

Right Atrial Appendage Clot in Atrial Fibrillation Complicated by Pulmonary Thromboembolism: A Case Report

Hedieh Alimi^{1,*}, and Asal Yadollahi¹

¹ Qaem Cardiovascular Medical and Research Center, Mashhad University of Medical Sciences, Mashhad, Iran

* **Corresponding author:** Hedieh Alimi, Department of Cardiology, Cardiovascular Research Center, Qaem Hospital, Faculty of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran. Email: Alimih@mums.ac.ir

Received 2018 February 15; Accepted 2018 October 29.

Abstract

Introduction: Nonvalvular atrial fibrillation (AF) can affect both right and left atria. Left atrial thrombus is more common in patients with nonvalvular AF. The presence of a large right atrial thrombus is a rare condition, which is seldom demonstrated as an etiology of pulmonary embolism, especially without any deep vein thrombosis at the presence of only atrial fibrillation, as was observed in this case. Right atrial thrombus is an underdiagnosed entity with a high mortality and morbidity rate. The best management modality has not yet been documented, and it sometimes resolved by anticoagulant therapy alone or needed surgical removal.

Case Presentation: Here, we report a case of pulmonary thromboembolism with the complaints of coughing and blood-stained sputum without clinical or sonographic signs of deep vein thrombosis. Permanent AF rhythm and a large clot in RAA were detected as the sources for pulmonary embolism. We started anticoagulant therapy and the clot got smaller using a transesophageal echocardiogram as a diagnostic tool.

Conclusions: Right atrial appendage (RAA) assessment should be considered in patients with recurrent or acute pulmonary embolism and AF rhythm in the absence of clinical and sonographic signs of deep vein thrombosis.

Keywords: Atrial appendage, Clot, Pulmonary embolism, Transesophageal echocardiography

1. Introduction

Nonvalvular atrial fibrillation (AF) in contrast to valvular AF could affect equally both atria. Both left and right atrial appendage function and emptying velocities are impaired in atrial fibrillation. In patients with AF, spontaneous echocardiographic contrast and thrombus formation may occur in left atrial appendage (LAA), and it may happen in right atrial appendage (RAA) with less frequency (1, 2). Here, we present a case of acute pulmonary embolism as a complication of right atrium appendage thrombosis in a patient with persistent AF. The relationship between atrial fibrillation and pulmonary embolism is unknown, whether the atrial fibrillation is the chicken or the egg; however, in this case, the priority of atrial fibrillation before embolism is the initial diagnosis (3, 4). Large right atrial thrombus was an unexpected finding as a source for pulmonary embolism without any deep vein thrombosis and associated left atrial thrombus in the presence of atrial fibrillation. Therefore, in patients with chronic nonvalvular AF, the RAA and LAA assessment should be considered which could be a source for recurrent or acute pulmonary thromboembolism (1).

2. Case Presentation

A 74-year-old female with no specific past medical history presented to the Emergency Department with the complaints of coughing and blood-stained sputum from three days ago. She did not use any kind of medicine; however, she had pleuritic chest pain and

mild dyspnea. Her presenting blood pressure, respiratory rate, and heart rate in the emergency room were 120/70 mmHg, 20 breaths/min, and 114 beats/min, respectively. There were no symptoms of volume overload, such as dyspnea on exertion, leg edema, or other symptoms of congestive heart failure exacerbation. An electrocardiogram showed atrial fibrillation (Figure 1).

Furthermore, a bedside transthoracic echocardiogram revealed mildly reduced left ventricle ejection fraction (EF) (EF=45%) with abnormal septal motion and no regional wall motion abnormalities. In addition, the right ventricle size and function were normal; however, the left and right atria were both severely enlarged. Moreover, mild to moderate aortic regurgitation, moderate functional mitral regurgitation, and moderate tricuspid regurgitation were detected in this case. A suspected mass was also found in the right atrium (RA) from a subxiphoid view.

Initial laboratory data showed hypokalemia of 2.6 meq/L (normal value 3.5-5.5 meq/L), glomerular filtration rate of 45 mL/min/1.73 m², normal liver function tests, normal complete blood count, and a serum troponin of <0.01 ng/mL (normal value <0.04 ng/mL).

