

Phrenic Nerve Injury Post Catheter Ablation for Atrial Fibrillation

Payal Sen, MD¹
Uddalak Majumdar, MD²
Ali Imran Saeed, MD¹

¹University of New Mexico
Albuquerque, NM USA

²Cleveland Clinic Foundation
Cleveland, Ohio USA

Abstract

Objective: Phrenic nerve injury (PNI) is a complication of catheter ablation treatment of atrial fibrillation (AF). This condition can mimic that of comorbid conditions like congestive heart failure (CHF) and chronic obstructive pulmonary disease (COPD).

Case details: A 77-year-old woman with past medical history of heart failure with preserved ejection fraction and mild COPD, presented with dyspnea for 8 days. One week ago, she had undergone radiofrequency catheter ablation for persistent symptomatic AF. After the ablation, she reported dyspnea during PCP and pulmonary office visits and was given increasing doses of diuretics and inhalers since her symptoms were attributed to acute exacerbation of heart failure in the setting of COPD. However, a chest x-ray showed elevation of the right hemidiaphragm, and she had a positive sniff test. She was thus diagnosed with right sided phrenic nerve palsy and was treated with oxygen therapy.

Discussion: Phrenic nerve injury can be diagnosed via clinical exam, chest x-ray and sniff test. A sniff Test which shows paradoxical elevation of the paralyzed hemidiaphragm with inspiration, compared with the rapid descent of the normal hemidiaphragm.

Conclusion: Phrenic nerve palsy is a complication which occurs in 6.6 percent of cases, post catheter ablation procedure for atrial fibrillation. This condition can mimic pulmonary conditions like acute exacerbation of COPD. Not keeping this complication in mind can lead to biased diagnostic reasoning and missed or delayed diagnosis.

Introduction

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia (1). In the past decade, catheter ablation of AF has evolved from an investigational procedure to a frequent therapeutic one (2). Phrenic nerve injury (PNI) is a complication of ablation that pulmonologists should be familiar with, due to its increasing incidence (3). This condition can mimic that of comorbid conditions like congestive heart failure (CHF) and chronic obstructive pulmonary disease (COPD). Hence it is important to develop clinical suspicion of phrenic nerve injury, and correlate onset of symptoms to the ablation, to prevent missed or delayed diagnosis, and to avoid falling prey to availability bias.

Case Report

History of Present Illness: A 77-year-old Caucasian woman with past medical history of heart failure with preserved ejection fraction and mild COPD (GOLD Stage 1), presented with dyspnea and right sided chest discomfort for 7 days. One week ago, she had undergone radiofrequency catheter ablation at the University of New Mexico, for persistent symptomatic AF. After the ablation, she reported dyspnea during PCP and pulmonary office visits, which was attributed to acute exacerbation of heart failure in the setting of COPD. She had been given increasing doses of diuretics which did not relieve her symptoms. A short course of azithromycin and prednisone had also been prescribed for possible acute exacerbation of COPD, but her symptoms had remained unchanged. Review of systems were negative for fever, chills, cough, leg swelling and hemoptysis. She led an active lifestyle, did not require oxygen, and had quit smoking 10 years ago. There was no history of cardio- respiratory diseases in the family.

Physical Examination:

Vitals: Temperature: 97.1°F, Pulse – 88/minute, RR 22/minute, BP – 140/70 mm Hg., Spo2 – 90% in Room air (baseline >95 percent). She appeared to be in mild distress. Baseline dry weight had not increased. She had no clinical signs of heart failure- no peripheral edema, no JVD, no S3, no bibasilar crackles. There were decreased breath sounds in the base of the right lung but no rales or rhonchi. No significant wheezing was heard in any of the lobes of the lungs. No clubbing or cyanosis was noted. The rest of the exam was unremarkable with a normal abdominal, and skin exam. There was no lymphadenopathy.

