

Case Report

Gas embolism: an exceptional complication of radial arterial catheterization

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Background: We report a rare complication of radial arterial catheterization in a 74-year-old man who had undergone retroperitoneal surgery for an infra-renal aortic aneurysm. A right subclavian venous catheter and a right radial artery catheter were inserted for hemodynamic monitoring.

Results: The patient suddenly went into a coma, with dyspnea and bradycardia, 1 day postsurgery, as a result of a cerebral gas embolism produced by the accidental entry of pressurized air into the artery via the arterial catheter. Cerebral Magnetic Resonance Imaging revealed multiple, diffuse brain lesions. Six months later the patient still had a left hemiparesis and a cognitive deficit. He walks with assistance.

Conclusion: Misuse of an arterial catheter can lead to a severe gas embolism. The infusion system used to flush arterial catheters should be checked regularly to ensure it contains no gas.

Accepted for publication 19 May 2004

Key words: Gas embolism; hyperbaric oxygen treatment; iatrogenic; post operative complication; stroke.

© Acta Anaesthesiologica Scandinavica 48 (2004)

AN iatrogenic gas embolism can result in serious injury or death. Patients undergoing many medical and surgical procedures are exposed to the risk of a gas embolism. Anesthesiologists and intensive care practitioners are often concerned by this complication and must be aware of it (1–3). Arterial catheterization is very often performed in the operating room or in the intensive care unit to monitor arterial blood pressure. Its main complications are vascular insufficiency (spasm, thrombosis or pulselessness), bleeding and infection (abscess insertion site, bacteremia or sepsis) (4). This report describes an exceptional complication in a patient who developed a retrograde systemic gas embolism due to the accidental injection of pressurized air via a radial arterial catheter. Venous gas embolisms are common, but arterial gas embolisms are rare.

Case report

A 74-year-old man was admitted to the Intensive Care Unit due to the sudden onset of a postoperative coma. He had a history of arterial hypertension, hyperlipidemia, diabetes mellitus, obesity and leg arteritis. He underwent retroperitoneal surgery for an infra-renal

aortic aneurysm. A right subclavian venous catheter and a right radial arterial catheter were inserted in the operating room (5). There was no evidence of pneumothorax on the chest X-ray. The tracheal tube was removed 1 h after the operation. On the following day, while the patient was sitting up, he suddenly went into a severe coma (Glasgow Coma Scale: 3/15) with acute dyspnea, bradycardia (50 beats min^{-1}) and hypotension (systolic arterial pressure: 80 mmHg). Simultaneously, the radial catheter infusion bag, which was kept at a constant pressure of 300 mmHg (Infusor system®, Alton Dean Medical, INC. North Salt Lake, UT), was found to be empty and the visible part of the tube was full of air. The trachea was intubated, the lungs were ventilated manually and fluid loading was performed. Arterial pressure and heart rate were normalized (130/85 mmHg and 70 beats min^{-1}). Pulmonary auscultation revealed reduced breath sounds over the right lung field. The resuscitated patient remained in a coma (GCS 3/15), with bilateral Babinski reflexes and unreactive, mid-sized pupils. The ends of one finger on his left hand and one toe on each foot were cyanosed. Thoracic X-ray showed a complete right pneumothorax, which required drainage. The cerebral CT-scan was normal. A gas embolism was suspected and the patient was

transferred to our intensive care and hyperbaric oxygen therapy unit. Hyperbaric oxygen therapy was started 4 h after the event. The patient had not regained consciousness after 5 h of hyperbaric oxygen therapy. Two sessions of hyperbaric oxygen therapy were given. The cyanotic appearance of the finger and toes disappeared after 24 h. A transoesophageal sonocardiogram ruled out a patent foramen ovale. The patient regained consciousness progressively and was quoted 9/15 on the Glasgow Coma Scale on day 20 postarrest.

Cerebral magnetic resonance imaging (MRI) performed on day 6 revealed ischemic lesions in the thalamic region, the right frontal, temporal and occipital cortices and both cerebella (Fig. 1). A cerebral MRI performed 2 months later showed atrophy of the right hemisphere cortex and persistent ischemic lesions in both cerebella.

Six months later the patient still had left hemiparesis and a cognitive deficit. He could walk with assistance.

Discussion

A gas embolism was diagnosed for several reasons: sudden onset of clinical symptoms concomitant with the discovery of pressurised air in the tubes, multifocal nature of the patient's neurological damage when sitting, and simultaneous damage to the brain, heart, lung and peripheral vessels (1-3, 6). The gas embolism could have three possible causes: a central venous catheter, a pneumothorax, or air entering the arterial catheter under pressure, as indicated by the air in the line (1-3, 6).

It seems very unlikely that the central venous catheter could have caused the gas embolism since the perfusion tube was tightly connected and *transoesophageal cardiac sonography* showed no patent oval foramen, a possible explanation for a paradoxical embolism (1, 2, 7).

The central venous catheter had been inserted before surgery and no pneumothorax was seen on the chest X-ray taken after 5 h of positive pressure ventilation for anesthesia. Thus, a pneumothorax due to catheter insertion was very unlikely. The pneumothorax was probably caused by resuscitation maneuvers (3). A pneumothorax must be under pressure to produce a gas embolism (8, 9). This was not the case in our patient. Without a patent foramen ovale, a gas embolism due to a pneumothorax cannot explain the signs of the massive, diffuse arterial gas embolism observed in this patient.

