# An unusual cause of ischemic stroke - Cerebral air embolism

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**Abstract**

Air embolism is a preventable, often undiagnosed but potentially treatable cause of ischemic stroke with a high morbidity and mortality. It is usually iatrogenic ocurring especially in patients in ICU setting. We describe the case and neuroimaging of a patient with ischaemic stroke due to air embolism during manipulation of central venous line. We also review the literature with respect to aetiology, incidence pathophysiology, diagnosis, and treatment options for venous and air embolism. Cerebral air embolism should be considered in patients with sudden neurological deterioration after central venous or arterial manipulations or certain neurological procedures. Prevention, as well as early diagnosis and management, may reduce morbidity and mortality.

**Key Words**

Central venous catheter, cerebral air embolism, hyperbaric oxygen therapy, ischemic stroke

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*Ann Indian Acad Neurol 2014;17:89-91*

## Introduction

Air embolism or the entry of air into the circulation is a serious and often fatal event occurring usually iatrogenically in the ICU setting. Air commonly enters the venous system but may also enter the arterial system with disastrous cardiac, pulmonary or neurological effects and is associated with a high morbidity and mortality.[1] Air embolism is caused by the entry of air through central venous cannulae, pulmonary artery catheters and by hemodialysis catheters. Alternatively air may enter during certain surgical procedures especially neurosurgical procedures in sitting position (cervical disc and posterior fossa tumor surgeries),[2] laparoscopic surgeries and cardiothoracic surgery. In the non-iatrogenic setting, air embolism can occur in deep-sea divers. We report a case of right hemispherical large infarct secondary to air embolism through a central venous catheter placed in the right subclavian vein.

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Quick Response Code:

Website: www.annalsofian.org

DOI: 10.4103/0972-2327.128562

## Case History

A 71-year-old hypertensive and diabetic male had been diagnosed with carcinoma bladder and underwent radical cystectomy with ileal conduit construction. Subsequently he made good recovery and was shifted to ward on the 5th post-op day in a stable condition. On the 7th post-op day the patient was mobilized on a chair and intravenous antibiotics were injected through the central venous catheter by the nursing staff in a sitting position. Immediately following the injection, patient developed abrupt onset tachypnea, profuse sweating followed by unresponsiveness. Examination revealed a stuporose patient with dense left hemiplegia and extensor left plantar (NIHSS = 18). An urgent NCCT [Figure 1] head revealed massive air embolism in the right hemispherical vessels [Figures 1 and 2]. A possibility of vascular air embolism secondary to embolism through the central venous line was considered. TEE did not reveal any air or PFO. Patient was managed with immediate removal of the central line, placing the patient in Trendelenburg and left lateral position, 100% oxygen, IV methylprednisolone and other supportive measures. Further management with hyperbaric oxygen was considered but could not be performed due to hemodynamic instability. Repeat NCCT head after 12 hours revealed spontaneous resolution of the air in the vessels with an evolving infarct in the right parieto-temporal region [Figure 3]. Repeat NCCT head after 4 days revealed a large right hemispheric infarct with significant midline shift and subfalcine herniation [Figure 4].

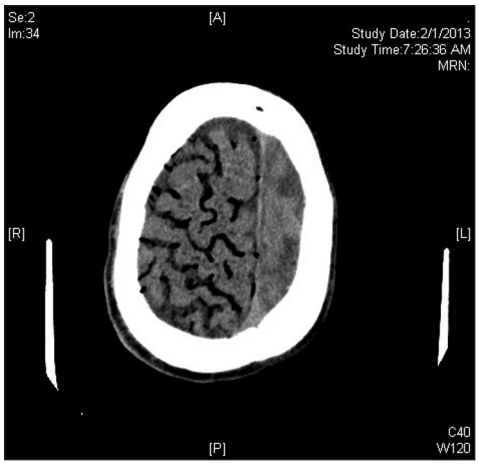


**Figure 1: NCCT head demonstrating massive air embolism in the right hemispherical vessels**

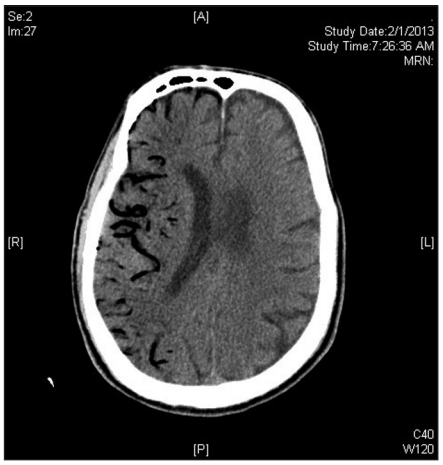


**Figure 2: NCCT head demonstrating massive air embolism in the right hemispherical vessels**

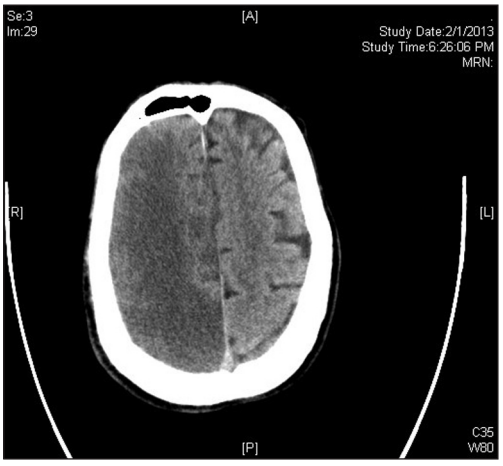


**Figure 3: Repeat NCCT head after 12 hours demonstrating spontaneous resolution of air in the vessels with an evolving infarct in the right parieto-temporal region**

