

Cerebral and Spinal Air Embolism following Percutaneous Nephrolithotomy

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Abstract

We present a case report of cerebral and spinal air embolism following percutaneous nephrolithotomy in a patient without evidence of intra-cardiac defects or prepulmonary A-V shunts. The position of the patient during the incidence determined the site of eventual lodgement of air emboli in the arterial circulation. We suspect that the time of onset of symptoms following the procedure may be the clue to the path followed by air emboli.

Introduction

Cerebral air embolism as a complication of PCNL has been observed in patients with intra-cardiac defects¹ as well as in patients without such defects.² Pyelovenous backflow causes displacement of air from the renal pelvicalyceal system into the renal veins. This phenomenon was first described by Lopez, who noted the passage of fluid from the calyces into the renal veins³. The postulated route of entry for gas emboli through to arterial circulation causing paradoxical embolism is suggested to be via the prepulmonary AV shunts or directly through the pulmonary capillary bed.² However the onset of symptoms in such cases is likely to be delayed by some hours in view of time taken for passage of emboli through the pulmonary vasculature. We present a case report of Cerebral and spinal air embolism following percutaneous nephrolithotomy in a patient with no evidence of intra-cardiac defects or A-V shunts.

Case Report

Forty eight years old male was admitted to a private hospital

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for percutaneous Nephrolithotomy (PCNL) indicated for a right side staghorn calculus. PCNL was done under general anesthesia with the patient in prone position and the surgical approach was supracostal. PCNL was supplemented with D-J stenting. Post procedure during recovery from anesthesia, he complained of neck pain followed by weakness in both lower limbs. Over next half an hour he developed weakness in both upper limbs. He complained of breathing difficulty, hence was shifted to our hospital. There was no history of sensory, bowel – bladder complaints (patient was already catheterized at the time of PCNL). There was no history of seizures, loss of consciousness or hypotension during PCNL.

On examination, patient was conscious, obeyed commands. Pulse was 88/min regular, equal on both sides, all peripheral pulses were palpable. Blood pressure was 110/70mmHg right arm supine position. There was no pallor, cyanosis, icterus, edema, clubbing. Patient was tachypneic with shallow breathing and respiratory rate of 30/min. Accessory muscles of respiration were used by the patient. Neurological examination revealed gaze preference to right side, hypophonic speech, poor cough and gag reflexes, pupils bilaterally symmetrical 1 mm reacting equally to light and there was no nystagmus. Motor system examination showed hypotonia in all four limbs with power 0/5, absent reflexes and equivocal planter responses. Rest of

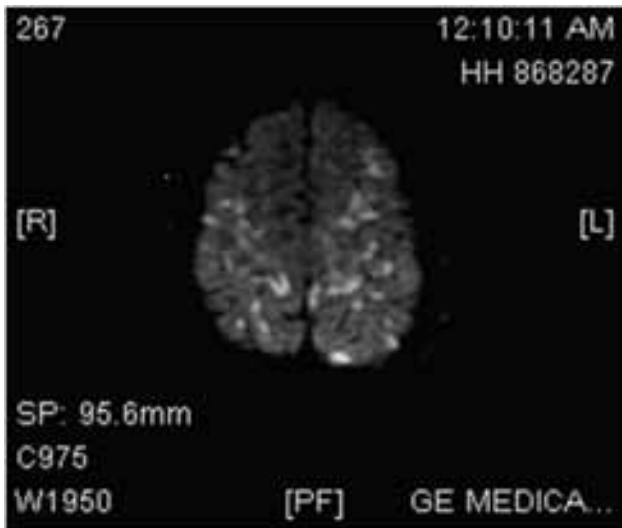


Fig. 1 : Diffusion weighted axial image through high frontoparietal region reveals multiple small foci of restricted diffusion, showing a gyriform pattern. These are suggestive of recent infarction.



Fig. 2 : Sagittal sequence through the whole spine reveals diffuse hyperintensities through out the cord more marked in the cervical region.

cranial nerve examination was normal. Sensory examination was normal.

On day of admission, patient underwent MRI brain which revealed multiple hyperintensities on diffusion weighted images (DWI) in bilateral cerebral cortices in a gyriform pattern which were hypointense on ADC (Apparent Diffusion Coefficient) suggestive of recent infarction. Intramedullary T2 hyperintensities were noted in spinal cord at multiple levels more marked in the cervical region. Hyperintensity was also noted in Left cerebellar hemisphere. MRI Angiography revealed no evidence of vascular abnormality (Figs. 1-4).

Patient was intubated and put on a ventilator (IPPV mode).

2D Echocardiogram, Doppler using agitated saline did not show any evidence of right to left shunts. Complete blood counts and metabolic parameters were normal. EEG revealed slowing of background activity. Nerve conduction studies and somatosensory evoked potentials were normal.

Patient was administered heparin 0.6mg subcutaneously twice daily and recovered with residual spastic paraplegia and is currently wheel chair bound with an indwelling catheter.

