Respir Case Rep 2024;13(2):86-89 DOI: 10.5505/respircase.2024.46548

A Case of Pulmonary Arterial Air Embolism: A 10-hour Cruise

Pulmoner Arteryel Hava Emboli Olgusu: On Saatlik Seyir

Uğur Yüregir, Ege Güleç Balbay, Mustafa Boğan, Ali Can Kara

Abstract

Air embolism is a condition that often goes unnoticed, and although it is potentially life-threatening, it is rarely reported. The condition usually develops iatrogenically and resorbs spontaneously, although complications such as pulmonary edema and parenchymal destruction can develop. In our case, in lung tomography images taken 10 hours apart, the air seen in the pulmonary artery and right atrium disappeared, while areas of increasing consolidation were identified in the left lung. It is thought that air embolisms may be a cause of pneumonia etiologies in patients undergoing intravenous (IV) procedures, while other studies have referred to the condition, considered pneumonia, as an inflammatory process that develops due to the destruction following an air embolism. There is a need to investigate the frequency of complications and pneumonia in cases that develop air embolisms following IV procedures.

Keywords: *Air embolism, iatrogenic air embolism, arterial air embolism, intravenous access complications, etiology of pneumonia.*

Öz

Hava embolisi çoğunlukla fark edilmeyen bu nedenle nadir olarak bildirilen ancak hayati tehlikesi olan bir durumdur. Genellikle iyatrojenik gelişir ve kendiliğinden rezorbe olur. Hava embolilerinde akciğer ödemi veya parankimal destrüksiyon gibi komplikasyonlar gelişebilmektedir. Olgumuzda on saat ara ile çekilen akciğer tomografisi görüntülerine göre pulmoner arter ve sağ atriumda görülen hava kaybolurken, sol akciğerde artan konsolidasyon alanları mevcuttu. Takibinde enfektif kliniğinin de gelişmesi ödem ve destrüksiyonun da pnömoni açısından kolaylaştırıcı neden olabileceğini düşündürmektedir. Intravenöz (IV) işlem yapılan hastalarda pnömoni etyolojisinde hava embolilerinin bir neden olabileceğini düşünülmektedir. Bir başka açıdan da pnömoni olarak değerlendirilen tablonun hava embolisi sonrası dekstüriksiyona bağlı gelişen enflamatuar süreç olarak da düşünülebilir. IV işlem yapıldıktan sonra hava embolisi gelişen olgular üzerinde ortaya çıkan komplikasyonlar ve pnömonilerin görülme sıklığının araştırılmasına ihtiyaç vardır.

Anahtar Kelimeler: *Hava embolisi, iatrojenik emboli, arteriyel hava embolisi, ıntravenöz damar yolu komplikasyonları, pnömoni etyolojisi.*

Düzce University Faculty of Medicine, Department of Pulmonary Diseases, Düzce, Türkiye

Düzce Üniversitesi Tıp Fakültesi, Göğüs Hastalıkları Ana Bilim Dalı, Düzce

Submitted (Başvuru tarihi): 12.12.2023 ***Accepted (Kabul tarihi):*** 27.02.2024

Correspondence (İletişim): Uğur Yüregir, Düzce University Faculty of Medicine, Department of Pulmonary Diseases, Düzce, Türkiye

e-mail: uguryuregir@gmail.com

Air embolisms are rare but can be life-threatening, and usually resorb spontaneously and unnoticed (1). Air entering the body via the venous route is usually filtered in the lungs if there is no shunt between right and left, but if the amount is excessive, it passes through the lungs and enters the left heart, and potentially other organs, via systemic circulation. The danger to life increases during this period (1,2). The first animal studies of air embolisms revealed that air bubbles increased vascular permeability (3).

It is thought that air embolisms in the pulmonary artery, which are considered high risk in terms of mortality in literature and are usually encountered incidentally, may be responsible for the etiology of unexplained deaths in emergency room patients. .