Laboratory: White blood cell count 10,000/mm³, hemoglobin 11 g/dL, with normal electrolytes, liver function tests and negative troponins. Arterial blood gases on room air showed a pH 7.38, paO₂ of 62 mm Hg, pCO₂ 41 mm Hg and HCO₃⁻ 25.

EKG: negative for signs of ischemia.

Radiography: Chest radiography showed an elevated right diaphragm (Figure 1).

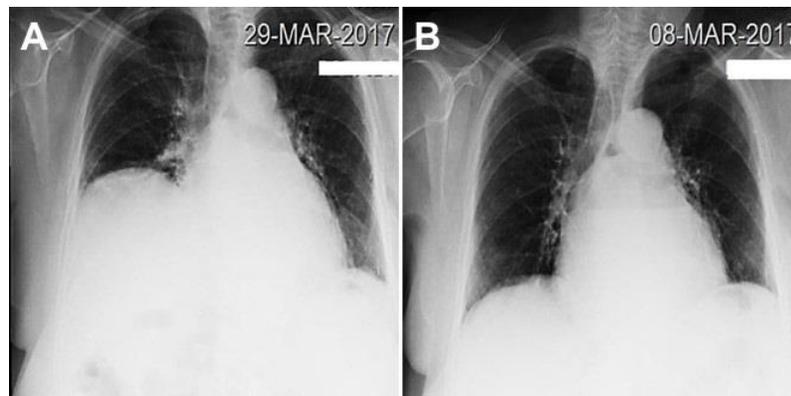


Figure 1. A: PA chest radiograph and B: 3 weeks earlier for comparison.

Sniff test performed under fluoroscopy showed paradoxical elevation of the right hemidiaphragm with inspiration, compared with rapid descent of the left hemidiaphragm, confirming right hemidiaphragm paralysis (Figure 2).

