatic cerebral embolism after LACA [16]. Anticoagulant therapy was resumed in this case in the absence of cerebral infarction because of the ongoing atrial fibrillation.

Corticosteroid use has been controversial, and although used in this case, it is typically not employed given its limited use in this form of cytotoxic cerebral edema [11].

Lidocaine therapy has proven cerebral protective effects from ischemia induced by AE and reduces elevated intracranial pressure in animal studies [10]. The cerebral protective effects of perioperative lidocaine infusion assessed by changes in neuropsychological test scores has been demonstrated in a clinical trial [17]. This patient did not receive lidocaine. However, administration of lidocaine in a bolus dose of 1.5 mg per kg with maintenance infusion thereafter can be recommended in patients with significant cerebral artery AE burden [11].

Hyperbaric-oxygen therapy (HBOT), first documented in 1662 by Henshaw, consists of therapy with 100% oxygen at a pressure greater than 1 atm [18]. Figure 1 shows a typical hyperbaric chamber. This therapy leads to a decrease in the volume of the intravascular air bubbles due to its facilitation of a greater ambient pressure. At



Fig. 1 Typical hyperbaric oxygen chamber. Photo is courtesy of Caryn Oldham, Sechrist Industries, <http://www.sechristind.com>.

2.8 atm, the air bubble volume is reduced by approximately two-thirds. HBOT also leads to a diffusion gradient with an increase of oxygen dissolution in the plasma and therefore improved delivery of oxygen to ischemic peripheral tissues. HBOT may also minimize brain edema from cerebral AE by stabilizing the blood–brain barrier and reducing the permeability of the cerebral vessels [13]. HBOT treatments are termed “tables”; the most common are for wound healing, decompression sickness, and carbon monoxide poisoning. The initial treatment in this case was the protocol used to treat scuba divers with decompression sickness. Initial HBOT may last up to 8 h, depending upon symptom relief. The chamber is brought to a pressure equivalent to 60 ft under seawater for up to 100 min. Immediate therapy is intuitively preferred, however, delayed treatment (over 48 h later) has been shown to provide significant clinical benefits [19–21]. In this case the time from recognition until initiation of HBOT was 77 min. Multiple treatments may be required before the maximum clinical benefit is noted. This patient's scuba diving experience may have been clinically beneficial.

The only absolute contraindication to HBOT is untreated pneumothorax. Also, patients should not undergo HBOT if they have recently taken doxorubicin, disulfiram, *cis*-platinum, or mafenide acetate. Relative contraindications include upper respiratory infections, high fevers, seizure disorders, emphysema with CO₂ retention, specific thoracic

abnormalities, uncontrolled hypertension, asthma, hypoglycemia, and pregnancy. Despite the lack of prospective clinical trials with HBOT in iatrogenic arterial AE, the known physiologic and therapeutic benefits warrant its use as first-line therapy for cerebral AE [11, 22, 23].

To our knowledge, the literature contains three reported cases of cerebral AE occurring during radiofrequency ablation procedures which were treated with HBOT [7, 8]. Table 1 shows the clinical features of these three and our one patient. These were all during treatment of PAF with TC access for LACA. In all but our case, the initial neurologic symptoms seemed to wax and wane. Accuracy of neurologic assessment during anesthesia may have influenced recognition. We could not find these three cases reported in the electrophysiology literature. Air bubbles were reported in all cases, which coupled with acute neurologic changes, supports the diagnosis of cerebral AE. All of the neurologic imaging studies obtained were normal, suggesting that delay of HBOT to obtain neurologic imaging studies may not be necessary given the clinical diagnosis of cerebral AE.

