EURASIAN JOURNAL OF EMERGENCY MEDICINE
DOI: 10.4274/eajem.galenos.2023.49002
Case Report
Eurasian J Emerg Med. 2023;22(4): 248-50

Seizure and Altered Mental Status After Thoracentesis: Cerebral Air Embolism

Çağrı Safa Buyurgan, Hüseyin Narcı, Cüneyt Ayrık

Mersin University Faculty of Medicine, Department of Emergency Medicine, Mersin, Turkey

Abstract

Cerebral air embolism (CAE) is a rare life-threatening condition. It may clinically mimic acute ischemic stroke by decreasing cerebral perfusion pressure and brain tissue oxygenation and may cause impaired consciousness and epileptic seizures. In its etiology; iatrogenic causes such as central venous catheterization, endoscopy, sclerotherapy, major surgeries, and invasive lung interventions are mostly seen. The most useful imaging method for diagnosis is cranial computed tomography (CT). In this presentation, we present the case of a 63-year-old male patient admitted to the emergency department (ED) with complaints of mental status changes and seizures and weakness in the left upper and lower extremities on physical examination. The patient had a history of thoracentesis performed three days ago and was discharged. Brain CT of the patient showed signs of newly developing CAE, and diffusion magnetic resonance imaging showed findings consistent with right middle cerebral artery infarction in his second admission. We thought that the symptoms of the patient, who had no history of additional intervention, were due to CAE, which developed as a complication of thoracentesis. Thoracentesis is one of the invasive procedures that can be applied for diagnostic/therapeutic purposes in the ED and does not have complications such as pneumothorax, hemothorax, soft tissue infection, or intra-abdominal organ injury. Physicians should be aware that CAE may also occur in patients who develop neurological deficits after thoracentesis. The clinician's high suspicion, prompt diagnosis, and treatment can be lifesaving.

Keywords: Cerebral air embolism, emergency department, thoracentesis

Introduction

Thoracentesis is a frequent invasive procedure in emergency departments (EDs) for diagnosis and treatment. Although it is generally a non-hazardous procedure, it may cause life-threatening complications such as bleeding, pneumothorax, re-expansion pulmonary edema, spleen, or liver perforation. In addition to these complications, cerebral air embolism (CAE) is an extremely rare complication of thoracentesis (1,2). CAE is a rare life-threatening condition, and most are iatrogenic. In literature, it is defined that it may be related to antral venous catheterization, endoscopy, sclerotherapy, major surgeries, and invasive lung procedures (3,4). CAE may be 20% mortal in the acute period and may mimic acute ischemic stroke by decreasing cerebral perfusion pressure and brain tissue oxygenation. Because the symptoms and signs of air embolism are not specific, diagnosis may be difficult. Neurological symptoms may include seizures, stroke, loss of consciousness, and altered mental status (5). The most useful imaging method in diagnosis is cranial computed tomography (CT), which shows intracranial air bubbles in 2/3 of the patients (6). Magnetic resonance imaging (MRI) also should be performed in patients with suspected infarcts with air embolism (3). The mechanism of CAE after thoracentesis is unclear. However, some hypotheses have been suggested. Air particles can pass through the arterial system in three different ways: directly through the venous to arterial system through pulmonary veins with a physiological shunt and cardiac septal defect (7,8). Arterial circulating air may obstruct microcirculation and cause ischemic endorgan damage. The most vulnerable organs affected by the microbubbles that cause arterial ischemia are the brain and heart. It is believed that 2 mL air in the cerebral arteries and 0.5 to 1 mL in the coronary circulation can be fatal (9,10). Because the brain consumes 15-20% of the cardiac volume, a large portion of the gas bubbles are transported to the brain by

Corresponding Author: Çağrı Safa Buyurgan MD, Mersin University Faculty of Medicine, Department of Emergency Medicine, Mersin, Turkey
Phone: +90 545 260 26 24 **E-mail:** cagribuyurgan88@yahoo.com.tr **ORCID ID:** orcid.org/0000-0001-8662-0670

Cite this article as: Buyurgan ÇS, Narcı H, Ayrık C. Seizure and Altered Mental Status After Thoracentesis: Cerebral Air Embolism. Eurasian J Emerg Med. 2023;22(4): 248-50.

Received: 01.03.2023
Accepted: 08.05.2023

blood flow and cause the occlusion of cerebral vessels, such as in cerebral arterial gas embolism (11).

For the management of CAE in the EDs, vital functions should be supported and 100% oxygen therapy should be initiated. Positioning of the patient, a control of seizures, and intubation if necessary are other supportive treatment methods. The patient should be placed in the supine position. In the past, the trendelenburg position was used for treating CAE, but nowadays it is no longer recommended because it increases cerebral ischemia. Hyperbaric oxygen (HBO) treatment is recommended for definitive treatment. It increases the arterial partial pressure of oxygen, facilitates the dissolution of oxygen in plasma, and increases diffusion into the tissue. It can be used in patients with neurological deficits, end organ damage and hemodynamic deterioration (1,3,4).

