

A RARE COMPLICATION OF CENTRAL VENOUS CATHETER REMOVAL: CEREBRAL AIR EMBOLISM

Santral Venöz Kateterin Çıkarılmasının Nadir Bir Komplikasyonu: Serebral Hava Embolisi

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ÖZET

Serebral hava embolisi (SHE) diyaliz kateteri kullanımının nadir görülen bir komplikasyonudur. SHE sıklıkla santral venöz kateter (SVK) yerleştirilmesinden sonra ortaya çıksa da, nadiren SVK çıkarılması sonucunda da oluştuğu bildirilmiştir. 67 yaşında bir erkek hasta, oturur pozisyondayken sağ juguler vene yerleştirilen SVK'nın çıkarılmasından hemen sonra bilinç değişikliği, konuşmama ve sağ tarafında güçsüzlük yaşaması üzerine hızla acil servise getirildi. Muayenesinde konfüzyon ve spontan göz açma vardı, kooperasyon yoktu, sol üst ve alt ekstremiteler plejikti, sol plantar refleks (PR) ekstansör yanıtı vardı. Beyin bilgisayarlı tomografisinde (BT) sağ serebral hemisferde, oksipital bölgede ve sağ temporal bölgede hava görüntüleri saptandı. Toraks BT'de sağ juguler vende lümen içinde hava yoğunluğu saptandı. Difüzyon manyetik rezonans görüntüleme sağ parietal lob posteriorunda, özellikle subkortikal alanda, belirgin subakut iskemi ile uyumlu difüzyon kısıtlaması saptandı. Hastaya KAE tanısı konuldu ve tedavisi için hiperbarik oksijen başlandı. Hasta takiplerinde kötüleşmesi üzerine entübe edilerek yoğun bakım ünitesine alındı.

Anahtar Kelimeler: Diyaliz kateteri, İnme, Venöz hava embolizasyonu

ABSTRACT

Cerebral air embolism (CAE) is a rare complication of dialysis catheter use. Although CAE often occurs after central venous catheter (CVC) insertion, it has also been reported to rarely occur as a result of CVC removal. A 67-year-old male patient was quickly brought to the emergency room after he experienced altered consciousness, inability to speak, and weakness on the right side immediately after removal of the CVC inserted into the right jugular vein while the patient was in a sitting position. In his examination, he had confusion and spontaneous eye opening; there was no cooperation; left upper and lower extremities were plegic; and he had left plantar reflex (PR) extensor response. In the brain computed tomography (CT), air images were found in the right cerebral hemisphere, occipital region, and right temporal region. Thorax CT revealed air density within the lumen in the right jugular vein. Diffusion magnetic resonance imaging revealed diffusion restriction in the right parietal lobe posterior, especially in the subcortical area, consistent with pronounced subacute ischemia. The patient was diagnosed with CAE, and hyperbaric oxygen was started for his treatment. The patient was intubated upon worsening in the follow-ups and taken to the intensive care unit.

Keywords: Dialysis catheter, Stroke, Venous air embolization

INTRODUCTION

Cerebral air embolism (CAE) after removal of the central venous catheter (CVC) is a rare condition that is associated with high morbidity and mortality (Chuang et al., 2019). Because there is a significant difference between the densities of air and blood, intravascular air bubbles cause symptoms according to the organ to which they have migrated; these include neurological sequelae (i.e. stroke), respiratory failure, pulmonary hypertension, acute cor pulmonale, complete cardiovascular collapse, and death (Pandurangadu, et al., 2012 ; Sahutoglu, T., et al. 2017). We present a case of CAE that occurred following removal of the hemodialysis catheter.

CASE REPORT

A 67 year old male patient underwent dialysis through his existing fistula in our hospital's dialysis center for chronic renal failure. Immediately after removal of CVC, which had previously been inserted into the right jugular vein while the patient was

in a sitting position, the patient experienced altered consciousness, inability to speak, and weakness on the right side, upon which he was quickly brought to the emergency room. At the first examination of the patient in the emergency room, he had confusion, spontaneous eye opening, did not comply with verbal commands, and there was no cooperation. Neurological examination revealed no facial asymmetry or spontaneously moving upper and lower extremities; left upper and lower extremities of the patient were plegic, and there was right TCR flexor response and left TCR extensor response. The National Institutes of Health Stroke Scale score was calculated to be 21. The patient's vital findings were as follows; temperature: 36.9°C (98.4 F), heart rate: 86 beats/min, blood pressure: 145/79 mm Hg, respiratory rate: 17 breaths/min, and saturation in room air: 94%. After the patient was placed under observation with an oxygen mask, non-contrast emergency computed tomography (CT) and diffusion magnetic resonance imaging (MRI) scan were quickly performed. Brain CT scan showed no hemorrhage or infarction, but air images were present in the occipital region of the right cerebral hemisphere

