CASE REPORT

Paradoxical air embolism and neurological insult during removal of a pulmonary artery catheter introducer

Philippe Frederick Bowles,¹ Christopher Lear,² Marco Maccario,² Robert Kong²

¹Department of ENT, Brighton and Sussex University Hospitals NHS Trust, Brighton, UK

²Department of Anaesthetics, Brighton and Sussex University Hospitals NHS Trust, Brighton, UK

Correspondence to

Dr Philippe Frederick Bowles,
philbowles@doctors.org.uk

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SUMMARY

A 54-year-old man was admitted to the intensive care unit following cardiac surgery. On day 6 postoperatively, approximately 2–3 min following the removal of the pulmonary artery (PA) catheter introducer sited in the right internal jugular vein, the patient became agitated, confused and then unresponsive. He was urgently moved onto the bed, laid supine, sedated with a propofol infusion, intubated and mechanically ventilated. A bedside transthoracic echocardiogram revealed extensive multiple air emboli in all cardiac chambers and review of the patient's intraoperative imaging confirmed the presence of patent foramen ovale (PFO). The following morning, sedation was discontinued and the trachea extubated. The patient was later transferred to the ward and then discharged home with no evidence of neurological deficit. This case reminds us of the importance of strict adherence to safety protocols when manipulating centrally positioned catheters and the high prevalence of undiagnosed PFO.

BACKGROUND

Pulmonary artery (PA) catheters provide a valuable means of monitoring cardiovascular function during cardiac surgery.¹ Complications associated with the insertion, use and removal of PA catheters are well documented.^{1–2} We report a case of paradoxical air embolism (PAE) and neurological insult after the removal of a PA catheter introducer in a patient with patent foramen ovale (PFO).

This case demonstrates the risk of serious and potentially fatal consequences that may be associated with routine procedures if safety protocols are not followed. This case also reminds us of the high incidence of undiagnosed PFO. The imaging in this case illustrates one mechanism by which air embolism may lead to neurological insult.

CASE PRESENTATION

A 54-year-old man was admitted to the intensive care unit after triple coronary artery bypass grafting and mitral valve annuloplasty having suffered an acute inferior myocardial infarction 1 month earlier. He presented for surgery with a globally enlarged heart, poor left ventricular function, mitral regurgitation, pulmonary hypertension and renal impairment. A PFO with left to right shunt was discovered during routine intraoperative transoesophageal echocardiography (figure 1). The PFO was not closed intraoperatively as it was not considered to be haemodynamically important.

Cardiopulmonary bypass lasted for 151 min, with an ischaemic time of 94 min.

The patient required mechanical ventilation until the first postoperative day. Inotropic support, consisting of dobutamine, norepinephrine and milrinone, was weaned over the following days and discontinued on day 4. The PA catheter was removed on day 5 but the introducer was left in situ as a precaution for ease of access should further venous cardiac monitoring be required. The patient's neurocognitive function was clinically normal by this time.

During the afternoon of day 6, the introducer catheter was removed while the patient was awake, breathing spontaneously and semirecumbent in a chair. The insertion site appeared to be dry after withdrawal of the introducer and an occlusive dressing was not applied. Approximately 2–3 min later, the patient became agitated, confused and then unresponsive. A rapid radial pulse was just palpable however, a non-invasive blood pressure recording was unobtainable. An arterial line was inserted and a blood pressure of 108/60 was recorded. Five-lead ECG monitoring showed sinus tachycardia with a pulse of 120 bpm. Inotropic support was not restarted. The patient remained unresponsive and the trachea was intubated to enable mechanical ventilation.

INVESTIGATIONS

A bedside transthoracic echocardiogram was performed by a cardiologist within 10 min of the onset of the neurological event. This revealed extensive multiple air emboli in all cardiac chambers (figure 2).

A CT of the brain approximately 2 h later was reported by a consultant neuroradiologist as showing no significant abnormality.

