Paradoxical gas embolism by transpulmonary passage of venous emboli during hysteroscopic surgery: a case report and discussion

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After an episode of apparent venous gas embolism in a patient undergoing surgical hysteroscopy, transoesophageal echocardiography revealed air in the left but not in the right heart. Contrast echocardiography failed to demonstrate anatomical right-to-left shunts, making it likely that venous emboli overwhelmed the capacity of lungs to filter emboli, resulting in paradoxical embolization.

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Surgical hysteroscopy is an established procedure of minimally invasive surgery. Although risks and complications during hysteroscopy are rare, gas embolism is a potentially life-threatening complication.1 Gas emboli may arterialize, resulting in paradoxical embolism. Although paradoxical embolism has been described under several circumstances including gynaecological laparoscopy,2 there are no reports of paradoxical emboli during hysteroscopy. The arterialization of gaseous products may develop by passage of gas through an anatomical right-to-left shunt such as an open foramen ovale or through pulmonary arterio-venous (AV) malformations. In addition, transpulmonary passage of venous air emboli has been described through the apparently intact pulmonary vasculature.3

We used transoesophageal echocardiography (TOE) in a patient undergoing surgical hysteroscopy during an episode of apparent venous gas embolism. TOE revealed air in the left but not in the right heart. Because further echocardiographic evaluation did not show evidence for a morphological substrate of a right-to-left shunt, it seems reasonable to assume that transpulmonary passage of venous air emboli through the apparently intact pulmonary vasculature resulted in paradoxical gas embolism.

Case report

A 42-yr-old woman (height 1.76 m, weight 86 kg) ASA I, with a history of menorrhagia-related anaemia (haemoglobin 5.5 mmol litre−1) presented for hysteroscopy and transcervical endometrial resection (TCR-E). Her past medical history was uneventful, with no known allergies, and no risk factors for cardiac or pulmonary disease apart from mild obesity.

The patient received total i.v. anaesthesia (propofol, sufentanil, and rocuronium), and standard non-invasive monitoring was used. Mechanical ventilation was with oxygen in air without nitrous oxide (FIO2=0.45), resulting in an arterial oxygen saturation (SaO2), as measured by pulse oximetry, of 98% and an end-tidal CO2 (Eco2) of 4.8 kPa. For TCR-E, a bipolar electro-surgical instrument was used with normal saline (0.9% NaCl) as the uterine distension fluid.

After approximately 20 min of apparently uneventful TCR-E, a 50% decrease in Eco2 was noticed (from 4.8 to 2.4 kPa) followed by a decrease in SaO2 to below 90%. A venous gas embolism was suspected, after excluding other possible explanations for the decreases in Eco2 and SaO2, such as hypovolaemia, ventilatory changes, and artifacts. With a stethoscope, a loud mill wheel murmur was heard precordially confirming the diagnosis of a large venous gas embolism. Although arterial pressure was not affected, an ongoing decrease in arterial oxygenation (lowest value SaO2 40%) and changes on the ECG were noticed. On lead II, a significant 1.3 mm ST-segment elevation was observed, accompanied by frequent ventricular multifocal extrasystoles. Treatment for venous gas embolism was instituted immediately. To prevent any further gas entrainment, surgery was stopped and the patient was put in Trendelenburg position in order to avoid gas from occluding the outflow tract by placing the right
ventricle more superior. Ventilation was continued with 100% oxygen, and a central venous catheter was inserted in the internal jugular vein for gas retrieval. No air or gas was withdrawn. Approximately 15 min after the start of the event, a 7.4 MHz multiplane TOE probe was inserted in order to confirm the diagnosis and eventually directing further treatment. At that moment, the TOE (Vivid-i GE Healthcare, Cardiovascular Ultrasound System) showed no air, gas, or any other particulate matter in the right atrium or right ventricle. However, paradoxical emboli were observed as indicated by echo-dense material that was observed in left atrium (LA) and left ventricle but not in the right side of the heart (Fig. 1).