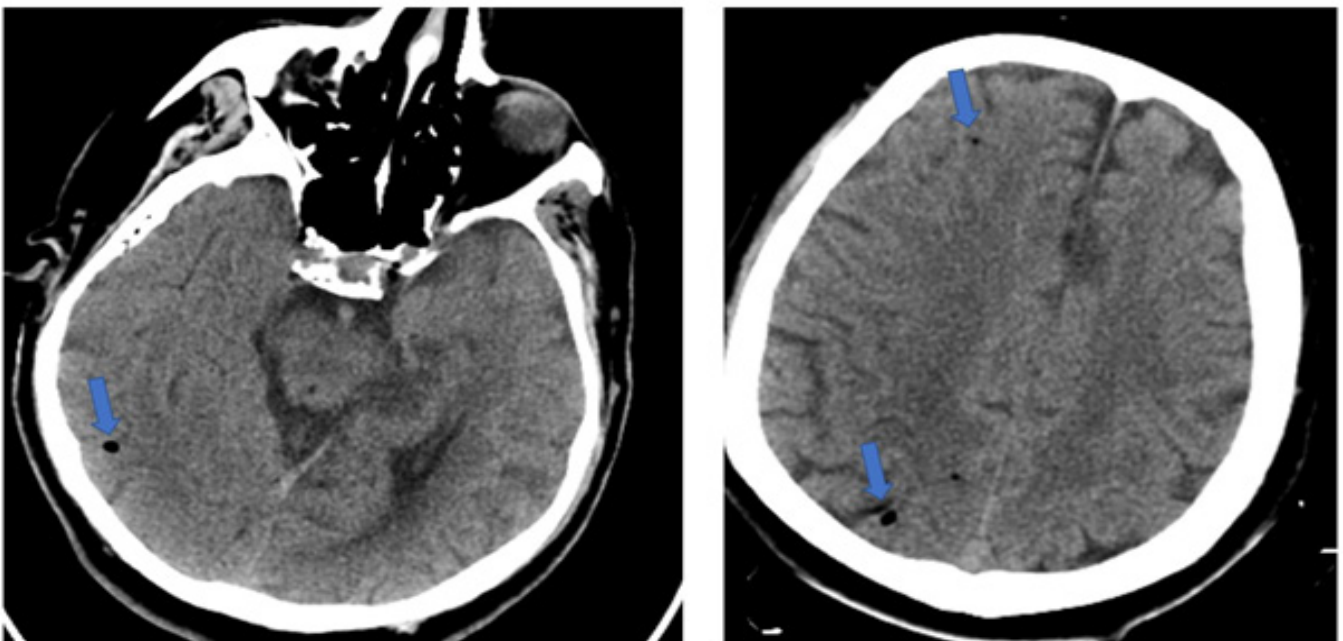


Figure-1: Aerial images in the brain

and in the right temporal region -371HU (Hounsfield unit) (Figure-1).

In addition, thorax CT scan revealed air densities within the lumen in the right jugular vein (Figure-2).

