Case Report

Cerebral venous air embolism – Rare complication after central venous catheterization

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Abstract

Cerebral venous air embolism (CVAE) is a rarely reported but potentially disastrous complication associated with manipulation or accidental disconnection of a central venous catheter. This case report brings up one such case of a 62-year-old male who presented with altered sensorium post central venous catheterization and was promptly diagnosed to have developed CVAE based on multi-detector computed tomography imaging.

Keywords: Air foci, central venous catheterization, cerebral venous air embolism, complications, imaging

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INTRODUCTION

Cerebral air embolism (CAE) is an infrequently encountered entity that has very rarely been documented in the literature. It may be associated with insertion, manipulation, withdrawal, or accidental disconnection of central venous catheter (CVC).[1-3] The term CAE refers to air locules in cerebral vasculature–arteries or veins–veins much less frequently than arteries.[4]

Given its catastrophic consequences, a high-degree of suspicion of CAE, prompt imaging-based diagnosis, and adequate treatment are imperative.

It is a rarely documented complication; hence, the purpose of this case report is to make radiologists aware of it as well as the imaging findings, including the importance of changing of window settings of the computed tomography (CT) images to easily detect the presence of air along the course of blood vessels and also not to mistake it for pneumocephalus.

CASE REPORT

A 62-year-old male, a case of multiple myeloma with back pain, was diagnosed with lumbar canal stenosis on magnetic resonance imaging (MRI) and posted for surgery. A central line was introduced successfully in the right internal jugular vein under ultrasound guidance. The patient later developed altered sensorium, drowsiness gradually becoming more aggressive and irritable. Noncontrast enhanced multi-detector CT scan of the brain performed revealed air foci (−1000 HU in density) in the right internal jugular vein, cavernous sinuses, bilateral pterygoid muscles, masticator spaces, infratemporal fossae, and medullary cavity of a few skull base bones (along the emissary's veins) suggesting a diagnosis of cerebral venous air embolism (CVAE) [Figure 1]. High-flow oxygen

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therapy, trendelenberg's position, and supportive therapy were given with gradual recovery in the patient's condition. Follow-up MDCT for the brain performed 1 week later confirmed complete resolution of previously noted air foci within the cerebral venous system [Figure 2].

DISCUSSION

Cerebral venous catheter-associated air embolism may affect the arterial vasculature in the presence of intracardiac or intrapulmonary right to left shunts.[5,6] Rarely, may a retrograde embolism of air bubbles into the venous system from a major central vein occur. The air emboli within the vasculature act as potent vascular irritants and create a local vasoconstrictive response that augments ischaemic damage. Hence, it is a great mimic in acute ischemic stroke.[1,4]

CNS involvement in venous air embolism may have diverse clinical manifestations. It may vary from nonspecific neurological symptoms such as headache, dizziness, and

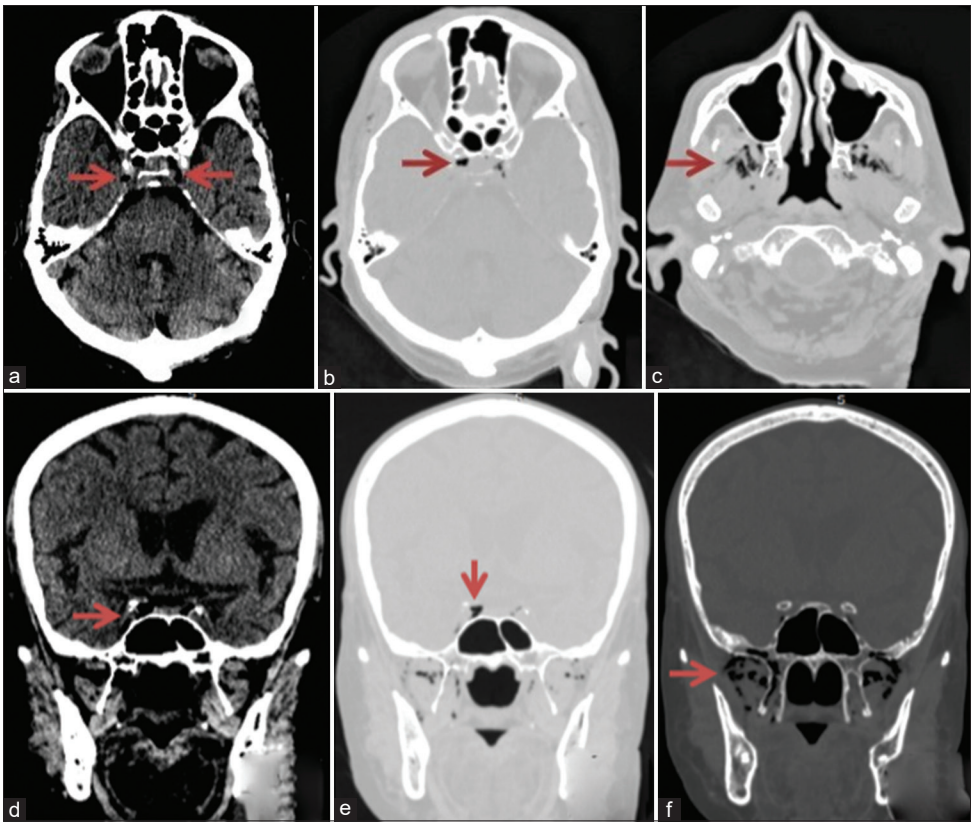

Figure 1: Noncontrast enhanced axial (a-c) and coronal (d-f) MDCT images (brain, lung, and bone window) showing air foci (arrows) in cavernous sinuses, masticator spaces, infratemporal fossae, and medullary cavity of skull base bones (along emissary veins)

