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Air Embolism Following Percutaneous Nephrolithotripsy: An Unusual Complication

Mufti Mahmood Ahmed, Zahid Mohd Rather, Bashir Ahmed Sanai, Nazir Ahmad Salroo, Riyaz Munawar Wani, Imran Nazir Salroo

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ABSTRACT

We present a case report of a seizure and neurological deficit following percutaneous nephrolithotripsy (PCNL) due to a venous air embolism in a patient who was previously healthy and not suffering from any intracardiac defects (foramen ovale) or prepulmonary AV shunts. An air pyelogram has the potential risk of an air embolism, and the time of seizure onset and neurological deficit following the procedure may be the clue to the path followed by air emboli. In our case, the time of seizure onset and neurological deficit following the procedure was 10 hours, and the volume of air injected exceeded the recommended dose (10 to 15 ml). The clinical diagnosis of venous air embolism was confirmed by magnetic resonance imaging (MRI). To our knowledge, this is the first reported case of such a complication.

INTRODUCTION

Standard urological textbooks do not mention the risk of air embolisms following the injection of air during percutaneous nephrolithotripsy (PCNL). A venous air embolism is a rare but potentially fatal complication of percutaneous renal surgery. Air enters the venous system and passes through the right heart into the pulmonary circulation, blocking the output of the right heart, which results in hypoxemia, hypercapnia, and depressed cardiac output and hypotension. Air can also pass through a patent foramen ovale into the arterial system, which can result in neurological deficits [6]. Pyelovenous backflow causes displacement of air from the renal pelvicalyceal system into renal veins. This phenomenon was first described by Lopez, who noted the passage of fluid from the calyces to the renal veins [7]. The postulated route of entry for gas emboli into arterial circulation causing paradoxical embolism is suggested to be via the prepulmonary AV shunts or directly through the pulmonary capillary bed [10]. The onset of seizures in such patients are likely to be delayed by some hours in light of the time taken for the passage of air emboli through the pulmonary vasculature. The adverse effects of such micro bubbles of air on the pulmonary vasculature may not be discernable, owing to the availability of adequate pulmonary reserve compensating for the same.

CASE REPORT

A 52 year-old-male presented to our hospital with complaints of recurrent episodes of right flank pain and urinary tract infection. Patient had undergone right pyelolithotomy 8 years back.

Detailed investigations included urinalysis, urine culture, coagulation profile, abdominal ultrasound, and intravenous urography (IVU). An abdominal ultrasound revealed right nephrolithiasis measuring 25 mm with grade II hydronephrosis. IVU revealed a right renal pelvic calculus with grade II hydronephrosis and normal excreation, as shown in Figure 1 and Figure 2.

The nature of the disease and the possible therapeutic options were discussed with the patient. Right PCNL was planned. General anesthesia was administered and the patient was placed

KEYWORDS: Renal stones, PCNL, air embolism, seizures

CORRESPONDENCE: Mufti Mahmood Ahmed (MCh), Assistant Professor and Chief Urologist, Division of Urology, Department of Surgery, Government Medical College, Srinagar, Jammu and Kashmir, India (muftimahmood61@rocketmail.com)

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Figure 1. Kidney, ureter, and bladder film showing right nephrolithiasis.

