**International Journal of Medical Science and Research**
www.medicaljournals.in
Online ISSN: 2664-8768, Print ISSN: 2664-875X
Received: 19-10-2021, Accepted: 04-11-2021, Published: 20-11-2021
Volume 3, Issue 2, 2021, Page No. 27-31

# Concomitant paradoxıcal air embolism, in intensıve care unit: A rare case report

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**Abstract**
**Introduction:** Paradoxical air embolism (PAE), as well as being a rare case, can often be fatal. It is mostly the consequence of an iatrogenic error related to various clinical, blood vessel-related procedures. There are still a significant number of deaths with a 1-year mortality of 21%, and almost 43% of intensive care unit (ICU) survivors had neurological sequels.
**Case:** 56-year old male patient who underwent regular dialysis treatment due to the diagnosis of chronic renal failure, gastrectomy was performed due to gastrointestinal stromal tumor. On the postoperative 7th day, he was admitted to the ICU due to respiratory distress and change in consciousness after intermittent hemodialysis. APACHE II score was 39, SOFA score was 14 and GCS was 8. He was intubated and followed up with invasive mechanical ventilation, predicting that he would not be able to protect the airway due to change in consciousness and type I respiratory failure. The catheter was quickly changed after a fracture was detected in the permanent hemodialysis catheter. Due to hypotension, noradrenaline treatment was started in addition to fluid resuscitation. There were air bubbles into the main pulmonary artery in his thorax computed tomography and acute hypodense areas consistent with air embolism in the brain magnetic resonance imaging. Patent foramen ovale as demonstrated by injection of agitated saline in his transthoracic echocardiography. So, the change in consciousness was associated with paradoxical (concomitant) air embolism. The patient was extubated on the 3rd day of intubation and was discharged to the ward on the 26th day of the ICU follow-up with GCS 15.
**Conclusion:** It is important to consider the diagnosis of air embolism in any patient with sudden hemodynamic or neurological deterioration in whom risk factors for air entry are present. Close follow-up in ICUs can provide satisfactory results in recovery.

**Keywords:** air embolism, concomitant, paradoxical embolism, intensive care unit, central venous catheter

## Introduction

Air embolism is the presence of air or gas in the vascular system and was described as early as 1885 by Senn [1]. Cohnheim reported the first case of paradoxical embolism with a cardiac septal defect in 1877 [2]. On the other hand, Zahn presented a cadaver with concomitant uterine vein thrombosis, multiple systemic embolism, and patent foramen ovale (PFO) in 1881 [3]. In 1885, the paradoxical embolism was described as a final term [4].

In paradoxical air embolism (PAE), air entering the venous circulation passes into the arterial system through an intracardiac shunt or pulmonary artery venous malformation/shunt [5]. Increased pulmonary artery pressure secondary to venous gas embolism may elevate right atrial pressure and may cause right-to-left flow through PFO or atrial septal defects (ASD) [6].

Paradoxical embolism is a rare clinical condition, representing less than 2% of arterial emboli [7]. The real prevalence of PAE is unknown, as embolic events are difficult to diagnose [8]. In general, PAE should be suspected in patients with arterial end-organ injuries. Cerebrovascular event, chest pain, migraine, cold extremity or mesenteric ischemia may occur [5].

PAE is a serious complication that can be observed as a result of interventions such as placement of arterial or venous catheters (eg central venous, hemodialysis, pulmonary artery etc.), contrast injection and thoracentesis [5, 9]. There is a risk of PAE in scuba divers and patients undergoing neurosurgery in a sitting position [10, 11, 12]. The incidence of cerebral air embolism associated systemic venous catheter- ranges from 15% to 54% [13, 14].

PAE should be considered when sudden-onset of respiratory distress, hemodynamic collapse or a neurological event occur in a patient with a known risk factor (eg intravenous catheter insertion, trauma). Computed tomography (CT) of the brain, and chest, magnetic resonance imaging (MRI) and / or echocardiography (transthoracic or transesophageal) can be helpful for diagnosis [9, 15, 16]. Appropriate supportive therapies in intensive care unit (ICU) and definitive treatment such as hyperbaric oxygen can be administered.