According to the presence of a suspected mass in RA and acute onset non-massive hemoptysis and pleuritic chest pain, one possible diagnosis was pulmonary thromboembolism. Additionally, lung computed tomography angiography revealed filling defects suggesting pulmonary embolism in the left superior and the right inferior lobar branches of pulmonary circulation. Other findings were patchy

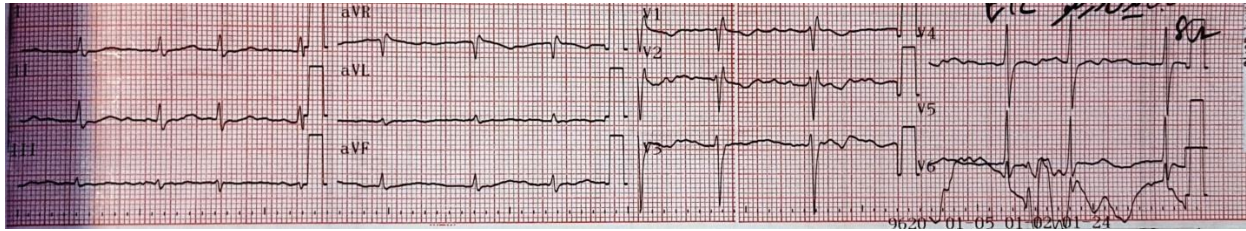


Figure 1. Electrocardiogram showing atrial fibrillation rhythm.

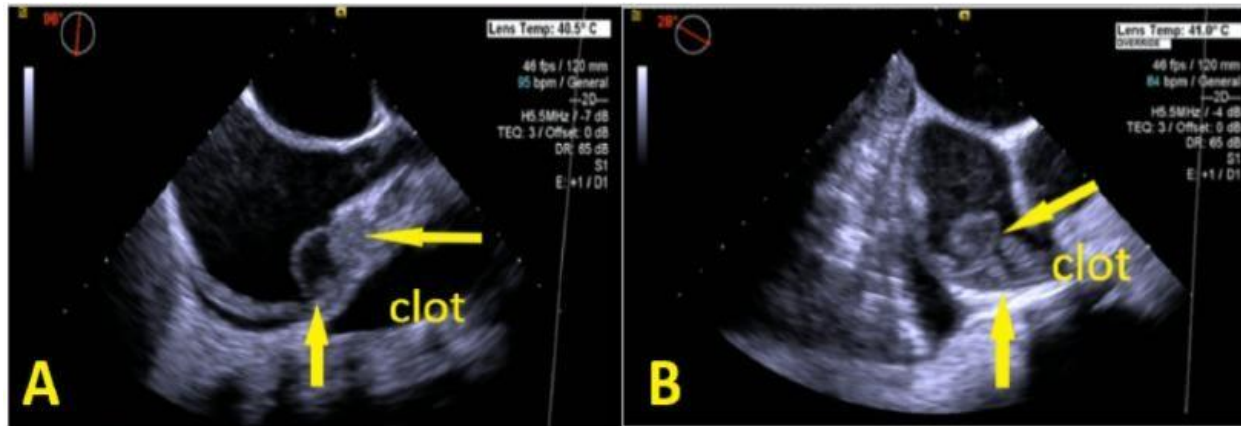


Figure 2. Intransepophageal echocardiography, a large size multilobulated mass (A) with central echolucency (B). A fresh clot is most likely observed in right atrial appendage in two views. Maximum size of the clot is 2.3*1.8 cm marked by yellow arrows.