4 Conclusion

With the advent and growth of radiofrequency techniques for atrial fibrillation ablation, the volume of cardiac trans-

Table 1 Summary of clinical findings from patients who underwent HBOT for cerebral AE during TC for LACA. The first three patients were reported by one institution. The fourth patient triggered this report

Clinical features of cerebral air emboli patients treated with hyperbaric oxygen therapy				
Patient age/gender	29 years/male	60 years/male	62 years/male	44 years/male
Indication/procedure	PAF/LACA	PAF/LACA	PAF/LACA	PAF/LACA
Neurologic signs	Syncope; apnea; confusion; left hemiparesis; left homonymous hemianopsia; waxing-waning	Confused; agitated; aphasic; right hemiparesis; right facial droop; waxing-waning	Agitated; object agnosia; aphasia; right hemiparesis; waxing-waning	Left hemiparesis
Air bubbles seen	Yes	Yes	Yes—in sheath	Yes—fluoroscopic
Associated with MI	Unknown	Unknown	Yes	Yes
CNS studies (Pre-HBOT)	CT; Cerebral Angiography	CT	CT	None
CNS studies (Post-HBOT)	DWI-MRI	DWI-MRI x2; SPECT	None	CT
Time to HBOT	6 h	3 h	3 h	77 min
Symptom resolution	@ 1 month had left arm weakness	@ several weeks impaired language returned to baseline	<48 h	<24 h
Heparin during LACA	Unknown	Unknown	Yes	Yes

CNS is central nervous system, CT is computed tomography, DWI-MRI is diffusion-weighted magnetic resonance imaging, SPECT is single photon emission computed tomography.

septal procedures have increased dramatically in the last several years. Despite careful technique by experienced operators, the potential for complications remains [1, 4–6, 16]. Although the incidence of cerebral AE appears to be small, it can have devastating consequences. Broadened knowledge of management options is paramount for the timely prevention of long term neurologic effects in the rare patient who might suffer cerebral AE.

We present here a case of cerebral and coronary arterial AE with their dramatic acute clinical consequences, where prompt intervention aborted long term disability. Cerebral AE phenomena likely represent not only an underreported, but underappreciated clinical scenario with significant possibility for improvement in clinical outcomes. Rapid recognition, clinical diagnosis and prompt initiation of therapy, including 100% oxygen, volume loading, intravenous lidocaine, and prompt HBOT can reverse the neurologic injury. Similar to other acute ischemic syndromes, time to treatment appears important in the outcome, and transfer to a regional HBOT center should be considered if necessary. According to estimates by the Undersea and Hyperbaric Medicine Society, which maintains a list of hyperbaric chambers worldwide on their website, there are approximately 458 hyperbaric centers in the United States [24]. It seems imperative that as clinicians striving to optimize patient outcomes, we should build knowledge about HBOT centers near our institutions to minimize the potentially reversible morbidity and mortality in our patients from arterial AE.