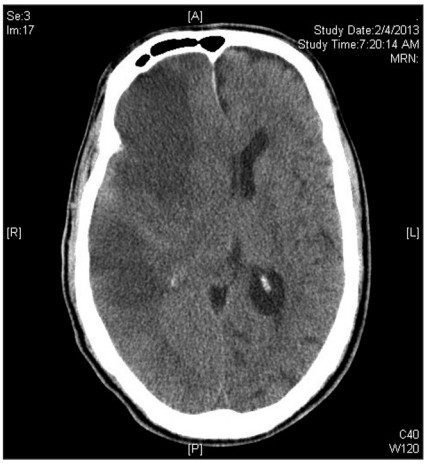


**Figure 4: NCCT head on day 4 revealing a large right hemispheric infarct with significant midline shift and subfalcine herniation**

## Discussion

Cerebral air embolism is a potentially catastrophic event and can involve the venous and arterial vasculature. The entry of air into the blood stream requires a pressure gradient favoring the passage of gas into the blood vessels occurring when the venous pressure is negative relative to the atmospheric pressure for example in deep inspiration, upright position of the patient or hypovolemia at the time of insertion, manipulation or removal of a central line.[3] When air enters the venous system, it can reach the cerebral vasculature in a number of ways. Firstly the presence of a PFO or an atrial sepal defect could facilitate a paradoxical air embolism into the cerebral arterial tree.[4] Secondly, air could enter the cerebral circulation through retrograde cerebral venous air embolism due to lower specific weight of air compared to blood allowing the bubbles to rise to the cranium in an upright positioned patient with an ability to overcome the opposing blood flow in the vein.[5] A more central origin of the right carotid artery and the right internal jugular vein predispose the right hemisphere to a higher risk of air embolism. Thirdly, air can enter into the systemic arterial circulation even in the absence of a cardiac septal defect on account of incomplete filtering of the air in the normal pulmonary capillaries or presence of pulmonary arterial-venous malformation allowing the air to enter into the cerebral arterial circulation.[6]

Air in the vessels causes a foreign body like blockade and also induces an inflammatory reaction secondary to platelet activation and release of vasoactive substances. Cardiac manifestations include chest pain, mill-wheel murmur and ECG evidence of non specific ST-T wave changes with evidence of right ventricular strain and/or tachy/bradyarrythmia.[7] Respiratory manifestations include dyspnea or tachypnea and evidence of hypoxemia and hypercarbia.[7] Neurological manifestations include seizures, encephalopathy and ischemic infarcts with resultant focal neurological deficits.

The frequency of air embolism reported with central venous catheters has ranged from 1 in 47 to 1 in 3000 patients.[7] The diagnosis of air embolism should be suspected whenever a sudden neurological deterioration along with hypotension,

hypoxia or bradycardia occurs in the setting of manipulation of a central venous catheter or immediately following certain neurological procedures like cervical disc or posterior fossa surgeries in the sitting position.[2] Air bubbles can be detected in the cranial vasculature by CT scan or MRI and in the pulmonary and cardiac circulation by transthoracic or transesophageal echocardiography. In our patient drug administration in the central line in the sitting position seems to be the mechanism through which cerebral air embolism occurred. In view of entry of air into the right hemisphere, a possibility of retrograde venous air embolism into the right cerebral hemisphere seems to be the plausible cause of the cerebral air embolism.

Early detection and thereafter prompt management is essential to reduce morbidity and mortality. The source of air must be identified and further vascular entry of air must be stopped promptly. Air must be evacuated from the circulation by aspiration followed by removal of the central venous catheter. Patient should be placed in left lateral and Trendelenburg decubitus position (Durant's maneuver)[8] thereby, allowing the right ventricular outflow tract to be placed below the right ventricular cavity.[2,9] One hundred percent oxygen should be administered to treat hypoxia and to reduce the bubble size by reducing the nitrogen content of the bubbles. Administration of a bolus dose of dexamethasone or methylprednisolone has variably been used to prevent cerebral edema and the inflammatory vascular cascade. There have been variable reports of a better prognosis in patients receiving hyperbaric oxygen and it has been proposed as a treatment of choice. The rationale for hyperbaric oxygen therapy is based on the reduction of the size of the air bubble with compression to 6 atmospheres reducing the size of the air bubble to 1/6 of its original size and hence reducing vascular obstruction and improving the cerebral perfusion.[1,10] In one series of 16 patients treated with hyperbaric oxygen, 50% patients had complete recovery, 31% had partial relief and 19% had no benefit with death in 12.5% patients.[11] This unusual cause of neurological decline in the ICU setting should be kept in mind as an important differential. Appropriate steps like avoiding manipulation of the central venous catheter with patient in the sitting position or during deep inspiration should be avoided. Prompt diagnosis and management which can occur only with a high index of suspicion and awareness about this possible complication can reduce the morbidity and mortality of the disease.

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**How to cite this article:** Suri V, Gupta R, Sharma G, Suri K. An unusual cause of ischemic stroke - Cerebral air embolism. Ann Indian Acad Neurol 2014;17:89-91.
**Received:** 27-08-13, **Revised:** 25-09-13, **Accepted:** 14-10-13
**Source of Support:** Nil, **Conflict of Interest:** Nil