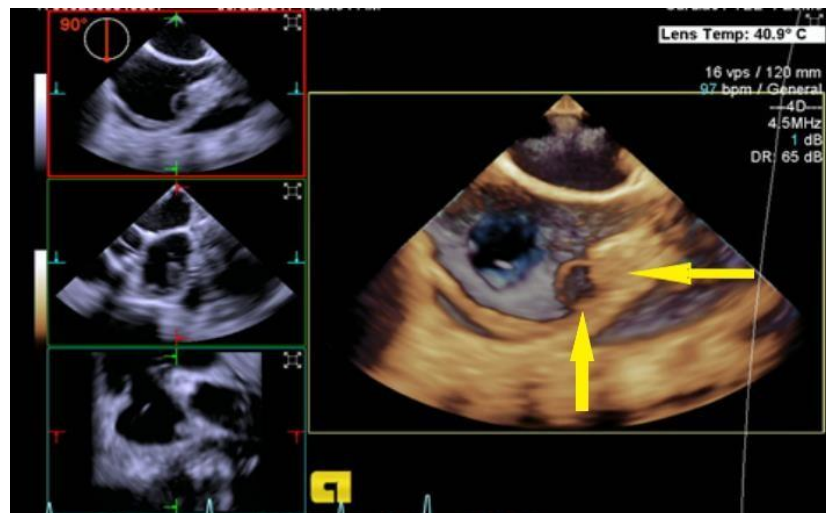


Figure 3. Mass location confirmed by a 3-dimensional study marked by yellow arrows.

consolidation in the same areas, which could be explained by pulmonary infarction in these regions.

Both AF rhythm and pulmonary thromboembolism were the indications for anticoagulant therapy. Therefore, the intravenous heparin therapy was started, hemoptysis was not repeated and hypokalemia was corrected. The patient had no risk factor and no clinical sign of deep vein thrombosis; however, Venous Doppler sonography was done, and no vein thrombosis was detected in lower limbs.

Additionally, a transepophageal echocardiogram (TEE) was performed to evaluate the RA suspicious mass in more details. There was no left atrium (LA) or left atrial appendage (LAA) clot but a large size multilobulated mass with central echolucency. In addition, a fresh clot was most likely observed in RAA with a maximum size of 2.3*1.8 cm whose location was confirmed by three dimensional and biplane views (figures 2 and 3).

During hospitalization, the patient remained in

permanent AF. Warfarin was started in association with heparin, and the patient was discharged after 10 days. The TEE was repeated 14 days later and revealed that the clot got smaller in size (i.e., 1.9*1.7 cm). Warfarin was continued considering the target international normalized ratio range between 2 and 3.

3. Discussion

In a transesophageal echocardiographic study, the prevalence rate of the patients with atrial arrhythmia and right atrial thrombus was reported to be less than 1% as a rare finding; moreover, spontaneous echo contrast had a prevalence rate of 14% (5). In a study conducted by De Divitiis on patients with nonvalvular AF, spontaneous echo contrast was reported to be 66%, 57%, and 100% in the LA, RA, and patients who had RAA thrombi, respectively. Nonvalvular AF patients in comparison with those in sinus rhythm had larger RA chamber, right atrial appendage area, and lower tricuspid annular excursion, as well as peak emptying velocities, and right atrium ejection fraction. Equivalent differences were found for the LA appendage.

The presence of the RAA thrombi was associated with a larger RA area, lower RAA ejection fraction, and emptying velocities, compared to AF patients without thrombi (6). Subramanian showed that patients with AF rhythm had the lower right and left atrial appendage ejection velocities than patients in sinus rhythm. He revealed that the prevalence of the thrombus in the right atrium appendage was lower than that on the left. The larger RAA width and lack of anatomic remodeling may partially explain the substantially lower prevalence of RAA thrombus found among patients with AF (7).

The incidence of perfusion defects in pulmonary scintigraphy as a marker of silent pulmonary embolism is predominantly higher in AF patients with right atrium spontaneous contrast, compared to AF patients without it. This indicates that the right atrial spontaneous echo contrast in patients with AF may be a predictive factor for being at high risks for pulmonary embolism (8).

In a study performed by Ogren on 23796 autopsies, the prevalence rate of right atrial thrombosis was the same as the left atrium thrombosis. Therefore, in all patients with pulmonary embolism, atrial thrombi should be mentioned as a possible source for embolism. This is more prevalent in patients with myocardial infarction or atrial fibrillation in the absence of confirmed deep vein thrombosis (9).

6. Conclusion

This case underlines the importance of considering a possible right atrial appendage thrombosis in patients with AF rhythm. It may affect

our management in different clinical situations, such as the assessment of a patient with pulmonary embolism, performing cardioversion or atrium appendage device closure, or starting anticoagulant therapy. It is important to remember that although RAA thrombi are less frequent than LAA thrombi, it may become large enough to be a source for clinically significant pulmonary embolism. Therefore, it is recommended to perform TEE for the evaluation of right atrium appendage in patients with recurrent or acute pulmonary embolism and AF rhythm in the absence of clinical and sonographic signs of deep vein thrombosis.