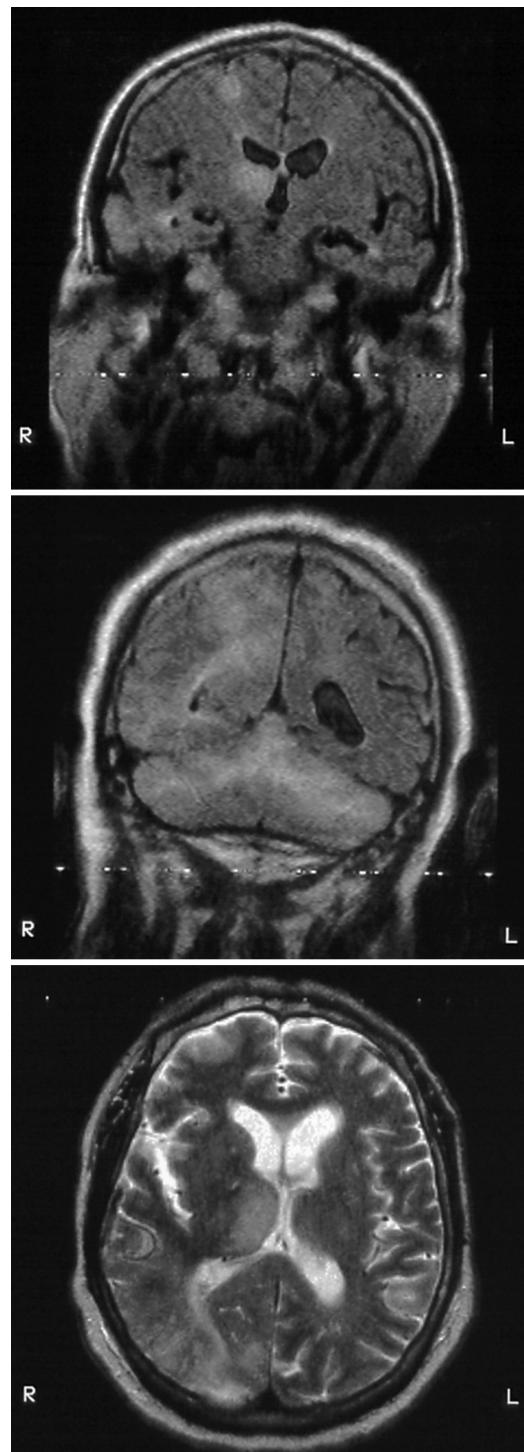


Fig. 1. Cerebral magnetic resonance imaging performed on day 6: FLAIR sequence, coronal slice (A,B). Fast-spin echo-weighted T2, horizontal slice (C). (A) Right frontal-temporal-cortical and thalamic hypersignals. (B) Right occipital and bilateral cerebellar hypersignals. (C) Right occipital-cortical and thalamic hypersignals.

Several indications point to the arterial catheter. Infusion bags from the same batch were found to contain an abnormal amount of air, up to 150 ml per

bag (the bag manufacturer was notified and batches of faulty bags were withdrawn from the market). The clinical symptoms were sudden, multifocal and concomitant with the presence of air in the arterial line (1). The MRI showed multiple, diffuse and not clearly circumscribed ischemic lesions in the vertebral and right carotid artery anatomic territories with no lesions in the left carotid artery. These are topographic arguments that support our assumption. Lastly, these various findings and the absence of cardiac rhythm disorders argue against an ischemic stroke. The topographic multiplicity of the lesions could have been due to a dichotomy in the embolized bubbles (6). Almost all the gas embolus went through the right vertebral artery, and only a small quantity reached the homolateral carotid artery. The distal ischemia of the finger and toes points clearly to a disseminated arterial embolism.

The most frequent complication arising from using arterial catheters is vascular insufficiency due to arterial thrombosis or vascular spasm leading to the occlusion of the distal artery (4, 5). This mainly affects the upper limb arteries due to their small diameter, thus making the air bubbles move against the blood flow (4). Studies on anesthetized monkeys in a sitting position have shown that air can migrate from an arterial catheter towards the brain after mechanical occlusion of the distal artery (10).

The infusion bag for this patient was under a constant 300 mmHg pressure. The infusion system used (Pressure Monitoring Kit, manufactured by BaxterTM, Utrecht, the Netherlands) delivers 3 ± 1 ml saline h^{-1} . If the saline is accidentally replaced by air, the air flow measured upstream by the fixed resistor in the tubing (under a constant pressure of 300 mmHg) is much higher: 1.8 ± 0.1 ml min^{-1} (unpublished data: Dr L. Dubé, 2003). According to Boyle Mariotte's law, a volume of air of an initial 300 mmHg pressure increases 1.23-fold when the pressure drops to 100 mmHg. This is, in part, why the symptoms appeared so rapidly. The volume of air injected into the patient was not measured, but Chang et al. found air in the cerebral circulation after injecting only 2 ml into monkeys weighing 7 kg (10).

Any diagnosis or suspicion of a gas embolism requires the immediate initiation of compression in a hyperbaric oxygen therapy unit, as this will mechanically reduce the size of the gas bubble and facilitate nitrogen removal (1, 3, 6). Time is the key element in

patient management: the shorter the delay, the better the outcome. Platelet-inhibiting drugs and hemodilution have been proposed as adjuvant treatments (1, 3).

The protocols for inserting arterial lines and their maintenance should include regular monitoring for the absence of gas from the infusion bag and line. Perhaps a bubble detector should be used on this type of line with a constant pressure system.

Acknowledgements

We thank Florence Lobrot-André for help with the English language version of the manuscript, and Drs Alain Mercat and Emmanuelle Gras for technical advice.

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