DIFFERENTIAL DIAGNOSIS

A patient with a history of coronary vascular disease recovering from cardiac surgery may become acutely unresponsive and haemodynamically unstable for several reasons. Compromise to cardiac, respiratory or the central nervous systems may all manifest in this way, for example, acute coronary syndrome, cardiac tamponade, postoperative venous thromboembolism, acute respiratory distress syndrome (bronchiectasis) and acute neurological insult. In this case, the preponderance of neurological symptoms along with the absence of any pain or overt respiratory distress, favoured a

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Figure 1 Intraoperative transoesophageal echocardiogram showing patent foramen ovale with left to right atrial shunt.

neurological diagnosis. The availability of a bedside transthoracic echocardiogram showing multiple air emboli in the left atrium further supported this.

TREATMENT

Following symptom onset, the patient was urgently moved onto the bed and laid supine. An oropharyngeal airway was inserted and a bag and mask circuit was used to provide oxygen and assist his shallow breathing. The patient was sedated with a propofol infusion, intubated and mechanically ventilated.

OUTCOME AND FOLLOW-UP

The patient awoke after the sedation was stopped and the trachea was extubated the following day. On day 9 he was transferred to the ward and subsequently discharged home with no evidence of neurological deficit. Routine follow-up of the patient 3, 6 and 12 months, respectively, confirmed complete resolution of this episode and normal neurological function postoperatively.

Figure 2 Transthoracic echocardiography after removal of the pulmonary artery catheter introducer showing multiple air emboli in the left atrium and left ventricle.

DISCUSSION

An EMBASE and MEDLINE database search and review of the literature confirmed air embolism as a well recognised complication of central venous catheter (CVC) use. However, despite the wide availability of guidance on the best practice and safety protocols for the safe removal of CVCs, preventable errors occur. The case underlines the need for particular caution in the removal of central devices such as CVCs and pulmonary artery catheters as probe PFO may be present in 25–30% of the population.^{3 4}

Usual intra-atrial pressures suggest that in the presence of an interatrial communication the resulting shunt would be from left to right.⁵ However, the effects of mechanical ventilation, thromboembolism, air embolism, inferior vena cava repositioning and increased intra-abdominal pressure are all known to cause a reversal of this shunt by increasing right atrial pressure above that of the left atrium.⁶ Pathological conditions increasing right ventricular pressure (eg, pulmonary hypertension) and physiological manoeuvres (eg, Valsalva manoeuvre) also favour right to left shunting by raising right atrial pressure.⁷ A further mechanism thought to encourage right to left shunting is the direction of blood flow toward the atrial septum by eustachian valves and Chiari networks, are associated with PFO.⁷ The resulting right to left shunt can lead to the passage of deoxygenated blood, thrombi and air from the venous side into the systemic circulation.

We suggest that massive air embolism in the pulmonary artery, in a patient with pre-existing severe pulmonary hypertension, precipitated a rise in the right ventricular pressure and subsequently in the right atrial pressure. Although PAE may occur in the absence of PFO or atrial septal defect via intrapulmonary right to left shunting, in this case it seems likely that as right atrial pressure increased to exceed that in the left atrium a reversal in the direction of the shunt through the PFO resulted. Air communicated into the right side of the heart by the removal of the PA catheter introducer that may have traversed the atrial septum to the systemic circulation causing a neurological event.

Further to the effects of mechanical obstruction venous air emboli are known to cause damage to the vascular endothelium, resulting in the release of vasoactive and proinflammatory mediators that affect haemodynamic stability by increasing

pulmonary hypertension and right ventricular afterload.⁸ Specifically, endothelin isopeptides have recently been identified in rat models as potent vasoconstrictors produced by the pulmonary endothelium in response to endothelial injury by embolic material such as entrained air.⁹ As a result, pulmonary hypertension and subsequent increase in right heart pressure is greater than that determined by mechanical obstruction alone. Such factors may have contributed to a reversal of blood flow through the PFO.