CASE

The case presented here relates to an 89-year-old female patient with diabetes mellitus, hypertension, chronic renal failure and Alzheimer's who had been immobile for 5 years due to Alzheimer's and who ate her meals with the support of her caregiver. The patient was started on valproic acid 2x500 mg by a neurologist following an epileptic seizure, and was subsequently admitted to the emergency department due to seizures and impaired consciousness. Upon admittance, the patient's vitals were: Fever: 36.5°C, pulse: 77/min, blood pressure: 120/70 mmHg and oxygen saturation in room air: 88 %. Nasal oxygen (4 L/min) support was administered, intravenous access was established, blood was taken, and intravenous treatment was started. The laboratory parameters were measured as hemoglobin: 8.46 g/dL, leukocyte: 10,400 /mm^3, thrombocyte: 129,000 /mm^3, neutrophil: 8630 /mm^3, CRP: 11.31 mg/dL, creatine: 1.7 mg/dL, pH in arterial blood gas: 7.47, PCO_2: 29.3 mmHg, SO_2: 99.2 %, PO_2: 111 mmHg and HCO_3:23.1 mEql/L. No additional pathologies were detected in other laboratory parameters. The patient underwent cranial imaging, and the neurologist confirmed that no new pathologies had developed, and suggested that the current medications should be continued. Epileptic seizures have been stated to occur secondary to infections. The patient was referred to a cardiologist following tachycardia and a new diagnosis of atrial fibrillation, and was prescribed only acetylsalicylic acid due to the risk of bleeding. It is recommended to use it as. The patient was subsequently evaluated by the infectious disease and chest disease departments, no antibiotic treatment was recommended in the absence of infective focus. Thorax computerized tomography (CT) revealed an appearance consistent with air density in the pulmonary artery and right atrium (Figure 1). Upon the identification of an air embolism in the patient, she was placed in the left lateral decubitus and Trendelenburg position and ventilated with 100% FiO_2, and based on the high mortality rate risk, it was recommended that she be followed up in intensive care. A thorax CT taken approximately 10 hours later in the intensive care unit revealed that the majority of the areas identified as air embolisms had been resorbed, while minimal areas were noted in and around the right atrium, as well as newly developed and increasing bilateral and peripheral consolidated areas (Figure 2). On the second day of follow-up, increases in CRP and in the amount of sputum and cough were noted, despite non-specific antibiotic therapy, suggesting the development of pneumonia. Upon the growth of Klebsiella Pneumoniae in the culture of a deep tracheal aspiration sample taken from the patient, the patient's moxifloxacin treatment was changed for 3 days, after which Levofloxacin was administered for 5 days, followed by piperacillin-tazobactam for 11 days and meropenem for 11 days, considering the lack of clinical response. The patient was discharged after her clinical and laboratory values had remained stable for 8 days. No additional pathologies were detected in any of the other control respiratory tract cultures.

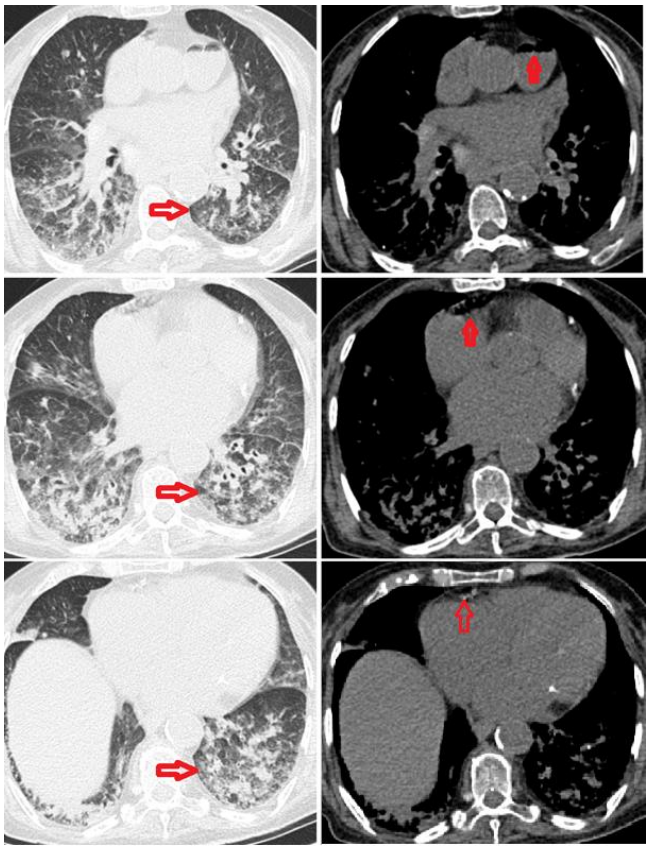

Figure 1: A slight consolidation can be seen in the left lung parenchyma in the first column, and air densities in the pulmonary artery and right atrium in the second column (08.04.2023, time: 01:47)