Acknowledgments

We thank Mashhad University of Medical Science for its support in order to get access to the data, which was required for preparing this study.

Conflicts of interest

The authors declare no biomedical, financial, or potential conflicts of interests.

References

1. Bilge M, Eryonucu B, Güler N, Erkoç R. Right atrial appendage function in patients with chronic nonvalvular atrial fibrillation. *Jpn Heart J*. 2000;**41**(4):451-62. doi: [10.1536/jhj.41.451](https://doi.org/10.1536/jhj.41.451). [PubMed: [11041096](https://pubmed.ncbi.nlm.nih.gov/11041096/)].
2. Benjamin MM, Afzal A, Chamogeorgakis T, Feghali GA. Right atrial thrombus and its causes, complications and therapy. *Proc (Bayl Univ Med Cent)*. 2017;**30**(1):54-6. doi: [10.1080/08998280.2017.11929526](https://doi.org/10.1080/08998280.2017.11929526). [PubMed: [28127133](https://pubmed.ncbi.nlm.nih.gov/28127133/)].
3. Flegel KM. When atrial fibrillation occurs with pulmonary embolism, is it the chicken or the egg? *CMAJ*. 1999;**160**(8):1181-2. [PubMed: [10234351](https://pubmed.ncbi.nlm.nih.gov/10234351/)].
4. Lohani S, Tachamo N, Timilsina B, Nazir S. Pulmonary embolism and atrial fibrillation: a complicated Relationship. *Int J Case Rep Images*. 2017;**8**(6):376-9. doi: [10.5348/ijcri-201751-cr-10790](https://doi.org/10.5348/ijcri-201751-cr-10790).
5. Bashir M, Asher CR, Garcia MJ, Abdalla I, Jasper SE, Murray RD, et al. Right atrial spontaneous echo contrast and thrombi in atrial fibrillation: a transesophageal echocardiography study. *J Am Soc Echocardiogr*. 2001;**14**(2):122-7. doi: [10.1067/mje.2001.108668](https://doi.org/10.1067/mje.2001.108668). [PubMed: [11174446](https://pubmed.ncbi.nlm.nih.gov/11174446/)].
6. deDivitiis M, Omran H, Rabahieh R, Rang B, Illien S, Schimpf R, et al. Right atrial appendage thrombosis in atrial fibrillation: its frequency and its clinical predictors. *Am J Cardiol*. 1999;**84**(9):1023-8. doi: [10.1016/s0002-9149\(99\)00492-0](https://doi.org/10.1016/s0002-9149(99)00492-0). [PubMed: [10569657](https://pubmed.ncbi.nlm.nih.gov/10569657/)].
7. Subramanian B, Riley MF, Panzica PJ, Manning WJ. Transesophageal echocardiographic assessment of right atrial appendage anatomy and function: comparison with the left atrial appendage and implications for local thrombus formation. *J Am Soc Echocardiogr*. 2006;**19**(4):429-33. doi: [10.1016/j.echo.2005.10.013](https://doi.org/10.1016/j.echo.2005.10.013). [PubMed: [16581482](https://pubmed.ncbi.nlm.nih.gov/16581482/)].
8. Yasuoka Y, Naito J, Hirooka K, Chin W, Miyatake K, Kusuoka H, et al. Right atrial spontaneous echo contrast indicates a high incidence of perfusion defects in pulmonary scintigraphy in patients with atrial fibrillation. *Heart Vessels*. 2009;**24**(1):32-6. doi: [10.1007/s00380-008-1084-3](https://doi.org/10.1007/s00380-008-1084-3). [PubMed: [19165566](https://pubmed.ncbi.nlm.nih.gov/19165566/)].
9. Ogren M, Bergqvist D, Eriksson H, Lindblad B, Sternby NH. Prevalence and risk of pulmonary embolism in patients with intracardiac thrombosis: a population-based study of 23796 consecutive autopsies. *Eur Heart J*. 2005;**26**(11):1108-14. doi: [10.1093/eurheartj/ehi130](https://doi.org/10.1093/eurheartj/ehi130).