

Rare Neurological Complication Due to Air Embolism Facilitated by Prone Position During PCNL Surgery and its Successful Management

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ABSTRACT

Venous air embolism during percutaneous nephrolithotomy (PCNL) following air pyelogram or saline irrigation has been occasionally reported. We present a case of suspected venous air embolism during air pyelogram in a patient undergoing PCNL. The clinical diagnosis of air embolism was made by fall in end tidal carbon dioxide and blood pressure and was conservatively managed. The patient developed a neurological complication, monoparesis, secondary to the embolisation. Early diagnosis with rapid resuscitation is the key to management of a patient with air embolism.

Keywords: Air Embolism, PCNL.

INTRODUCTION

Venous air embolism during percutaneous nephrolithotomy (PCNL) following air pyelogram or saline irrigation has been reported previously.^[1] We describe a case of suspected air embolism during saline irrigation pyelogram in a patient undergoing PCNL leading to a neurological complication.

CASE REPORT

A 30 year old female, weighing 52 kilograms, with no co-morbidities or positive findings in examination, with all routine investigations within normal limits, who was posted for percutaneous lithotripsy for a 8 mm × 14 mm middle calyx calculus was accepted as ASA PS I with General Anaesthesia and controlled mechanical ventilation in prone position as the plan.

On the day of surgery, after ensuring adequate fasting, she was wheeled into the Operation theatre. Emergency resuscitation equipment, anesthesia machine and equipment were checked. Baseline monitors were noted: Blood Pressure(BP)- 118/62 mm hg, Heart Rate (HR)- 78 beats per minute(bpm), room air saturation (sPo2)- 100%, electrocardiogram(ECG)- Normal sinus rhythm. Intravenous (iv) access was secured. One mg Midazolam and 100 mcgs Fentanyl was given iv. Preoxygenation was done for 3 minutes with 100% oxygen (O₂) at 6 litres/min. Induction of anesthesia was started with graded dose of propofol (total 80mg

was given), after checking for ventilation, muscle relaxant was given (5 mg iv vecuronium). Intermittent positive pressure ventilation was done for three minutes with 100% O₂. Patient's trachea was intubated with 7mm ID endotracheal tube under direct laryngoscopic vision, checked with end tidal carbon dioxide (EtCO₂) trace, bilateral air entry, chest rise, and connected to ventilator on volume control ventilation. Patient was monitored with ECG, SpO₂, ETCO₂, heart rate, BP measured at 5 min intervals as per the recommendation.² For maintenance of anesthesia, boluses of vecuronium (1mg) as per requirement, sevoflurane (1-2%), O₂ + N₂O mixture(50%) and fentanyl boluses (10 mcg) were given.

The patient was placed in a lithotomy position for ureteric catheter placement in the left kidney through a cystoscope under fluoroscopic guidance. The patient was then carefully turned prone taking all precautions and PCNL started. Following identification of the calyx, 18 G needle was directed to the targeted calyx. The tract was dilated and nephroscopy was commenced. Pyelogram was done by injecting saline under high pressure for proper identification of the pelvi-calyceal system. During pelvicalyceal dilatation, there was a sudden drop of EtCO₂ from 35 to 14mm Hg in a span of 3-4 minutes. B/L air entry was checked, airway pressures and exhaled tidal volume were normal, and the patient was hemodynamically stable. Following this, the heart rate dropped from 78 to 42 bpm, and blood pressure fell from 98/64 to 78/46 mm Hg with a saturation of 100% throughout. In view of hypotension and bradycardia, 100% oxygen given, nitrous oxide discontinued and patient treated with intravenous fluids, anticholinergics and vasopressors restoring the hemodynamic stability and EtCO₂ level to 30mm Hg. Surgery was abandoned. Patient

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was then positioned back to the supine position, extubated after extubation criteria fulfilled, and shifted to the PACU.

Two hours after extubation, patient was drowsy with a GCS of 10/15 and there was associated right hemiparesis and extensor plantar reflex on the right foot although she was hemodynamically stable with a BP of 100/64 mm HG, HR 89 bpm, and room air saturation of 100%. ABG,CBG and 2D Echo were done which were all found to be normal. MRI was immediately done which revealed multiple restricted diffusion defects in both cortex, left more than right, suggestive of recent infarcts as shown in [Figure 1].