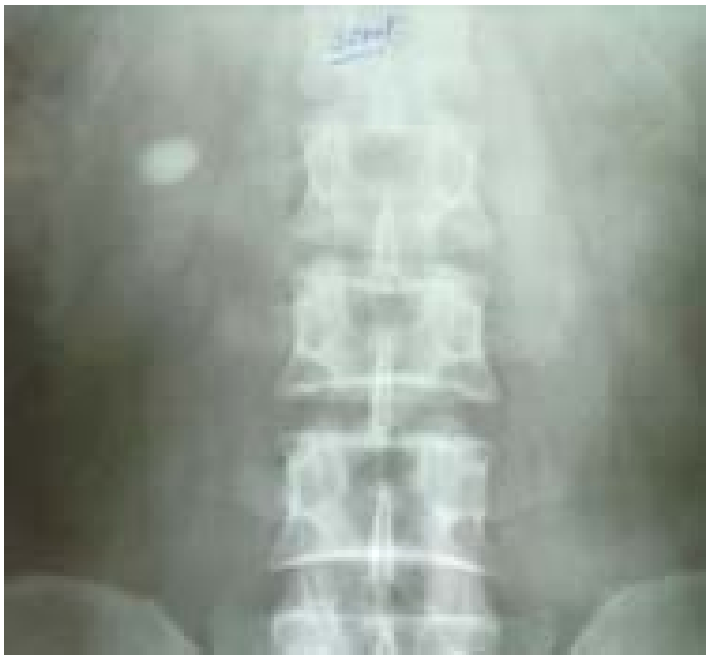
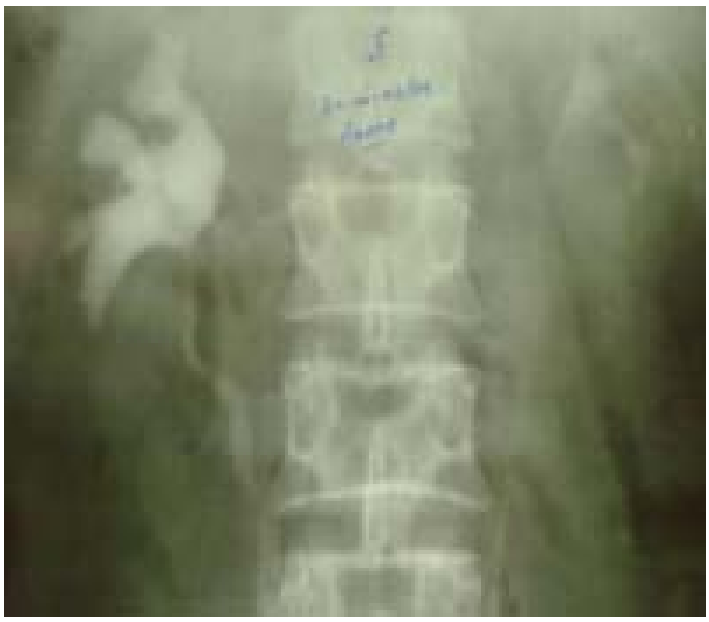


Figure 2. IVU film showing normal excretion of contrast by both kidneys with grade II hydronephrosis and a filling defect in the right side.



in the lithotomy position. A 6 Fr ureteric catheter was placed in the right ureter under cystoscopic guidance. The patient was turned into the prone position, and an air pyelogram was obtained by injecting about 20 mL of air into the right pelvicalyceal system. An inferior calyceal puncture was done, and the tract was secured with a 0.035" floppy tip, hydrophilic guide wire. The tract was dilated to 26 Fr and an Amplatz sheath was placed. The stone was visualized and fragmented with a pneumatic lithotripter into multiple small fragments. The fragments were retrieved, and complete clearance was ensured upon fluoroscopy followed by the placement of a 6 Fr double-J stent. A nephrostomy tube drain was kept in the pelvicalyceal system, which was clamped and secured in place. The patient was turned back into the supine position and extubated. The recovery was uneventful.

In the immediate postoperative period, the patient was conscious and well oriented in time, place, and person. The general condition was fair; his pulse was 90 beats/min; his blood pressure was 130/88 mmHg; his urine output was 100 mL, which was high-colored; and his postoperative hemoglobin was 12.5 gm%. Ten hours following the procedure, the patient developed generalized tonic-clonic seizures and a change in sensorium, followed by weakness on the left side of his body and slurred speech. An injection of diazepam was administered to the patient (10 mg) intravenously followed by an injection of phenytoin (100 mg) intravenously. He was shifted to the surgical intensive care unit (SICU) where he was actively convulsing with unremarkable vital parameters, such as a pulse of 120 beats/min and blood pressure of 130/92 mmHg. The patient was tachypneic, with shallow breathing, and a respiratory rate of 28/min. His oxygen saturation (SPo2) was 64%. The patient was anaesthetized in the surgical intensive care unit with 5 mg of midazolam and 100 mg of scoline, intubated with 8 mm of cuffed endotracheal tube, and coupled to a ventilator. Neurological examination was not possible, because the patient was paralyzed. An investigation done in SICU was unremarkable. Computed tomography (CT) of the brain revealed subtle hypodensity in the area of the right posterior cerebral artery. Patient was given IV fluids, an injection of meropenem (1gm IV, 8 hourly), an injection of phenytoin (100 mg IV, 8 hourly), an injection of mannitol (100 ml IV, 12 hourly), and an injection of omeprazole (40 mg IV, once a day).

