Cerebral Air Embolism Following A Small Volume Of Air Inadvertently Injected Via A Peripheral Arterial Cannula: Case Report

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Citation

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Abstract

CASE REPORT

A fit and healthy 27 year old woman presented for a diagnostic laparoscopy as a day case. She was enrolled into a clinical trial investigating the peri-operative haemodynamic response to various mixtures of thiopentone and propofol. This included placement of a radial arterial cannula prior to induction. Local ethics committee approval was obtained. A full verbal and written explanation of the procedure was given and informed consent obtained.

Once the patient was in the theatre holding area, a 20 gauge cannula was placed under local anaesthetic in her right radial artery. The patient was semi-reclined on a theatre trolley at approximately 30 degrees to the horizontal with ECG, pulse oximetry and NIBP monitoring. All cardiovascular parameters were within normal limits. After successful insertion of the cannula it was connected to the pressure monitoring line and the apparatus flushed with the inline flushing device adjacent to the transducer. As he flushed the line the operator noticed that the monitoring line had been incompletely run through with heparinised saline. The most distal portion of the line from the three-way tap onward therefore contained air.

Within a period of 3 seconds the patient volunteered that she ‘felt funny’, her gaze then turned to the left and her level of consciousness became depressed, although she remained responsive. The patient was immediately laid in the horizontal left lateral position and oxygen administered via a face mask. Her blood pressure increased from 110/65 to 140/95 and her heart rate remained unchanged at 80 bpm. Retrograde cerebral air embolism via the brachial artery was thought to be the most likely explanation considering the time scale of the onset of symptoms.

Apart from the depressed level of consciousness there were no other adverse neurological signs and particularly no evidence of focal neuropathy. Over the following hour the patient’s neurological observations returned to normal although her systemic blood pressure remained elevated. The volume of air injected was estimated from an identical piece of apparatus and was approximately 1.6 ml.

After discussion between the anaesthetist, patient and surgeon it was decided to continue with the anaesthetic and surgery.

Anaesthesia was induced with thiopentone, maintained with isoflurane in oxygen and supplemented with fentanyl. Muscle relaxation with vecuronium facilitated tracheal intubation and positive pressure ventilation to an end tidal carbon dioxide of 4.5 kPa.

The anaesthetic, surgery and subsequent recovery were uneventful. The patient remained in hospital overnight and a full explanation of the untoward events was made later on the day of operation and also the next morning. She also underwent a neurological examination which was unremarkable and was then discharged. However, after 5 days she returned complaining of blurred vision and clumsiness. She was readmitted, referred to a neurologist, and fully investigated including EEG and MRI. All investigations were normal, her symptoms abated within 24 hours and she was discharged.

DISCUSSION

Use of an indwelling arterial catheter for monitoring the cardiovascular system and arterial blood gas analysis is common in both the operating room and the intensive therapy unit. The presence of such a catheter provides a

potential source of air embolism. Air introduced into the arterial tree may cause cerebral air embolism in a retrograde fashion, via a patent foramen ovale (or other septal defect) or, in the case of large volumes, via the pulmonary vasculature.

Cerebral air embolism from a peripheral arterial cannula has been previously reported in humans and in primates but it is unclear what the minimum volume required for significant injury is. (1) Chang et al were unable to state the volume of air inadvertently injected arterially which resulted in the death of an adult human female. In their animal study they demonstrated that 2 ml of air was needed to cause air embolism in a 7 kg macaque. By extrapolation the critical volume for 70 kg human should be well in excess of 2 ml.

Cerebral air embolism may cause a spectrum of adverse effects ranging from the absence of or very mild symptoms and signs to severe neurological injury or death. Signs which have been described include focal motor deficit, sensorial disturbance and visual deficit. Murphy et al described 16 patients with cerebral air embolism treated with hyperbaric oxygen. (2) The most common presenting symptom was a sudden change in sensorium. Smaller numbers exhibited focal motor deficit or visual changes. Respiratory arrest, seizures and severe headache were described in a minority of cases. Cerebral air embolism in an anaesthetised or sedated patient may, therefore, be hard or impossible to detect as the patient is unable to report changes in the sensorium or visual defects and abnormal motor function may not be detectable.

The available techniques for the detection of air embolism are predominantly aimed at the venous circulation as, in the anaesthesia setting, this route of entry is more common than the arterial. In ascending order of sensitivity, these include measurement of blood pressure, cardiac output monitoring, end-tidal carbon dioxide, precordial doppler ultrasound and transoesophageal echocardiography (TOE). (3) Of these methods, doppler and TOE are quantifiable in their ability to detect air embolism. Doppler may detect volumes of air down to 0.5ml and TOE may be 5 to 10 times as sensitive.

The management of cerebral air embolism is largely supportive. Attenuation of bubble size is the prime objective and 100% oxygen should, therefore, be administered in all suspected cases as this favours nitrogen ‘washout’ from the bubble into the alveoli. The administration of nitrous oxide should be discontinued immediately as it diffuses rapidly into any air space including bubbles and significantly increases their size. Hyperbaric oxygen has been shown to be of benefit in up to 80% of cases of cerebral air embolism but controlled trials have yet to be performed. (2) However, the effectiveness of therapy is dependent on the volume and precise position of the embolism as well as any pre-existing morbidity.

In conclusion, cerebral air embolism is an uncommon yet potentially fatal complication associated with arterial catheterisation. The volume of air necessary to cause patient detriment is unknown but may be lower than previously described. In the case reported here, the volume of the monitoring line from the three-way tap to the distal luer lock was measured as 1.6 ml and this was the volume inadvertently injected into the radial artery. It therefore appears that quite small volumes of air may cause significant cerebral injury if inadvertently injected into the peripheral arterial tree at high pressure. All physicians, nurses and paramedics should be aware of the potential for disaster and exercise due care in the management of arterial cannulae.

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