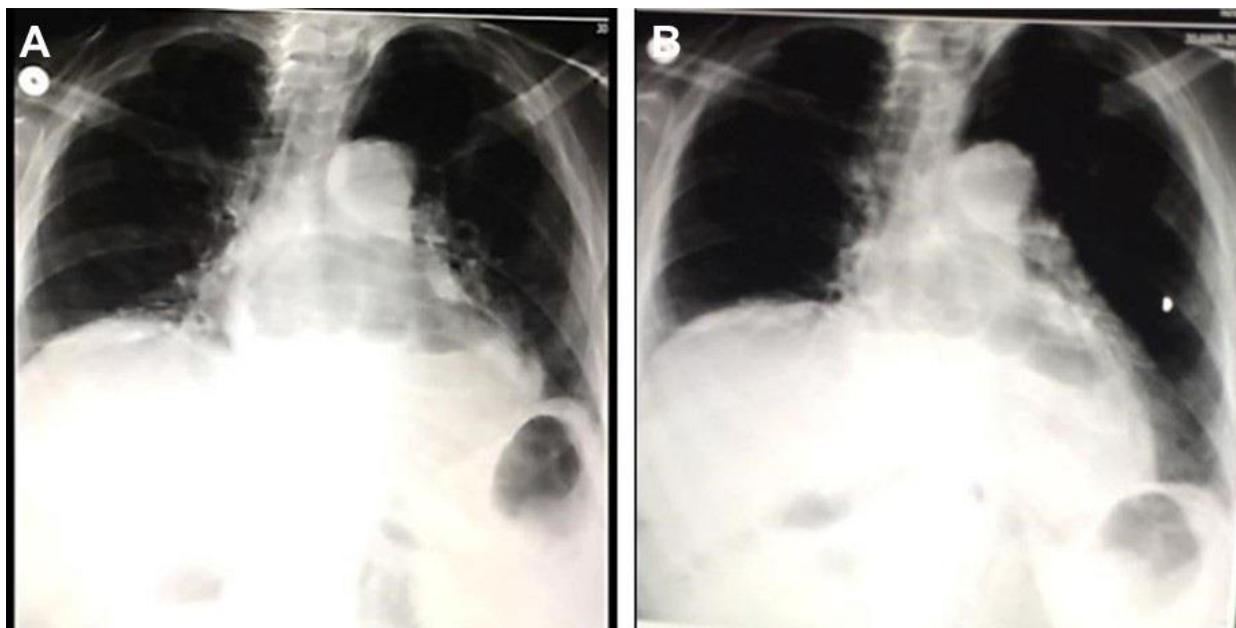


Figure 2. Static images from sniff test under fluoroscopy: A: pre-sniff. B: post-sniff. When the patient sniffs in, the left hemidiaphragm moves downwards but right hemidiaphragm does not (actually moves upwards very slightly).

After obtaining proper imaging, the patient was finally diagnosed with right-sided diaphragmatic paralysis due to phrenic nerve injury from the catheter ablation procedure done to treat AF. She was discharged with home oxygen and her symptoms have resolved. Follow up clinic visits revealed complete resolution of symptoms.

Discussion

Ectopic discharges from pulmonary veins are an important cause of atrial fibrillation, the most common sustained cardiac arrhythmia (1). Calkins *et al.* (4) carried out a study in 2009, where they showed statistically significant improvement in symptoms and quality of life in patients receiving ablation therapy versus those patients who received anti arrhythmic drugs (4). Traditionally, isolating the pulmonary vein by point-by-point radiofrequency catheter ablation was the cornerstone of catheter ablation strategies for the treatment of atrial fibrillation (2). However, this procedure had various complications such as thromboembolism, cardiac perforation, injury to adjacent structures and pulmonary vein stenosis (5). Hence, with the hope of finding an effective alternative approach with less complications, cryothermal ablation was started. This particular procedure involves electrically isolating pulmonary veins, by creating circumferential lesions by means of a cryoballoon catheter (6). Nonetheless,

in both techniques, the most common complication is hemi-diaphragmatic paralysis, due to phrenic nerve injury. This especially occurs whilst trying to isolate the right superior pulmonary vein (3). The approximate incidence of this complication is close to 3–11% (7). It is thought that the phrenic nerve gets injured due to the close anatomic relationship of the phrenic nerve to the heart (Figure 3).

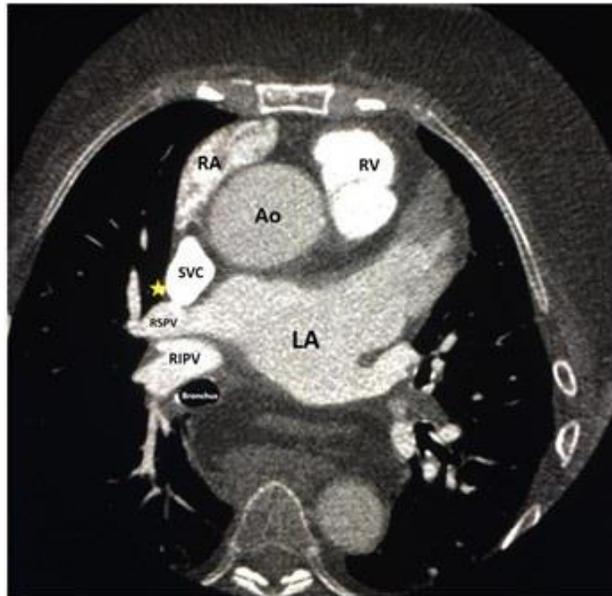


Figure 3. Thoracic CT scan showing anatomical relationships (yellow star is the right phrenic nerve).

Both the right and the left phrenic nerves can get damaged - the right phrenic nerve is specifically at risk when ablations are carried out in the superior caval vein and the right superior pulmonary vein, and the left phrenic nerve is liable to damage during lead implantation into the great cardiac and left obtuse marginal veins (8). In our patient, the right phrenic nerve, which runs along the lateral surfaces of the superior vena cava and right atrium, was injured by energy delivered to the adjacent area during ablation.