Case Report

A 63-year-old male presented to the ED with seizure and loss of consciousness. He had no history of additional disease. His blood pressure was 150/90 mmHg, pulse was 98 beats/min, respiratory rate was 22 breaths/min, and oxygen saturation was 92% (in the room air) as vital signs. Physical examination revealed disorientation with left upper and lower extremity weakness. His laboratory values were unremarkable. Three days ago, the patient underwent diagnostic thoracentesis for a newly diagnosed mass in the lung after presenting to the ED with dyspnea. Thoracentesis was not performed using an ultrasonography guide, and approximately 50 cc pleural fluid was removed. In his second admission, the patient was placed on 100% nonrebreather oxygen and CT scan with MRI was planned for intracranial hemorrhage or infarction. No signs of intracranial hemorrhage were detected on CT, but air bubbles on the right cerebral hemisphere confirmed the diagnosis of CAE (Figure 1). Brain diffusion-weighted MRI also showed hyperintense lesions in the right hemisphere compatible with the right middle cerebral artery (MCA) infarct (Figure 2). As the patient had no history of other intervention, we thought that air embolism had developed due to thoracentesis performed three days ago. After consultation with a neurologist, 0.6 mg intramuscular enoxaparin and 1,250 mg phenytoin intravenously in 250 mL saline over 45 min were given as medication. For etiological research, echocardiography was performed on the patient and it showed no patent foramen ovale (PFO) or atrial septal defect. Because the patient was unstable, he could not be transported for HBO treatment. He was admitted to the intensive care unit (ICU) for further examination and treatment. Diffusion MRI of the patient performed in the ICU also confirmed MCA infarction secondary to air embolism. The patient's ischemic cerebrovascular event and side findings were associated with air embolism. After being hospitalized and followed up for 45 days in the ICU, he was discharged with left hemiparesis sequelae and a peg tube inserted. Antiepilectic and anticoagulant treatments were prescribed.

Discussion

Our patient had no etiologic factors that could cause CAE, such as trauma, surgical intervention, catheterization, and PFO. We therefore thought that CAE may be due to thoracentesis. He had an air embolism in the right hemisphere on CT imaging and infarct findings in the same area detected on MR imaging. We thought that the infarct area developed secondary to ischemia caused by air embolism.

In a study evaluating 15 patients with CAE; it was observed that 80% of the patients presented with focal deficit and/or coma (53%). Seizures were detected in 47% of the patients (12). Although there are studies proving that brain CT scans show CAE in 2/3 of the patients (6), the brain CT scans performed in this study were sensitive in only 4 of 13 patients, whereas MRI scans were found to show multifocal diffusion restriction (12). In the patient we presented, air embolism was detected in the brain CT performed in the acute period, and diffusion limitations in MRI. In studies in the literature, imaging findings such as intracranial air embolisms and ischemic or edematous lesions were associated with increased mortality but were not statistically significant (6). Heckmann et al. (9) showed that the presence of focal neurological signs, encephalopathy, or epileptic seizures were not individual markers of mortality. Similarly, CAE detected in our case who presented with the complaints

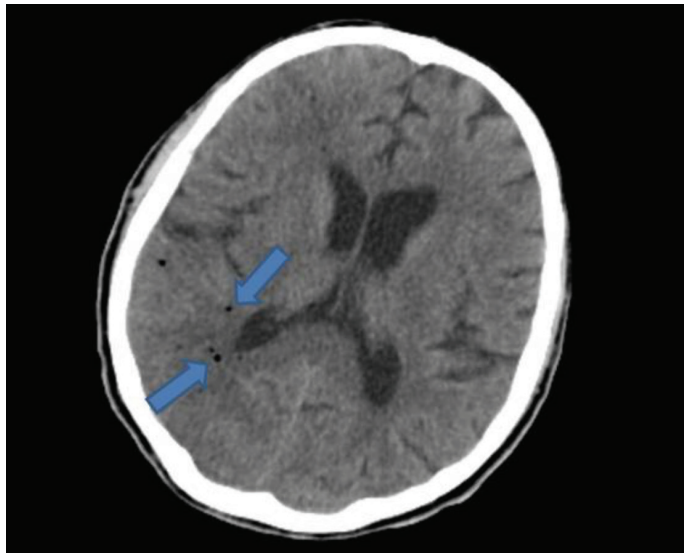

Figure 1. Arrows show cerebral air embolism on the right cerebral hemisphere in computed tomography imaging

