Pulmonary artery air embolism after permanent pacemaker implantation

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DESCRIPTION
A woman in her 60s with a history of type 2 diabetes mellitus and systemic hypertension was admitted with giddiness and syncope. ECG showed sinus bradycardia with a heart rate of 32 beats per minute with long sinus pauses. Serum electrolytes, thyroid function test and serum creatinine were normal. Transthoracic echocardiogram revealed normal left and right ventricular systolic function, no regional wall motion abnormality and trivial tricuspid regurgitation with systolic pulmonary artery pressure of 12 mm Hg (true estimated value). Mitral, aortic and pulmonary valves were normal. The patient underwent a temporary pacemaker insertion through the inferior vena cava route. Two extrathoracic left subclavian punctures were taken under local anaesthesia with fluoroscopic guidance. When the guidewire and dilator were removed, the operator immediately used his thumb to occlude the lumen of the sheath to prevent bleeding or air entry. A low ventricular lead threshold in the outflow tract and apical positions could not be obtained and ventricular lead was screwed in the mid-ventricular septum. After prolonged manipulations to get a good ventricular lead position and threshold, the patient developed sudden onset dyspnoea, tachypnoea and desaturation. On fluoroscopy large amount of air was seen above the pulmonary valve in the proximal pulmonary artery (video 1 and figure 1A).

The patient was managed with high flow 100% oxygen and was put in Trendelenburg position. Repeat fluoroscopy and cineangiogram later showed the disappearance of the air bubbles from the pulmonary artery (video 2 and figure 1B).
Transient pulmonary air embolism occurs during permanent pacemaker implantation due to deep inspiration during the lead insertion.¹ The intrathoracic pressure fluctuation during respiration is transmitted to large neck veins which leads to pulmonary air embolism.² Sudden cardiovascular collapse can be managed with cardiopulmonary resuscitation, intravenous fluid challenge and high flow oxygen.

Risk factors include snoring, deep sedation and low intravascular volume status. Patient lying in the supine position during operation results in airway collapse due to posterior displacement of tongue and soft palate. Pulmonary air embolism leads to elevated pulmonary artery pressures, ventilation-perfusion mismatch, reduced cardiac output, hypoxia and hypercapnia. The degree of impairment depends on the rate and the volume of air entered, position of the patient and the type of gas. Massive air embolism can result in acute right ventricular outflow tract obstruction and failure. Prevention includes adequate hydration, intravascular volume expansion, proper positioning and positive end-expiratory pressure.

**Contributors**

VG—planning, conception and design, acquisition of data or analysis and interpretation of data. UCV—conduct, acquisition of data or analysis and interpretation of data. PS—reporting, conception and design and interpretation of data. AMK—conception, design, analysis. VA—conduct, reporting, analysis.

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**Competing interests**

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

**REFERENCES**


**Patient’s perspective**

This gave me a good opportunity to understand the disease condition and to discuss in detail with the doctor regarding the available treatment options.

**Learning points**

- Pulmonary air embolism is a possible complication of the insertion of a transvenous pacemaker should be recognised.
- Early detection and prompt treatment helps to avoid catastrophic complication.