Sinuses around CVCs have been shown to develop after 24 h and may lead to an increase the risk of air embolism.^{9 10} The PA catheter in this case had been in place for 6 days and may have led to sinus formation creating an open communication between the venous circulation and the atmosphere. The semirecumbent positioning of the patient at the time of catheter withdrawal meant that the right atrium was below the level of the catheter site creating a pressure gradient that resulted in air being entrained into the venous circulation.¹¹ In this case, the attending healthcare professional neglected to follow the Trust safety protocols for central line removal.¹² Removal of the introducer with the patient in a non-supine position and without the application of an airtight dressing to the opening significantly contributed to PAE. Such events can be prevented

by adhering to safety guidelines. Removal of the catheter should be performed with the patient atleast supine, if not in the Trendelenburg position.¹² The catheter should be removed while the spontaneously breathing patient is exhaling. An occlusive dressing should be applied immediately following catheter removal with the patient still in this position and gentle pressure applied until haemostasis is achieved. The dressing should be left in place for a minimum of 24 h.¹²

Competing interests None.

Patient consent Obtained.

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REFERENCES

- 1 American Society of Anesthesiologists. Task force on pulmonary artery catheterization practice guidelines for pulmonary artery catheterization. *Anesthesiology* 1993;78:380–94.
- 2 Swan HJC, Ganz W, Forrester JS, *et al.* Catheterisation of the heart in a man with the use of a flow directed balloon tipped catheter. *N Engl J Med* 1970;283:447.
- 3 Moorthy SS, Tisinai KA, Speiser BS, *et al.* Cerebral air embolism during removal of a pulmonary artery catheter. *Crit Care Med* 1991;19:981–3.
- 4 Augoustides JG, Weiss SJ, Ochroch AE, *et al.* Analysis of the interatrial septum by transesophageal echocardiography in adult cardiac surgical patients: anatomic variants and correlation with patent foramen ovale. *J Cardiothorac Vasc Anesth* 2005;19:146–9.
- 5 Cohn LH. *Cardiac surgery in the adult*. 3rd edn. McGraw-Hill Publishing, 2008:1433–4.
- 6 Sukernik MR, Mets B, Bennett-Guerrero E. Patent foramen ovale and its significance in the perioperative period. *Anesth Analg* 2001;93:1137–46.
- 7 Libby P, Bonow RO, Mann DL, *et al.* *Braunwald's heart disease—a textbook of cardiovascular medicine*. 8th edn. Philadelphia: Saunders Elsevier, 2008:1580–3.
- 8 Riedel M. Venous thromboembolic disease; acute pulmonary embolism 1: pathophysiology, clinical presentation, and diagnosis. *Heart* 2001;1:229–40.
- 9 Battistini B, Verreault M, Ayach B, *et al.* Role of the endothelin system in secondary pulmonary hypertension related to air embolism: lessons learned from testing four classes of endothelin blockers in a rat model. *J Cardiovasc Pharmacol* 2004;44 (Suppl 1):S386–9.
- 10 Mennim P, Coyle CF, Taylor JD. Venous air embolism associated with removal of central venous catheter. *BMJ* 1992;305:171–2.
- 11 Smith T, Pinnock C, Lin T. *Fundam Anaesthesia*. 3rd edn. Cambridge, UK: Cambridge University Press, 2009:54–5.
- 12 Preventing air embolus on central venous catheter (CVC) removal. Metavision Information Management System—ITU department, Brighton & Sussex University Hospitals NHS Trust. Metavision/Care Protocols/Care for the safe removal of CVC (accessed 14 May 2014).

Learning points

- Particular caution is advised in the removal of central venous catheters as inexperienced removal of this routine procedure can lead to serious and potentially life-threatening complications.
- Adherence to the safety protocols with regard to patient position during, and wound closure after, catheter removal is necessary to minimise risk of complications.
- Undiagnosed, probe-patent, foramen ovale may be present in 25–30% of the general population. This case highlights the added risks that unknown and known patent foramen ovale represent in the use of central venous catheters.

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