Case Report

Gyriform Infarction in Cerebral Air Embolism: Imaging Mimicker of Status Epilepticus

Konark Malhotra, Appaji Rayi

Department of Neurology, Charleston Area Medical Center, West Virginia University Charleston Division, Charleston, West Virginia, USA

Abstract

Cerebral air embolism (CAE) is a potentially fatal iatrogenic complication related to common procedures including central venous catheter (CVC) removal. We report an interesting case of CAE related to CVC removal that was further complicated with status epilepticus. Neuroimaging of CAE and status epilepticus could pose diagnostic dilemmas and require consideration of wide diagnostic differentials. We discuss the clinical presentation, mechanism, and diagnostic approach, especially neuroimaging to differentiate various etiologies in CAE patients.

Keywords: Cerebral air embolism, gyriform, laminar necrosis, stroke

Introduction

Cerebral air embolism (CAE) is a rare, iatrogenic, potentially fatal complication with varied systemic and neurological sequelae.[1] Relatively, common procedures such as removal of central venous catheter (CVC) can predispose to CAE either through retrograde or paradoxical pathways. The neurological manifestations usually include headaches, encephalopathy, seizures, or stroke. The seizures are often brief in duration; however, rarely, they could evolve to status epilepticus. Status epilepticus in these patients is usually refractory to medications and results in poor clinical outcomes or withdrawal of care.[2,3] Clinical examination and neuroimaging in these patients remain critical during acute decision-making settings. We describe a case of paradoxical CAE after CVC removal that was further complicated with a prolonged course of neurological recovery due to status epilepticus.

Case Report

A 72-year-old male with a medical history of coronary artery disease, diabetes mellitus, hypertension, tobacco dependence, Bell's palsy, and mechanical aortic valve (magnetic resonance imaging [MRI] compatible) on warfarin presented with worsening chest pain and shortness of breath. On admission, the patient had a mild temperature elevation, positive blood cultures with coagulase-negative *Staphylococcus aureus*, and elevated cardiac enzymes (troponin I = 18.9 ng/mL). A right internal jugular venous catheter was inserted due to poor venous access. Due to the history of mechanical aortic valve and clinical suspicion for bacterial endocarditis, a transesophageal echocardiogram (TEE) was performed. TEE revealed moderate-severe left ventricular dysfunction with a tiny right-to-left cardiac shunt through patent foramen ovale; however, vegetations were not observed. Repeat blood cultures were negative, suggesting a possible contaminated initial blood sample. The patient was diagnosed with non-ST elevation myocardial infarction and required multi-vessel coronary angioplasty. Triple antithrombotic therapy was initiated after the procedure, and the patient was deemed stable for discharge. The CVC was removed in upright position after applying mechanical pressure for approximately 2 min. Fifteen minutes later, acute worsening of neurological status was noted and a stroke code was activated. The initial National Institute of Health Stroke Scale was 14 involving deranged level of consciousness, aphasia, gaze deviation to the right, left upper and lower extremity weakness, and left hemisensory loss (excluding the baseline right facial droop).

Address for correspondence: Dr. Konark Malhotra, Department of Neurology, Charleston Area Medical Center, West Virginia University, Charleston, WV 25301, USA.
E-mail: Konark.malhotra@yahoo.com

Access this article online

Quick Response Code:

Website: www.annalsofian.org

DOI: 10.4103/aian.AIAN_94_17

How to cite this article: Malhotra K, Rayi A. Gyriform infarction in cerebral air embolism: Imaging mimicker of status epilepticus. Ann Indian Acad Neurol 2017;20:313-5.

Immediate noncontrast computed tomography (NCCT) of the head was negative for frank hypodensity or hemorrhage; however, air emboli in bilateral cerebral hemispheres were noted. Intracranial and extracranial vasculature was negative for critical stenosis, occlusions, or cerebral aneurysms. On clinical examination, the patient was observed to have left hand shaking with a fluctuating mental status, raising a possibility of seizure. He was treated with intravenous lorazepam followed by loading dose of levetiracetam, initiated on high flow oxygen, and transferred to the Neuroscience Intensive Care Unit (ICU). Initial routine electroencephalography (EEG) demonstrated right greater than left hemispheric slowing. However, continuous video EEG monitoring revealed discrete focal seizures emanating from the right parasagittal region with occasional generalization, consistent with complex partial status epilepticus (CPSE) [Figure 1]. The patient was intubated for airway protection and required multiple anti-epileptic medications including fosphenytoin and zonisamide for seizure control without much success. Eventually, general anesthesia with midazolam was introduced with successful control of seizures. The patient was maintained on prolonged EEG monitoring during ICU care and discontinued after deemed seizure free for 24 h. On day 5 of the index event, MRI of the brain (1.5 Tesla) showed multiple confluent, gyriform restricted diffusion lesions that were located in the right cortical hemisphere [Figure 2]. The clinical status of our patient gradually improved and later was discharged to a rehabilitative facility with mild left-sided hemiparesis. Our patient remained clinically stable during the hospital course and on 3 month follow-up appointment.