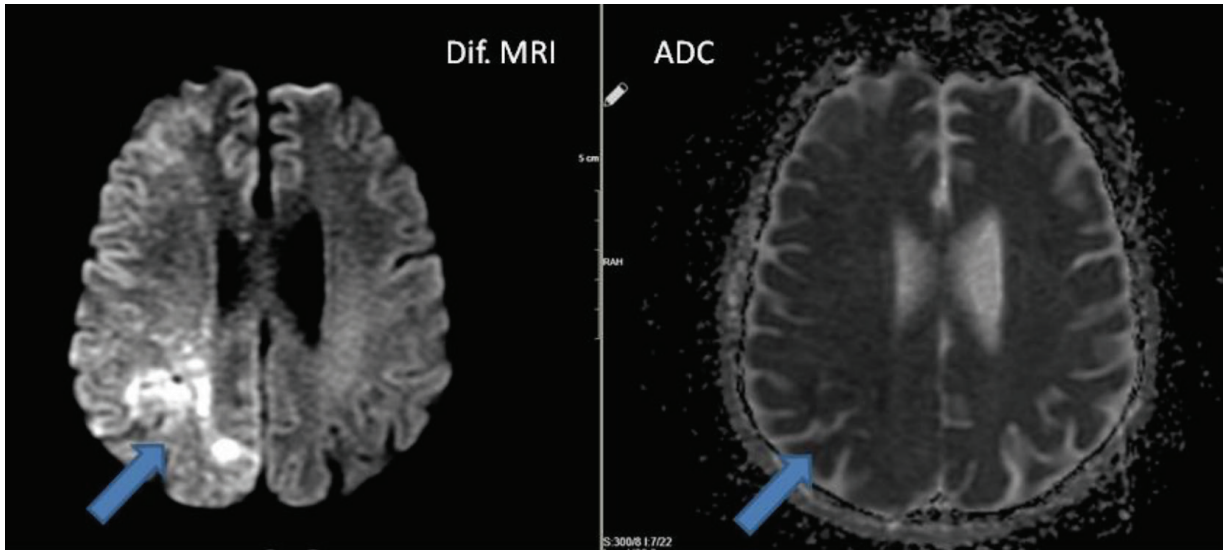

Figure 2. Arrows show acute cerebral infarct on the right cerebral hemisphere in magnetic resonance imaging

of altered consciousness, seizure, and left hemiparesis did not cause mortality but caused morbidity.

Conclusion

CAE is a rare but life-threatening condition. When a neurological event (altered mental status, seizure, focal neurologic signs) develops after venous catheterization, neurosurgical, vascular, and cardiac surgeries, gastroenterology procedures, pulmonary barotrauma, and invasive lung procedures, it should always be suspected. Rapid diagnosis and supportive treatment with HBO therapy can be lifesaving.

Ethics

Informed Consent: Informed consent was not required as no personally identifying information was used this case.

Peer-review: Externally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: Ç.S.B., Concept: Ç.S.B., Design: H.N., Data Collection or Processing: H.N., Analysis or Interpretation: H.N., Literature Search: C.A., Writing: C.A.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

References

1. Cantey EP, Walter JM, Corbridge T, Barsuk JH. Complications of thoracentesis: incidence, risk factors, and strategies for prevention. Curr Opin Pulm Med. 2016;22:378-85.
2. Diamond S, Kaplitz S, Novick O. Cerebral Air Embolism As A Complication of Thoracentesis. GP. 1964;30:87-91.
3. Yesilaras M, Atilla OD, Aksay E, Kilic TY. Retrograde cerebral air embolism. Am J Emerg Med. 2014;32:1562.
4. Parker CD, Shea CM. Cerebral air embolism. Am J Emerg Med. 2017;35:374.
5. Mirtchev D, Mehta T, Daniel A, Finstein T, McCullough L. Pearls & Oy-sters: Enhancing vigilance for detection of cerebral air embolism: From syncope to death. Neurology. 2018;91:717-20.
6. 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.
7. Muth CM, Shank ES. Gas embolism. N Engl J Med. 2000;342:476-82.
8. Bou-Assaly W, Pernicano P, Hoeffner E. Systemic air embolism after transthoracic lung biopsy: A case report and review of literature. World J Radiol. 2010;2:193-6.
9. 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.
10. Orebaugh SL. Venous air embolism: clinical and experimental considerations. Crit Care Med. 1992;20:1169-77.
11. Radecka E, Stjernqvist P, Nyren S, Nekludov M. Iatrogen cerebral arteriell gas embolism after lung biopsy. Lakartidningen. 2017;114:D93A.
12. Brown AE, Rabinstein AA, Braksick SA. Clinical Characteristics, Imaging Findings, and Outcomes of Cerebral Air Embolism. Neurocrit Care. 2023;38:158-64.