References

- Marine, J. E., Doug, J., & Calkins, H. (2005). Catheter ablation therapy for atrial fibrillation. *Progress in Cardiovascular Diseases*, 48(3), 178–192.
- Moak, J. P., Moore, H. J., Lee, S. W., Giglia, T. M., Sable, C. A., Furbush, N. C., et al. (2000, December). Case report: Pulmonary vein stenosis following RF ablation of paroxysmal atrial fibrillation: Successful treatment with balloon dilation. *J Interv Card Electrophysiol*, 4(4), 621–631.
- Swartz, J. F., Tracy, C. M., & Fletcher, R. D. (1993). Radio-frequency endocardial catheter ablation of accessory atrioventricular pathway atrial insertion sites. *Circulation*, 87(2), 487–499.
- Cauchemez, B. (2004). High-flow perfusion of sheaths for prevention of thromboembolic complications during complex catheter ablation in the left atrium. *Journal of Cardiovascular Electrophysiology*, 15(3), 276–283.
- De Ponti, R., Cappato, R., Curnis, A., Della Bella, P., Padeletti, L., Raviele, A., et al. (2006, March). Trans-septal catheterization in the electrophysiology laboratory: Data from a multicenter survey spanning 12 years. *Journal of the American College of Cardiology*, 47, 1037–1042.
- Gonzalez, M. D., Otomo, K., Shah, N., Arruda, M. S., Beckman, K. J., Lazzara, R., et al. (2001). Transseptal left heart catheterization for cardiac ablation procedures. *J Interv Card Electrophysiol*, 5(1), 89–95.
- Augoustides, J., Mancini, D. J., & Marchilinski, F. (2002). An unusual cause of intraoperative confusion in the electrophysiology laboratory. *Journal of Cardiothoracic and Vascular Anesthesia*, 16(3), 351–353.
- Hinkle, D. A., Raizen, D. M., McGarvey, M. L., & Liu G. T. (2001). Cerebral air embolism complicating cardiac ablation procedures. *Neurology*, 56, 792–794.
- Palmon, S. C., Moore, L. E., Lundberg, J., & Toung, T. (1997). Venous air embolism: A review. *Journal of Clinical Anesthesia*, 9, 251–257.
- Evans, D. E., Kobrine, A. I., LeGrys, D. C., & Bradley, M. E. (1984). Protective effect of lidocaine in acute cerebral ischemia induced by air embolism. *Journal of Neurosurgery*, 60, 257–263.
- Muth, C. M., & Shank, E. S. (2000) Gas Embolism. *NEJM*, 342(7), 476–482.
- Turgeman, Y., Antonelli, D., Atar, S., & Rosenfeld, T. (2004). Massive transient pulmonary air embolism during pacemaker implantation under mild sedation. *PACE*, 27, 684–685.
- Fowler, M. J. (2005). Diffuse cerebral air embolism treated with hyperbaric oxygen. *Journal of Neuroimaging*, 15, 92–96.
- Van Liew, H. D., Conkin, J., & Burkard, M. E. (1993). The oxygen window and decompression bubbles: Estimates and significance. *Aviation, Space, and Environmental Medicine*, 64, 859–865.
- Kytta, J., Tanskanen, P., & Randellm, T. (1996). Comparison of the effects of controlled ventilation with 100% oxygen, 50% oxygen in nitrogen, and 50% oxygen in nitrous oxide on responses to venous air embolism in pigs. *British Journal of Anaesthesia*, 77, 658–661.
- Lickfett, L., Hackenbroch, M., Lewalter, T., Selbach, S., Schwab, J., Yang A., et al. (2006, January). Cerebral diffusion-weighted magnetic resonance imaging: A tool to monitor the thrombogenicity of left atrial catheter ablation. *Journal of Cardiovascular Electrophysiology*, 17(1), 1–7.
- Mitchell, S. J., Pellett, O., & Gorman, D. F. (1999). Cerebral protection by lidocaine during cardiac operations. *Annals of Thoracic Surgery*, 67, 1117–1124.
- Henshaw, N. (1664). Aero-chalinos. Dublin, *Dancer*.
- Blanc, P., Boussuges, A., Henriette, K., Sainty, J. M., & Deleflie, M. (2002) Iatrogenic cerebral air embolism: Importance of an early hyperbaric oxygenation. *Intensive Care Medicine*, 28(5), 559–563.
- Moon, R. E, de Lisle Dear, G., & Stolp, B. W. (1999). Treatment of decompression illness and iatrogenic gas embolism. *Respiratory Care Clinics of North America*, 5, 93–135.
- Shank, E. S., & Muth, C. M. (2000). Decompression illness, iatrogenic gas embolism, and carbon monoxide poisoning: The role of hyperbaric oxygen therapy. *International Anesthesiology Clinics*, 38, 111–138.
- Benson, J., Adkinson, C., & Collier, R. (2003). Hyperbaric oxygen therapy of iatrogenic cerebral arterial gas embolism. *Undersea & Hyperbaric Medicine*, 30, 117–126.
- “<http://www.virginiamason.org/dbhyperbaricoxygen/default.htm>” Source site accessed March 1, 2006.
- “<http://www.uhms.org/Chambers/CHAMBER%20DIRECTORY2.asp>” Source site accessed March 19, 2006.