Short-term mortality is about 12% in retrospective studies. There are still a significant number of deaths with a 1-year mortality of 21%, and almost 43% of ICU survivors had neurological sequels [14]. We present a successful clinical report of a patient admitted to the ICU with the concomitant paradoxical air embolism due to central venous catheter fracture during intermittent hemodialysis.

## Case Presentatıon

56-year old male patient who underwent regular dialysis treatment for the diagnosis of chronic renal failure, gastrectomy was performed due to gastrointestinal stromal tumor. On the postoperative 7th day, he was admitted to the ICU due to

respiratory distress and change in consciousness after dialysis. In the medical history, he had hypertension, diabetes mellitus type 2 (DM), chronic kidney failure and gastrointestinal stromal tumor. His hemodialysis program was 3 times a week.

On physical examination, his vitals were as follows: heart rate 138 beats /minute, respiratory rate 32 /minute, and blood pressure 70/50 mmHg and body temperature (axillary measurement) 36.5 ˚C. Acute physiology and chronic health assessment (APACHE II) score was 39, sepsis related organ failure (SOFA) score was 14 and Glasgow coma scale (GCS) was 8. Oxygen saturation ($SaO_2$) was 64% under nasal oxygen therapy of 5 lt/min at ICU admission. The patient, who could not protect the airway due to change of consciousness and had type 1 hypoxemia, was intubated and followed up with invasive mechanical ventilation. The catheter was quickly changed after a fracture was detected in the permanent hemodialysis catheter. The hemodynamic status was instabile so, norepinephrine infusion was started at 0.1 μg/kg/min and rapidly increased to 1 μg/kg/min, in combination with dobutamine infusion at 5 μg/kg/min in addition to fluid resuscitation. Propofol sedation was initiated to prevent mechanical ventilator incompatibility. There were air bubbles in the main pulmonary artery in his thorax CT (Figure-1). Cortical ischemic changes at the vertex level of bilateral frontoparietal lobes were detected on brain diffusion-weighted MRI images performed 48 hours after the event (Figure-2). He was consulted with the neurology department. Low molecular weight heparin (LMWH) and acetylsalicylic acid treatment were administered. The dose of LMWH was adjusted by monitoring the antifactorXa level. A functional shunt was demonstrated by early passage of injected, aerated, saline microbubbles from the right-to-left atrial chambers and minimally reduced right ventricular function was detected in his transthoracic echocardiography (TTE). PFO was diagnosed and so, the change in consciousness was associated with arterial air embolism concomitantly.