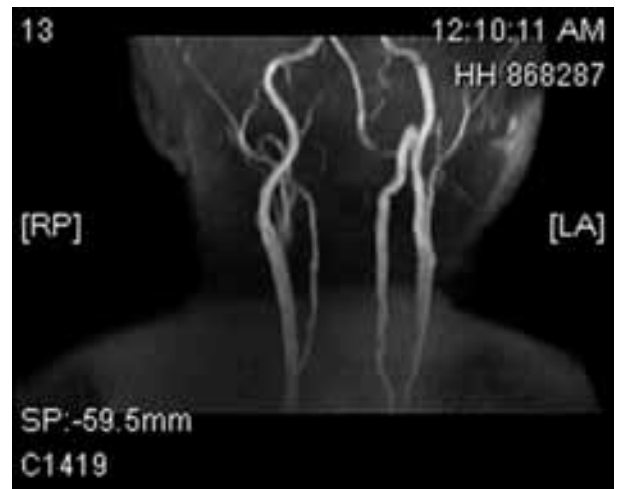


Fig. 3 : 2D TOF neck projection image showing hypoplastic right vertebral artery with other major arteries appearing normal.

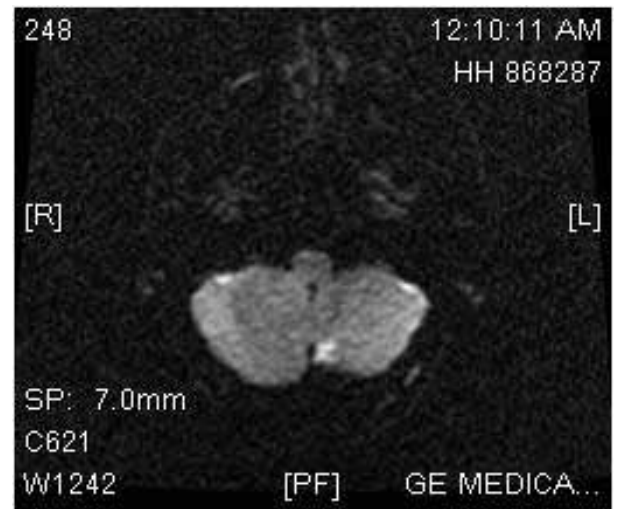


Fig. 4 : Diffusion weighted axial image through posterior fossa showing hyperintensity in left Cerebellar hemisphere

Discussion

In our patient the symptoms started during recovery from anesthesia. This suggests that the air emboli may have directly entered the pulmonary venous system due to the supracostal approach adopted during the procedure. Site of eventual lodgement of air emboli in brain depends on position of patient at the time of incidence. The prone position of the patient during the procedure provided a gravitational gradient between the posterior vertebro-basilar circulation and the left side of the heart facilitating the entry of air emboli into the posterior vertebro-basilar arterial circulation including the spinal artery.

Cerebral arterial air embolization typically involves small arteries (average diameter, 30 to 60 μm).⁴ The emboli cause pathologic changes by two mechanisms: a reduction in perfusion distal to the obstruction and an inflammatory response to the air bubble. This results in a transient decline of cerebral blood flow and neural function⁴. Clinical manifestations range from minor motor weakness to hemiparesis, headache, confusion, convulsions, loss of consciousness. Asymmetry of the pupils, hemianopia, impairment of the respiratory and circulatory centers (manifested as bradypnea, Cheyne–Stokes breathing, cardiac arrhythmias, and circulatory failure) have also been described⁴. In patients who have undergone surgical procedures that carry a risk of gas embolism, a delayed recovery from general

anesthesia or a transitional stage of impaired consciousness may be a clue to diagnosis of cerebral air embolism.⁴

Brain CT is diagnostic only if obtained immediately because air is rapidly resorbed from the brain arterioles.⁴ In our patient brain CT could not be performed due to urgency of the situation. MRI picture was characteristic showing multiple areas of restricted diffusion along the cortical gray matter in a gyriform pattern involving both cerebral hemispheres. These were suggestive of recent infarction. Diffusion Weighted Image (DWI) findings depend on the size and number of the air emboli. The gyriform pattern seen may be the result of multiple air emboli lodged into end-artery territories of the brain, causing ischemia along the cortex. The combination of CT and DWI MRI appears ideal for evaluating suspected cerebral air embolism in an acute setting.⁴

There are no specific guidelines for treatment of cerebrospinal air embolism. Options include- hyperbaric oxygen therapy, lidocaine, heparin, Infusion therapy. Hyperbaric oxygen is suggested to induce mechanical compression of air bubbles to a much smaller size and also results in delivery of high doses of oxygen to ischemic brain tissue.⁵⁻⁶

It has been suggested that neurological injury from cerebral air embolism may not only be the result of temporary vessel occlusion but rather is more likely the result of secondary thrombo-inflammatory responses at sites of air-injured endothelium. Both ultrastructural and functional studies indicate that there is a complex interaction among bubbles, blood elements (platelets, fibrinogen, and leukocytes), and endothelium, which results in local fibrin deposition and adherence of platelets and leukocytes to both bubbles and air-injured endothelium. Hence, heparin is supposed to be helpful.⁷

There is some evidence that gas embolism may cause

hemoconcentration, due to intravascular leak following endothelial damage. Infusion therapy is used to achieve normovolemia and prevent hemoconcentration.

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