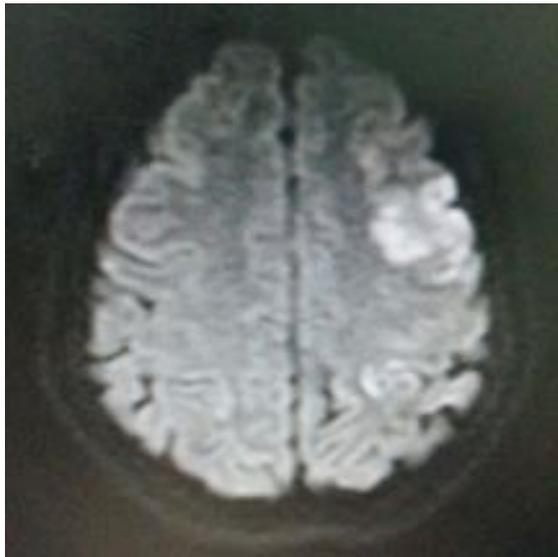


Figure 1: MRI of the patient within three hours

Immediately treatment was started with anti platelets, mannitol, anti epileptic drugs and heparin. She had residual weakness in the right upper limb and was sent back to the ward. After two weeks the patient was reviewed on an outpatient basis. She had a power of 4/5 in the right upper limb with no other focal neurological deficit.

DISCUSSION

Venous air embolism (VAE) is a potentially life threatening condition that has been reported in several surgeries like neurosurgeries, spine surgeries, laparoscopic, arthroscopic procedures etc.^[3] Although it has been reported in PCNLs as well, it is rare and has caused long term complication in only one other reported case where patient developed quadriparesis,^[4] whereas our patient developed monoparesis.

Air pyelogram has the potential risk of an air embolism. Although in our setup we use saline as the irrigating fluid, the high pressure of the irrigating fluid containing potential air bubbles was the most probable source of the air embolism. The site of eventual lodgement of air emboli in the brain depends on the position of the patient at the time of

incidence. The prone position of the patient facilitated the VAE by producing a significant gravitational gradient between the right side of the heart and the renal pelvis, possibly resulting in air being drawn into open veins by the negative pressure.^[5] Furthermore, decreased caval pressure due to the position of the lower limbs could have facilitated the embolism. Several sophisticated modalities of confirmation of diagnosis of VAE are available like Doppler ultrasound, Right Atrial Catheter, Pulmonary artery catheter, Transesophageal Echocardiography etc.^[6] The application of end tidal carbon dioxide monitoring during general anaesthesia is a routine practice and provides a sensitive and specific method to detect VAE.^[7] Since PCNL is a low risk surgery for the development of VAE, we used routine monitoring and not the other monitors for VAE. As soon as the ETCO₂ and hemodynamic instability ensued, air embolism as a probable cause was considered and nitrous oxide immediately turned off. We ruled out other possibilities like cardiac arrhythmia, pneumothorax, displacement of the endotracheal tube, ventilatory or mechanical failure that can simulate air embolism.^[8] Treatment that supports right heart function may allow sufficient time for redistribution of embolized air and produce a good outcome when access for central aspiration of air is not available, as was in our case hence we used vasopressors till hemodynamics were stabilised.^[9] The delayed manifestation of symptoms in this patient a few hours after extubation could be explained by the fact that air emboli may have taken time to pass through the pulmonary vasculature, delaying the onset of systemic embolic manifestations.

CONCLUSION

Air embolism should be kept in mind following air pyelogram during PCNL surgery and other surgeries where fluid or air is introduced under high pressure. In our case, we ensured that saline was used for irrigation, made immediate diagnosis and abandoned surgery, and got a timely MRI done, even then the patient had a neurological complication. This may be avoided or minimized by using smaller amounts of air, saline or other alternate gas like CO₂. Vigilant monitoring with available monitors, particularly end tidal CO₂ and prompt treatment could result in favorable outcome following air embolism.

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