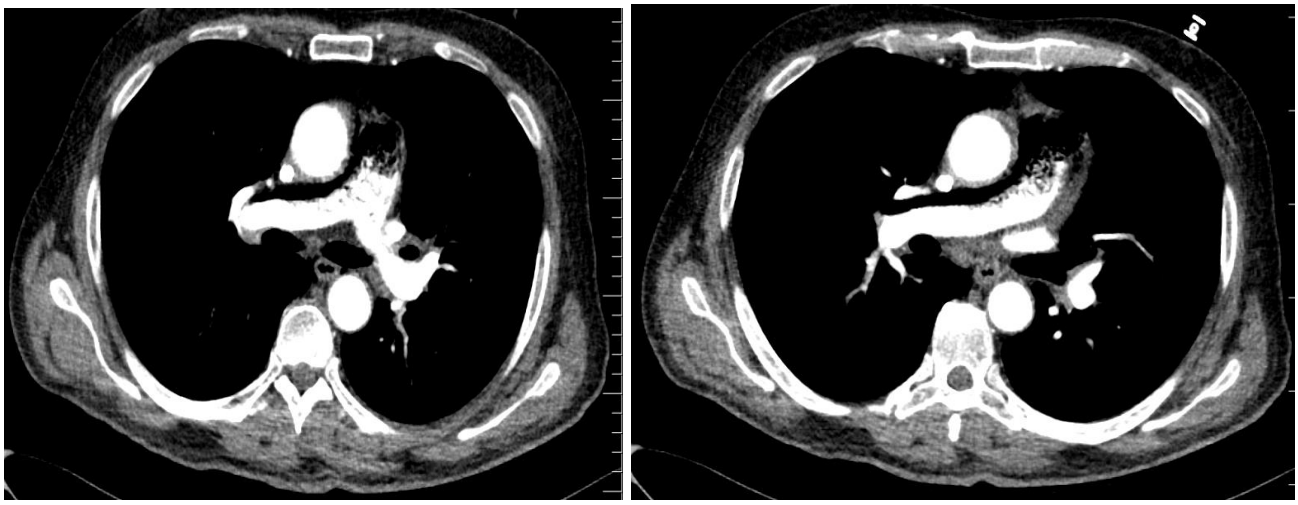
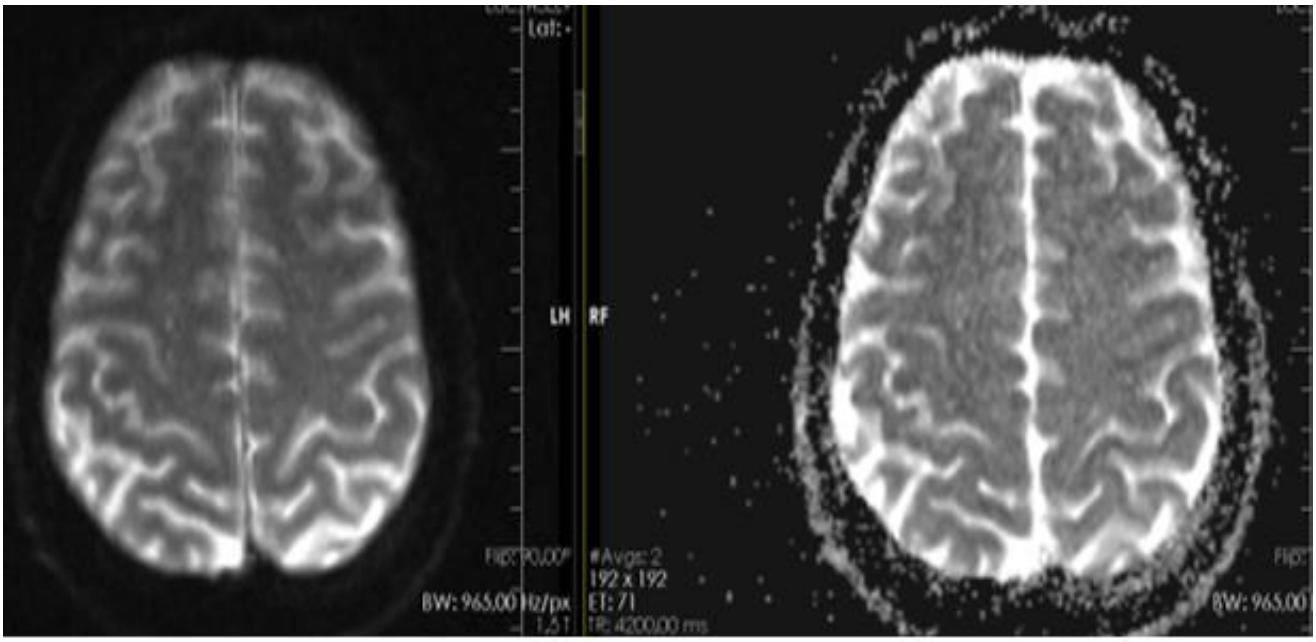