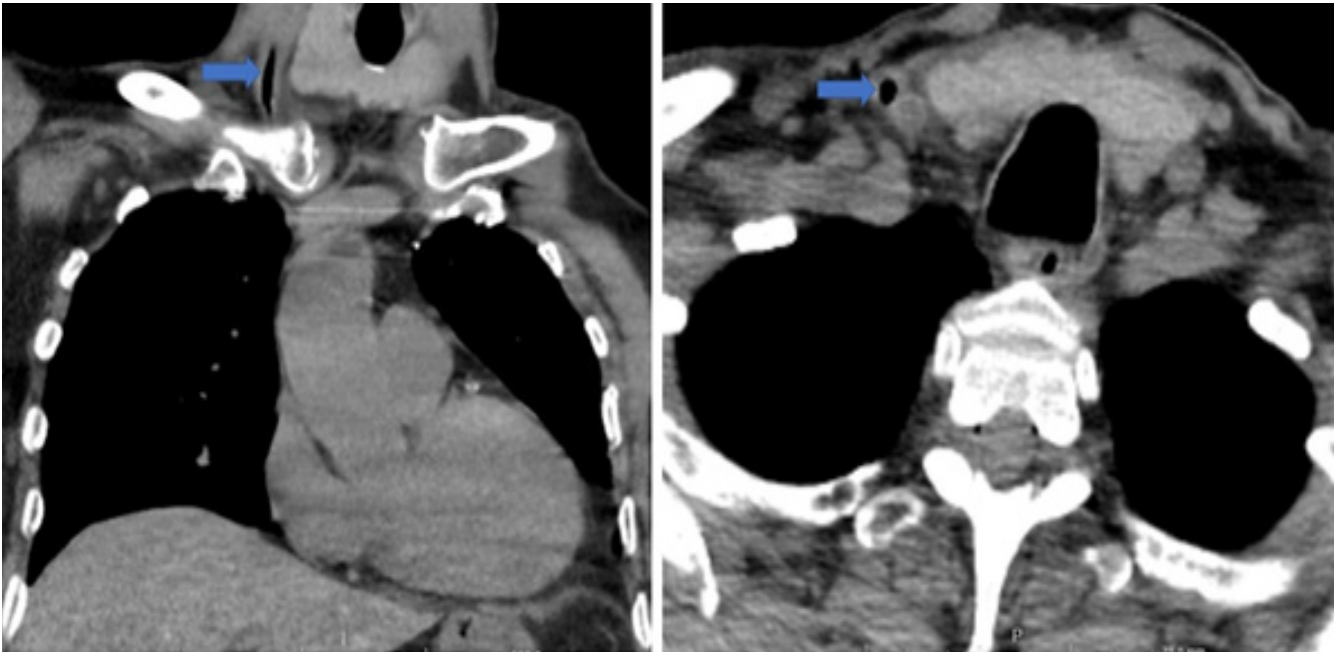


Figure-2: Aerial view inside the right jugular vein

Diffusion MRI revealed diffusion restriction consistent with pronounced subacute ischemia in the right parietal lobe posterior, especially in the subcortical area (Figure-3).