Discussion

CVC removal is a relatively common iatrogenic procedure with potential complications of CAE and status epilepticus. The risk of CAE increases with a patient being in sitting position.[4] Usually, patients should be in end of inspiration phase and placed in Trendelenberg position before CVC removal. Slow and continuous withdrawal followed by mechanical pressure for at least 5 min with occlusive dressing is critical to prevent CAE.[5] Our patient developed CAE likely due to CVC removal performed in upright position and probably was not in expiratory phase. Although the incidence and mortality of CVC-related CAE are approximately 0.2%–1%[6] and 23%, respectively,[7] lethal volumes of cerebral air of as low as 2 ml have been reported.[1] Various patient-related factors during CVC removal that reduce the central venous pressure and predispose to CAE include upright positioning, deep inspiration, and hypovolemia.[8] Factors associated with unfavorable outcome in CAE include old age, encephalopathy, hemiparesis, and evidence of gyriform air on initial brain imaging.[2] Proposed mechanisms leading to CAE include either retrograde flow or right-to-left paradoxical passage of cerebral air embolus. The retrograde mechanism involves reflux of air through jugular venous system leading to venous CAE, while paradoxical embolism involves trespassing of venous air to arterial system through cardiac or pulmonary shunts leading to arterial CAE.[9]

Intravascular cerebral air can predispose to neurological injury through following mechanisms: (a) mechanical obstruction of distal cerebral vasculature causing neuronal damage and cytotoxic edema, (b) endothelial irritation by air bubble causing blood-brain barrier disruption, (c) immune response activation by the surface of air bubbles.[1] These mechanisms lead to reduced blood oxygen and glucose transport to small cerebral arterioles (30–60 μm). Variations in cerebral perfusion and cortical neuronal injury lead to epileptogenic alterations that could predispose to poststroke epilepsy and status epilepticus.[10]

Neuroimaging remains paramount to discern litany of differentials in cerebrovascular disorders.[11,12] Cheng *et al*. have described different imaging patterns in CAE patients including gyriform, parenchymal or subarachnoid, and venous sinus air bubbles.[2] Diagnosis could be established with NCCT or diffusion-weighted imaging sequences of MRI; however, rapid resorption of air emboli through tiny arterioles on repeat neuroimaging could pose a challenge for clinicians. The gyriform-restricted diffusion in CAE coincides with the disruption of BBB and cytotoxic edema;[13] however, these imaging patterns often mimic as cortical laminar necrosis and pose a diagnostic dilemma. Cortical laminar necrosis ensues with various energy-depleted conditions including hypoxia, ischemia, and seizures.[14] A high index of clinical suspicion is essential as encephalopathy and laminar necrosis confound the clinical picture in CAE. However, evaluation of these

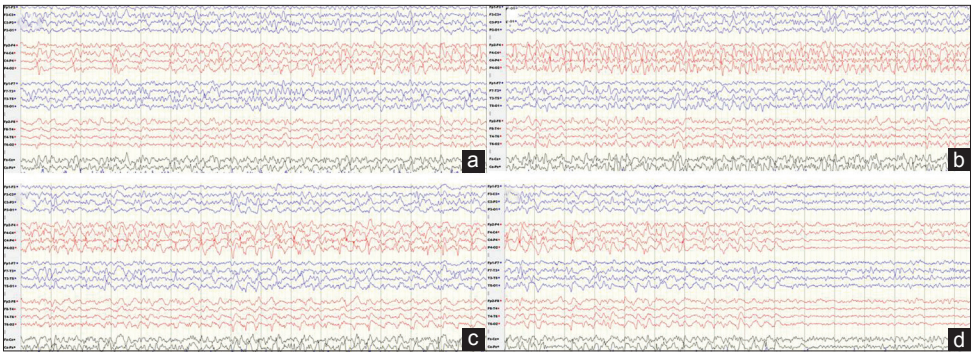

Figure 1: (a-d) Consecutive epochs of electroencephalography illustrating right parasagittal (F4/C4 leads) epileptogenic focus with eventual resolution