In 2005 Bunch *et al.* (9) investigated the specific mechanism of phrenic nerve injury. Their study revealed that the phrenic nerve tended to retain heat after ablation. This phenomenon resulted in higher local temperatures with subsequent energy deliveries, causing early transient injury. Andrade *et al.* (3) in 2014, were the first to define this phrenic nerve injury histopathologically. According to them, phrenic nerve injury consisted of Wallerian degeneration characterized by loss of large myelinated axons with variable degrees of endoneurial edema, vacuolated macrophages, myelin ovoids, and myelin digestion chambers (6).

Phrenic nerve injury can be diagnosed on clinical exam, and via a chest X-ray. Thereafter one can confirm the diagnosis with the sniff test or phrenic nerve stimulation/diaphragm electromyography. An upright chest x-ray will reveal an

elevated diaphragm on the affected side. This test is sensitive, but not specific for the diagnosis of unilateral diaphragmatic paralysis (10). Another frequently done test is the sniff test which shows paradoxical elevation of the paralyzed hemidiaphragm with inspiration, compared with the rapid descent of the normal hemidiaphragm (11). The sniff test has more than 90 percent sensitivity (11). In 2014, Linhart *et al.* (12) performed studies to show that fluoroscopic assessment of diaphragm movement during spontaneous breathing was more sensitive for the diagnosis of phrenic nerve injury as compared to SVC pacing (12). It has also been seen that EMG-guided approach results in less damage to the phrenic nerve and a significant reduction in hemi-diaphragmatic paralysis as compared to current methods of abdominal palpation and fluoroscopy (13).

In unilateral diaphragmatic paralysis, patients are usually asymptomatic, have good prognosis and do not always need treatment. This is specifically true in the absence of underlying lung disease (14). Another procedure often done is the surgical plication of the affected hemidiaphragm (15). In bilateral diaphragm paralysis, ventilatory failure often occurs and these patients may require continuous positive airway pressure or mechanical ventilation and tracheostomy (16). According to Kauffman (17) in 2014, functional restoration of the paralyzed diaphragm should also be part of the standard treatment algorithm in managing symptomatic patients.

Conclusion

Phrenic nerve palsy is a complication which occurs in about 6 percent of cases post catheter ablation procedure for atrial fibrillation. This condition can mimic pulmonary conditions like acute exacerbation of COPD. It is important to develop clinical suspicion and correlate onset of symptoms to the ablation. Not keeping this complication in mind can lead to biased diagnostic reasoning and missed or delayed diagnosis.

References

1. Yamazaki M, Filgueiras-Rama D, Berenfeld O, Kalifa J. Ectopic and reentrant activation patterns in the posterior left atrium during stretch-related atrial fibrillation. *Prog Biophys Mol Biol.* 2012 Oct-Nov;110(2-3):269-77. [\[CrossRef\]](#) [\[PubMed\]](#)
2. Pedrote A, Acosta J, Jauregui-Garrido B, Frutos-Lopez M, Arana-Rueda E. Paroxysmal atrial fibrillation ablation: Achieving permanent pulmonary vein isolation by point-by-point radiofrequency lesions. *World J Cardiol.* 2017 Mar 26;9(3):230-40. [\[CrossRef\]](#) [\[PubMed\]](#)
3. Andrade JG, Dubuc M, Ferreira J, Guerra PG, Landry E, Coulombe N, *et al.* Histopathology of cryoballoon ablation-induced phrenic nerve injury. *J Cardiovasc Electrophysiol.* 2014 Feb;25(2):187-94. [\[CrossRef\]](#) [\[PubMed\]](#)
4. Calkins H, Reynolds MR, Spector P, *et al.* Treatment of atrial fibrillation with antiarrhythmic drugs or radiofrequency ablation: two systematic literature reviews