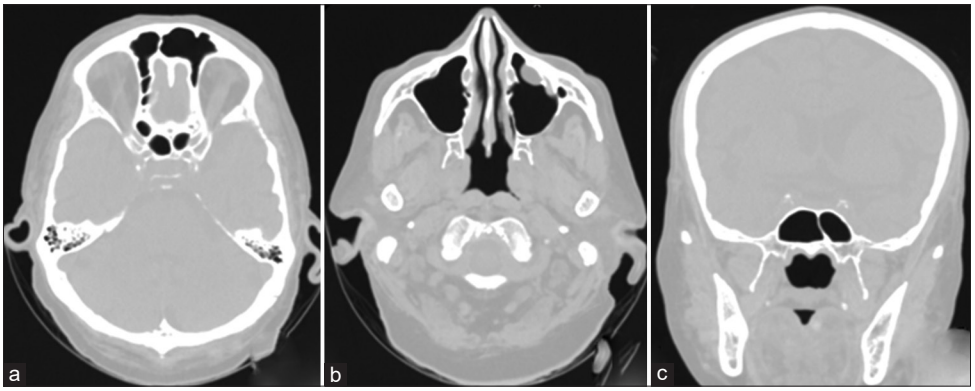

Figure 2: Noncontrast enhanced axial (a and b) and coronal (c) images (lung window) of MDCT brain performed 1 week later confirming complete resolution of previously noted air foci within the cerebral venous system

vertigo to altered mental state, sensory and motor deficits, seizures, and coma.[3]

CAE may be easily diagnosed with the help of imaging. However, a high degree of suspicion and timely imaging is necessary. In a systematic review of iatrogenic CAE related to CVC, the most common locations for emboli, in decreasing order of frequency, are in the subarachnoid space, cerebral parenchyma, and venous sinus.[7]

CT scan of the brain provides the most direct method for confirming the diagnosis of CAE as it can easily detect the air density in CVAE.[8] However, it might be diagnostic only in acute settings[6] as the gas, if present, may get absorbed rapidly. It might reveal foci of air in the arterial or venous system. Lung windows can be helpful to increase the detection of CAE, as seen in the images provided by us, better depicting the extent and distribution of air locules.

MRI is not the primary imaging modality to diagnose CAE as it is not as good as CT in diagnosing air as well as due to its low patient compliance, lack of availability, and high cost. However, when an MRI is performed first, we should be able to consider CVAE as a possibility. According to Hwang and Kim, venous CAE presents as engorged tubular dark signal intensities with aliasing and blooming artifacts on susceptibility weighted imaging (SWI) due to marked susceptibility artifacts caused by air. It is necessary to differentiate air emboli from hemorrhage, which also presents as dark signal intensities and even aliasing artifacts on SWI. Integrated interpretation considering imaging findings on SWI and T1- and T2-weighted images can be helpful to differentiate CVAE from hemorrhage as unlike hemorrhage, air foci appear as dark signal intensities both on T1- and T2-weighted images in addition to blooming on SWI, i.e. dark signal intensities on all MR sequences with aliasing artifacts on the susceptibility weighted (SW) filtered phase image should point toward air embolism.[9] Apart from this, MRI might commonly help in evaluating complications of CAE rather than directly detecting air. It might reveal– (i) ischemia due to interruption of cerebral arterial flow with associated cytotoxic edema on diffusion weighted imaging (DWI) (ii) ischemia due to vasogenic edema and venous infarcts (iii) vasogenic edema without acute ischemia due to inflammation and breakdown of the blood–brain barrier. Studies have shown a right hemispheric predominance of these ischemic/edematous lesions in CAE[1] probably because air bubbles follow the bloodstream directly from the aorta to the right innominate artery or the right vertebral artery.[3]

Kaichi *et al*. in their two case studies of changes over time in intracranial air in patients with CAE advise early CT scan of brain for diagnosis of CVAE. Use of DWI is advocated in suspected CVAE despite the absence of intracranial air on CT scans as cerebral infarction observed mainly in cortical areas is coincident with edema due to CVAE although the air was quickly absorbed.[10]

Pinho *et al*. in their systematic review of 158 cases of CAE associated with CVCs noted an association of mortality and the presence of air bubbles in CT and the presence of any cerebral lesion secondary to gas embolism.[1]

In suspected CAE, any obvious source of air entry, if found, should be appropriately occluded, preventing further air entry into circulation. Trendelenberg's position and general supportive measures like hyperbaric oxygen therapy[2,3,8] may help. Prevention of CAE is crucial and proper clinical protocol and technique must be strictly observed to avoid such complications.

CONCLUSION

CVC-associated CAE may be a catastrophic event which can be prevented by proper and judicious use of CVCs and training of health professionals. Diagnosing CAE requires a high degree of suspicion and prompt MDCT imaging of the brain which will reveal foci of air density along the course of vessels. These air foci may be subtle, hence changing window settings will be of help to easily detect them. In a patient with CVC with sudden neurological deterioration, this complication needs to be borne in mind by means of which appropriate treatment can be started to reduce mortality and morbidity.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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