# Case Report: Spontaneous Arterial Gas Embolism with Pulmonary Arteriovenous Malformation and Obstructive Sleep Apnea

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## INTRODUCTION:

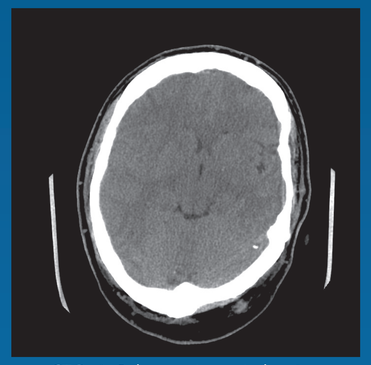
Pulmonary barotrauma can cause cerebral air gas embolism (CAGE) from pulmonary over-distension of alveoli forcing gas into pulmonary capillaries, which may arterialize. Causes include positive-pressure ventilation, central lines, surgery, trauma or a complication of SCUBA diving or hyperbaric/hypobaric exposures.

Spontaneous arterial gas embolism is rare: a single case has been reported in a diver.[1] We report a case of spontaneous CAGE in a man with occult pulmonary arteriovenous malformation (AVM) and undiagnosed obstructive sleep apnea (OSA).

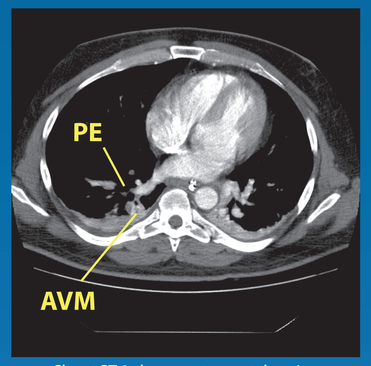
## CASE PRESENTATION:

A 46 year-old man was admitted to the hospital for an acute seizure occurring shortly after going to bed, followed by left-sided weakness. He had multiple telangiectases on his lower lip and tongue. Brain CT showed intravascular gas in the superior sagittal sinus, right anterior parietal lobe, right temporal lobe, and right aspect of the cavernous sinus. Brain MRI found right hemispheric cerebral edema and right frontal gray matter hypodensities.

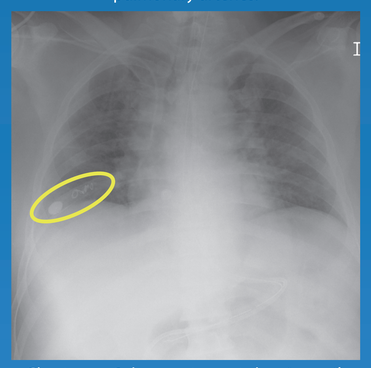
Physical exam and radiographic findings increased suspician for pulmonary AVM, an abnormal communication between a pulmonary artery and vein causing a right-to-left shunt. AVM is associated with hereditary hemorrhagic telangectasia (Osler-Weber-Rendu), an inherited disorder of blood vessels



CT Brain 2 days post-event, showing cerebral edema of the right hemisphere and early hypodensities involving the right frontal lobe gray matter.




Chest CT 3 days post-event showing prominent artery extending towards a 1.8 cm hypodense lesion in the lateral right costophernic sulcus consistent with AVM, and a 4 mm nodular opacity which could represent another AVM. Moderate clot is present within pulmonary arteries.



Chest x-ray 6 days post-event showing coil, normal cardiac size, decreased lung volumes, and mild patchy perihilar opacities bilaterally.

## CASE PRESENTATION (CONTINUED):

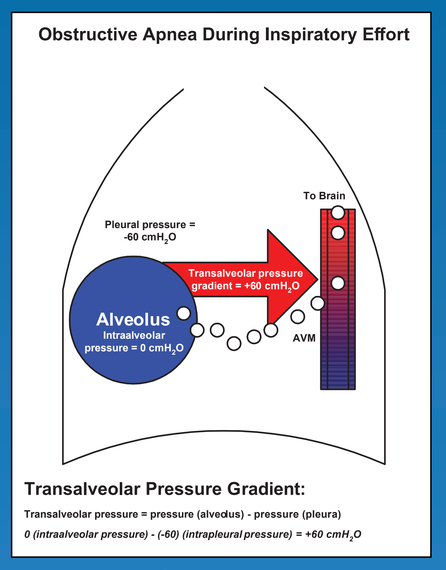
Chest CT revealed a 1.8-cm pulmonary AVM in the posterior right costophrenic sulcus. By transthoracic echocardiogram he had no intracardiac shunt or patent foramen ovale. He was treated with Dilantin, Lidocaine (48 hours) and hyperbaric oxygen for CAGE. He received 3 hyperbaric oxygen sessions following the US Navy Treatment Table 6 protocol and 1 session at 2.4 atm abs for 100 minutes. Pulmonary angiography demonstrated an AVM, which was occluded with a detachable balloon followed by coil embolization.

The patient's wife reported snoring, and polysomnogram revealed severe obstructive sleep apnea, which was treated with CPAP. Upon discharge he had a normal neurological exam, but cognitive impairments. Seven years later his primary care physician reports he is back to his pre-event baseline including employment.

## HYPOTHETICAL MECHANISM FOR AGE:

The intraalveolar pressure equals 0 at the end of expiration, and with normal resting inspiration, the intrapleural pressure is -10 $cmH_2O$. Therefore the transalveolar gradient is +10 $cmH_2O$.

With obstructive sleep apnea, the intrapleural pressure may exceed -60 $cmH_2O$ during inspiration. Therefore, with OSA, the transalveolar pressure gradient would be +60 $cmH_2O$. In the presence of pulmonary AVM, this pressure is sufficient to cause the passage of air from alveoli to the AVM circulation, with arterialization.




## CONCLUSION:

We propose this CAGE was caused by high negative intrathoracic pressures while breathing against an obstructed upper airway, with air entrainment into the AVM, and subsequent arterialization. Alternatively, cough may have contributed to CAGE. This case represents a rare report of spontaneous CAGE.

## REFERENCES:

1. Hsu YL, et al. Br J Sports Med. 2004 Aug;38(4):E6