On the second day of admission, the patient was weaned off the ventilator. Neurological examination revealed mild drowsiness, slurred speech, poor cough and gag reflex, and both pupils were symmetrically reacting to light. The patient developed left upper motor-neuron, seventh-cranial nerve palsy and hypotonia in the left upper and lower limbs, with a strength of 0/5 (MRC), absent reflexes, and left planter extensor. The rest of the cranial nerve examination was normal. The sensory examination was normal. The patient subsequently

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Figure 3. MRI of the brain; T1W (axial section) showing infarct in the right PCA territory.

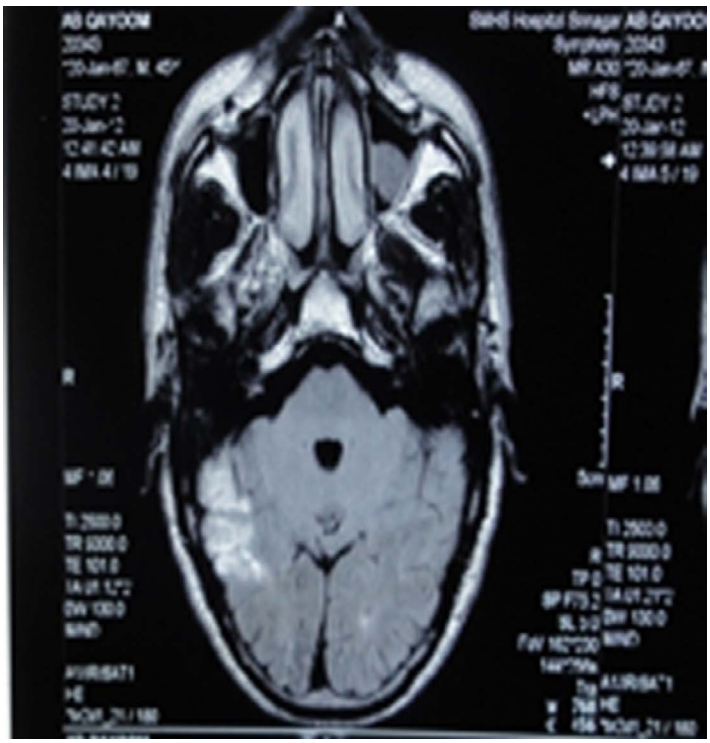
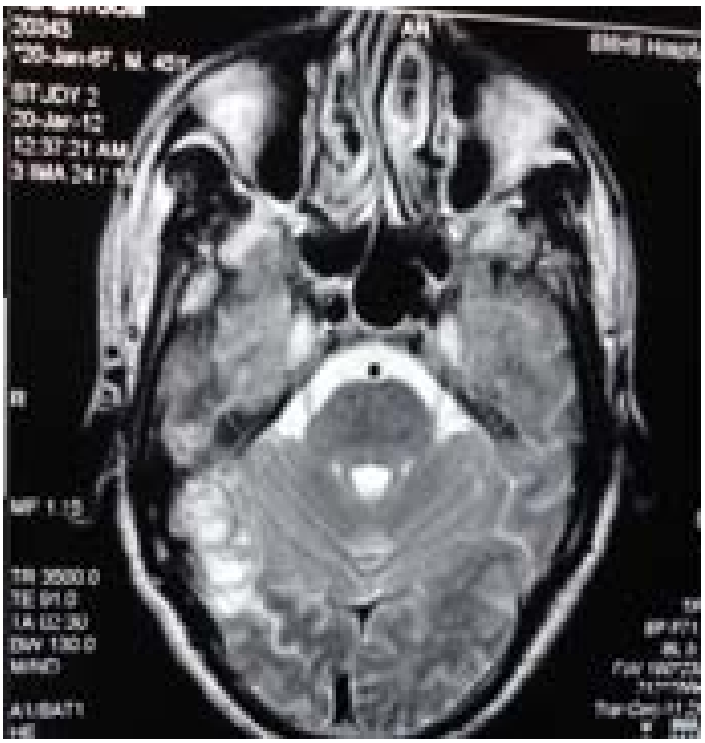


Figure 4. MRI of the brain; T2W (axial section) showing infarct in the right PCA territory.



underwent MRI of the brain that showed acute infarct in both T1,T2 weighted and Flair images in the territory of the right posterior cerebral artery. In addition, bilateral, non-specific, small multifocal hyperintensity in the subcortical areas of the brain were seen, as depicted in Figure 3, Figure 4, and Figure 5. On the fourth day, the patient recovered with residual left hemiparesis, which resolved within six months.