**Fig 1:** Computerized tomography of the thorax on intensive care unit admission



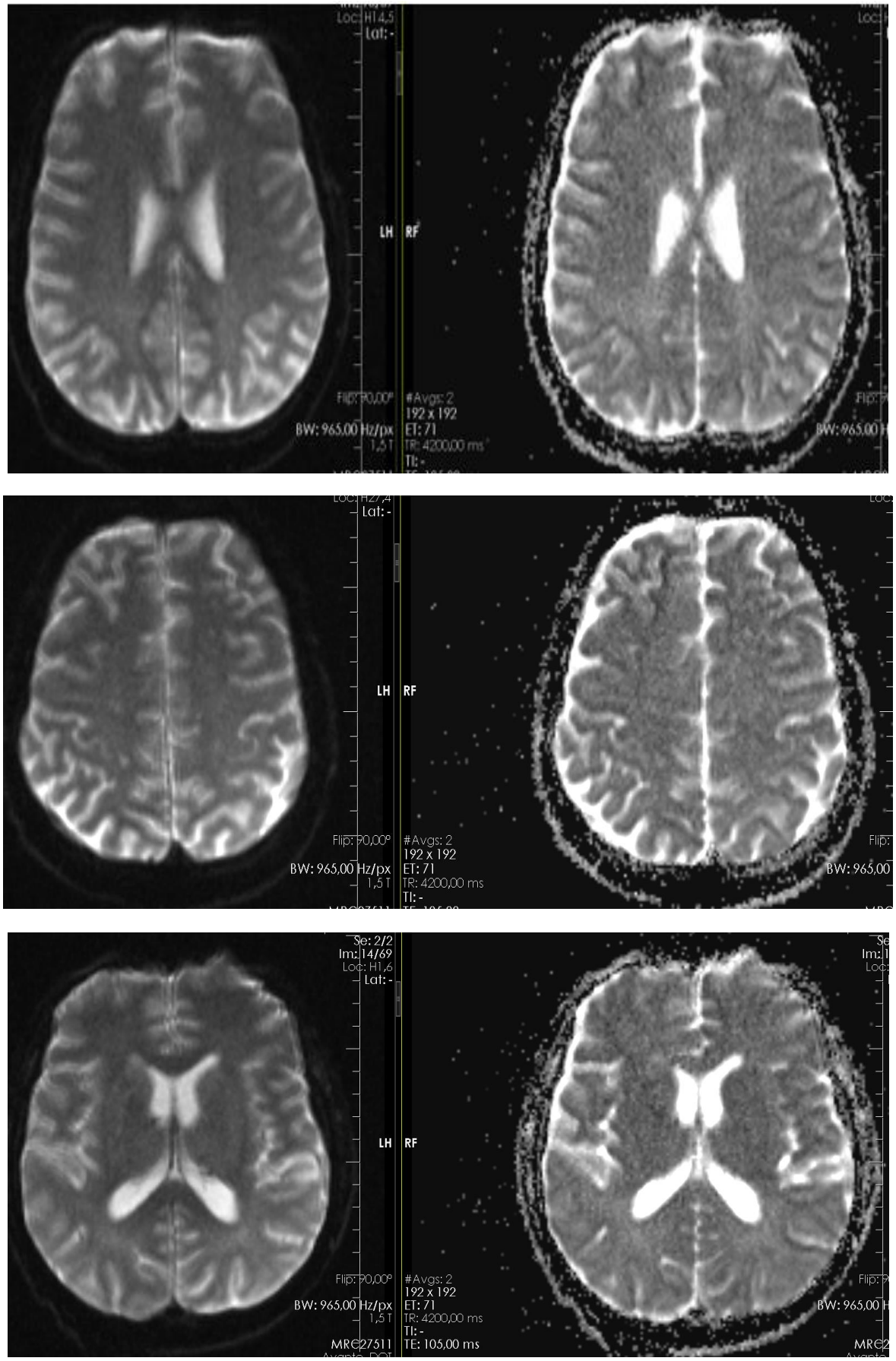



**Fig 2:** Diffusion-weighted magnetic resonance imagings of the brain and ADC map

Continuous renal replacement therapy was applied due to chronic renal failure and hemodynamic instability for 6 days. Gastroenterologic consultation for elevated liver enzymes revealed as ischemic hepatitis. In the follow-up, his liver function tests regressed. Multidrug-resistant klebsiella pneumoniae from the abdominal drain culture was identified. Antimicrobial therapy with intravenously meropenem for 18 days, teicoplanin for 19 days, fluconazole for 10 days, and amikacin for 5 days were administered.

The air bubbles in the main pulmonary artery were resorbed on control thorax CT (Figure-3). Propofol sedation was stopped daily and neurological examination was performed. The patient was extubated 3 days after intubation. On the 12th day of ICU admission, laparotomy was performed due to intestinal anastomotic leakage.