During the next 20 min, changes in $S_{aO_2}$, $T_{Paco_2}$, and ST-segment slowly recovered and sinus rhythm returned. Because the patient’s condition in the following hour was stable, the decision was made to proceed to the recovery room, in the meantime ventilating with 100% oxygen. A repeat TOE did not show emboli anymore and standard TOE views, including colour-flow Doppler imaging revealed good right and left ventricular contractility without septal defects or valve abnormalities. Contrast TOE, under mechanical ventilation, could not demonstrate an intracardiac shunt. Given the patient’s stable haemodynamic and pulmonary condition on mechanical ventilation, it was decided to stop all sedatives. The patient recovered uneventfully from anaesthesia and her neurological examination was unremarkable.

Several weeks after successful recovery, a thorough transthoracic echocardiography (TTE) investigation was performed to exclude any possible missed intracardiac or intra-thoracic shunt. All heart chambers and valves appeared to be of normal size and with colour-flow Doppler imaging (Nyquist limit 30 cm s$^{-1}$), no patent foramen ovale (PFO) could be detected. Agitated saline contrast was injected peripherally. No intracardiac shunting was observed as indicated by the absence of microbubbles in the LA within three cardiac cycles. A repeat contrast echocardiography, while performing a Valsalva manoeuvre (transiently increasing right atrial pressure provoking the opening of a PFO), again failed to detect an intracardiac shunt. In addition, contrast echocardiography could not delineate intrapulmonary shunting through AV malformations, while agitated saline bubbles did not arrive in the LA with a delay of three to eight cardiac cycles after right atrial opacification. Taken together, no shunting through a PFO or intrapulmonary AV malformation could be detected.

**Discussion**

This case report describes the development of paradoxical emboli in a patient, despite the apparent lack of an anatomical right-to-left shunt undergoing hysteroscopic surgery. To the authors’ knowledge, this is the first report of paradoxical embolism during hysteroscopy, demonstrated by echocardiography. This is important because paradoxical emboli may be a serious clinical threat and may well explain some of the cardiovascular and neurological complications that can take place during the event of large venous gas embolization.

The cardiovascular burden of a paradoxical embolism is important, given its potential lethal impact. Although the coronary arteries are vulnerable for paradoxical air embolization, cardiac failure and cardiac arrest are all possible, depending on the amount of gas embolized. Coronary artery embolization induces electrocardiographic changes typical of ischaemia and infarction, dysrhythmias, and myocardial suppression. Embolization into the right coronary artery is evident by ST changes in the inferior leads. Indeed, in our patient, ECG monitoring showed evidence of right coronary artery embolism as indicated by ST elevations in lead II and frequent ventricular multifocal extrasystoles. It is in our opinion important to realize that coronary air emboli may occur during the event of paradoxical embolization and may at least partly explain some of the events that accompany the clinical picture of large venous emboli.

Neurological sequelae during an episode of severe venous embolism may well be related to the cardiovascular collapse, but paradoxical emboli may also play a role. Cerebral arterial gas embolization may lead to a wide variety of symptoms ranging from mild headache and mild encephalopathy on one side of the spectrum to hemiparesis and frank coma on the other. Indeed, a case of temporary blindness after hysteroscopy was associated with paradoxical embolism. We did not use any cerebral monitoring in our patient during the event, so we cannot comment on the possibility that cerebral emboli did occur. Fortunately, she recovered from anaesthesia without any neurological deficits.
There are several explanations for the development of paradoxical emboli. First, gas may enter the systemic circulation through abnormal venous-to-arterial communications. These may be intracardiac, such as seen in a ventricular septal defect, atrial septal defect, or PFO. A PFO, which is detectable in about 25% of the general population, is the most commonly known cause for emboli to cross from right to left. Under mechanical ventilation, a PFO might go undetected, despite using two-dimensional TOE imaging in multiple views and using colour-flow Doppler. Therefore, we decided for an extensive TTE with agitated saline contrast examination, including Valsalva manoeuvre, several weeks after the event. Our patient did not have a detectable PFO as indicated by this TTE study. Although, short of necropsy and visual inspection, there is no true gold standard for detecting a PFO, TTE contrast studies detect an intracardiac shunt with a sensitivity of 90.5% and specificity of 96.5% and are considered as accurate as TOE with saline solution contrast in determining the presence of a right-to-left shunt. We concluded that passage of emboli through a PFO did not play a role in the development of paradoxical emboli in our patient.