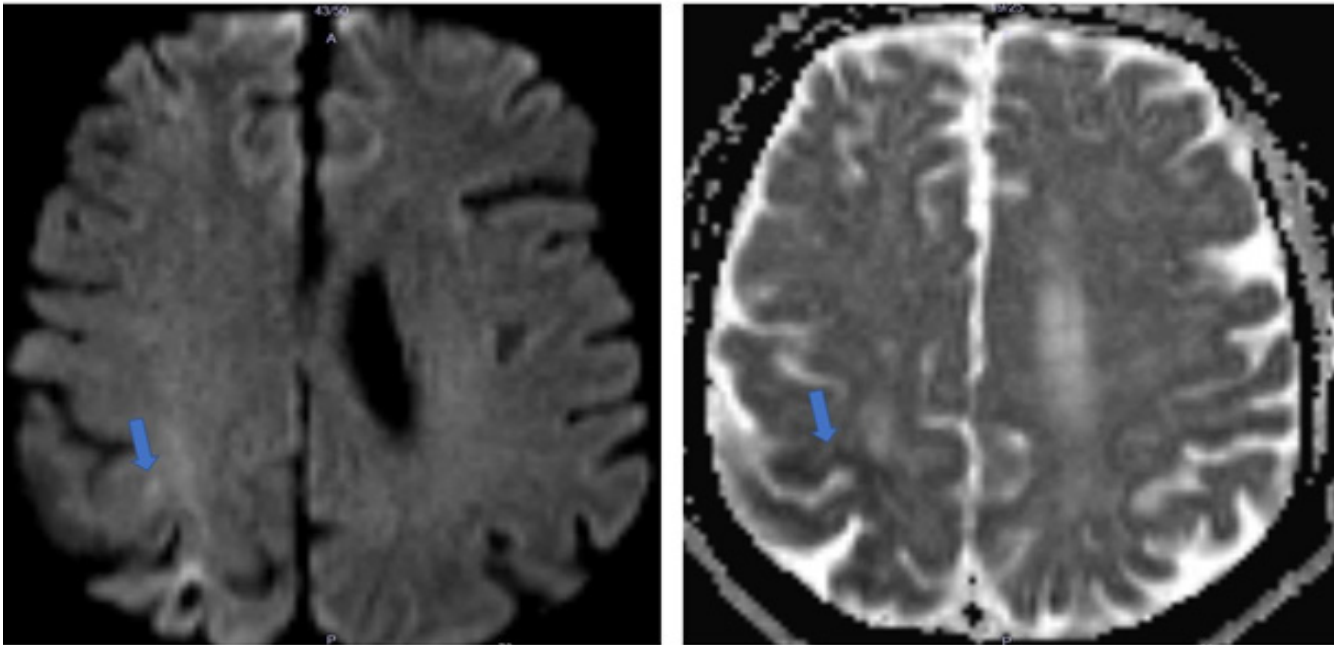


Figure-3: Subacute ischemia in the right parietal lobe posterior, especially in the subcortical area

CT angiography and interventional cerebral angiography performed for thrombus retraction were normal. Echocardiography showed no pathology such as patent foramen ovale and atrial septal defect (ASD), or any air images. The patient was diagnosed with

CAE and hyperbaric oxygen (HBO) was started for his treatment. The patient was given HBO treatment 3 h a day for 3 days. The patient was intubated due to worsening during the follow-ups and was taken to the intensive care unit.

The patient died on the 18th day of hospitalization in the intensive care unit due to sepsis associated with pneumonia.

DISCUSSION

CAE is a rare complication of dialysis catheter use. Although CAE often occurs after CVC insertion, it has also been reported to rarely occur as a result of CVC removal (Oyama, N., et al., 2012). Our patient developed air embolism due to catheter removal. Removal of CVC can result in CAE with paradoxical or retrograde mechanisms. There are many mechanisms for air to enter into the arterial system; air can enter the arterial bed directly, usually as a result of trauma to the chest or abdomen, or iatrogenically during medical procedures. Alternatively, venous emboli can enter the arterial system through intracardiac shunts (e.g. Patent foramen ovale or ASD) or intrapulmonary shunts (e.g. arteriovenous malformation/fistula). Rarely, venous air embolism can pass into the arterial system through extrafiltration by pulmonary capillaries (Khan, & Zaidi 2013; Oyama, et al., 2012; Schlimp, et al., 2014). In addition, another mechanism that causes CAE is the retrograde elevation of air in the vein due to the lower density that overcomes the opposite blood flow (Pandurangadu, et al. (2012). There must be a pressure gradient that supports the passage of air into the vein for air to enter the vein during the process of installing and removing CVC. This pressure gradient is greater when the patient is in an upright position, during inspiration, and in hypovolemic conditions, in which the central venous pressure is low. In these cases, greater amounts of air enter into the vein (Bartolini et al., 2015; Pandurangadu et al. 2012). In our case, the fact that the catheter was removed after dialysis (decreased central venous pressure compared with normal time) and in a sitting position, that is, under conditions where the pressure gradient is elevated, suggests that air entered the venous structure after CVC removal. The normal echocardiography also supports the idea that air caused CAE retrogradely.

Pathological effects of CAE occur with blockage of cerebral arteries. In addition, the foreign body response to air bubbles causes more damage to the ischemic tissue. Venous emboli can block the flow of blood in the last artery, causing cortical vascular zones and

infarctions. In addition, contact of the embolism with the endothelium of the blood-brain barrier leads to disruption of the barrier and cerebral edema, resulting in a decrease in blood flow and an inflammatory response (Bartolini et al., 2015; Khan et al. 2013). In our case, a generalized cerebral edema also presented over time due to CAE.

In CAE, as in our case, symptoms tend to occur suddenly. Neurological symptoms may occur with encephalopathy, coma, and seizures; or focal manifestations such as hemiparesis, hemianopia, dysarthria, aphasia, and hemihypesthesia, may also present (Chuang, et al. 2019; Pandurangadu, et al. 2012). Treatment of CAE is mainly supportive. The overall goal of treatment is to increase oxygenation and reduce complications. Administration of oxygen not only reduces hypoxemia but also reduces the size of air bubbles. Ventilation support may sometimes be required. Several studies have shown that HBO treatment is usually useful in cases of CAE, but accessing HBO treatment may be difficult (Bartolini et al., 2015; Oyama, et al., 2012).

In conclusion, we can minimize negative consequences with early diagnosis of CAE, which is associated with high mortality and morbidity, and by taking preventive measures during catheter removal. We recommend that physicians keep in mind that CAE may develop when removing CVC and take preventive measures.

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CONFLICT OF INTEREST

There is no conflict of interest between the authors.

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