DISCUSSION

An air pyelogram has the potential risk of an air embolism [5,8]. Saline injected after air will compound the problem as it increases the pressure in the renal pelvis, predisposing it to pyelovenous back flow. The prone position of the patient in this case produced 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. This case demonstrates the problems associated with a combination of a gravitational gradient, decreased caval pressure due to the position of the lower limbs, and possible high pressure of the irrigating fluid containing potential air bubbles [1]. In our patient, the symptoms started 10 hours after the procedure, suggesting that the air emboli may have taken time to pass through the pulmonary vasculature. An increase in pulmonary artery pressure could have been induced during retching episodes. While the patient was in the head-up position, air crossing to the systemic arterial circulation could have migrated into the carotid and cerebellar arteries, and then to cerebral and cerebellar hemispheres [10]. Cerebral arterial air embolization typically involves small arteries (average diameter of 30 to 60 μm).The emboli causes pathological changes by 2 mechanisms: a reduction in perfusion distal to obstruction and inflammatory response to the air bubble. This results in cerebral blood flow decline and neural function [2] leading to variable clinical manifestations. A CT scan of the brain is helpful in detecting arterial air in the acute stage, and finer cuts are found to increase the detection rate [3,11]. A brain CT is diagnostic only if obtained immediately because air is rapidly reabsorbed from the brain arterioles [2]. In our patient, a brain CT was performed 24 hours following convulsions. MRI helps

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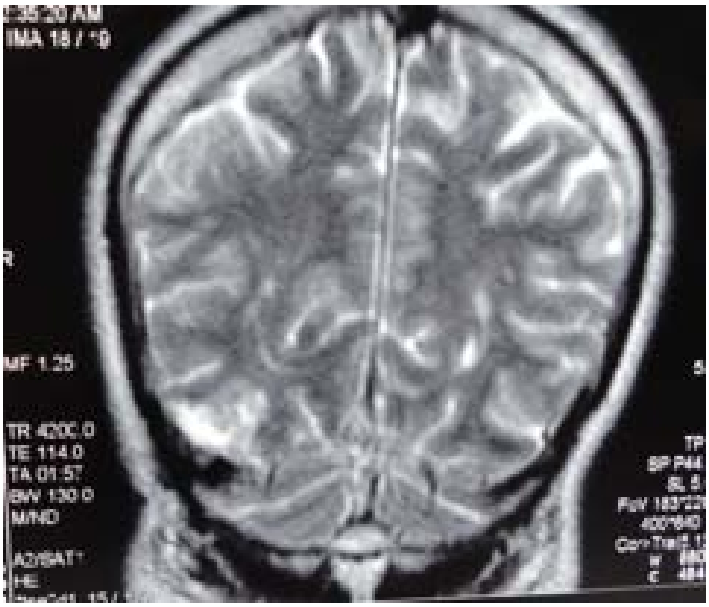
in detecting areas of infarction in the acute stage, identifying areas of reversible ischemia and hemorrhagic transformation of infarcts, discriminating between acute and chronic infarcts, and identifying tissue edema less than 1 hour after an ischemic event [11]. In our patient, a brain MRI showed acute infarct in both T1,T2 weighted and Flair images in the right posterior cerebral artery area. There was also non-specific, small multifocal hyperintensity seen in bilateral subcortical areas of the brain. The combination of CT and T1,T2 weighted and Flair MRI images appear ideal for evaluating suspected cerebral air embolisms in the acute setting [2]. At the present time, there are no consensual guidelines on the optimal management of cerebrospinal air embolisms. Options include hyperbaric oxygen therapy, Lidocaine, heparin, and infusion therapy.

It has been suggested that neurological injury from cerebral air embolisms may not be the only result of temporary vessel occlusion, but more likely the result of secondary thrombo-inflammatory responses at the site of the air-injured endothelium. Both ultra-structural and functional studies indicate that there is a complex interaction among bubbles, blood elements (platelets, fibrinogen, and leukocytes), and endothelium, which result in local fibrin deposition and adherence of platelets and leukocytes to both bubbles and air-injured endothelium. Hence heparin is supposed to be helpful [9].

Urologists who prefer to use air during PCNL under general anesthesia should be aware of the potential risks of air embolisms. Injection of a small amount of air (10 to 15 mL) for pneumopyelography is strongly advisable, if necessary for delineation of the posterior calyx.

Figure 5. MRI of the brain; T2 coronal section showing infarct in the right PCA territory.



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