A second explanation is that the patient developed paradoxical emboli through an extra-cardiac pathway such as a pulmonary AV malformation. This, however, is unlikely. The patient was not known to have any symptoms that may have indicated an existing pulmonary AV malformation. She had no history of shortness of breath, haemoptysis, haemothorax, or oxygen desaturations. Also the TTE could not demonstrate a right-to-left shunt. In this respect, saline contrast TTE is considered to be the most sensitive (93%) non-invasive diagnostic test for the screening of pulmonary AV malformations.9 Finally, the prevalence of pulmonary AV malformations (although high in patients known with hereditary haemorrhagic telangiectasia) is low in the general population (1 in 5000). Indeed, our patient’s family history was negative for the presence of hereditary haemorrhagic telangiectasia.

This leads us to the third possible explanation for the development of paradoxical emboli: venous emboli may have overwhelmed the capacity of lungs to filter emboli, resulting in paradoxical embolization. In considering the lungs as a blood filter, two questions determine its capacity to do so. First of all, the diameter of the micro-bubbles is of importance. Deformable emboli larger than 14–22 μm will be filtered in capillaries of the lung having diameters of 3–15 μm.3 The hysteroscopically derived emboli are likely the result of electrosurgical vapours and consist of the highly soluble gases, hydrogen, carbon monoxide, and carbon dioxide.10 The size of these gaseous micro-bubbles is not known, but it is well possible that a large amount of these bubbles did not exceed the aforementioned threshold size of 14–22 μm and developed into paradoxical emboli.

A second question is the maximum volume of gas that the pulmonary vasculature can filter. The pulmonary circulation has a filtration function for small amounts of air and gas. What the filtering threshold of the human lung is for emboli is not known. Studies in dogs showed that the pulmonary filtration threshold may be exceeded when either a large bolus of gas (20 ml kg⁻¹ or more) or a small continuous amount (larger than 0.30 ml kg min⁻¹) is introduced into the venous system. In vitro studies revealed that, during electrosurgical vaporization, gas is produced in volumes up to 60 ml min⁻¹, which is well above the aforementioned pulmonary filtration threshold.14 We assume that a large amount of gas must have been entrained during the hysteroscopy.

In this respect, it is important that transpulmonary transport of gas has been described in humans. Morphological studies in humans demonstrated the existence of direct vascular conduits between pulmonary arteries and veins in human lungs. Indeed, in healthy volunteers, Eldridge and colleagues17 demonstrated exercise-induced passage of contrast bubbles through the pulmonary circulation. They proposed that contrast emboli travelled through direct AV intrapulmonary shunts. Lee and colleagues18 described paradoxical emboli during hepatic surgery in a cirrhotic patient, suggesting intrapulmonary shunting due to cirrhosis-induced pathological dilated pulmonary vessels. Paradoxical emboli were described during several other procedures in patients without a PFO.2 19 20 These case reports do not always mention transpulmonary passage as a pathophysiological mechanism for paradoxical emboli, but it is conceivable that in these cases transpulmonary passage also played a role. Given the absence of a detectable abnormal venous-to-arterial communication in our patient and the amount and composition of the electrosurgical gas by-products, we consider transpulmonary passage of emboli the best likely pathophysiological explanation for the occurrence of paradoxical emboli.

In conclusion, this case report demonstrates that paradoxical emboli may develop during an episode of severe venous emboli during surgical hysteroscopy in a patient without a morphological right to left shunt. Transpulmonary passage is considered a possible pathophysiological mechanism. Cardiovascular and neurological complications resulting from episode of severe venous embolism may at least partly be related to the development of these paradoxical emboli.

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References