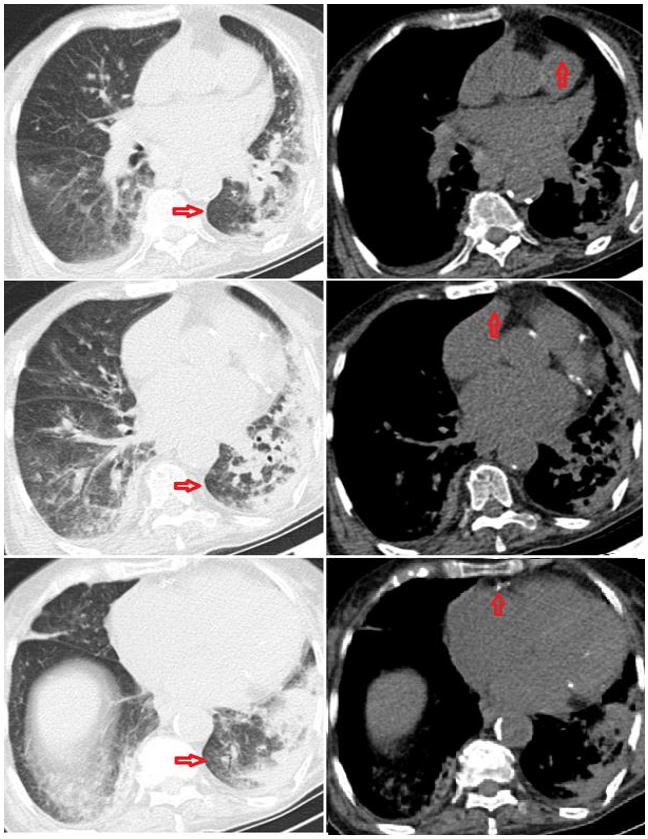

Figure 2: In the first column, it was observed that the consolidation in the left lung parenchyma had decreased, while the appearance of air densities in the pulmonary artery and right atrium decreased in the second column (08.04.2023, time:12:07)

DISCUSSION

Pulmonary air embolism is a rare but life-threatening condition that often goes unnoticed and is often iatrogenic (4,5). Complications can be significantly improved by rapid diagnosis and appropriate treatment (4).

The mechanism of lung damage resulting from a pulmonary air embolism involves several interrelated processes:

-Vascular Obstruction: When air enters the pulmonary circulation, it can travel through blood vessels and obstruct the flow of blood. Such obstructions can occur in small to larger pulmonary arteries, impeding blood supply to portions of the lung tissue.

-Ischemia and Infarction: A blockage of a pulmonary artery or its branches can lead to ischemia (lack of blood supply) in the affected lung tissue. A significantly compromised blood supply can result in localized lung tissue death, known as a pulmonary infarction, which can cause areas of the lung to become non-functional or scarred.

-Inflammatory Response: The presence of air within the blood vessels triggers an inflammatory response in the surrounding tissues. The immune system may subsequently react to the foreign substance (air) within the vasculature, leading to an inflammatory cascade. Such inflammatory responses can contribute to further lung tissue damage.

-Pulmonary Hypertension: Severe or repeated emboli can lead to an increase in pressure within the pulmonary arteries – a condition known as pulmonary hypertension – and this increased pressure can put strain on the right side of the heart, leading to right heart failure and other complications.

-Acute Respiratory Distress Syndrome (ARDS): In cases where blood flow is substantially compromised or a severe inflammatory response occurs, ARDS can develop, being a severe lung condition characterized by widespread inflammation in the lungs that leads to fluid accumulation and impaired oxygen exchange with the potential to lead to severe respiratory failure.

The severity and extent of damage depend on various factors, including the size and number of emboli, the patient's overall health, the promptness of treatment, and any underlying lung or cardiovascular conditions. Immediate medical interventions can mitigate the extent of damage and improve outcomes (6-9).

Venous air embolisms are more common than arterial air embolisms, and can occur during venous infusions or catheter manipulation. Arterial air embolisms, on the other hand, can occur as a complication of lung biopsy, vascular catheterization or cardiopulmonary bypass, or from the venous system to the arterial system beyond the lung (2). Oxygen support is recommended for the treatment of both conditions, although it is recommended to use the right lateral decubitus position for arterial embolisms, and the left lateral decubitus and/or Trendelenburg position for venous embolisms (1,4,10,11). Hyperbaric oxygen therapy is the optimum treatment approach, reducing the size of the air embolism by facilitating gas reabsorption, while also improving tissue oxygenation and reducing ischemic reperfusion injury, although cardiovascular and respiratory supportive treatments should also be provided (1,12). It is thought that the risk to life is attributable to the large amount of air flowing to the end organs via the arterial system, which destroys the lung parenchyma before entering the venous system, the arterial system and the end organs. It is thought that the extent of parenchymal destruction in air embolisms may parallel the amount of air, revealing the importance of parenchymal destruction in terms of vital risk (2). Other complications associated with air embolisms that are often overlooked include post-emboli pulmonary edema (1,2).