- and metaanalyses. *Circ Arrhythm Electrophysiol.* 2009 Aug;2(4):349-61. [\[CrossRef\]](#) [\[PubMed\]](#)
5. Sarabanda AV, Bunch TJ, Johnson SB, *et al.* Efficacy and safety of circumferential pulmonary vein isolation using a novel cryothermal balloon ablation system. *J Am Coll Cardiol.* 2005 Nov 15;46(10):1902-12. [\[CrossRef\]](#) [\[PubMed\]](#)
 6. Andrade JG, Khairy P, Guerra PG, *et al.* Efficacy and safety of cryoballoon ablation for atrial fibrillation: a systematic review of published studies. *Heart Rhythm.* 2011 Sep;8(9):1444-51. [\[CrossRef\]](#) [\[PubMed\]](#)
 7. Omran H, Gutleben KJ, Molatta S, *et al.* Second generation cryoballoon ablation for persistent atrial fibrillation: an updated meta-analysis. *Clin Res Cardiol.* 2018 Feb;107(2):182-92. [\[CrossRef\]](#) [\[PubMed\]](#)
 8. Sanchez-Quintana D, Cabrera JA, Climent V, Farre J, Weiglein A, Ho SY. How close are the phrenic nerves to cardiac structures? Implications for cardiac interventionalists. *J Cardiovasc Electrophysiol.* 2005 Mar;16(3):309-13. [\[CrossRef\]](#) [\[PubMed\]](#)
 9. Bunch TJ, Bruce GK, Mahapatra S, *et al.* Mechanisms of phrenic nerve injury during radiofrequency ablation at the pulmonary vein orifice. *J Cardiovasc Electrophysiol.* 2005 Dec;16(12):1318-25. [\[CrossRef\]](#) [\[PubMed\]](#)
 10. Chetta A, Rehman AK, Moxham J, Carr DH, Polkey MI. Chest radiography cannot predict diaphragm function. *Respir Med.* 2005 Jan;99(1):39-44. [\[CrossRef\]](#) [\[PubMed\]](#)
 11. Alexander C. Diaphragm movements and the diagnosis of diaphragmatic paralysis. *Clin Radiol.* 1966 Jan;17(1):79-83. [\[CrossRef\]](#) [\[PubMed\]](#)
 12. Linhart M, Nielson A, Andrie RP, *et al.* Fluoroscopy of spontaneous breathing is more sensitive than phrenic nerve stimulation for detection of right phrenic nerve injury during cryoballoon ablation of atrial fibrillation. *J Cardiovasc Electrophysiol.* 2014 Aug;25(8):859-65. [\[CrossRef\]](#) [\[PubMed\]](#)
 13. Miyazaki S, Ichihara N, Nakamura H, *et al.* Prospective evaluation of electromyography-guided phrenic nerve monitoring during superior vena cava isolation to anticipate phrenic nerve injury. *J Cardiovasc Electrophysiol.* 2016 Apr;27(4):390-5. [\[CrossRef\]](#) [\[PubMed\]](#)
 14. Piehler JM, Pairolero PC, Gracey DR, Bernatz PE. Unexplained diaphragmatic paralysis: a harbinger of malignant disease? *J Thorac Cardiovasc Surg.* 1982 Dec;84(6):861-4. [\[PubMed\]](#)
 15. Kuniyoshi Y, Yamashiro S, Miyagi K, Uezu T, Arakaki K, Koja K. Diaphragmatic plication in adult patients with diaphragm paralysis after cardiac surgery. *Ann Thorac Cardiovasc Surg.* 2004 Jun;10(3):160-6. [\[PubMed\]](#)
 16. Davis J, Goldman M, Loh L, Casson M. Diaphragm function and alveolar hypoventilation. *Q J Med.* 1976 Jan;45(177):87-100. [\[PubMed\]](#)
 17. Kaufman MR, Elkwood AI, Colicchio AR, *et al.* Functional restoration of diaphragmatic paralysis: an evaluation of phrenic nerve reconstruction. *Ann Thorac Surg.* 2014 Jan;97(1):260-6. [\[CrossRef\]](#) [\[PubMed\]](#)