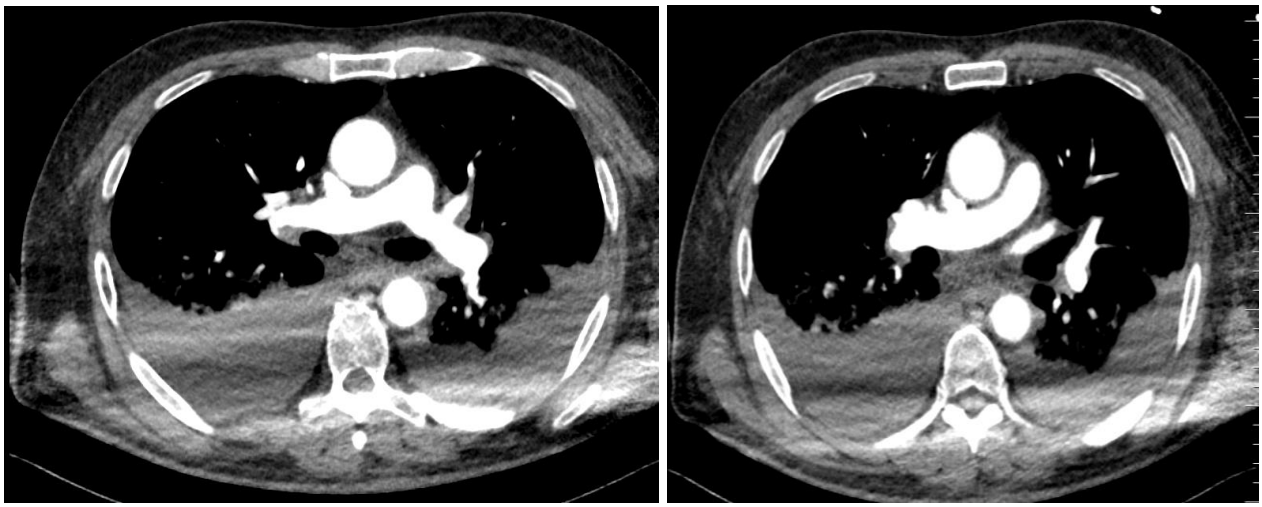


**Fig 3:** Control computerized tomography of the thorax 3rd day of intensive care unit follow-up

At that time, it was noted that patient has marked muscle wasting and diffuse weakness of his proximal left upper and lower extremities than right extremities. His sensory systems and reflexes were normal. The muscles of the neck and respiratory system were not weak, The patient was provided with a physiotherapist-assisted neuro-rehabilitation program consisting of conventional and respiratory physiotherapy. His muscle strength improved on the 40th day of hospitalization. He was discharged to the ward on the 26th day of the ICU follow-up with GCS 15.

## Dıscussıon

Paradoxical air embolism (PAE) occurs when an air or gas crosses an intracardiac defect into the systemic circulation (5). PAE is most commonly seen as a complication of central venous catheterization. However, causes include disconnected central venous catheters, air travel, ERCP, hemodialysis, trauma, laparoscopic air blowing, open heart surgery, lung biopsy, radiological procedures, obstetrics, head and neck surgery, and diving (11, 14). PAE has nonspecific signs and symptoms and to diagnose is difficult. For these reasons, its real incidence is unknown. The prevalence of PAE is difficult to measure because of the difficulty in proving embolic events. The incidence in patients with central venous catheters is between 0.2% to 1%. The incidence of cerebral air embolism associated with central venous catheter ranges from15% and 54%reported in previous studies [13].

PAE may be suspected when sudden shortness of breath, hypoxemia, chest pain and electrocardiography changes and neurological dysfunction occur in a patient with central venous catheter. It may occur during insertion, usage, or maintenance of the catheter, after catheter removal and disconnection [5, 15, 16]. Our patient had respiratory distress and change in consciousness with the usage of central venous catheter during intermittent hemodialysis. Therefore, air embolism was diagnosed in our patient with a central venous catheter.