Many risk factors are associated with pulmonary air embolism, but in the presented case, the only risk factor was the patient's receipt of intravenous therapy. Following the air embolism diagnosis, the patient was placed on oxygen therapy and was followed up in the left lateral decubitus and Trendelenburg position. A CT scan taken 10 hours later revealed that the air had been largely resorbed, although it was uncertain whether the resorption had

been spontaneous or due to the treatment given. As suggested in literature, when an air embolism is detected, it is important to place the patient on oxygen and position therapy, regardless of the size (1,4,10-12). In our case, the patient developed pneumonia on the third day of follow-up, and it was thought that the pulmonary edema and destruction in the lung parenchyma may have been responsible, or that the pneumonia was a complication of the air embolism. The patient was duly started on treatment for pneumonia. It has been suggested that areas considered pneumonic consolidated could actually be considered destruction, and that the high infective parameters recorded may be associated with the air in the parenchyma initiating and continuing the inflammatory process.

To reduce the development of such iatrogenic cases, appropriate training should be provided to healthcare providers in the use of appropriate techniques and equipment, while the close monitoring of patients during procedures can help reduce all other air embolism risks (2,13).

CONFLICTS OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

Concept - E.G.B., U.Y., M.B., A.C.K.; Planning and Design - E.G.B., U.Y., M.B., A.C.K.; Supervision - E.G.B., U.Y., M.B., A.C.K.; Funding - E.G.B., U.Y.; Materials - U.Y., M.B., A.C.K.; Data Collection and/or Processing - U.Y., M.B., E.G.B., A.C.K.; Analysis and/or Interpretation - U.Y., E.G.B., A.C.K.; Literature Review - E.G.B., U.Y.; Writing - E.G.B., U.Y.; Critical Review - E.G.B., U.Y., M.B., A.C.K.

REFERENCES

1. Mirski MA, Lele AV, Fitzsimmons L, Toung TJ. Diagnosis and treatment of vascular air embolism. Anesthesiology 2007; 106:164-77. [CrossRef]
2. Marsh PL, Moore EE, Moore HB, Bunch CM, Aboukhaled M, Condon SM, et al. Iatrogenic air embolism: pathoanatomy, thromboinflammation, endotheliopathy, and therapies. Front Immunol 2023; 14:1230049. [CrossRef]
3. Takeoka M, Sakai A, Ueda G, Ge RL, Panos RJ, Taniguchi S. Influence of hypoxia and pulmonary air embolism on lung injury in perfused rat lungs. Respiration 1996; 63:346-51. [CrossRef]
4. Shah J, Jiwa N, Mamdani N, Hill D. Venous and arterial air embolism: a rare phenomenon with fatal consequences. BMJ Case Rep 2016; 2016:bcr2016217550. [CrossRef]
5. Austin LS, VanBeek C, Williams GR. Venous air embolism: an under-recognized and potentially catastrophic complication in orthopaedic surgery. J Shoulder Elbow Surg 2013; 22:1449-54. [CrossRef]
6. Ishak BA, Seleny FL, Noah ZL. Venous air embolism, a possible cause of acute pulmonary edema. Anesthesiology 1976; 45:453-5. [CrossRef]
7. Holmes CM, Baker AB. Pulmonary edema associated with severe venous air embolism. Anaesth Intensive Care 1985; 13:435-7. [CrossRef]
8. Lam KK, Hutchinson RC, Gin T. Severe pulmonary oedema after venous air embolism. Can J Anaesth 1993; 40:964-7. [CrossRef]
9. Smelt WL, Baerts WD, de Langhe JJ, Booij LH. Pulmonary edema following air embolism. Acta Anaesthesiol Belg 1987; 38:201-5.
10. Malik N, Claus PL, Illman JE, Kligerman SJ, Moynagh MR, Levin DL, et al. Air embolism: diagnosis and management. Future Cardiol 2017;13:365-78. [CrossRef]
11. Lanfranco J, Romero Legro I, Freire AX, Nearing K, Ratnakant S. Pulmonary air embolism: an infrequent complication in the radiology suite. Am J Case Rep 2017; 18:80-4. [CrossRef]
12. Jørgensen TB, Sørensen AM, Jansen EC. Iatrogenic systemic air embolism treated with hyperbaric oxygen therapy. Acta Anaesthesiol Scand 2008; 52:566-8. [CrossRef]
13. Ahn Y, Lee SM, Kim HJ, Choe J, Oh SY, Do KH, et al. Air embolism in CT-guided transthoracic needle biopsy: emphasis on pulmonary vein injury. Eur Radiol 2022; 32:6800-11. [CrossRef]