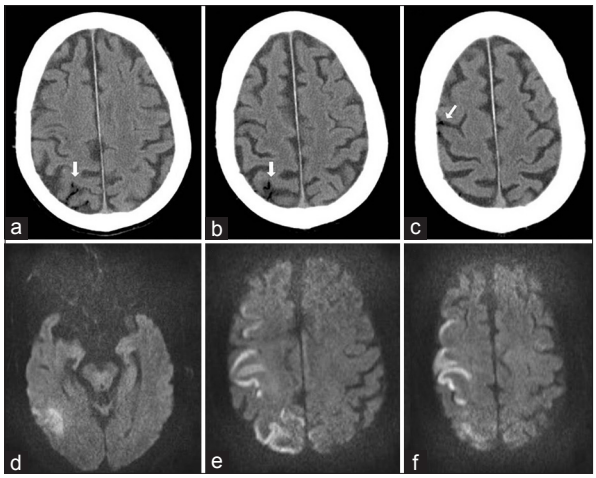

Figure 2: (a-c) Noncontrast computed tomography, axial sequences, showing sulcal air in the precentral and central sulci of right frontal cortex and intraparietal sulci of right cerebral hemisphere. (d-f) Brain magnetic resonance imaging axial diffusion-weighted sequences demonstrating gyriform restricted diffusion corresponding to sulcal air

patients for status epilepticus with prolonged EEG monitoring remains critical to prevent worse clinical outcomes and future prognostication. Severe encephalopathy in our patient was initially correlated to CAE; however, an exhaustive search of other differentials led to the diagnosis of CPSE with prompt management and improved clinical outcome. Treatment of CAE usually includes (Trendelenberg) positioning of the patient and high flow oxygen therapy; however, endovascular aspiration of the air embolus has been discussed as well.[15]

Conclusion

This case highlights to maintain a wide diagnostic differential and a high index of clinical suspicion to assess the temporal relationship between iatrogenic procedures and development of encephalopathy in CAE patients. Encephalopathy in these patients should be carefully discerned from other clinical and radiological mimickers to avoid worse prognosis.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Muth CM, Shank ES. Gas embolism. N Engl J Med 2000;342:476-82.
2. Cheng CK, Chang TY, Liu CH, Chang CH, Huang KL, Chin SC, *et al.* Presence of gyriform air predicts unfavorable outcome in venous catheter-related cerebral air embolism. J Stroke Cerebrovasc Dis 2015;24:2189-95.
3. Cordeiro I, Teodoro T, Peralta R, Bentes C. Cerebral gas embolism presenting as refractory status epilepticus. Epilepsia 2013;54:122.
4. Pinho J, Amorim JM, Araújo JM, Vilaça H, Ribeiro M, Pereira J, *et al.* Cerebral gas embolism associated with central venous catheter: Systematic review. J Neurol Sci 2016;362:160-4.
5. Hsiung GY, Swanson PD. Cerebral air embolism after central venous catheter removal. Neurology 2000;55:1063-4.
6. Gordy S, Rowell S. Vascular air embolism. Int J Crit Illn Inj Sci 2013;3:73-6.
7. Heckmann JG, Lang CJ, Kindler K, Huk W, Erbguth FJ, Neundörfer B. Neurologic manifestations of cerebral air embolism as a complication of central venous catheterization. Crit Care Med 2000;28:1621-5.
8. Vesely TM. Air embolism during insertion of central venous catheters. J Vasc Interv Radiol 2001;12:1291-5.
9. Mishra R, Reddy P, Khaja M. Fatal cerebral air embolism: A case series and literature review. Case Rep Crit Care 2016;2016:3425321.
10. Velioglu SK, Ozmenoglu M, Boz C, Alioglu Z. Status epilepticus after stroke. Stroke 2001;32:1169-72.
11. Malhotra K, Liebeskind DS. Imaging in endovascular stroke trials. J Neuroimaging 2015;25:517-27.
12. Liebeskind DS, Malhotra K, Hinman JD. Imaging as the nidus of precision cerebrovascular health: A million brains initiative. JAMA Neurol 2017;74:257-8.
13. Caulfield AF, Lansberg MG, Marks MP, Albers GW, Wijman CA. MRI characteristics of cerebral air embolism from a venous source. Neurology 2006;66:945-6.
14. Saito Y, Ogawa T, Nagaishi J, Inoue T, Maegaki Y, Ohno K. Laminar cortical necrosis in adrenal crisis: Sequential changes on MRI. Brain Dev 2008;30:77-81.
15. Belton PJ, Nanda A, Alqadri SL, Khakh GS, Chandrasekaran PN, Newey C, *et al.* Paradoxical cerebral air embolism causing large vessel occlusion treated with endovascular aspiration. J Neurointerv Surg 2017;9:e10.