Severe cardiovascular and pulmonary failure are often seen after PAE. An "air lock" occurs in the right heart and obstructs the outflow. Hence, acute right heart failure associated with hypo-oxygenation and cardiovascular depression is seen [16, 17]. Paradoxical (concomitant) air embolism occurs under cardiac defect conditions and via physiologic pulmonary arteriovenous shunts. Paradoxical embolism is uncommon and causes less than 2% of arterial embolism. PFO has been described in 30% of patients with paradoxical embolism, and several studies suggest that PFO is a risk factor for paradoxical embolism [5, 18, 19]. For this reason, the presence of PFO may be investigated during diagnosis [14, 17]. Our patient also had hemodynamic and respiratory instability. He had ischemic cerebral complications and PFO was detected at TTE. So, PAE was diagnosed.

CT is a valuable tool that shows the presence of air bubbles in both the cerebral circulation and pulmonary arteries, especially when performed immediately after the event [4, 13, 15, 16]. Bilateral and multiple acute ischemic infarctions can be observed in cerebral manifestations of PAE. Diffusion-weighted MRI of the brain is diagnostic [4, 5, 11, 13]. In our patient, there were air bubbles in the pulmonary circulation in his thorax CT and bilateral cortical ischemic changes consistent with air embolism in his brain diffusion-weighted MRI images.

Cerebral manifestations in PAE involves symptoms such as encephalopathy, coma, seizures, hemiparesis, hemianopia, hemi-hypoesthesia and aphasia. In diagnosis, brain CT may be normal. Infarct areas or multiple bilateral areas of decreased diffusion in cortical gray matter detected on brain MRI may be diagnostic [4,

[20, 21]. Cerebral manifestation was considered due to the change in consciousness observed in our patient after she was admitted to the ICU. It was confirmed by the presence of cerebral infarctions in the brain MRI performed 48 hours after the event. PFO which caused a right-to-left shunt that allowed air to enter the systemic arterial circulation was detected by TTE [13, 20].

Transesophageal echocardiography is the most sensitive method for the diagnosis of venous air embolism. Although TTE is a less sensitive method in diagnosis, it can be easily performed at the bedside in ICU. Intracardiac shunts causing PAE can be detected by both echocardiographic methods [20, 22]. Similar to the literature, in our patient PFO was diagnosed by agitated saline injection in TTE.

Treatments included for the management of air embolism: 1) stopping the air intake; 2) aspiration air from the right ventricle if there is a central venous catheter; 3) placing the patient head down in the left lateral position (Durant's maneuver) [23]; and 4) application of intensive care supporttive treatments, including oxygen support therapies and cardiopulmonary resuscitation [5, 20]. In patients with cerebral air embolism, hyperventilation may be preferred to maintain brain perfusion and metabolism [24]. We also stopped the hemodialysis procedure in our patient and closed the hemodialysis catheter. A central venous catheter was changed quickly. We intubated for respiratory control due to a change in consciousness. We started vasopressor and inotropic support to ensure hemodynamic stability. Close follow-up and appropriate supportive treatments were applied in the ICU. Hyperbaric oxygen therapy (HOT) is one of the treatment methods in patients with arterial gas embolism [5, 20]. Although HOT is recommended by expert opinions, there are no controlled clinical trial to support its efficacy and routine use [6, 20].

Mortality was associated with the presence of encephalopathy and advanced age in patients with cerebral air embolism. While the mortality rate was 23% in one trial [13], it was 93% in another study in which no therapeutic intervention was performed [20, 25]. As of 2006, there are no human randomized controlled trials for the treatment of cerebral air embolism.

## Conclusıon

In summary, we present a 56-year-old male patient who was admitted to ICU due to PAE that caused hypoxia, hemodynamic collaps and encephalopathy as a result of rupture of the central venous catheter during intermittent hemodialysis. This case demonstrates the use of CT and MRI to confirm the presence of air embolism in the venous and arterial circulation and the importance of echocardiography in the etiology of concomitant air embolism. Finally, it is important to consider the diagnosis of air embolism in any patient with sudden haemodynamical or neurological deterioration in whom risk factors for air entry are present. Close follow-up in ICUs can provide satisfactory results in recovery.

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